The African Journal of MEDICAL SCIENCES

Volume 3

1972

BLACKWELL SCIENTIFIC PUBLICATIONS
Oxford London Edinburgh Melbourne

Afr. J. med. Sci. (1972) **3**, 1–12.

The Pattern of Acquired Heart Disease in Nigerian Children

A. U. Antia, C. E. Effiong and A. H. Dawodu

Department of Paediatrics, University College Hospital, Ibadan, Nigeria

(*Received* 15 *October* 1970)

Summary. The results of a prospective study of acquired heart disease in Nigerian children from birth to age 5 years for a period of 4½ years are presented. The three common acquired heart disorders were rheumatic heart disease, idiopathic cardiomegaly and endomyocardial fibrosis. The less common disorders included tuberculous and pyogenic myopericarditis and left ventricular submitral aneurysm. Although heart failure was present in all these disorders, it was possible to make accurate diagnosis based on the clinical features, chest radiographs and electrocardiograms.

Résumé. On présente les résultats d'une étude prospective sur les maladies du coeur acquises chez des enfants nigérians depuis la naissance jusqu'à l'âge de 5 ans pendant une période de quatre ans et demi. Les trois cardiopathies acquises les plus répandues étaient le rhumatisme cardiaque, la cardiomégalie idiopathique et la fibrose endomyocardiale. Parmi les désordres moins répandus on a remarqué la myopéricardite tuberculeuse et pyogénique et l'anévrisme submitral du ventricule gauche. Bienque l'insuffisance cardiaque fût présente dans tous les désordres, il était possible de construire un diagnostic exact fondé sur examens cliniques, radiogrammes thoraciques et électrocardiogrammes.

A number of previous studies in the tropics on acquired heart disease have concentrated on adults alone and sometimes on adults and children together (Beet, 1956; Abrahams, 1959; Halim & Jacques, 1961; Binder, 1961; Miller, Spencer & White, 1962; Harling, Marsden & Ridley, 1965; Rowland, 1965; D'Arbela, Kanyerezi & Tulloch, 1966). In some of those studies which included adults and children, the latter constituted a considerable proportion of the subjects, while in others no attempt was made to show the proportion of children in the series. Such studies which group children and adults together may inevitably obscure the true incidence of the disease in these two age groups.

Certain important differences exist in the clinical features of some of the tropical cardiomyopathies between children and adults and it is possible that such differences might provide significant clues to the aetiology and pathogenesis of these diseases. For example, we

Correspondence: Professor A. U. Antia, Department of Paediatrics, University College Hospital, Ibadan, Nigeria.

have shown that in idiopathic cardiomegaly in children there are some features which differ from those in adults with the same disease. Because of such differences in the two age groups we have suggested that the aetiological factors in children may not be the same as in adults (Antia, Cockshott & Thorpe, 1969). In this communication we are presenting the results of a prospective study of acquired heart disease in Nigerian children over a period of 4½ years.

SUBJECTS AND METHODS

There has been a cardiac register in the Department of Paediatrics, University College Hospital (UCH), Ibadan, since July 1966. All types of heart disease in children from birth to the age of 12 years are entered into this register. However, occasionally children over the age of 12 years have also been entered into the register. The present study includes those cases of heart disease entered into this register between July 1966 and February 1970. All cases of heart disease secondary to anaemia, renal disorders, hypertension and tumours have been excluded from the study.

The diagnosis in each case was made clinically including chest X-rays and electrocardiogram (ECG). In some cases the diagnosis was confirmed by autopsy and by cardiac catheterization and angiocardiography. Haemoglobin, white blood count and differentials, and haemoglobin electrophoresis (genotype) were carried out routinely on every patient. Anti-streptolysin-O titre (ASO titre), throat swabs, serum electrolytes, and proteins in the serum and serous fluids were undertaken whenever the test was indicated.

RESULTS

In the 4½-year period of the study, seventy-two patients with acquired heart disease were entered into the register. The cases consisted of rheumatic heart disease (thirty-one), idiopathic cardiomegaly (nineteen), endomyocardial fibrosis (fifteen), tuberculous myopericarditis (three), pyogenic myopericarditis (two) and left ventricular submitral aneurysm (two). Thus, rheumatic heart disease, idiopathic cardiomegaly and endomyocardial fibrosis were the three common acquired heart disorders in this study. The different types of the disorders and their frequency are shown in Table 1.

