Current Practice

MEDICINE IN THE TROPICS

Diagnosis and Management of Heart Failure in the Young Adult African

E. H. O. PARRY,* M.D., M.R.C.P.

In many contexts the use of the word "African," referring to all the indigenous inhabitants of Africa together, is rightly being abandoned, but it is still relevant in cardiac disease. Whereas some disorders appear to be limited to certain fairly well defined parts of the continent—for example endomyocardial fibrosis in the hot and wet countries—most of the other common diseases occur in every country. One can therefore write about "the adult African" with cardiac failure, and it is also reasonable to consider the typical patient as a peasant farmer or an unskilled labourer, because such men or their families are seen far more commonly than any other class of patient in the hospitals of tropical Africa.

The patient usually seeks help when he is so beleaguered by a failing heart that he can go no further, and the problem is one either of pulmonary oedema demanding urgent action or of massive peripheral oedema. The man with pulmonary oedema is a familiar sight—distressed and coughing, he breathes quickly and has a rapid heart beat, and showers of crepitations in the chest may overshadow all other signs. It may be impossible to get a clinical history at this stage, but in less severely ill patients a long history of nocturia may indicate chronic renal disease, while pain in the front of the chest, which may be confused with dyspnoea by the patient, may point to pericardial disease, and in a woman a history of a recent pregnancy or of continued breast-feeding may make "puerperal" heart disease possible.

Physical Examination

Even in the acutely ill patient peripheral and general physical signs should be defined, because these can often help in diagnosis and may materially affect treatment: fine silken hair, opaque finger-nails, and abnormally pigmented skin, which may be shiny, cracked, and desquamating over swollen legs, make significant hypoalbuminaemia probable; uraemic frost—the pigmented face and trunk look as if a white powder has been dusted lightly over them—can be a useful sign of terminal renal failure; the active breasts of a woman may point to "puerperal" heart disease; clubbed fingers can be the only peripheral sign of bacterial endocarditis, or there may be the remains of a carbuncle which has been the source of a bacteraemia; profound anaemia may be missed if the tongue and mucosae are not examined; and fresh scarifications or the marks of "therapeutic" burns may show the site of recent pain in the chest.

Pulmonary Oedema

The adult African with pulmonary oedema commonly has either a failing left ventricle or mitral stenosis. When the cardiovascular system and the chest are examined an aetiological or pathological diagnosis is less important at first than a functional and an anatomical diagnosis, because treatment is primarily directed towards reversing the disorder of function which threatens life. The diagnosis of pulmonary oedema is not difficult when the orthopnoeic patient coughs up copious frothy pink sputum. Occasionally, patients with rapidly advancing lobar pneumonia present in this fashion. In such patients when cardiac signs are not found the sputum should be stained by Gram's method and a thin blood film stained by Leishman's stain. Gram-positive diplococci in the sputum and many young neutrophils in the blood should leave no doubt of the diagnosis of pneumonia. Impending pulmonary oedema may be more difficult to define because the patient may have only a cough, a tachypnoea, and a tachycardia, and no auscultatory signs in the chest at all. In such a patient signs of abnormality in the heart are a great help to the correct diagnosis.

"African Cardiomyopathy"

The young adult African who presents with pulmonary oedema may well have the cardiomyopathy which is common throughout Africa. In this disorder there is no primary lesion of the valves or of the endocardium, but the muscle of the heart is abnormal. No specific cause has been found, though excessive drinking of alcohol may be contributory, particularly among palm-wine tappers.

This disorder has been named "cryptogenic cardiomyopathy," "African cardiomyopathy," or "heart muscle disease." At necropsy the heart is hypertrophied and dilated, but there are no other specific findings. Though it is commoner in older adults, the young adult is not spared this disorder, and he may be first seen in actual or impending pulmonary oedema. The most prominent physical signs are often an abnormal blood-pressure (systolic pressure around 140 mm. Hg, and diastolic pressure around 100–120 Hg), a rapid arterial pulse with a small pulse pressure, a high jugular venous pressure with no consistent pattern in the pulse wave, and an abnormal heart in which the left ventricle either is palpable as a forceful impulse displaced to the left in the 6th or 7th interspace, or is very quiet. A gallop sound at the apex of the heart and a pansystolic murmur of mitral or tricuspid regurgitation can be heard in most patients. The cardiac shadow in the chest radiograph may be difficult to define in a patient with pulmonary oedema, but in the typical case the entire heart is enlarged, particularly the left ventricle. If an electrocardiogram can be taken it may show either signs of left ventricular hypertrophy or a low voltage graph with flat or inverted T waves in most leads.

