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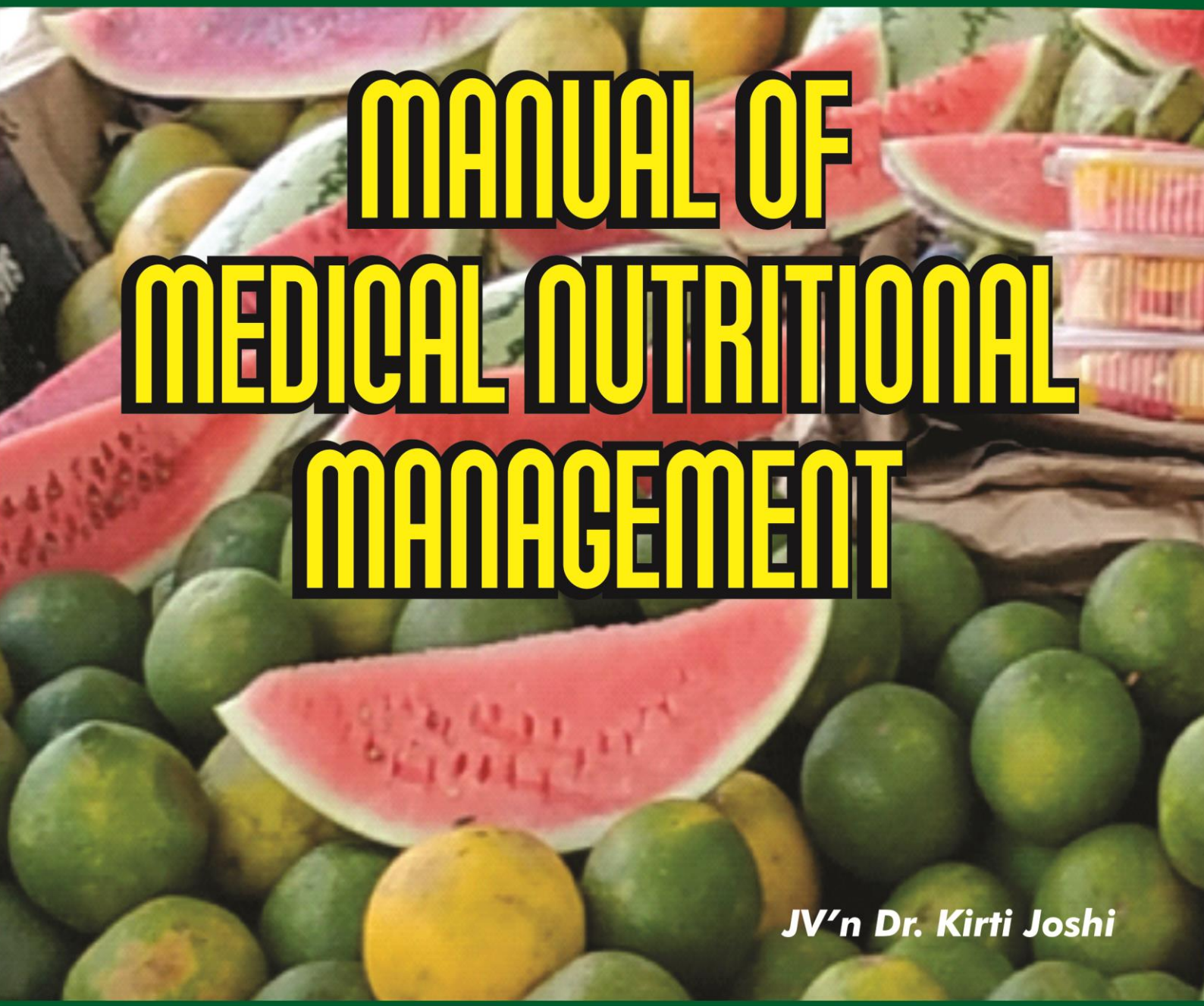
MANUAL OF MEDICAL NUTRITIONAL MANAGEMENT



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Index

OBESITY AND WEIGHT MANAGEMENT

Approximately 35% of women and 31% of men age 20 years and older are overweight or obese. One-fourth of children and adolescents ages 6 to 17 years is considered overweight or obese. The prevalence of overweight and obesity in the India has increased substantially. Obesity contributes to many adverse health outcomes, including type 2 diabetes; cardiovascular disease; hypertension; stroke; osteoarthritis; gallbladder disease; sleep apnea and respiratory problems; and endometrial, breast, prostrate, and colon cancers. Obesity-related conditions are estimated to contribute to 300,000 deaths yearly, ranking second only to smoking as a cause of preventable death. The total cost of obesity amounted to $99.2 billion in 1995, with $51.6 billion being direct medical costs.

Obesity is a complex multifactorial disease that results from the positive energy balance that occurs when energy intake exceeds energy expenditure. Lifestyle and environmental factors, including excessive energy intake, high fat intake, and physical inactivity, are associated with the pathophysiology of obesity. Growing evidence suggests a strong link between genetic factors and the pathogenesis of obesity. Genes involved in energy regulation such as leptin, a signal protein for satiety produced in the adipose tissue, and other hormones or peptides, such as neuropeptide Y, may have important implications for understanding the causes of obesity. Ongoing research is required to determine the role of genetic factors in obesity treatment.

Weight reduction is desirable in patients not only as obesity relates to increased mortality but also because weight loss reduces risk factors for disease. Thus, weight loss may help not only control diseases worsened by obesity but also decrease the likelihood of developing these diseases. Strong and consistent clinical evidence supports weight loss in persons who are overweight or obese and who have hypertension, hyperlipidemia, or type 2 diabetes mellitus. In overweight and obese persons, weight loss is recommended to

• lower blood pressure in patients with hypertension

• lower total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglyceride levels in patients with hyperlipidemia

• lower blood glucose levels in patients with type 2 diabetes mellitus

Fat is lost when the body is in a state of negative energy balance due to a reduced energy intake or increased energy output through muscle work or both. A reduction in

energy intake is an important modality for the treatment of obesity or lesser degrees of overweight.

The discussion below will pertain to indications for the treatment of obesity and overweight. Body mass index (BMI) defined as weight (kg)/height (m2) is the best determinant of weight status and health risk (1,2). For optimal health, a BMI goal of 19 to 25 is recommended, based on evidence that this range is associated with minimal risk of disease. Table IC-3 outlines the health risks associated with the BMI level.

Table1 : Weight Classification by BMI, Waist Circumference, and Associated Health Risks

			Health Risk Relative to Waist Circumference	
Weight	**BMI**	**Risk Class**	**Men ≤102 cm (≤40 inches) Women ≤88 cm (≤35 inches)**	**Men >102 cm (>40 inches) Women >88 cm (>35 inches)**
Underweight	<18.5	--	--	--
Normal	18.5-24.9	0	--	Increased
Overweight	25.0-29.9	0	Increased	High
Obesity	30.0-34.9	I	High	Very high
	35.0-39.9	II	Very high	Very high
Extreme obesity	≥40	III	Extremely high	Extremely high

Source: National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Available at: http://www.nhlbi.nih.gov/nhbli/htm. Accessed June 24, 1998.

Weight reduction is not recommended for the following:

• pregnant women because energy restriction during pregnancy sufficient to produce weight loss can be dangerous

• patients with unstable mental or medical conditions

• patients with anorexia nervosa or a past history of the disorder

• terminally ill patients

• children whose statural growth and central nervous system development could be impaired by a prolonged catabolic state; growth of the child's lean mass should be supported, and the child's adipose mass should be held constant

Moderate energy restriction is recommended. When combined with exercise and behavioral therapy, individualized meal plans of 1,200 to 1,500 kcal/day for women and 1,400 to 2,000 kcal/day for men can promote retention of lean body mass while facilitating weight reduction. To increase long-term compliance and avoid unnecessarily large energy deficits that require major dietary changes, a modest (500-kcal) energy deficit can be established. When setting goals of weight loss with

patients, the dietician should establish a realistic and practical target, such as a decrease of 2 BMI units.

Planning the Diet

The dietitian should plan a calorie-controlled diet to meet the individual needs and lifestyle of the client. Suggestions to reduce daily energy intake include the following:

• Reduce intake of foods with high-caloric density (eg, alcohol and fat). Follow the Dietary Guidelines of less than 30% energy from fat, 10% to 20% from protein, 50% to 60% from carbohydrates.

• Reduce the total amount of food consumed by decreasing portions and frequency of consumption. Employ behavior modification techniques to improve the client's control over the food selection process and the act of eating.

• Establish self-management training techniques that will enhance the satiety of meals but reduce the energy intake. For example, encourage eating more slowly so the patient’s brain can register that the stomach is full, or recommend eating ample amounts of low-caloric density vegetables (eg, salads with small amounts of salad dressing or fat-free dressing) to provide chewing satisfaction and fill the stomach.

www.hindustantimes.com%2Ffitness%2F

The principles of the 20% Fat Diet (see Food Guide), which consists primarily of fruits, vegetables, grains, lean chicken and fish, legumes, non-fat dairy products, oil-based salad dressings (eg, olive oil), and egg whites, are

• 20% or less of calories from fat (poly- and monounsaturated),

• 60-65% carbohydrate (mainly from complex sources),

• 15-20% protein,

• approximately 100 mg cholesterol,

• high in fiber,

• no restriction in calories.

