# St. Cyres Lecture

ON

# HEART AND CIRCULATION IN A TROPICAL AVITAMINOSIS (BERI-BERI).

*Delivered at the National Hospital for Diseases of the Heart, London,*

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FORTY years ago, as a young doctor in my own country, I was intimately acquainted with the two Dutch scientists, Pekelharing and Winkler, who, as a government committee, had studied beri-beri in the Dutch Indies. In those days nothing was known of its ætiology; my authorities believed it to be an infectious disease. Eykman and Grijns had not yet discovered its causation, but many of its clinical features had been described, and it was generally known that, to quote a British poet—

> "Beri-beri is very, very
> Hard on the nerves——,"

and, moreover, very "hard on the heart" also. I was greatly impressed by the astonishing facts that patients seem to die of complete failure of the right heart only; that the opinion (accepted as a dogma) was that this heart failure must be due to a degeneration of the vagus nerve; that patients may die 24 hours after having been in seemingly good health; that there was an "idiopathic" hypertrophy of the whole heart with evident lack of contractile strength as the chief characteristic; and that vitamin B had a miraculous effect on a heart which refused to react to any cardiac drugs—all these facts suggested to me the existence of some quite peculiar morbid process. Some seven years ago I explained this important problem to a young friend, Dr. Aalsmeer, a lecturer on internal medicine at the medical school for native doctors at Surabaya (Java). He devoted a good deal of time and interest to the question and last autumn returned from the Dutch Indies with complete clinical records of the cardiac aspect of the disease. These data, illustrated by X ray photographs and electrocardiograms, provide us with a veritable "natural history" of the circulatory apparatus in beri-beri cases.

## CAUSE, SYMPTOMS, AND TREATMENT OF BERI-BERI IN GENERAL.

The generally acknowledged ætiology lies in the absence of an accessory food factor (vitamin B), contained in the bran of rice and cereals. The disease makes its appearance where polished rice is the exclusive diet. Proof of this pathogenesis is furnished by the results of the sole treatment—viz., addition to the diet of vitamin-containing foodstuff, such as rice-bran, bran extracts, or a certain bean, known in Japan for over ten centuries (*Phascolus radiatus*. Malayan: katjang idjoe). Even in very severe cases it relieves the serious symptoms. The principal pathological changes are:—

(*a*) Degenerative processes in the peripheral nervous system, damaging and even destroying the peripheral motor and sensory, as well as the sympathetic and parasympathetic elements. The symptoms are those of extensive polyneuritis.

(*b*) Anasarca and serous effusion in the large cavities. In chronic cases there may be a condition of extreme general atrophy and dryness of skin and tissues, the so-called "dry form."

(*c*) Heart disease, characterised by general feebleness of the circulation, palpitations on the slightest exertion, swelling of the liver and œdema, with general swelling of the limbs. This heart failure is very often the cause of death.

## CLINICAL ASPECTS OF HEART AND CIRCULATION.

I rely on the work of Aalsmeer, correlating it with the earlier work of Vedder, Castellani, Chambers, and Manson, in English; of Scheube, Nocht, and Mense in German; of the Dutch authors Pekelharing and Winkler, Kieviet de Jonge, de Langen; and the many Japanese contributions, culminating in the important work of Shimazono, whose conceptions are in many respects identical with those of my collaborator.

The following points may now be taken as well established:—

1. From the beginning of the disease the whole heart, right and left, is enlarged; X rays show not only an enlargement to the right, but also bulging of the left auricle in the so-called "taille" (left upper border) of the heart shadow. The left ventricle, too, is clearly larger than normal. It is impossible to decide whether, in this first period, the enlargement is due to dilatation alone or to hypertrophy of the muscle wall also.

2. This enlargement and corresponding failure of the heart increases more or less rapidly, sometimes in a few hours, *but chiefly involves the right heart.* The prevalence of right heart failure and right heart widening and hypertrophy is mentioned by everyone who has studied the disease at the bedside and post mortem. A remarkable clinical manifestation is the disappearance of the apex-beat and the substitution for it of a curious wriggling, fluttering motion of the heart muscle, which the patient himself feels and which is easily seen on inspection and felt by palpating with the whole palm of the hand.