The age and sex distribution of the three common conditions are presented in Table 2. There was no significant difference in the sexes among patients with rheumatic heart disease (fifteen males and sixteen females). There was, however, a preponderance of males over females among patients with idiopathic cardiomegaly (twelve males and six females) and those with endomyocardial fibrosis (ten males and five females). The ages of patients with rheumatic heart disease ranged between 5 and 11 years (mean 7·8 years); of patients with idiopathic cardiomegaly the ages ranged between 10 months and 15 years (mean 4·5 years) and of those with endomyocardial fibrosis the ages ranged between 4 and 14 years, with a mean of 8 years. The three patients with tuberculous myopericarditis were males, aged 3, 6 and 8 years respectively. The two patients with pyogenic myopericarditis were a male and a female infant, and the two with left ventricular submitral aneurysm were females aged 7 and 10 years respectively.

Endomyocardial fibrosis

The clinical diagnosis of this condition was confirmed by angiocardiography in seven patients and by autopsy in two others. All the fifteen patients lived in widely scattered parts

TABLE 1. Types of acquired heart disease in seventy-two Nigerian children

Type	No. of cases
Rheumatic heart disease:	31
(a) Acute	5
(b) Chronic	26
Idiopathic cardiomegaly	19
Endomyocardial fibrosis:	15
(a) Right ventricular	11
(b) Biventricular	4
Miscellaneous conditions:	7
(a) Tuberculous myopericarditis	3
(b) Pyogenic myopericarditis	2
(c) Left ventricular submitral aneurysm	2
Total	72

TABLE 2. Age and sex distribution in children with rheumatic fever, idiopathic cardiomegaly and endomyocardial fibrosis

Type of heart disease	No. of cases	Sex		Age range (yr)	Mean age (yr)
		Male	Female		
Rheumatic heart disease	31	15	16	5–11	7·8
Idiopathic cardiomegaly	19	13	6	10/12–15	4·5
Endomyocardial fibrosis	15	10	5	4–14	8

of the Western State of Nigeria (within 20 miles of Ibadan, six patients; Ondo, two; Ilesha, two; Ijebu-Ode, three; Epe, one; Eruwa, one). There was therefore no concentration of the cases in any particular part of the State. All the children were from low socio-economic families.

The various symptoms and their frequency in the fifteen patients are presented in Table 3. The onset of symptoms was insidious in every case. The duration of the illness before hospitalization varied between 4 weeks and 2 years. The patient with the shortest duration of symptoms had cough as the main complaint and she was referred from the local tuberculosis clinic where she had been for medical attention because of persistent cough thought to be of tuberculous origin. The clinical features of this patient, whose main complaint was cough, have been reported elsewhere (Antia, 1968b). The patient who is alive 3 years after the diagnosis by clinical and angiographic studies, has biventricular endomyocardial fibrosis and chronic massive pericardial effusion. The patient is of further interest since she has not developed any ascites in the course of the disease. Progressive distension of the abdomen

was the commonest complaint. There were only three patients who complained of abdominal pains due to either the massive ascites or gross hepatomegaly. Cough was a prominent complaint by about half of the patients. This was obviously not due to pulmonary venous congestion since none of these patients had clinical or radiological evidence of pulmonary venous congestion. Recurrent fever in the course of the illness was complained of by five patients. One intelligent and educated father gave a history of definite fever antedating the onset of abdominal swelling in his 14-year-old son. Nearly half of the patients complained of facial swelling in the morning.

TABLE 3. Symptoms in fifteen children with endomyocardial fibrosis

Symptom	No. of cases
Progressive abdominal swelling	14
Cough	7
Facial swelling	6
Fever	5
Abdominal pains	3
Exertional dyspnoea	3
Tiredness	3
Pedal swelling	2
Chest pain	1
Weight loss	1

The physical signs are summarized in Table 4. Ascites with relatively little or no peripheral oedema (Fig. 1), hepatomegaly, and raised jugular venous pressure were present in all the patients except in one already referred to above who had no ascites. Ascites was gross in twelve patients and moderate in two. Peripheral oedema was moderate in two patients and minimal in four others. Digital clubbing was observed in only one patient whose haemoglobin electrophoresis (genotype) was SS. Atrial fibrillation confirmed by ECG occurred in two patients. Supraventricular tachycardia developed in one patient nearly 4 years after the diagnosis. The patient first presented with massive ascites in 1966, and after treatment with paracentesis abdominis and diuretics the ascites disappeared and has not recurred since. The patient is still alive.

