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NUTRITION

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NEPHRITIS

Nephritis is a disease of the kidney that is due to inflammation or degenerative changes. Different parts of the nephron may be hurt in different degrees. It may be acute, subacute, or chronic, and be worsened by edema or uremia (resulting from accumulation of protein waste products in the blood). It may be short in duration, as in acute, or of many years, as in chronic. So there is no one diet to apply to all kinds of nephritis.

Acute nephritis is caused by an infection following a contagious disease in childhood. It may also follow tonsillitis, diphtheria, sinusitis, pneumonia, etc. More frequently this is found in childhood. The characteristics are: less urine (anuria), nausea, vomiting, edema, and hypertension.

Dietary treatment aims for a rest for the kidneys, even to disregard for a few days the general nutrition of the patient. Fluids should exceed only a little the output. Citrus fruit juices may substitute water. If edema is present sodium is restricted. During the first days little food is given except juices, followed with a milk diet of three to four glasses per day, and with later additions of cereals with cream, toast with unsalted butter, jelly, and fruits. For the first two to three weeks protein is restricted. Some doctors today, however, are more liberal with protein.

Chronic nephritis begins ususally with mild symptoms so the subject is not aware of the condition. Urine is abundant

in quantity and has a low specific gravity. There may be morning headaches. Later symptoms are more severe- blood pressure rises and the edema may become worse.

The diet is adjusted as the disease progresses. Due to the great reserve power in the kidneys, no special restrictions are made at first. Little is known as to the cause and there is no cure, dietary or otherwise. The main hope is to keep the body in good condition and prolong life on the highest level. Diet should maintain body weight with adequate protein (80-100 gms.) and of good quality. Edema may require salt and sodium restriction. Water is not restricted, as it is used for toxic material elimination. It may be increased to four to six liters. Foods rich in vitamins are needed as deficiencies in riboflavin, ascorbic acid, and vitamin A are known to cause renal damage. Fat is limited to easily digested forms. Overeating is avoided. Condiments (pepper, mustard, horshradish, and spices) are avoided due to the irritating effects. Alcohol is prohibited. Tea and coffee are used only in limited quantities.

When the body is losing protein in the form of albumin in the urine, the loss must be, if possible, made up with an increase in the protein intake.

GALACTOSAEMIA

Galactosaemia is a well-established disorder due to a genetically determined inborn error of metabolism. There is a specific deficiency of an enzyme called galactose-1-phosphate uridyl transferase which is required for the conversion of galactose to glucose. A deficiency of this enzyme can be shown in the red blood cells and the liver of patients with this inborn error. Hence when the patients with galactosaemia are given a test dose of galactose, the level of galactose in the blood rises steeply, to be followed later by the excretion of galactose in the urine.

As in the case of other inborn errors of metabolism, the clinical picture varies from case to case in severity. In its most severe form the disorder may be manifest in infants within 2 or 3 weeks after birth, as indicated by vomiting, difficulty in feeding, loss of weight, and the onset of jaundice. In such patients the spleen may be palpable and the liver greatly enlarged and very firm. Examination of the urine will show the presence of sugar (galactose) and also protein. Without immediate dietetic treatment of such severe cases death rapidly occurs. In mild to moderate cases the diagnosis may be missed because of the failure of the doctor to examine the urine of an infant who has infrequent vomiting and who is not thriving. If the disorder is allowed to continue for months cataracts are likely to develop and these may lead in time to blindness. Mental and physical retardation are also likely to occur.

Successful treatment requires early diagnosis and the

complete exclusion of lactose and galactose from the diet. If this is accomplished successfully and in time, the hepatic, mental, physical, and ocular changes can be prevented. Even when pathological alterations have occurred in the liver, kidney, and eye, these may be reversed or improved.

