change and vulvectomy is not justified as a cancer preventive. And as an empirical treatment it does not give satisfactory results even when the skin change appears to be strictly limited and demarcated. Unless the cause is determined and eradicated, epithelial dystrophy recurs in approximately 50% of cases treated by partial or total vulvectomy.

For the searching of records and the follow-up of patients we are indebted mainly to Dr. Winifred Francis; but also to Dr. Mary Mayeur and Dr. R. E. Rewell for details of individual cases. Cases 1 and 3 were under the care of Mr. E. Parry-Jones, to whom we are grateful for permission to report them.

ADDENDUM

Since this paper was completed one more of the 98 women under observation has developed cancer of the vulva, and the circumstances confirm the above conclusions. Histological examination of biopsy specimens of the chronically affected vulval skin was carried out in 1956, and showed epithelial activity of a degree which one of us (A.S.W.) considered disturbing. The lesion was so widespread and ill demarcated clinically as to be unsuitable for excision, so it was treated conservatively with some improvement. In view of the histological findings, however, the patient was observed regularly, and when symptoms relapsed in 1960 further biopsy specimens were examined without any more alarming epithelial changes being found. In April, 1961 (after five years' observation), however, the patient reported that she had had pain in one area of the vulva for one week. This area then had a red appearance, and on section showed cancer *in situ* with possibly early invasion of malignant cells at one point.

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SYNDROME OF MITRAL INCOMPETENCE, MYOCARDITIS, AND PULMONARY HYPERTENSION IN NIGERIA

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[WITH SPECIAL PLATE]

Rheumatic mitral-valve disease has been studied intensively during the past 25 years. Mitral stenosis is rare in Nigerians, but one of us (Abrahams, 1959) described a peculiar form of mitral-valve disease which is common in West Africa but unusual elsewhere. In this paper is reported a more detailed study of the syndrome (presenting as mitral incompetence and pulmonary hypertension), which may be caused by an unusual form of rheumatic carditis.

Material

The study covered 50 patients with signs of mitral incompetence and pulmonary hypertension observed over a relatively short period; most of them were examined by both authors, and many were admitted to hospital for treatment and more prolonged observation. Electrocardiograms were recorded and a radiological examination was made in all cases. Right heart catheterization was carried out successfully in 15 cases. Necropsy was performed in 13 cases.

All the patients came from the western region of Nigeria. Most of the tribes of the south were represented, but Yorubas, who are the majority people of the western region, predominated. All were young by Western standards, but not in their own community, where the expectation of life is short. At the last official census in 1952 the population of Ibadan Province was over 1½ million, and less than 8% were over the age of 50. The diet of these people consists mainly of carbohydrate and is deficient in protein, but none of the patients was obviously malnourished at the time of examination.

Clinical Features

Women were affected at least twice as frequently as men, and over three-quarters of the series were under 30 years of age. It was not easy to obtain an accurate history, because these people have little sense of time and find it difficult to describe symptoms. However, the majority complained of substernal pain; in some cases this was clearly cardiac pain, but generally it was impossible to distinguish pain from the sense of tightness which accompanied effort dyspnoea. Nocturnal dyspnoea was uncommon, and most patients could lie flat without discomfort. Cough was a constant complaint, but haemoptysis was very rare.

Swelling of the legs and abdomen was the presenting symptom in half the cases, and in some had already

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assumed gross proportions. We were unable to obtain a history of preceding acute illness in any patient, and in particular sore throat and arthralgia were conspicuously absent.

The physical signs were those of mitral incompetence and pulmonary hypertension of varying severity, with or without right heart failure. The radial pulse was small in volume, and the average systemic arterial pressure was only 112/76 mm. Hg over the whole series. When it appeared that mitral incompetence was severe the pulse was small and had a collapsing quality. Sinus rhythm was present in all except one patient, who had atrial fibrillation. Ventricular extrasystoles caused pulse irregularity in many patients. The mean jugular venous pressure was always raised when there was oedema with ascites. In a number of patients a large *cv* wave indicated tricuspid incompetence, and some of these subsequently showed evidence of an unusual lesion affecting the right ventricle. When there was severe pulmonary hypertension the *a* wave was increased, but giant *a* waves were not seen.

