HEART DISEASE IN THE TROPICS

BY

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TO THE MEMORY
OF
CHARLES PEIRIS
A GREAT GENTLEMAN,
WISE COUNSELLOR,
ADMIRABLE FRIEND.

PREFACE.

The object of this small volume is best declared negatively. It is not intended to give a description of the various diseases of the heart which are met with in the tropics. These are dealt with very comprehensively in the works of many well-known men which are either in use or are available for reference by both the medical student and the practitioner in tropical countries. But although these authors record fully the present state of our knowledge of these diseases as they occur in the West, very scant reference or none at all is made to the influence of the tropics on their incidence, course, prognosis or to modifications in treatment, which they require. Reference is also not often made adequately to tropical diseases which give rise to serious cardiac disability. Such a condition is ankylostoma infection which is comparable as a common cause of heart disease with rheumatic fever of temperate climates and which for this reason has been allotted a good deal of space in the following pages. Another condition not peculiar to the tropics which has been given ample consideration is high blood pressure as it

is responsible for a far larger number of cases of heart disease than is rheumatic fever. The aim generally is to indicate features in cardio-vascular diseases induced by tropical conditions and by diseases peculiar to the tropics.

There appears to be more than a little justification for this volume. In the first place there is no single book in which an attempt has been made to deal with this subject *per se*. In the second place it is bound to inspire a more detailed study of an aspect of disease hitherto too much neglected and this little work may, I hope, be an incentive to a larger and a more complete study. In the third place facts relative to important diseases such for instance as rheumatic fever, ankylostoma infection and high blood pressure are shown up on a background in which their scientific value becomes obvious. If for any one of these reasons this book proves to be of use to the student and the practitioner of medicine in the East its publication will have been thoroughly justified.

I have to acknowledge here my gratitude and thanks to the visiting physicians of the General Hospital, Colombo, Dr. E. M. Wijerama who has so kindly helped me in many ways, to Dr. P. B. Fernando for reading the proofs and for

many useful suggestions, to Mr. Mendis of the General Hospital for his photographs, to Messrs. M. L. B. Caspersz and J. Fernandopulle, my two Assistants in Ceylon, and to the Publishers for so much kindness and help.

H. O. GUNEWARDENE.

COLOMBO,
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CHAPTER I

THE INCIDENCE OF HEART DISEASE IN THE TROPICS.

The picture of heart disease in England side by side with that of the same disease in the tropics would show very many points of great interest. While in England rheumatic fever plays so prominent a part in the causation of cardiac lesions, we see the same etiological factor operating much less frequently in the countries blessed with the perpetual tropical sun. It is not unusual in private practice in England to have to visit in the morning round two or three patients laid up with acute rheumatic fever or chorea and the attendant cardiac affections. Rheumatic valvular lesions with the disabilities which come as their sequelæ loom large in our minds when we reflect on the array of cardiac affections we have met with in England both outside and inside the hospitals. But in the tropics they get no such prominence. Indeed though they do occur not infrequently,[1] *contrary to the view held by some that rheumatic fever is unknown*

[1] Hughes, B M J., October 22nd, 1932, p. 773.

in the tropics[1] they form but a small percentage when actual figures are studied. Of 400 cases observed in private practice, a good many of which were in consulting practice, the figures are as follows :—

High Blood Pressure	15%
Myocardial Degeneration	12%
High Blood Pressure with Renal Disease	10%
Angina Pectoris	10%
Dilatation	8%
Extra Systoles	8%
Mitral Stenosis and Regurgitation ..	8%
Congenital Heart Disease ..	6%
Rheumatic Fever	5%
Syphilis	5%
Coronary Thrombosis	4%
Auricular Fibrillation	4%
Other lesions (Chorea 2 cases) ..	5%

Rheumatic fever according to these figures, which are approximately correct, has therefore a low incidence in the tropics. But experience teaches us that lesions like mitral stenosis do occur in larger numbers than one would be justified in anticipating on considerations of the incidence of rheumatic fever alone. It would appear that the sequelæ of rheumatic fever is less virulent in the

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[1] Tertius Clarke, B.M.J., October 1st, 1932, p. 650.

B.M.J., November 12th, 1932, p. 900.

tropics, is often latent, if I may say so, and not infrequently non-progressive. In favour of the non-virulent nature of the infection I may mention that rheumatic aortic regurgitation is relatively uncommon. When we say the rheumatic processes are latent we mean that they appear to go on insidiously without the signals of their presence, more so than in temperate climates. That the lesions are not infrequently non-progressive is evident in more ways than one. Off and on I had under my observation a Singhalese lady with mitral stenosis for the last twelve years. The diagnosis was made in London, and since then within this period she was seen by many well-known cardiologists. On the last occasion I sent her to Dr. Thomas Cotton with the observation that as the patient has developed no new signs or symptoms I thought the case was one of non-progressive mitral stenosis. Dr. Cotton wrote back to say that he agreed with me, that 'it is one of those rare cases of non-progressive mitral stenosis'. Such cases are not uncommon. Owing to their non-progressive nature these hearts are able to stand greater strain than those apparently similarly affected in Europe. For instance if the patients are married women, they appear to bear the burden

of a larger number of pregnancies, ceteris paribus, than their European sisters. I have often made the error of making no allowance for this factor, to be told later on, by the obstetrician, on more than one occasion, that the lady you said should not have another child had one the other day without any trouble. In cases of mitral stenosis therefore the condition of the heart must be carefully considered apart from the diagnosis itself and prognosis should be based on a close scrutiny of the signs and symptoms of cardiac disability which were present at the last confinement.

Another striking observation in support of the slow progress of the changes following a rheumatic infection is the relative infrequency of auricular fibrillation. The majority of cases of the latter condition have been non-rheumatic, and of both kinds there have been only seven in this series in consulting practice. Indeed they have been so few that they have been included in the 'other lesions' group.

Before commenting on the other groups I will take the figures for 1932, kindly given me by Dr. E. M. Wijeyrama, the Medical Registrar of the General Hospital in Colombo. These figures are for the non-paying section of the institution :—

Cardiac failure due to ankylostoma infection		?	
Myocarditis		183	40%
Mitral stenosis and Regurgitation		93	20%
High Blood Pressure ..		66	13%
Cardiovascular Syphilis ..		45	10%
Irregular heart action including auricular fibrillation ..		20	5%
Aortic Lesions	Regurgitation..	13	3%
	Stenosis ..	1	
Angina Pectoris		11	2·5%
Infective Endocarditis ..		9	2·5%
Pericarditis		5	1%
Aortic aneurysm ..		5	1%
Congenital Heart Disease ..		4	1%
Rheumatic fever (63 cases) ..		?	

From the above group, cases of cardiac disability due to ankylostoma infection are unfortunately excluded, for the reason that these have not received any special attention from the point of view of the cardiac manifestations. But the fact remains that this infection is the commonest cause of heart disease in the poor, so much so that it may be called the 'poor man's heart disease'. The largest group in the Hospital series is myocarditis, a term which has apparently been taken to connote all kinds of conditions giving rise to cardiac symptoms and signs the cause of which could not be associated with a definite lesion, like for instance mitral disease. In this

group, there can be little doubt, are a fair number of cases of high blood pressure.

In my series, cases of myocarditis have been included under myocardial degeneration, fibroid and fatty. The incidence of this condition is here too high forming 12% of my cases. But the highest in my series is high blood pressure, both renal and non-renal. This disorder, or disease, whatever one likes to call it, is among those who are not too poor, the commonest cause of cardiac disability. Even in the Hospital series it is not too low, the percentage being 13, but it will be observed that it is much higher outside hospital practice, namely 25%. As elsewhere, so in Ceylon we have been slow in recognizing its frequency and its numerous sequelæ. Mitral stenosis and regurgitation form 20% in the Hospital series while amongst the better classes it is only 8%. The higher incidence of both this condition and its common antecedent, rheumatic fever among the poorer classes would explain its greater frequency in hospital practice. As in Russell-Wells[3] series at The National Hospital for Diseases of the Heart, London, a history of rheumatic fever is

[3] Sydney, Russell-Wells and others. B M J, September 7th, 1918, p. 249.

frequently absent. The figures for the tropics are not so high however, with a negative history in cases of definite mitral stenosis. Angina pectoris forms about 10% in both series and among some of these there are probably included cases of coronary thrombosis. Those referred to under the latter category are ones in which the clinical diagnosis (not verified by the electrocardiograph) appeared very clear. Syphilis in my series forms but a small percentage. This tallies with the fact that aortic regurgitation is relatively very rare. As stated elsewhere even rheumatic aortic regurgitation is rare. But in hospital wards the incidence is twice as high as in my series. So is that of aortic regurgitation. I have classified under Dilatation a number of my cases amounting to 10% of the series, in spite of extreme reluctance to attach any importance to mitral systolic murmurs. It is so contrary to the teaching in European schools and almost a sacrilege against the revered names of well-known cardiologists but my conviction is that conditions in the tropics, for instance the recurrent febrile affections, do give rise to affections of the myocardium indicated by apical systolic murmurs and symptoms referable to a slightly disabled myocardium. It is

probably a large number of these cases too that raise the percentage of the 'Myocarditis' group in hospital practice. The relative infrequency of the other lesions both in hospital and private practice are shown by their low incidence. But under 'Other lesions' I have included such conditions as infective endocarditis, heart block and paroxysmal tachycardia.

From the point of view of cardiac disease and disorders therefore we have to turn our minds in the tropics more seriously to conditions the incidence of which is high in these parts of the world. Ankylostoma infection, high blood pressure, recurrent tropical fevers, malaria for instance, have not received due prominence. From the suffering patient's point of view the discomfort is his, and whether we label a condition a disease or not, he pays his fee to secure relief, which can only be given if we are in possession of a thorough knowledge of these conditions.

CHAPTER II.

THE CARDIO-VASCULAR PHENOMENA IN TROPICAL CONDITIONS.

From the scant reference in text-books on tropical medicine to the cardio-vascular system it would appear that there are but few conditions which bring the heart to the forefront in their symptomatology or complications. But not infrequently the physician gets anxious about the pulse, the heart is found dilated, a murmur arouses interest and extra systoles cause alarm. More rarely, as in beriberi, there is extreme dilatation of the heart and fatal failure, and persistent or paroxysmal tachycardia. One of my patients had auricular fibrillation which judging by the history followed on an attack of smallpox. On the other hand ankylostoma infection, recurrent attacks of tropical fevers, like malaria, the numerous conditions which give rise to secondary anæmia, these directly or indirectly give rise to symptoms such as palpitation and shortness of breath which bring the patients to the physician whose duty it becomes to ascertain the primary cause and its

sequelæ. Some patients come in so advanced a stage of anæmia that the heart requires the closest attention, if sudden death is to be averted. Treatment for and management of the heart then becomes an essential part of the treatment for the primary condition. Ankylostoma infection affords many such examples.

There is another group of cases of great interest to the cardiologist. In this category are patients who present signs and symptoms of cardio-vascular disease, for example, senile changes or evidence of old rheumatic carditis. In the tropics, infections peculiar to these parts of the world exaggerate these conditions. By a secondary anæmia being grafted on him or by being subjected to the strain of recurrent bouts of malaria, the patient is carried to the threshold of symptoms referable to the heart. In assessing the degree of cardio-vascular damage, it becomes necessary only too often to eliminate the purely 'tropical element' in the patient before a final pronouncement on the primary condition is made.

The study of the heart and pulse in typhoid fever has aroused not a little interest. Since Marris in 1915 described his test for the diagnosis of enteric

fever the subject has been studied more closely. Rogers[1] give the following table in 224 cases of typhoid fever with a temperature rising to 103 or over:—

Pulse rates in 224 cases of Typhoid fever with a temperature rising to 103 or over.

	Pulse not over 100 throughout		Pulse not over 100 for 2 days or more		Pulse over 100 throughout during high fever.	
	No	Per centage	No	Per centage	No	Per centage
Men	57	41·6	52	38·0	28	20·0
Women	1	2·9	4	11·8	29	85·3
Children	1	1·9	0	..	52	98·1

According to him a slow pulse in children and in adult females is rare during typhoid fever. He stresses however the importance of recognizing the fact that in 80% of men 'a pulse not exceeding 100 during pyrexia reaching over 103 F for two days or more, most frequently in the early stages of the disease, is of great diagnostic importance because such a relatively slow pulse is rare in other fevers liable to be confused with typhoid with the single exception of 7-day fever'.

[1] Rogers, Sir Leonard—Fevers in the Tropics, 2nd ed., p. 114.

Heart: Of the heart he says, 'the tendency of the first heart sound to become short and faint should be looked upon as an indication for cardiac stimulants—a fact pointed out by G. H. Young in 1887'. Extra systoles, vagal irregularities and tachycardia are not infrequently seen in the later stages of the disease. Their importance lies in that these signs too often cause alarm and tempts the practitioner to resort unnecessarily to cardiac stimulants and other drugs. These signs by themselves should not influence the prognosis, unless the general picture is one of circulatory failure. The blood pressure is generally low. But my observations led me to think that no reliable conclusion as to the degree of toxæmia or as to prognosis could be made on the pressure figures alone.

In support we may mention Potain,[2] who stated that 'arterial pressure does not bear any relation to the severity of the malady'. J. D. Rolleston[3] on the other hand finds that the depression of pressure is in direct relation to the severity of the attack.