Foods to avoid or restrict with the 20% Fat Diet include

• foods high in saturated fat (eg, all red meat, fried foods),

• high and low-fat dairy products (eg, whole and reduced fat milk, yogurt, butter, cheese, egg yolks, cream)

• alcohol.

Questions / Case Studies

1. When does a person lose or gain weight?
2. Why is it important to regulate weight?
3. A female 21 years old, 5.3 feet height, 82 kgs, having sedentary life style, what will be your suggestions with reference to diet and lifestyle modifications?

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IRON DEFICIENCY ANEMIA

Treatment should focus primarily on the underlying disease or situation leading to the anemia. The chief treatment of iron deficiency anemia is oral administration of inorganic iron in the ferrous iron form. The most widely used preparation is ferrous sulfite, and the dose is calculated in terms of the amount of elemental iron provided. Depending on the severity of the anemia, daily dosage of elemental iron should be 50 to 200 mg for adults and 6 mg/kg for children. Ascorbic acid greatly increases iron absorption. It takes 4 to 30 days to note improvement with iron therapy, especially the hemoglobin level. Iron therapy should be continued for several months, even after the hemoglobin level is restored, so that the body iron reserves are replete.

In addition to medication, attention should be given to the amount of absorbable iron in food. Dietary modification can be adjunctive to iron administration or can be prophylactic in the individual who is at risk for iron deficiency anemia. The diet can be modified to increase the iron intake for any individual.

Dietary strategies involve:

1. Providing foods that have a higher iron density

2. Increasing the iron absorption from food

Iron Density

The normal mixed diet has been said to have an iron density of around 6 mg/1000 kcal. Beef, legumes, dried fruit, and fortified cereals are foods that rank the highest in iron content.

In general, foods that obtain most of their calories from sugar, fat, and unenriched flour have a low iron density. Foods made from whole grain and enriched flour, as well as unrefined foods (fruit, vegetables, and meats), have a higher iron density. Dairy products have a low iron density.

Iron Absorption

The iron content of the body is highly conserved and in the absence of bleeding, little is lost each day. For men and postmenopausal women, for whom the RDA is 8 mg/day of iron, 1 mg of absorbable iron per day will meet this requirement (1).

Dietary iron is provided in the diet in two forms: heme and nonheme. Heme iron constitutes 40% of the iron present in meat, fish, and poultry. Nonheme iron constitutes the balance of the iron in meat, fish, poultry and all the iron present in plant food, eggs, milk, and cheese. Heme iron is better absorbed than nonheme iron. The absorption of nonheme iron is influenced by several dietary enhancing factors, particularly ascorbic acid and meat, fish, and poultry. Ascorbic acid binds iron to form

a readily absorbed complex. Good sources of ascorbic acid include, but are not limited to, citrus fruit and juices, tomatoes and tomato juice, greens, broccoli, strawberries, and sweet potatoes.

Approaches

Guidelines to increase iron intake and absorption are as follows:

- Increase ascorbic acid at every meal.
- Include meat, fish, and poultry at each meal, if possible.
- Avoid drinking tea or coffee with meals.
- Avoid foods with high quantities of EDTA by checking food labels.
- Increase food selections that have a high iron density.

Table 2: Iron Content of Common Foods

Food	Amount	Iron (mg)
Sources of heme iron:		
Beef, cooked, lean	1 oz	0.7
Chicken, cooked	1 oz	0.4
Cod, cooked	1 oz	0.14
Egg	1 large	0.6
Liver, beef, cooked	1 oz	1.9
Liver, chicken, cooked	1 oz	2.4
Oysters, cooked	6 medium	5.0
Pork, cooked, lean	1 oz	0.33
Sources of nonheme iron:		
Apricots, dried	4	0.6
Bread, enriched	1 slice	0.7
Bread, whole wheat	1 slice	0.8
Cereal, dry, fortified	1 cup	4.5 – 18.0
Cream of wheat, cooked	¾ cup	9.0
Farina, cooked, enriched	½ c	7.4
Green beans	½ cup	0.9
Greens, turnip, cooked	½ cup	1.0
Kale, cooked	½ cup	0.6
Kidney beans, cooked	½ cup	2.6
Lentils, cooked	½ cup	3.3
Molasses, blackstrap	1 tbsp	3.5
Pasta, cooked, enriched	½ cup	1.25
Peanut butter	2 tbsp	0.6
Prunes	5	1.0
Prune juice	½ cup	1.5
Raisins	1/3 cup	1.0
Spinach, cooked	½ cup	1.4

Source: Pennington JAT. *Bowes & Church's Food Values of Portions Commonly Used*. Philadelphia, Pa: Lippincott;1998

https://www.google.comfoods-good-for-anemia

Questions / Case study:

A young pregnant woman, aged 27 years and in her third trimester, has approached you for advice about improving her weight since she is slightly underweight. She is suffering from morning sickness and showing signs of toxaemia. She is also slightly anaemic. Write a note as to how you will guide her for the same.

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FEVER

Fever often accompanies infection. The patient may have chills due to fever and may complain of feeling cold. But all fevers are not a result of infection. All elevations in body temperature are not fever. For example, there is elevation of body temperature in heat stroke, as the body is unable to eliminate heat. Fever may be acute and of short duration as in colds, intermittent as in malaria or chronic as in tuberculosis.

Diet:

The dietary treatment varies with the kind of fever and its duration. When fever is acute and of short duration, the most important aspect is to feed sufficient fluids and electrolytes to make up for the losses from the body. As appetite is usually poor, frequent small feeds of liquid and soft foods need to be given to ensure adequate intake. As the condition improves, the size of the feed is increased to meet nutritional needs. The critical problem is protein breakdown, which occurs in infection.

A high protein, high calorie diet is prescribed). Liquid and soft foods need to be fed often to ensure sufficient food intake as appetite is poor. Food allowances for such a diet are indicated in figure. The drugs given (antipyretics) help to bring the temperature to normal. Most of the drugs contain ingredients to relieve pain also. The medication is taken with food to minimise gastric irritation.

Planning the Diet:

The diet is planned as a Regular Diet with addition of between-meal supplements that increase energy intake by at least 500 kcal and protein intake by 25 g for adults. Examples of high-protein, high-energy supplements are milk shakes, eggnogs, puddings, custards, and commercial supplements.
For children, the diet generally should provide 120% to 150% of the Dietary Reference Intakes (DRIs) for energy and protein. The actual amounts of energy and protein provided will depend on the child's or adolescent's age, height, weight, medical status, and nutrition goals.

Dietary modification in fevers:

Energy should be increased by 50%, carbohydrate should be high and as liberal as possible, add glucose in all liquids and fruit juices given to the patient, high protein diet supplying about 1.25 to 1.5 g protein per kg bogy weight day, loss of the two electrolytes, sodium and potassium, should be replaced. Soft texture and fluid to semi-solid consistency re desirable to promote apprtite and patient to consume a nutritionally adequate diet.

https://www.menopausenow.com/night-sweats/articles/relieving-menopausal-fever-night-sweats

Questions/ case study:

1. Define fever. Why do caloric needs increase during fever?
2. How do you plan a diet for a patient suffering from tuberculosis?

PANCREATITIS

Acute pancreatitis involves a systematic immunoinflammatory response to a localized process of autodigestion of the pancreatic gland and can include the involvement of other tissues and organ systems. The onset of pain usually occurs 24 to 36 hours after the peak of cytokine production which leads to the inflammatory response. Distant organ failure can occur in 1 to 3 days.

Nutritional Assessment and Diagnosis:

Acute pancreatitis produces a hypermetabolic response that alters carbohydrate protein, fat, and energy metabolism leading to rapid deterioration of nutritional status. Reduced oral intake can occur from abdominal pain, food aversion, nausea, vomiting, gastric atony, paralytic ileus, or partial obstruction of the duodenum from enlargement of the pancreatic gland. Pancreatitis can also produce a hemodynamic response, which is similar to that of sepsis. This response includes increased cardiac output, decreased peripheral resistance, and increased oxygen consumption, currently known as the systemic inflammatory response syndrome (SIRS). Energy expenditure reportedly increases by 139% of that predicted by the Harris-Benedict equation and can further be increased by 15% if the pancreatitis is complicated by sepsis. Patients with acute pancreatitis are more hypermetabolic when compared to those with chronic pancreatitis.

