community, which correlates well with the national population statistics but not with the previously estimated numbers.

It would seem that as far as the Wadoma of the Kenyemba area are concerned this deformity is one affecting a particular family as opposed to the tribe itself. Since only the males of one generation appear to be affected, it is possible that this is an example of the rarer autosomal recessive type of inheritance. X-linked inheritance, with Mr Maburani's mother as gene carrier, is also possible (Fig. 3). On the other hand the reports emanating from Botswana would seem to indicate the more common autosomal dominant type of inheritance but this has not yet been properly investigated.

REFERENCES

Pancreatic calcification not due to alcohol consumption
A case report

C. M. STEIN

Summary
An 18-year-old woman presented with diabetes, pancreatic calcification and evidence of malabsorption. The features of calcific pancreatitis unrelated to alcohol consumption are discussed.

Pancreatic calcification in Africa has been well documented; in many countries the finding thereof implicates alcohol consumption as the underlying cause. Other conditions, such as hyperparathyroidism, hereditary pancreatitis and biliary tract disease, are extremely uncommon causes of calcific pancreatitis. Even when a history of excessive alcohol consumption is not obtained, it is difficult to exclude this with any certainty, and most cases of calcific pancreatitis in which the cause is not determined are presumed to be due to alcohol. However, there is a well-defined group of patients peculiar to underdeveloped countries in whom alcohol consumption can definitely be excluded as the underlying cause.

Case report
An 18-year-old Black Zimbabwean woman from the Mutare area was first admitted to Parirenyatwa Hospital, Harare, in December 1981 with a 3-year history of weight loss, secondary amenorrhoea and frequent passage of bulky, pale, offensive stools. Direct questioning elicited a story of acute episodes of epigastric pain radiating through to the back, lasting several days and occurring at about 3-monthly intervals for 3 years. The patient's medical history was not significant, except that she may have had mumps as a child. She was single and unemployed, was living in the communal areas and had never drunk any alcohol. Her diet was unremarkable except that the family occasionally ate cassava which they cultivated on a small scale.

On examination she looked ill, was malnourished and weighed only 34 kg. Apart from bilateral parotid gland enlargement no other abnormalities were found. A provisional diagnosis of malabsorption/tropical sprue was made and appropriate investigations were undertaken. Routine urinalysis showed glycosuria. A random blood glucose value was 24 mmol/l. A full blood count, blood urea and electrolyte, calcium, phosphate, cholesterol and triglyceride levels and thyroid function were all normal. The chest radiograph was normal and the Heaf test was negative. The serum albumin level was 32 g/l and the total protein level 64 g/l, while other liver function tests gave normal results. The condition of the patient was stabilized by the administration of 40 U soluble insulin twice daily.

A 3-day faecal fat collection showed a loss of 36 g/24 h (normal 4 - 6 g/24 h). A D-xylose test gave a negative result. A plain radiograph of the abdomen showed gross pancreatic calcification, and the lateral view confirmed this. At this point the patient insisted on being discharged from hospital and, unfortunately, was lost to follow-up. She was readmitted 5 months later via Mutare Hospital with a blood glucose level of 43 mmol/l, having received no treatment for several months. Her diabetes was again controlled by the administration of large doses of soluble insulin and education about diet. On this occasion the 24-hour faecal fat excretion was 8 g/24 h. An oral cholecystogram was normal. When the diabetes had been reasonably controlled the patient was transferred back to Mutare Hospital for further management.

Department of Clinical Pharmacology, University of Zimbabwe, Harare, Zimbabwe

C. M. STEIN, M.B. CH.B., M.R.C.P., Lecturer
Discussion

Pancreatic calcification is not uncommon in Black Africans. In Kampala, Uganda, Shaper found pancreatic calcification at 2 of 500 consecutively performed autopsies and in 8% of patients attending a clinic for diabetics. In developed countries with adequate protein intake, alcohol consumption correlates well with the incidence of calcific pancreatitis. This condition is found in patients of approximately 40 years of age with a daily alcohol intake of 150 ml or more. The type of alcohol does not seem to be of importance, but a diet rich in fat and protein does seem to be linked to the disease. An accurate history of alcohol intake is notoriously difficult to obtain. In adults with pancreatic calcification alcohol consumption is the suspected cause even when no history of excessive alcohol intake is obtained. Many of the cases reported from Africa and other underdeveloped countries do not fit into this category and seem to form a well-defined separate group.

