Acute Reversible Heart Failure in Africans

By H. Grusin, M.B., B.Ch., M.R.C.P. (Lond.)

Sixteen cases of acute reversible heart failure were observed in the native African. On the basis of their responses to dietary and vitamin therapy they were divided into various etiologic groups. The clinical manifestations are described and the relationship of these cases to idiopathic cardiac hypertrophy of Africans is discussed.

Acute reversible heart failure is common among Africans (Bantu) living in and around Johannesburg. In some cases the temporary heart failure is due to some obvious cause such as acute nephritis, anemia, diphtheria, or pulmonary embolism, but in many patients the etiology is obscure. Gillanders described a type of heart disease that in its early stages could be reversed by an adequate diet, but usually progressed to chronic heart failure when the patient returned to his deficient home diet. Other types of heart failure are seen, however, that apparently disappear rapidly and completely (fig. 1). In this study a series of such cases has been collected and an attempt made to classify the various syndromes observed.

Material and Methods

The subjects of this study were Bantus living in or near Johannesburg. Like many of their kind they had subsisted for years on an inadequate diet containing small quantities of animal protein and fat and a high proportion of carbohydrate in the form of maize. Most were alcoholics who drank brandy and unmatuerd homemade brews known locally as “babaton” and “skokiaan.” The following groups of patients were studied: 1. Sixteen successive cases of acute heart failure, i.e., patients with a short history who made a complete clinical and radiologic recovery within 3 to 4 weeks. Obviously many had to be judged retrospectively. 2. Ten cases of edema not due to renal or cardiac causes. 3. A control group of 7 cases of chronic, recurrent heart failure.

All patients were confined to bed; their weight and urinary output were recorded daily. They were maintained for 5 to 10 days on a diet low in thiamine content but adequate in other respects.* If they showed no clinical improvement on this regime, then vitamin B1 was added for a further 5 to 10 days (thiamine hydrochloride 100 mg. twice a day intramuscularly). If there was still no response, digitalis and mersalyl were administered (tablets of digitalis leaf, 9 gr. daily for 3 days and 3 gr. daily thereafter; mersalyl 2 ml. intramuscularly on alternate days; ammonium chloride 15 gr. three times a day).

The rice was washed, boiled rapidly in 500 ml. of water for half an hour. After cooking it was strained and washed under running water until completely free of cooking water. It was divided into 3 equal portions for each of the 3 meals.

The meat used was thin cuts of sirloin steaks fried in a portion of the butter ration. Both rice and meat were reheated in steam ovens for 10 minutes before serving.

Canned peaches were drained of syrup and washed with cold running water. Dried peaches were washed and boiled rapidly for three quarters of an hour in water. The cooking water was drained off and the peaches were again washed under running water, reheated with additional water and sweetened with sugar. Desserts were prepared with either tapioca, sago or cornflour, sugar, butter, and water. They were flavored by caramelizing the sugar or by the use of commercial jelly crystals.

Weak tea with sugar but no milk was served with breakfast, midmorning, midafternoon, and supper meals.

Observations. On the basis of their response to these regimens the 16 cases of acute heart failure fell into the following groups (table 1): 1. Ten cases responded completely to thiamine (beriberi heart disease). 2. Three cases were unresponsive to thiamine (syndrome A). 3. Three cases responded completely on a thiamine-low diet (syndrome B).

<table>
<thead>
<tr>
<th>Food</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice (polished)</td>
<td>200 Gm.</td>
</tr>
<tr>
<td>Canned peaches</td>
<td>100 Gm.</td>
</tr>
<tr>
<td>Cheddar cheese</td>
<td>25 Gm.</td>
</tr>
<tr>
<td>Sugar and glucose sweets</td>
<td>100 Gm.</td>
</tr>
<tr>
<td>Tapioca, sago or cornflour</td>
<td>100 Gm.</td>
</tr>
<tr>
<td>Meat</td>
<td>100 Gm.</td>
</tr>
<tr>
<td>Dried peaches</td>
<td>100 Gm.</td>
</tr>
<tr>
<td>Butter</td>
<td>50 Gm.</td>
</tr>
<tr>
<td>Jelly (commercial jelly crystals)</td>
<td>60 Gm.</td>
</tr>
<tr>
<td>Ascorbic acid</td>
<td>0.05 Gm.</td>
</tr>
<tr>
<td>Halibut liver oil</td>
<td>0.2 ml.</td>
</tr>
</tbody>
</table>

From the Baragwanath Hospital and the University of the Witwatersrand, Johannesburg, South Africa.

