NUTRITIONAL ASPECTS of HEART FAILURE

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THE CARDIAC patient, like the normal person, should be maintained in nutritional homeostasis. His diet should be patterned as closely as possible after his "normal" diet, provided three objectives are achieved: reducing the work of the heart, preventing or eliminating edema, and establishing nutritional balance for calories, protein, and essential food factors. In addition, the food plan offered must be acceptable to the patient in order to insure its successful enforcement.

The problem of maintaining the nutritional status of patients with congestive heart failure is complicated by several factors. One of the least understood, yet probably most important, is the alteration in cellular metabolism resulting from chronic congestive circulatory failure. This is manifested by electrolyte shifts and a change in water balance within the body.[1-4] A reduction of cellular protein and potassium and an increase in intracellular sodium and water have been demonstrated. Alterations in the osmotic relationships between intra- and extracellular fluid compartments have been postulated on the basis of these metabolic adjustments.[2] Another important contributing factor is the loss of body protein observed in cardiac decompensation. Not only is this phenomenon manifested by the frequent finding of hyproteinemia and hypoalbuminemia,[5-9] but also it is apparent in the muscle atrophy and wasting, especially pronounced in the upper extremities and pectoral regions, of the chronically decompensated patient. Since the cellular electrolyte structure is not altered in compensated cardiac patients, it is probable that the shifts observed in heart failure are, in part, related to the disturbance in protein metabolism.

The total body protein deficits calculated from the value of total circulating albumin indicate an extensive reduction in congestive failure. Assuming a normal blood volume as shown by Ross *et al.*,[10] the total circulating albumin can be estimated. According to Elman,[11] each gram of circulating protein below normal is equivalent to a loss of 30 Gm. of tissue protein. With movement of water into the cells, the values for tissue protein may actually be somewhat lower than these estimates. The loss of muscle mass seen on examination may be a gross reflection of this protein deficiency. Actual measurements of the nitrogen content of skeletal muscle in congestive failure patients at biopsy disclosed reductions of protein below control values.[1]

The loss of cellular protein, and the associated electrolyte and fluid shifts, may prevent the effective utilization of co-enzyme by the cells in the formation of active enzyme.

EFFECT OF CIRCULATORY FAILURE ON NUTRITION

In heart failure, there are basic difficulties in the absorption and utilization of foodstuffs. The reduced appetite limits the actual intake of food, particularly meat and other protein sources. The assimilation of digested foods is impaired by the reduced transport rates in the absorption pathways of the gastrointestinal and portal systems. The dynamic activity of the gastrointestinal mucosal cells may be af-

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fected by an alteration in electrolyte concentrations and by anoxia. Furthermore, the rate of utilization of the absorbed nutrients by the liver is impaired due to the alterations in liver function—a common finding in heart failure.[12] In each of these crucial phases, it is the protein and vitamin metabolism which are especially susceptible to the damaging effects of cardiac failure. Thus, using radioactive iodine and sulphate labeled amino acids, the rate of protein turnover in congestive failure has been shown to be strikingly prolonged by Armstrong and co-workers.[13]

Therapeutic Agents

In addition to the nutritional impairment wrought by anoxia and the circulatory defects of cardiac failure, certain problems arise in connection with the agents employed in its treatment. The use of digitalis and ammonium chloride may produce anorexia and nausea which further reduce the dietary intake. Sodium restriction and the administration of mercurial diuretics under certain circumstances may lead to disturbances in electrolyte balance which further impair cellular function.

Thiamine Metabolism

We have recently studied the effect of mercurial diuretics upon thiamine excretion in nine patients with chronic cardiac decompensation.[14] These subjects manifested signs of progressive heart failure despite treatment with digitalis, diuretics, salt-poor diets, and vitamin supplementation. The excretion of thiamine was measured in 24-hour urine samples obtained after vitamins and mercurial diuretics were withheld for three days. During the control period the mean 24-hour thiamine output was 134.9 μg. The mean 24-hour thiamine excretions on the day of the mercurial injection and the day following the mercurial were 403.2 μg. and 419.4 μg., respectively. These observations suggest that one effect of mercurial diuretics is to increase both the concentration and absolute amounts of thiamine in the urine. Our data also indicate that the mercurial-induced thiamine loss is not a function of the increased urine volume. The most likely explanation is depression of renal tubular reabsorption of thiamine.[15]

In order to study the status of thiamine nutrition in patients with heart disease, 35 subjects admitted to the Philadelphia General Hospital for the treatment of congestive failure were examined by means of a thiamine loading test using an intramuscular injection of 0.35 mg. of thiamine per square meter of surface area.[16] The thiamine excretion over a four-hour period after the loading dose was measured by the method of Melnick and Field.[17] Similar studies were performed in a group of 17 normal controls. The mean thiamine excretion for the 35 cardiac patients was 40.6 μg.; the mean thiamine excretion for controls was 139.5 μg. These findings demonstrate a significant difference between the two groups. The failure of the cardiac patients to excrete thiamine in amounts comparable to normal subjects is interpreted as retention of the vitamin within the tissues to restore the depleted cocarboxylase (diphosphothiamine) levels. Our results are in agreement with those of Robinson and Melnick[18] and others who have found evidence of thiamine deficiency in patients with cardiac decompensation.