Until recently the preparation of a galactose-free diet for infants presented great difficulty as this means the rigid exclusion of milk, which is the main dietary source of galactose. Many technical difficulties are encountered when an attempt is made to remove the lactose from casein. There are, however, available commercially prepared synthetic milk powders in which lactose has been replaced by dextrin, dextrose, and maltose. For those infants with a hypersensitivity to even the very smallest amount of lactose in the diet, a malt and soya flour food has been recommended (Wanderlac). It is still not known whether children with galactosaemia must remain on a galactose-free diet for life or whether after a few years the diet may be modified.

PHENYLKETONURIA

Phenylketonuria is now known to be due to a genetic deficiency of the enzyme phenylalanine hydroxylase which is essential for the conversion of phenylalanine to tyrosine. In consequence the blood and also somewhat, the cerebrospinal fluid and urine of phenylketonurics contain amounts of phenylalanine and its pyruvate, lactate and acetate derivatives greatly in excess of normal. As a result of this accumulation, damage to the brain occurs which may be severe and irreversible unless treatment is instituted at an early age. Of the various clinical features which develop, mental deficiency, usually marked in degree is the more important, but convulsions, tremors, rhythmic rocking of the body and posturing of the hands in front of the body also occur. Skin lesions such as dryness, roughness, and eczema are common. In addition to these mental, neural and dermal changes, phenylketonuric children are often blond, fair-skinned, and blue-eyed, consequent on a defect in melanin metabolism.

Treatment consists of giving the child a diet consisting of very small amounts of phenylalanine. Since this is one of the essential amino acids, normal growth will not occur if excluded entirely from the diet. However, if the daily intake of an infant is reduced from the normal of about 100mg./kg. body weight to approximately 15 to 25 mg./kg. body weight, the child will grow satisfactorily and the level of serum phenylalanine will fall to a normal or low value. Since the amino acid is present in all dietary proteins (2g. of milk

protein will contain about 100 mg.) there are great technical difficulties in providing sufficient protein to keep the patient in nitrogen balance and at the same time ensure a sufficient reduction in the intake of phenylalanine. In addition, such a diet is expensive and difficult to prepare. Fortunately, there are commercial preparations available which provide the essential nutrients and yet keep the diet low in phenylalanine- Minafen (Trufood Ltd.), Lofenalac- (Mead Johnson), etc.

Patients can tolerate different amounts of phenylalanine in the diet and the tolerance may vary in the same patient at different ages. This amount can only be determined by frequent analyses of the phenylalanine concentration of the blood.

At the time of this writing it is not possible to say how long the patient must adhere to the diet. There is some evidence to suggest that it can be relaxed partially or completely when the patient has reached the age of 13 to 15 years. The cost of the diet is very high. Apart from humane reasons, it is cheaper to treat these infants efficiently than to allow them to deteriorate into mental defectives with all the expense of institutional and nursing care which will be required for life.

There is good reason to believe that if efficient treatment is begun in time the clinical features can be prevented, or if already present, can be ameliorated. Unfortunately, the results are far from satisfactory in regard to the mental state, especially, if treatment has been delayed beyond the first year. Mental retardation can be arrested but not reversed. Detection of phenylpyruvic acid in the urine by means of the "Phenistis" paper strip test has been advocated.

ADDISON'S DISEASE

This is rare and the prognosis is grave, though new methods of treatment make possibilities more hopeful. Characteristics are bronze skin, progressive anemia, low blood pressure, dehydration, nausea, diarrhea, and digestive disturbances. The disease is due to impairment of destruction of the cortex of the adrenal glands, caused chiefly by tuberculoses and atrophy. The cortex is believed to control water metabolism and excretion of sodium, potassium, and carbohydrate metabolism.

It has been shown that sodium content of the blood of patients suffering from Addison's Disease is greatly decreased. Formerly a low potassium diet, which is hard to prepare, was used, but now cortical extracts, acetates, sufficient amounts of Sodium Chloride water and an adequate diet are used.

