



Medical Nutrition Therapy for Upper Gastrointestinal Tract Disorders



Normal Function of GI Tract

- n Digestion
- n Absorption
- n Excretion



Normal Function of GI Tract

- Digestion
 - Begins in mouth & stomach
 - Continues in duodenum & jejunum
 - Secretions:
 - Liver
 - Pancreas
 - Small intestine

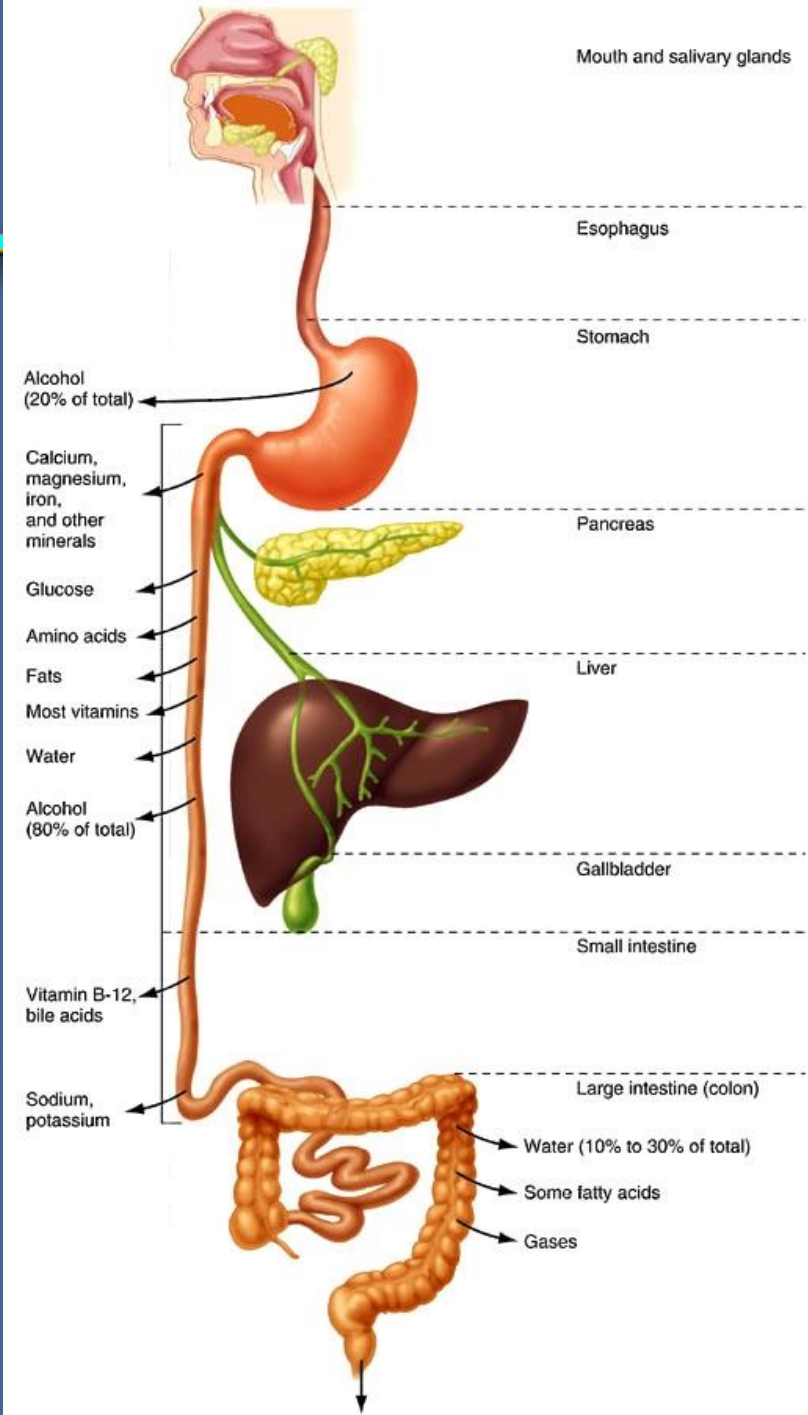


Normal Function of GIT

- Absorption
 - Most nutrients absorbed in jejunum
 - Small amounts of nutrients absorbed in ileum
 - Bile salts & B₁₂ absorbed in terminal ileum
 - Residual water absorbed in colon