* Diet consisted of raw edible weights of the following:
patients had signs of mild congestive cardiac failure. None looked ill or distressed and only 2 had a few crepitations over the lung bases. The venous engorgement was usually moderate (jugular venous pressure to the level of the hyoid with the patient sitting at 60°), the liver was enlarged 1 to 4 fingerbreadths and was slightly tender. All patients had edema of the feet and sacrum; the face was affected in 5. They lost from 10 to 25 pounds in weight during their hospital stay.

Seven patients had no signs of polyneuritis, 1 had tender calves, and 2 absent knee and ankle jerks.

Nine patients had throbbing carotid pulses, hot hands and feet, water-hammer brachial pulses, and audible pistol-shot sounds over the femoral arteries. Only 1 patient had cool hands and full, rather than collapsing, pulses.

The blood pressure ranged from 110 to 170 systolic and 65 to 90 diastolic; the pulse pressure ranged from 45 to 80 and in 8 cases was 60 to 80 mm. Hg. The pulse rate ranged from 80 to 120. No heaves or thrusts were felt over the precordium; in 5 a mild tumbling sensation was appreciable. Slight cardiac enlargement to the left could be elicited by percussion in only 2 cases. Eight had a grade II late systolic murmur at the mitral area. The pulmonary second sound was usually loud and split. A protodiastolic third sound was heard in 4 cases.

Mild exercise usually accentuated the circulatory phenomena and uncovered a third heart sound. Carotid sinus pressure produced no striking effects on the pulse rate. In 4 with more florid signs, pitressin 1 ml. intramuscularly produced dramatic effects in a few minutes; it slowed the pulse, elevated the diastolic pressure, reduced the throbbing, and caused disappearance of the third heart sound. The effects usually wore off in 15 minutes and were sometimes followed by diuresis. Saccharin arm-to-arm circulation times in 6 cases ranged between 10 and 20 seconds. The electrocardiogram usually showed tachycardia but no arrhythmias. Inversion of T waves, S-T deviations, and serial changes in the electrocardiogram were frequently seen but were probably not specific for beriberi. The Q-T and P-R intervals were within normal limits.

None of the 10 patients suffered from anemia, nephritis, fever, thyrotoxicosis, or other conditions associated with a hyperkinetic circulation. No evidence of other vitamin deficiencies was noted. No acute pernicious cases (shosin) were seen.

The heart was moderately enlarged and of a fairly characteristic shape (fig. 2). The right border showed 2 curves, an upper one due to a dynamically expanded aorta (it usually disappeared with recovery), and a lower one due to right atrial or ventricular enlargement. The left border showed a prominent main pulmonary artery and a rounded and elongated left lower segment. The secondary branches of the pulmonary artery were never grossly distended. Three cases showed mild pul-

Clinical Features and Responses of Patients with Heart Failure

Controls. These consisted of 7 cases of chronic heart failure (2, rheumatic mitral stenosis; 2, syphilitic aortic incompetence; and 3, hypertension).

On the thiamine-low diet or after thiamine was administered, 2 types of response were observed. Four patients lost no weight but the remaining 3 lost 4 to 10 pounds. In all patients signs of heart failure, namely edema, congestion, and hepatomegaly, persisted and were improved only when digitalis and mersalyl were administered.

Beriberi Heart Disease, Response to Thiamine.