A closer study of the low arterial

[2] Potain, La pressure artinelle chez l'hensen, 1902

[3] Rolleston, J. D., Medical Press and Circular, 1916, p. 234.

pressure in typhoid fever may prove to be interesting, although our present knowledge of its variations does not throw any light on the better management of a patient suffering with the disease. A few facts are however worth mentioning. It is stated by Rolleston [3] that 93·8% of the cases show subnormal pressures; the systolic pressure in the majority being under 100; in 50% a low pressure is said to persist 5 to 23 weeks after the disease. An observation which might be useful in gauging the return of normal vascular tone is that in 93·1% of convalescent cases the reading in the recumbent positions was higher than in the erect, and it remains higher in the recumbent until normal health was established. This 'hypotension of effort' is believed to persist until the patient is discharged. Cooks and Briggs state that in the early stage of typhoid perforation there is a striking rise in pressure, whilst hæmorrhage and myocarditis produce a fall. Halls Dally [4] quoting from Janeway and Barach give the following figures for the course of the disease:—

[3] Rolleston, J. D., Medical Press and Circular, 1916, p. 234.

[4] Halls Dally, Low Blood Pressure, 1928, p. 142, Heinemann.

	Janeway.	*Barach.*
Systolic Pressure in 1st Week	115	93
,, ,, ,, 2nd ,,	106	92
,, ,, ,, 3rd ,,	102	83
,, ,, ,, 4th ,,	98	83
,, ,, ,, 5th ,,	96	85
,, ,, ,, 6th ,,	90	85

Below is given a brief description of the heart and pulse in some of the commoner tropical affections. *Malaria*—The heart is very seldom affected in malaria. Cases of marked anæmia develop hæmic murmurs.

As a result of defective nutrition from prolonged anæmia and recurring fever, the muscular tissue of the heart in chronic malarials may degenerate, the ventricles dilate and in time the lower extremities become œdematous. For this reason the subjects of valvular diseases of the heart whether compensated or otherwise must be discouraged from residing in malarial districts.

In rapidly fatal cases of subtertian malaria Dudgeon and Clarke have found a diffuse fatty degeneration of the heart similar to that occurring in acute diphtheria poisoning whilst Gaskell has observed the actual presence of subtertian parasites within the cardiac capillaries.

According to Stitt [5] those which affect

[5] Stitt, Tropical Medicine, 5th ed., 1927, p. 681.

cardiac muscle itself or those which cause a disturbance in the note or rhythm which may be of importance in diagnosis, treatment or prognosis, are :—

1. **Ankylostomiasis** (dealt in Chapter on the subject).

2. **Beriberi**—early palpitation on slightest exertion—loss of normal cardiac rhythm with advancing vagal degeneration ; pulsating jugulars ; blowing systolic murmurs, embryocardial rhythm with tumultuous action and weak pulse—low blood pressure—in acute pernicious beriberi, pulmonary œdema. According to Maxwell[4] patients with heart symptoms are always on the brink of disaster till these heart symptoms clear up, and at any time, in the course of a few hours may develop an attack of acute and fatal dilatation. Others who recover do so with permanently dilated hearts.

3. **Blackwater fever**—a rapid, weak, low tension pulse.

4. **Cholera**—rapid pulse in stage of evacuation and with onset of algid stage almost cessation of circulation—low blood pressure 65-70.

5. **Heat-Stroke**—Cardiac tetany—severe anginal pain with sudden death.

[4] Maxwell. The Diseases of China, 2nd ed., 1929, p. 101.

6. **Leprosy**—a rapid pulse specially in the morning is thought to be a feature of active leprosy.

7. **Malaria**—a small rapid high tension pulse in the cold stage ; full bounding in the hot stage—Cardiac type of pernicious malarial fever has been described by the Frends.

8. **Plague**—rapid, soft dicrotic pulse soon becoming thready—sudden death on getting up from bed may occur.

P.M. Petechial hæmorrhages in pericardium—and fatty degeneration of heart muscle.[7]

9. **Trypanosomiasis**—rapid pulse with or without fever ; manifestations of myocardial disease due to invasion of heart muscle by the parasite and low blood pressure from affections of the adrenals (along with other signs of Addison's disease).

10. **Typhus**—tends markedly to affect the heart—faint heart sounds—rapid low tension pulse.

11. **Yellow Fever**—At first a high blood pressure. The pulse rate shows Faget's sign—a falling pulse with a constant temperature or a constant pulse with a rising temperature. It is marked-

[7] Jewell and Kauntze, Handbook of Tropical Fevers, 1932, p 293

ly slow after the 3rd day. Low blood pressure in the asthenic stage.

Reference must be made here to a condition frequently diagnosed in the tropics as 'dilatation of the heart'. The condition has also been called the 'toxic heart' of the subacute and chronic stages of infection. The diagnosis so made is not based on a radioscopic examination of the heart, particularly of alterations in the size of the different chambers; nor is it made on an accurate clinical assessment of the size of the heart as a whole. Medical men in the tropics or elsewhere are fully aware of the fact that neither dilatation nor hypertrophy is easy to determine accurately in this type of case where there is hardly any demonstrable enlargement but the term is used however in the absence of a scientific license for its adoption. Such a condition is similar to that seen often after influenza and after dengue in the tropics but the condition occurs so frequently that the term is resorted to in order to allow a clinical grouping of this kind of case. The patient usually gives a history of a frequently repeated febrile affection or is obviously the victim of some parasitic infestation. He complains of palpitation and shortness of breath on exertion, and occasionally slight pre-

cordial distress. Examinations reveal almost invariably an apical systolic murmur, sometimes basal systolic murmurs and often slight tachycardia ; the blood shows a secondary anæmia : other examinations will often reveal evidence of parasitic infection.

These cases are frequently dosed with digitalis : in my opinion this is a quite unnecessary procedure. If the patient is treated adequately for his infection, is reassured and asked to cut down his physical exertions for a time, and is treated with preparations of iron, both the cardiac and general symptoms will disappear. To send such a patient after malarial infection to the hills without adequate treatment is to invite a recrudescence of the infection.

Prognosis and Treatment. Fortunately for those who live in the tropics Providence has decreed that serious complications shall be very rare indeed in the common tropical diseases as regards the effect of these on the human heart. A tiring reference to points on which to base a prognosis is not necessary. Wilson * in his study of some of the cardio-vascular phenomena which follow these infections

* Wilson, R. M., Byam and Archibald. Practice of Medicine in the Tropics. 1922. Vol III, p. 2300.

particularly malaria, dysentery and trench fever, has attempted to base prognosis on the effects of inspiration on the pulse observed in some of his patients when he stated that 'if, with a normal temperature there is neither slowing nor loss of volume, a very favourable prognosis is justifiable; if only a slowing is present the outlook is good'. This information might be useful but in the vast majority of cases, patience, reassurance, and a thorough elimination of the toxins and their origin is all that is required.

No elaborate technique as regards prognosis and treatment need be described under this heading but the following observations should serve as a reliable guide to those in doubt as to the management of a patient.

1. The help of a colleague well-versed in tropical medicine should be sought when the primary cause is not obvious or when the measures adopted fail to give relief.

2. 'Organic disease of the heart accompanying or following tropical fevers is excessively rare.' But cardiac symptoms which are secondary phenomena form part of a vicious circle which has to be broken if the patient is to get relief.

3. 'It is astonishing how quickly, if the toxin is removed—i.e., if the disease is cured—the cardiac symptoms disappear in all but a few very weak and disabled cases.'

CHAPTER III.

THE CARDIAC COMPLICATIONS OF ANKYLOSTOMA INFECTION WITH SPECIAL REFERENCE TO A PRESYSTOLIC MURMUR OCCURRING IN THESE CASES.*

In adding more to the already abundant medical literature, ample justification seems to lie in the fact that standard text-books appear to be wanting in a full description of so common a disorder in the tropics as the cardiac complications due to infection with ankylostoma. For instance, *Manson's Tropical Medicine*, edited by *Manson Bahr*, merely states 'the circulation is irritable and bruits can be heard over the heart and large blood-vessels'. As for treatment relative to the heart the reference made is only 'Thymol is contra-indicated—and in active heart disease'. *Castellani and Chalmers*, in their *Manual of Tropical Medicine*, refer to the subject with 'patients always complain of palpitation or difficulty in breathing. The

* From the Journal of Tropical Medicine and Hygiene, Feb. 14, 1933.

lungs will be found normal, but the heart will be found displaced downwards and to the left and be feeble, with a hæmic bruit at the base'. The vessels of the neck may be found to pulsate markedly. 'The pulse is quick and may be weak, thready, dicrotic and intermittent. The liver is very often enlarged specially in children.'

Such a description, though perhaps a conglomeration of the symptoms and signs which may be the sequelæ of this infection, does not appear to give too faithful a picture of the cases as seen in the wards of a large hospital. The students working in the hospitals and practitioners serving among the poorer classes must often come across those advanced cases in different stages, which in a large percentage so resemble the clinical picture of heart failure due to rheumatic fever, that diagnosis in some cases becomes not only difficult but impossible. The occurrence of an enlarged liver due to other causes in the tropics only renders it more difficult to isolate the enlarged liver of congestive failure; the occurrence of fever so common in this infection often gives a picture only too suggestive of infective endocarditis; the occurrence of not only the so-called hæmic murmurs, but others,

even in the absence of organic disease, leads to the suspicion of organic valvular disease; and finally the occurrence of ankylostoma infection in a patient with organic valvular disease, rheumatic or otherwise, adds further puzzling circumstances.

The murmurs which occur in these cases, though classed *en bloc* as hæmic, whatever the word means, are not always only systolic murmurs at the base or the apex or at both sites. They are no doubt the commonest. But many variations are present and they are interesting not merely in the fact of their variety but in the peculiarity of their incidence. Why, for instance, should some of the most suddenly fatal cases have none at all, or at the most, faintly audible ones? Why should they be absent in some and be present in others with the same degree of dilatation or hypertrophy and the same degree of anæmia? It is the occurrence of variety which to my mind appears most responsible for the confusion of these cases with organic heart disease. Going through the notes of several reliable clinicians one finds in the bed-head tickets descriptions of the murmurs as diastolic and presystolic. I made a careful study of these cases and came to the conclusion

that, whatever the explanation, such murmurs do occur. Whether they are produced by relative stenosis or whether in our ignorance of the factors responsible for the production of these murmurs we can enunciate no acceptable explanation, they do in my opinion occur. The murmur is not so definite as in mitral stenosis nor is there a thrill; it is more like the murmur named after Austin Flint, on auscultation. My experience does not appear to be unique. The clinical notes of so well-qualified a clinician as Dr. Cyril F. Fernando contains similar notes and in one of his cases of this type a post-mortem was done and the valves were found to be normal (Case No. 3, see p. 30). Like notes are found in the tickets of my colleague, Dr. V. P. de Soysa. Looking back on my own experience, I can recall a case under the care of the late Dr. Lionel de Silva which was demonstrated by me and later carefully followed as one of infective endocarditis with ankylostomiasis. A presystolic murmur at the apex and a diastolic murmur over the sternum were present. The fever lingered for months but diminished gradually, the anæmia slowly improved, the murmurs became less numerous and less distinct, and the boy finally felt well. I have reported

below a case in which all the murmurs, including a presystolic, disappeared. This case happened to be admitted as one of mitral stenosis and regurgitation with ankylostomiasis and was under different visiting men who had independently noted their presence. Even if some are diffident about accepting the presence of such presystolic murmurs, contrary to our indelible text-book beliefs that they are present only in organic heart disease, their further study seems imperative if only to shift some of the confusing features of the cardiac complications of ankylostomiasis.

To a physician who was sceptical about the existence of a presystolic murmur in these cases, the observation and treatment of one of this particular type was entrusted. The case was sent to me by Dr. Cyril Fernando from the Out-patient Department, and I in turn handed him over to the in-patient physician. All three of us were agreed that a presystolic murmur was definitely present. After a few weeks in the wards it was observed that in addition to the presystolic murmur disappearing, the suspicion of infective endocarditis was aroused. A full report of the case, No. 6, with a photograph of the temperature chart, is given (see pp. 34-35).

In some institutions in the tropics ankylostoma infection is a source of dread anxiety to the medical man. I mean lying-in institutions in particular. In small out-station hospitals and in the practice among the poor folk in Ceylon, ankylostomiasis is a serious menace to the life of the pregnant mother. I am grateful to the officers in charge of the Lying-in-Home, Colombo, for the demonstration of some of these cases and the gravity of this infection as a cause of unexpected death during, immediately after delivery, and in the first few days of the puerperium. It would seem that such sudden catastrophies occur not only in the very advanced cases exhibiting obvious dyspnœa of a severe infection but even in those in which the dyspnœa occurs only on exertion, and the anæmia does not appear to be profound. Two illustrative cases deserve mention. One was a Sinhalese woman delivered of her second child, with a moderate degree of anæmia, a pulse of 120, slight enlargement (apex in the fifth space just outside the mid-clavicular line) and with no murmurs. The other was a young primipara of whom a grave prognosis was pronounced by my friend Dr. Wickramasuriya. The pulse was 80, there were systolic murmurs

at the apex and at the base, the apex beat was half an inch outside the nipple line, there were no signs of failure and with no apparent dyspnœa even on walking round the room once. In both these cases a grave prognosis was given. I called at the home a week later to see other cases and inquired about these two. To my great surprise I was told that both were dead; one had died immediately after delivery (the primipara), the other, two days after I had seen her. While leaving a profound study of the part ankylostoma infection plays in pregnancy to those who have better opportunities, we will merely record here the gravity of the condition as a cause of sudden death through the damage it does to the myocardium.