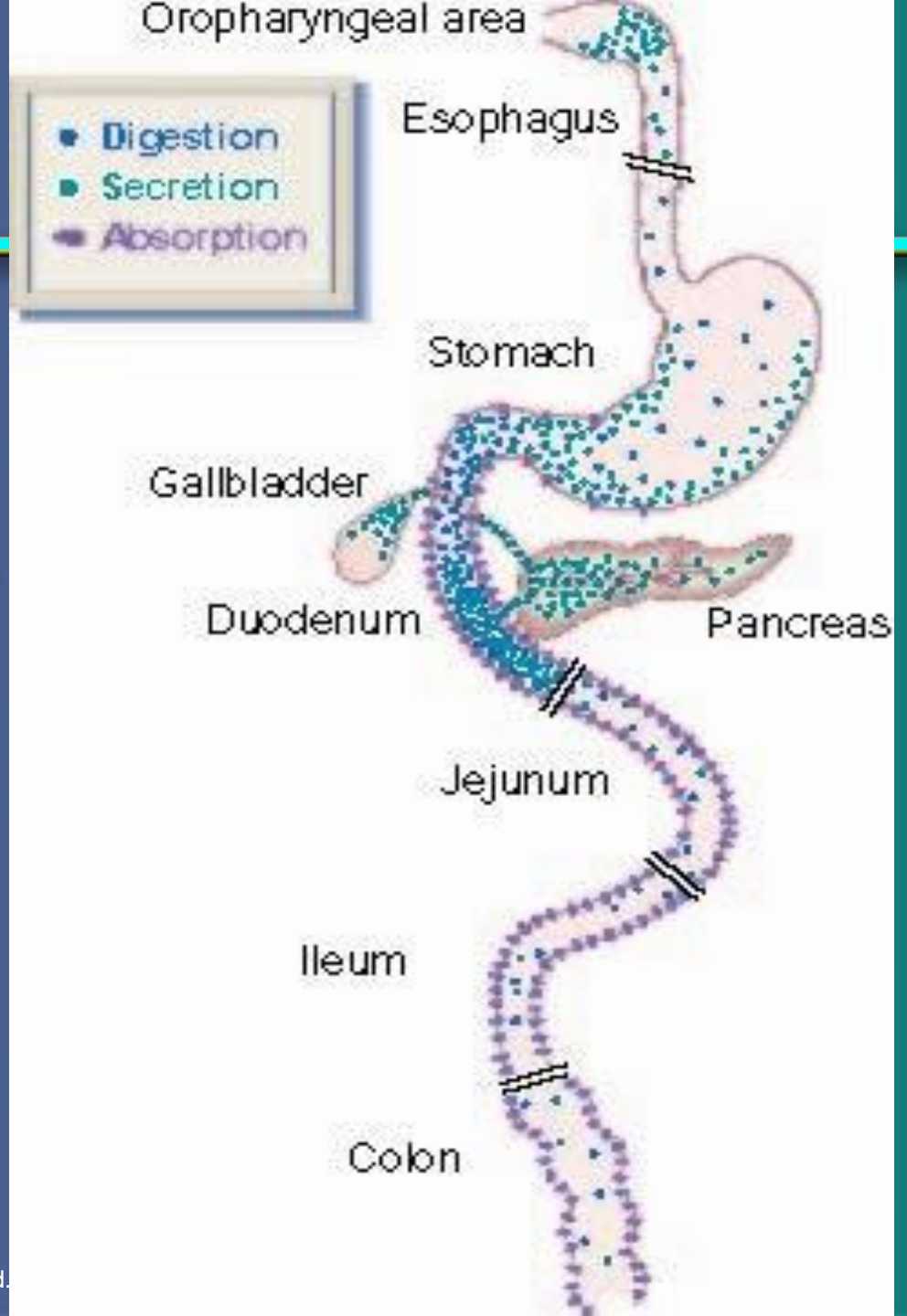


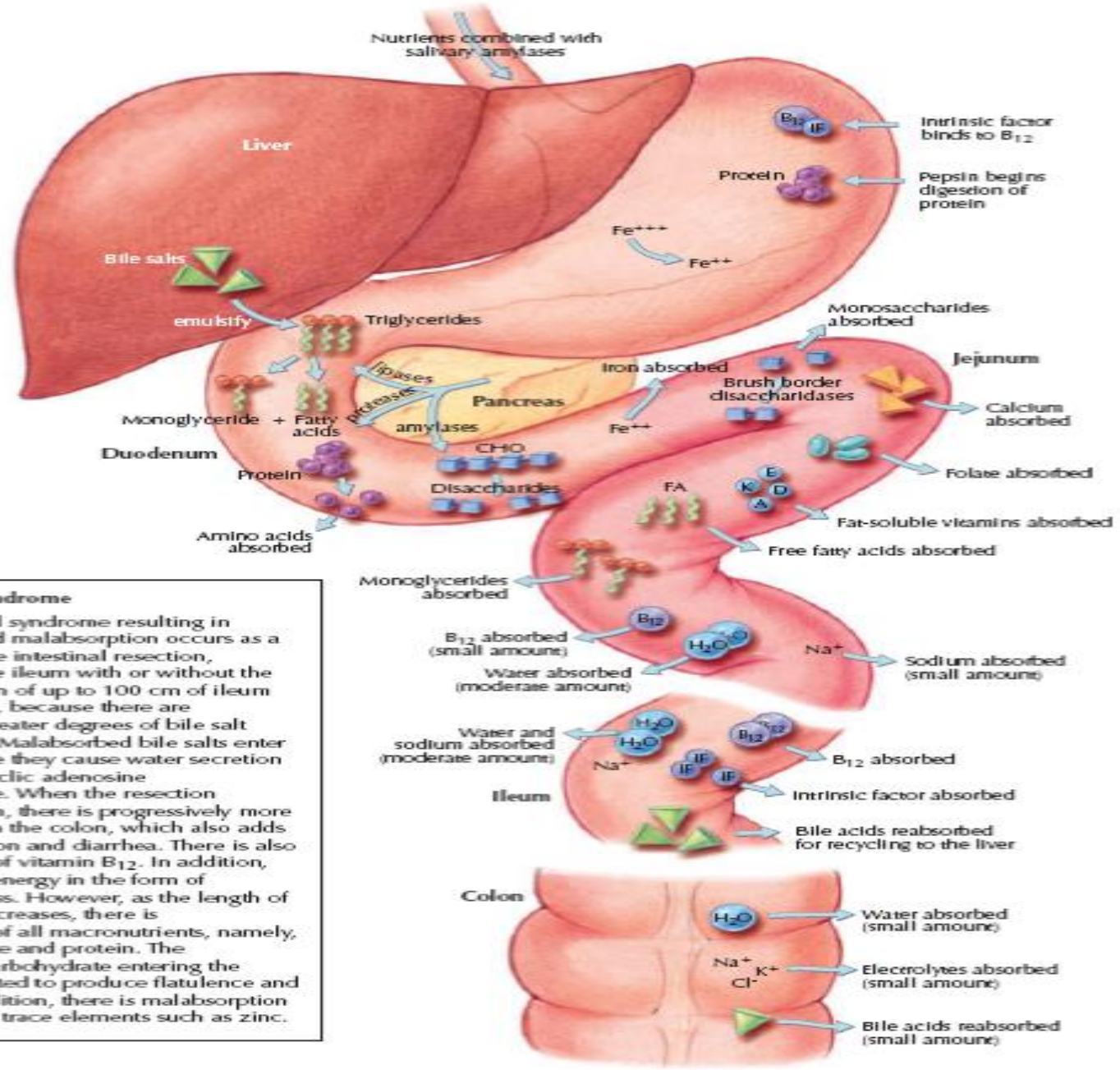
Sites of Digestion and Absorption





Sites of Secretion, Digestion and Absorption





Short bowel syndrome

The short bowel syndrome resulting in dehydration and malabsorption occurs as a result of massive intestinal resection, especially of the ileum with or without the colon. Resection of up to 100 cm of ileum causes diarrhea, because there are progressively greater degrees of bile salt malabsorption. Malabsorbed bile salts enter the colon where they cause water secretion by activating cyclic adenosine monophosphate. When the resection exceeds 100 cm, there is progressively more fatty acid loss in the colon, which also adds to water secretion and diarrhea. There is also malabsorption of vitamin B₁₂. In addition, there is loss of energy in the form of increased fat loss. However, as the length of the resection increases, there is malabsorption of all macronutrients, namely, fat, carbohydrate and protein. The malabsorbed carbohydrate entering the colon is fermented to produce flatulence and diarrhea. In addition, there is malabsorption of vitamins and trace elements such as zinc.