There was pulsation in the pulmonary area in two patients and in the apical region in six. Systolic murmurs were present in the apical region in four patients and in the apical and tricuspid areas in four others. There was no diastolic murmur in any of the fifteen patients. There was no murmur in seven patients. Apical triple or gallop rhythm was present in six patients. A prominent third heart sound was observed in three patients with no murmurs.

The total protein content in ascitic fluid was determined in seven patients and this ranged between 0·1 g and 4·9 g/100 ml (mean 2·7 g). The total protein content in the pericardial fluid in one patient was 3·9 g/100 ml and in the pleural fluid in another patient 2·1 g/100 ml. The total serum protein was determined in fourteen patients and the value was above 5·0 g/100 ml in eleven and below 5 g in three. Antistreptolysin-O titre was normal in eleven patients in whom this test was carried out. The distribution of haemoglobin electrophoresis

(genotype) was AA (thirteen), AS (one) and SS (one). The histopathology of the liver in two patients showed cirrhosis; in one of these two patients the haemoglobin genotype was SS.

The electrocardiograms of fourteen patients were available for analysis. Some abnormalities were present in all cases. Regular sinus rhythm was present in all except two patients who had atrial fibrillation. The P-wave was of the 'pulmonale' configuration in seven patients and normal in the others. There was no instance of P–R interval prolongation. The QRS complex was dwarfed in the majority of the patients. By voltage criteria three patients had right ventricular hypertrophy. Right bundle branch block was present in four patients.

TABLE 4. Physical signs in fifteen children with endomyocardial fibrosis

Sign	No. of cases
(A) Extracardiac	
Ascites	14
Hepatomegaly	15
Puffiness of the face	9
Pedal oedema	6
Splenomegaly	6
Central cyanosis	3
Exophthalmos	3
Digital clubbing	1
(B) Cardiac	
Visible praecordial pulsation	
1. Apical	6
2. Pulmonary area	2
Systolic murmurs	4
1. Apical alone	4
2. Apical and tricuspid	4
No murmurs	7
Apical triple or gallop rhythm	6
Prominent third apical heart sound	3
Atrial fibrillation	2
Raised right atrial pressure	14
Small pulse volume	12

The abnormalities in the T-wave included flattening (four), dwarfing (three), inversion (three), gothic (one) and normal in the rest. There was no instance of S–T segment elevation or depression.

The clinical features, radiological and electrocardiographic findings of the cases with idiopathic cardiomegaly and rheumatic heart disease have been published elsewhere (Antia, 1968a; Antia, *et al.*, 1969; Antia, 1970). The salient features of these two conditions may be summarized as follows.

Idiopathic cardiomegaly. This is not an uncommon cause of heart failure in children. In all the nineteen patients studied varying degrees of cardiomegaly and cardiac failure were present. The most frequent presenting symptoms were fever, cough and breathlessness. A majority of the patients sought medical attention under a month from the onset of their

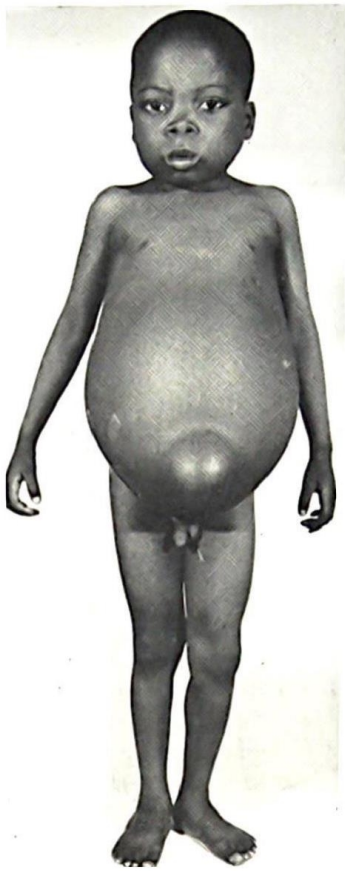

FIG. 1. A 6-year-old boy with right ventricular endomyocardial fibrosis. The abdomen is grossly distended with ascitic fluid. Note the absence of swelling of the legs.

symptoms. The most common physical signs included pyrexia, tachycardia, a gallop heart rhythm and hepatomegaly. The electrocardiogram was abnormal in every patient, T-wave inversion in the left praecordial leads being the most common abnormality.