In 1955 Hugh-Jones described a new clinical presentation of diabetes which he termed 'J-type' or Jamaican diabetes; these patients were young and thin, not prone to ketosis, and seemed to require unusually large doses of insulin. Zuidema linked J-type diabetes with pancreatic calcification in 16 patients, the majority of whom were less than 30 years old; alcohol could not be implicated as the underlying cause. No quantitative fat excretion studies were performed, but exocrine pancreatic function did not seem to be markedly disturbed. He also suggested that the emphasis should be placed not on a type of tropical diabetes but on the underlying pancreatic disease which was probably caused by malnutrition.

Several other reports outlining a similar clinical picture followed. Kinnear from Nigeria reported pancreatic calcification in 30 out of 266 diabetic patients. The majority of these patients were less than 30 years old; again, alcohol did not seem to be a factor and many needed more than 80 U insulin daily. All of the 10 patients whose faecal fat excretion was measured were losing more than 10 g/24 h. Olurin and Olurin, again from Nigeria, described 37 patients with diabetes and pancreatic calcification. The majority were less than 20 years of age and needed large doses of insulin; half of them had a history of chronic abdominal pain, while evidence of malabsorption was found in one-third. An interesting autopsy finding in 1 case was schistosomiasis.

The South Indian state of Kerala has the highest incidence of calcific pancreatitis in the world — 10 times that in Marseilles, which has the second highest incidence. In 5 years Pitchumoni saw more than 400 patients in Kerala with diabetes and pancreatic calcification. Most of these patients were 16-20 years of age, had a history of chronic upper abdominal pain and were found to be emaciated with enlarged parotid glands. Alcohol and biliary tract disease did not play a role. A curious sign found in many of his patients was a blue tinge to the lips.

Very few similar cases have been reported south of the Zambezi. Joffe, from Johannesburg, described pancreatic calcification in a 12-year-old Black boy who was not diabetic but did have steatorrhoea. Prinsloo (Pretoria) reported pancreatic insufficiency masquerading as kwashiorkor in a 5-year-old Black boy. This patient had pancreatic calcification but was not diabetic, nor was his faecal fat loss abnormal. Wicks, looking for pancreatic calcification in Zimbabwe, described pancreatic calcification with diabetes and malabsorption in a 12-year-old schoolgirl.

The aetiology of this unusual form of pancreatic calcification has been widely debated. Its occurrence in underdeveloped countries raised the possibility that malnutrition was the cause, since malnutrition can certainly affect the pancreas. Davies described the disappearance of acinar tissue and islet involvement in advanced cases of kwashiorkor. Barbezat showed that patients recovering from acute malnutrition seemed to regain normal exocrine pancreatic function, whereas those with chronic malnutrition did not recover normal pancreatic function even after prolonged dietary therapy. Malnutrition is widespread but pancreatic calcification is localized to certain geographical areas. A local toxic cause, possibly cassava consumption, was postulated. The root of this plant (Manihot esculenta) supplies 10% of global caloric needs. A geographical analysis of dietary cyanide ingestion and pancreatic calcification with diabetes supports the hypothesis that a combination of cyanide ingestion and a low-protein diet may be the cause of this clinical syndrome. In animal experiments temporary diabetes has been induced in rats by administering cyanide. The cyanogenetic glycosides in cassava are concentrated in the integument of the tubers and are thought to be responsible for its toxic features.

*Manihot esculenta* is grown in Zimbabwe, but usually only on a small scale by individual families to vary the diet and provide a reserve source of food in times of drought. There are no figures available which accurately document cassava cultivation in Zimbabwe, but it is not a staple food or widely cultivated (Mr A. Rowe, Department of Crop Science, University of Zimbabwe — personal communication). This may account for the relative infrequency of calcific pancreatitis unrelated to alcohol consumption in Zimbabwe.

REFERENCES