Nutrition Intervention in Acute Pancreatitis:

Treatment of acute pancreatitis has drastically changed over the past decade. Parenteral nutrition (PN) and bowel rest has traditionally been the primary management approach based on the hypothetical reasons that enteral feeding may stimulate the synthesis of pancreatic enzymes and worsen the severity of disease. However, current evidence suggests that the provision of enteral nutrition (EN) has a dramatic impact on patient outcome compared to the provision of parenteral nutrition (PN). Two landmark meta-analyses demonstrated that use of EN reduces infection by as much as 52%, hospital length of stay by as much as 4 days, need for surgical intervention by as much as 52%, and trend toward reduced organ failure by as much as 41% when compared to use of PN. Experience from the literature suggests that efforts to promote pancreatic rest as the sole management strategy to treat pancreatitis is ineffective and does not have impact on patient outcome.

Nutrition Approaches and Intervention in Chronic Pancreatitis:

Chronic pancreatitis is a chronic, persistent inflammatory state resulting in progressive, irreversible fibrosis and destruction of the endocrine and exocrine tissue. What differentiates chronic pancreatitis from acute is evidence of permanent damage to the anatomy or function of the gland (1). The etiologies that can lead to chronic pancreatitis are nearly identical to those for acute pancreatitis. A flare up of chronic pancreatitis is identical to acute pancreatitis , however, after the acute episode patients may go on to have recurrent abdominal pain

complicated by diarrhea, steatorrhea, and weight loss. Chronic pancreatitis causes many digestive and metabolic disturbances and can compromise the patient's nutritional status over time. Malnutrition occurs late in the disesase course and is a result of a reduction in nutrient absorption, and an increase in metabolic activity. Nutrition intervention and management should focus on maintaining the patient's weight, nutritional status and controlling abdominal pain through symptom management.

Questions/ Case study

1. List metabolic changes which occur in Pancreatitis

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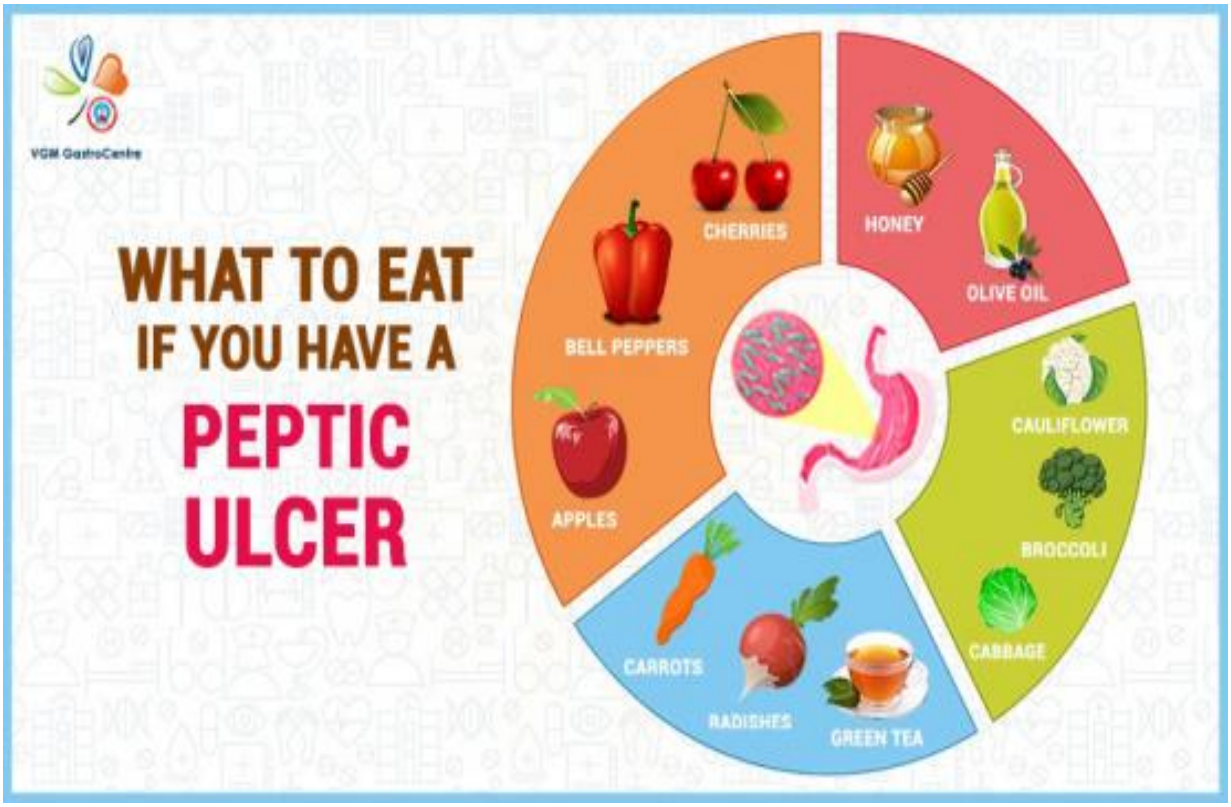
PEPTIC ULCER

Discussion

Peptic ulcer disease includes esophageal, gastric and duodenual ulcers. Research identifies the Helicobacter (H.) pylori bacteria as the primary cause in 95% of gastric and duodenal ulcers. The remaining 5% is caused by non-steroidal anti-inflammatory medication usage (eg, aspirin and ibuprofen) and excessive production of stomach acid. The treatment for individuals infected with the H. pylori bacteria includes healing the ulcer with acid suppressing medication and curing the infection by using antibiotics.

Medical Approaches	Rationale
Avoid foods not tolerated. (See Section ID: Gastrointestional (GI) Soft Diet)	Eliminate foods that cause pain or discomfort to the patient during the acute phases
Antisecretory medication (histamine H_2 antagonist blocker) Cimetidine (Tagamet), Ranitidine (Zantac) Famotidine (Pepcid), Nizatidine (Axid)	Reduces gastric acid and pepsin secretion
Antibiotics	Inhibits growth and destroys microorganisms, ie, H. pylori bacteria
Antacids	Buffers acidity
Sucralfate (carafate)	Forms protective coating over ulcer



https://www.vgmgastrocentre.com/blog/eat-peptic-ulcer-stomach-ulcer/

Questions/ case study:

1. List foods to be avoided in low residue diet.
2. What are the factors which affect food tolerance?

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GLUTEN-FREE DIET

Description

The Gluten-Free Diet is the primary treatment for celiac disease, which is also called gluten-sensitive enteropathy or celiac sprue. The only treatment for celiac disease is lifelong adherence to a gluten-free meal pattern, including strict avoidance of prolamins, which are proteins found in wheat, rye, barley, and triticale (1). Dermatitis herpetiformis is the term for the skin manifestation of celiac disease. The Gluten-Free Diet also helps to control most cases of dermatitis herpetiformis associated with gluten-sensitive enteropathy .

Indications

Celiac disease is an immune-mediated disease characterized by chronic inflammation of the small intestine mucosa that results in malabsorption due to atrophy of the intestinal villa. Although celiac disease was once thought to be a rare childhood disease, it is now recognized as a fairly common multisystem disorder that occurs in one in 133 people. Individuals with celiac disease have an immunologic reaction to proteins termed prolamins, which are found in wheat, rye, and barley. When foods containing gluten are consumed by a person with celiac disease, the digestive process fails and an immunologically reactive protein fragment remains.

Classical celiac disease:

This class is characterized by predominant gastrointestinal symptoms and sequelae including malabsorption, significant weight loss or gain, failure to grow (in children), diarrhea, constipation, excessive gas, bloating, and abdominal pain. The diagnostic testing reveals positive serologic test results and biopsy-proven intestinal atrophy. Symptoms improve after a patient adopts a gluten-free diet.

Celiac disease with atypical symptoms:

This class is characterized by predominantly extraintestinal manifestations and few or no gastrointestinal symptoms. Non-gastrointestinal symptoms include anemia, osteoporosis, peripheral neuropathy or neurological symptoms, dental enamel defects, and fatigue. The diagnostic test results and treatment response are consistent with classical celiac disease.

Silent celiac disease:

This disease is characterized by a lack of clinical symptoms in spite of positive serologic test results and biopsy-proven villous atrophy. Diagnosis of silent celiac disease usually results from screening high-risk individuals, eg, family members and individuals with associated conditions such as type I diabetes mellitus, Down syndrome, or Williams syndrome. A clear outcome benefit of treating these individuals has not emerged from current data analysis.

Latent celiac disease:

This class is characterized by positive serologic test results, the absence of villous atrophy on intestinal biopsy, and no clinical symptoms of celiac disease. These individuals may develop intestinal changes and symptoms of celiac disease later in life.