Fig. 1: The relative locations of digestion and absorption of nutrients in the healthy gastrointestinal tract. CHO = carbohydrate.



Principles of Nutritional Care

Intestinal disorders & symptoms:

- Motility
- Secretion
- Absorption
- Excretion



Principles of Nutritional Care

Dietary modifications

- To alleviate symptoms
- Correct nutritional deficiencies
- Address primary problem
- Must be individualized

Common Symptoms of Gastrointestinal Disease

SYMPTOM	POSSIBLE DISORDER
1. Ingestion of solid food causes distress, but liquids do not.	Esophageal stricture or tumor
2. Difficulty swallowing; food sticks in throat	Esophageal spasm; achalasia
3. Epigastric pain when eating	Gastric ulcer
4. Pain 2–5 hours after a meal; pain relief after eating	Duodenal ulcer
5. Abdominal pain several hours after ingesting a fatty meal	Pancreatic or biliary tract disease
6. Cramps, distention, and flatulence 18–24 hours after drinking milk	Lactose intolerance, due to lactase deficiency or rapid transit time
7. Heartburn after eating a large or fatty meal	Esophageal reflux



Disorders of the Esophagus

1. Gastroesophageal reflux disease (GERD)

- Backward flow of the stomach and/or duodenal contents into the esophagus
- Burning sensation after meals; heartburn
- Possible discomfort during and after eating, change in eating habits, especially in the evening



FACTORS AFFECTING LES PRESSURE IN GERD

REDUCE (OPEN)

Alcoholic beverages

Caffein

Chocolate

Cigarettes

Dietary fat

Mint oils

High pressure on stomach: overeating, drinking

Hormone level: progesterone (late pregnancy, late phase menstrual cycle)

Medication: antichollinergics, bronchodilators

INCREASE (CLOSE)

Dietary protein

Medication: bethanechol, metoclopramide



MEDICATIONS THAT SLOW GASTRIC EMPTYING

Calcium channel blocker

Adalat, calan, cardene, cardizen, nimotop, Norvasc

Opiates/opioid

Alfenta, buprenex, codeinetc

Tricyclic antidepressants

Anafranil, adapin, norpramin, etc

Other substrates

Alcohol, marijuana, tobacco

Nutritional Care Guidelines for Patients with Reflux and Esophagitis

1. Avoid large, high-fat meals, especially 2 to 3 hours before retiring.
2. Avoid acidic and highly spiced foods when inflammation exists.
3. Avoid chocolate, alcohol, and caffeine-containing beverages, such as coffee.
4. Avoid peppermint and spearmint oils.
5. Stay upright and avoid vigorous activity soon after eating.
6. Avoid tight-fitting clothing, especially after a meal.
7. Avoid smoking.



Disorders of the Esophagus—cont'd

2. Hiatal hernia

- An outpouching of a portion of the stomach into the chest through the esophageal hiatus of the diaphragm
- Heartburn after heavy meals or with reclining after meals
- May worsen GERD symptoms



Disorders of the Esophagus—cont'd

3. Cancer of the oral cavity, pharynx, and esophagus
 - Existing nutritional problems and eating difficulties caused by the tumor mass, obstruction, oral infection and ulceration, or alcoholism
 - Chewing, swallowing, salivation, and taste acuity are often affected.
 - Weight loss is common.



Disorders of the Stomach

1. Indigestion/dysphagia

- Epigastric discomfort following meals
- Abdominal pain, bloating, nausea, regurgitation, and belching
- Eat slowly, chew thoroughly, and do not eat or drink excessively.



Disorders of the Stomach—cont'd

2. Gastritis

- Helicobacter pylori*
- Infection and inflammation
- Acute gastritis: rapid onset of inflammation and symptoms
- Chronic gastritis: occurs over period of time
- Symptoms: nausea, vomiting, malaise, anorexia, hemorrhage, and epigastric pain



Disorders of the Stomach—cont'd

3. Peptic ulcer disease

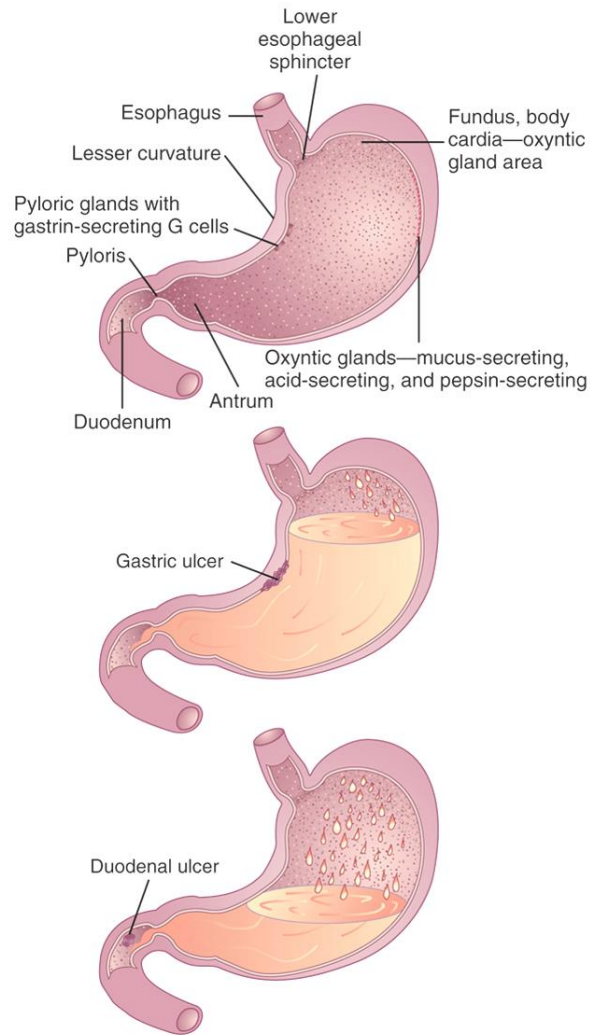
- Primary causes: *H. pylori* infection, gastritis, use of NSAIDs, corticosteroids, and so-called stress ulcers
- Involves gastric and duodenal regions
- Gastric ulcers: in stomach; normal or low acid secretion
- Duodenal ulcers: in duodenum; high acid secretion



