MYOCARDIAL INFARCTION IN THE AFRICAN

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Myocardial infarction is one of the most important single causes of death among most White populations including that of Southern Rhodesia where, according to death certificates, the disease accounted for 15% of all White deaths during 1962. By contrast it is generally agreed that it is rare in the indigenous populations of underdeveloped countries, and reports certainly indicate that this is so in the southern half of Africa. It may, therefore, be of interest to report a case of myocardial infarction—the first clinically diagnosed in an African at Harare hospital, though 3 have been found at autopsy in Salisbury during the last 4 years.[1, 2]

In 1958 a 'possible case of coronary insufficiency' was reported in an African female by Gelfand and Kaplan,[3] but electrocardiographic proof of infarction was not obtained. At that time they predicted that within the next few years many more cases of coronary infarction would be published. It is perhaps surprising that no cases have been reported in Salisbury until now. In Bulawayo Baldachin[4] reviewed 564 cases of cardiovascular disease admitted to the Memorial Hospital and later to Mpilo Hospital during the period September 1957—September 1960 and only 1 was thought to have 'possible myocardial infarction'.

Singh[5] drew attention to the rarity of coronary heart disease among Africans in East Africa and reported a case, while Patel[6] recorded that he had seen only 2 proved cases of coronary thrombosis in Uganda Africans in 23 years of medical practice.

In South Africa a similar pattern is found. In a series of 1,000 consecutive African patients over 8 years of age admitted to Edendale Hospital in Natal with heart disease from June 1959 to March 1961, Cosnett[7] found only 6 cases of myocardial infarction, 3 of which were diagnosed at necropsy. In Cape Town Bronte-Stewart[8] reported only one authenticated case of ischaemic heart disease in the Bantu in the 5-year-period preceding his paper, while an analysis of ECGs of 9,507 patients attending Groote Schuur Hospital during the years 1958 and 1959 showed only 3 cases of definite myocardial infarction in Bantu patients.[9] The largest series of cases comes from Johannesburg, where Seftel and his associates[10] conducted an intensive search through the clinical and pathological records of Baragwanath Hospital for the period 1951 - 1961 and discovered 30 cases (27 males and 3 females) of proved myocardial infarction in Bantu patients.

CASE REPORT

Jonas, an African male aged about 55 years, was born in Tete, Portuguese East Africa and came to Southern Rhodesia in 1957. At the time of admission to hospital he was living with his wife and 3 children in Mufakose township and was working in Salisbury as a builder's labourer. His diet was of the usual African type, low in animal protein and fat:

Breakfast — tea with milk and sugar, bread with margarine or jam.
Lunch — usually nothing; occasionally a piece of dry bread.
Supper — mealie meal, cabbage and meat.

He never ate eggs; he said he had given up alcohol 7 years previously but drank about 1 bottle of 'coca-cola' daily. He was a non-smoker.

At 8 a.m. on the day of admission he was lifting a heavy cement block when he experienced a retrosternal 'heavy feeling' which went through to between his shoulder blades. He continued working, and the feeling persisted and gradually became worse until at 3 p.m. it became a very severe pain, which radiated down to the epigastrium but not up into the neck nor down the arms. At this time also he sweated pro-

fusely, felt nauseous and vomited and became short of breath. He had never had chest pain previously.

On admission the patient was obviously distressed but not shocked. He had a pulse rate of 55/min. and a BP of 170/100 mm.Hg. There was very slight epigastric tenderness, but no other abnormal physical signs were found and the heart sounds were normal.

ECG (Fig. 1) showed the changes of a large anterior myocardial infarct with pathologic Q waves, elevated ST segments and inverted T waves. Serum lactic dehydrogenase, estimated

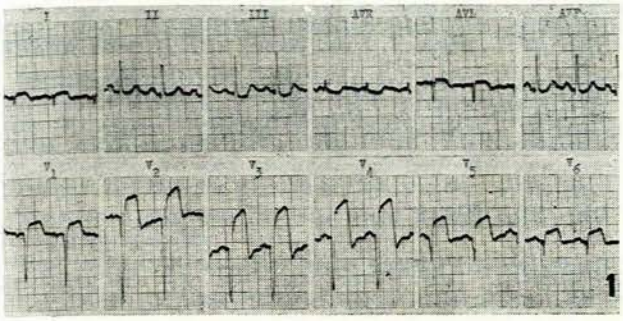

Fig. 1. ECG showing extensive anterior myocardial infarction.

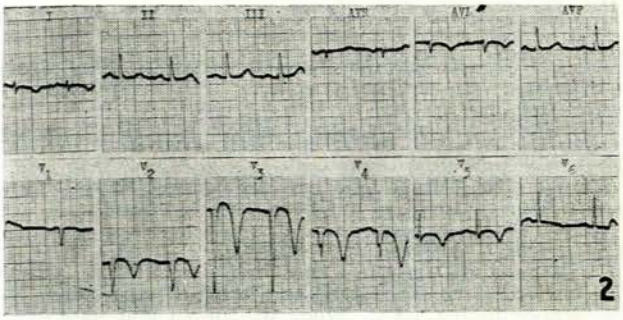

Fig. 2. Repeat ECG, taken after 5 weeks.

