

LEFT VENTRICULAR ANEURYSM IN THE AFRICAN

BY

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Left ventricular aneurysm is a very rare finding in the African and it is the purpose of this paper to describe four such cases, including one following coronary thrombosis.

Sternberg (1914) has shown that 85 per cent of cardiac aneurysms in white patients follow on myocardial infarction. A different picture is presented in the African. In a series of fifteen African patients with left ventricular aneurysm, Jacobs (1952) found that none were due to coronary thrombosis, whereas his 65 cases of aneurysm in white patients were all due to this cause.

The low incidence of coronary thrombosis (and consequently of left ventricular aneurysm) in the African appears to be due to the notable absence of severe coronary atherosclerosis (Wainwright, 1958). This last observation has also been the experience of workers at other centres in Africa (Becker, 1946; Higginson and Pepler, 1954; and Gelfand, 1948).

CASE REPORTS

Case 1. An African woman, aged 30 years, was first seen in 1951 and died nine months later. She was admitted to hospital on three separate occasions during this period and each time gave a history and presented with signs typical of congestive cardiac failure. B.P. 120/85 mm. Hg. Pulse rate 104/min. There was a harsh systolic murmur at the apex with an accentuated pulmonary second sound. On each admission, except the last, there was a good response to mercurial diuretics, digitalis, and rest, but on the last there was poor response to therapy and the patient died suddenly during the fourth month in hospital. X-ray and screening of the chest (Fig. 1) showed an enormously enlarged heart with ill-defined pulsation in all areas. The left ventricle was much enlarged with an irregular bulge which showed paradoxical pulsation. The electrocardiogram (Fig. 2) showed low voltage, incomplete left bundle-branch block, and slightly inverted T waves in leads 1, AVL, and V5.

Necropsy. Apart from generalized congestive changes, the only abnormal findings were in the heart. A straw-coloured effusion was found in the pericardial sac. The heart was enlarged. There was a large aneurysm, slightly lobulated and about 7 cm. in diameter, situated antero-laterally at the level of the atrio-ventricular groove (Fig. 3). The wall of the aneurysm was thin and fibrous and was lined on the inside by an adherent organized thrombus. The left ventricular myocardium showed some hypertrophy and dilatation and the right ventricular myocardium some hypertrophy. There was no lesion of the coronary arteries and the valves were normal. The aorta was thickened and wrinkled, but the coronary ostia were not involved in any disease process.

Histology. The wall of the aneurysm was formed by vascular fibrous tissue with numerous dilated thin walled vessels and was lined by laminated thrombus. The wall was very thin at some points. The adjacent myocardium showed degenerating fibres and areas of fibrosis. No changes were seen in the vessels and no active inflammatory process was present. The aetiology of these changes was not apparent.

Case 2. An African woman, aged 24 years, was admitted with the typical symptoms and signs of congestive cardiac failure. In addition, she complained of severe pain in the ankle and knee joints a week before the onset of failure. The heart was enlarged. The pulse was of low amplitude, with a rate of

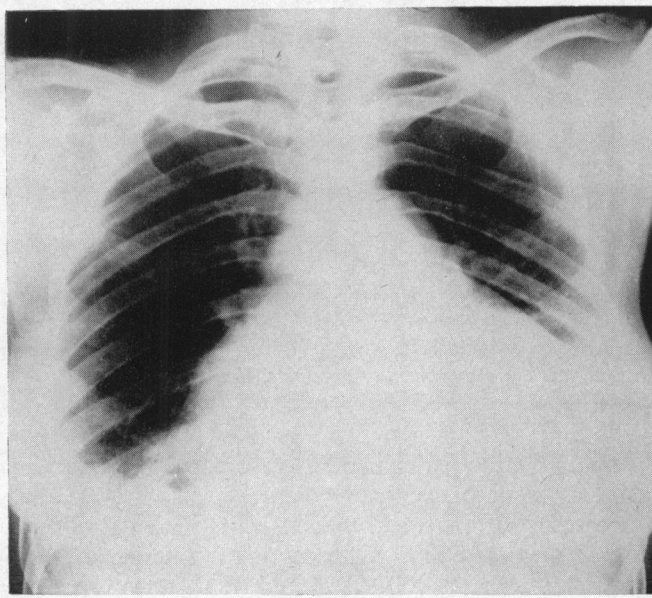


FIG. 1.—Radiograph of Case 1, showing enormously enlarged heart. The aneurysm was seen only in the left oblique position.