Nine patients admitted to heavy drinking and 5 had eaten little food in the preceding 6 to 18 months. All were male, aged 24 to 48 years. No cases were seen in women although many African females drink heavily. A similar predominance among males has been noted in India.2

Nine patients had been ill for 1 to 3 weeks and one for 3 months. The usual complaints were swelling of the feet and breathlessness. Two gave a history suggestive of paroxysmal nocturnal dyspnea. All

Fig. 1. Top. Acute heart failure. Bottom. The same patient 3 weeks later. Four years later the appearances were unchanged.
monary congestion; 2 had small pleural effusions. Radioscopy in 8 cases revealed active throbbing (so called “aortic” movement) of the ventricles, aorta, and main pulmonary artery. In the oblique views the ventricular enlargement appeared to involve both ventricles equally in 7 cases but mainly the right in the remaining 3. In 2 cases the contraction of the whole heart appeared much reduced probably because of the presence of a small pericardial effusion.

**FIG. 2.** Top. Beriberi heart disease. Middle. The oblique view shows enlargement of both ventricles. Bottom. The same patient 3 weeks later. One year later there was no change.

**Response to Thiamine**

Six of the 10 patients lost 0–3 pounds in 5 days on the low-thiamine diet and their physical signs remained unchanged. During this time the daily urinary output was usually low, but in some cases rose as high as 80 ounces in 24 hours, possibly because the patients were allowed to drink unrestricted amounts of water.

The diuresis (60–80 ozs.) occurred within 12 hours in 2 cases but was delayed for 24 hours in 4 others. In these 6 cases the physical signs and all the edema disappeared in 5 to 9 days; the pulse rate slowed to 50–60 per minute, the blood pressure remained at the same level or rose but the pulse lost its water-hammer quality. The heart size returned to normal within 3 weeks. Two cases developed a mild fever for a few days. Tenderness of the calves persisted in 2 cases for 6 weeks and reflexes when absent did not return.

Three patients showed an incomplete response on the low-thiamine diet. They lost most of their edema and systemic venous congestion over a period of 5 to 9 days but the tachycardia and the water-hammer pulse persisted. One patient started vomiting persistently, the second developed a fever and looked ill, and the third complained of extreme calf tenderness. The administration of thiamine at this stage dispersed these signs, except calf tenderness, in 2 or 3 days. It was possible that these patients may have lost their signs without any treatment and to test this point a fourth patient who had partially responded was given digitalis and mersalyl and kept on the low-thiamine diet. He lost all his edema but the water-hammer pulse and tachycardia persisted. On the fifth day he collapsed and became cold and sweaty. He was given intravenous thiamine and recovered in half an hour and lost his abnormal circulatory signs after a further 3 days of thiamine therapy.

Five cases have been followed for 6 to 15 months. One has relapsed but none has developed an enlarged heart.

**Syndrome A**

*Cases of Heart Failure Unresponsive to Thiamine* (fig. 3). All 3 cases were males aged 32 to 45 years and only 1 was an alcoholic. They had
been ill with edema and breathlessness for 1 to 21 days and 1 gave a history suggestive of paroxysmal nocturnal dyspnea.

They had moderately severe heart failure with normal blood pressures. There was no cyanosis, valvular defect, or indeed any obvious cause for the heart failure. Two had triple rhythm and 4 had crepitations over the lung bases. X-ray showed enlargement of the heart without, however, a characteristic shape. On radioscopy the heart contracted actively in 2 cases but appeared virtually immobile in the third. On the therapeutic regime 2 lost most of their edema after 10 days of thiamine; unlike cases of beriberi however, the elevated jugular venous pressure and hepatomegaly persisted and disappeared only after a further 2 weeks of bed rest. The third case showed no improvement at all until digitalis and mersalyl were administered. In all cases the heart size became normal in 4 weeks. All 3 have been followed for 9 to 15 months and none has shown recurrence of signs or increase in heart size.

