temporary porphyrinuria after ethanol administration. I feel that my patient manifested Zieve's syndrome and a variety of secondary acquired porphyria with abdominal pain, paraesthesia, and porphyrinuria, and that this may be responsible for the abdominal pain in Zieve's syndrome. Clearly future patients with Zieve's syndrome need to be investigated for abnormalities of porphyrin metabolism.

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Home-made Cider: Source of Lead-poisoning

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Lead-poisoning is an industrial disease reportable under the Factories Act, 1961. Because of the Public Health Act, 1936, there is little chance of commercially prepared food and drink or public water supply containing lead in appreciable quantities, and of course certain factories in Great Britain are subject to regulations governing the use of lead and lead-containing materials. There are no regulations, however, governing such domestic use of lead as is here described.

CASE REPORT

A 49-year-old man was admitted to hospital in April 1968 with severe epigastric pain of 24 hours' duration. The pain was constant, did not radiate, and was becoming progressively worse. He had vomited several times, but had had no diarrhoea. For five years before admission he had been having similar attacks of abdominal pain; he thought he had been slower mentally since Christmas 1967, and he had noticed that a tremor had developed in his hands.

He was a slight pale man, but was not clinically shocked; his pulse was regular, 100/min., blood pressure 140/85, temperature 36.6° C. He was writhing in bed and appeared to have considerable epigastric pain. Rectal examination showed nothing abnormal and there was no evidence of intestinal obstruction either clinically or on x-ray examination. His haemoglobin was 56%, the packed cell volume 25%, and there was basophilic stippling of the red cells. Closer examination showed a dark blue line round the gingival margins. It was thought that the patient might be suffering from plumbism.

There was no radiological evidence of lead-poisoning in the skeleton, red cell fragility and plasma electrolytes and proteins were normal. Routine urinary analysis showed no protein, sugar, or acetone, and the creatinine clearance was within the limit of normal; however, qualitative urinary analysis showed porphyrins to be grossly in excess of normal. Blood and urine samples sent to the department of pathology at the London Hospital for lead estimation gave these results: blood lead level 375 µg./100 ml. (normal 60–80 µg./100 ml.) and urinary lead 260 µg./100 ml. (normal 8–12 µg./100 ml.).

On questioning, the patient stated that he drank about a quart (1.1 litres) of his own home-brewed cider each day, and that he had done so for about 30 years. During the process of making the cider, apple juice was poured on to a lead-lined tray at the base of the press. Here presumably was the source of the poison.

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A diagnosis of lead-poisoning was made and treatment instituted. This consisted in giving 2 g. of sodium calciumedetate each day intravenously. Seven doses were given. By the end of treatment his blood lead level had fallen to 55 µg./100 ml., and his urinary lead to 12 µg./100 ml. At this stage his haemoglobin had risen to 69%, he felt very much better, the tremor in his hands had disappeared, and he seemed to be sharper mentally.

Six weeks later the following figures were obtained: blood 12 µg./100 ml., urine 13.5 µg./100 ml. He had, of course, been counselled to drink no more of his own brew.

DISCUSSION

Beer and cider drawn through lead pipes, wine from lead-glazed earthenware pots, and soft or acidic water allowed to stand for any length of time in contact with lead pipes or cisterns have all been reported as causing lead-poisoning. The press which had been used to make our patient's cider was 120 years old and made of oak. Over the years the collecting-tray at the bottom of the press had shrunk, and so a watertight lead covering had been put on some 30 years ago to prevent loss of the apple juice through the cracks. While the slightly acidic juice was in contact with the tray a certain amount of lead was dissolved, as evidenced by a concentration of 5.95 mg. of lead in each litre of cider. This is considerably less than the lead content in home-made wine (120 mg./l.) reported by Lane and Lawrence (1961) from this hospital, although their patient had been drinking the wine for only four months.

Both our patient and his father had drunk the cider, and it is interesting to note that the latter had been admitted to hospital in 1948 and 1958 with acute abdominal pain and had had laparotomies on both occasions. Nothing pathological had been found at either operation, but in 1958 his blood film had shown basophilic stippling. Unfortunately, no investigations could be carried out on him, since he died in 1966 of an unrelated illness. We must assume that he too was suffering from chronic lead-poisoning and that this had been the cause of his abdominal pain.

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