ABSTRACTS ON RELATION OF VITAMIN DEFICIENCIES TO HEART DISORDERS

"In rats a Vitamin B1-free ration produces a distinct bradycardia, which disappears on addition of the Vitamin (as yeast) to the ration. In underfeeding, bradycardia develops as a result of the inanition and is not relieved by administration of the vitamin."

Parade, Vitamin B Researches. I. Relation Between Deficiency of Vitamin B1 and Bradycardia, Z. Vitaminforsch, 6:327-334, 1937, as Abstracted by Chemical Abstracts, 32, 7:2577, April 10, 1938.

"CARDIOVASCULAR MANIFESTATIONS OF VITAMIN B1 DEFICIENCY: The Cardiovascular manifestations of beriberi most commonly encountered are dyspnea and palpitation on exertion, tachycardia and edema. The heart is generally enlarged both to the right and to the left. Systolic murmurs are common. Basilar pulmonary rales are frequent. The arterial blood pressure is usually normal or low, frequently with an increased pulse pressure. On palpation a bounding quality is noted in the larger arteries and 'pistol shot' sounds may be heard on auscultation. The venous pressure is generally increased but may be normal. The skin is usually warm and of normal color. Cyanosis is rare; edema may be mild and only in dependent parts, or diffuse and extreme. Electrocardiograms generally show alterations, chiefly in the T waves. Circulatory failure may be predominantly right sided or left sided. Sudden circulatory collapse (shock) has been observed, as well as syncope due to hypersensitivity of the carotid sinus.

"It is thus apparent that the cardiovascular manifestations of Vitamin B1 deficiency do not, at least in the present state of our knowledge, comprise a rigid and easily recognized clinical syndrome. Furthermore, not infrequently rheumatic, arteriosclerotic or syphilitic heart disease may have superimposed injury due to Vitamin B1 deficiency.

"However, there are technical measurements of the circulation which may prove to be of significant value in the differentiation of beriberi from other types of cardiovascular disorders. The circulatory minute volume and circulation time are both increased in the cardiovascular complications of beriberi whereas other causes of congestive failure, except hyperthyroidism, result in conspicuous slowing of the circulation. The important points in establishing the diagnosis of cardiovascular disease dependent on deficiency of Vitamin B1 are, in addition to those points already presented, the presence of other manifestations of Vitamin B1 deficiency, such as polyneuritis, or of deficiencies of other portions of the B complex, such as glossitis and pellagrous skin changes. Indeed it is rare to observe 'beriberi heart' without at least minimal signs of polyneuritis; the history of dietary inadequacy or of conditioning factors which lead to Vitamin B1 deficiency in spite of an apparently normal diet; the disappearance of signs and symptoms following adequate B1 therapy."

Strauss, Therapeutic Use of Vitamin B1 in Polyneuritis and Cardiovascular Conditions: Clinical Indications, Journal of the American Medical Association, 110, 13:953-956, (955), March 26, 1938.

"Nutritional disorders may affect the heart along with other tissues, sometimes seriously. Beriberi, a disease which is primarily the consequence of Vitamin B deficiency (tropical avitaminosis), has been shown to cause hydropic degeneration (intracellular edema) of the myocardium, particularly of the right ventricle, with cardiac dilation and failure. Relief is obtained neither by digitalis nor by diuretics but by the administration of antineuritic Vitamin B."

White, Heart Disease, Page 437, MacMillan Co., 1936.

"Evans found that Vitamin C increased the urinary output in each of 8 cases of cardiac failure and in another with considerable edema of the lower extremities of unknown etiology. In 2 cases the increase was slight; in 4 it was either moderate or considerable and in 3 cases it was great. When a quantitative estimate was made of the excess of urinary output over fluid intake in the 9 cases over a period of 173 days, it was found that Vitamin C induced greater diuresis than digitalis but less than theobromine, theobromine with sodium salicylate, and ammonium chloride. In each of 3 patients in whom cardiac failure had occurred with auricular fibrillation, Vitamin C induced diuresis actually in excess of that produced by digitalis, although never with the same degree of clinical improvement nor with reduction of the ventricular rate. These results direct attention to the need of providing an adequate supply of Vitamin C for all patients with cardiac failure. To ensure a constant state of Vitamin C saturation in heart failure it is probably enough to include in the patient's diminished fluid intake an adequate proportion of lemon and orange juice."