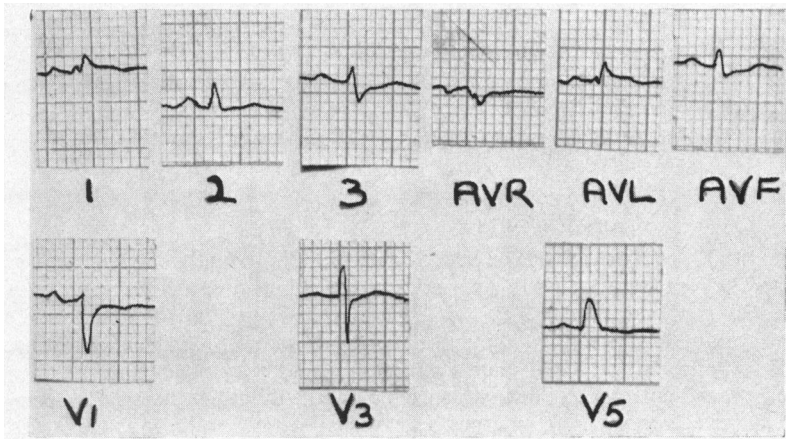


FIG. 2.—Electrocardiogram from Case 1, showing low voltage, incomplete left bundle-branch block, and inverted T waves in leads I, AVL, and V5.

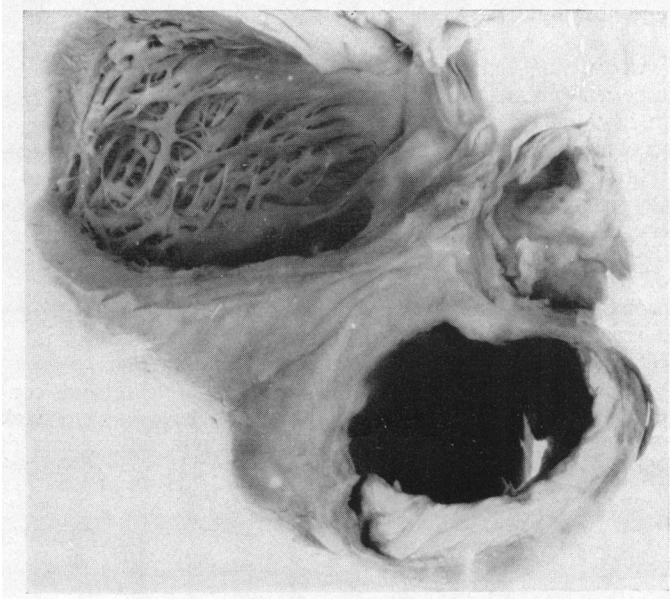


FIG. 3.—Photograph of the heart of Case 1, showing the left ventricle and the large aneurysm at the level of the atrio-ventricular groove.