3. Increasing stasis takes place in the liver and venæ cavæ, and in acute cases this causes an enormous, painful swelling and bulging of the liver, ascites, and sometimes sudden death.

4. Even in the most serious cases there is never any irregularity of the heart, no extrasystoles, flutter, or fibrillation. The electrocardiogram never shows any disturbance of stimulus conduction. The ventricular complex is perfectly normal, and the size of the different waves rather exaggerated, the only peculiarity being a certain degree of preponderance of the right ventricle.

5. The pulmonary circulation is completely free from any sign of stasis, œdema of the lungs only showing itself in the last moments before death. There is no other disease of the lungs, no true emphysema, no arterio-sclerosis or spasm of the pulmonary arteries, which might cause right heart failure.

6. Apart from variations in the course, dependent on the relative intensity of neuritic manifestations on the one hand and of cardiac disease on the other, two forms of beri-beri are generally recognised—namely, a chronic and an acute, so-called pernicious and usually fatal form. The latter is now much less often seen than it used to be, since specific treatment has almost abolished it.

7. The whole series of heart remedies and diuretics (digitalis, strophanthin, caffeine, theobromine, thyroid, novasurol, salyrgan, &c.) is of no real use for the beri-beri heart. Vitamin B alone is an immediate and usually complete success. At autopsy the heart shows enlargement of its cavities and apparent hypertrophy of its walls, both much more pronounced in the right side of the heart. Microscopically there are no striking changes in the heart muscle-fibres and no inflammation.

## DILATATION AND FAILURE OF THE RIGHT HEART.

The first unsolved problem of the beri-beri heart is the extreme widening and failure of the right heart in contrast with the relative smallness of the left ventricle and the absence of pulmonary stasis. This seems the more enigmatic, as there is proof that both sides of the heart suffer at the beginning of the disease. The soft and frequent pulse, however, the curious wriggling motion of the enlarging heart, and the progressive over-filling of the proximal parts of the venous system (v. cavæ and liver), are undeniable signs of the heart's incapacity to convey the whole quantity of affluent blood from the

venous into the arterial side of the vascular system. Clinical experience has taught me *that in such cases of increasing insufficiency of the whole heart the right heart is doomed beforehand to suffer much more than the left, and even to suffer to the profit of the latter.* If the strength of systole is decreasing in both ventricles equally, the moment will come at which both ventricles will fail to discharge their contents completely. A larger amount of residual blood than normal is retained in the heart. This larger post-systolic ventricular blood volume, together with a normal onflow of blood during the diastole, must necessarily result in a greater filling of the heart at the beginning of the next systole. This increased load will soon become too much for a heart already unable to discharge a normal quantity of blood. The output will become smaller and smaller, the filling of the arteries will be less, and the diastolic filling of the heart and the venous stasis before the entrance port of the heart must both increase. This being so, two factors will establish a marked difference between the right and left heart.

The first of these is the anatomical inequality of the two ventricles, the right being of lighter build, and provided with a thinner wall. The tricuspid valves are implanted on a much weaker ground than the mitral; there is no strong fibrous ring supporting them, they are condemned to share the distension of the right ventricle, and consequently are more liable to incompetence. Once this incompetence is established, blood passes from the right heart backwards into the veins and liver and the arterial output of the right ventricle is greatly reduced.

A still more important factor is at work. In the blood stream, moving "quasi in circulo," the right heart is located higher up the river than the left. As a consequence, the right heart has to deal under all circumstances with the whole volume of blood returning from the periphery to the heart, *the left heart not receiving more blood than the right heart is able to transmit.* Consequently the right heart is all the time struggling for life against an ever-increasing overfilling and widening, but the left heart is spared a similar burden. *The more the right heart suffers and fails, the safer the left heart becomes,* working under easy conditions and decreasingly threatened by such factors as might lead to overfilling and dilatation.

In such circulatory disturbances, despite a smaller output of the left heart, this smaller minute volume will reach the great veins and the right heart in due time. This holds good even in severe degrees of mitral stenosis, in which an extremely high arterial blood pressure may be maintained. Even after the final standstill of the heart in the first moments of death this function of the arterial wall (perhaps the last of life) is maintained, so that post mortem the arteries are found empty and the venous system filled. Therefore a smaller supply of blood to the aortic system cannot be expected to relieve the difficulties of the right heart in the same way as a smaller supply to the pulmonary arteries relieves the left heart.