Preliminary studies on the thiamine and cocarboxylase content of tissues of 9 patients who died in cardiac failure and 7 control patients who died from noncardiac causes showed statistically significant lower concentration of both total thiamine and cocarboxylase in the heart tissue of patients with heart failure than in the control patients. In liver tissue the difference between the groups was significant for total thiamine but not for cocarboxylase. In kidney tissue the differences were not statistically significant.[19] The determination of blood cocarboxylase in cases of heart failure by Goodhart and Sinclair[20] revealed subnormal values in 6 of 13 patients examined. Thus, ample evidence has accumulated to suggest the presence of subclinical thiamine deficiency in at least some patients with severe congestive failure. The mechanisms operating to produce this condition may include inadequate dietary intake, interference with absorption and utilization of thiamine for cocarboxylase formation, and the increased excretion of thiamine

resulting from the use of mercurial diuretics.

The depletion of tissue cocarboxylase may prevent the utilization of carbohydrate-derived substrate for energy production. However, large decreases of thiamine are required before pyruvate utilization is impaired.[21] This results in a rise in blood lactate and pyruvate which has been demonstrated to exist in congestive failure.[22,23] Furthermore, other nutrients may be used by the myocardium for purposes of energy formation.[24] However, if the energy derived from noncarbohydrate sources were sufficient to maintain the requirements of the myocardium, the existence of beriberi heart disease would be difficult to explain. It is well known that patients with this disease respond poorly to digitalis but recover upon administration of thiamine.

A possible effect of these changes upon the myocardium may be that of further reducing efficiency and rendering the maintenance of compensation more difficult. Furthermore, with a rise of blood pyruvate and lactate there is an associated vasodilation with a resultant rise in cardiac output.[25] There is experimental evidence that deficiency of thiamine, as well as of other fractions of the vitamin B complex (biotin and pantothenic acid), results in lessened ability of heart slices to utilize acetate and pyruvate.[26] The work of Bing[27] using coronary sinus catheterization suggests that there is no defect in oxidative metabolism in heart failure but that the primary difficulty lies in the conversion of the energy derived from these sources into mechanical work. Although this may be true in the early stages of decompensated heart disease, it is possible that in the more resistant later phases of heart failure the biochemical defect of a low capacity for energy formation is also involved.

Relation to Potassium

It is interesting to note that the electrocardiographic changes observed in thiamine deficiency resemble those described in hypopotassemia; namely, a prolonged QT interval with low, broad T waves.[28] Experimental thiamine depletion in animals was found by Pecora to be associated with abnormally low concentrations of potassium in the myocardium.[29] These animals also manifested an increased intracellular sodium content and evidence of water retention.[29] Similar findings in the biochemical analysis of tissues obtained from congestive failure patients suggest that the low thiamine content observed in this condition may be related, in some way, to electrolyte alteration.

Dietary Problems

The recognition of protein and vitamin deficiencies in chronic congestive failure has prompted the administration of large quantities of dietary protein and supplemental vitamins to patients with this disorder. Some difficulties are encountered in augmenting the protein component of the diet because of the necessity for restricting the salt intake. Several dialyzed salt-free milk preparations, flavored with various agents, have been employed, and natural protein foods, such as meats, eggs, and cheese have been fed together with sodium-exchanging resins. The intravenous administration of amino acids and salt-free albumin has been cautiously attempted in a few instances. Vitamin supplementation has been used orally and parenterally in most of the patients examined in the thiamine excretion studies. It has been recommended that 100 mg. of thiamine daily be given to patients with congestive failure. Our present investigations indicate that there is no advantage in the administration of thiamine in quantities larger than 10 mg. daily, parenterally, in correcting the subclinical deficiencies detected by the loading test method.

The patients found to be in congestive failure resistant to the usual therapeutic measures have not always been benefited by efforts at protein and vitamin replacement. However, long-term observations on patients seen in early phases of heart failure with special attention to the maintenance of the nutritional status has not been made. The problem of overcoming anorexia, absorption and utilization defects, and alterations in cellular metabolism are formidable obstacles confronting the physician concerned with the nutritional aspects of cardiac disease.

