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Cook, Libby, "Nutrition and Disease" (1973). *Honors Theses*. 570.

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NUTRITION AND DISEASE

LIBBY COOK

HONORS 1973

CARE OF THE SURGERY PATIENT

Preoperative Nutrition

With time permitting, nutritional preparation of a patient should correct any deficiencies and provide necessary reserves for the surgery itself and the recovery until oral feedings can be begun.

Protein is a deficiency most related to surgery. Reserves are necessary for blood losses and tissue catabolism.

Calories should be provided for any weight deficit. It is necessary for glycogen stores and the protein-sparing action. Overweight should be corrected, also, to a degree to reduce surgical risks.

Tissue stores of vitamins and minerals are necessary for metabolism of carbohydrate and protein. Deficiencies should be corrected, as should be acidosis, ketosis, or dehydration.

During the immediate preoperative period no food is given by mouth for at least eight hours prior to the surgery, so the stomach will be empty during the operation.

A low residue or residue-free diet may be given to the preoperative gastrointestinal patient for two to three days prior to the surgery so there will be no fecal residue at the operative site.

Postoperative Nutrition

Normally the body tissues undergo a continuous turnover, with losses regained by the food we eat. At times of disease or surgery, losses are especially great and yet food intake is lessened or even absent for a time. Therapeutic nutrition becomes very important as a means of aiding recovery.

Protein- It has already been said that it is necessary to have adequate preoperative protein intake to replace losses and supply increased needs.

Progressively increasing protein deficiency is common in surgical patients. Negative nitrogen balances of up to 20 gm. per day may occur and this loss represents an actual tissue loss of over one pound a day. There are also plasma proteins lost by hemorrhage, bleeding, and exudates. If there occurs extensive tissue inflammation, or infection, there will be further losses. If there has been a prior malnutrition or chronic infection, the protein deficit may become severe.

Following is a list of the body's protein needs.

- (1). Tissue synthesis in wound healing.
- (2). Avoidance of shock - due to reduced blood volume.
- (3). Control of edema - due to a low serum protein level, edema results from a loss of colloidal osmotic pressure to keep a normal shift of fluid from capillaries

and surrounding tissues.

(4). Bone healing, as in orthopedic surgery, protein is needed for proper calcification.

(5). Resistance to infection - amino acids are constituents of proteins involved in body defense.

(6). Lipid transport - to protect the liver from damage by fatty infiltration.

If there is an inadequate amount of protein many clinical problems may develop easily. Among these are poor wound healing and dehiscence (splitting along a suture), delayed fracture healing, anemia, depressed pulmonary and cardiac function, extensive weight loss, liver damage and increased mortality risks.

Calories- Carbohydrate must be supplied to ensure use of protein for tissue protein synthesis and to supply energy needed for increased metabolic demands. Studies done by Calloway and Spector have shown that a minimum of 2,800 calories per day must be provided before protein will be used for tissue repair and not converted for energy use. During great stress, as in extensive radical surgery or burns, from 4,000 to 6,000 calories per day, with 250 grams of protein included, are required. In addition to its protein-sparing action, carbohydrate helps avoid liver damage from depletion of glycogen stores.

Fat calories should be adequate but not excessive as fatty tissue heals poorly and is more susceptible to complications.

Fluid- Fluid therapy is necessary to insure against dehydration. Fluid losses are brought on, following an operation, from vomiting, hemorrhage, exudates, diuresis, fever, or drainage. Intravenous therapy will supply initial needs, but oral intake should be begun as soon as can be, and maintained.

Minerals- Replacement of mineral deficiencies and continued adequacy is a must. Potassium and phosphorus are lost in tissue catabolism. Fluid losses result in electrolyte imbalances in sodium and chloride. Iron deficiency may develop from blood loss or faulty absorption.

Vitamins- Vitamin C is an absolute for wound healing. It is used for formation of cementing material in connective tissue, in capillary walls, and new tissue. Extensive tissue regeneration may require as much as 1 gram daily (15-20 times the normal daily requirement). As protein and calories are increased, to aid in metabolism of the carbohydrate and protein, the B vitamins must be increased. Vitamin K is for blood clotting.

The National Research Council has said that a patient who is not eating well and/or has a previous malnutrition record may need one to two times the normal daily requirement of vitamins. A patient, fed intravenously only, should receive one to two times the minimum requirement for parenteral (other than the intestines) injections

with additional amounts of vitamin C. In serious illness or severe burn, the vitamin requirement is five to ten times the usual amount for the first few days. Thereafter two to three times is basic until recovery is complete.

CHOLECYSTITIS & CHOLELITHIASIS

Cholecystitis is a gallbladder inflammation, which usually results from a low grade chronic infection. The process of infection produces changes in the gallbladder mucosa and this affects its absorptive powers. Normally, the cholesterol in bile (which is insoluble in water) is kept in solution by action of other bile ingredients, especially the bile acids. However, these mucosal changes alter the absorptive powers of the gallbladder and thus affect the solubility ratios of the bile ingredients. Excess water may be absorbed, or excess bile acids may be absorbed.