The praecordium and left chest was deformed by great cardiac enlargement in some of the younger patients. Palpation at the left sternal edge showed increased pulsation from right ventricular hypertension and hypertrophy. Pulsation over the pulmonary artery was palpable in most cases. Some degree of left ventricular hypertrophy could be felt at the apex in the majority, and in those with great mitral incompetence a powerful heave was followed by a diastolic rapid inflow beat.

All patients had an apical pansystolic murmur; in some this was palpable. A mitral diastolic murmur was not generally heard; however, in a few patients in whom active carditis was suspected a short mid-diastolic murmur was heard at some time. A third heart sound was audible in half the cases. The pulmonary second sound was always louder than normal, and in patients with evidence of severe pulmonary hypertension "splitting" was close and there was a pulmonary diastolic murmur in nine. There was no clinical evidence of aortic-valve disease in any of the patients, and this was confirmed by the absence of aortic-valve disease in the 13 cases that came to necropsy.

Congestive cardiac failure was present in half the patients when first examined; that these patients had not presented earlier appeared to be due to poverty and ignorance rather than the natural history of the disease. In 16 patients there were signs suggesting an active carditis in addition to those of established mitral incompetence and pulmonary hypertension. These patients had slight fever and tachycardia out of proportion to the rise in temperature; some had a transient mid-diastolic murmur, and others pericardial friction. Six of these patients had a high venous pressure, swelling of the face, and a tender liver without dependent oedema or ascites. Pulmonary oedema developed in two, which was in contrast with the rest of the patients in whom the evidence of selective left heart failure was minimal.

Radiology

The radiological signs were remarkably uniform. Pulmonary venous congestion (hilar congestion) was absent or minimal (Special Plate, Fig. 1). Septal lines were not seen. The peripheral lung fields were clear, as in other kinds of pulmonary hypertension. The main

pulmonary arteries were large and pulsated vigorously in some patients, but the branches were attenuated and there was never any suggestion of plethora. In some of the older patients with tricuspid incompetence and very large hearts the pulmonary arteries were less conspicuous (Special Plate, Fig. 2). The aorta was small. The heart shadow was always larger than normal. The right atrium was invariably larger and much more conspicuous than the left, which was never greatly enlarged. However, there was often a left auricular bulge below the prominence of the right ventricular outflow tract and pulmonary artery (Special Plate, Fig. 1). Systolic expansion of the left atrium was seen in a few patients. In advanced cases the great enlargement of the right atrium, the convexity of the upper left border, and the small vascular pedicle gave the heart a globular appearance.

Electrocardiography

Sinus rhythm was usual, but ventricular extrasystoles were common, as in other forms of mitral incompetence. One patient had atrial fibrillation, and another, who was thought to have active carditis, had transient first-degree heart-block. Electrocardiographic signs of right ventricular hypertrophy were present in all patients (Text Fig. 1). Right bundle-branch block occurred

TEXT FIG. 1.—Electrocardiograms from six patients, mounted from left to right in ascending order of right ventricular dominance. Notched and biphasic P waves indicate right and left atrial hypertrophy.

twice, and was also considered to indicate right ventricular hypertrophy. There was additional evidence of left ventricular hypertrophy in most cases. Monophasic R waves were present in V7 in most, and even when evidence of right ventricular hypertrophy was extreme only a shallow S appeared in V7. Large P waves indicated right atrial hypertrophy in all cases, and there was additional evidence of left atrial hypertrophy in the majority. Biphasic P waves in V1 were almost invariable (Text Fig. 1).