Planning the Diet:

Eliminate all foods containing wheat, rye, oats, barley, and their derivatives. In addition, evaluate the patient for lactose intolerance, which sometimes appears secondary to celiac disease. If the patient is lactose intolerant, see the discussion of the Lactose-Controlled Diet later in this section. Usually lactose intolerance will normalize within weeks to months of a gluten-free diet. Other nutrient recommendations include the following:

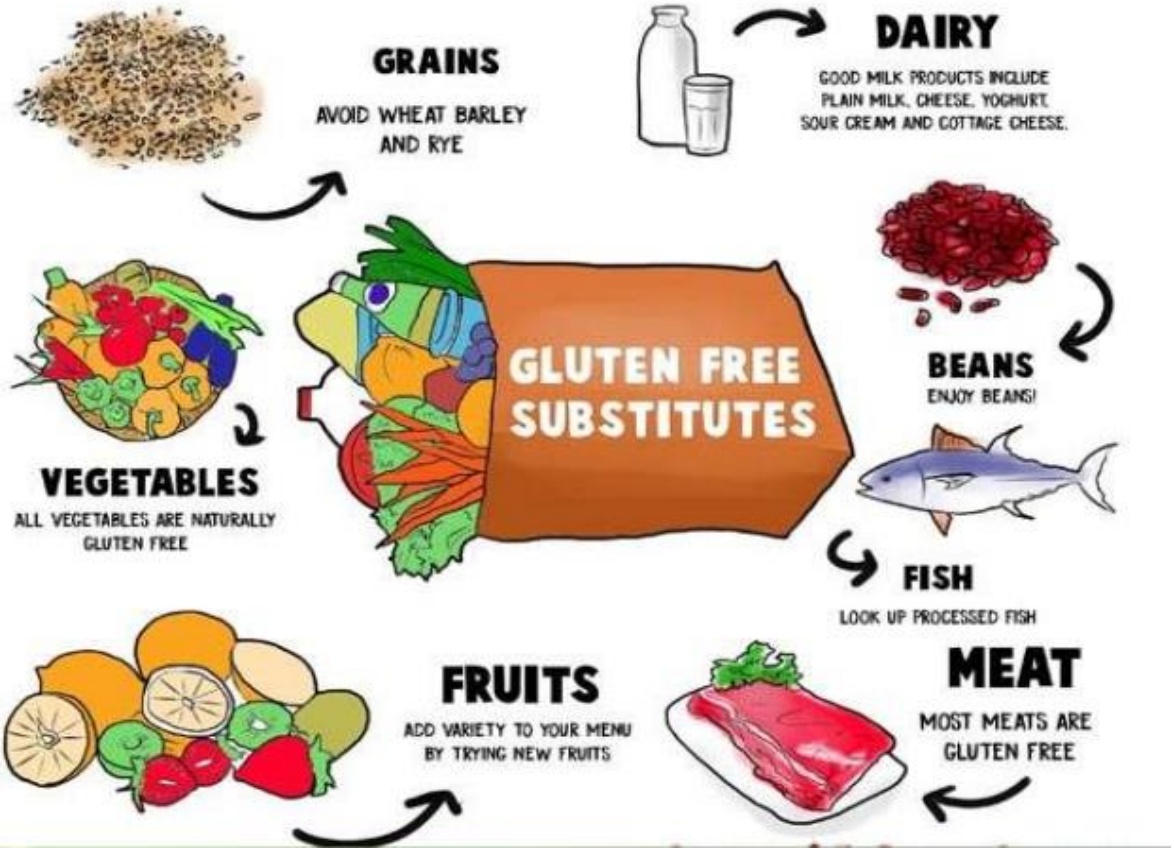
• Protein intake: 1 to 2 g protein/kg of body weight for adults. Use proteins with high biological value.

• Energy intake: 35 to 40 kcal/kg of body weight for adults. Adults may use simple carbohydrates, such as flavored gelatins, fruit juices, simple cornstarch pudding, and fruit. Infants may tolerate banana
powder.

• Evaluate the need for a medium-chain triglyceride supplement, especially in adults.

The food label on all prepared items should be screened. Some hidden sources of gluten include hydrolyzedvegetable protein, flavorings, malt flavoring, brown rice syrup, modified food starch, vegetable gum, soy sauce,monoglycerides and diglycerides in dry products, maltodextrin, emulsifiers, distilled vinegar, and alcohol-basedextracts (eg, vanilla). Some additional components that contain gluten and are often overlooked are broth, breading,
croutons, pasta, stuffing, flours, sauces, coating mixes, marinades, thickeners, roux, soup base, self-basting poultry,imitation seafood, and imitation bacon.
Some medications also contain gluten. All prescribed and over-the-counter medications should be examined by a knowledgeable pharmacist or physician.
Ingredients used as part of packaging are not required to be listed on the label. Gum wrappers are dusted with flour to prevent the gum from sticking. The flour isn’t listed as an ingredient because it is part of the packaging.

Likewise, hidden sources may come in how food is prepared. Food that is prepared on a grill or in a fryer with wheat-breaded items, can pass some of the gluten onto other foods.



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Questions/ Case study:

1. What are the factors which affect food tolerance?
2. Plan a days meal for a patient suffering from Celiac disease.

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LACTOSE-CONTROLLED DIET

Description

The Lactose-Controlled Diet limits intake of milk and milk products to the amount tolerated by the individual. Refer to Lactose Maldigestion medical nutrition therapy protocol for medical nutrition intervention strategies.

Indications

The Lactose-Controlled Diet is indicated in patients who are lactose intolerant; they are deficient in the enzyme lactase and are unable to tolerate ingested lactose. Lactose maldigestion occurs when digestion of lactose is reduced as a result of low activity of the enzyme lactase, as determined by the breath hydrogen test. Interpretation of the terms used to describe lactose maldigestion varies. For example, lactose intolerance refers to the gastrointestinal symptoms resulting from consumption of too much lactose relative to the body's ability to break it down by the intestinal enzyme lactase. Lactose maldigestion or its symptoms (lactose intolerance) should not be confused with a milk allergy, which is an allergy to milk proteins, not lactose. Lactose maldigestion is present in 70% of the world's adults and 20% to 25% of the US population. It is most prevalent among African-Americans, Asians, Hispanics, Native Americans, and people of Jewish descent. Lactose not hydrolyzed by lactase in the small intestine passes into the large intestine, where it is broken down by bacteria. The products of bacterial degradation can irritate the mucosa and raise the osmolality of the intestinal contents, causing a net secretion of fluid. Symptoms include bloating, abdominal pain, flatulence, and diarrhea, usually within 30 minutes after ingestion of lactose-containing foods.

Lactose maldigestion is not a disease, but a normal physiologic pattern (3). Primary lactase deficiency is the most common type and occurs as a normal physiological process in which lactase production in the brush border of the small intestine is reduced (3). Lactase deficiency may be secondary (secondary lactase deficiency) to significant protein-energy malnutrition, acquired immunodeficiency syndrome (AIDS), or iron deficiency anemia. Secondary lactase deficiency has also been observed following the use of antibiotics and anti-inflammatory drugs for arthritis. A transient secondary lactase deficiency may occur following viral gastroenteritis. It has been observed following surgical resection of the stomach or small bowel when there is a decrease in the absorptive area, following radiation therapy to the gastric or pelvic area, and after prolonged disuse of the gastrointestinal tract (eg, with total

parenteral nutrition). However, the lactase activity may return to normal in the latter conditions over time. In children, it is typically secondary to infections or other conditions, such as diarrhea, AIDS, or giardiasis. Lactose intolerance may also be secondary to conditions that produce intestinal damage, such as celiac sprue, regional enteritis, Crohn's disease, and gluten-sensitive enteropathy.

Treatment is aimed at the underlying disorder in order to restore the patient's tolerance to lactose and to eliminate lactose restrictions. Evidence suggests that people with medically confirmed lactase maldigestion can include the recommended number of servings of milk and other dairy foods in their diet, which may actually improve their tolerance to lactose.

In feeding malnourished hospitalized patients and other patients with lactose intolerance, intolerance to 12 g of lactose can be clinically relevant. The following are used to determine the presence of lactose intolerance:

- *A diet history* can reveal symptoms of lactose intolerance following ingestion of lactose. Relief of symptoms following trial of a reduced lactose intake also indicates lactose intolerance.
- *A breath hydrogen analysis test* is the gold standard, or method of choice, to diagnose lactose maldigestion, especially in children. An increase in breath hydrogen concentration, generally 10 to 20 ppm above baseline, warrants a diagnosis of lactose maldigestion.
- *A lactose tolerance test* gives an oral dose of lactose equivalent to the amount of 1 quart of milk (50 g). In the presence of lactose intolerance, the blood glucose level increases less than 25 mg/dL of serum above the fasting level, and gastrointestinal symptoms may appear.
- *A biopsy* of the intestinal mucosa to determine lactase activity.

Planning the Diet

The important consideration is how much lactose can be tolerated without developing intestinal symptoms.