We will now go on to study the different clinical types of myocardial affections which are the result of this infection or which are complicated by it. In the first type the suspicion of cardiac involvement arises from the complaint of breathlessness on exertion associated with systolic murmurs at the base or the apex or at both sites. Usually there is no enlargement nor is there any physical sign of failure. We have no proof that the breathlessness is not due to changes in the blood. But

for practical purposes and effective treatment, this kind of case should, in my opinion, be regarded as being due to early myocardial involvement.

The second type is one which presents a picture of congestive failure; in the early stages with râles at the bases of the lungs and slight œdema of the legs, in the advanced stages with marked œdema of the legs, very much enlarged liver and râles at both bases. There is generally moderate cardiac enlargement with all kinds of murmurs. This is the kind of case in which the presystolic murmur previously described occurs. The picture of Case No. 2 (see fig. 1) shows a patient of this type to all appearances, but for the anæmia, like a case of congestive failure due to mitral stenosis. Eleven qualified men were shown this photograph and of these only one, judging by the picture, suggested heart failure due to ankylostoma infection as the cause, and this in a country in which such a disease is so common. The signs and symptoms in these cases are those common to congestive failure.

Case No. 1 (*Type* 2).—A case of anæmia with early heart failure.

L. N. Admitted under Dr. Gunaratnam Cooke, M.D. (Lond.). Aged 51

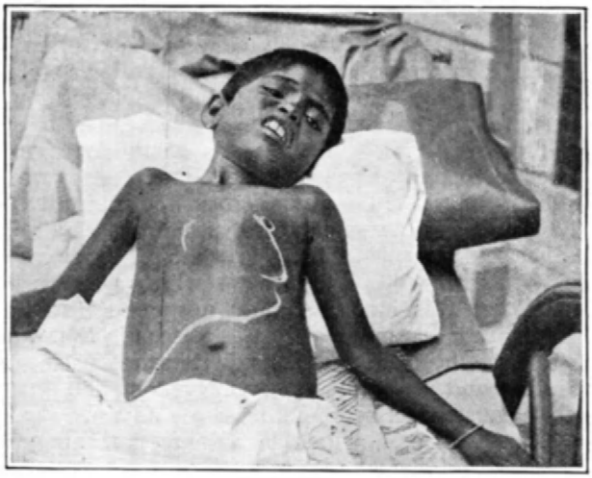


Fig. 1.—Case No. 2 (Type 2)

years, with cough and fever of 10 days' duration.

Previous History.—Nothing of significance.

Present Condition.—Tongue and conjunctivæ pale, legs swollen, breathless. Lungs: râles at both bases. Heart: within normal limits. Systolic murmurs at base and apex; pulse: regular (100). Liver and spleen: not palpable. Blood: red-cell count, 1,500,000; leucocytes, 7,500; polymorphs, 65 per cent.; S. lymphocytes, 10 per cent.; L. lymphocytes, 10 per cent.; hyalines, 1 per cent.; eosinophils, 14 per cent. Urine: specific gravity 1008, re-action, acid. Albumin and sugar, nil. Deposit, few acid crystals. Egg-count: 105 hookworm.

Case No. 2 (*Type* 2).—C. N., aged 9 years. Admitted on November 6th, 1930, with a history of cough and breathlessness of four years' duration; conjunctivæ and tongue pale. Heart: right border, lateral sternal line, the left in anterior axillary line; systolic murmur over pulmonary base, presystolic and systolic in the mitral area (?). Lungs: few râles at both bases. Liver: three fingers below costal margin. Slight œdema (puffiness) of feet and the face. Hookworm + +, round-worms +.

Treatment.—Rest; tinct. digitalis, 4 minims in half an ounce of water once in six hours; Fe and As injections half doses; thymol and mist. alba, chinnapodium later.

History.—Temperature varied between 98° and 101° F. till December 20th; normal after this except for one day, until discharge on February 6th, 1931, when the child was apparently well, with no ova in the stools, no murmurs, a normal pulse, and only slight enlargement remaining.

The third type is met with mostly in adults. The anæmia and breathlessness are very marked. There is often very little or practically no clinically discernible enlargement except in rare cases of this group. Murmurs are often absent; if present, only faint systolic ones, other murmurs being present only very occasionally as in the case described below. The first sounds are generally very poor. Sudden deaths are most common in this type of case and signs of congestive failure are generally absent.

Case No. 3.—J. P., aged 36. Admitted on October 4th, 1930, under Dr. Cyril F. Fernando, M.D. (Lond.), M.R.C.P. (Lond.), with the complaint of breathlessness and swelling of the body for

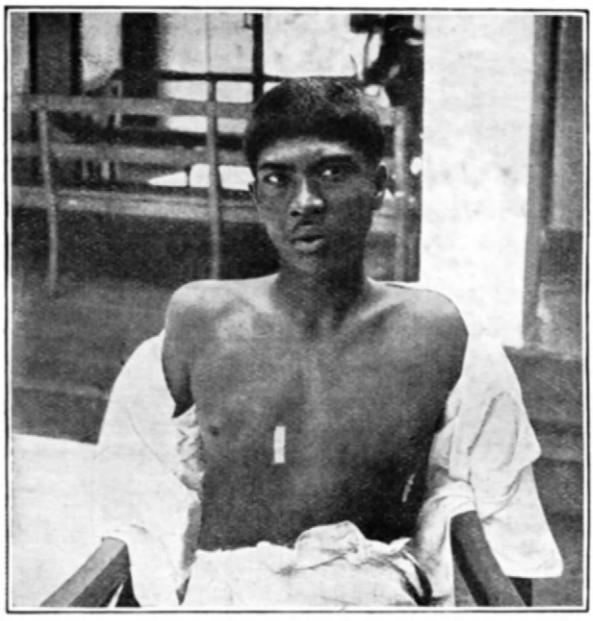


Fig. 2.—Case No. 1 (Type 4).

the last two years. Conjunctivæ pale, tongue pale and flabby.

Previous History.—Nothing of significance. No malaria. No rheumatic fever.

Heart: Apex sixth space 4 in. from the middle line. Presystolic in mitral area. Pulse 100, regular. Lungs: few crepitations at both bases. Urine: albumin, a trace, nothing else; hookworm and round-worm ova +.

Temperature normal till the 14th; 15th, 103° F. (a.m. and p.m.). On the 16th the patient got suddenly dyspnœic and cyanosed and died within a few minutes. Pulmonary embolism was suspected.

Post-mortem disclosed a pale fatty myocardium, no emboli, no evidence of any valvular disease. There was slight œdema of the lungs.

The fourth type is that in which ankylostoma infection complicates organic heart disease.

Case No. 4 (*Type* 4).—E. F., aged 21 years. Admitted on May 6th, 1931, under Dr. Cyril F. Fernando, M.D. (Lond.), M.R.C.P. (Lond.), with aortic regurgitation, mitral stenosis and regurgitation, auricular fibrillation, complaining of pain in the region of the heart and breathlessness, difficulty in speaking and pain

in the region of the shoulder, of four months' duration.

Previous History.—A relapsing fever with pain in the joints and profuse sweating at 14. Nothing else of significance.

Present State.—Somewhat pale: wants to be propped up. No œdema, no cough. Pulse: 78, weak and occasionally irregular. Heart: upper border third rib: right border one finger's breadth external to right sternal border; apex sixth space half an inch outside the nipple line. Systolic and early diastolic murmurs at the apex; diastolic third left interspace. Spleen and liver: not palpable. Urine: nil.

Reported to have left the hospital suddenly, after developing a hemiplegia. (Removed by relatives.)

The fifth type of case met with is the one in which the murmurs of valvular disease appear to be present and the clinical picture is complicated by the presence of an irregular pyrexia. Taken as a whole the clinical picture is in many ways like one of subacute infective endocarditis, specially when the patient has a moderately enlarged (malarial?) spleen. Two illustrative cases are reported. Valvular disease is often diagnosed in these cases and consequently

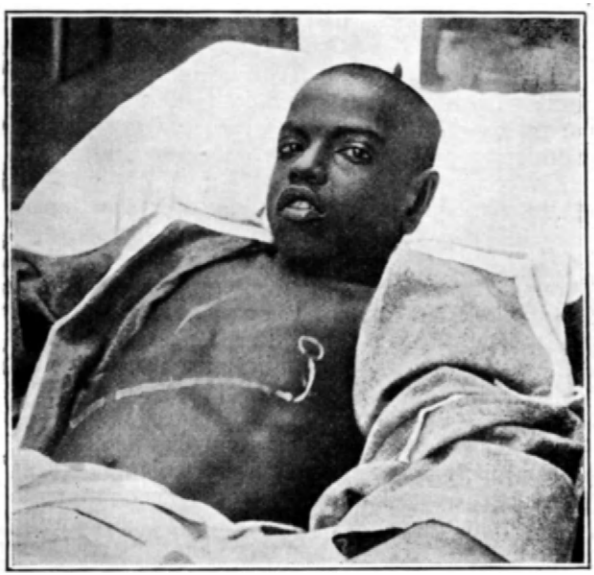


FIG. 3.—Type 5. Case No. 5. Before Treatment.

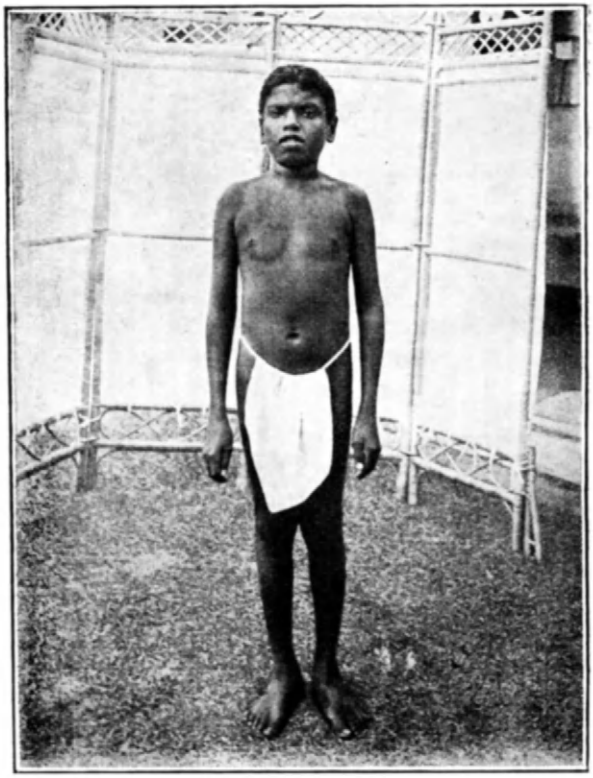


Fig. 4.—Type 5. Same Case. After Treatment

a very bad prognosis is given when the outlook should be one of complete cure in the end. Two such illustrative cases are given.

Case No. 5 (Type 5).—Joseph, aged 15 years. Admitted under Dr. Cyril F. Fernando, M.D. (Lond.), M.R.C.P. (Lond.), as mitral stenosis and regurgitation with ankylostomiasis, complaining of breathlessness on exertion, swelling of the legs, abdomen and face of two months' duration.

Previous History.—Two attacks of fever with pain in the joints, one a year ago and the other two months ago.

Heart: moderately enlarged with systolic and diastolic murmurs and gallop rhythm in the mitral area; also a pulmonary systolic murmur. Liver: three fingers below costal margin.

Temperature on admission 101° F. Pulse 120, regular. Hookworms 59,600 per c.c., round-worms 12,400. Red-cell count: 1,490,000.

Left hospital after a few months, perfectly fit and well, with no ova, no murmurs lying down, standing or on exercise. The photographs of the patient on admission and on his discharge are shown opposite. This patient was examined before he left by the three visiting

physicians of the hospital, under whose care he was during his stay in hospital.

Case No. 6 (*Type* 5).—Saineris, aged 35 years. Admitted by Dr. Cyril F. Fernando, M.D. (Lond.), M.R.C.P. (Lond.), under the care of Dr. J. R. Blaze, M.D. (Lond.), M.R.C.P. (Lond.), for advanced ankylostomiasis with a presystolic murmur, complaining of dyspnœa, pain in the chest, and œdema of both legs of a year's duration.

Previous History.—Malaria one year ago.

Conjunctivæ anæmic and muddy coloured. Heart: apex beat quarter of an inch outside the nipple line. Presystolic murmur in the mitral area, no thrill; systolic in the pulmonary and aortic areas. Spleen and liver: not palpable. Urine: nil. Fæces: ankylostoma and ascaris ova; eggs, necator 40,800, ascaris 45,000. Van den Bergh: direct and indirect nil. Blood: hæmoglobin 20 per cent., red blood-cells 2,300,000. Lungs: clear.

November 13.—On hexyl resorcinol tablets, mist. alba twenty-four hours later.

November 14.—Worm-count 55, ascaris nil, pulse 110, volume and tension fair.

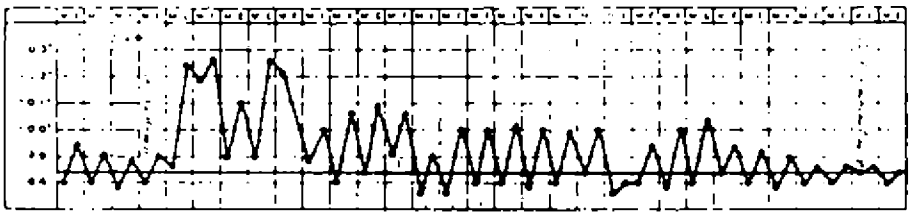


FIG. 5.—Case 6. Temperature Chart

November 18.—Electrocardiograph, on digitalis.