In addition to more salt than is the normal, preparation of food, tablets or capsules of salt may be given. Salt has a sparing effect on the hormonal needs so it helps in the expense.

Diet should have adequate nutrition. There's a tendency to hypoglycemia so a diet high in carbohydrate is advised. It should be served in frequent feedings and include a late evening nourishment.

Protein should be normal, some recommend an allowance of $1\frac{1}{2}$ -2 gm. per kilo. This is necessary if due to digestive disturbances; the diet is low in fats.

Low potassium intake, recommended a few years ago, is no longer necessary.

Appetite is fickle and catering to the desires of the patient maybe necessary to maintain sufficient food intake.

UNCOMPLICATED INFECTIOUS HEPATITIS

Experience with the United States Army has shown that best results are obtained when a full dietary regimen is begun as soon as the diagnosis is made. It is true, however, that anorexia is a cardinal symptom of infectious hepatitis but even those severely anorexic patients will rapidly improve if they will force themselves to eat. Thus, it should be explained to the patient, in some detail, the reasons for the type of diet that is used. He should be told of the dangers of starvation and an unbalanced diet and should be persuaded to eat properly. In other words, the patient should be aware that the dietary prescription provides "medicine" and not just "food". This importance of diet should be stressed to the patient by the physician, the dietitian, the nursing staff, and by anyone who is in contact (such as family and friends) with the patient.

A well-balanced, nutritious, mixed diet contains all of the essential nutrients needed by patients with acute hepatitis. This should be served as attractive meals at regular intervals (as 7:30a.m., 12:30p.m., and 6:00p.m.), when the patient is most likely to be hungry, and by interval feedings of milk shakes or eggnogs (10:00a.m., 3:30p.m., and 10:00p.m.).

Hot food must be served hot; and cold food, cold. But, unfortunately this sort of meal is not often standard diet for the hospital patient. The diet for the patient with infectious hepatitis should be a "special diet" ordered for the individual. It is important to know what the patient has consumed each day

and so the dietitian or nursing staff should weigh or measure and record on a hospital chart plate-wastage after each meal or small in-between-meal feeding. Food should not be brought to the patient by relatives or friends.

If the above measures do not indicate an adequate intake of food, then food must be given by tube feedings, but intravenous feedings will rarely be needed if the patient is urged to eat. Occasionally this threat is necessary to get a patient to eat.

The patient should actually consume (not be served) 40 to 45 calories per kg. of body weight per day. These required intakes can also be calculated on the basis of approximately 20 calories per pound per day.

The protein intake of the patient with infectious hepatitis will be satisfactory if he consumes between $1\frac{1}{2}$ to 2gm. of protein per kg. body weight per day. This is easily obtained from a well-planned, mixed diet. The protein should come from both animal and vegetable sources.

There is no need to restrict fat intake in patients ill with acute hepatitis. Fat is beneficial when supplied at a normal level, at 30 to 35 per cent of the calories per day, they add palatability to the diet. Patients ill with infectious hepatitis are not usually intolerant to fatty foods, but it is wise to provide fat through dairy products instead of through fatty meats and fried foods.

An adequate carbohydrate intake should be easily obtained, but the custom of hospitals allowing hard candy at will to

the infectious hepatitis patient should not be allowed, as this continuous sucking of sweets disturbs the appetite and provides an unbalanced source of calories.

A liberal diet provides all the vitamins and minerals required by this patient. Care should be taken, however, to insure adequate intake of ascorbic acid and the B-complex vitamins. Although some advocate vitamin B₁₂ as a specific measure for the treatment of infective hepatitis, data from the United States Army do not support its special use.

Consumption of alcoholic beverages should not be allowed during the illness and for six months after recovery. This should be discussed with the patient.