Cardiac Catheterization

Cardiac catheterization was performed by the usual technique, but in some instances there was difficulty in entering the right ventricle when the right atrium was greatly enlarged. Even when the right ventricle and pulmonary artery were successfully entered (15 cases) it was not always possible to obtain a satisfactory "wedge" pressure (10 cases). The pulmonary arterial

pressure was abnormally high in each case, and the "wedge" pressure, which was assumed to reflect the left atrial pressure, was also high. Cardiac output was low and the pulmonary vascular resistance was raised to 527, 713, and 541 dynes/sec./cm.⁵ in three cases (Nos. 16, 18, and 19) where calculation of this factor was possible. Other results are shown in Table I.

TABLE I.—Pressures (mm. Hg) Obtained at Catheterization in Right Atrium (R.A.), Ventricle (R.V.), Main Pulmonary Artery (M.P.A.), and Wedged Pulmonary Artery in 15 Patients

Case No.	Age (Yrs.)	Sex	R.A. Pressure	R.V. Pressure		M.P.A. Pressure		Wedge Pressure
				Absolute	Mean	Absolute	Mean	
2	27	F	+5	60/2	25	62/30	44	19
4	47	F	+4	92/4	46	90/38	61	—
5	8	M	+12	60/5	26	63/22	37	22
6	16	F	0	55/0	27	57/25	40	15
7	16	M	+4	62/4	35	60/35	45	24
9	28	F	+6	60/0	—	60/35	42	—
12	32	F	+12	85/5	43	83/44	58	28
14	14	F	+5	104/0	48	102/42	69	—
15	22	M	+8	70/5	—	68/30	—	20
16	41	F	+2	65/0	31	65/37	45	16
17	25	F	+4	80/0	31	81/38	51	—
18	15	M	+6	75/3	—	75/34	56	23
19	12	F	0	60/0	21	58/25	41	23
20	11	F	+2	100/0	42	98/43	62	28
21	27	M	+4	—	40	—	53	—

Other Investigations

All the patients had anaemia. Half of the men and one-third of the women had haemoglobin levels below 70%, and six children under 10 years of age had the lowest levels. The anaemia was always of the iron-deficiency type, though it was often refractory to oral administration of iron. The total and differential cell counts were within the normal range for this population. There was no evidence that significant unexplained eosinophilia was associated with the heart disease from which these patients suffered.

The incidence of abnormal haemoglobins was determined. One-quarter of the patients carried the sickle-cell haemoglobin, which agrees with the incidence in the general population of the region* (24.4%—Watson-Williams, 1960).

TEXT FIG. 2.—Relationship of antistreptolysin-O titre to age in 27 patients.

The erythrocyte sedimentation rate, measured in a number of patients, was always moderately raised; it seemed to bear no relation to the patient's heart condition or to the progress of the disease when serial estimations were made. As malaria, ancylostomiasis,

*Figures taken from an analysis of 5,730 blood donors.

and other infections are rife in Nigeria the significance of the E.S.R. in our cases could not be assessed.

It is well known that the plasma-protein pattern of Africans is unlike that of Europeans in that the albumin/globulin (A/G) ratio is reversed. Observations were made on only nine of our patients, and the plasma-protein values corresponded to the usual pattern for this population as described by Edozien (1958). The mean value for total proteins was 6.7 g. per 100 ml. The average serum albumin was 2.5 g. and the serum globulin 4.7 g. per 100 ml., giving an average A/G ratio of 1/1.88 (the increase being mainly due to γ -globulin).

Before determining the antistreptolysin-O (A.S.O.) titre the local normal was established in 100 Nigerian blood donors of all ages and both sexes, and was found to be the same as in Europeans (upper limit of normal 166 Todd units). The results in 26 patients in the present series are shown in Text Fig. 2. The relation between the onset of symptoms or start of the disease and the level of antibody titre could not be determined owing to the paucity of history. Over 50% (14) of the 26 patients had an A.S.O. titre above normal, and in 90% it was over 100 Todd units, whereas only 13% of the controls had an A.S.O. titre above normal, and in more than 50% it was below 50 units. Eight patients with a titre of 250 units or more were under 25 years of age. A.S.O. titres had been determined in six of the patients who came to necropsy (Table II).