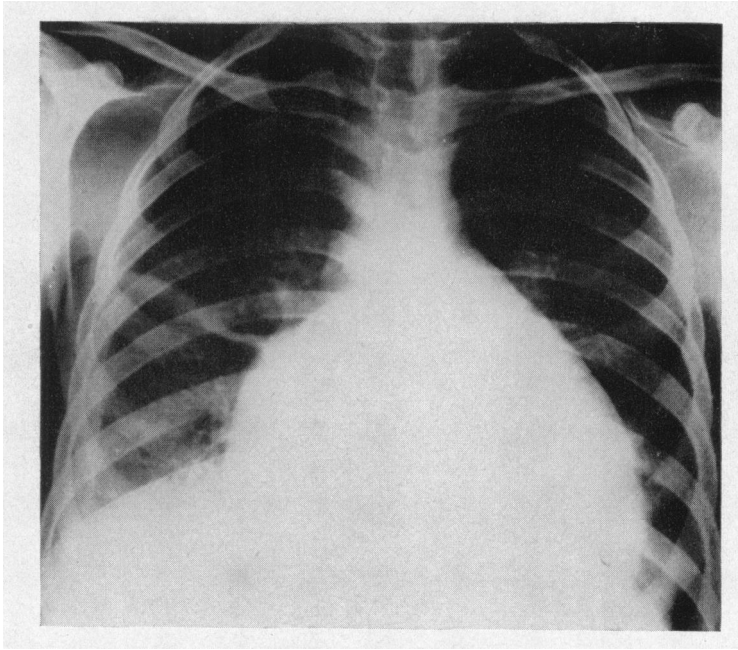


FIG. 4.—Radiograph of Case 2, showing the enlargement of the cardiac shadow and the right pleural effusion. The aneurysm is not visible.

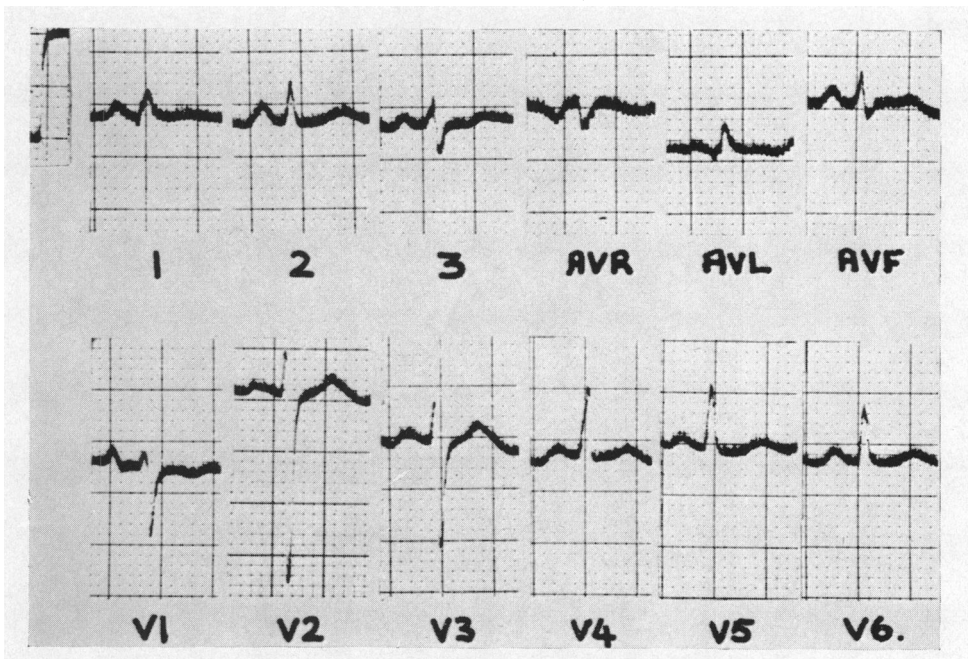


FIG. 5.—Electrocardiogram of Case 2. The tracing is within normal limits in spite of the gross changes found at necropsy.