Between 80% and 100% of people with lactase deficiency experience the symptoms described if they drink 1 quart of milk a day. Research indicates that most people with low levels of lactase can comfortably ingest at least 1 cup (8 oz) of milk (12 g of lactose) with a meal and even 2 cups of milk in a day (5,6). One study has found that people with lactose

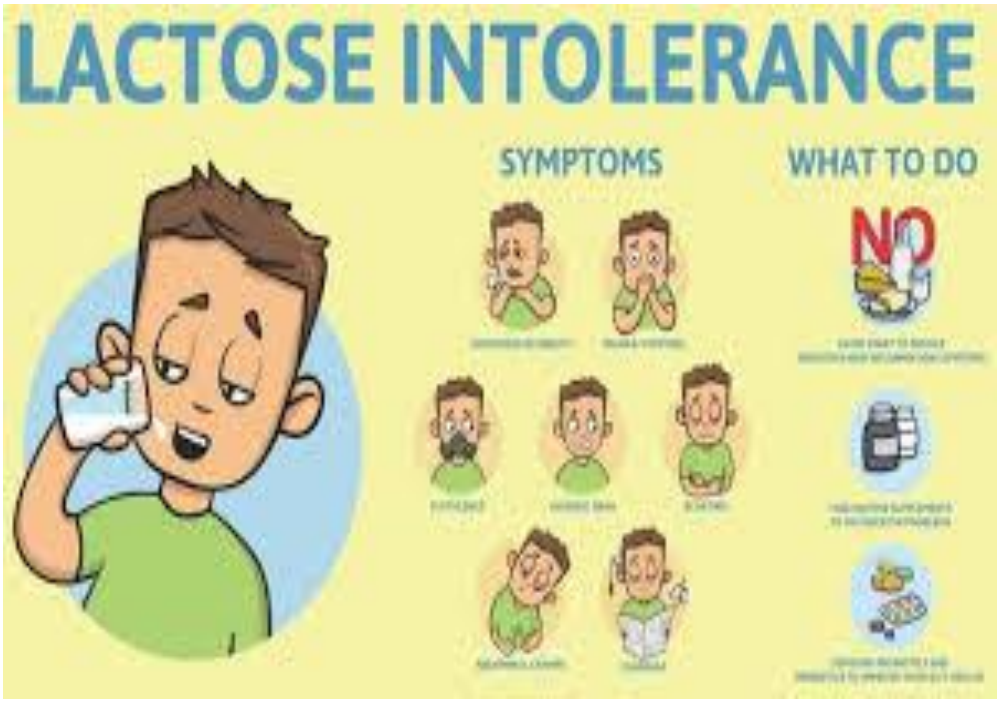
maldigestion can consume 1500 mg of calcium per day if the dairy products are distributed between the three meals and provided partially in the form of yogurt and cheese (2 cups of milk, 2 oz of cheese, and 8 oz of yogurt) (7). Tolerance to milk products is greater when they are consumed with other foods and spaced throughout the day. Whole milk is better tolerated than lower fat milk, and chocolate milk is better tolerated than unflavored milk (8,9). Generally, cheeses and ice cream are better tolerated than milk because of its lower lactose content. Adults with lactose intolerance can usually tolerate the amounts of milk in many prepared foods, such as breads, luncheon meats, and creamed foods, if these foods are given at intervals throughout the day.

Milk contributes a number of important nutrients to the diet, and dairy products are a major source of calcium, protein, and riboflavin. The maximum amount of milk products that can be taken without adverse effects should be included in the diet of persons with lactose maldigestion. Tolerance to lactose can be improved by gradually increasing intake of lactose-containing foods such as dairy products (3).

Commercial lactase enzyme preparations (eg, Lactaid® and Dairy Ease®) will hydrolyze 70% to 90% of the lactose in milk depending on the amount added. Lactose-reduced milks (reduced-fat, nonfat, calcium-fortified, and chocolate) with 70% to 100% of their lactose hydrolyzed are available. Lactose-reduced cottage cheese, pasteurized processed cheese, and some ice creams are available in some markets. Lactaid® caplets and Dairy Ease® tablets, which can be taken before ingestion of milk or milk products, are also available. Products made from soy, eg, tofu, calcium and vitamin fortified soy milk, tofu-based ice cream substitutes, and pasta entrees, are also available.

The following ingredients contain lactose and can be identified on the product's food label: (dry) milk solids/curds, casein, whey (solids), and lactose.

Other compounds that may appear on the food label but do not contain lactose are calcium compounds, kosher foods marked "pareve" or "parve," lactate, and lactic acid.



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Questions/ Case Study:

1. What do you understand by lactose intolerance?
2. Plan a day's meal for patient suffering from lactose intolerance.

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GASTROESOPHAGEAL REFLUX DISEASE (GERD)

GERD involves the symptomatic reflux of gastric contents- particularly acid, pepsin, and bile- into the esophagus which results in damage to the esophageal mucosa and leads to esophagitis, regurgitation, and heartburn. Heartburn is often elicited by lying flat or bending over. If the reflux is severe enough, the same positions may evoke actual regurgitation of gastric fluid into the mouth, causing choking, coughing, and possible pulmonary aspiration. Other symptoms may include dysphagia, pain on swallowing and water brash (when the mouth suddenly fills with a large amount of fluid secreted from the salivary glands).

Ordinary the esophagus is protected from reflux of gastric contents by contraction of the lower esophageal sphincter (LES). In persons with chronic esophageal reflux, the sphincter pressure tends to be lower. Either increased intragastric pressure or decreased LES pressure causes GERD.

Treatment is aimed at modifying the factors that promote gastroesophageal reflux and irritation. Treatment requires a multifactorial approach and is aimed at nutrition and lifestyle modifications, drug therapy, consisting of antacids and hydrogen antagonists and, rarely, surgery.

Management goals are as follows:

1. Limit intragastric pressure.

2. Avoid substances that decrease the LES.

3. Decrease acidity of refluxed material to prevent irritation of the esophagus

Therapeutic treatment is usually provided in three phases.

Phase 1

Approaches: Consume small-volume meals; this may necessitate dividing meals into smaller meals and midmorning and mid afternoon snacks, or consuming fluids between meals. Avoid tight fitting clothing, frequent bending Avoid lying down after eating; consume bedtime snacks or meals at least 2 hours before retiring Elevate head of bed at least 6 inches when

sleeping. Limit fat in diet. Avoid peppermint and spearmint These substances decrease LES pressure. Avoid gastric stimulants:

• Cigarette smoking

• Alcohol

• Chocolate

• Coffee, regular

• Caffeine

Limit food constituents that the patient claims cause discomfort; these may include citrus fruits and juices, tomato products, and carbonated beverages.

Phase 2

Medical Approaches

• Treat with Histamine H2 antagonists

- Cimetidine, rantidin
  • Omeprazole (Prilosec)
  • Bethanechol (Urecholine)
  • Metoclopramide (Reglan

Phase 3

Antireflux surgery

Occasional use for the patient in which maximal medical therapy is not successful, and persistent severe symptoms and complications are present. Although, significant improvement is seen postoperatively, recurrence of symptoms as well as histologic evidence of esophagitis is reported as time progresses.

http://skynewswire.com/7-foods-to-help-your-acid-reflux

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HEPATIC ENCEPHALOPATHY

Description:
Adjustment of the amount and type of protein characterizes the Protein-Controlled Diet for Hepatic Encephalopathy. Energy and protein are provided to attempt maintenance of nitrogen balance and support liver regeneration.

Indications
The diet is used in the treatment of acute and refractory hepatic encephalopathy associated with hepatic disorders, which may include the following:

- hepatitis
- cholestatic liver disease
- cirrhosis with acute and/or chronic encephalopathy

Liver disease causes numerous metabolic problems that can affect all major nutrients and the assessment parameters commonly used to evaluate nutritional status of the patient with hepatic disease. The classic signs of liver disease are anorexia, weight loss, and nausea with marked deficiencies in energy, protein, vitamins, and minerals. Because of the high risk for malnutrition in persons with hepatic diseases the American Society for Enteral and Parenteral Nutrition (ASPEN) recommends protein restriction be no less than 0.6 to 0.8 g/kg and reserved to those patients during acute or refractory episodes of encephalopathy. Normal protein intake should be resumed of 1 to 1.2 g/kg after the cause of encephalopathy has been identified and treated. The widespread practice of protein restriction for all patients with cirrhosis is not justified and often leads to iatrogenic protein malnutrition.

Although malnutrition does not correlate with the type of liver disease, therapeutic modifications vary according to the type and severity of hepatic insufficiency. Generally, fatty liver requires little to no nutrition intervention, while cirrhosis necessitates major changes in the patient’s food intake. A major goal of medical nutrition therapy in liver disease is to prevent and treat hepatic encephalopathy.

Hepatic disease can profoundly affect the nutritional status of the patient because of its effects on carbohydrate, fat, protein, vitamin, and mineral metabolism. Metabolic disorders of the following are commonly seen in the clinical setting of patients with hepatic insufficiency:

- Carbohydrates: Adverse effects can include hypoglycemia or hyperglycemia. Hypoglycemia is most frequently seen in acute hepatitis or fulminant liver disease, probably due to impaired gluconeogenesis. Hyperglycemia is commonly observed secondary to counteracting catabolic hormones and insulin resistance when superimposed by acute stress and injury. Soluble fiber may be beneficial in managing hepatic encephalopathy. Soluble fiber is fermented in the colon by the same mechanism as lactulose, which eliminates ammonia in the form of ammonium ion and bacterial proteins.

- Fats: Malabsorption may occur because of inadequate production of bile salts. This may lead to steatorrhea, which could lead to deficiencies in fat-soluble vitamin and calcium levels. Researchers have found an increase in serum lipids, reflecting lipolysis.