TABLE II.—Antistreptolysin-O Titre and Necropsy Findings in Six Fatal Cases

Case No.	Sex and Age	A.S.O. Titre	Histology
14	F 15	100	Many Aschoff nodules
15	M 23	100	Aschoff nodules scanty and much fibrosis
24	F 15	250	Many Aschoff nodules
28	F 26	200	Non-specific myocarditis and endocardial thickening
29	F 25	100	Aschoff nodules and endocardial thickening
45	M 7	250	Aschoff nodules scanty and endocardial thickening

Necropsy Findings

Confirmation of the clinical diagnosis of mitral-valve disease causing mitral incompetence was obtained in the 13 patients who came to necropsy. No other valve was affected. However, examination of these hearts suggested that more than one disease process had led to an identical clinical picture. There was evidence of myocarditis in each heart; in some this was indistinguishable from rheumatic carditis, while in others the reaction appeared to be of a non-specific interstitial variety. Endomyocardial fibrosis of varying degree was present in some hearts irrespective of the kind of inflammatory reaction found histologically. All the patients had died of congestive cardiac failure, and one had recent ulcerative valvulitis.

The left atrium was moderately enlarged and its wall was thicker than normal, being up to 5 mm. in one case. This left atrial thickening was mainly due to muscle hypertrophy rather than endocardial hyperplasia, which was, however, present on the posterior wall in two cases. Each heart showed some degree of left ventricular hypertrophy (Text Fig. 3). There was a variable amount of macroscopic endomyocardial fibrosis affecting the inflow tract of the left ventricle (Text Fig. 4) and varying from a slight patchy opacification of the endocardium to a gross silting up of the apical lumen (Text Fig. 5). In the most severely affected hearts the

posterior papillary muscle and the valve chordae were incorporated in this process, and the posterior cusp of the valve was then greatly deformed (Text Fig. 5). In all cases the mitral-valve cusps were thickened, and in one case ulcerative endocarditis had arisen on the deformed valve.

TEXT FIG. 3.—Mitral valve and left ventricle. Chordae tendineae, particularly of posterior papillary muscle, are shortened; hypertrophy of left ventricle with patchy fibrosis. (P.C.=posterior and A.C.=anterior cusp.)

TEXT FIG. 4.—Endomyocardial fibrosis in left ventricle. Posterior papillary muscle is affected in this process; the outflow tract is almost free of endocardial thickening.

TEXT FIG. 5.—Endomyocardial fibrosis of left ventricle. A large thrombus—part calcified—can be seen at apex. Posterior papillary muscle is incorporated in ventricular wall (P.C.) and valve is grossly distorted and fibrotic.

A curious but common finding was an increase in the number of trabeculae carneae, which were thinner than normal and gave the endocardial surface a lace-like appearance. Fresh thrombi and partially organized ones were commonly found in the spaces between the trabeculae. The right ventricle was always thicker than normal, and in some hearts hypertrophy of the trabeculae had greatly diminished the size of the ventricular cavity; in other cases the right ventricular outflow tract was much dilated, producing a large oblique bulge over the anterior surface of the heart (Text Fig. 6).

TEXT FIG. 6.—Right atrium and ventricle, showing thickened trabeculae and dilated outflow tract. (T.V.=tricuspid valve; P=pulmonary valve.)

None of the hearts showed evidence of coronary-artery disease. The diameter of the pulmonary artery was always greater than that of the aorta, which was normal. Macroscopic evidence of previous pericarditis was present in only two cases.

On microscopical examination one or more of the following lesions were found: (a) "rheumatic" carditis with Aschoff bodies; (b) a non-specific inflammatory myocarditis; (c) fibrosis which was maximal in the endomyocardium.

(a) The presence of Aschoff bodies was the sole histological criterion for recognition of "rheumatic" carditis. They were typical of the Aschoff body found in the usual form of rheumatic carditis, having characteristic basophil cells with ragged edges and occasional fibroblasts. There was granular collagen between the cells, and scattered round cells occurred around the mass (Special Plate, Fig. 3). The nodes were usually spindle-shaped and adjacent to small blood-vessels (Special Plate, Fig. 4). This reaction was found mainly in the ventricular myocardium and was present in eight of the 13 hearts; in four it was the only abnormality, while in three it was associated with endomyocardial fibrosis, and in one other an intense rheumatic reaction was accompanied by a "non-specific" myocarditis. In contrast to the usual form of rheumatic carditis the mitral valve was only slightly affected. The other valves were normal. Aschoff nodules were also found in the muscle and endocardium of the atria.