Characteristics and Comparisons Between Gastric and Duodenal Ulcers

- n Gastric ulcer formation involves inflammatory involvement of acid-producing cells but usually occurs with low acid secretion.
- n Duodenal ulcers are associated with high acid and low bicarbonate secretion.
- n Increased mortality and hemorrhage are associated with gastric ulcers.

Gastric and Duodenal Ulcers



Factors That Affect Gastric Acidity

Box 29-2. Factors That Affect Gastric Acidity

Increased Gastric Acidity

Cephalic Phase of Digestion

Thought, taste, smell of food, and chewing and swallowing initiate vagal stimulation of the parietal cells in the fundic mucosa, resulting in secretion of gastric acid.

Gastric Phase of Digestion

Effect of food in the stomach: Distention of the fundus stimulates the parietal cells to produce acid. Increased alkalinity of antrum causes the release of gastrin, which stimulates gastric acid secretion. Distention of the antrum causes release of gastrin. Substances in certain foods and digestive products increase acidity (e.g., coffee, both with or without caffeine; alcohol; polypeptides and amino acids [products of protein digestion]).

Decreased Gastric Acidity

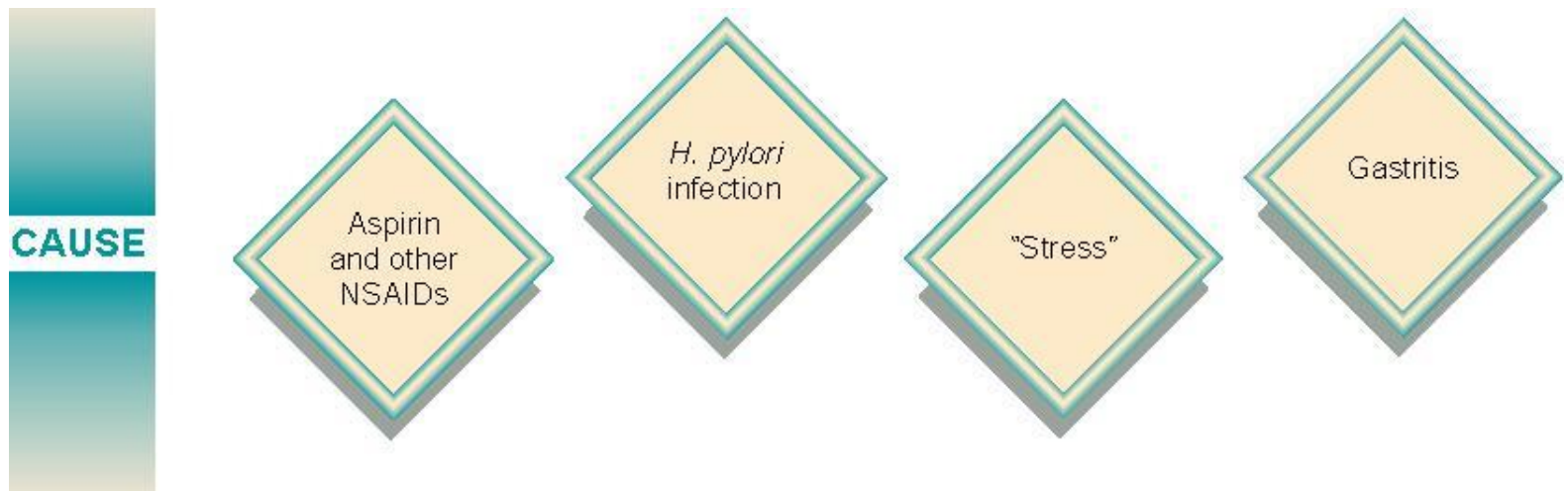
Gastric Phase of Digestion

Acidification of the antrum reduces gastrin release and thus gastric acid secretion. Food, especially protein, has an initial buffering effect.

Intestinal Phase of Digestion

Fat, acid, and protein in the small intestine stimulate release of one or more gastrointestinal hormones that inhibit gastric acid secretion.

Peptic Ulcer–Cause



Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Peter L. Beyer, 2002.

Peptic Ulcer—Pathophysiology

PATHOPHYSIOLOGY

Erosion through
muscularis
mucosa into
submucosa or
muscularis
propria

Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000.

Peptic Ulcer—Medical and Nutritional Management

MEDICAL MANAGEMENT

- Reduce or withdraw use of NSAIDs
- Use antibiotics, sucralfate, antacids
- Acid secretion suppression with:
proton pump inhibitors or
H₂-receptor blockers

BEHAVIORAL MANAGEMENT

Avoid tobacco products

NUTRITIONAL MANAGEMENT

Decrease consumption of:

- Alcohol
- Spices, particularly red and black peppers
- Coffee and caffeine

Increased intake of n-3 and n-6 fatty acids which may have a protective effect

Good dietary/nutritional status helps decrease *H. pylori*

Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Peter L. Beyer, 2002.