These explanations make it possible to understand the cardiac and circulatory phenomena of beri-beri, the initial widening of the whole heart, the ever-increasing bulk of the right heart, the ever-decreasing cardiac systolic output, and the progressive engorgement of the proximal venous system, venæ cavæ, and liver. It is now easy to see how in acute attacks of beri-beri a sudden failure of the right ventricle with tricuspid incompetence puts the great veins and the liver under systolic ventricular pressure. These are distended to the highest degree and sudden death ends a condition of extreme suffering, the horror of which was so dramatically described by Manson in his handbook of tropical diseases. Throughout this unsuccessful struggle of the right heart the lung fields on the X ray plate are quite clear; there is no lung stasis, no râles, and no pulmonary œdema, and the left heart is not greatly enlarged but, on the contrary, is smaller and poorly filled. A small and rapid, but fairly palpable, pulse gives proof of the relative competence of the left ventricle, until at the last moment before death the latter loses its systolic power, succumbs, and leaves the lungs with stagnating blood and signs of ante-mortem œdema.

This simple mechanism, which explains the whole circulatory complex of beri-beri and the post-mortem findings, has great importance for general cardiology. The so-called safety-valve action of the tricuspid valve, hepatic stasis relieving the lung from threatening high tide, is familiar to a good number of us, but well-defined conditions of functional heart disease, involving both sides of the heart equally, are rather rare. They may be found in the course of acute infectious diseases (pneumonia, diphtheria, and others), but here so many different toxic and reflex influences complicate the picture that the separate influence of each is not easily discriminated. Another condition in which both sides of the heart are hampered in their action to exactly the same extent is rapid paroxysmal tachycardia and auricular flutter and fibrillation. In otherwise normal hearts it has been possible to show that here also there is marked stasis in the large veins and liver coincident with absence of lung stasis, râles, and dyspnœa. Since James Mackenzie discovered the change of circulation caused by the onset of what he called auricular paralysis, now known as fibrillation, in long-standing mitral stenosis, we have been acquainted with sudden, painful swelling of the liver; from personal experience this change is often accompanied by a striking improvement in respiration, and relief of the suffocating dyspnœa.

Once I recognised this mechanism, I was able to find it in a great many cases of slowly progressive, general myocardial weakness. Careful treatment, with well-apportioned doses of digitalis and diuretics, may cause lung œdema and swelling of the liver to disappear, even in extreme conditions, but only for a time. These intervals, during which equilibrium between the right and left heart is maintained with difficulty, become shorter and shorter, until at last treatment fails and the patient dies in a few hours. In many of these cases of recurrent heart failure the first and chief symptom is a rapid and very painful swelling of the liver. Even in rapidly fatal cases the same symptoms may be found, râles and lung œdema only showing themselves during the last hours of life.

In all these conditions the curious relationship between right and left heart failure may be recognised, but nowhere is it so uncomplicated, so regular, and so easily demonstrable as in the beri-beri heart. Thus there is a law of circulatory correlation between the two sides of the heart which may be formulated as follows: *In equal and increasing feebleness of the whole heart muscle the patient suffers most and dies from the failure of the right side of the heart only.*

Shimazono and Aalsmeer do not accept the conventional view that degeneration of the vagus fibres is the cause of the heart failure. Considerable dilatation of the heart may be present long before the first manifestations of polyneuritis. There is no parallelism between the degree of degeneration of nerve-fibres and that of the heart affection; indeed, it is the strong, apparently still healthy patient who seems to be most liable to the serious sudden acute attacks, in contrast with the patient whose first symptoms are those of palsy from polyneuritis. Aalsmeer is probably right in thinking that the relatively good nerve-fibres of the strong man enable him to undertake an amount of exercise which asks too much of his already weakened heart and induces the sudden attack of heart failure.