November 26.—Heart and liver +; mist. diuretic and tr. digitalis ℥ 20 t.d.s.

November 28.—Improving.

December 4.—Œdema improving; presystolic practically absent.

December 6.—Œdema much less, but patient still anæmic.

December 8.—Hexyl resorcinol tablets; mist. alba.

December 13.—Improving; egg-count, ascaris nil, ankylostoma ova nil.

December 15.—Discharged; no presystolic murmur. Patient not completely well, but left of his own accord.

The temperature chart is shown.

In the sixth category are included the five kinds of cases described above, but occurring in pregnant women. This kind is common in lying-in hospitals, and in these sudden death is a frequent termination. It appears obvious that the cause of sudden death is often not the physical strain of labour. Could it be the sudden release of pressure of the pregnant uterus on the abdominal veins, a consequent increase of the blood-volume entering the heart, throwing too much of a strain on the damaged myocardium already exhausted by the strain of labour? This we can verify only

with further study and observation. For the present our concern should be to free the pregnant woman in the earlier months both from the infection and the toxæmia and blood loss which is a result of it.

If I attempt to detail the treatment necessary for these cases I will be going beyond the object of this contribution, namely, to describe the cardiac complications which follow infection with ankylostoma. So much has been written on the treatment of this condition that a course could easily be outlined which would reckon with the attendant cardiac disability in order to avoid those measures which through their violent reactions will only add to the burden of the heart-muscle. Rest is essential until long after the patient is rid of his ova and worms. Iron and arsenic injections form part of the routine treatment. Good nourishment and fresh air are necessary. With these and careful management, many a heart seriously damaged by the toxins of these parasites will regain both good function and strength.

Heart disease due to ankylostoma infection might justly be called the 'poor man's heart disease' of the tropics. In practice, in high society they are rare. For six years I have not seen one

such case. Its frequency among the poor, and often the helpless poor, should be an incentive to further study, particularly in view of the fact that the sequelæ of this infection can, as I hope I have shown, be easily mistaken for organic valvular disease and consequently labelled 'incurable'. To stress this point I have deliberately chosen cases which, according to the recorded observations of competent and highly qualified observers, have shown some of the physical signs of rheumatic valvular disease.

CHAPTER IV.

RHEUMATIC HEART DISEASE, DIABETES, DIPHTHERIA, AND SYPHILIS.

SECTION 1.

RHEUMATIC HEART DISEASE.

It is stated by some, but on no reliable pathological data that rheumatic fever does not occur in the tropics.[1] Others have gone further and appear to lend colour to the contrary view by stating that the disease is the result of a protozoan infection,[2] the Aschoff body being regarded as manifestation of the presence of such a parasite. The evidence in favour of both these views is so meagre that if they are not ignored for any other reason, they should be for the reason that there is overwhelming clinical evidence that rheumatic fever and its complications are certainly met with in the tropics. A fever following a recent sore throat with joint pains associated with or followed by valvular lesions more often in young children, is not uncommonly met with.

[1] Tertius Clarke

[2] Arbuer Stephens, B.M.J. August 25th, 1928.

In a paper read before the Ceylon Branch of the British Medical Association quite recently Dr. Cyril Fernando, M.D., M.R.C.P. (Lond.), gave notes of 40 cases of rheumatic fever showing convincing evidence that all the usual sequelæ of this condition are met with in the tropics. Confusion as to their existence appears to have arisen from the fact that :—

1. The initial attacks of fever or joint pains, or both, are not so severe as in the West.

2. The tendency to recurrence of attacks is strikingly much less.

3. The cardiac complications are not generally so serious, nor as judged by the later sequelæ, so progressive.

4. Clinically evident pericarditis is uncommon.

5. Other sequelæ of infection with the organism responsible for rheumatic fever are very rarely seen, e.g. nodules and chorea.

6. As elsewhere, but evidently much more frequently the only evidence of the previous infection are the sequelæ.

Two facts then should be remembered in connection with the cardiac sequelæ of rheumatic fever in the tropics.

First that in a few cases the course of the fever, its tendency to recurrence and

its complications may be the same as those in the West.

Secondly the disease as a rule runs a mild course, the cardiac complications being evident only in some. Short notes illustrative of cases of the two groups are:—

First Group.

1. A boy aged 12 years was seen by me in his second attack of fever with joint pains. The tonsils had been removed since the first attack. He had also mitral regurgitation. On the second occasion he developed a carditis, pericarditis being a prominent feature, and after being seriously ill for three months he died. Post-mortems proved there were old mitral disease and a recent pancarditis.

2. A young girl of 20 under a colleague of mine suddenly developed a right-sided hemiplegia soon after the death of her father. Examination revealed mitral valve disease. After a long illness with complications during which she was given large dose of rheumatic phylacogen she recovered. Two and a half years later she was left with a homiplegia and fully developed mitral stenosis. There was no history of rheumatic fever or joint pains.

3. The youngest case of mitral ste-

nosis I have seen was seen in a village-girl, 5 years of age. There was a presystolic thrill, a much presystolic murmur and a bifid P in the electrocardiogram. She was brought to the out-patient department for slight breathlessness on exertion. (Reported in journal Ceylon Branch of British Medical Association). There was no history of joint pains or influenza.

Second Group.

1. A lady patient of mine was first seen by me in London 13 years ago for slight pre-cordial pain and occasional palpitation. Examination revealed a presystolic murmur and mitral stenosis was diagnosed. The late Sir Sydney Russell Wells saw her in consultation and confirmed the diagnosis. She has been seen later by cardiologists in London and there is no doubt whatever as to the diagnosis. All these years she has carried on fulfilling her social obligations with apparently no alteration in the state of her heart.

2. A young man of 20 consulted me for 'thumping of his heart' and for not feeling very fit. Three months before he was laid up for two weeks with a low fever without joint pains. Influenza and paratyphoid fever were suspected. His

fever left him and a few days later he developed extra-systoles and reliable physicians then found no evidence of valvular disease. Subsequently he stood a minor operation (hydrocele, under local anæsthesia). At the time I saw him he had extra-systoles and was pale in colour. In addition he had very slight cardiac enlargement (to percussion) and definite diastolic and systolic murmurs at the apex. The temperature varied from 98-99·4. This boy was under observation for two months, during which time the character of the murmurs changed very often, a short diastolic murmur developing along the left border of the sternum, in addition to the others. There was no clinical evidence beyond the changing murmurs to suggest an infective endocarditis, but the possibility of the case proving to be one had to be entertained. A full report of the case was sent to Dr. T. F. Cotton in London who suggested the condition was probably a sequel of rheumatic infection. The boy was given rest and was treated, in the usual manner, as a case of rheumatic carditis; rheumatic phylacogen was also given in big doses. At the end of 9 months he was reported to be well and to having put on 23 pounds in this time. Eighteen months from the beginning of his illness his

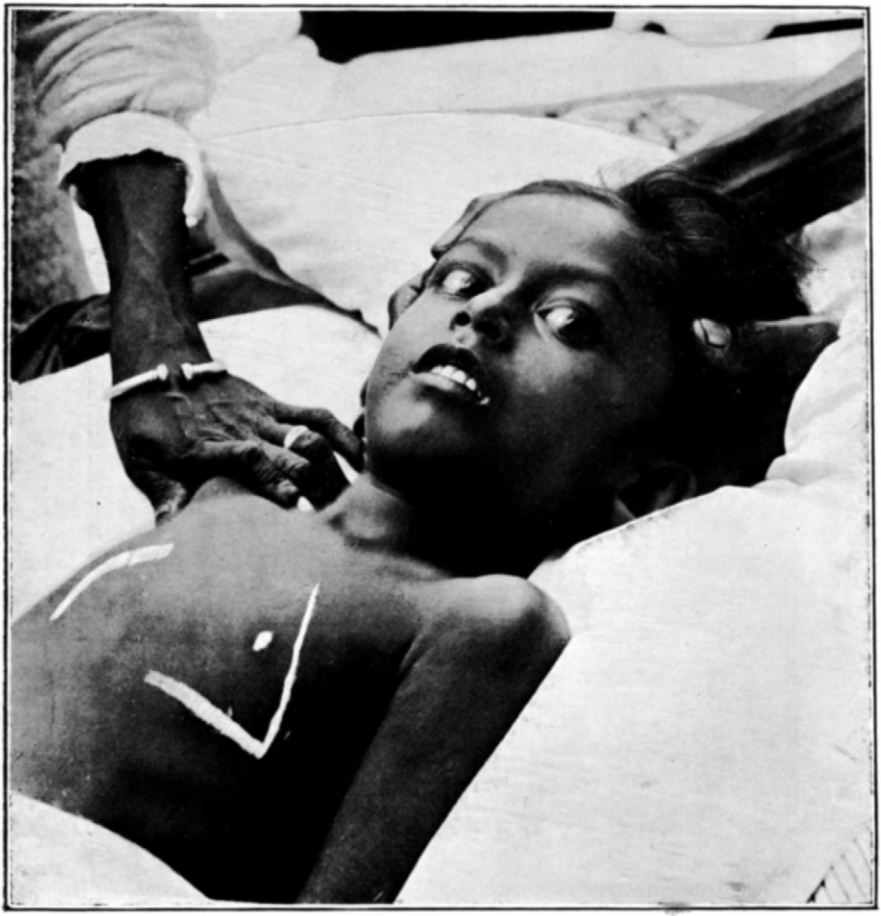
heart showed no abnormality on clinical examination.

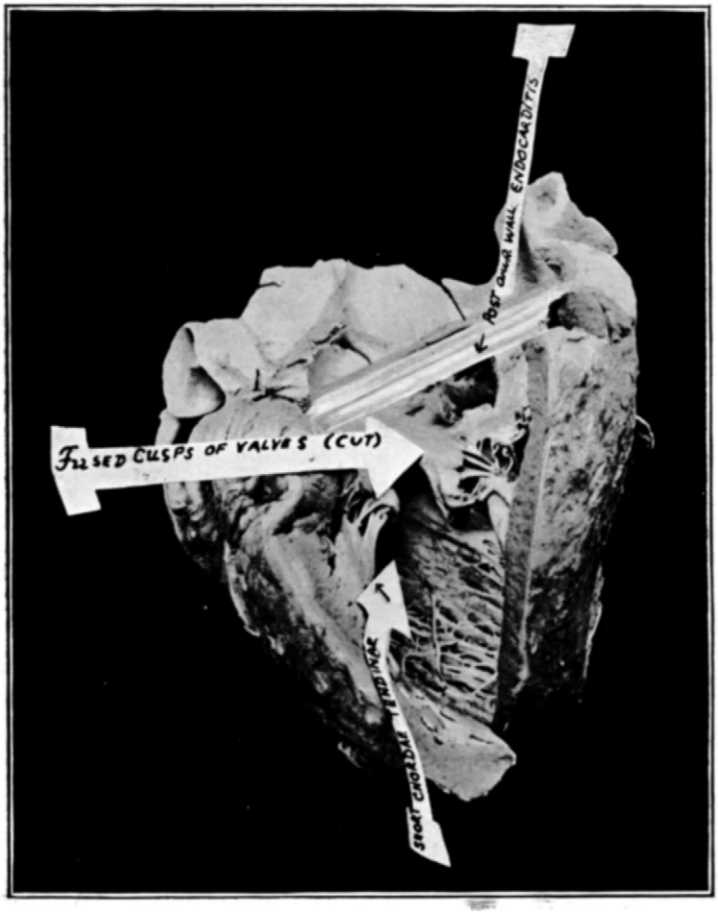
3. Many cases have been under my care, including three cases of chorea some of which have developed no cardiac disability whilst of those others which suggested valvular involvement, the majority have no valvular trouble now.

If the existence of both these types of cases with a striking predominance of the latter is borne in mind it would be wise to look upon rheumatic cardiac affections as being less serious in the tropics than in Western countries. The rarity of auricular fibrillation, the incidence of which is low besides the number of cases of mitral stenosis met with, also seems to favour this view. The latter condition which is met with more frequently than one expects to, in view of the fact that definite rheumatic fever is not so prevalent, should always mean the recognition of a very definite cardiac disability but again with a better prognosis than one would give, for instance, in a similar case in England. In any case, especially when it is seen for the first time, it is better to defer a final opinion for some months whilst the patient is left under observation and every measure advocated for arresting the rheumatic process is resorted to.