Under these abnormal absorptive conditions, cholesterol may precipitate, causing gallstones to form, a condition called cholelithiasis. (Gallstones are almost pure cholesterol.) Also, a high fat intake over a long time predisposes to gallstone formation due to the constant stimulation to produce more cholesterol as a necessary bile ingredient to metabolize the fat.

When either inflammation, stones, or both are present in the gallbladder, contraction from the cholecystokinin (contractions due to a hormone and thus bile is secreted) mechanism causes pain. Sometimes the pain may be severe. There is fullness and distention after eating, and difficulties especially with

fatty foods.

Surgical removal of the gallbladder is usually indicated. But, the surgeon may wish to postpone the surgery until the inflammation has subsided. If the patient is obese, as many persons with gallbladder disease are, some weight loss before surgery is advisable. Thus, the supportive therapy is largely dietary.

Diet Therapy - Since fat is the chief cause of contraction of the diseased organ and the subsequent pain, it should be reduced. Calories will come mostly from carbohydrate foods, especially during acute periods. The day's diet should be limited in fat to 20 - 30 grams. Later, the patient may tolerate 50 - 60 grams, and thus the diet will be more palatable.

Two additional modifications usually found on traditional low fat diets for gallbladder disease concern restriction of foods containing cholesterol and foods labeled "gas-formers". Neither modification has valid rationale, the body synthesizes daily several times more cholesterol than found in an average diet. Cholesterol restriction has no appreciable effect in reducing gallstone formation. Total dietary fat reduction is more to the point.

Blanket restriction on so-called gas-formers seems without reason, also. Food tolerances are very individualized.

GLUTEN - INDUCED ENTEROPATHY
(Celiac Disease)

The celiac syndrome seen in children is thought to be due to an enzymatic defect or metabolic error in the intestinal mucosal cells, brought out by wheat or rye gluten. It is believed to be caused genetically but the mechanism is not known. In adults, this condition is called nontropical sprue. By the process of this disease, the intestinal mucosa has villi that atrophy, which greatly reduces the absorptive and secretory surface. Tissue changes bring on pathologic lesions of various sorts. It is not decided as to whether or not these lesions can be reversed. The efforts of some investigators to return patients to regular diets after initial improvement on a low-gluten regimen have been successful with a few children, but most have not been helped by this. Most children seem to recover from the overt disease by school age, but it must be only in remission, as it can reoccur during the adult years. Most adults with sprue had celiac disease as a child.

The protein gluten is found mostly in wheat and rye. It is composed of two parts - glutenin and gliadin. The gliadin is mainly responsible for the malabsorption in gluten-induced enteropathy. About 47% of the weight of wheat gliadin has been found to

be the amino acid glutamine. Studies seem to point to this amino acid as having part in the biochemical defect. It is now apparent that the steatorrhea is a secondary manifestation caused by the primary biochemical reaction to gliadin in sensitive parents.

Clinical symptoms - In children who develop gluten-induced celiac disease, the onset occurs between the ages of six months to eighteen months, with symptoms appearing later in breast-fed babies. It usually begins with a chronic course, which may be worsened by celiac crisis very suddenly, usually triggered by an infection.⁴ This is severe dehydration and acidosis with large, watery stools and copious vomiting. It is an acute medical emergency. There is chronic diarrhea with passage of characteristic foul, foamy, bulky, greasy stools. About 80% of ingested fat appears in the stools, usually in the form of soaps and fatty acids. There will be progressive malnutrition with signs of deficiencies secondary to the malabsorption - anemia, rickets, and increased tendency to bleeding. The abdomen is greatly distended. There is loss of subcutaneous fat tissue, leaving the buttocks flattened and wrinkled with folds of skin. The child takes on the emaciated, apathetic, and fretful appearance of malnutrition.⁴

Idiopathic steatorrhea is the name given to the disease clinically identical to gluten-induced enteropathy.

It is sometimes called idiopathic celiac disease, which is often confusing. The only distinction is in etiology. Idiopathic steatorrhea is not induced by gluten and hence does not respond to clinical trial with a low gluten regimen.

Dietary management of gluten-induced enteropathy would be better defined as low-gluten rather than gluten-free, because it is impossible to remove all the gluten completely and there is evidence that a small amount of gluten is tolerated by most patients. Wheat and rye are the main sources of gluten and it is also found in oats and barley. Therefore, these four grains are eliminated from the diet. Corn and rice are the substitute grains used. The offending grains are obvious in cereal form, but they are also used as ingredients (thickeners and fillers) in many commercial products. Therefore, specific instructions must be outlined to the child's parents, giving the principal omissions in each food group and a basic meal pattern. Commercial products involving gluten and careful label reading habits should also be discussed.

Good dietary management varies with the age of the child, his clinical status, and pathologic conditions.