Clinical Aspects

The indices which denote the precarious nutritional status of many patients with chronic congestive failure are (1) hypoproteinemia, which may act synergistically with salt and water retention and increased venous pressure to produce edema, (2) evidence of muscle wasting reflecting the losses of tissue protein, and (3) the presence of subclinical vitamin deficiencies, especially of thiamine. Our studies have demonstrated and reaffirmed the findings of a deficiency of thiamine nutrition in cardiac patients and have revealed a high incidence of hypoproteinemia and muscle wasting. In addition, we have shown that the use of mercurial diuretics has increased the urinary excretion rate of thiamine.

Diet Patterns—Therapeutic Salt Restricted Diet

The basic foods required in the diet to provide adequate nutrients must receive primary consideration:

> Milk—1 pint; meat—4 ounces; vegetables—2 servings; fruit and fruit juices—3 servings; carbohydrate and fat to fulfill caloric needs.

In order to restore depleted protein levels, it is necessary to increase the protein component by adding meat servings. The caloric requirement of the diet is based upon the body weight of the patient with respect to the desired weight. One may encounter hypoproteinemia in overweight persons. In these instances, we have encouraged high level protein feedings (1.5 to 2.0 Gm. per Kg. body weight) with restriction of carbohydrate. There is evidence to indicate that protein repletion can occur despite caloric restriction under these circumstances, the energy requirements being satisfied by endogenous fat sources.[30,31]

Having fulfilled the dietary requirements for protein, accessory food factors, and calories, the problem of the sodium restriction must be resolved. By employing the following measures the sodium intake can be restricted to an average of 0.6 Gm.:

> Avoid canned foods unless prepared without salt. Use unsalted breads and fats. Avoid frozen peas and lima beans; avoid fresh beets and celery. Use no preserved foods, meats, fruits, flour mixes, or relishes containing salt. Avoid instant coffees and Dutch processed cocoa. Shellfish, except oysters, should be excluded.

For more strenuous salt restriction to the level of 200–400 mg. per day, the use of a low sodium milk such as Lanolac® or Lesofac® is necessary. For high-level protein feedings with sharp sodium restriction various low sodium meat and milk products must be employed. Several salt substitutes such as Co-Salt,® Neocurtasal,® and Diasal® may be recommended for flavoring. These contain ammonium and potassium chloride and may be used in the absence of serious renal insufficiency. Lemon juice, dry mustard, and pepper may also be employed for the seasoning of food. If the basic foods are included in the diet, vitamin supplementation may not be required. However, if diuretics are necessary in the management of fluid retention, the administration of a balanced vitamin preparation orally, or preferably parenterally, is recommended.

Attention to these details should provide an adequate diet to accomplish the objectives of the dietary management of heart disease. However, our ability to influence the defects of absorption and utilization of ingested foodstuffs has not improved. Further study of these aspects of the problem is indicated.

Summary

The nutritional status of patients with congestive heart failure becomes increasingly important as cardiac decompensation advances. While it is true that early in the course of decompensated heart disease no nutritional defects may be encountered, in the resistant phases of chronic heart failure serious nutritional alterations occur. These include changes in electrolytes and water balance, hypoproteinemia, and subclinical thiamine deficiency. Factors contributing to thiamine deficiency

are anorexia and impaired absorption and utilization of nutrients. The conventional use of mercurial diuretics may constitute an additional factor by increasing thiamine excretion, although it is realized that mercurial diuretics are of basic importance in cardiac therapy. Suggestions are made for supplying a dietary program including an adequate protein intake, restricted in salt, supplemented by oral or parenteral vitamin therapy.

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RESUMEN

Aspectos nutricionales de la insuficiencia cardíaca

El estado nutricional de los pacientes con insuficiencia cardíaca congestiva se vuelve más importante cuando la descompensación progresa. Mientras al principio de la descompensación pueden no encontrarse defectos nutricionales, en las fases resistentes de la insuficiencia cardíaca ocurren serias alteraciones en la nutrición. Estas incluyen cambios en los balances del agua y de los electrolitos, hipoproteinemia y deficiencia subclínica de tiamina. Los factores que contribuyen para la producción de la deficiencia de tiamina son la anorexia y la defectuosa absorción y utilización de las substancias nutritivas. El uso convencional de los diuréticos mercuriales puede constituir un factor adicional incrementando la excreción de tiamina, aunque as reconocido la importancia básica que los diuréticos mercuriales tienen en la terapía cardíaca. Se sugiere un programa dietetico que incluye una cantidad adecuada de proteínas, restricción de sal y adición de vitaminas administradas por vía oral y parenteral.