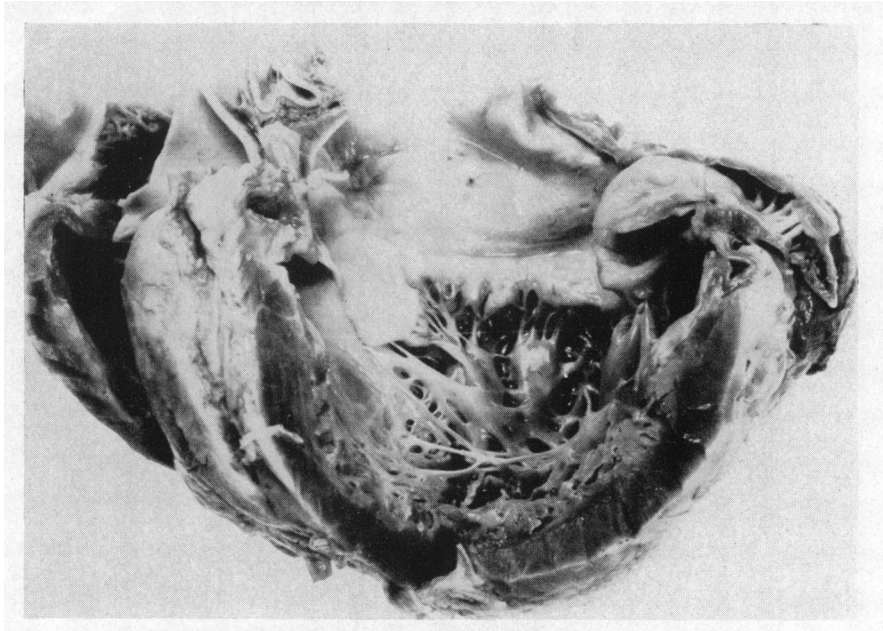


FIG. 6.—Photograph of the heart of Case 2. The aneurysm and its opening from the left ventricle are clearly seen at the top right.

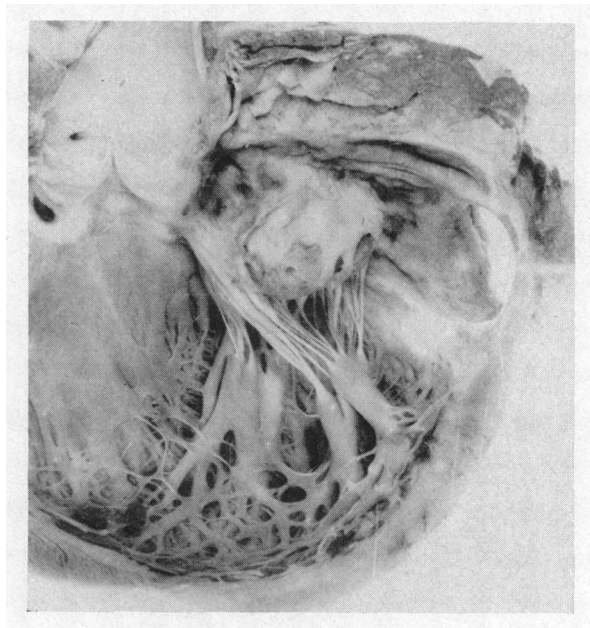


FIG. 7.—Photograph of the heart of Case 3, showing the left ventricle with the large opening into the aneurysm. Aneurysmal dilatation of the mitral cusp is also clearly seen.

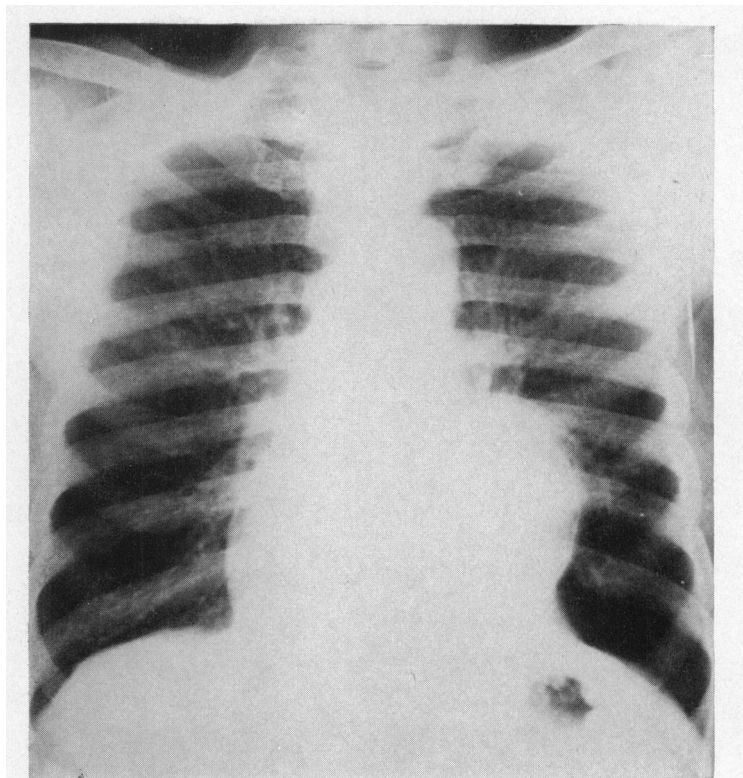


FIG. 8.—Radiograph of Case 4, showing the large bulge of the left ventricular aneurysm with calcification in its wall.