(b) Three hearts showed a non-specific myocarditis which was focal and interstitial, with infiltration by eosinophil and mononuclear cells.

(c) Some degree of endomyocardial fibrosis extending for a varying depth into the left ventricle was present in three hearts with no other histological abnormalities; it was associated with scanty Aschoff-nodule formation in three hearts and with non-specific myocarditis in two others.

Histological examination of the lungs showed only slight evidence of vascular hypertrophy; most small arteries and arterioles were normal. Furthermore, the vascular pattern shown by post-mortem pulmonary angiography was normal in the case of a 15-year-old girl whose pulmonary arterial pressure was 102/42 mm. Hg (Case 14) one year before death.

Discussion

The constant clinical findings of a mitral pansystolic murmur, an overactive left ventricular apex-beat, a loud pulmonary second sound, and palpable pulmonary arterial pulsation, together with the radiological and electrocardiographic signs, clearly indicated the presence of mitral incompetence and pulmonary hypertension in these patients. Confirmatory evidence was obtained from cardiac catheterization, and the necropsies showed that there was severe myocardial disease in addition to organic mitral valvulitis and pulmonary hypertension. This syndrome appears to be common in Nigeria, in contrast to its rarity in Great Britain. Two features must be kept distinct in considering the pathogenesis of the condition: one concerns the aetiology of the cardiomyopathy, and the other the mechanism of the pulmonary hypertension and its association with mitral incompetence.

There are many causes of heart-muscle disease, and it is probable that at least two, and perhaps more, are concerned in this disease. Aschoff nodules in the myocardium and rather high antistreptolysin-O titres suggest that there was a form of rheumatic carditis in some cases, though rheumatic fever is very uncommon in Nigeria. It is rare for these people to complain of a sore throat, but it is possible that the haemolytic streptococcus may gain entry by an entirely different route, such as the common tropical leg ulcer. Chorea is rare and arthralgia is equally uncommon, but those affected would not complain unless there was extreme disability. In any case, we might expect that the rheumatic reaction would be different in people suffering from chronic anaemia and chronic infection, and in whom the globulin pattern of the plasma proteins is quite different from that of Europeans. Heart-muscle disease is certainly very common in West Africans, and it may be that the myocardium, having suffered from the effects of anaemia and chronic infection from birth, may be more vulnerable than the endothelium of valves or the soft tissues of joints.

Necropsy on 13 patients in the series showed that a significant proportion had some degree of endomyocardial fibrosis. It is not suggested that this is caused by rheumatic carditis, but it appears that cardiomyopathy, whether due to inflammation, nutritional deficiency, or other metabolic disorder, may during an acute phase precipitate mural intratrabecular clots, which subsequently become organized. This process may be recurrent and lead to endomyocardial fibrosis.

There have been many accounts of cardiomyopathy in West Africans since Bedford and Konstam (1946) first drew attention to this condition. Davies (1948) described an apparently similar form of heart disease in Uganda,

calling it endomyocardial fibrosis, and subsequent papers from Uganda have extended the original observations (Ball *et al.*, 1954; Williams *et al.*, 1954). Endomyocardial fibrosis has now been reported from many parts of the world, including the Sudan (O'Brien, 1954), Nigeria (Nwokolo, 1955), Rhodesia (Gelfand, 1958), Ceylon (Nagaratnam and Dissanayake, 1959), Malaya (La Brooy, 1957), Hawaii (Stemmermann, 1957), America (Fienberg and Holzman, 1951; Clark *et al.*, 1956; Fisher and Davis, 1958), Great Britain (Gray, 1951; Lynch and Watt, 1957; Evans, 1957), and Holland (Van Buchem *et al.*, 1959). Most of these reports have been concerned with morbid anatomy and theoretical discussion of aetiology. Recently one of us* has seen the syndrome of mitral incompetence and pulmonary hypertension in the French Congo at Brazzaville, where it is common (Peuchot, 1959), and in Uganda, where both clinical cases and necropsy specimens were seen. Some of these cases were identical with those described here. Patients with the same condition were also seen in Khartum and Accra, but none was seen in South Africa or the Rhodesias (Baldachin, 1959); the cardiovascular collagenosis with endocardial thrombosis described by Becker and his colleagues (1953) is different.

