Summary

A case of acute nitrobenzene poisoning is recorded in which there was methaemoglobinaemia and aminoaciduria. Urinary chromatography revealed a transient gross excess of all the normal amino-acids. The literature on the symptomatology and treatment of the condition is reviewed briefly. The patient made a rapid recovery.

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ACUTE TETRACHLORETHANE POISONING

A REPORT ON TWO FATAL CASES

BY

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Cases of acute tetrachlorethane poisoning, following its ingestion by mouth, are unusual. Only three such cases are reported in the English literature (Hepple, 1927; Elliott, 1933; Forbes, 1943). Acute tetrachlorethane poisoning is generally suicidal, its distinctive odour rendering its accidental ingestion unlikely. One case of acute accidental poisoning has, however, been recorded (Hauser, 1939). There is no record of tetrachlorethane having been used as a homicidal agent.

Tetrachlorethane is a colourless, non-inflammable, oily liquid, boiling at 146.3° C., and with a specific gravity of 1.600 at 20° C. It is soluble in fats, oils, resins, and waxes, miscible with alcohol, and soluble in water to the extent of 1 part in 5,000. It has a sweetish smell and taste, somewhat resembling trichlorethylene, carbon tetrachloride, and chloroform, but quite distinct to those who are familiar with it. The commercial preparations have a yellow tinge. As a solvent, tetrachlorethane has many applications in industry, but, owing to its intensely poisonous properties, being the most toxic of the halogenated hydrocarbons (Hamilton, 1934; Browning, 1937), it is falling into disuse. At one time it was issued to the Army as "silk cleansing fluid," and two of the three acute cases recorded in the English literature occurred in soldiers. Its widest application outside industry is in horticulture—as an effective insecticide against white fly. In the two cases described below, the tetrachlorethane was originally purchased as an insecticide.

Chronic tetrachlorethane poisoning has been described and reviewed by a number of authors (Willcox, 1915; Minot and Smith, 1921; Legge, 1934; van Oettingen,

1937; Forbes, 1943). Methods for the early detection of chronic tetrachlorethane poisoning by examination of the blood and urine have been described by Minot and Smith (1921), and Gurney (1943). Toxic jaundice due to tetrachlorethane poisoning became compulsorily notifiable in this country after January 1, 1916, as the result of cases occurring amongst aircraft workers, who used it as "dope" for the treatment of fuselage. Twenty-six cases, five of which were fatal, were notified in 1916, but no further cases were notified until a fatal case occurred in 1929. Although chronic tetrachlorethane poisoning is becoming virtually unknown in this country, cases are still occurring abroad. Seven such cases, one of which was fatal, have been recorded by Coyer (1944).

When taken in large quantities by mouth, tetrachlorethane kills by its narcotic action before recognizable changes can take place in the liver and other organs. Consciousness is rapidly lost, and this is followed by a gradual depression of the vital centres, death occurring some 10 to 20 hours after ingestion. In these cases, except for the presence and smell of tetrachlorethane, the necropsy findings are those of narcotic poisoning, with changes in the gastric mucosa depending on the quantity taken and the presence or otherwise of food. In the two cases to be described the tetrachlorethane had been deliberately taken by mouth.

Case 1

A gardener aged 57 had become depressed because the estate on which he had been employed for thirty years was about to change hands. He left home one day as usual at 7 a.m., taking his sandwiches with him. As his mother, with whom he lived, was often in bed before he returned home in the evening, he was not missed until the next morning. At about 10 a.m. on that day he was found dead on the floor of his potting-shed; an uncorked bottle of tetrachlorethane lay by his side and a short farewell note was found scrawled in chalk across his work-bench.

Necropsy was carried out the next morning. A strong smell of tetrachlorethane was apparent on entering the mortuary. The body was that of a well-nourished man. There was an intense cyanosis of the post-mortem livid stains. Anoxic haemorrhages were present in the scalp.

There was no corrosion of the mouth, lips, or tongue. The lower third of the oesophagus was congested, and early superficial necrosis of the mucosa was present. At the cardiac end of the stomach extreme congestion of the entire gastric mucosa was found, with superficial necrosis and frank erosion. At the pyloric end of the stomach the exposed ridges of the rugae were principally affected. From the stomach and the first half of the duodenum 15 oz. (425 ml.) of tetrachlorethane was recovered. The tetrachlorethane had not passed beyond this point. The stomach also contained a further 5 oz. (142 ml.) of fluid consisting mainly of mucus. Food was not present.

Numerous anoxic haemorrhages were scattered throughout the brain, and there was early basal collapse of both lungs. No material natural disease was present in other organs.

Case 2

A carpenter aged 20, recently discharged from the R.A.F., was depressed over an error he had made at work. On the day before his death he returned home as usual for lunch. He had a good meal and left the house at 1.20 p.m. to return to work. At 2.30 p.m. it was noticed that his bicycle was still in the garden, and almost immediately afterwards he was found unconscious in a wheel-barrow which was standing close to a greenhouse. Later a medicine bottle which contained tetrachlorethane was found in the greenhouse, with approximately 1 to 2 oz. (28 to 57 ml.) missing.