TREATMENT OBJECTIVES & RECOMMENDATIONS

OBJECTIVES

Eradicate *H pylori* infection if present

Reduce gastric acidity and gastric secretion

Avoid gastric irritants

Promote ulcer healing



TREATMENT OBJECTIVES & RECOMMENDATIONS

NUTRITIONAL RECOMMENDATION

Individualize the diet

Check for anemia, correct if exist

Check for B12 deficiency, correct if exist

Avoid large meals

Avoid excessive alcohol, coffee, decaffeinated

Avoid cigarette

Include extra protein and vitamin C to promote healing



GASTRIC IRRITANTS AND GASTRIC ACID STIMULANTS

IRRITANTS

Pepper (all type)

Chilli

Alcohol

STIMULANTS

Regular and decaffeinated coffee

Frequent meals (3 meals preferred)



Drugs Commonly Used to Treat Gastrointestinal Disorders

- n Antacids: lower acidity
- n Cimetidine (Tagamet), ranitidine (Zantac):
block acid secretion by blocking histamine
H₂ receptors
- n Prostaglandins
- n Sucralfate: coats and protects surface
- n Colloidal bismuth: coats and protects surface
- n Carbenoxolone: strengthens mucosal barrier
- n Tinidazole: antibiotic



Disorders of the Stomach— Nutritional Care

- n Lifestyle changes are an important component of the nutrition care plan.
- n Patients with dyspepsia should avoid high-fat foods, sugar, caffeine, spices, and alcohol.



Summary

- n Upper GI disorders—*H. pylori* plays an important role
- n Maintain individual tolerances as much as possible.

n Taken from Krause's



Medical Nutrition Therapy for Disorders of the Lower Gastrointestinal Tract



Common Intestinal Problems

- n Intestinal gas or flatulence
- n Constipation
- n Diarrhea
- n Steatorrhea





Constipation

- n Defined as hard stools, straining with defecation, infrequent bowel movements
- n Normal frequency ranges from one stool q 3 days to 3 times a day
- n Occurs in 5% to more than 25% of the population, depending on how defined



Causes of Constipation - Systemic

- Side effect of medication, esp narcotics
- Metabolic Endocrine abnormalities, such as hypothyroidism, uremia and hypercalcemia
- Lack of exercise
- Ignoring the urge to defecate
- Vascular disease of the large bowel
- Systemic neuromuscular disease leading to deficiency of voluntary muscles
- Poor diet, low in fiber
- Pregnancy



Causes of Constipation - Gastrointestinal

- Diseases of the upper gastrointestinal tract
 - Celiac Disease
 - Duodenal ulcer
 - Gastric cancer
 - Cystic fibrosis
- Diseases of the large bowel resulting in:
 - Failure of propulsion along the colon (colonic inertia)
 - Failure of passage through anorectal structures (outlet obstruction)
- Irritable bowel syndrome
- Anal fissures or hemorrhoids
- Laxative abuse



Diagnostic Tests Constipation

Begins with a physical exam including a digital rectal exam. Other tests can include the following:

- Thyroid tests
- Barium enema x-ray: colonic contrast study
- Sigmoidoscopy
- Colonoscopy
- Colorectal transit study
- Anorectal manometry tests to measure anal sphincter muscle tone and contraction.
- Evacuation proctography



Treatment of Constipation

- n Encourage physical activity as possible
- n Bowel training: encourage patient to respond to urge to defecate
- n Change drug regimen if possible if it is contributory
- n Use laxatives and stool softeners judiciously
- n Use stool bulking agents such as psyllium (Metamucil) and pectin



Food History / Recall / Frequency

A complete food history and 24-hour recall should be completed. Specific areas of concern should include the following:

- Number of daily servings from grains, fruits, vegetables, nuts, and legumes
- Caffeine intake
- Fluid intake
- Evaluation of exercise and activity patterns



To quickly estimate fiber intake from a food record (Marlett, 1997):

- n Multiply number of servings of fruits and vegetables by 1.5 g
- n Multiply number of servings of whole grains by 2.5 g
- n Multiply number of servings of refined grains by 1.0 g
- n Add specific fiber amounts for nuts, legumes, seeds, and high-fiber cereals
- n Total = estimated fiber intake



MNT for Constipation

- Depends on cause
- Use high fiber or high residue diet as appropriate
- If caused by medication, may be refractory to diet treatment





Nutrition Intervention for Constipation

- Eat adequate insoluble fiber (gradually increasing daily total fiber to 25-38 g/day)
- The major sources of insoluble fiber include cellulose, psyllium, inulin, and oligosaccharides. These types of fiber are primarily found in the skins of fruits, vegetables, wheat and rice bran, and whole wheat.
- Increase fluid intake to a minimum of 64 oz each day.
- Participate in daily physical activity.
- Use bulk-forming agents such as Psyllium, Calcium polycarbophil, or Methylcellulose.
- Avoid stool retention and initiate bowel retraining program if required



Fiber, roughage, and residue

- Fiber or roughage
 - From plant foods
 - Not digestible by human enzymes
- Residue
 - Fecal contents, including bacteria and the net remains after ingestion of food, secretions into the GI tract, and absorption



High-Fiber Diets

- n Most Americans = 10 – 15 g/day
- n Recommended = 25 g/day
- n More than 50g/day = no added benefit, may cause problems





High-Fiber Diet

- Increase consumption of whole-grain breads, cereals, flours, other whole-grain products
- Increase consumption of vegetables, especially legumes, and fruits, edible skins, seeds, hulls
- Consume high-fiber cereals, granolas, legumes to increase fiber to 25 g/day
- Increase consumption of water to at least 2 qts (eight 8 oz cups)



High-Fiber Diets: cautions

- Gastric obstruction, fecal impaction may occur when insufficient fluid consumed
- With GI strictures, motility problems, increase fiber slowly (~1mo.)
- Unpleasant side effects
 - Increased flatulence
 - Borborygmus
 - Cramps, diarrhea



Diarrhea

- n Characterized by frequent evacuation of liquid stools
- n Accompanied by loss of fluid and electrolytes, especially sodium and potassium
- n Occurs when there is excessively rapid transit of intestinal contents through the small intestine, decreased absorption of fluids, increased secretion of fluids into the GI tract



Diarrhea Etiology

- Inflammatory disease
- Infections with fungal, bacterial, or viral agents
- Medications (antibiotics, elixirs)
- Overconsumption of sugars
- Insufficient or damaged mucosal absorptive surface
- Malnutrition
- Should identify and treat the underlying problem



Diagnositics in Diarrhea

Stool cultures:

- Fecal fat: qualitative and quantitative to rule out fat malabsorption
- Occult blood
- Ova and paracytes
- Bacterial contamination (*Clostridium difficile*, foodborne illnesses, etc.)
- Osmolality and electrolyte composition



Diarrhea Diagnostics

Intestinal Structure and Function

- Sigmoidoscopy
- Colonoscopy

Evaluation of hydration status and electrolyte balance:

- Serum electrolytes, serum osmolality
- Urinalysis
- Physical examination
- Current weight, Usual weight, % weight change



Diarrhea Nutritional Care Adults

Restore normal fluid, electrolyte, and acid-base balance.