Differential Diagnosis

The distinction of this cardiomyopathy from other disorders in which there is left ventricular hypertrophy can sometimes

* Wellcome Fellow, Department of Medicine, Haile Sellassie I University, Addis Ababa.

be very difficult. Hypertensive heart failure can usually be recognized by a sustained and even higher blood-pressure than that found in some cases of the cardiomyopathy, and also by retinopathy. (Examination of the ocular fundus is of inestimable value in the diagnosis of heart disease in the adult African, though bright sunlight may make it difficult.) The urine of the young hypertensive in Africa almost invariably contains protein, and the centrifuged deposit should give further evidence of the chronic renal disease which is so commonly the cause of his hypertension.

When the pansystolic murmur of mitral regurgitation occurs in patients with African cardiomyopathy it is probably due to a dilated mitral ring, and thus often disappears after effective treatment. Such patients, however, must be distinguished from those who have other diseases in which mitral incompetence and an easily palpable left ventricle are found. Rheumatic heart disease, widespread in Africa, is probably the commonest single cause of mitral incompetence. It can be diagnosed easily if there are also signs of mitral stenosis, or of an aortic valve lesion. If an associated valvular lesion is not found and if the patient is a young adult from the countries of the west coast of Africa, Uganda, or the Congo territories, lone mitral incompetence due to rheumatism may be impossible to distinguish from mitral incompetence due to left ventricular endomyocardial fibrosis (E.M.F.). Some patients with E.M.F. have rapidly advancing disease and pulmonary oedema may occur early. In others a functional diagnosis can be made early, but only a partial anatomical diagnosis can be made without the aid of angiocardiography—a very rare luxury in tropical Africa. When an early and relatively high-pitched third heart sound is heard at the cardiac apex it may help in the diagnosis of E.M.F. The chest radiograph shows a heart which is a little enlarged but in which the left atrial shadow is usually smaller than in similar cases of rheumatic mitral incompetence.

A rare condition described from the tropics and defined more particularly at Ibadan, Nigeria, is annular subvalvular aneurysm of the left ventricle. The pathogenesis of this condition is uncertain, but it may lead to catastrophically acute mitral incompetence. The diagnosis has academic but little practical value, and it can be suspected if there is a peculiar double impulse at the apex of the heart and an unusual bulge on the border of the heart in the chest radiograph.

Bacterial Endocarditis

One of the most devastating heart diseases in the young adult African is bacterial endocarditis. If the aortic valve is infected, the patient may be first seen in pulmonary oedema, and his large left ventricle may lead to an erroneous diagnosis of "African cardiomyopathy." The diagnosis of bacterial endocarditis can be suspected, however, if obvious signs of aortic valve disease are found together with clubbing of the fingers, pallor, and fever. In some patients with aortic regurgitation there is such a tachycardia that the aortic murmurs have the cadence of a pericardial rub: in others, the familiar peripheral signs of a wide pulse pressure and a "collapsing" arterial pulse may not be found. In a patient in whom infective endocarditis is suspected the ocular fundi should always be searched for haemorrhages or exudates. Though the sophisticated history of a dental extraction will not be obtained, there may well be evidence of a source of organisms—for example, pelvic sepsis in a woman recently delivered of a baby, more than the usual pyorrhoea, or the remains of a recent boil or carbuncle.

It is dangerous to think of "subacute" bacterial endocarditis in the young African: by the time the patient is first seen his disease may be advanced and advancing. Simple side-room investigations can be a great help. In most cases there is a high erythrocyte sedimentation rate and a lowered haemoglobin: when the pneumococcus or the staphylococcus is the responsible organism there is neutrophil leucocytosis. The deposit from a centrifuged sample of clean mid-stream urine should always be examined. Red blood cells may be found in possible cases of bacterial endocarditis, but in patients in whom the disease advances rapidly excess pus cells are often a more valuable sign. If there are facilities for blood culture this should be done.

It is essential to be alert for bacterial endocarditis in any young African patient with obscure cardiac signs and evidence of infection, and there must be good reasons if antibiotics are to be withheld from him when bacterial endocarditis is possible.