120 a minute, and on the day of admission, pulsus paradoxus was present. B.P. 90/70 mm. Hg. A loud, rough pan-systolic murmur was heard at the apex, with a short rumbling mid-diastolic murmur. The pulmonary second sound was accentuated. The patient was treated with digitalis, mercurial diuretics, salicylates, and steroids, but there was no improvement in the signs or symptoms and she died a month after admission. X-ray of the chest (Fig. 4) showed much cardiac enlargement and the heart was globular in shape. Bilateral basal congestive changes and right pleural effusion were noted. The electrocardiogram (Fig. 5) was within normal limits.

Necropsy. Apart from generalized congestive changes, the only abnormality was found in the heart, which was enlarged (Fig. 6). There was a lobulated aneurysm 3½ cm. in diameter just below the atrio-ventricular groove and slightly to the left of the intraventricular groove. The aneurysm had numerous adhesions on its outer surface. It was lined on the inside by organized blood clot. One cusp of the mitral valve showed aneurysmal dilatation and was bulging into the left ventricle. The right ventricle showed some dilatation and hypertrophy. The left ventricle was hypertrophied and also showed well-marked subendocardial fibrosis. There was patchy fibrosis of the left ventricular myocardium. Dissection of the coronary arteries showed no evidence of disease. The aorta was normal. There was no fluid in the pericardium, but there was evidence of old, healed pericarditis.

Histology. The wall of the aneurysm consisted of dense collagenous fibrous tissue with foci of calcification. There were deposits of blood pigment in the wall and the lining of the aneurysm consisted of organized blood clot. Numerous dense adhesions were present over the outer surface of the aneurysm and there was evidence of a non-specific chronic pericarditis. The heart muscle in the area near the interventricular septum showed fibrosis and chronic inflammatory cell infiltration. A marked degree of subendocardial fibrosis was present. There was hypertrophy of the heart muscle. The coronary arteries showed no evidence of disease. In the sections examined, there was no evidence of any specific disease process that could account for the formation of an aneurysm.