Rheumatic heart disease. The majority of the patients with rheumatic heart disease sought medical attention when they were in advanced cardiac failure. Evidence of recent acute rheumatic fever was found in approximately 13% of the subjects, while a past history of acute rheumatic fever was obtained in 23% of others. The clinical manifestations of acute rheumatic fever were similar to those in non-tropical countries.

The problems of diagnosis and management of the disease in Nigerian children were similar to those reported from most developing countries of the world.

The chest radiographs of twenty-six patients with rheumatic heart disease, sixteen with idiopathic cardiomegaly, and of twelve with endomyocardial fibrosis were reviewed. Cardiomegaly was present in every case. The cardiothoracic ratio (Fig. 2) for the rheumatic heart disease group ranged from 52·8 to 80% (mean 67·96%), for the idiopathic cardiomegaly patients from 56 to 74% (mean 66·5%) and for the endomyocardial fibrosis group the ratio ranged from 61 to 80% (mean 69·37%). The enlargement of the cardiac shadow in most of the patients with endomyocardial fibrosis was undoubtedly contributed to by dilatation of

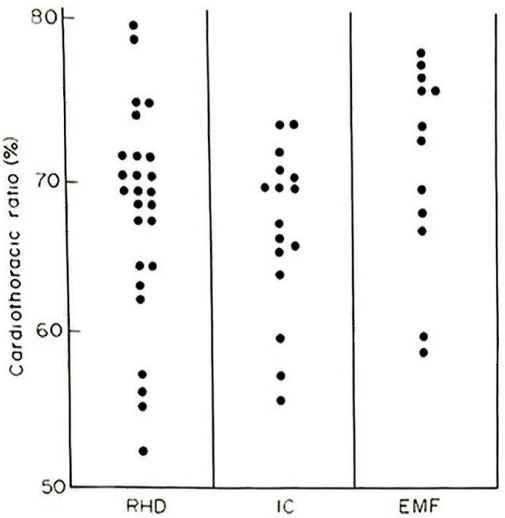

FIG. 2. The cardiothoracic ratio in patients with rheumatic heart disease (RHD), idiopathic cardiomegaly (IC) and endomyocardial fibrosis (EMF).

the right atrium and sometimes by pericardial effusion. A significant amount of effusion was found in four of the fifteen patients with endomyocardial fibrosis and none in the other patients. The cardiac silhouette in patients with right ventricular endomyocardial fibrosis was globular in shape and the lungfields were in all cases strikingly oligaemic as described by Abrahams (1962) and in *Bulletin of the World Health Organization* (1965). In patients with rheumatic heart disease the cardiac silhouette was in most cases of 'mitral' configuration with pulmonary vascular congestion and evidence of left atrial dilatation.

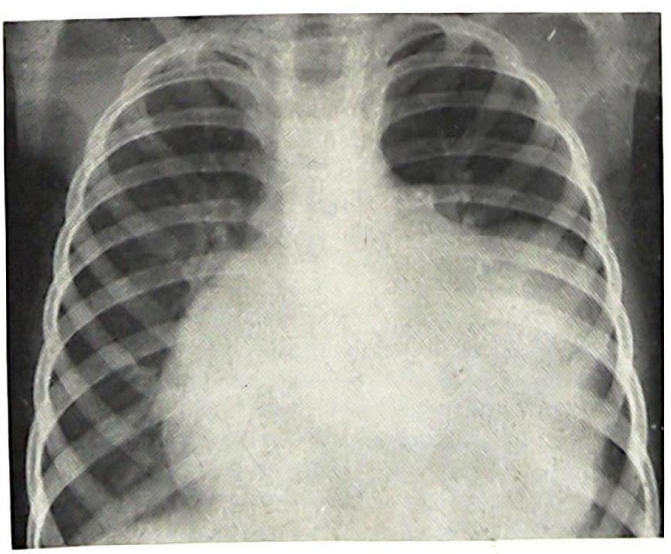

FIG. 3. Chest radiograph of a 6-year-old girl with left ventricular submitral aneurysm. Note the characteristic aneurysmal shadow in the left heart border and also the cardiac enlargement.