Syndrome B

Heart Failure Responding Completely to the Low Thiamine Diet (fig. 4). All 3 patients were males aged 34 to 40 years and were heavy drinkers; they had been ill for 1 or 2 weeks with swelling of the legs and breathlessness. Clinically, these patients were indistinguishable

![Fig. 3. Top and middle. Acute heart failure. Poor pulsation radioscopically. Bottom. Four weeks later. No response to low-thiamine diet or to thiamine. Response after 10 days of digitalis and mercurials. Twelve months later the x-ray appearances were unchanged (syndrome A).]

![Fig. 4. Top. Acute heart failure. Bottom. Seventeen days later. Response on bed rest and low-thiamine diet (syndrome B). Fifteen months later there was no change.]
from cases of beriberi heart failure. They were all in a state of mild congestive cardiac failure. They all had wide pulse pressures, soft mitral systolic murmurs, loud and split pulmonary second sounds, and 1 had a protodiastolic third sound as well. In 2, saccharin circulation times measured 11 and 15 seconds respectively. The electrocardiograms showed nonspecific changes. Pitressin tests in 2 were equivocal. X-ray revealed moderate enlargement of the heart but not a characteristic shape. (One had a long ventricular outflow tract, 1 had a boot shape, and the third resembled beriberi heart.) Only 1 had pulmonary congestion. Radioscopically the heart contracted actively in all 3 cases.

These patients differed from cases of beriberi in their immediate and complete response on the low-thiamine diet and bed rest. Within 24

| Table 1.—Laboratory Findings in Sixteen Cases of Acute Heart Failure and Eight Cases of Hunger Edema |
|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|
| Alkaline phosphatase (K.A. units) | Liver function tests | Serum cholesterol (mg. %) | Serum proteins Hemo- globin (Gm. %) | Erythrocyte Sedimentation Rate (Win-trobe) (mm./1 hr.) |
|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|
| TT | TF | CR | TA | Total | Albumin | Globulin | Mean | TT | TF | CR | TA | Total | Albumin | Globulin | Mean | TT | TF | CR | TA | Total | Albumin | Globulin | Mean |
|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|
| Beriberi 3.7 | 3.5 | 3+ | 4+ | 3+ | 115 | 6.7 | 3.0 | 3.7 | 16.6 | 19 |
| 14.0 | 5.5 | 3+ | 4+ | 3+ | 110 | 7.0 | 3.4 | 3.6 | 17.6 | 24 |
| 8.5 | 4.5 | 4+ | 4+ | 3+ | 145 | 8.5 | 4.7 | 3.8 | 16.4 | 9 |
| 4.4 | 3.5 | 0 | 1+ | 0 | — | — | — | — | — |
| 6.0 | — | — | — | — | 140 | 6.7 | 3.6 | 3.1 | 15.6 | 3 |
| — | 4.5 | 1+ | 4+ | 3+ | 200 | 6.6 | 3.4 | 3.2 | 16.3 | 18 |
| — | 2.5 | 1+ | 3+ | 3+ | 110 | 6.6 | 3.4 | 3.2 | 15.0 | — |
| 6.6 | 2.5 | 0 | 0 | 3+ | 145 | 6.5 | 2.2 | 4.3 | 13.8 | — |
| — | 1.5 | 0 | 3+ | 0 | — | 7.1 | 3.6 | 3.5 | 15.8 | 2 |
| — | — | — | — | — | — | 7.0 | 2.5 | 4.5 | 15.6 | 16 |
| Mean | 138 | 7.0 | 3.3 | 3.7 |
| Syndrome A 7.5 | — | — | — | — | 125 | 6.6 | 3.0 | 3.6 | 16.1 | 28 |
| — | — | — | — | — | 140 | 7.5 | 4.1 | 3.4 | 16.0 | — |
| — | 4.0 | 3+ | 4+ | 2+ | 170 | 7.0 | 3.3 | 3.7 | 15.6 | 31 |
| Mean | 145 | 7.0 | 3.5 | 3.6 |
| Syndrome B — | 4.5 | 1+ | 4+ | 3+ | 200 | 6.6 | 3.4 | 3.2 | 16.3 | 18 |
| — | — | — | — | — | 7.3 | 3.5 | 3.8 | 17.4 | 3 |
| 5.1 | 3.5 | 3+ | 3+ | 0 | 200 | 6.7 | 3.0 | 3.7 | 15.0 | 30 |
| Mean | 200 | 6.9 | 3.3 | 3.6 |
| Hunger edema 8.4 | 3.5 | — | 4+ | 1+ | 195 | — | — | — | — |
| 7.2 | 1.5 | 0 | 1+ | 1+ | 165 | 7.7 | 3.6 | 4.1 | 17.9 | 17 |
| 6.2 | 5.0 | 4+ | 4+ | 1+ | 120 | 7.4 | 3.7 | 3.7 | 18.0 | 2 |
| — | — | — | — | — | 7.3 | 3.5 | 3.8 | 17.4 | 3 |
| 5.8 | 3.5 | 4+ | 3+ | 0 | — | 7.4 | 3.3 | 4.1 | 14.4 | 3 |
| 9.8 | 4.0 | 0 | 4+ | 3+ | — | 6.4 | 3.0 | 3.4 | 16.7 | 7 |
| 10.0 | 3.5 | 0 | 3+ | 3+ | — | 6.2 | 2.5 | 3.7 | 13.0 | — |
| — | — | — | — | — | — | 6.7 | 2.4 | 4.3 | — |
| Mean | 160 | 7.0 | 3.1 | 3.9 |