- Use oral rehydration solutions such as Pedialyte, Resol, Ricelyte, and Rehydralyte
- The World Health Organization has a standard recipe for an oral rehydration solution: $\frac{1}{3}$ - $\frac{2}{3}$ tsp table salt, $\frac{3}{4}$ tsp sodium bicarbonate, $\frac{1}{3}$ tsp potassium chloride, $1\frac{1}{3}$ Tbsp. sugar, 1 liter bottled or sterile water.



Nutritional Intervention Diarrhea

- Decrease gastrointestinal motility
- Avoid clear liquids and other foods high in simple carbohydrates (i.e., lactose, sucrose, or fructose) and sugar alcohols (sorbitol, xylitol, or mannitol)
- Avoid caffeine-containing products
- Avoid alcoholic beverages
- Avoid high-fiber and gas-producing foods such as nuts, beans, corn, broccoli, cauliflower, or cabbage



Nutrition Intervention Diarrhea

- n Stimulate the gastrointestinal tract by slow introduction of solid food without exacerbation of symptoms
- n Low-residue, low-fat, lactose-free nutrition therapy should guide initial food selections
- n If there is no evidence of lactose intolerance, then these foods should be added back to the meal plan (Steffen, 2004).



Diarrhea Treatment for Adults

Repopulate the GI tract with microorganisms

- Prebiotics in modest amounts including pectin, oligosaccharides, inulin, oats, banana flakes
- Probiotics, cultured foods and supplements that are sources of beneficial gut flora



Low- or Minimum Residue Diet

- Foods completely digested, well absorbed
- Foods that do not increase GI secretions
- Used in:
 - Maldigestion
 - Malabsorption
 - Diarrhea
 - Temporarily after some surgeries, e.g. hemorrhoidectomy



Foods to Limit in a Low- or Minimum Residue Diet

- Lactose (in lactose malabsorbers)
- Fiber >20 g/day
- Resistant starches
 - Raffinose, stachyose in legumes
- Sorbitol, mannitol, xylitol >10g/day
- Caffeine
- Alcohol, esp. wine, beer



Restricted-Fiber Diets

- Uses:
 - When reduced fecal output is necessary
 - When GI tract is restricted or obstructed
 - When reduced fecal residue is desired



Restricted-Fiber Diets

- Restricts fruits, vegetables, coarse grains
- <10 g fiber/day
- Phytobezoars
 - Obstructions in stomach resulting from ingestion of plant foods
 - Common in edentulous pts, poor dentition, with dentures
 - Potato skins, oranges, grapefruit



RECOMMENDATION FOR IRRITABLE BOWEL SYNDROME

1. In case of acute irritable colon, an elemental diet may be necessary
2. Progress to soft, bland diet when tolerated
3. Progress to high fiber diet slowly to avoid discomforts such as bloating and flatulence
4. Foods to avoid: alcohol, black pepper, caffeine, chili, cocoa, chocolate, coffee, cola, garlic, spicy food, sugar (fructose, lactose), sorbitol
5. Avoid gas producing foods (apples, artichoke, asparagus, avocados, barley, beer, bran, brussel sprouts, cabagge, caulie flower, soda, celery, coconut, cucumbers, eggplant, high fat meals, pastries, onions, wheat, soybean, yeast, nuts)



RECOMMENDATION FOR IRRITABLE BOWEL SYNDROME

1. Avoid lactose if not tolerated
2. Avoid wheat or yeast if not tolerated
3. Common food allergies
4. Avoid excess fat
5. Drink 2 – 3 quarts of water (consume 20 – 30 g fiber daily)
6. 1 tbs bulking agent (Metamucil)
7. Probiotics
8. Supplement (B complex, vit D, riboflavin)



MNT for Infants and Children

- Acute diarrhea most dangerous in infants and children
- Aggressive replacement of fluid/electrolytes
- WHO/AAP recommend 2% glucose (20g/L) 45-90 mEq sodium, 20 mEq/L potassium, citrate base
- Newer solutions (Pedialyte, Infalyte, Lytren, Equalyte, Rehydralyte) contain less glucose and less salt, available without prescription



MNT for Infants and Children

- Continue a liquid or semisolid diet during bouts of acute diarrhea for children 9 to 20 months
- Intestine absorbs up to 60% of food even during diarrhea
- Early refeeding helpful; gut rest harmful
- Clear liquid diet (hyperosmolar, high in sugar) is inappropriate
- Access American Academy of Pediatrics Clinical Guidelines
<http://aappolicy.aappublications.org/cgi/reprint/pediatrics;97/3/424.pdf>



Nutrition Intervention Diarrhea in Children

- Thicken consistency of the stool
- Banana flakes, apple powder, or other pectin sources can be added to infant formula
- If the infant has begun solid foods, use of strained bananas, applesauce, and rice cereal are the best initial food choices
- AAP no longer recommends the BRAT diet (bananas, rice, applesauce, and toast) for diarrhea in children



Lactase “Deficiency”

- n 70% of adults worldwide are lactase deficient, especially Africans, South Americans, and Asians
- n Maintenance of lactase into adulthood is probably the result of a genetic mutation
- n Diagnosed based on history of GI intolerance to dairy products



Lactose Intolerance Diagnostics

Lactose breath hydrogen test

- Baseline breath hydrogen concentration is measured.
- Patient consumes 25 to 50 grams lactose.
- Breath hydrogen concentration is re-measured in 3 to 8 hours. An increase >20 ppm suggests lactose malabsorption (90% sensitivity).



Lactose Deficiency Diagnostics

Lactose tolerance test

- After 8-hour fast, baseline serum glucose is measured.
- Patient consumes 50-100 grams of lactose
- Serum blood glucose levels are measured at 30, 60, and 90 minutes after lactose ingestion
- No increase in blood glucose levels suggests lactose malabsorption (Pagana, 2004).