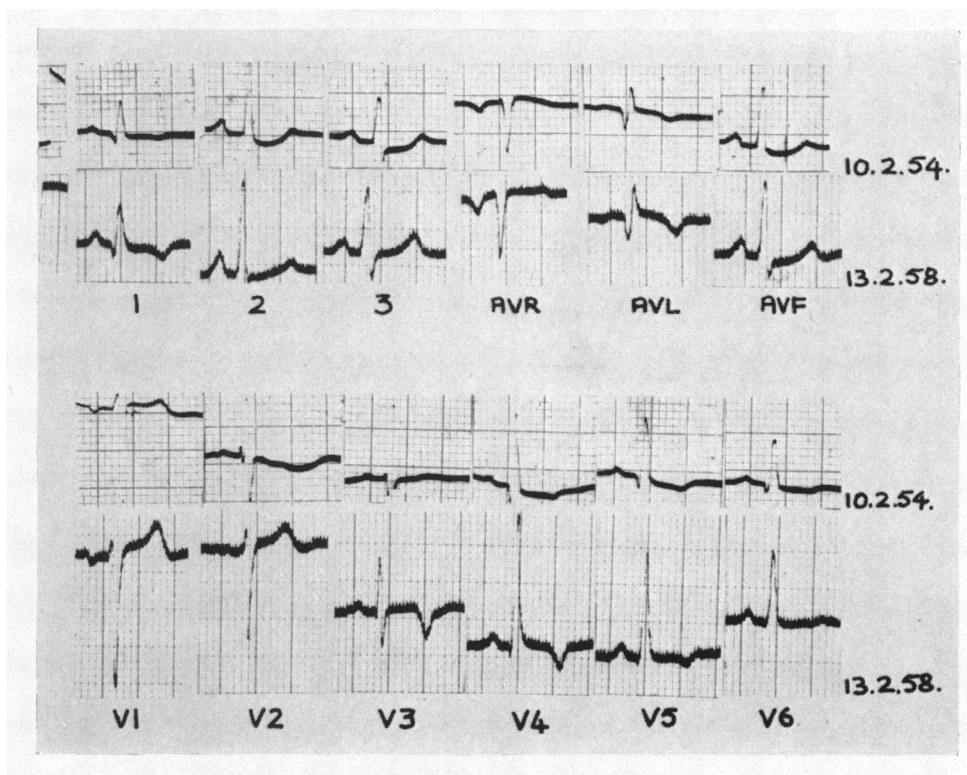


FIG. 9.—Electrocardiogram from Case 4, showing evidence suggestive of anteroseptal infarction and the changes that have occurred during the four-year period between the two tracings.

Case 3. An African man, aged 31 years. His main complaints were pain in the chest for three weeks, dyspnoea on exertion, orthopnoea, a cough productive of blood-stained sputum, and swelling of the ankles. There was a history of pain in the elbows, knees and ankles about six months before admission. On examination the patient was found to be pyrexial (100° F.). Jugular venous pressure was slightly raised. The pulse rate was 96 a minute, the B.P. 110/80. Clinical evidence of cardiomegaly was present. A systolic murmur was heard at all areas, but maximally at the apex. There was some evidence of pulmonary congestion and tender hepatomegaly was found. There was some oedema of the legs. The patient was digitalized and given mercurial diuretics, but there was no response to treatment and he died suddenly a month after admission. X-ray of the chest showed that the heart was enlarged with a hump-shaped shadow in the region of the atrial appendage. On screening the bulge was seen on the left border of the ventricle and showed paradoxical pulsation. The lung fields showed evidence of pulmonary oedema. The electrocardiogram showed a P-R interval of 0.28 sec. but was otherwise within normal limits.

Necropsy. Apart from generalized congestive changes the only abnormality was found in the heart, which was enlarged (Fig. 7). There was an aneurysm about 5 cm. in diameter, involving the anterolateral wall of the left ventricle at the level of the atrio-ventricular septum. The aneurysm had a thin fibrous wall, lined by a laminated thrombus. Fibrosis of the myocardium adjacent to the aneurysm was evident. There was also an aneurysmal dilatation of a cusp of the mitral valve. A further small aneurysm was noted in the interventricular septum immediately below the aortic valve, and extending into the muscle of the septum. The muscle of the left ventricle was hypertrophied and dilated and there was also hypertrophy of the muscle of the right ventricle. Fibrosis was not present in other parts of the myocardium apart from that noted near the site of the aneurysm. Dissection of the coronary arteries revealed a thrombus in the circumflex branch of the coronary artery.

Histology. The aneurysm showed a lining of organizing blood clot. The wall was thin and consisted of fibrous tissue with areas in which deposition of blood pigment had occurred. An organized thrombus of

the circumflex branch of the left coronary artery was present. The coronary arteries showed no evidence of atherosclerosis. Some myocardial fibrosis was observed in a section taken near the site of the aneurysm.

Case 4. An African man, aged 54 years. This patient is still alive and was first seen in January 1954 when he was admitted to hospital in congestive cardiac failure, the cause of which was thought to have been hypertension, the blood pressure being 140/110 mm. Hg. On X-ray it was established that he had a large posterior left ventricular aneurysm with calcification in its wall (Fig. 8). The electrocardiogram of that period and a tracing taken four years later show evidence suggestive of an anteroseptal infarction (Fig. 9). There was no history of an acute episode of chest pain prior to his admission. The Wassermann reaction was negative. There were three further admissions, during each of which he was successfully treated for congestive cardiac failure. Blood pressures recorded were never more than 140/90 mm. Hg. When last seen the patient was feeling fairly well, but complained of pain in the chest very suggestive of angina pectoris. X-ray and screening of the chest showed a large bulge on the left cardiac border with calcification of the wall. From its appearance and the fact that paradoxical pulsation was noted, there was little doubt that it represented a cardiac aneurysm.