TT = Thymol turbidity; TF = Thymol flocculation; CR = Colloidal red test; TA = Uckos' modification of the Takata-Ara test.
hours they started losing weight and passed large quantities of urine (94–120 ozs.). Within 6 to 8 days they had lost all their edema (12–25 lbs.) and physical signs. One patient who started losing weight was kept on a low-thiamine diet and was treated with digitalis and mersalyl as well; he recovered completely in 5 days. All 3 patients have been followed for periods up to 15 months and have shown no recurrence of symptoms or signs and no radiologic enlargement of the heart.

**Edema without Cardiac Enlargement, 10 Cases.** Two patients, both males, showed no improvement on the low-thiamine diet but when thiamine was added they recovered rapidly and completely, indicating that the edema was due to vitamin B₁ deficiency.

The remaining 8 cases of edema responded completely on the thiamine-low diet and bed rest. There was one female in this group. Their ages ranged from 30 to 50 years. Six were admitted alcoholics and 1 had been in jail for 18 months. All had developed swelling and breathlessness in the previous 3 to 21 days.

Four of these patients, 1 female and 3 males, in addition to their edema showed striking circulatory phenomena, namely moderately raised jugular venous pressure, tender hepatomegaly (1–3 fingerbreadths), hot extremities, waterhammer pulses, and tachycardia (80–120). On auscultation 3 had soft systolic mitral murmurs and 1 had triple rhythm. Circulation times in 3 measured 12, 13, and 17 seconds respectively. Pitressin tests were dramatically positive in 2. Clinically, they were indistinguishable from cases of beriberi heart failure or from syndrome B but differed from both these types in showing no radiologic enlargement of the heart. They resembled syndrome B, in their immediate and dramatic response while on a low-thiamine diet. All recovered in 3 to 7 days. No subsequent enlargement of the heart was observed in periods of 4 to 12 months.

Of the remaining 4 cases of edema none had clinically abnormal circulation. The pulse rates ranged from 65 to 90. On a low-thiamine diet all had a brisk and immediate diuresis and lost their edema (10–20 lbs.) in 5 to 7 days. In 2 the edema recurred in 2 and 6 weeks respectively.

No renal lesion was detected and there was no anemia. Liver function tests and blood proteins were abnormal but did not differ significantly from values found in the other groups (table 1).