He was admitted to hospital at 4.15 p.m., where he was found to be unconscious; his limbs were flaccid and all reflexes were absent. His pupils were contracted and unreactive. He was cyanosed. His pulse was good and the blood pressure was 140/90. There was a sweet smell in his breath which was thought to be due to trichlorethylene. He was given oxygen and admitted to a ward, where his stomach was washed out with water. Three and a half litres of stomach washings were retained. During the wash-out he vomited the whole time, but was prevented from inhaling the vomit by tipping. The wash-out was stopped as his breathing was becoming weaker. Eventually breathing stopped completely. An intratracheal tube was passed, and artificial respiration was carried out with Boyle's apparatus. His heart then stopped beating, but recommenced after an injection of nikethamide. Breathing began naturally after two injections of 12 mg. of picrotoxin, but the respiratory excursion remained very poor in spite of two further injections that day. His condition deteriorated during the night and his heart again stopped, but started spontaneously. Later his breathing stopped and picrotoxin had no further effect. Death occurred at 5.30 a.m. the following day—about fifteen hours after he had ingested the poison.

Necropsy was carried out on the same day. A distinct smell of tetrachlorethane was present at the lips. A fine froth was found in the mouth and issuing from between the lips. There was an intense cyanosis of the post-mortem livid stains.

The brain was oedematous and congested, and a number of small anoxic haemorrhages were scattered throughout the white matter. Both pleural cavities contained a small clear exudate. The air passages were filled with copious frothy oedema fluid, and the lower lobes of both lungs were virtually airless as a result of extreme congestion and oedema. There was a small clear pericardial effusion. Numerous anoxic petechial haemorrhages were present on the epicardial and endocardial surfaces of the heart, and were most noticeable on the interventricular septum. No natural disease of the cardiovascular system was found.

There was no corrosion of the mouth or throat. The lower end of the oesophagus was congested. The stomach contained 440 ml. of fluid, consisting of water, mucus, a little undigested food, and free tetrachlorethane. Pronounced congestion of the gastric mucosa was present, affecting principally the ridges of the rugae. There was no necrosis, and the gross appearances resembled nicotine poisoning. A small blood-tinged peritoneal exudate was present, and congestion of the duodenum and upper jejunum. The other organs were normal, as was the histology of the liver and kidneys.

Free tetrachlorethane was recovered from the alimentary tract as follows:—Stomach: total contents, 440 ml.; free tetrachlorethane, 23 ml. Duodenum: total contents, 115 ml.; free tetrachlorethane, 21.5 ml. Small intestine (jejunum and upper ileum): total contents, 550 ml.; free tetrachlorethane, 4 ml. There was no free tetrachlorethane in the 3,500 ml. of stomach washings retained by the hospital.

Discussion

Acute and chronic tetrachlorethane poisoning are two distinct entities. In the latter, tetrachlorethane acts on the liver, heart, kidneys, and haemopoietic system, differing from the other halogenated hydrocarbons only by its higher toxicity, death resulting from liver necrosis a few weeks after the onset of symptoms. In its acute form of poisoning tetrachlorethane behaves as a narcotic, death usually occurring some 10 to 20 hours after its ingestion. Consciousness, however, seems to be lost early. The interval between ingestion and death, and the appearances of the gastric mucosa, depend on the quantity taken and the presence or otherwise of food in the stomach. In Case 1 it would seem that death occurred with extreme rapidity after the

ingestion of a very large volume of tetrachlorethane taken on an empty stomach. In Case 2 a much smaller quantity of tetrachlorethane was taken immediately after a large meal, and, although consciousness appeared to have been rapidly lost, death did not occur for some 15 to 16 hours. Vigorous resuscitative measures undoubtedly prolonged life in this case. It is quite clear from Case 2 that, owing to the insolubility of tetrachlorethane in water and its very high specific gravity, it is unlikely to be satisfactorily evacuated from the stomach by the normal methods of lavage.

Summary

Two cases of acute fatal tetrachlorethane poisoning have been described. The differences between acute and chronic tetrachlorethane poisoning are discussed.

Ordinary methods of gastric lavage are unlikely to be satisfactory.

I wish to thank H.M. coroner, West Surrey, for permission to publish these two cases.

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ANAL FISTULA WITH EXTENSION TO THE THIGH

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A case of chronic ulcerative colitis is presented in which a fistula extended from the anal canal through the space between the sacro-spinous and the sacro-tuberosus ligaments and then along the obturator internus tendon to form an abscess in the thigh.

Case Report

A man now aged 37 first complained of diarrhoea and the passage of blood and mucus per rectum in 1938. In 1939, following a haemorrhoidectomy and excision of a fistula-in-ano, the diarrhoea increased in severity, and he also began to pass pus per rectum. In 1940 signs and symptoms of an anal stricture developed, and for its relief a caecostomy was performed. Later that year he also developed a vesico-colic fistula, and his weight fell to 6 stone (38.1 kg.). A transverse colostomy was then constructed and the caecostomy closed by operation. Since 1940 the transverse colostomy had acted many times a day; and during that time he had complained of pain in the region of the right hip.

On April 26, 1949, he was first seen by me at Sydney Hospital. He stated that he had had more severe pain over the right hip for the previous three months, and for six months had noticed a swelling over the right greater trochanter.

On examination his general condition was fairly satisfactory, his weight was 9 stone (57.2 kg.), and his height 5 ft. 7 in. (170 cm.). The abdomen was lax and not tender,