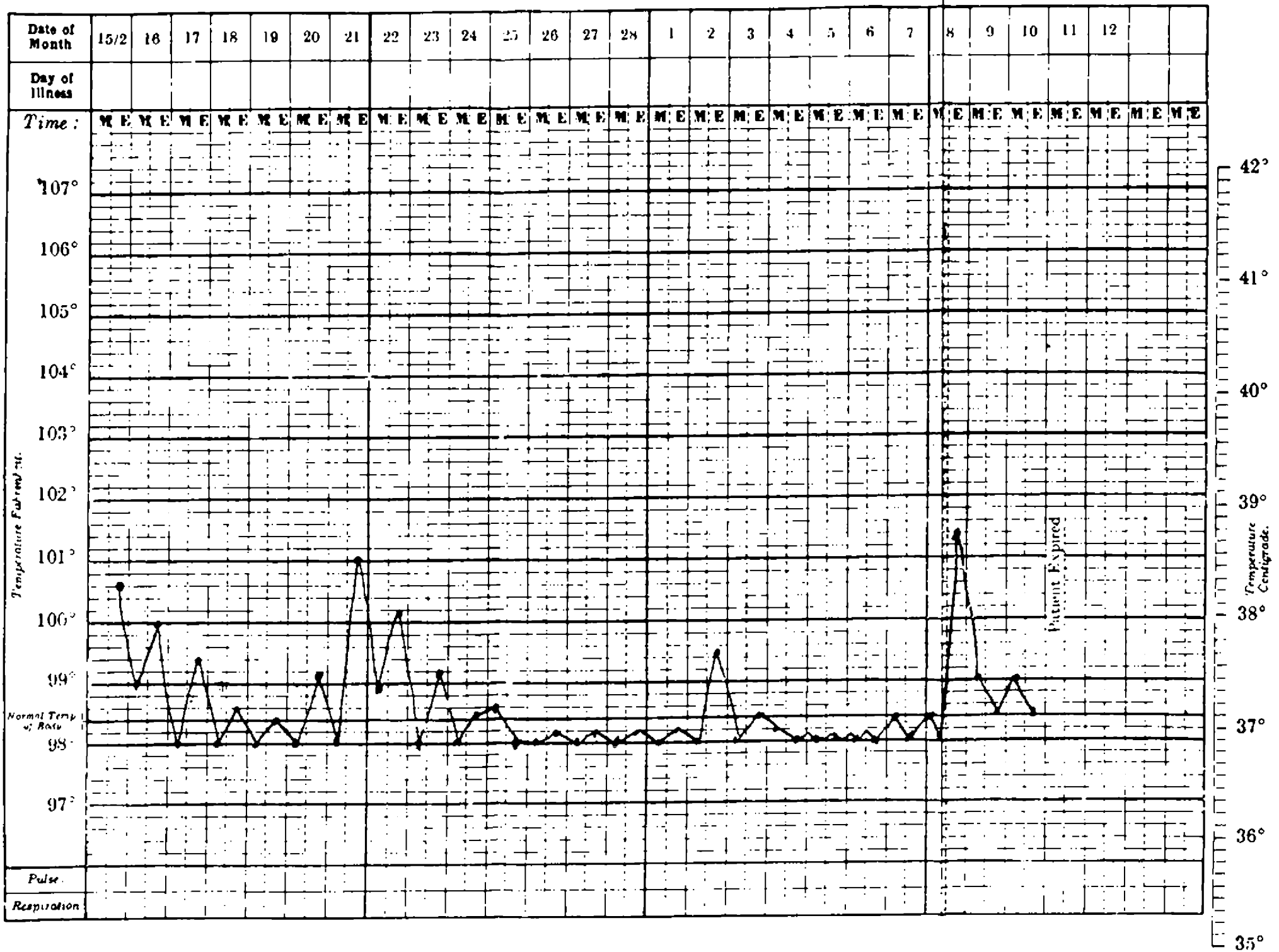
Case 2261 | 5 | 15-2-34.

A typical case which illustrates the occurrence of acute rheumatism with its various manifestations is described overleaf.




POST. CHORD WALL ENDOCARDITIS
FUSED CUSPS OF VALVES (CUT)
ANT. CHORD ENDOCARDITIS

Name of Patient : Hemalatha Mahanama **Age :** 9. **Sex :** F. **Disease :** Chorea and Rheumatic Carditis.



Case [illegible] S. *Hemalatha* [illegible]. Female [illegible] years. Singhalese.

(Admitted under the care of Dr. E. Garvin Mack, to whom I am indebted for these notes.)

Admitted with a history of spasmodic irregular movements of the limbs of one week's duration.

History of Previous Illness

Attacks of fever with pain in the joints two years ago and two months ago. Details of these illnesses are not available.

Condition on Examination

Patient has severe spasmodic involuntary movements of both upper and lower extremities, involuntary twitchings of the facial muscles on both sides. The movements are very irregular, increased by attention and are typical of chorea.

Tongue moist and clean. Patient finds it difficult to hold the tongue protruded for any length of time and so finds it difficult to keep it still.

Throat. Healthy looking. No enlarged tonsils.

Heart.

3rd rib

[illegible] right lateral sternal line | [illegible] outside the mid-clavicular line

No adventitious sounds heard.

Pulse [illegible] V. & T. good. Regular.

Lungs
Abdomen N.A.D.
Liver
Spleen

Patient finds it difficult to talk. No paralysis of the external ocular muscles, no abnormalities of the pupil reactions.

Knee jerks—present, not sustained.

Plantar reflex. Flexor.

Wrists are kept in a position of flexion but there is no marked hyperextension of the fingers.

Micturition normal.

Urine—Sp. [illegible]
Albumin nil
Sugar [illegible]
Reaction acid

The patient was put on the following mixture:

℞ [illegible]
[illegible]
[illegible] ʒi
[illegible]
[illegible] ʒi
ʒi [illegible]

℞ Half a tablet of [illegible] for a dose four times a day.

The patient made steady improvement; on the [illegible] the choreic movements greatly improved and on 26.2.[illegible] [illegible] were omitted. On the same day the child complained of pain of both wrists and movements were painful. The patient appeared to improve up to the 6.3.[illegible]. The choreic movements were less and the temperature had remained normal for about ten days.

[illegible] Heart. A soft systolic murmur (mitral area) was detected.

[illegible] Heart.

2nd interspace

Rt. lateral sternal line | H. A. B. [illegible] outside Mid. Clav. line in the sixth space.

[illegible] over the precordium. Systolic and diastolic murmurs heard over the mitral area.

2.40 p. A severe attack of dyspnoea lasting a few minutes, patient sat up and gasping for breath. The attack passed off with oxygen inhalation.

[illegible]

[illegible] p.m. Patient quiet, oxygen is present. Respiration [illegible] per minute.

[illegible] Patient [illegible] dyspnoea, unable to speak.

2.2[illegible] p.m. [illegible]

Pulse 130. V. & T. poor.

Heart sounds very rapid, double murmur at apex.

Lungs: [illegible] heard over right lung, upper part posteriorly.

Patient expired 2.[illegible] p.m.

Post Mortem Examination

Heart. [illegible]

Mitral valve: [illegible] and adherent and [illegible].

[illegible] of the [illegible] of left auricle immediately above posterior cusp [illegible].

Dry pleurisy present.

Treatment should be carried out on the usual lines. Even in the tropics it is best to err on the side of too much rest than too little. For the reason that two cases known to me gave such good results, phylacogen might be tried. Perhaps in the tropics, conditions being favourable to the arrest of the disease, therapeutic measures of this kind may give better results than in temperate climates. If the child is living in the hills he should be brought down. As Sir Thomas Lewis[1] says, 'Patients in whom the disease is running a long course should be removed whenever possible to a hot sunny climate. Experience is beginning to show, too, that recurrences are rare so long as the child is kept in tropical or sub-tropical countries.' This latter fact and the possibilities which it opens out have been referred to by me elsewhere in reference to Ceylon:[2] 'If we can prove that it (rheumatic fever) runs a mild course without the deadly complications so frequent in the West we may at least make this Island an attraction to the very large numbers

[1] Lewis, Sir Thomas—Diseases of the Heart, (1933) Macmillan, p. 201

[2] Journal of the Ceylon Branch of the British Medical Association—Presidential Address, June, 1933.

of wealthy people abroad whose children are crippled by the sequelæ of the disease, and who live in constant dread of a recurrence. If the disease as we see it is clearly studied and our knowledge of its complications disseminated throughout the world, we may find sanatoria for the victims of acute rheumatism studded throughout the island.'

Section 2.

DIABETES.

This is another condition which is common in the tropics and deserves mention here on account of the fact that cardio-vascular changes are a frequent accompaniment of it. Boyd states, 'Arteriosclerosis is a common accompaniment of diabetes . . . . . severe myocardial degeneration is common in diabetes over 40 years of age[1] . . . . . the renal arteries and kidneys may be affected'.[2] With the use of insulin and the more widespread adoption of the modern methods used in controlling the condition the cardio-vascular picture is bound to come more into prominence

[1] Boyd. Pathology of Internal Diseases, 1931, p. 391.

[2] Boyd. Text Book of Pathology, 2nd Ed. 1934, p. 597.

as the years roll by. Hamilton and Root[3] stress this fact when they state that 'the increase in duration of life of diabetic patients will have at least an equal effect in swelling the incidence of arteriosclerotic heart disease as a cause of death in diabetes that increasing prolongation of life has in the general population'. According to these authors 'patients who succumb in the early stages of coma may die because of complicating cardio-vascular disease rather than because of the coma itself', a fact which is not often borne in mind when the condition of coma stands out so prominently and for the moment at any rate claims all the attention of the practitioner. The necessity of absolute rest for patients in a condition of coma has been recently stressed by several writers, some like D. E. Bedford maintaining that the collapse is due to peripheral and not cardiac failure. It is also pointed out by Hamilton and Root 'that two factors serve to disguise the severity of the cardiac damage: 1. diabetic patients notably in the past, less so since the use of insulin, have been weak, inactive and hence less likely to

[3] Hamilton, B. and Root, H., in Treatment of Diabetes Mellitus by Joslin, 4th ed., 1928, p. 701.

impose strain on the heart muscle to provoke anginal symptoms; 2. the sensitiveness of the diabetic nervous system to pain is less than that of the normal'.

Following on those changes in the vascular system symptoms are occasioned both in the periphery and in the heart itself although not always in a measure which appears to be proportionate to the degree of changes in the vessels. We are all very familiar with the picture of diabetic gangrene but what frequently occasions more alarm both to the patient and to his doctor are the cardiac manifestations. Generally speaking, it is wise to look upon the diabetic as one prone to any one of the sequelæ of coronary sclerosis. Congestive failure, coronary infarction or occlusion and auricular fibrillation have all been met with rarely. But a symptom not uncommonly met with is angina pectoris. When this does occur without placidly resigning oneself to the condition of coronary sclerosis it is well to bear in mind that two other factors should always be eliminated. The first is an infective condition which in the diabetic finds the circumstances for working evil when in the ordinary individual it may not cause any trouble. A case of mine illustrates how important it is to

recognize this possibility. A very influential man, aged 65, for 20 years a diabetic, complained of mild attacks of substernal pain associated with unpleasant feelings and faintness. The frequency and severity of these attacks gradually increased till it came to a time when he had 8 or 10 a day even when he was completely at rest lying down in bed. The usual measures, like amyl nitrite, which used to give him relief now answered but sluggishly or not at all. Owing to his age, his long standing diabetes and among other observations, the fact that he had many attacks a day the prognosis was considered so grave that it was thought unwise to attempt to remove at the time a few of his teeth which were very septic. As days went by without improvement it was decided to have these removed one by one under local anæsthesia. Practically each one opened out a pocket of pus. In the course of a few days the patient improved and within a few weeks insisted on getting up and going about. For two years subsequently he had no further attacks and considering himself well again, went back to a fairly active political life. It is now 4 years since his illness; but for these mild attacks he has been quite well.

The second condition is what one may describe as a relative ischemia of the heart muscle induced by the diabetic state. Although in the more severe stages of diabetes the heart muscle has an adequate supply of glycogen, its utilisation is interfered with possibly owing to insulin deficiency. Whatever the actual cause, that some such disability obtains is shewn by the valuable observations of Shirley Smith[4] to whose contributions on the subject the reader is referred to for further information. Vandenberg[5] believes that the sclerotic heart may require an unusually high level of blood sugar. Although the latter is present in diabetes in excess of the normal the heart muscle is not able to convert it to its use so competently as in the normal subject. The changes in the functional efficiency of the heart muscle attendant on this disability have been studied by several authors and some of these are demonstrated by Smith and Hickling[6], in the electrocardiographic changes observed in many cases of diabetes undergoing adequate treatment. These tracings show the increase in amplitude

[4] Shirley Smith, Lancet March 25th 1933.

[5] Vandenberg, Julius Springer Berlin, 1926 p. 222

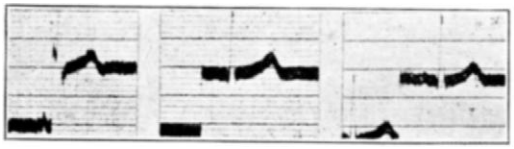
[6] Shirley Smith and Hickling, R. A., Lancet, March 5th 1932

of the T. wave within a week of treatment and what is more important reversion to the normal of the definitely inverted T. wave observed before treatment. (*See* tracings on opposite plate.) The importance of these changes will be appreciated when one bears in mind the serious prognosis and the liability to sudden death generally associated with the inversion of the T. wave in leads 1 and 2. These authors conclude that 'the electrocardiographic changes produced by diabetes represent mainly a parenchymatous damage to the heart muscle resulting from defective nutrition.'

The observations rendered above must therefore be borne in mind in the treatment of the cardio-vascular complications of diabetes which are bound to figure more prominently in the van of a multiple symptomatology as the number of diabetics efficiently treated increased. The first step is to resort to the scientific treatment of the diabetic. This becomes all the more necessary in view of Aschoff's[1] finding that the early intimal fatty changes frequent in the diabetic are reversible. This essential first measure will obviate also the haphazard reduction of carbohydrate diet with the earliest

[1] Aschoff, Lectures on Pathology, New York, 1924, p. 133.