**Discussion**

The first point to decide is whether the patients who responded on a low-thiamine diet were mild cases of beriberi responding to small quantities of vitamin B₁ in the diet. It must be pointed out that it was not found practical to maintain these African patients for any length of time on a lower vitamin B₁ intake, for such a diet was not bulky enough and made the subjects hungry and uncooperative. The diet used in this study supplied 0.25 to 0.3 mg. of vitamin B₁ in 24 hours and it has been estimated that an adult weighing 70 Kg., requires 0.65 to 1.25 mg. of vitamin B₁ per day to prevent the development of deficiency. On the other hand, patients at rest in bed probably require less vitamin B₁ than more active people and, moreover, it is possible that synthesis of vitamin B₁ may occur in subjects subsisting on a low-thiamine diet. Although these Africans were maintained on a diet that theoretically could not cure beriberi, nevertheless this possibility cannot be entirely dismissed.

If these syndromes are not evidence of thiamine deficiency, what are they? Edema without cardiac enlargement or venous congestion is common among Africans. It is aggravated by exercise, reduced or dispersed by bed rest, and often recurs within a short time after the patient leaves the hospital. It is not due to heart failure or renal disease. It is commonly associated with abnormal liver function tests and a comparatively low albumin concentration in the serum—features however that are also found among outwardly healthy Africans and in many medical patients without edema. From its clinical features it corresponds with the description of hunger edema, which occurred in undernourished Europeans after the last 2 great wars. The studies of McCance and others have established that in hunger edema the extracellular space is expanded; McCance postulated that the excess fluid moves freely about the body and may invade the intravascular compartment to produce changes in
blood volume. There is already some evidence that scorbutic Africans suffering from edema show variations in blood volume as judged by elevation of jugular venous pressure and wide fluctuations in hemoglobin levels from day to day. The clinical features of the patients with noncardiac edema and with certain of the heart failures (syndrome B) in this study are so similar as to suggest that they may represent gradations of the same underlying fault, namely hypervolemia. Maneuvers that increase the blood volume both in man and in animals are known to produce a hyperkinetic state and ultimately heart failure.10

The 3 cases of heart failure in syndrome A did not respond to thiamine, no obvious cause was apparent for the heart failure and radiography showed poor cardiac pulsation in 1 patient. In these features they resembled the early stage of nutritional heart disease described by Gillanders.1 All have been at home for periods of 9 to 15 months and presumably have been living on their accustomed home diet. None have relapsed and in this respect they differ from Gillanders’ group whose heart failure recurred within a short time after return to the deficient home diet. Whether these examples represent a homogeneous group of heart disease is not yet known.

*Idiopathic cardiac hypertrophy* is commonly found at postmortem examination among Africans in South Africa.11 Both ventricles are hypertrophied, there is a tendency to mural thrombosis, and histology reveals no specific lesion. No obvious cause for the hypertrophy can be found. A similar type of cardiac hypertrophy has been described among West Africans.12 In East Africans cardiac hypertrophy is commonly associated with extensive endocardial fibrosis,13 probably due to massive organization of mural thrombi. In Johannesburg, Becker’s group14 have described a heterogeneous group of heart failures, some of which are probably examples of idiopathic cardiac hypertrophy studied by special histochemical methods.

In the Bantu, Gillanders1 and Higginson11 studied the etiology of the condition and excluded such factors as glycogen-storage disease and deficiency of vitamins B1 and E. At an early stage of the disease Gillanders was able to reverse the cardiomegaly by feeding the patient an adequate diet, and he could reproduce the heart failure by returning the patient to his deficient home diet. Many of his patients suffered from liver disease. For these reasons he concluded that the condition was due to malnutrition. Liver disease, however, is commonly found in the Bantu and in many who do not have idiopathic cardiac hypertrophy; idiopathic cardiac hypertrophy is found in young children who have not suffered from malnutrition15 and certainly many adults with idiopathic cardiac hypertrophy do not show the stigmata of malnutrition in the accepted sense.

Idiopathic cardiac hypertrophy is found throughout Africa. As far as can be ascertained, the condition has not been reported in other undernourished communities in the Far East and India. Although the mechanism is not understood, it seems reasonable to assume that the condition is in some way due to malnutrition, possibly to a particular dietetic pattern.