Evans, Vitamin C in Heart Failure, Lancet London, 1:308, February 5, 1938, as Abstracted by Journal of the American Medical Association, 110, 18:1526, April 30, 1938.

". . . . Sudden death from heart disease in relatively young or middle-aged persons is caused, in a measure, by blood vessel and heart disease resulting from a lack of sufficient foods containing Vitamin C . . . ."

Quigley, Notes on Vitamins and Diets, Page 126, Consolidated Book Publishers, Inc., Chicago, 1933.

"An analysis was made of the cardiac rate, electrocardiographic complexes and the response to drugs of rats in the non-deficient state and in repeatedly induced Vitamin B1 deficiency. The heart rate fell slowly during feeding on a deficient diet, but returned to normal within a few hours after an adequate dose of Vitamin B1. In most cases of deficiency, changes occurred in the electrocardiographic complexes. Exercise did not produce more rapid heart changes in deficiency. The cardiac responses of deficient and normal rats to adrenaline were the same. Atropine and section of the vagus nerve did not abolish the changes caused by deficiency. Deficient rats were more sensitive to the toxic effects of strophanthin."

Weiss, Haynes and Zoll, Electrocardiographic Manifestations and the Cardiac Effect of Drugs in Vitamin B1 Deficiency in Rats, American Heart Journal, 15:206-220, 1938, as abstracted by Chemical Abstracts, 32, 22:9202, November 20, 1938.

"The cardiac disturbance is of great diagnostic importance in distinguishing beriberi from other, unrelated forms of neuritis and is the most serious threat to life in the patient not already endangered by infectious disease. Cardiac symptoms come and go but are always present at some stage of beriberi. They consist of shortness of breath, precordial pain, boring in nature, which may be as severe as that in angina pectoris.

"The heart is enlarged and the liver swollen and tender. The veins of the neck are engorged. The pulse is weak. The cardiac symptoms, when severe, cause intense suffering and discomfort. The patients toss about in bed unable to compose themselves. They may die suddenly in such attacks. Respiration is so laborious it resembles that of respiratory obstruction."

Eddy and Dalldorf, The Avitaminoses, The Chemical, Clinical and Pathological Aspects of the Vitamin Deficiency Diseases, Pages 92-93, The Williams and Wilkins Company, Baltimore, 1937.

"The symptoms of acute cardiac beriberi closely resemble those of infantile beriberi. Patients with predominating cardiac symptoms usually die suddenly, and often without a history of prodromal symptons. At any time during the course of the disease, and often without warning to the physician, symptoms of acute cardiac failure may suddenly appear. This cardiac failure is characterized by paroxysmal onset of precordial pain, dyspnea, cyanosis, increased venous pressure, tachycardia, murmurs, and a small, thready pulse. Transient elevation of the systolic and diastolic blood pressure and electrocardiographic changes are frequently described. Examination of the heart shows striking enlargement, especially to the right, associated with pulmonary congestion, cyanosis and vasomotor collapse . . . . . ."

Williams and Spies, Vitamin B1 and Its Use in Medicine, The Macmillan Medical Monographs, Page 21, The Macmillan Company, New York, 1938.

"THE HEART. The most common cause of sudden death from Vitamin B1 deficiency is acute cardiac failure. The classical picture at postmortem is a dilated and hypertrophied heart. The right auricle and right ventricle are conspicuously enlarged; the chambers are filled with blood and the valves are normal. At times, when the enlargement of the right auricle and ventricle is pronounced, the left heart may be small. In such instances, the walls are 'paper thin' as a result of the dilatation of the right auricle and conus arteriosus. Chronic passive congestion of the liver, spleen, kidneys, and intestines is nearly always present, and pulmonary edema occurs in at least 50% of the cases."

Ibid Page 62.

"THE MECHANISM OF CARDIOVASCULAR FAILURE IN VITAMIN B1 DEFICIENCY. The beriberi heart results from failure to utilize sufficient quantities of Vitamin B1. It is characterized by two outstanding features: 1, the predominance of cardiac enlargement on the right side; 2, diminished contractile power. Lack of satisfactory anatomical or physiological explanations for these findings has led to the following hypotheses as explanations of the mechanism of the heart failure.

"1. Degeneration of the vagus nerve . . . . .

"2. Respiratory paralysis . . . . .

"3. Water retention or edema hypothesis . . . . ."

Ibid Pages 64 and 65.