Mitral Stenosis

Rheumatic heart disease is common throughout Africa, and many patients with mitral stenosis are seen for the first time with pulmonary oedema. This is the only common disorder in which pulmonary oedema can occur in the African without evidence of an abnormal left ventricle. There should therefore be little problem in differentiating such patients from those described above with left ventricular hypertrophy and pulmonary oedema. When the patient is first seen rapid atrial fibrillation and signs of pulmonary oedema may obscure all other signs, and so a functional diagnosis only can be made. The anatomical diagnosis should not remain in doubt for long, however, because a diastolic murmur at the cardiac apex will be heard when the ventricular rate is slowed by digoxin; an inconspicuous apex beat and prominent pulsation of the right ventricle at the left border of the sternum may also be helpful signs.

Cardiac Failure with Systemic Venous Congestion

It is in some ways artificial to distinguish between patients who have systemic oedema and those who have pulmonary oedema, particularly when one disease may be responsible for both clinical presentations. But the young African with much systemic oedema may be the victim of one of a number of diseases which do not affect the heart at all, but which can sometimes be confused with cardiac failure. Thus the puffy-faced oedematous patient may have the nephrotic syndrome: heavy and consistent proteinuria should confirm this diagnosis. If the nephrotic syndrome is due to amyloid disease secondary to tuberculosis it will be difficult to prove unless a renal or rectal biopsy can be studied histologically.

The young African who has protein malnutrition and an associated anaemia may first be seen swollen all over and short of breath, and it is often only when no abnormal cardiac signs are found that the history is elicited of deplorably little protein in the diet or of persistent diarrhoea which has led to excessive loss of protein in the stools. If signs of hypoalbuminaemia are found first in such patients, a quicker diagnosis may be possible. When the causes of oedema due to general disease are remembered, the diagnosis of patients with cardiac failure and widespread oedema, with or without evidence of valvular disease, will be simpler.

Valvular Disease

The diagnosis of rheumatic heart disease with obvious valvular lesions is not difficult unless there are signs of mitral and tricuspid regurgitation only, when the distinction from biventricular E.M.F. may be impossible at the bedside. Before a diagnosis of E.M.F. is suggested its probable geographical limits in Africa should be remembered. Because the endocardial fibrous tissue in E.M.F. occurs in the inflow tract of the ventricle—either left or right or both, and because it anchors the papillary muscles at their bases, atrio-ventricular valvular incompetence is produced, and stenosis cannot occur. The effect of treatment may help in differentiating the two diseases, as tricuspid incompetence due to E.M.F. is irreversible, while in rheumatic heart disease it may disappear.

When E.M.F. affects the right ventricle alone, evidence of valvular disease may be missed altogether. Such patients have much ascites, little or no oedema of the ankles, and are often confused with those who have cirrhosis of the liver because the cardiac signs may be difficult to elicit. Thus the systolic wave in the jugular pulse due to tricuspid regurgitation is often so high that it can be seen only as a pulsation under the lobe of the ear even when the patient is standing up; the cardiac impulse is very quiet because the aneurysmal right atrium occupies the front of the heart; no murmur of tricuspid regurgitation may be heard because the right atrium and ventricle are virtually one chamber; and the early third heart sound may be mistaken for a "split" second heart sound. Often the confusion is complete unless a chest radiograph can be done: this will show a very large heart, which may resemble a pericardial effusion. Some cases of right ventricular E.M.F. may be mimicked by constrictive pericarditis, but in this condition instead of a single systolic wave in the jugular pulse there are two waves, and a chest radiograph should decide the issue if there is a small heart with calcium round it.

Myocardial and Other Diseases

The young African adult who is not in pulmonary oedema, who has no manifest valvular lesion, and yet who has signs of cardiac failure can be a difficult problem. Hypertensive heart disease should not be difficult to define, but some cases of African cardiomyopathy can be puzzling. For example, patients whose left ventricle has a feeble impulse instead of the more usual forceful beat may well be confused with those with a pericardial effusion. In both disorders inspiration often makes the venous pressure rise and the arterial pulse pressure fall (pulsus paradoxus), the cardiac impulse may be impalpable, a tachycardia and a gallop sound may be heard, and the heart may be diffusely enlarged on the chest radiograph. The electrocardiogram may not help, because a low voltage graph with flat T waves can occur in both conditions, although more commonly in cases of cardiomyopathy there are electrocardiographic signs of left ventricular hypertrophy.