Three patients in this series had tuberculous myopericarditis and presented with cardiomegaly and heart failure. The diagnosis in each case was made from the chest radiograph which showed hilar lymphadenopathy, a strongly positive Heaf tuberculin test and satisfactory response to antituberculous chemotherapy.

Pyogenic myopericarditis occurred in two young infants who presented with pyrexia, marked tachypnoea, tachycardia, cardiomegaly and heart failure. Pericardiocentesis confirmed the diagnosis.

A diagnosis of left ventricular submitral aneurysm was made in two patients who presented with congestive cardiac failure. Confirmation of the diagnosis was obtained from left ventricular angiocardiography in one patient, and from necropsy in the second. A characteristic bulge in the left cardiac border of the chest X-ray (Fig. 3) suggested the diagnosis in each case.

TABLE 5. Summary of essential clinical features in rheumatic heart disease, idiopathic cardiomegaly and right ventricular endomyocardial fibrosis

Feature	Rheumatic heart disease	Idiopathic cardiomegaly	Right ventricular endomyocardial fibrosis
Onset of symptoms	Insidious but may be acute	Acute in most cases but may be insiduous	Insiduous
Age distribution	Not seen in present series under the age of 5 years	A majority of the patients under the age of 5 years	Same distribution as in rheumatic heart disease Few cases seen under the age of 5 years
Sex	Equal sexes	More males than females	More males
History of fever at onset of illness	Common	Common	Uncommon
Cough	Very common	Common	Uncommon
Peripheral oedema	Marked	Moderate to marked	Mild or absent
Ascites	Mild to moderate	Mild to moderate	Massive
Apex beat, apical impulse	Easily located, displaced apex beat, and left or right ventricular impulse	Easily located and displaced apex beat; left or right ventricular impulse	Difficult to locate apex
Triple or gallop rhythm	Common	Very common	Common
Hepatomegaly	Present and firm depending on the duration of the symptoms	Present, tender and soft	Present, firm to hard
Chest radiography	Moderate to very marked cardiomegaly with mitral configuration, venous congestion in lungfields	Moderate to marked cardiomegaly, globular heart shape; venous congestion in lungfields	Marked cardiomegaly, globular heart shape, strikingly clear lungfields
Electrocardiogram	P-mitrale; prolongation of P–R interval, biventricular hypertrophy	Normal P-wave and P–R interval; T-inversion in $V_{5\cdot6}$ common	Dwarfed QRS, nonspecific ST changes, P-pulmonale, RBBB
ASO Titre	Very commonly raised	Normal	Normal

RBBB, right bundle branch block.

The essential clinical features of the three common acquired heart diseases in the present study are presented in Table 5. Although it was sometimes impossible at the bedside to distinguish one of these conditions from the other, an accurate clinical diagnosis of each of the conditions was easily made from a combination of their clinical features, chest radiographs and electrocardiographic studies.

DISCUSSION

Acquired heart disease in children and adults is an important and common health problem throughout the tropics and subtropical parts of the world. The so-called tropical cardiomyopathies constitute the largest number of acquired heart disorders encountered in these areas.

Medical studies in children in the tropics have been rightly concentrated on the more common and devastating conditions of malnutrition, infections, and other childhood diseases. With the control of some of these diseases in childhood, acquired and congenital heart disease, malignancy, inherited disorders and so forth will engage the attention of workers more than ever before. Since there has to be a beginning of detailed studies of these conditions, it was thought necessary to attempt to define the pattern of acquired heart disease as seen in Nigerian children.

The comments on the present study will be devoted more to endomyocardial fibrosis than to the other two common acquired heart diseases which have been the subject of previous communications (Antia, 1968a; Antia *et al.*, 1969; Antia, 1970; Antia *et al.*, unpublished data). Much has been written about endomyocardial fibrosis from East and West Africa where the incidence of the disease is higher than elsewhere (Davies, 1948; Williams, Ball & Davies, 1954; Davies & Ball, 1955; Somers, Brenton & Sood, 1968; Shaper, Hutt & Coles, 1968; Nwokolo, 1955; Abrahams, 1959, 1962; Abrahams & Parry, 1965). From these various studies the pathology, clinical and haemodynamic features of the disease have been well-established. Yet its pathogenesis and aetiology remain essentially unknown. In children the clinical features of the disease are the same as in adults except for a few details as shown in this study.