- Protein: The effect of hepatic injury on protein metabolism is more dramatic than is carbohydrate or fat metabolism. There is a decrease in synthesis of serum albumin, the transportation of proteins, and the clotting factors. The ability of the liver to synthesize urea decreases, which results in an accumulation of ammonia and a decrease in serum urea level. This derangement in metabolism elevates the serum aromatic amino acids (AAAs) (phenylalanine, tryptophan, and tyrosine) and methionine and decreases the serum branched-chain amino acids (BCAAs) (valine, isoleucine, and leucine). The only enzymes that metabolize AAAs are located in the hepatocytes. In hepatic insufficiency, there is a decrease in hepatic oxidation of AAAs, leading to an increase in circulation of AAAs in the plasma. In contrast, BCAAs are metabolized primarily by the skeletal muscle. There is an increase in BCAA oxidation in the peripheral tissue during stress, causing a drop in plasma circulation.

- Vitamins and minerals: Hepatic injury results in decreased absorption, transport, and storage and may alter the metabolism of vitamins and minerals. Cirrhotic livers have been reported to store decreased levels of thiamin; folate; riboflavin; niacin; pantothenic acid; vitamins B6, B12, and A; zinc; and cobalt (1,4). In chronic liver disease, the hydroxylation of dietary and endogenous vitamin D to the active form (25-hydroxy derivative) is impaired and may lead to a deficiency state with concomitant osteomalacia. Although there are possibilities of vitamin and mineral

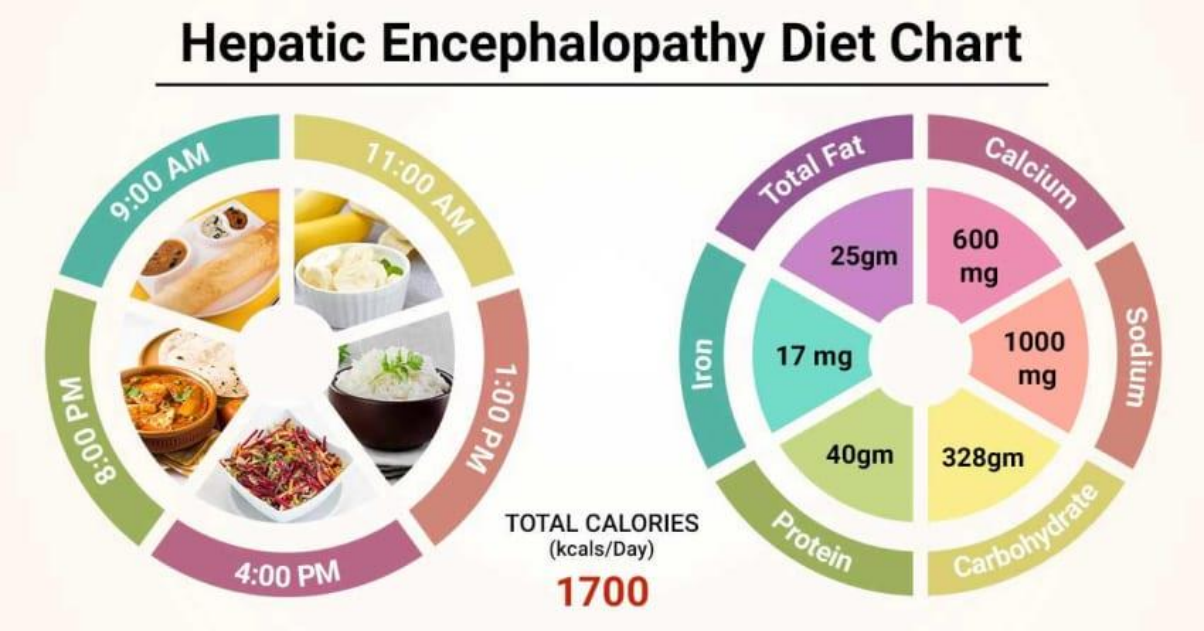
deficiencies, supplementation should be administered only when a specific nutrient deficiency is identified. Supplementation should be monitored. Vitamin K deficiency may be induced from malabsorption with steatorrhea, dietary deficiency, impaired hepatic storage, and/or decreased production of gut flora due to intake of antibiotics. If vitamin K deficiency occurs, the rate at which prothrombin is converted to thrombin is affected, thus hampering the coagulation process and producing inadequate clotting factors (1). Intravenous or intramuscular vitamin K often is given for 3 days to rule out hypopothrombinemia due to deficiency.

Nutritional Adequacy:

Diets containing less than 50 g of protein may be inadequate in thiamin, riboflavin, calcium, niacin, phosphorus, and iron based on the Statement on Nutritional Adequacy in Section IA. Supplementation may be indicated but should be assessed on an individual basis. This diet should be considered a transitional diet. Normal protein intake should be resumed soon after the cause of encephalopathy has been identified and treated. Long-term protein restriction should only be considered in patients with refractory encephalopathy.

Meal size and frequency:

Some patients require small portions and frequent feedings because ascites limits the capacity for gastric expansion. Studies have shown that the metabolic profile after an overnight fast in patients with cirrhosis is similar to normal individuals undergoing prolonged starvation without any associated stress. Cirrhosis can be considered a disease of accelerated starvation with early recruitment of alternative fuels. A small-scale study showed patients with cirrhosis who received an evening snack to supply energy during sleeping hours were able to maintain a greater positive nitrogen balance than did other patients who were fed less frequently



https://www.lybrate.com/topic/hepatic-encephalopathy-diet

Questions / Case study:

1. List metabolic changes which occur in hepatic failure.
2. Plan a diet for a man aged 31 years who is convalescing from hepatic encephalopathy. Outline the nutritional considerations for his diet regime.

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ENTERAL NUTRITION SUPPORT THERAPY FOR ADULTS

Definition

Enteral nutrition support therapy is the provision of nutrients to the gastrointestinal tract via a feeding tube, catheter, or stoma to maintain or replete the patient's nutritional reserves. Enteral nutrition is the preferred route for the provision of nutrition for patients who cannot meet their needs through voluntary oral intake. This section pertains to nutrition support via enteral tube feeding.

Nutrition Assessment

Indications

Enteral nutrition support via tube feeding should be considered as a proactive therapeutic strategy for patients who are unable to ingest adequate amounts of nutrients orally and have an adequately functioning gastrointestinal tract. The advantages of enteral nutrition over parenteral nutrition include:

- a much lower cost (Grade II) and shorter length of hospital stay
- the avoidance of complications associated with parenteral feedings (eg, infectious complications (Grade I), pneumothorax, catheter embolism, and cholecystitis) the support of the metabolic response to stress and a favorable modulation of the immune response in critically ill patients
- the maintenance of gastrointestinal mucosal integrity and prevention of bacterial translocation
- **Contraindications**
- Enteral nutrition support should be avoided in patients who do not have an adequately functioning gastrointestinal tract *or* who are hemodynamically unstable. Specific contraindications include:
- intractable vomiting

- severe diarrhea

- high-output enterocutaneous fistula (greater than 500 mL/day) and distal to site of feeding tube tip placement

- conditions warranting total bowel rest, such as severe acute necrotizing pancreatitis (unless jejunal enteral feeding can be provided beyond the ligament of Treitz)

- severe inflammatory bowel disease

- upper gastrointestinal hemorrhage (caused by esophageal varices, portal hypertension, or cirrhosis)

- short-bowel syndrome (less than 100 cm of small bowel remaining)

- intestinal obstruction (depending on location)

- a prognosis that does not warrant aggressive nutrition support

The initiation of enteral feedings is not contraindicated by a lack of bowel sounds, flatus, or stool passage. Paralytic ileus is the temporary loss of contractile movements of the intestinal wall that results in an absence of bowel sounds or flatus. Ileus was once considered a contraindication to enteral feedings; however, it is now known that ileus has different effects on different areas of the intestine. For example, postoperative ileus appears to affect colonic and stomach function to a greater extent than small bowel function. The period of time that a patient's oral intake is prohibited due to diagnostic tests or procedures should be minimized. A delay in the resumption of feeding or oral intake may exacerbate the potential for ileus. The clinical condition of the patient is an important consideration in the decision to initiate enteral nutrition. A soft, nontender abdomen, adequate perfusion, and hemodynamic stability are indicators of the potential for the safe administration of enteral nutrition. For most patients, lower gastrointestinal bleeding does not affect the administration of enteral support.