CANCER OF THE STOMACH

Carcinoma of the stomach seems to occur where mechanical insult and chemical irritation are more prone to occur or where a lesion already exists. Some say that the great incidence of this disease in those who previously appeared healthy may be explained on the possibility of minor lesions of the stomach being frequently overlooked. Among those "precarcinomatous" lesions, ulcer is thought to rank number one. However, this theory is not universally accepted. Today there is only one reasonably sure etiologic factor in cancer of the stomach - an inherited predisposition.

The tragedy of cancer of the stomach lies in the paucity of symptoms and difficulty of recognition during the early months of development.

Some of the symptoms listed in a study of patients were those that could be fairly common in any group of Americans. Some were: weakness, tired feeling, nausea, vomiting, flatulence, and indigestion. There is no short cut to early diagnosis of cancer of the stomach. It is determined only by x-ray examination, surgical exploration, and microscopic examination.

The dietary treatment of cancer of the stomach is undertaken in order to promote the comfort and nourishment of the patient after operation or when, for any reason, operation is not advised.

The dietary regimen should be determined in some way

by location of the cancer and the nature of the functional disturbance. If depression of gastric secretion is a feature, a diet suitable for chronic gastritis is best. In this milk is a good food. Too much food is not taken at one time and coarse particles are excluded. Coarse, stringy or woody vegetables, tough meats, and bran or whole wheat are not allowed. A good part of the carbohydrate may be taken in dry toast. Vegetables are pureed, as are the fruits. Tender meats are used but fats are taken in the form of butter and cream. In general, it can be said that, with regard to the nature of the secretory disturbance, the same foods and dietary precautions should be prescribed in carcinoma of the stomach as in chronic gastritis. The diet would be largely soft foods, with no roughage. Meats are tender, preferably in finely divided form. Gruels, toast, zwieback, and preserves are prescribed. Feedings, which are small, are given at frequent intervals (5 - 6 times daily)

Anorexia is a frequent problem, and so the food should be as appetizing as possible and of good variety. Tea, coffee, and weaker alcoholic beverages may even be of help. His tastes and inclinations, unless distinctively harmful, should be given wide play.

GOUT

The cause of gout is not definitely known, generally it is thought to be a disturbance of purine metabolism. Purines are formed in the breakdown of certain kinds of proteins, especially those of animal origin. Purines may be produced within muscular tissues of the body or may enter the body via foods in which they occur. Uric acid is the end product of purine metabolism. It is found in blood and tissues and accumulates like glucose does in the body.

Diet is the chief remedial measure. Often patients are overweight and so the diet is usually low in calories.

Diet prescribed by the doctor will depend upon the condition of the patient at that time. An acute attack of gouty arthritis will usually demand a purine free or low purine diet. Eggs, milk, and cheese will be the protein source. The diet is usually low fat to prevent trophi. and foods must be prepared so as to be easily digested and without seasonings, except salt. Some will omit tea, coffee, chocolate, and condiments.

Typically each patient is treated individually according to the stage and severity of the disease, and also according to his temperament. Generally the diet is free of purines on two days and low in purines five days a week. Normal diets have 600- 1000 mg. purines daily, a low purine diet would be 100- 150 mg. daily. Deficiencies in protein, iron, and vitamin B complex should be avoided. Generally the diet is

reinforced with the vitamin B complex. Following is a list of foods free or nearly free of purines:

fruit juices	okra
string beans	potatoes
beets	pumpkin
cabbage	broccoli
tomato	lettuce
turnips	carrots
cucumber	macaroni
white bread	eggs
crackers	cheese
corn bread	fruits
corn	milk
rice	

Acute gout probably is not very common in the United States. However, there are probably many persons with unrecognized cases of mild gout or with inherited gout tendencies. Recent papers show the disease to be more common than realized. It occurs chiefly in middleaged, robust, but overweight individuals, who have a family history of gout. One new idea is that the problem is more one of excretion than one of production of uric acid. When uric acid is held in the body, uric acid content of the blood increases above normal levels, and deposits of sodium urate takes place in the tissues, especially in the joints. There results pain and swelling about the joints and thus gout is often confused with rheumatism or arthritis.