The most common presenting symptom was progressive abdominal swelling. The duration of the symptom varied between 3 months and 2 years before patients sought medical attention. All the patients with this complaint had varying degrees of ascites. The unusual features regarding ascites in two patients already referred to call for further comments. One of these two patients, alive 3 years after the diagnosis of biventricular lesion, has not developed ascites in the course of the illness. The other patient with lone right ventricular lesion initially presented with massive ascites which was managed with paracentesis abdominis and diuretics. Four years after the disappearance of the ascites the patient is alive and there has been no recurrence of the ascites even though the patient has not been maintained on any diuretics. To our knowledge there has been no previous record of such an unusual course of this disease.

There was no instance of either nocturnal dyspnoea or haemoptysis. Abrahams (1962) has stated that nocturnal dyspnoea never occurs in right ventricular endomyocardial fibrosis. However, Somers *et al.* (1968) have reported seven patients with this symptom out of twenty-eight cases studied. Other symptoms reported in adults which were absent in children in our series included joint pains, palpitations and sore throat. The complaint of 'fever' at

the initial stage of the disease was given by about 43% of adults reported by Somers *et al.* (1968). Patients studied by Abrahams (1962), Abrahams & Parry (1965), Ive *et al.* (1967), consisted of children and adults, and these authors commented that 'fever' was a common complaint.

In the present study consisting entirely of children, fever was complained of by five out of the fourteen children (35·7%). Analysis of the temperature charts on admission and throughout the period of hospitalization in twelve patients in this series revealed low grade pyrexia (temp. 99–100°F) in six. One of the six patients with a continuous low grade pyrexia for 4 weeks died at the end of that period and necropsy showed a well established right ventricular lesion. This case illustrates the fact that even in well-established disease, patients may have and do complain of fever; therefore, this symptom is not necessarily a manifestation of an early stage in the natural history of the disease as has been suggested (Abrahams & Parry, 1965; Ive *et al.*, 1967).

The physical signs of the disease in children, are similar to those in adults. However, Somers *et al* (1968), and Abrahams (1962) have included digital clubbing as a sign in some of their patients. The only patient in our series with this sign had an abnormal haemoglobin SS. Since children with haemoglobin SS sometimes develop digital clubbing we are inclined to attribute this sign to the abnormal haemoglobin and hepatic fibrosis in our single patient with this sign.

The characteristic chest radiograph and angiocardiogram of the disease were as described by others (Abrahams, 1962; Cockshott, 1965; *Bulletin of the World Health Organization*, 1965; Cockshott, Saric & Ikeme, 1967). There was, however, no case of calcification within the cardiac shadow as described by other authors.

The non-specificity of the changes in the ECG in this disease has been reported by others (Williams & Somers, 1960; Abrahams, 1962). Most of our findings were in agreement with these previous reports. Right bundle branch block occurred in four of our patients. This abnormality was not mentioned in the report by Abrahams (1962), nor in that by Somers *et al.* (1968). The incidence of P-mitrale was much higher in patients studied by Williams & Somers (1960) than in the present series.

The total protein content of the ascitic fluid was raised in all seven patients in whom the test was carried out. Although the rise was in keeping with the findings of others, the mean (2·7 g/100 ml) in our series was lower than the mean of 4·6 g/100 ml reported by Abrahams (1962), and the mean of 3·5 g/100 ml reported by Somers *et al.* (1968). It seems likely that this difference is due to the duration of the ascitic fluid accumulation—the longer the accumulation the higher the protein content.

The present study does not provide significant clues to the aetiology or pathogenesis of the disease. None of our patients showed clinical evidence of malnutrition, although they all came from the low socio-economic families. Ingestion of plantain was once considered an aetiological factor in the disease, but experimental evidence does not lend support to this theory (Ojo & Parratt, 1966; Antia, Talbert & Paplanus, 1967). Thus, the available clinical and experimental evidence does not suggest a nutritional cause in the disease. Other aetiological factors which have been suggested include the ethnic origin of patients studied in Uganda (Shaper & Coles, 1965), and filariasis in Nigerian patients (Ive *et al.*, 1967). All the children in the present study were of the Yoruba ethnic group, the predominant tribe in the Western State of Nigeria, where the UCH is situated. The majority of the patients attending this hospital are of this ethnic group. The suggestion of a possible aetiological relation-

ship between endomyocardial fibrosis and filariasis is an attractive one which should be pursued in future studies.