Nutrition Intervention

Enteral feedings can be nutritionally adequate if an appropriate formula is selected with consideration of each patient's individual estimated requirements. Energy requirements may be calculated by predictive equations or measured by indirect calorimetry. Predictive equations should be used with caution, as they provide a less accurate measure of energy requirements than indirect calorimetry. In the obese patient, the predictive equations are even less accurate. (Refer to Section II: Estimation of Energy Requirements.) Tube feedings may be used as the sole source of nutrients or as a supplement to inadequate oral nutrition. Enteral nutrition should be initiated within 24 to 48 hours of injury or admission to the ICU, and the average intake delivered within the first week should be at least 60% of the total the estimated energy requirements, as determined by the nutrition assessment (Grade II). Provision of enteral nutrition within this time frame and at this intake level is associated with fewer infectious complications (Grade II). Guidelines for critically ill patients from ASPEN and the SCCM include similar recommendations. These guidelines recommend the provision of more than 50% to 65% of the estimated energy requirements during the first week of hospitalization to achieve the clinical benefits of enteral nutrition The impact of a specific threshold of enteral nutrition delivery on mortality, hospital length of stay (LOS), and days on mechanical ventilation is unclear due to inconsistent results produced by existing studies.

Based on limited evidence available, permissive underfeeding rather than overfeeding obese critically ill patients may produce better medical outcomes. In obese, critically ill adults, the Registered Dietitian (RD) may consider prescribing hypocaloric, high protein enteral feedings (Grade III). According to the Academy of Nutrition and Dietetics (AND) guidelines, very limited research in patients receiving enteral nutrition shows that the effect of hypocaloric, high protein feeding (< 20 kcal per kg adjusted body weight (ABW) and 2 g protein per kg ideal body weight (IBW) promoted shorter intensive care unit (ICU) stays, although total hospital length of stay (LOS) did not differ. In the group receiving hypocaloric, high protein feedings, nitrogen balance was not adversely affected (Grade III). Guidelines from ASPEN and the SCCM recommends permissive underfeeding or hypocaloric feeding with enteral nutrition in the critically ill obese patient. For patients with a body mass index (BMI) greater than 30 kg/m2, the goal of the enteral nutrition regimen should not exceed 60% to 70% of target energy requirements or 11 to 14 kcal/kg actual body weight per day (or 22 to 25 kcal/kg ideal body weight per day).

In addition to the delivery of energy, the adequacy of protein provision should be assessed on an ongoing basis. The use of additional modular protein supplements is a common practice, as standard enteral formulations tend to have a high ratio of nonprotein energy to nitrogen. The protein requirements of critically ill patients with a BMI less than 30 kg/m2 are 1.2 to 2.0 g/kg actual body weight per day; these requirements may be higher in burn patients or multiple-trauma patients. Critically ill patients who are obese have higher protein requirements to maintain an adequate nitrogen balance and accommodate the needs for wound healing. The protein requirement for class I and II patients (BMI, 30 to 40 kg/m2) is greater than 2.0 g/kg ideal body weight per day, and the protein requirement for class III patients (BMI >40 kg/m2) is greater than 2.5 g/kg ideal body weight per day.

Antioxidant vitamins (including vitamin E and ascorbic acid) and trace minerals (including selenium, zinc, and copper) may improve patient outcome, especially in burns, trauma, and critical illness requiring mechanical ventilation. A combination of antioxidant vitamins and trace minerals (specifically selenium) should be provided to all critically ill patients receiving specialized nutrition therapy.

How to Order the Diet

The physician in collaboration with the dietitian determines the appropriate prescription for the enteral nutrition regimen, including the route and type of formula. A dietitian should facilitate the selection of the formula type and goal rate for tube feeding. Once the goal rate is reached, a nutrient intake study may be beneficial to verify that the total nutrient intake (oral plus enteral) is adequate.

The order specifies:

- product, either by name or as "Standard Tube Feeding," according to hospital protocol

- volume, rate, and timing, including the initial volume and rate, as well as the progression and goal volume and rate (At a standard dilution of 1.0 kcal/mL, the volume will be roughly equal to the number of kilocalories specified.)

- administration and monitoring, following either the facility's standard procedures or individualized orders, including the administration of extra water to flush the tube or meet fluid requirements

Routes of Access for Enteral Tube Feeding

The type and route of feeding tube should depend on the patient's needs and the route that optimizes nutrient delivery (stomach or small bowel) for disease management. The smallest tube possible should be used for patient comfort, and correct placement of the feeding tube should be confirmed by X-ray prior to use (4). When the anticipated need for enteral nutrition exceeds 4 to 6 weeks, a more permanent enteral access device is indicated.

There are several types of feeding tube placements:

- **Orogastric:** The feeding tube is inserted through the mouth, with the tip resting in the stomach.

- **Nasogastric:** The feeding tube is inserted through the nose, with the tip resting in the stomach.

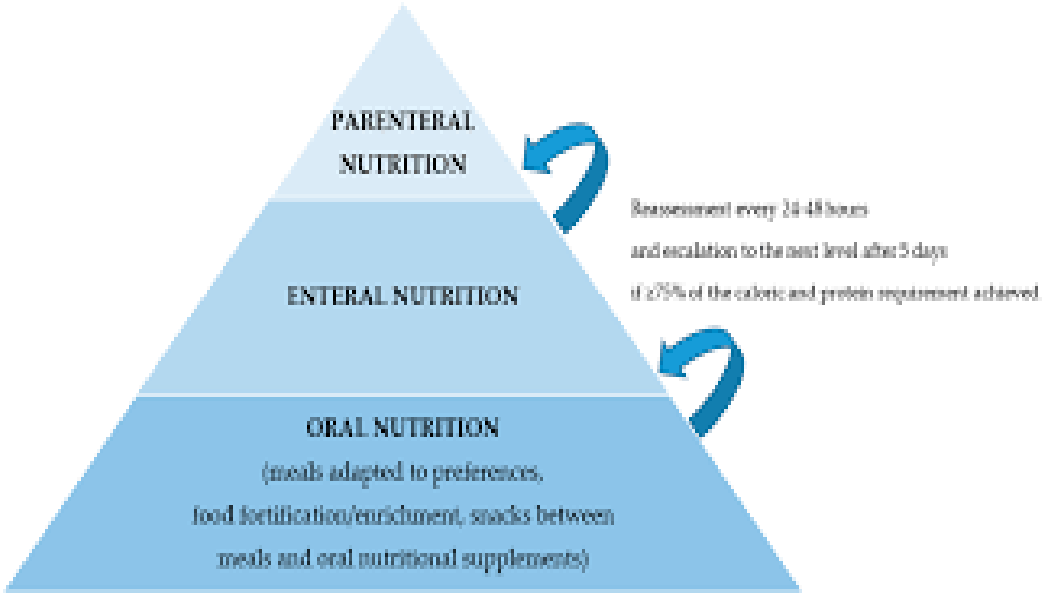
- **Nasoduodenal:** The feeding tube is inserted through the nose, with the tip resting in the duodenum.

- **Nasojejunal:** The feeding tube is inserted through the nose, with the tip resting in the jejunum.

- **Esophagostomy:** The feeding tube is inserted through a surgical opening in the neck and passed through the esophagus, with the tip resting in the stomach.

- **Gastrostomy:** The feeding tube is inserted through the abdominal wall into the stomach via percutaneous endoscopic guidance or surgical placement (surgical "open" gastrostomy).

- **Jejunostomy:** The feeding tube is inserted through the abdominal wall into the jejunum via percutaneous endoscopic guidance or surgical placement (surgical "open" jejunostomy).



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Questions/ Case study:

1. A 32-year-old patient has undergone surgery for gallstones. Prescribe a postsurgery diet. How will you allow progression of the diet?
2. Plan a diet for an old woman who is 65 years old and using enteral feeding.

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PARENTERAL NUTRITION SUPPORT

Total parenteral nutrition (TPN), also known as central parenteral nutrition (CPN), and historically known as intravenous hyperalimentation (IVH), is the delivery of nutrients via a central venous access (subclavian or internal jugular vein). TPN permits the delivery of hyperosmolar nutrient solutions for long-term nutrition support.
Supplementation via a peripheral vein is defined as peripheral parenteral nutrition (PPN) and is a temporary access for the administration of dilute nutrient solutions. (See section entitled "Peripheral Parenteral Nutrition (PPN)".

Indications
Total parenteral nutrition (TPN) is indicated for patients who are unable to take adequate nutrients via the enteral route (eg, patients who have a nonfunctional or severely compromised gastrointestinal tract). Specifically, conditions may include:

• severe malabsorption

- massive bowel resection (> 70% resected)

- severe diarrhea

• intractable vomiting,

• moderate to severe pancreatitis

• ileus

• complete intestinal obstruction

• enterocutaneous fistula

• severe inflammatory bowel disease (IBD)

• malnourished patient undergoing high-dose chemotherapy, radiation, bone marrow transplantation.

Contraindications:

TPN is not indicated for patients

• with a fully functional and accessible GI tract

• inability to obtain venous access

• whose prognosis does not warrant aggressive nutritional support

• whose sole dependence on TPN is expected to be less than 5 days

• when the risks of TPN exceed the potential benefit to the patient.

Nutrient Sources of PN Solutions

Carbohydrate in the intravenous form is available as a monohydrous dextrose. Stock solutions range from 5% to 70% concentration of dextrose in water. Each gram of dextrose yields 3.4 Calories. Dextrose solutions in the TPN mixture usually range from 10% to 35% final concentration. The ability of the body to oxidize glucose is in the range of 2-5 mg/kg BW/min. Provision of carbohydrate in excess of this range may result in overfeeding and can lead to increased CO2 production, minute ventilation, and respiratory quotion (RQ) > 1.0 (5). This may be hazardous for the pulmonary-compromised patient. Overfeeding of dextrose can also lead to fatty liver (steatosis) and unnecessary hyperglycemia.