If a pericardial effusion is suspected the pericardial sac should be tapped. The epigastric approach is the safest and easiest. A fine hypodermic needle is used to infiltrate the skin and subcutaneous tissues just below the xiphisternum with 1% or 2% procaine hydrochloride. The syringe is then fitted with a No. 1 needle, which is directed through the skin upwards, backwards, and slightly to the left: it should be long enough to puncture the pericardial sac easily. Throughout the procedure the patient should be sitting up with his head and chest supported by a pile of pillows. If fluid is obtained, a thin film should be stained with Leishman's stain to define the morphology of the white cells. The total white cell count of the fluid should be done in the same way as the white cell count in blood. The total protein content should be estimated, but if this is impossible the fluid can be left in a pot, and if a fibrous clot forms it is safe to conclude that the total protein content is high, which indicates an inflammatory process.

Only in cases in which an acute pericardial effusion causes cardiac tamponade is it necessary to aspirate the effusion as completely as possible. In all other cases a diagnostic tap of 10 ml. is quite adequate. Unless it is a complication of lobar pneumonia or of an amoebic liver abscess, an acute pericardial effusion is almost invariably due to tuberculosis; and it is patients with an acute effusion, rather than those with a chronic effusion, who present a picture like that of myocardial failure.

One particular form of myocardial failure which has signs very similar to those described above is limited to women at or around childbirth or while they are breast-feeding: it is called either "puerperal" heart disease or "heart failure of the nursing mother." Signs of cardiac failure develop quickly. The pathogenesis of this disorder is unknown. It may be related in some way to continued lactation, because if this is suppressed—a desperate and drastic measure in the young African woman—some patients get better. Lactation should be allowed to continue unless rigorous routine treatment for cardiac failure does not help.

Finally, patients with severe anaemia in tropical Africa may present with cardiac failure: for example, the young farmer with hookworm infection, or the pregnant mother with megaloblastic anaemia of pregnancy. The classical signs of a hyperkinetic state may not be found in patients with cardiac failure due to hookworm disease, probably because some have hypoalbuminaemia, and in these the blood volume may be low. Other causes of cardiac failure in the young adult African are much less common than the everyday conditions which have been described above.

Treatment

Pulmonary Oedema

The principles of emergency treatment are: (1) to reduce the work of the heart and to improve its power to contract, (2) to relieve the patient's anxiety and the indulgent family's distress, and (3) to promote a rapid diuresis.

Action must be rapid and decisive. The patient should be helped to sit up, resting against pillows, with his legs down and the head of the bed raised on blocks (12 in.—30 cm.) high. Venous occlusive tourniquets should be tied round the thighs as close to the groin as possible: these will produce a substantial "bloodless venesection," so that an actual venesection of about 500 ml. will not be necessary. For a tourniquet anything that will tie will be all right—rubber tubing, linen, or a shirt—but at all costs the arterial pulses in the legs must not be obliterated. The tourniquets should be left on for 30-minute periods and should be released alternately from left and right legs for a few minutes each at the end of 30 minutes: they can be taken off when the patient is quiet and florid signs of pulmonary oedema have gone. Next, 1 mg. of digoxin in 10 ml. normal saline should be injected intravenously in 1–2 minutes. The needle should be left in the vein for the next drug—morphine.

Most patients are restless and will need morphine hydrochloride in a dose of 10–20 mg.: it should be injected through the needle used for digoxin. There is no place for the family, who should be put outside and kept outside as courteously and as firmly as possible at the beginning.

A brisk diuresis can be produced if a rapidly acting diuretic —for example, frusemide 80 mg. or ethacrynic acid 100 mg.— is given. If these drugs are not available, mersalyl 2 ml. by intramuscular injection is the best drug to use. If in spite of all that has been done the patient is slow to improve aminophylline should be given intravenously in a dose of 0.5 g. in 20 ml. over a period of about 10 minutes: if this drug is given too quickly the patient may sweat profusely and the blood-pressure may drop.

After the immediate crisis is over further treatment can be planned. Digoxin 0.5 mg. by mouth should be given at six-hourly intervals for four doses and then the drug should be continued at a dose of 0.25 mg. twice daily. These doses and the intravenous dose of 1 mg. will be adequate whether there is atrial fibrillation or sinus rhythm. If renal failure is suspected the doses should be reduced by half because the drug will be excreted more slowly.

For the first few days rest in bed is desirable but may be difficult to enforce. A diuretic should be continued: mersalyl 2 ml. on alternate days is probably the best drug, and it will satisfy both the patient's need for a diuresis and his appetite for an injection. The effects of diuretics are easily measured if the patient is weighed daily: if the drug which is being used is not effective another should be tried.