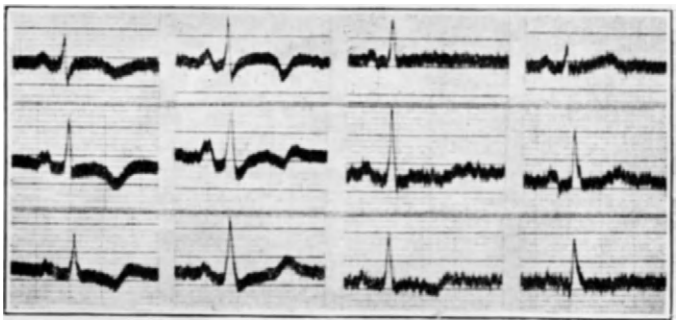
FIG. 1 (Case 6) *p. 52*



1 week 5 weeks

Successive records (lead I) showing stabilization and increased amplitude of T-wave after one week of treatment

FIG. 2 (Case 11) *p. 52*



1 days. 5 weeks 15 weeks

Female diabetic, aged 39. First ECG taken before treatment shows concave inversion of T-waves, their form in leads I and II suggesting "coronary" T-waves. Later records during treatment show gradual reversion to normal after 15 weeks.

manifestations of cardiac symptoms. 'In diseased hearts or in the sclerotic diabetic heart long accustomed to an increased blood sugar it seems likely that sudden alterations in blood sugar may be dangerous whether induced by restriction of diet or by insulin.' The diabetic victim of angina has observed this himself. More than one patient of mine has remarked 'I am better when I take a little rice'. But the extent to which carbohydrates should be allowed should depend on the method of treatment employed. The signs and symptoms referable to the heart must be taken into account in assessing the degree of cardiac disability and the patient's activity restricted or his mode of life altered to suit not only his capacity for physical work but also his general condition.

A point I have already referred to is the importance of attending to and eliminating septic foci with the necessary precautions, recognizing that in a diabetic even mild operative measures of any kind should be undertaken only when absolutely necessary. Smith has described the beneficial effects of insulin in the treatment of angina pectoris in the elderly.* He advises giving 5 units of

* Shufflebotham & Smith: B.M.J. April 22nd, 1933, p. 695.

insulin before breakfast and the evening meal, with 30 grams of glucose taken with the meal for over 2 weeks. Virchow and Muller[9] have also shown that in normal mammalian hearts insulin causes a slight increase in the power of contraction. Results similar to those described by Smith have been obtained in the diabetics also but in the latter case it must be remembered that both anginal attacks and heart failure have been precipitated by the use of insulin—or as we should say by the irrational use of insulin—either by inducing what Parsonett and Hyman[10] have described as 'insulin angina' or possibly by inducing a hypoglycæmia. I can recall a case of a high priest, 65 years old, whom I electrocardiographed one afternoon four years ago. The tracing showed flattened T.s in leads 1 and 2 but no inversion. He was being given insulin by his doctor. The same night he died suddenly and I was allotted no small share of discredit because my tracing had not revealed the possibility of sudden death. It is obvious now that an alternative explanation could have been

[9] Virchow and Muller, Journal of Physiology, 1927, 62,341.

[10] Parsonnet, A. E. and Hyman, A. S., Archives Internal Med., 1931, IV, 1247

found particularly in view of the fact that insulin was being administered in the careless way it is often used.

Section 3.

DIPHTHERIA.

As in rheumatic fever the course and sequelæ of this disease are modified in the tropics. Its cardiac complications are seldom serious, the membrane does not become so easily evident, the toxæmia is much less than in temperate climates and the cardiac complications only mild. Fatal cases occur much less frequently than in the West. Overlooking exceptions it may be said that the condition when it begins and ends in inflammation of the throat is often not diagnosed. In the majority of cases with cardiac complications seen by me the initial diphtheria had been overlooked. In several cases one child in the family was away with the disease whilst another was laid up with cardiac symptoms giving a definite history of recent sore throat. Fatal cases from heart failure except in the very young or unless the patient is in the hills is extremely rare. The usual story is one of a tachycardia of fairly sudden onset associated with a feeling of faintness. The child appears to be

uncomfortable, the pulse is fast, generally about 120, and there is a rapid diffuse apex beat with no ascertainable enlargement. The parents as well as the doctor gets alarmed, and the latter very rightly when every text-book in his possession refers to the possibility of sudden death. Fortunately this occurs very rarely. The figures of the Infectious Diseases Hospital, Colombo, into which the poorer classes are admitted, suggest that sudden death preceded by some kind of irregularity is not uncommon. The total admissions to this institute for the six years ending 1933 were 112. Among these there were 30 deaths, 17 with cardiac failure, 2 with respiratory distress, 1 with broncho-pneumonia. In 1932 there were 6 deaths, 5 from cardiac failure. In 1933 there were 8 deaths, 7 from cardiac failure. I have seen only one case in which myocardial changes seem to date from an attack of diphtheria at 4 years of age. Of this infection being the cause one could hardly be sure seeing that the patient is now 25 years of age and exhibiting periodically attacks of auricular fibrillation and heart block associated with mild anginal pains. The patient appeared to be in good health, had a fairly good exercise tolerance, was

slightly on the fat side, and gives a very definite history of heart trouble since diphtheria, a picture which in the whole hardly warranted the diagnosis of the more serious forms of myocardial degeneration, but one which justified the question, could this be the result of some damage inflicted by diphtheria such as fibrosis as has been observed by Warthin[1]? Serious cardiac complications however are rare. The author himself was a victim to diphtheria thirty-three years ago; the second in the family to get it, after the first, a younger sister, died of it. He suffered from many of the usual sequelæ, paralysis of the palate, diplopia and weakness of the leg muscles. He can recall very vividly and with amusement how the doctors attending on him used to make him run in order to afford themselves the fun of seeing him stumble within a few yards of the start. The circumstances would suggest that the heart never came into serious reckoning, and these in the days before serum came into use!

For treatment the child is given complete rest, flat in bed, and proper nursing. The bedpan should be used and under no

[1] Warthin A. S., Journal of the Tech. Dis., 1924, XXXV, 1932.

circumstances must the child be allowed to get up for at least a fortnight. Attention to the bowels, general health measures, plenty of fluids, a mixture containing a little bromide to act as a sedative, a few months of restraint from physical exercise and a final exercise tolerance test are all that is required.

But usually neither the failure in the early toxæmic stage described by Schwentker and Noel[2] nor the grave form of the late stage with vomiting, slight enlargement and heartblock are observed.

Section 4.

SYPHILIS.

In the cardio-vascular manifestations of syphilis in the tropics there are certain features which are of no little interest. For a reason unknown to us general paralysis of the insane and even tabes are not nearly so common in the tropics as in Western countries, at any rate among the indigenous population. Possibly for a similar reason aortic regurgitation is relatively uncommon, although meningo-vascular syphilis is commonly

[2] Schwentker and Noel, Bulletin John Hopkins Hosp., Nov., 1929, XLV—(April and June), 1930, XLVI

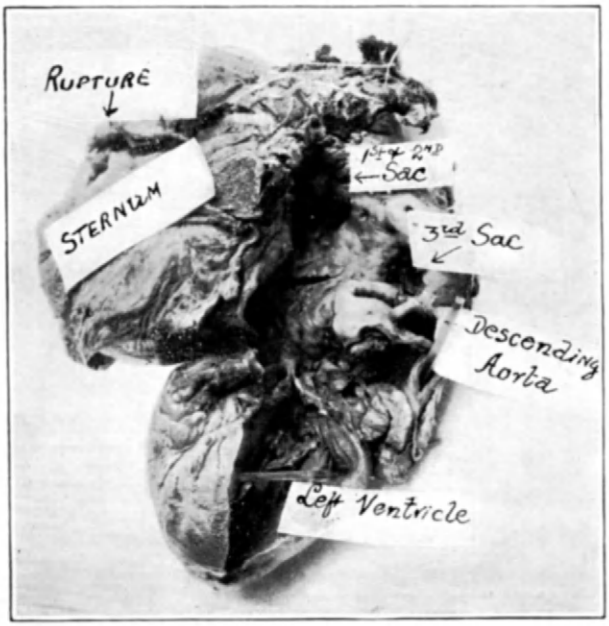
met with. William Fletcher[1] has stated that 'lesions of the heart and aorta are particularly common and contrary to experience in temperate climates—where acute rheumatism is common and the mitral valve more often affected than any of the others—in the tropics it is the aortic valve which is most often diseased, and the cause is nearly always syphilis'. It is not clear what incidence in actual statistics this statement implies, in any case it does not indicate the frequency of aortic regurgitation which according to my clinical experience is uncommon.

Aneurysm of the aorta occurs again less often than in Europe. In the absence of reliable statistics it would be unwise to do more just now than suggest further enquiry and a closer study of the incidence of syphilitic cardio-vascular disease in the tropics. In a review of my experience the infrequency of aortic regurgitation both in hospital and specially in consulting practice would tempt me to doubt the American figures which show the spironema pallidum to be responsible in so large a percentage but that one is

[1] W. Fletcher—Practice of Medicine in the Tropics, by Byam and Archibald. Vol II, p. 1282.

familiar with the peculiarities of the latter in its treatment of the various races. All the manifestations of syphilis appear earlier than one meets with in Western clinics. We trace the fact either to earlier maturity or the easier access under conditions of life in certain quarters to the sources of infection. Aneurysm, for instance, occurs earlier. I do not like to exaggerate this impression by the photograph shown opposite, namely, one of three aneurysms in a woman of 26, who showed no evidence of congenital syphilis. But the fact I think is generally admitted.

Treatment is given with the usual care necessary in the case of cardiac affections. Reference is made in *Chap. VII, Therapeutic Considerations* to the precautions necessary in the use of potassium iodide in the tropics.


RUPTURE
STERNUM
1st & 2nd Sac
3rd Sac
Descending Aorta
Left Ventricle

CHAPTER V.

HIGH BLOOD PRESSURE.

High blood pressure is a very common cause of cardiac disability in the tropics. In private practice it accounts for at least 20% of all patients who seek medical aid with symptoms referable to the cardio-vascular system.

We may say that a patient has high blood pressure when the pressure is persistently above certain figures, namely a diastolic of 100 and a systolic of 150. The elevation of the systolic pressure alone is not an indication of high pressure. For the condition to be diagnosed the diastolic pressure must be 100 or over. For the patient's safety a diastolic of 90 should be regarded as indicating a tendency to high pressure. A high systolic pressure with a diastolic lower than normal is always suggestive of aortic regurgitation. Similar figures are often obtained in some cases of arteriosclerosis but in this latter condition the diastolic is slightly higher. In elderly men when figures in the neighbourhood of 100 for the diastolic is obtained with a high systolic of 170 or 180, or even higher, nearly always thickened arteries

are present particularly the brachials, the radials and the temporals. Such are not genuine cases of high blood pressure, the elevation of pressure appearing to be a compensatory phenomenon to extensive changes in the arteries. Everything considered, the diastolic pressure is the one to be relied on for guidance and therefore its persistent elevation above 90 may be taken as a tendency to high arterial pressure and one above 100 as high blood pressure in its early stage. As the elevation of pressure may be transient particularly when it is due to psychological factors, the presence of persistently high pressure will have to be ascertained by repeated examination.

How to Determine Blood Pressure.

There is in my opinion only one method of accurately determining the blood pressure in the human being, and that is by the use of a reliable blood pressure machine. The method of approximately gauging the pressure with the finger is hopelessly fallacious. I have only too often seen the ice bag placed on the head, croton oil administered and leeches applied to patients with low blood pressure exhibiting pareses due probably to cerebral thrombosis.

consequent to a diagnosis being made by the finger over the radial, of hypertension with threatened haemorrhage. Serious mistakes have been made by the experienced as well as the inexperienced. To those who have no blood pressure instrument or where one is unexpectedly called upon to examine a patient, the aortic second sound will give far more reliable information. But no room will be left for doubt if a reliable instrument is used. I feel disposed to say that in the interests of his patients every doctor ought to have an instrument. Cabot has not put it too strongly when he said 'If I were allowed to have only two instruments of precision for my aid in physical diagnosis, they would be the stethoscope and the blood pressure machine'.

What is the best instrument to use? By far the most useful and the most accurate is a simple form of mercurial manometer. The present-day Riva-Rocci instrument is as good as any for practical purposes. It has one disadvantage, namely that of having figures which cannot be easily seen at night in the dim light of a patient's room. But an instrument which is almost perfect in its accuracy and its appearance is the Acossan manometer.

The desk model unfortunately gives figures up to 260 only. Why, I do not know. It is not rare to get higher systolic pressures than this and one is often left with the desire to know, specially when records are kept for purposes of investigation, what the actual figure is. For this reason I would prefer the model with a scale reading up to 300.

If I were advising anyone I would say positively 'do not buy an aneroid machine for use in the tropics'. I have only too often seen errors up to 50 mm. in my private practice as well as in hospital wards. On one occasion a whole series of investigations was rendered useless by a later discovery that this machine was inaccurate. Compactness and portability are often sought at the expense of scientific accuracy. If these are sought, let the machine be a mercury one, and as simple as possible. After experience with a variety of instruments I am convinced that this type of machine is the most reliable one.

There are a few practical points to be observed before the reading is taken. The latter should always be done last. The preliminary conversation with the patient, the writing of the history, the ordinary examination, they all help the patient to settle down while the instru-

ment is tested and the readings are adjusted. He ought to be told quietly that the armlet will press on the arm and that any unusual sensation will be due to the pressure. The bag should not be left inflated till a feeling of numbness is experienced. If the readings cannot be easily obtained it is best to release the air and start again. When there is any doubt, specially about lower figures, as to whether a higher pressure than normal is present or not, the examination should be repeated after ten minutes.