This vagal factor being unacceptable, we have to seek other clinical facts that would reveal the one potent unknown factor of this disease. The first hint is given by reasoning that, in our explanation of the development of myocardial failure in beri-beri, there seems to be no place for the hypertrophy of the heart muscle so often described. Why should the muscle that from the very beginning is showing all the signs of reduced contractile force and lessened output become hypertrophic? Such conceptions as

"hypertrophie passive par faiblesse du myocarde," or "the individual fibres becoming weaker . . . an attempt is therefore made to compensate by increasing the number of fibres—i.e., hypertrophy," do not elucidate the problem. The only way out of the difficulty seemed to be to look for another explanation of the post-mortem findings of increased weight of the whole heart and increased thickness of the muscle walls.

Aalsmeer found that, even in the worst conditions of failure, the beri-beri heart gives a perfectly normal electrocardiogram, without arrhythmia, extrasystoles, heterotopic rhythms or disturbance of conductivity in the bundle or in the conducting fibres of the ventricle. This fact reminded me that the old observations of MacWilliam record non-contracting heart muscle without disturbance of conductivity. Thirty years ago Engelmann found that the frog's heart muscle when immersed for a short time in water loses its contractility but retains its excitability and its power of stimulus conduction, showing that this phenomenon, discovered by Biedermann in voluntary muscle, holds good also for heart muscle. Later de Boer demonstrated that the swelling of the heart muscle, caused by the water, reduces mechanically the contractile power of the muscle-fibre, but at the same time the electrocardiogram may continue completely unchanged. Skramlik, Lusanna, Zondek, and Kraus demonstrated the same phenomenon for other forms of water retention in muscle-fibres, and even found that the size of the waves of the electrocardiogram may be enlarged and the rate of conduction increased. The presence in beri-beri also of this strange contrast between evident weakening of the heart muscle and the undisturbed or accelerated automatic excitatory processes suggests that here also *the nature of the affection might be water retention* ("Quellung") *with consequent swelling of the heart muscle-fibres*, and that this swelling, rather than a true hypertrophy, may be the cause of the increased weight and thickness of the heart muscle wall as found post mortem.

### The Water Retention Hypothesis.

Many arguments support this hypothesis. A few months ago Tiemann showed that striated, in contrast with smooth muscle-fibres, swell in water and in all hypertonic solutions. The gain in weight and the swelling, due to the quantity of water taken up, is less in heart muscle than in common voluntary muscle. If the same process is at work in beri-beri we should expect to find the skeletal muscles swollen at the same time as the heart. This swelling is, in fact, typical of the disease, especially at the beginning, long before general tissue œdema or signs of nerve degeneration appear. Swelling is most conspicuous in the muscles of the calf, which are very painful in walking and on being pinched. Pekelharing, Winkler, and Nocht compared the condition with the "pseudo-hypertrophic stage" in progressive muscular atrophy of childhood.

On this hypothesis we should expect that no treatment can succeed which does not free the heart muscle from its surplus water and drain the muscle-fibre. When vitamin B is given, the heart rapidly diminishes in size and recovers its power of contraction, and the skeletal muscles share in this process. The hard, thickened, and painful muscles of the calf melt away, as snow in the sun, to muscles of normal size and function. De Langen, in his new monograph on beri-beri, mentions this rapid disappearance of the swelling and equally rapid cure of the motor paralysis, less than 24 hours after taking vitamin B-containing food. This remarkable improvement is accompanied by a marked increase in the secretion of urine. Scheube states that katjang idjoe had the reputation in Japan a thousand years ago of being a good diuretic. Of recent years the diuretic action of rice-bran and katjang idjoe has been specially investigated by Japanese scientists.[1] From the extensive work on the regulation of water exchange by Magnus and by the Vienna Pharmacological Institute (E. P. Pick, Molitor Baer) we learn that striated muscle may take up and, under certain circumstances, can retain large quantities of water injected into the blood or taken by the mouth. Our muscular system may be regarded as one of the biggest water reservoirs, and its contents do not appear on the balance of intake and output.

Recent research work by Tsunoda and Kura[2] gives important information concerning the anatomy and treatment of beri-beri. In pigeons exclusively fed on polished rice the sensory nerves and their endings in the skin show curious primary regressive metamorphosis accompanied by swelling and other changes in their microscopical appearance. After the injection of small doses of vitamin B all these changes and their clinical consequences disappear rapidly. The endings of the sensory and motor nerves improve rapidly within five hours after the injection. Twenty-four hours later the histological appearance is practically normal.