MNT for Lactose Intolerance

- n Most lactase deficient individuals can tolerate small amounts of lactose without symptoms, particularly with meals or as cultured products (yogurt or cheese)
- n Can use lactase enzyme or lactase treated foods, e.g. Lactaid milk
- n Distinct from milk protein allergy; allergy requires milk free diet



MNT Strategies for Lactose Intolerance

- Start with small amounts of lactose containing foods ($\frac{1}{4}$ cup of milk or $\frac{1}{2}$ ounce of cheese)
- Start with foods lower in lactose content (see table)
- Only include 1 dairy food a day and gradually increase the amount as the days go by*
- Only eat 1 lactose-containing food/meal
- Drink milk or eat dairy foods with a meal or a snack, but not alone
- Space lactose-containing foods several hours apart
- If drinking milk, heating the milk may improve tolerance

A vertical image on the left side of the slide shows a close-up of several ears of corn. The husks are partially removed, revealing the golden-yellow kernels. The background is a soft, out-of-focus gradient of yellow and green.

MNT Strategies for Lactose Intolerance

- Try lactose-free or lactose-reduced milk
- Use lactase enzyme drops if you are drinking milk , however, they must be added at least 24 hours before drinking the milk or take lactase tablets before eating dairy foods
- Aged cheeses that are naturally lower in lactose than a processed cheese, such as Velveeta or cheese spread
- Yogurt, which contains bacteria that break down the lactose may be easier to digest
- Buttermilk may also be easier to tolerate as it is a fermented dairy food
- *A good strategy is to add in the equivalent of a maximum of 2-5 grams of lactose at a time.



Lower GI Disorders Summary

- n Food intolerances should be dealt with individually
- n Patients should be encouraged to follow the least restrictive diet possible
- n Patients should be re-evaluated frequently and the diet advanced as appropriate



Chapter 31

**Medical Nutrition
Therapy for
Liver, Biliary
System, and
Exocrine
Pancreas
Disorders**



Common Laboratory Tests Used to Test for Liver Function

- Hepatic excretion
 - Total serum bilirubin, urine bilirubin
- Cholestasis tests
 - Serum alkaline phosphatase
- Hepatic enzymes
 - ALT, AST



Common Laboratory Tests Used to Test for Liver Function—cont'd

- Serum proteins
 - PT, PTT, serum albumin
- Markers of specific liver diseases
 - Serum ferritin, ceruloplasmin
- Specific tests for viral hepatitis
 - IgM anti-HAV, anti-HBS, HCV-RNA



LIVER FUCTION TEST

FUNCTION	TEST	DERANGEMENT
Bile synthesis	bilirubin	Bilirubin not excreted in feces, indirect bilirubin high, direct bilirubin high in biliary tract disease
detoxification	bromsulphalein	Retention high and urinary excretion, low in liver disease
Carb metab	OGTT, blood glucoe	Normal until advanced disease, low in acute disease, high in chronic disease
Lipid metab	TG, lipoprotein/cholesterol, ketones	All low in severe disease
Protein metab	Urea (BUN, NH ₃)	BUN low, NH ₃ high in advance disease
	Plasma protein	Low protein levels. AA ratio skewed toward higher AAA
	Enzymes	high



Diseases of the Liver

- n Acute viral hepatitis
- n Fulminant hepatitis
- n Chronic hepatitis
- n Alcoholic liver disease, alcoholic hepatitis, and cirrhosis



Diseases of the Liver—cont'd

- Cholestatic liver diseases
 - Primary biliary cirrhosis
 - Sclerosing cholangitis
- Inherited disorders
- Other liver diseases



Acute Viral Hepatitis

- Widespread inflammation of the liver that is caused by hepatitis viruses A, B, C, D, and E
- Four phases of symptoms:
 1. Prodromal phase
 2. Preicteric phase
 3. Icteric phase
 4. Convalescent phase



Fulminant Hepatitis

- n Syndrome in which severe liver dysfunction is accompanied by hepatic encephalopathy



Chronic Hepatitis

- n At least 6-month course of hepatitis or biochemical and clinical evidence of liver disease with confirmatory biopsy findings of unresolving hepatic inflammation



DIETARY RECOMMENDATION FOR HEPATITIS

- n High energy (30 – 35 kkal/kg BBI)
- n High protein (1 – 1,2 g/kgBBI)
- n Carbs (50 – 55% TE)
- n Supplement: B comp, vit C, K, and zinc
- n Adequate liquids



Alcoholic Liver Disease


- n Disease resulting from excessive alcohol ingestion characterized by fatty liver (hepatic steatosis), hepatitis, or cirrhosis



Alcoholic Liver Disease

Metabolic Changes


- n Steatorrhea
- n Wernicke-Korsakoff syndrome
- n Peripheral neuropathy
- n Pellagrous psychosis
- n Folate deficiency



End-Stage Alcoholic Liver Disease

Possible Characteristics

- n Malnutrition
- n Portal hypertension with varices
- n Ascites
- n Hyponatremia
- n Hepatic encephalopathy
- n Glucose alterations



End-Stage Alcoholic Liver Disease

Possible Characteristics—cont'd

- n Fat malabsorption
- n Osteopenia
- n Thrombocytopenia with anemia



Cholestatic Liver Diseases

- Primary biliary cirrhosis (PBC)
 - An immune-mediated chronic cirrhosis of the liver due to obstruction or infection of the small and intermediate-sized intrahepatic bile ducts, whereas the extrahepatic biliary tree and larger intrahepatic ducts are normal
 - 90% of patients are women



Cholestatic Liver Diseases—cont'd

- Sclerosing cholangitis
 - Fibrosing inflammation of segments of extrahepatic bile ducts, with or without involvement of intrahepatic ducts