"NUTRITIONAL HEART DISEASE IN CHILDREN. Waring presents thirteen cases of cardiac disturbances of nutritional origin. The diagnosis of nutritional cardiac disease was made on the basis of the following observations: cardiac enlargement, shown by percussion and by roentgen examination, without accompanying murmur; negative results of urinalyses; absence or sluggishness of reflexes; a history of deficient diet, and a more or less rapid response to rest and sufficient food. Dietetic histories in most cases were vague, but malnutrition was generally evident. Twelve of the thirteen patients were negro children aged from 14 to 48 months. The cause of cardiac enlargement does not appear in these cases. Whether deficiency of Vitamin B1, deficiency of protein or general dietetic deficiency is the fundamental cause is still a matter of some disagreement. All the patients improved on adequate diets strengthened with materials rich in Vitamin B1. It appears that the cardiac changes are not entirely due to a lack of Vitamin B1 but to a rather more general deficiency of dietary materials and that a satisfactory response to dietetic treatment may be expected in cases in which the illness is not too far advanced."

Waring, Nutritional Heart Disease in Children, American Journal of Diseases of Children, 55:750, April, 1938, as Abstracted by Journal of the Amercan Medical Association, 110, 24:2033-2034, June 11, 1938.

". . . . The heart beat is affected in both pigeons and rats. There is bradycardia and heart block which clear up promptly when the Vitamin (B) is given."

MacLeod, Physiology in Modern Medicine, Page 475, The C. V. Mosby Company, St. Louis, 1935.

"A few years ago, Dr. Drury and I discovered that the hearts of rats suffering from lack of Vitamin B1 beat only half as fast as those of normal rats—i.e. they had what clinicians would call bradycardia.

". . . The interesting point I am leading up to is this, that further investigations in my laboratory have shown that the low heart-rate has to do with the excess of lactic acid, which in the absence of Vitamin B1 cannot be got rid of. The lactic acid seems to poison the heart muscle and prevent it functioning at full rate. Give Vitamin B1, the lactic acid can be disposed of, and the heart is soon beating at its normal rate again."

Harris, Vitamins in Theory and Practice, Pages 65-67, The Macmillan Company, New York, 1935.

"A most interesting finding recently made in England is that a deficiency of Vitamin B produces abnormal slowness of the heart beat. It was proved that this was produced by the specific effect of Vitamin B deficiency, not by insufficient food intake. Since heart failure in mankind is a common cause of death, and frequently occurs in persons apparently in excellent health, the application of such experimental results to human ailments may very well be expected."

Sure, The Vitamins in Health and Disease, Page 41, The Williams and Wilkins Co., Baltimore, 1933.

"Experimentally it has been demonstrated that Vitamin B deficiency produces an abnormal rhythm of the heart, known as 'bradycardia.' In this disease the heart rate in the rat is reduced from a normality of 500-550 beats per minute to 300-350. Since the American diet is often low in Vitamin B, is it not possible that some forms of cardiac diseases are due to cumulative effects of Vitamin B deficiency? No clinical evidence of this is available, however. A statistical investigation of the dietary history of men dying of cardiac diseases would be of medical interest."

Ibid Page 191.

Tohoku states that Vitamin B deficiency is also known to cause heart enlargement.

Tohoku, Journal of Experimental Medicine, Page 184, 1935.

". . . . Bradycardia is common (in Vitamin B deficiency)."

Wiggers, Physiology in Health and Disease, Page 960, Lea and Febiger, Philadelphia, 1934.

"Vitamin-B1 deficiency in rats caused bradycardia, not of vagus origin, and depression of the T wave and the ST segment of the electrocardiogram. It is not clear whether or not the bradycardia is due to certain metabolites produced in excess in this condition (lactic and pyruvic acids and alpha-ketoglutamic acid). Synthetic vitamin administration in normal dogs is reported to produce, on the other hand, a marked and sustained bradycardia. The addition of Vitamin C to the perfusion solution of the isolated frog's heart increases the extent of contraction, especially when added after perfusion has been carried out for some time. Acute Vitamin-C deficiency in the guinea pig is reported to be associated with proliferative lesions along the margins of the heart valves. No changes in the electrocardiograms of normal children receiving therapeutic doses of Vitamin D over a long period of time occur, contrary to the results of previous workers."

Eyster, Heart, Annual Review of Physiology, 1:317-344, 1939.