DISCUSSION

Apart from myocardial infarction, syphilis is given as the most frequent cause of cardiac aneurysm (Aronstein and Newman, 1941; Benson *et al.*, 1933; Braunstein *et al.*, 1940; Brink and Barnard, 1954; Cookson, 1929; Higginson and Keely, 1951; Morris, 1927; Sohval, 1935). Less often, it may be due to: congenital weakness of the myocardium (Bland *et al.*, 1933; Berlin and Hollén, 1938; Burn *et al.*, 1943; Clearkin and Bunje, 1955; Hunter and Benson, 1933); mycotic aneurysm (Pirani, 1943); trauma (Crawford, 1943; French, 1912; Joachim and May, 1927); rheumatic myocarditis (Crawford, 1943; Parkinson *et al.*, 1938); and endarteritis of unknown etiology (Higginson and Keely, 1951; Macfie and Ingram, 1920).

Jacobs (1952), investigating 15 cases of aneurysm in Africans, found that 6 were due to syphilis; 4 were undetermined; and one each due to congenital diverticulum, tuberculosis, Loeffler's parietal endocarditis, rheumatic myocarditis and mycotic aneurysm. The case reports presented in this paper do not follow any of these patterns of aetiology.

Cases 1 and 2 were young African women and both had ventricular aneurysms arising from a similar site, adjacent to the atrio-ventricular groove, but here the similarity ceases. Whereas Case 1 showed no evidence of a myocardial or pericardial inflammatory process, this was a striking feature in Case 2. The significance of the localized fibrosis in the myocardium adjacent to the aneurysm in Case 1 is difficult to assess, but might suggest that the aneurysm arose in an area weakened by fibrosis following on a localized infective or ischaemic lesion. Case 2, apart from the chronic inflammatory process present in the myocardium and pericardium, also had subendocardial fibrosis as a notable feature. It is impossible to say what type of inflammatory process the evidence represents. It will be recalled that the patient complained of severe joint pains prior to the onset of the symptoms of cardiac failure. The possibility of rheumatic fever with pancarditis was considered clinically and the patient treated with salicylates and steroids. There were doubts as to the aetiology, however, mainly because the history was atypical. Had this in fact been an acute rheumatic process, it would have been reasonable to expect some evidence of this to be found histologically, which was not the case. Moreover there was no evidence of valvulitis, but aneurysmal dilatation of one cusp of the mitral valve was observed. The aetiology of the ventricular aneurysm in Cases 1 and 2 is therefore unknown.

Case 3 is the only instance of ventricular aneurysm following on coronary thrombosis as yet described in an African. There are, however, some unusual features. First, the patient was only 31 years old when he died, an age at which coronary thrombosis is uncommon, even in white patients (Wood, 1956). Second, the site of the thrombus, the circumflex artery, is not as common as the anterior descending branch. Thirdly, the most remarkable feature of the case, is the complete absence of coronary artery atherosclerosis or other lesion that could be held responsible for the formation of a thrombus. Following on the last point mentioned, there are two further possibilities to be considered. Due to its close proximity to the circumflex artery, the aneurysm

may have stretched this artery and thus induced the formation of a thrombus: if this was the case, the thrombosis was *post* rather than *propter hoc*. The second possibility is that the thrombus was, in fact, an embolus to the circumflex artery and did not occur *in situ*, but embolization to the coronary artery is very rare, and only 45 cases have been reported (Moragues *et al.*, 1950). It seems reasonable to suppose that in this case coronary artery thrombosis did occur and that it was the cause of the ventricular aneurysm.

The fourth patient is still alive and the cause of his aneurysm is not known. The history of chest pain suggests attacks of myocardial ischæmia, for which there is some support in the electrocardiogram: this might, therefore, be the cause of the aneurysm.

SUMMARY

Ventricular aneurysm in Africans is rare. Four cases of left ventricular aneurysm in Africans are reported, of which the cause was shown to be coronary thrombosis in one and was unknown in the other three patients.

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