The conception of water-retention in the heart muscle-fibre explains all the symptoms and peculiarities of the beri-beri heart, which thus constitutes a hitherto unrecognised and unique form of heart muscle disease. Before trying to bring this curious pathogenesis into relation with other well-known facts of physiology and general pathology, I must mention an equally unique phenomenon in the symptomatology. Not only is the electrocardiogram normal during the whole course of the disease, but the rate of conduction is increased and the atrio-ventricular interval is shortened. Moreover, conduction quickens as the heart failure progresses. In the most serious and acute cases the shortest atrio-ventricular intervals were found when the patient seemed to be in imminent danger of death. With recovery the conduction slowed down to normal, even with slowing of the rate of the heart beat. The fact that the same phenomenon was found experimentally in water-swelling of the heart muscle is a strong argument in favour of the identity of this condition with beri-beri heart.

### Theoretical Considerations.

It seems very probable that water-retention may be the primary cause of other manifestations of beri-beri and, in the first place, of the important degenerative processes in the whole peripheral nervous system. We have already seen that the swelling of nerve-endings in chicken beri-beri disappears completely a few hours after vitamin treatment. The microscopical anatomy of nerve-fibres and ganglion cells in beri-beri accords well with this conception, and it should not be forgotten that although motor and sensory paralysis may be found, as in polyneuritis, the primary affection of the nerve element in beri-beri is not inflammation. It is the general opinion that the anasarca is not so much secondary to heart failure, as essentially an imbibition process of the tissues. Other organs may show temporary swelling at the same time. Some of the glands seem to possess a special predisposition for this kind of water-retention in beri-beri, just as striated muscle is the first tissue to swell while smooth muscle escapes. A familiar pathological finding in this disease is swelling of the mucous lining of the gall-bladder. Kieviet de Jonge mentions a curious broadness of the face, recalling parotitis, and he found that there is enlargement of both parotid glands, the nature of which, he says, is "not œdema." Stepp thinks it probable that some of the glands, especially those secreting saliva, ceased to function.

It would be astonishing if beri-beri were the only disease in which water-retention directly damaged the heart. When an ailing heart does not react to heart drugs we generally suspect a desperate anatomical and functional condition of the myocardium; it seems possible that the cause found in beri-beri plays a part in the development of such events and might be removed by analogous forms of treatment. The first disease to remember in this connexion

[1] Japan Jour. of the Med. Sciences, 1927, viii.

[2] Virchow's Archiv, 1928, p. 267.

is myxœdema, where there is water-retention as a consequence of insufficient thyroid secretion ("Quellung"), and where water is expelled ("Entquellung") by thyroid treatment. A case of atypical myxœdema may be shortly related here:—

Some 20 years ago a small Frisian community sent a poor man to the medical clinic in Groningen because he was more or less stupid and so easily tired that he was incapable of any bodily work. His face showed a certain degree of imbecility or apathy, and his muscular function was greatly diminished; but his build was Herculean. There was no trace of œdema, the face was not swollen, and the heart seemed normal. The muscles were not only big, but very hard and of abnormal consistence; I thought this might be myxœdema of uncommon localisation. A few thyroid tablets had a most astonishing effect; the firm voluminous muscles that were very painful on pinching became thinner, softer, and fit for use again. On palpation the peculiar sensation of rolling muscle bundles returned, leaving only a few hard nodes, which disappeared in a day or two. In a week's time the normal intellect of the man had returned and he was sent back to his village and advised to take small doses of thyroid indefinitely. To the great satisfaction of his community he proved completely cured and able to do regular work, but with less Herculean muscles.

The absence of heart and nerve lesions, on the one hand, and the inactivity of thyroid in beri-beri on the other, prove that this case was not beri-beri, and the two conditions of different nature.

Another important difference was noted by Aalsmeer. In both diseases there is a greatly increased skin-resistance in the patient to the electrocardiogram; more in beri-beri than in myxœdema. But the deflections of the string are very small in myxœdema and at the least normal, or sometimes much larger than normal, in beri-beri.

These clinical facts point to the quite peculiar and specific nature of the swelling of the striated and heart muscle, which is certainly not a condition of œdema.