One last thing to mention about diet is that fluid intake should be liberal, as uric acid is excreted better when the urine is of a large volume. Also, organ meats and small fish are especially rich in uric acid-forming substances. Protein allowances should be low but adequate, probably 60-75 grams daily.

HYPERTENSION

Hypertension by itself is not a disease. Rather, it is a symptom complex that may manifest itself in the course of many disorders and whose development may be based on one of several mechanisms.

Obesity is extremely common in patients who have essential hypertension. For this reason, total caloric intake should be restricted in such patients. Great fluctuations in blood pressure often occur after the ingestion of large meals because of decreased vasomotor regulation in patients who have essential hypertension. It is also known that filling of the stomach may result in embarrassment to the heart, or then the heart labors under the double load of hypertension and obesity. As a general rule, a diet restricted to 1,000 to 1,200 calories daily will bring about the desired results without harm to the patient. In most cases, reduction of excessive weight is not accompanied by more than a mild decrease in blood pressure, but in some cases the drop in blood pressure is notable or even striking. Even when the blood pressure is not lowered, improvement in subjective symptoms, such as fatigue and dyspnea, may be noted. It should be noted that dietary restriction should not be, however, carried out over too prolonged a period.

Less frequently, hypertension is seen in patients who are greatly underweight and undernourished. No evidence exists to support the thesis that hypertension is benefited by under-nutrition. So, in these patients it is important to bring the

weight and nutritional status of the patient to a normal level.

For years, sharp restriction in protein intake has been widely used in the treatment of hypertension among some members of the medical profession. The belief has been inculcated so firmly in the minds of the laity that many patients who have hypertension literally have a phobia concerning meat and its supposedly harmful effects. In fact, some patients have voluntarily, or by a physician's order, have made such drastic restriction of protein in their diets that they have experienced symptoms and signs of deprivation of protein, namely, weakness, wasting, hypoproteinemia with edema, and anemia. Up to the present time, evidence does not exist that ingestion of protein plays any role in the production or aggravation of hypertension. As in the case with all foods in the diet of hypertensive patients, moderation should be exercised in the amount of protein eaten, but there should be no objection to a reasonable amount of meat and the temperate use of other protein foods.

Opinion varies with regard to the practice of restriction of fluid in hypertensive patients. Evidence shows that the ingestion of extremely large quantities of fluid may contribute to further increase of blood pressure in hypertensive patients.

No evidence exists that alcohol as such plays any part in the causation of aggravation of hypertension. The use of coffee or tea in moderation is probably harmless to most patients who have essential hypertension. However, their use in patients who suffer from extreme nervousness, irritability, or insomnia should be sharply restricted or even prohibited.

The belief that restriction of the intake of salt is

beneficial to patients who have essential hypertension seems to have begun in the early part of the present century. It has been noted recently that the effectiveness of antihypertensive drugs is often enhanced when the patient is maintained on a diet moderately restricted in sodium, in such cases a diet containing .05 gm. of sodium daily was used. The majority of patients treated by means of diets in which salt is severely restricted have reported definite, and in some cases, pronounced relief of symptoms, particularly headache, tinnitus, vertigo and other distressing cerebral symptoms. In general, the consensus at present appears to be that the benefits to be derived in the management of essential hypertension by rigid restriction of salt are not sufficient to render worthwhile the general use of this form of treatment alone, but that considerable value may be derived by the combination of moderate restriction of salt with other forms of antihypertensive therapy.

In summary, no dietary treatment is known that has any specific favorable effect on the course of essential hypertension. The patient who has essential hypertension should eat as much as is necessary to maintain strength and nutrition but should avoid excess. This restriction of the quantity and quality of the diet should be extended to total calories, total volume of fluids, protein, and salt. Moderation is the key word and should apply to the diet and other forms of treatment.

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