One of the basic problems about the disease is why both right and left ventricles should hypertrophy in the absence of pulmonary or systemic hypertension or valvular defects. Can it be due to chronic vitamin B1 deficiency or to repeated attacks of cardiac dilatation associated with hypervolemia?

It has been suggested that vitamin B1 deficiency may be a cause of cardiac hypertrophy. This hypothesis was first advanced by Weiss16-18 who found examples of unresponsive heart failure associated with cardiac hypertrophy among a group of undernourished subjects who were probably suffering from vitamin B1 deficiency as well. The idea has been supported by others.19-21 The evidence is by no means conclusive and has been challenged by Wintrobe,22 who doubts whether chronic heart failure can ever be attributed to vitamin B1 deficiency. It is not known whether cardiac hypertrophy is common in the Far East or in India, where beriberi is endemic. On the contrary, there is evidence that prisoners of war who suffered from cardiac beriberi in Japan have not developed chronic heart disease.23 The evidence from these Africans also lends support to the view that the acute form of beriberi does not
lead to chronic heart disease. Nevertheless, the possibility exists that subacute (subclinical) deficiency may be a cause of cardiac hypertrophy although this has not been demonstrated in animals. As far as Africans are concerned, it is not known whether beriberi is an endemic disease or whether it is confined to urban natives who live in poor economic circumstances and drink alcohol to excess. Judging by food surveys of urban and rural Africans, it would seem that they obtain adequate quantities of vitamin B1 in their diet. Although hunger edema is commonly seen in this hospital, the same doubt exists whether it is a disease indigent to Africans or only occurs in urban areas and thus whether it could be reasonably considered a factor in the production of idiopathic cardiac hypertrophy.

SUMMARY

Sixteen cases of acute reversible heart failure in Africans have been studied. They have been arbitrarily classified into 3 groups on the basis of their response to a low-thiamine diet and to injections of thiamine. Only 10 cases were examples of vitamin B1 deficiency (beriberi heart disease).

Eight cases of nutritional edema without cardiac enlargement were studied in the same way. Four of these patients and 1 group of heart failures had similar clinical features and responded on a low-thiamine diet. It is suggested that these similarities were due to the same basic abnormality, hypervolemia.

The role of vitamin B1 deficiency and nutritional edema in idiopathic cardiac hypertrophy of the Bantu is briefly discussed.

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SUMMARIO IN INTERLINGUA

Esseva studiate 16 casos de acute reversibile disfallimento cardiac in africanos. Super del base del responsas a dietas de basse contento de thiamina e a injectiones de thiamina, le casos esseva classificate arbitrarimente in 3 gruppos. Solmente 10 del casos esseva exemplos de carentia de vitamina B1 (morbo cardiac beri-beri).

Octo casos de edema nutritional sin allargamento cardiac esseva studiata in le mesme maniera. Quatro de iste patientes e un del gruppos de casos de disfallimento cardiac habeva simile aspectos clinic e respondeva a dietas a basse contento de thiamina. Es suggerite que iste similitudes esseva le resultato de un identic anormalitate fundamental: hypervolemia.

Es presentate un breve discussion del rolo de carentia de vitamina B1 e de edema nutritional in idiopathic hypertrophia cardiac in le bantu.

REFERENCES

11 Higginson, J. S., Gillanders, A. D., and


Withering's prominence as a botanist is sometimes lost sight of because of his discovery of digitalis. His years at Stafford had culminated in the publication of a two-volume work entitled Botanical Arrangement of Vegetables (1776), which earned him the title of the English Linnaeus. On the Continent he was probably better known for his botany than he was for the introduction of digitalis.

Withering also achieved an eminent position as a mineralogist principally for his discovery of barium carbonate which is still known as "Witherite" in honor of the man who first proved its chemical constitution, it having been so named by the great German geologist, Werner.—John F. Fulton.—The Place of William Withering in Scientific Medicine. J. Hist. of Med. & Allied Sc., 8: 14, 1953.
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