In patients with hypertensive heart failure an effective fall of blood-pressure is desirable. Reserpine in a dose of 2.5 mg. by mouth or by intramuscular injection will lower the blood-pressure smoothly. More powerful hypotensive drugs have little place in the treatment of the young adult African because only exceptionally is there adequate nursing staff, and after the crisis is over the patient may live far away from the follow-up clinic, even if he could afford to buy the expensive drugs which he needs.

Unless associated or complicating diseases are tackled as soon as emergency treatment is over they may prevent the patient from getting home and back to work quickly, which for the labourer may mean the end of his job.

Systemic Oedema

Digoxin and a diuretic are the sheet anchors of treatment. The dose of digoxin is similar to that described above, but there is no need to give the first dose of 1 mg. intravenously unless there is atrial fibrillation with a rapid ventricular rate. Mersalyl is again ideal as a diuretic, but for treatment out of hospital an oral diuretic such as hydrochlorothiazide 50 mg. will be easier to manage. It should be given often enough to prevent oedema forming: the dose will vary from 50 mg. weekly to 50 mg. daily. If possible, potassium chloride 1 g. three times a day should be taken on days when the diuretic is taken. One of the least palatable measures, which is often necessary in the very oedematous patient, is to restrict salt in his food. When relatives bring food to him in hospital this may prove almost impossible, but it is well worth trying to stop his family putting salt into the pot or giving him foods rich in sodium. When there is much ascites, which is very common in the African with cardiac failure, it should not be tapped unless the abdomen is very tense: instead, diuretics and a diet without added salt should be tried first.

Summary

The young adult African is often seen with advanced cardiac failure.

When pulmonary oedema overshadows all other signs a functional diagnosis only is possible. Some of the common causes of pulmonary oedema in the African are mentioned and their diagnosis is discussed.

Patients with cardiac failure and much systemic oedema can be confused with those in whom oedema is due to other causes, for example hypoalbuminaemia. Problems in the diagnosis of these patients are discussed.

Treatment is urgent in cases of pulmonary oedema. Follow-up treatment is governed as much by the patient's means and the distance of his home from hospital as it is by his disease.

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DISEASES OF THE SKIN

Management of Disorders of the Nails

PETER D. SAMMAN,* M.D., F.R.C.P.

The management of nail disorders is far from satisfactory at the present time. Very little is known about the pathology of these conditions, and though most nail deformities are of little importance from a functional point of view they are of great cosmetic importance.

There are three nail diseases which are often seen by the dermatologist: chronic paronychia, fungal infections, and psoriasis. They are very often misdiagnosed. In addition, patients present with one of a number of nail symptoms, the causes of which are often quite unknown, and treatment for which must therefore be empirical.

Chronic Paronychia

Chronic paronychia is quite the most common nail disorder for which the patient seeks advice. It is a disease mainly of women, and of women who have their hands constantly in water. It embraces, therefore, housewives (and all others who do much housework), bar tenders, laundry hands, nurses, and many others. It is more likely to affect people who suffer from cold hands. The characteristic features are the loss of the cuticle, bolstering and discomfort of the posterior nail fold, and the appearance of occasional beads of pus in the groove at the nail base opened up by the loss of the cuticle. From time to time a more acute inflammation may occur for a few days. Nail deformity appears later and most often consists of roughness and discoloration of one edge of the nail. Less often the nail becomes cross-ridged, and in long-standing cases there may be greater deformity. Occasionally the nail may be reduced in size, a feature which is exaggerated by the great bolstering which may by now completely surround all but the free edge of the nail.

The aetiological factors in chronic paronychia are not universally agreed, but it is certainly not a true fungal infection. Monilia, usually *Candida albicans,* can be recovered from the nail fold in almost every case,[1] but bacteria also play a part, especially in the more acute exacerbations. On culture a variety of organisms may be found, including *Pseudomonas aeruginosa,* which is probably responsible for the discoloration at the edge of the nail.[2]

From what has already been said it is obvious that griseofulvin can play no part in the treatment of this disorder. By far the most important part of treatment is to keep the hands dry. Unfortunately, this is often not possible, and treatment accordingly is not successful. The patient should be advised to wear rubber gloves for all wet work, preferably with thin cotton gloves under the rubber. Local applications are of less importance. In the early stages nystatin ointment is often the most useful preparation, and it should be applied frequently to the groove between the nail and the nail fold. Later, greater benefit may be obtained from the use of 15% sulphacetamide in 50% surgical spirit or 2–4% thymol in chloroform. Both these preparations help to dry out the nail

* Physician, Dermatological Department, Westminster Hospital, London S.W.1, and St. John's Hospital for Diseases of the Skin, London W.C.2.