The patient should be reclining or seated in a position of comfort and relaxation; the armlet is then placed round the arm evenly and not too tightly and the cloth band carefully wound round. The end is tightened with clip or tucked in round the last few folds. After tightening the screw of the inflating bulb, the air is slowly pumped in. The figure at which the radial pulse disappears at the wrist is first noted. This procedure prevents the error of overlooking low pressures with such remarks as 'there is something wrong' or 'the instrument is not working'. Having thus obtained the systolic pressure figure roughly, the auscultatory estimation is made. The bag is deflated till the first definite tap

or click is heard. The figure at which this occurs gives the systolic pressure. Then while slowly deflating, the various phases described by Karotkoff are noted. The point of complete disappearance and the one at which the third phase described as a thud changes into a muffle, which is the fourth, are when recognizable noted. This latter figure is the diastolic pressure.

Too much noise has, in my opinion, been made of the nature of these phases and far too great stress is laid on the importance of recognizing them accurately. It is often exasperating to the practitioner when repeated attempts fail to reveal these stages. He is almost put off the study of blood pressure; he is left with the impression that his figures are unreliable and inaccurate. As stated by Korns[1] and endorsed by Marshall[2] 'It is the exception and not the rule to find the sound-sequences which display the supposedly characteristic phase differentiation. The determination of the diastolic pressure from the end of the fourth phase when it is present and prolonged certainly leads to a registration of a lower figure. But

[1] Korns' Physiology, XVI, 247.
[2] Marshall, B M J., March 12th, 1932.

this prolongation does not often occur. In my experience this occurs only in aortic regurgitation, arteriosclerosis, in children and in young adults; in the first condition it sometimes does not seem to disappear at all. Such cases, in spite of the frequency of arteriosclerosis, are rare. In nearly all cases the point of disappearance of all sounds occurs 5–10 mm. below the actual diastolic, so that if the latter number is added to the figure at the point of disappearance of sounds, a reliably accurate diastolic pressure is obtained. But whenever possible the greatest accuracy ought to be sought, these other manœuvres being resorted to only when the different phases cannot be easily defined by the ear. The determination of the figure accurately is more important as regards lower pressures than the higher ones. In the former, firstly, to ascertain whether early high pressure is present at all; secondly, to see how near we are to the figure which might be termed 'the symptoms and signs threshold in high pressure'. This is, in my experience, about a diastolic of 115, at the time symptoms are felt or signs are observed. When we reach higher figures, as for instance those over 135, we have entered the phases in which in men, any one of

the serious complications may occur, and an observation of the variation of 10–15 mm. does not help us so much as to allow us to prognosticate which is the one likely to occur. At this stage, the prognosis will have to be based on a general examination of the patient, which may reveal thickened arteries, cardiac enlargement, albuminuria, casts in the urine, and so forth. So long as we do not frighten our patients and we do not take so serious a view of the lower figures it is better for them that when we are in doubt a diastolic should be read slightly on the higher side then on the lower of the true figure.

Clinical Types.

It has been noted by most medical men that apoplexy which often results from high blood pressure occurs in the thick set, deep-chested man with a short neck. This observation has given origin to the term 'the apoplectic habitus' and to the belief that apoplexy is rare in other types of men. This notion is a mistaken one and the idea has led to a number of fallacies. As a matter of fact, our experience teaches us that the worst form of hypertension is commonest in a type of man neither so fat nor so thick-set as the one who is

'debited' with a tendency to cerebral hæmorrhage. Indeed the kind of case described as malignant hypertension occurs very frequently under 45, and in both types, but more commonly in the latter.

There are two other types of patients in whom hypertension occurs, and in these the disease takes unusual courses. One is the female, in whom either through the influence of menopausal changes or through endocrinous action high blood pressure, when it has no renal origin, often leads to almost a spontaneous cure. The other is the one in which there is generalized progressive thickening of the arteries with cardiac enlargement, the patient usually coming to us with his apex beat in the axillary line, and his vessels like lead pipes. I think this type approximates almost to Bishop's 'Cardio-arteriosclerosis'. A complete account of these types is given in my book on High Blood Pressure.

Etiology.

In a recent article on the etiology of high blood pressure, Waller[3] has given a very acceptable summary of

[3] Waller, 'Etiology of High Blood Pressure', B.M.J., Oct. 1930 p. 98.

the various factors implicated in its etiology. I have modified this to include nearly all the causes which have been thought to be responsible for the elevation of blood pressure in the human being. They are as shown below :—

The Etiology of High Blood Pressure.

(1) Intoxication. (Non-infectious)
- (*a*) Exogenous :
  - (i) Alcohol.
  - (ii) Lead.
  - (iii) Arsenic.
  - (iv) Diet—meat, general excess, condiment, constipation.
- (*b*) Endogenous :
  - (i) Pressor Bodies.

(2) Habits :
- (*a*) Sedentary occupations.
- (*b*) Insufficient physical exercise.

(3) Metabolic :
- (*a*) Endocrinous (menopausal).
- (*b*) Pregnancy.
- (*c*) Fatigue.

(4) Heredity and Diathesis.

(5) Infection. (Past and Present):
- (*a*) Generalized systemic.
- (*b*) Focal infection with dissemination of organisms, e.g. Syphilis.
- (*c*) Focal infection with dissemination of toxins.

(6) Obesity.

(7) Psychic.

(8) Arteriosclerosis.

(9) Supra-renal hyperplasia.

In practice, however, these numerous factors appear to be irregularly distributed so far as they are observed in patients. For instance, cases due to psychic causes do occur but that very rarely. Halls Dally reports an interesting case of an elderly man, once a hard liver and heavy drinker who became a total abstainer after a long and terrible struggle. He arrived at Paddington after a heavy day's work and feeling run down and tired out had two glasses of brandy and soda. On reaching home his young wife detecting the odour of the spirit remarked 'At your old drinking habits again, are you?' This upset him so much that his systolic blood pressure rose to 265 producing symptoms which necessitated the doctor being sent for. The following morning his pressure was normal. Under my care, I had a well-known lawyer, 49 years of age, who had congenital cystic kidneys which in this case were accidently discovered. He had seen me a month before his death when his diastolic pressure was 105 and his systolic 155. He was a very religious man, leading a quiet life with complete happiness in his family. One morning about 1 A.M. he dreamt that his wife was dead. He was so upset and excited that he ran into her room and awakened

her saying 'I dreamt that you were dead'. With these words he fell down unconscious with cerebral hæmorrhage of which he died the following morning. Elsewhere I have reported another instance when a man who hac been regularly robbing his employer, got a transient right-sided hemiplegia as soon as his defalcation was discovered. His systolic blood pressure a few hours after the stroke was 185, his diastolic 120. Two years later the diastolic pressure was 110 and his systolic 160. These are no doubt interesting but they form only a very small group. Factors like these though responsible for transient elevation of pressure can hardly account for the large number of cases in which the blood pressure is persistently raised. When we consider that the mechanism by which blood pressure is maintained is endowed with the power of altering it to suit not only the needs of different parts of the body but also the physical needs of the latter as a whole and the emotional state of the individual, variations of this kind must come under exaggeration of the factors normally at work. They cannot be said to have a real pathological basis as in those cases of high blood pressure with symptoms which are so common in

general practice. The former are like the tachycardia and fainting associated with emotional states, the latter like the tachycardia and fainting of myocardial disease.

The various possible causes of high blood pressure mentioned above do not include renal disease, which is generally accepted as accounting for a large number of cases of hypertension. It is the non-renal which one finds difficulty in attributing to a single cause. The author is of the opinion that so many factors are not usually responsible for the elevation of pressure. The intoxications are challenged without justification, alcohol is unduly suspected when many circumstances point to sedentary habits without physical exercise and overfeeding as being contributory to the production of high blood pressure. Syphilis as Sir Humphrey Rolleston has pointed out only causes elevation of pressure when its protean effects produce such changes as those in the kidneys. Arteriosclerosis is so often observed without high blood pressure as a concomitant feature that it cannot be looked upon as a cause. Heredity and diathesis appear to be contributory in 20% of cases. The stress and strain of life, such as prevails in 'a stock exchange atmosphere' are constantly brought into the etiology

more out of habit and tradition than from any justification for their inclusion based on a truly scientific study of these as causative factors. Supra-renal and other rare conditions have been mentioned among the causes but it is the privilege of a very few indeed to meet such cases even in the careful scrutiny of hospital practice. In the main it may be said that not a few are born with a tendency to develop high tension; with it or even in the absence of it, when renal disease is not the primary cause, sedentary habits, overfeeding, endocrine dysfunction and careless habits as regards diet and exercise contribute singly or in combination to give rise to essential hypertension.

There is no single special symptom which may be said to be pathognomonic of high blood pressure. Very often it is only discovered in the course of routine examination, a fact on which so many fantastic views are based as to the harmless nature of elevated pressure. But in a large number of cases serious sequelæ are noted and reference will be made here to some of those. We will leave out of consideration those very rare cases of hæmatemesis, hæmarthrosis, hæmaturia, to mention but a few, which are attributed to high blood pressure. The common sequelæ come under three

main headings: (1) The cerebral, (2) the cardiac, and (3) the renal. The cerebral manifestations are varied. There may be the symptoms of 'neurasthenia'. Headache and epistaxis, common symptoms in the colder countries, are very uncommonly complained of in the tropics. More frequently strokes of many different kinds occur, the motor attracting more attention than the sensory because of the associated paralysis or pareses. Their distribution may be monoplegic, hemiplegic or localized, as for instance the tongue only, and the duration of these may vary from a few seconds or hours to days or permanency. The first and last evidence may be a fatal hæmorrhage. The sensory manifestations of similar distribution as the motor may be tingling and numbness or paræsthesia. A full reference to these have been made in my book on 'HIGH BLOOD PRESSURE AND ITS COMMON SEQUELÆ'.* The cardiac manifestations are generally those due to enlargement and fatigue of the heart. In uncomplicated hypertension the heart if healthy goes on fighting this arterial pressure hypertrophying more each day and with passage of time giving way, showing dilatation. This process

* Bailliere Tindall & Cox, London.

finally ends in failure with congestion with a much enlarged heart. Any one of the ordinary symptoms of myocardial disease may therefore be met with. When associated with hypertension there are other factors which interfere with the functional efficiency of the heart such as fatty infiltration, arteriosclerosis (particularly coronary) or syphilis, and present the phenomena of myocardial involvement and breakdown when the pressure figures are not yet too high. Not an uncommon result of persistent hypertension is acute œdema of the lungs. The renal changes generally come on late in the course of high blood pressure for from diffuse hyper-plastic sclerosis renal efficiency is slowly impaired. But it must be remembered that the elevated pressure may be secondary to renal disease. The examination of the urine, the blood urea and the renal function tests will help in the elucidation of the cause and in the determination of the course of the disease. Albuminuria, retinal hæmorrhages, sudden blindness, and anæmia occur, much less frequently in essential hypertension than in high blood pressure associated with renal disease.

The prognosis of high blood pressure must be based on facts ascertained from an examination of the urine. The pre-

sence of renal impairment, or much albumen in urine always means a bad prognosis. When renal in origin those which last longest are cases of so-called granular kidney. The presence of retinal hæmorrhages or exudates or any of the other complications makes the prognosis infinitely worse. As regards essential hypertension it may be said that the author himself is in complete disagreement with those who take too light a view as regards prognosis, particularly when the diastolic is over 130. Generally speaking the symptoms present should be the guide. Transient pareses, early renal involvement, breathlessness or any other symptom referable to the heart generally mean trouble not far distant, particularly if no accommodation is made to the abnormal pressure. Marked cardiac enlargement usually renders cerebral hæmorrhage improbable, but makes the general outlook a matter of a few years. In women the prognosis is always better and cases which present serious symptoms live comfortably for years. Each case should be judged on its family history, the strain to which the patient is submitted by his routine of life, the presence of symptoms and obvious signs, and finally on the recurrence or persistence of one or more of these latter.

Treatment. High blood pressure needs both management of the patient as well as treatment. Rush methods which cause alarm should be avoided at all costs. The first step is to assure the patient that he need not worry about his state and that the condition is comparable to a mild grade of obesity which, however, should receive a little attention. Unless the pressures are very high and symptoms or signs are serious the patient need not be rested. If the latter are present a few weeks of rest in bed will invariably reduce the pressure. If no such trouble is evident the patient must be asked to lead a quieter life but as pleasant as he could make it—the idea being to cut off excess of work and worry with a view to providing adequate mental and physical rest. At the start a fuss should not be made about food but later reduction of the protein content and the daily quantity of food taken is desirable. Drugs are generally not of much value. The best mixture is an ordinary mild diaphoretic one with a little bromide. The numerous vaso-dilators may be used in small quantities. Big doses do not appear to do much good; they often upset the patient by producing peculiar sensations he has not previously experienced. There are numerous patent

medicines on the market, but none are reliable. Acetyl choline has been given intravenously but no uniform results are obtained and on this ground cannot be recommended. The sulphocyanates are often useful, 1½ grs. three times a day the first week, twice a day the second week, and once a day the third week. Diathermy is useful in all cases even when it does not reduce the pressure; it is particularly so in renal cases. A method the author finds of great value in non-renal hypertension is mobilization of the skeletal muscles. This may be effected on a Bergonnie chair or by some other contrivance, the aim being to produce contraction of the muscles of the trunk, upper and lower limbs, ten minutes for each part, daily. The pressure often falls rapidly. This treatment may be given to patients who are well enough to go about whilst they attend work. The size of the heart often diminishes under treatment and the symptoms disappear. Whenever possible the patient should be advised to take a restful holiday in a place where there is sufficient amusement to prevent boredom and not enough to encourage undue excitement or over-exhaustion. Renal cases require close observation and treatment directed from the point of view of disturbance of kidney function.