CYSTIC FIBROSIS of the PANCREAS

Cystic fibrosis is a generalized hereditary disease of children that involves the exocrine glands and affects many tissues and organs. In past years its prognosis was poor. Few children with early disease survived past ten years of age. However, with better knowledge of the disease and improved diagnostic tests, clinical treatment and antibiotic therapy, prognosis has improved. Cystic fibrosis usually produces characteristic clinical manifestations:

(1). Pancreatic deficiency with greatly diminished digestion of food caused by the absence of pancreatic enzymes.

(2). Malfunction of mucus-producing glands with accumulation of thick, viscid secretions and subsequent respiratory difficulty and chronic pulmonary disease.

(3). Abnormal secretions of the sweat glands containing high electrolyte levels.

(4). Possible cirrhosis of the liver arising from biliary obstruction and increased by malnutrition or infection.

Treatment, therefore, is based on three factors: (1) control of respiratory infection, (2) relief from the effects of extremely viscid bronchial secretions, and (3) maintenance of nutrition.

The digestive deficiency and malabsorption character of cystic fibrosis is evident in the nature of the child's stools. They are similar to those in celiac disease (typically bulky, mushy, greasy, foul, foamy), but they contain more undigested food. Only about half (50% - 60%) of the child's food is absorbed. Thus the child with cystic fibrosis has a much more voracious appetite.

The basic objective of nutritional therapy is to compensate for the large loss of nutrient material resulting from the insufficiency of pancreatic enzymes. Apparently protein hydrolysates, split fats (emulsified, simple fats), and simple sugars are used readily. There is a wide variation, however, in tolerance of fat, and the amount of fat intake is usually prescribed according to the character of the stools. Large increases of protein seem to be well tolerated and are needed for replacement of losses and for growth.

Dietary programs for cystic fibrosis are similar to those outlines for celiac disease, the food used varying in form according to the age of the child. The diet differs, however, in that gluten sources need not be restricted, and more emphasis is put on food quantity.

DIVERTICULOSIS & DIVERTICULITIS

Divertivula are blind pouches which may be present in the esophagus, stomach, and small and large intestines. They may be congenital in origin or be acquired during life. They are found most frequently in the colon, especially in the sigmoid section. The presence of diverticula is known as diverticulosis; when they are inflamed the condition is called diverticulitis.

About 5 - 10% of all persons have or develop diverticulitis. ^{Chronic} Cononic diverticula develop in middle-aged and elderly people, many of whom are obese. If stagnation in diverticula is followed by an infection, an inflammatory reaction occurs. Repeated attacks of diverticulitis result in a chronically inflamed bowel, with narrowing of the lumen and pericolic adhesions.

If symptomless diverticula are discovered during a routine radiological examination, no treatment is required and the patient should not be informed of their presence.

Many cases of diverticulitis have periodic attacks of mild left-sided abdominal pain, fever, and irregularity of the bowels. These should be treated medically by antispasmodics, antibiotics, and diet. Many of these patients are obese, in which case weight-reducing diets should be prescribed. The diet should be constructed

to prevent constipation, and yet it must not contain excess of roughage. Fruit and vegetables are suitable, provided they are sieved and served as purees. To increase the bulk of the intestinal contents Agar-agar, a product derived from seaweed, should be given, supplemented by the lubricant, liquid paraffin. A bland diet will be found suitable. Purgatives should not be used.

For a minority of cases surgical intervention will be required for features of obstruction, perforation, or abscess formation, or for severe and extensive involvement of the intestine.

FOLIC ACID DEFICIENCY
(Megaloblastic Anemia)

A deficiency of folic acid in human beings produces a macrocytic anemia associated with megaloblastic arrest in red blood cell production. Production of white blood cells and platelets is also hindered. Clinical manifestations include : (1) the weakness and pallor usually associated with anemias, & (2) degeneration of surface mucosal tissue, resulting in ulceration and secondary infections, sore tongue, and gastrointestinal disturbances such as diarrhea, and poor fat absorption. A similar type of megaloblastic anemia occurs with the deficiency of vitamin B₁₂ that is secondary to pernicious anemia. Folic acid deficiency anemia may be distinguished by trial therapy. If the anemia is due to a deficiency of folic acid, a reticulocyte response will be evident within seven to ten days after the administration of folic acid, and blood values will return to normal.

Folic acid deficiency may be due to one of several causes: (1) a primary dietary lack, (2) poor intestinal absorption of the vitamin, or (3) increased metabolic demands, as during late pregnancy and the rapid growth of early infancy, and in concurrent ascorbic acid deficiency.

The diets of persons evidencing nutritional folic acid deficiency are particularly lacking in animal protein foods and green vegetables. These should be supplied from adequate and varied food sources.

Folic acid deficiency occurs usually in conjunction with general malnutrition. The pregnant woman is especially susceptible. The infant is at risk due to stress of growth because of infections, or because of ascorbic acid deficiency resulting from a poor diet.

Generally diets adequate in the other B complex vitamins will also be in folic acid. Doses of five to twenty mg. of folic acid may be given with the deficiency.

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