The aetiology of endomyocardial fibrosis remains obscure. Infection, malnutrition, avitaminosis, parasitic infestation, and allergy have all been discussed in the papers mentioned. In the present series endomyocardial fibrosis of the left ventricle was clinically indistinguishable from rheumatic mitral incompetence, and in several instances both coexisted in the same heart. It is debatable whether this association is casual or causal. The possibility that endomyocardial fibrosis and rheumatic heart disease are related is now being considered in Uganda (Shaper and Williams, 1960). It seems probable that endomyocardial fibrosis is the "final common path" resulting from many different forms of myocardial damage. In Southern Nigeria we suggest that rheumatic carditis may be concerned with this process in some cases.

The dominance of mitral incompetence rather than stenosis is interesting. An analysis of patients with mitral-valve disease attending the hospital at Ibadan (Table III) shows that a combination of mitral incom-

TABLE III.—Analysis of Mitral-valve Disease in Out-patients (April–September, 1959)

Lesion	No. of Cases
Pure mitral stenosis (with or without pulmonary hypertension)	4
Pure mitral incompetence (without any evidence of pulmonary hypertension)	2
Mitral stenosis and incompetence	10
Combined mitral and aortic valve diseases	2
Mitral incompetence with pulmonary hypertension	43
Total	61

petence and pulmonary hypertension is common, in contrast to experience in the United Kingdom, where pure mitral incompetence is uncommon and rarely associated with pulmonary hypertension (Brigden and Leatham, 1953). Several factors, including valvulitis, endomyocardial fibrosis, and diffuse myocardial disease, are responsible for this dominance of mitral incompetence. Furthermore, it is probable that in some cases the myocarditis combined with so many unfavourable factors, including continued physical activity, results in death before stenosis can occur.

There were clinical, electrocardiographic, and radiological signs of pulmonary hypertension in all patients in this series. High pulmonary arterial pressures were

*D. G. A. (with a grant from the Rockefeller Foundation).

confirmed by cardiac catheterization. There was evidence of chronic elevation of left atrial pressure apart from the raised "wedge" pressures obtained at catheterization. The electrocardiogram showed left atrial hypertrophy as in mitral stenosis, and at necropsy there was great atrial hypertrophy without dilatation in some cases. Severe left atrial hypertension is in contrast to the simple systolic type of hypertension which exists in pure rheumatic mitral incompetence in Europeans. In these patients the ventricular diastolic inflow may be impeded by stiffness of the hypertrophied myocardium associated, in some cases, with the occlusive and splinting effects of endomyocardial fibrosis. It is therefore probable that the pulmonary hypertension is, as in many cases of mitral stenosis, due to the passive effects of the high left atrial pressure coupled with a vasoconstrictive element at a precapillary level. It remains to mention the possibility that pulmonary vasoconstriction is an excessive response to the pulmonary venous hypertension because of chronic anaemia or some other unknown factor. Left atrial hypertrophy, in contrast to dilatation, parallels the absence of atrial fibrillation. It is probable that atrial systole is an essential component in the maintenance of a cardiac output sufficient to sustain life in these patients.

Finally, we wish to stress the importance of the soil in which this form of heart disease thrives. Anaemia is usual; intestinal parasitism is rampant; in addition malaria is endemic, and the dysenteries, both bacterial and protozoal, are widespread. Protein malnutrition is rife, especially in infants and children. In the presence of persistent chronic ill-health of this nature it is difficult to believe that the myocardium remains unaffected and healthy. It is probable that anaemia alone, when severe and protracted, may lead to cardiomyopathy (Somers, 1959). On top of all this it must be emphasized that these patients are always ambulant, and never rest unless it is physically impossible for them to stand and walk. This continued physical activity may have further adverse effects upon an already damaged myocardium.