3 days after the initial episode, was 1,320 units. Other investigations included:

X-ray examination of chest	—	lung fields clear, heart size and shape within normal limits
Blood count: Haemoglobin	—	14·0 G/100 ml
WBC	—	9,700 cu.mm. with a normal differential count
ESR	—	15 mm. after 1 hour (Westergen)
Blood Wasserman reaction	—	negative
Blood urea	—	20 mg./100 ml
Serum cholesterol	—	155 mg./100 ml
Liver-function tests	—	normal
Serum proteins: Total	—	6·2 G/100 ml
Albumin	—	3·1 G/100 ml
Globulin	—	3·1 G/100 ml
Urine examination		no abnormality
Stool examination		no ova

The patient's stay in hospital was uneventful. Slight retrosternal discomfort persisted for about 36 hours after admission, but he was symptom-free and felt perfectly well thereafter. Anticoagulant therapy was discontinued after 4 weeks, and the patient was discharged after 6 weeks, arrangements having been made for him to do lighter work. An ECG taken before discharge (Fig. 2) shows the progression in ST segment and T-wave changes that occurred over 5 weeks.

DISCUSSION

The marked difference in the incidence of myocardial infarction between European and African population groups is of considerable interest. Gillman and his associates[11] suggested that the severe liver disease so widespread among Africans in South Africa might be of some importance in protecting them against coronary thrombosis, while Charters and Charters[12] drew attention to the tendency to an inverse ratio between the geographical incidence of liver cirrhosis and coronary heart disease. They suggested that the nutritional and post-necrotic varieties of cirrhosis, common among Africans, give better protection than the conspicuously fatty type seen in the USA and in White South Africans.

The racial difference in the incidence of the disease has aroused speculation as to whether the rapid socio-economic progress of the African will be accompanied by a correspondingly rapid increase in the incidence of myocardial infarction. The findings of Seftel *et al.*,[10] in their study of 30 Bantu subjects with proved myocardial infarction, support the view that Western modes of living, especially when associated with certain conditions common among Western communities such as diabetes, hypertension and obesity, are important factors in the pathogenesis of heart disease. By comparison with control groups their Bantu patients with infarction showed a higher incidence of hypertension, diabetes and obesity, were more advanced socio-economically and less active physically, ate more animal protein and fat, and had higher blood lipids. Furthermore, the increasing incidence of cases diagnosed during the 11-year-period of their review (12 cases in the first 7 years and 18 in the last 4) suggests that myocardial infarction, although rare, may be increasing. Outside of Africa similar findings have been reported. Keys[13] has shown that while the incidence of myocardial infarction is low among the Japanese in Japan, it steadily rises as they become progressively Westernized.

One might, therefore, expect the occasional case of myocardial infarction encountered in an African to involve one of the more sophisticated members of the community—perhaps a prosperous business or professional man who has largely adopted the European diet and way of life, who is overworried and overweight and who has a high blood cholesterol and atherosclerotic arteries. That this is not always the type afflicted is illustrated by the case reported here, and by a similar case reported by Singh[5]—a 60-year-old Gisu male of low socio-economic status whose diet contained little fat and whose serum cholesterol was 145 mg./100 ml. Furthermore, of Cosnett's 3 cases which came to necropsy,[7] only 1 had a moderate degree of atherosclerosis. Serum cholesterol was estimated in 4 of his 6 cases, and in none was it raised. An interesting suggestion with regard to pathogenesis in cases such as these was put forward by Seftel *et al.*[10] In their study of 15 Bantu cases of myocardial infarction coming to autopsy they found that in those patients who had diets low in animal fat and protein, were of low socio-economic status, relatively active physically, and who had low or normal blood lipids, the coronary occlusive lesion was predominantly thrombotic rather than atherosclerotic. Such patients still, of course, constitute the great majority of our hospital admissions.

The association between myocardial infarction and atherosclerosis is well established, and it is not surprising that the incidence and severity of coronary atherosclerosis are also lower among Africans than among Whites. Sacks[14]

examined the aortas and coronary arteries from 1,251 unselected adult autopsies carried out in Cape Town. Only 5% of Bantu males in the age group 40 - 59 years showed severe coronary atheroma compared with 37% in White males. Of these males none of the Bantu had severe lumenal narrowing, whereas 24% of the Whites did. Marked differences were also found in the older age groups.

The African inhabitants of Rhodesia and neighbouring territories still enjoy almost complete freedom from serious coronary heart disease. It seems safe to predict, however, that as their way of life changes towards that of the European, so will the incidence of atherosclerosis increase, with an accompanying rise in the incidence of myocardial infarction.

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