The only previous study in tropical Africa which attempted to define the pattern of acquired heart disease in children was that from Uganda by Caddell *et al.* (1966). Of their total number of fifty children with acquired heart disease, only six were cases of endomyocardial fibrosis. These workers showed that rheumatic heart disease was the most common acquired heart disorder in Ugandan children. In the present study this condition was also the commonest, constituting 40·8% of the total number of cases. Unlike the Ugandan workers, we have not found acute rheumatic heart disease to be common. About 84% of our patients had the chronic form of the disease in contrast to 34·8% in Ugandan children. This difference may well be due to Ugandan parents being more hospital-conscious than their Nigerian counterparts, who generally seek medical attention for their children at an advanced and chronic stage of the disease. Also in contrast to the Ugandan study, the sexes were equal in our series. However, Al-Bahrani and his associates (1966) have reported equality in the sexes in Iraq children with the disease. Of the thirty-one children with rheumatic heart disease in the present study, seven (22·6%) had significant mitral stenosis. This early development of mitral stenosis in rheumatic heart disease in the tropics has been observed by others (Beet, 1956; Roy *et al.*, 1963; Harling *et al.*, 1965). There is no satisfactory explanation for this phenomenon.

Idiopathic cardiomegaly was the next common acquired heart disease in the present study. Caddell *et al.* (1966) reported fewer cases in Ugandan children. The cause of the disease is unknown. However, on the basis of the acute onset of the symptoms and the presence of an initial fever in a large number of the patients, there has been a suggestion of an infective aetiology (Antia *et al.*, 1969).

Of the miscellaneous acquired heart disease there were three tuberculous and two pyogenic myopericarditis. The tuberculous cases presented with congestive cardiac failure, and the diagnosis in each case was based on the strongly positive tuberculin test, hilar adenopathy in the chest radiograph, and satisfactory response to antituberculous drug therapy. The diagnosis of pyogenic myopericarditis was suggested by the age of the patient and the acute onset of the symptoms. Both patients were under the age of 1 year and had pyrexia, marked tachycardia, cardiomegaly and muffled heart sounds. The diagnosis in each case was confirmed by pericardiocentesis and at necropsy.

The two patients with left ventricular submitral aneurysm presented with congestive cardiac failure. The diagnosis in each case was suggested by the characteristic bulge in the left cardiac border of the chest radiograph. In one of the two patients the diagnosis was confirmed by angiocardiogram and in the other by autopsy.

ACKNOWLEDGMENT

This study was given financial support by the World Health Organization.

REFERENCES

ABRAHAMS, D.G. (1959) An unusual form of heart disease in West Africa, its relationship to endomyocardial fibrosis. *Lancet*, **ii**, 111–113.

ABRAHAMS, D.G. (1962) Endomyocardial fibrosis of the right ventricle. *Quart. J. Med.* **31**, 1–20.

ABRAHAMS, D.G. & PARRY, E.H.O. (1965) The natural history of endomyocardial fibrosis. *Quart. J. Med.* **34**, 383–408.

AL-BAHRANI, I.R., THAMER, M.A., AL-OMERI, M.M. & AL-NAAMAN, Y.D. (1966) Rheumatic heart disease in the young in Iraq. *Brit. Heart J.* **28,** 824–828.

ANTIA, A.U. (1968a) Clinical diagnosis of idiopathic cardiomegaly in children. *Cardiologia,* **52,** 41–43.

ANTIA, A.U. (1968b) Endomyocardial fibrosis presenting as chronic pericardial effusion. *W. Afr. med. J.* **17,** 108–109.

ANTIA, A.U., TALBERT, J.L. & PAPLANUS, S. (1968) Etiology of endomyocardial fibrosis. An experimental evaluation of plaintain ingestion and chronic lymphatic obstruction. *Johns Hopk. med. J.* **122,** 87–93.

ANTIA, A.U., COCKSHOTT, W.P. & THORPE, G.J. (1969) Idiopathic cardiomegaly in Nigerian Children. *Brit. Heart J.* **31,** 178–183.

ANTIA, A.U. (1970) Idiopathic cardiomegaly in African children. Further experience with cases seen in Nigeria. *Path. Microbiol.* **35,** 44–48.

BEET, E.A. (1956) Rheumatic heart disease in Northern Nigeria. *Trans. roy. Soc. trop. Med. Hyg.* **50,** 587–593.