Summary

The clinical, radiological, and electrocardiographic features of 50 Nigerian patients who presented with heart failure due to mitral incompetence and pulmonary hypertension have been investigated. Necropsy examination of 13 cases showed that there was severe left ventricular myocardial disease and mitral-valve disease. Most hearts showed some degree of macroscopic endomyocardial fibrosis, and histological examination revealed either a diffuse non-specific inflammatory reaction or a myocarditis with Aschoff nodules which was indistinguishable from rheumatic carditis.

It is suggested that the peculiar pattern of this disease, comprising left ventricular myopathy, mitral valvulitis, and pulmonary hypertension, may be determined not only by its primary cause, but also by the background of chronic anaemia, protein malnutrition, and chronic infestation which is present in these patients and in the population from which they are drawn.

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ADRENOCORTICAL STEROIDS IN INTERMITTENT HEART-BLOCK

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Stokes-Adams attacks, by their dramatic nature alarming to the patient and his relatives, are always a matter of grave concern when the usual treatment with sympathomimetic drugs and atropine fails. Until recently there was no other effective simple treatment. There is now, however, growing evidence of the value of adrenocortical steroid therapy in this syndrome.

Prinzmetal and Kennamer (1954), in a paper devoted to treatment of various cardiac arrhythmias, described, almost as a curiosity, the response to corticotrophin of a patient with complete heart-block and Stokes-Adams attacks. A man of 60, after an acute posterior cardiac infarct, had been having repeated and prolonged attacks of syncope for a week with no relief from ephedrine or adrenaline. As a last resort, 40 units of corticotrophin was given: the attacks ceased and normal sinus rhythm returned in a few hours. Relapse a few days later again responded almost immediately, and after 10 days' maintenance treatment heart-block did not recur. Phelps and Lindsay (1957) described a very similar case wherein intramuscular cortisone was effective.

Since then, patients with Stokes-Adams attacks of long standing, associated with chronic heart disease, have been successfully treated with corticotrophin and adrenocortical steroids.

Litchfield *et al.* (1958), using corticotrophin, obtained immediate and permanent freedom from attacks in a man of 35 with ankylosing spondylitis who developed right bundle-branch block and intermittent complete heart-block. The attacks had occurred for nine months, continuing despite ephedrine and frequent injections of adrenaline during six weeks in hospital. A second patient, a man of 45 with rheumatic aortic valvular disease and paroxysmal complete heart-block, had

D. ABRAHAMS AND W. BRIGDEN: MITRAL INCOMPETENCE AND PULMONARY HYPERTENSION

FIG. 1.—Chest radiograph of a girl aged 16 with mitral incompetence and pulmonary hypertension. The aorta is inconspicuous; main pulmonary arteries and outflow tract of right ventricle enlarged.

FIG. 2.—Radiograph showing a late stage of mitral incompetence and pulmonary hypertension when tricuspid incompetence has supervened. Right auricle is large; peripheral vascular markings are diminished.

FIG. 3.—(a) Aschoff nodule showing collagen debris. ($\times 76$.) (b) Aschoff nodule showing one of the rare giant cells (G). ($\times 236$.)

FIG. 4.—Aschoff nodules adjacent to a small vessel. ($\times 228$.)

L. S. TAITZ *ET AL.*: ACUTE NEPHRITIS ASSOCIATED WITH CONGENITAL SYPHILIS

FIG. 1.—Glomerulus from kidney of Case 1, showing hypercellularity and adhesion between tuft and Bowman's capsule. (H. and E. $\times 260$.)

FIG. 2.—Markedly swollen glomerulus. (H. and E. $\times 260$.)

FIG. 3.—Eosinophilic necrosis of glomerular tuft. (H. and E. $\times 260$.)