CHAPTER VI.

SOME SIGNS AND SYMPTOMS.

Here it is desired to make reference in a very general way to some signs and symptoms met with in cardio-vascular diseases which are often modified by tropical conditions. I have made some reference to this in connection with high blood pressure where it has been noted that headache and epistaxis commonly associated with it in cold countries are rare in the tropics. Whether the condition of the peripheral circulation explains this has not been established. In all probability it does. It may also be said that symptoms referred to by the patient, such as palpitation and fluttering of the heart, are not so frequently met with owing probably to the infrequency of rheumatic auricular fibrillation. The main signs and symptoms evident to the patient in the majority of cases are pain or substernal discomfort, breathlessness, and œdema of the legs. The nature and distribution of pain are the same as one sees elsewhere but a few words may be said by way of caution as regards œdema of the legs and breath-

lessness. Whilst their presence should always arouse the suspicion of diseases of the heart a few facts in connection with these should be borne in mind before definite disease is diagnosed.

1. ŒDEMA OF THE LEGS (ANKLES).

Although the average book in use by the practitioner or the student makes reference to this sign as evidence of cardiac failure, little emphasis is made on the possibility of its occurrence compatible with a well-functioning myocardium. For instance, it is stated that in heart disease it first becomes apparent in the feet and ankles and when the patient is walking about. "Bolton's[1] recent work has shown that the œdema fluid primarily due to lack of nutrition of the capillary walls owing to stagnation of the blood is actually originated in the neighbourhood of the heart . . . . . It is purely due to gravity that it first becomes visible round the ankles." It usually commences in the most dependent part and generally the patient first notices some puffiness round the ankles in the evening . . . . . Even in the severe and prolonged case œdema is often confined to the lower extremities.

[1] Taylor's Medicine revised by Poulton, 14th Ed., 1930, p. 333.

In the tropics apart from the often noticeable forms of lymphatic œdema swelling of the ankles is common in both men and women of middle age who lead quiet easy lives from the point of view of physical exertion. This is not peculiar to the tropics for as Lewis states, 'Most important from the diagnostic standpoint is the fact that hydrostatic dropsy is a very common event in middle-aged and elderly people who are heavy, who stand or sit much but who otherwise are healthy'.[2] But its occurrence is more frequent and certainly more evident in the tropics possibly on account of some conditions which have a connection with the peripheral circulation and are not explained by Bolton's hypothesis. In support we may quote Lewis again, 'It is important to grasp that a certain grade of œdema is physiological'[3]. . . . . Another factor proved to influence the rate at which dropsy forms in man is temperature; the higher the temperature of the part the faster the exudate occurs; a clinical instance is that in which dropsy of the leg forms quickly in patients while they sit before the fire. If science has no positive proof of factors

[2] Lewis, Sir Thomas, Diseases of the Heart, 1933, p. 20.
[3] Ibid., p. 18.

contributing to a physiological œdema we may look for conviction to the reclining chair or the armchair found in almost every tropical bungalow with long arms for resting the legs on while one is reclining in the afternoons and evenings, or to the habit of the local middle-aged business man who employs his servant every night to massage his legs for the comfort he gets thereby. Other extra-cardiac condition which probably contribute to the causation of œdema in the tropics are the numerous widely prevalent causes of secondary anæmia. Œdema of the ankles should therefore not be looked upon when it is present alone, as a sign of myocardial weakness. It is a part of the story of congestive failure which begins only when a large proportion of one's cardiac reserve has been drawn upon, and for this reason other signs of failure are generally present.

2. BREATHLESSNESS.

Breathlessness here referred to is the mild form. Or if we may express it differently the breathlessness the patient complains of or that which is not very evident. Obvious breathlessness when it does not point to myocardial disease should point to some other serious condition. But the former while it may be

evidence of early cardiac affection may only be part of a syndrome which has been described as the syndrome of effort, namely, breathlessness, consciousness of the heart's beat, or giddiness, tremulousness or feeling of exhaustion, forcible and diffuse cardiac impulse, and sometimes soft basal and apical systolic murmurs. This syndrome which came into prominence during the training of recruits of the late war comprises symptoms and signs which may be produced by severe exercise in the healthy but which appear in the apparently healthy when they are subjected to milder grades of physical effort. The underlying cause in the latter is traced generally to sedentary occupations, latent infections or to sequelæ of acute infectious fevers. In the tropics these latter conditions are very widely prevalent and recurrent febrile affections of parasitic origin (and non-febrile ones) are common. Sedentary habits and a slower life are almost the rule in the majority of people. The stimulus of whipping up the sluggish circulation by an annual holiday is the exception in the routine of life. Under these circumstances breathlessness on exertion is met with as a common complaint. Before a young man is condemned for heartstrain or an old man

is suspected of having myocardial disease the true nature and the degree of breathlessness should be carefully gone into and diagnosis and treatment should be based on a thorough enquiry into the actual state of affairs.

CHAPTER VII.

SOME THERAPEUTIC CONSIDERATIONS.

Section 1.

THE DIGITALIS GROUP.

There are possibly no drugs apart from quinine which are so extensively used in the tropics as those of the digitalis group. Recently digitalis as the tincture, and now digifortis, creep into practically every prescription for any ailment associated with rapid heart action, or the slightest evidence of cardiac irregularity. The reason for this can possibly be due to such statements as Douthwaites[1] 'the weakly acting heart that is met with during pericarditis, pneumonia, typhoid fever . . . . . is often greatly benefitted by digitalis'. Or by the encouragement given by such assertions as Hyman's 'Digitalis affects some lowering of abnormal temperature'.

The tropics at any rate seems to pay no heed to Lewis' statement that[2] 'the use of digitalis for any febrile affection is not usually to be recom-

[1] Hale-White, Materia Medica revised by A. H. Douthwaite, 1932, p. 406

[2] Lewis, Sir Thomas, Diseases of the Heart, 1933, p. 201.

monded '. Nor to Cushney's[3] view that these remedies should be used with special care in high fever.

This indulgence in the drug has often made me wonder if in tropical conditions digitalis has some other effect than that observed in cases like auricular fibrillation or whether in fevers it effects a beneficial influence apart from its action on the heart. When I started practice in the tropics as an ardent student of the English School I used religiously to restrict the use of tincture of digitalis for conditions like auricular fibrillation but gradually I became a convert to the idea that the extensive use of the drug may be based on experience which could not be altogether overlooked. I think however that there is no justification for administering it on the scale in which it is being used at the present time. Fortunately however the drug is tolerated very well indeed and seldom causes ill effects in moderate doses. For those patients of mine with auricular fibrillation who are unable to afford to pay the chemists repeatedly for 'bottles of mixture' I prescribed the tincture to be taken in a wine-glass of coriander (in-

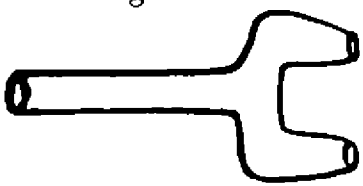
[3] Cushny—Digitalis and its allies, p. 91, Longmans Green & Co., Hyman—Pharmaceutical Therapeutics, p. 217.

fusion) water. This makes a most palatable mixture. The tincture of digitalis is bought few ounces at a time in a drop bottle and the dose is measured out at the necessary intervals. This procedure induces the patient to continue with his medicine and the deterioration of the latter is at the same time prevented.

Section 2.

OXYGEN AND POTASSIUM IODIDE.

Oxygen is not used nearly as often as it should be. Lack of means on one hand, and the difficulty of obtaining it on the other often from a source many miles from a patient's home probably account for this. To the expense incurred in purchasing is often added that due to rapid evaporation owing to the high temperature of the tropics. For this reason I often get it administered after passing it through a Woolf's bottle containing water and eau-de-cologne. (If pure spirits is used the contents of the bottle get too cold.) The tube carrying the oxygen from the Woolf's bottle carries a glass terminal shaped as shewn in the diagram below :--



It may be administered with a nasal catheter in the manner described by Bourne but Haldane's method is most unsuitable for the tropics. Both these methods are resented by the patients perhaps owing to the irritation of the very warm gas.

Potassium Iodide is a drug very commonly used for a number of conditions. It is prescribed freely in arteriosclerosis, coronary artery disease, high blood pressure and syphilistic cardio-vascular conditions. There is no drug which plays a bigger part in damaging the reputation of the practitioner than this. In the tropics it is very badly tolerated by the native population. Attention is not generally paid to this fact in the belief that increasing the dose gives the necessary relief. But only too often the patient does not give you the chance to revert to this for he promptly seeks the advice of another doctor to whom he says 'the medicine prescribed for me makes my headache, my eyes and ears burn'. Although increasing the dose of iodides gives relief in many cases it does not do so in so reliable a manner as to warrant our dependence for relief from this only. When Dixon states that[4] 'it is

[4] Dixon, W., A Manual of Pharmacology, 7th Ed., 1929, p. 359

commonly stated that on increasing the dose of iodide symptoms of iodism disappear and such is undoubtedly *sometimes* the cases'[4] it becomes obvious that one cannot always depend on an increased dose to give the necessary relief in a case of unexpected sensitiveness to the drug. This sensitiveness is more evident in the tropics and a practitioner who takes no notice of this peculiarity loses more patients than he is aware of. The safest course is to warn the patient of the possibility of iodism or use colloidal iodine as an alternative. I have had very good results with the latter and in one instance very conclusive proof of the complete disappearance of a large mediastinal tumour, gumma, in a man presenting the signs and symptoms of an aortic aneurysm. Cushny suggests the use of iodipin and sagodin as alternatives.[5]

Section 3.

THE HILLS.

'Sending a patient to the hills' for a few weeks is a commendable therapeutic measure in the tropics. For this reason it is often resorted to. The

[4] Dixon, W., A Manual of Pharmacology, 7th Ed., 1929, p. 359.

[5] Cushny, A. R., Pharmacology and Therapeutics of the action of Drugs, 9th edition, p. 567.

bracing climate of the higher elevation no doubt acts as a tonic, the victim of insomnia begins to sleep, the system gets refilled with energy and in many illnesses during the convalescent stage much benefit is obtained. But in cardio-vascular conditions both the altitude and the place itself should be carefully chosen. It would not do, for instance, to send a case of high blood pressure to a very high altitude, nor would such a place suit a patient whose condition would be associated with any degree of anoxæmia. Breathlessness easily elicited is generally a sign which contraindicates a hilly district or a high altitude. When such a symptom is not evident the patient will make an attempt to improve his physical condition by 'going for regular walks' forgetting or deliberately attempting to fight the strain of climbing hills and the effects of the rare atmosphere. I have seen many a catastrophe follow failure to recognize these facts. A patient of mine with severe myocardial degeneration went up to an altitude of 5,000 feet in a few hours contrary to advice and developed acute œdema of the lungs the following day. Another developed such severe breathlessness on the slightest exertion on the day after his arrival at a hill station that he had

to come down the next day. A third with high blood pressure died of a fatal hæmorrhage on returning from a long walk. These are a few instances. Generally speaking it may be said that for cases with low blood pressure where there is no definite evidence of a severe myocardial degeneration, a high altitude, even of 6,000 feet, may be beneficial. But where signs and symptoms of cardio-vascular diseases are present no patient should ever be removed to a higher altitude than 4,000 feet. In high blood pressure of renal origin a cold climate would be most injurious. There are a few people who with high blood pressure or even with renal disease will spend a holiday in the hills and come back feeling better, but such exceptions should be no encouragement to deviation from the advice which should be given in the average case by the doctor who desires to safeguard both his patient's interests and his reputation. The following case under the care of Dr. P. B. Fernando, M.B., M.R.C.P. (Lond.) is illustrative: 30-9-33. M.R.C. male 39, married.

History.—Has not been ill; rejected by Insurance Co., as high blood pressure was found.

Condition on examination—

H.A.B.

3rd. rib.

rt. st. line | ½" ext. to N.L.;

Pulse 72; B.P. 200·140; Lungs normal; Abdomen soft.

Spleen + + (malarial): Liver normal.

Urine: S.G. 1005; Albumen + ; Sugar nil.

Mict.

N 2 twice

D 4-5 times

Fundi—No arterial changes seen.

Treatment—Lacto-vegetarian diet with weekly fasts. Elixir.

Na. Sulpho. cyanate (P.D. & Co.) as directed.

Mixture—Pot. iodide, pot. bromide, sp. am. amon. mag. sulph. aq. ad.

He insisted on going to a hilly district, altitude 5,000 ft., for business reasons contrary to advice and died suddenly from cerebral hæmorrhage within two months.

It is not possible in a few pages to go into the details concerning the therapeutics and management of a case of heart disease in the tropics. Clothing, the utilization of fresh air and sunlight, as for instance in the treatment of subacute infective endocarditis, the selection of a dry place for the recurrent cases of

acute rheumatism, the regime as regards diet, so peculiar in its variety, and a number of other factors which determine satisfactory treatment have to be considered in every case. But in a work of this kind in its present stage, it is not possible to go into these factors in great detail, if only for the reason that hard and fast rules cannot be laid down in connection with them. We have yet to study a host of variations which are necessary from the point of view of a patient being in the tropics, temporarily or permanently. These will probably be different with each individual and the adjustment of these cannot be done better by anyone than by the general practitioner to whom I hope my observations will be helpful.

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