BINDER, E. (1961) Cardiovascular disease in Accra (Ghana) as suggested by analysis of post-mortem records. *W. Afr. med. J.* **10,** 158–170.

BULLETIN OF THE WORLD HEALTH ORGANIZATION (1965) *Cardiomyopathies,* **33,** 257–266.

CADDELL, J.L., WARLEY, A., CONNOR, D.H., D'ARBELA, P.G. & BILLINGHURST, J.R. (1966). Acquired heart disease in Ugandan children. *Brit. Heart J.* **28,** 759–765.

COCKSHOTT, W.P. (1965) Angiocardiography of endomyocardial fibrosis. *Brit. J. Radiol.* **38,** 192–200.

COCKSHOTT, W.P., SARIC, S. & IKEME, A.C. (1967) Radiological findings in endomyocardial fibrosis. *Circulation,* **35,** 913–922.

D'ARBELA, P.G., KANYEREZI, R.B. & TULLOCH, J.A. (1966) A study of heart disease in the Mulago Hospital, Kampala, Uganda. *Trans. roy. Soc. trop. Med. Hyg.* **60,** 782–790.

DAVIES, J.N.P. (1948) Endocardial fibrosis in Africans. *E. Afr. Med. J.* **25,** 10–14.

DAVIES, J.N.P. & BALL, J.D. (1955) The Pathology of endomyocardial fibrosis in Uganda. *Brit. Heart J.* **17,** 337–359.

HALIM, A.M. & JACQUES, J.E. (1961) Rheumatic heart disease in the Sudan. *Brit. Heart J.* **23,** 383–386.

HARLING, D.S., MARSDEN, P.D. & RIDLEY, D.S. (1965) Some observations on the pattern of heart disease in the Gambia. *Trans. roy. Soc. trop. Med. Hyg.* **59,** 628–641.

IVE, F.A., WILLIS, A.J.P., IKEME, A.C. & BROCKINGTON, I.F. (1970) Endomyocardial fibrosis and filariasis. *Quart. J. Med.* **36,** 495–516.

MILLER, D.C., SPENCER, S.S. & WHITE, P.D. (1962) Survey of cardiovascular disease among Africans in the vicinity of the Albert Schweitzer Hospital in 1960. *Amer. J. Cardiol.* **10,** 432–446.

NWOKOLO, C. (1962) The evolution of endomyocardial fibrosis. *W. Afr. med. J.* **11,** 51–57.

OJO, G.O. & PARRATT, J.R. (1966) Urinary excretion of 5-hydroxyin-doleacetic acid in Nigerians with endomyocardial fibrosis. *Lancet,* **i,** 854–856.

ROWLAND, H.A.K. (1965) Cardiovascular disease in Sierra Leone. *W. Afr. med. J.* **14,** 99–100.

ROY, S.B., BHATIA, M.L., LAZARO, E.J. & RAMALINGASWAMI, V. (1963) Juvenile mitral stenosis in India. *Lancet,* **ii,** 1193–1195.

SHAPER, A.G., HUTT, M.S.R. & COLES, R.M. (1968). Necropsy study of endomyocardial fibrosis and rheumatic heart disease in Uganda 1950–65. *Brit. Heart J.* **30,** 391–401.

SHAPER, A.G. & COLES, R.M. (1965) The tribal distribution of endomyocardial fibrosis in Uganda. *Brit. Heart. J.* **27,** 121–127.

SOMERS, K., BRENTON, D.P. & SOOD, N.K. (1968) Clinical features of endomyocardial fibrosis of the right ventricle. *Brit. Heart J.* **30,** 309–321.

SOMERS, K., BRENTON, D.P., D'ARBELA, P.G., FOWLER, J.M., KANYEREZI, B.R. & SOOD, N.K. (1968) Haemodynamic features of severe endomyocardial fibrosis of the right ventricle, including comparison with constrictive pericarditis. *Brit. Heart J.* **30,** 322–332.

WILLIAMS, A.W., BALL, J.D. & DAVIES, J.N.P. (1954) Endomyocardial fibrosis in Africa; its diagnosis, distribution and nature. *Trans. roy. Soc. trop. Med. Hyg.* **48,** 290–305.

WILLIAMS, A.W. & SOMERS, K. (1960) The electrocardiogram in endomyocardial fibrosis. *Brit. Heart J.* **22,** 311–315.