Protein is available in the intravenous form as crystalline L-amino acids. Stock solutions vary from 3% to 15% amino acids (AA). Commercial AA formulas usually contain 40-50% EAA, 50-60% NEAA. The calorie content of intravenous amino acids is 4 kcal/gram. Most patients require 1.0-2.5 g protein/kg BW/day to meet nitrogen losses (10-12). Exceptions may include patients with hepatic or renal failure who may not tolerate large doses of protein. (See "Specialized Amino Acid Solutions".) Balanced protein solutions (normal NEAA:EAA ratios) contain 16% nitrogen (1 g protein = 0.625 g nitrogen). Protein breakdown for energy seems to be inherent in metabolically stressed patients. It is difficult to determine exact non-protein calorie to nitrogen ratios for protein repletion in stressed patients; however, it is generally recommended to provide 80-150 calories for every gram of nitrogen (10). In the unstressed, stable patient, calorie to nitrogen ratios of 150:1 to 350:1 are recommended.

Fat in the intravenous form is available as long-chain triglycerides (LCT) derived from either soybean oil or as a combination of soybean and safflower oils. Lipid sources are emulsified with egg yolk phospholipid; therefore, use may be contraindicated in patients with known egg allergies. Lipids provide 9 kcal/gram and have been shown to be nitrogen sparing when provided with dextrose in PN solutions. Current stock solutions are available as 10% concentration (1.1 kcal/cc), 20% concentration (2.0 kcal/cc), and 30% concentration (3.0 kcal/cc). Intravenous lipids are isotonic, and therefore contribute minimally to osmolarity of PN solutions. Lipids provide an important source of essential fatty acids (EFA). Two to four percent of energy needs should be supplied as linoleic acid or 25- 100 mg/kg EFA daily. A minimum of 500 cc of 10% lipid stock solution or 200 cc of 20% stock solution given two to three times a week is sufficient to prevent EFA deficiency.

Providing a portion of the nonprotein calories as lipid in PN solutions without overfeeding in total calories has been shown to reduce insulin requirements in hyperglycemic patients (15) and may blunt excessive CO2 production, which has been associated with infusion of high carbohydrate solution. Recent evidence has indicated, however, that excessive rates of LCT infusions may have a detrimental effect on immune function by interfering with the reticulo-endothelial system (RES). Therefore, it is currently recommended that lipids be infused continuously over a 24-hour period and at 25-40% of calories or less, or no more than 1 g/kg BW/day. Exceeding 60% of the nonprotein calories as fat has resulted in fat overload syndrome. Lipids should be used cautiously in patients with hypertriglyceridemia (>250

mg/dL). Specialized Amino Acid Solutions: Use of these formulas is intended for special disease states in which conventional amino acid solutions may not be well tolerated (eg, renal failure, hepatic failure). The contribution of these formulas to improve overall clinical outcome is debatable. Since the cost of these formulas is usually much higher than conventional amino acid solutions, the clinician should evaluate the cost in light of potential benefit to the patient before recommending them for use. In 1993 ASPEN published guidelines for nutrition support in specific disease states. These guidelines serve as a basis for the following discussion of use of specialized or "designer" formulas.

Transitional Feeding

Parenteral to Enteral

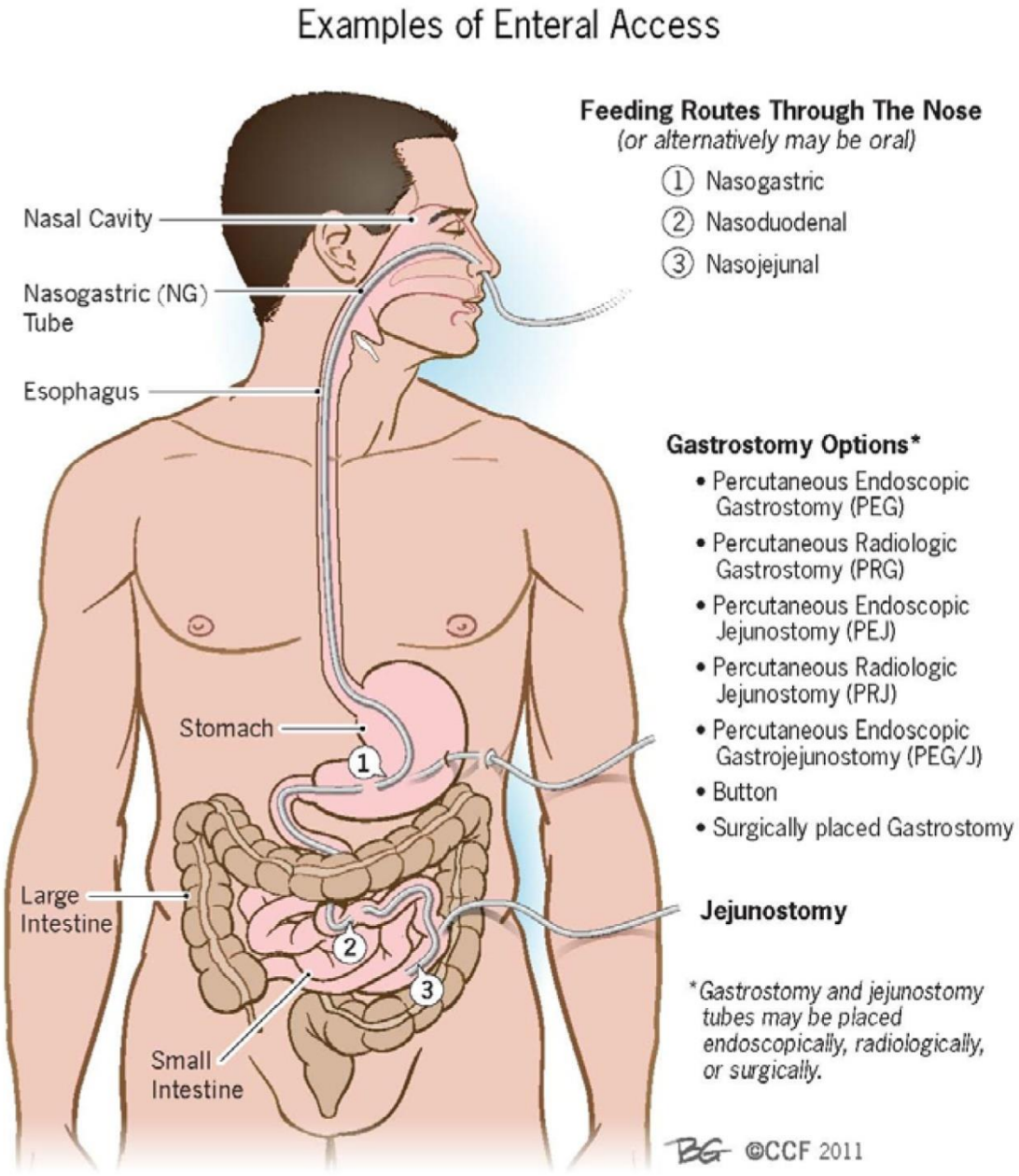
When the patient is transitioned from parenteral support to enteral support, the tube feeding should be initiated at full strength at 10-50 cc/hr. As the rate of tube feeding is increased, the rate of TPN is decreased proportionately. In general, the transition of TPN to TF is accomplished in 3-7 days, depending on the patient's tolerance.

Cyclic TPN

Cyclic TPN is the infusion of TPN over a limited amount of time (usually 12-18-hour periods). Cyclic TPN is indicated for patients who are metabolically stable and for patients requiring long-term TPN, such as home TPN patients. The advantages of cyclic TPN include feedings that more closely resemble physiological (discontinuous) feedings, which may reduce hepatic toxicity associated with continuous feedings, and improved quality of life as the patient is free from TPN equipment during the day.

Peripheral Parenteral Nutrition (PPN)

Peripheral parenteral nutrition is a solution of protein, calories, vitamins, trace elements, and electrolytes that is administered via the peripheral vein. PPN is indicated for use as a temporary measure (<2 weeks) of nutrition support in malnourished patients in whom central PN access has not yet been or cannot be obtained. PPN does not meet the total needs of most patients. Because of the inability of peripheral veins to tolerate soluntions of > 900 mOsm/L, nutrients must be limited in PPN. Dextrose may be provided in 5-10% final concentration, amino acids in 3.0-5.0% final concentration. Lipids are isotonic, and therefore contribute minimally to the osmolarity of the PPN solution. Lipids may also help protect the veins from irritation associated with PPN solutions of dextrose and amino acids. The maximum volume of PPN usually tolerated is 3 L/day (125cc/hr).



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Questions/ Case study:

1. A 32-year-old patient has undergone surgery for gallstones. Prescribe a postsurgery diet. How will you allow progression of the diet?

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