### Heart Hormones and Automatins.

Our subject forms a bridge between scientific research at the bedside and modern experimental work on heart "hormones" (Haberlandt) and "automatins" (Zwaardemaker). Rigler[3] found that vitamin B, isolated as a pure chemical substance by Jansen and Donath, belongs to this group of substances. He believed it to be identical with histamin. The fact that the organism itself produces substances which are able to bring an experimentally stopped heart and its automatic functions back to life has already induced experimental workers to recommend these substances for the treatment of heart patients. The beri-beri heart shows that at least in some rare conditions the revival of a seemingly lost heart is possible. There are doubtless a great number of different conditions causing standstill of the heart, and doubtless we shall hear, in the course of time, of a great number of different substances which may help. The beri-beri heart shows that not every heart that stops its systolic action has lost its regular automatic vital function. The automatism may go on undisturbed, but is prevented from manifesting itself by lack of conductivity or, as in beri-beri, by interference with contractility. In such cases the active substance which restores the regular function should not be called an automatin; what happens in the heart is not revival from death, but release from bondage.

### Summary.

My subject shows eloquently that careful study of the sick and their symptoms, and free experimental research are equally indispensable to the advancement of medicine.

It teaches us the existence of a law of circulatory correlation between the left and the right heart, working along simple mechanical lines of considerable clinical importance.

It leads us towards the better understanding of a disease, of which the successful treatment had already been found by experience at the bedside.

It brings us into close contact with the most modern biological problems of water exchange, of hormones, of sudden death, and revival of vital functions.

It seems permissible to formulate the hypothesis that the fundamental process in beri-beri is water-retention and the consequent swelling of certain vital tissues, especially nerve and striated muscle.

[3] Med. Klinik, 1928.

## THE LATER RESULTS OF PARTIAL GASTRECTOMY.*

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Few subjects have received more attention or have been the cause of intenser controversy during recent years than that of the nature and treatment of gastric and duodenal ulceration. From the surgical point of view the discussions have, for the most part, ranged around the relative merits of partial gastrectomy and gastro-enterostomy. It is not proposed in this article to consider partial gastrectomy as a treatment for new growths—for here the obligations of the surgeon are sufficiently clear—but to limit the review to cases of apparently non-malignant ulceration.

It will be well at the outset to summarise the standpoints taken by the several schools of thought at the present day. (1) The first school believe that operative treatment should never be adopted in the absence of acute complications such as perforation; they maintain that the symptoms can be adequately controlled by purely medical measures. For obvious reasons this group consists almost exclusively of physicians. (2) The second group comprises those who think that operation may be occasionally justified in very persistent cases, and especially in the presence of such complications as pyloric stenosis. Into this group fall many physicians and a few surgeons who have been unfavourably impressed by the recurrence of symptoms or the onset of jejunal ulceration after gastro-enterostomy. (3) The majority of English surgeons belong to the third group and favour gastro-enterostomy for chronic cases, after medical treatment has been given a trial, but would treat all acute cases medically. (4) Others advocate gastro-enterostomy, with or without pyloric exclusion, for practically all cases. (5) Yet another school consider that for the majority of cases of gastric ulceration partial gastrectomy is indicated, but perform gastro-enterostomy for duodenal disease. (6) The last school believe partial gastrectomy to be the operation of choice in all cases, and would always perform it for both gastric and duodenal ulceration if the technical difficulties are not insuperable.

With such diversity of opinion the only safe guide is a critical and, as far as possible, unbiased examination of the results. Since this article will deal only with the surgical aspect it is essential, as a prologue, to give reasons why the claims of the first school, that surgical treatment is unnecessary, cannot be entertained. Unfortunately, perhaps, for the surgeon's proper judgment, the great majority of the cases upon which he operates have already received a prolonged course of medical treatment, a circumstance likely to prejudice his opinion of these claims. The discovery of signs of old healed ulcers is offset by the presence of the fresh outbreak for which the operation is undertaken. The treatment therefore, even in such cases, has done little more than allow the original ulcer to heal without removing the cause. Post-mortem evidence of completely healed ulcers is, in my experience, much rarer than that found at operation for a fresh outbreak; it thus appears reasonable to conclude that medical treatment can rarely bring about permanent cure. Moreover, relief of symptoms is not necessarily accompanied by cure of the ulcer.

* Being a paper delivered before the South-West London Post-Graduate Association.