Inherited Disorders

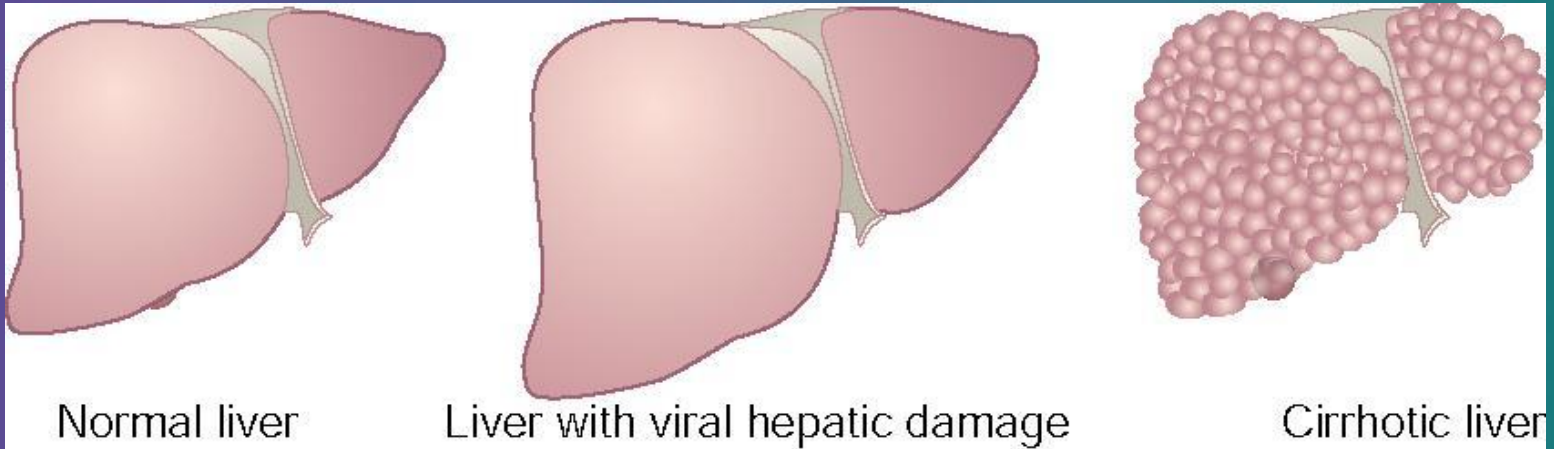
- Hemochromatosis
 - Inherited disease of iron overload
- Wilson's disease
 - Autosomal recessive disorder associated with impaired biliary copper excretion
- α_1 -antitrypsin deficiency
 - Causes cholestasis or cirrhosis and can cause liver and lung cancer



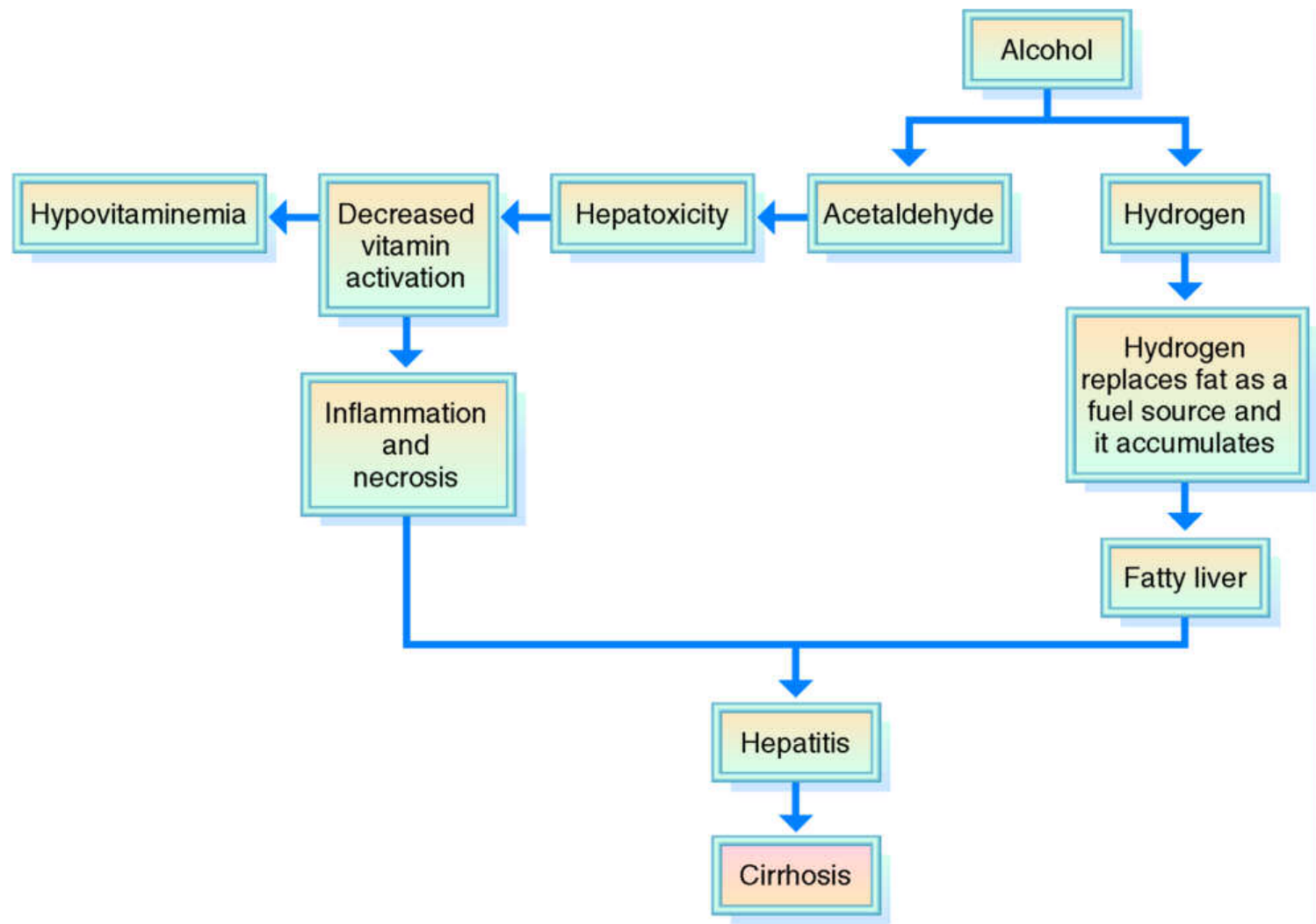
Other Liver Diseases

- Several other causes of liver disease, including
 - Liver tumors
 - Systemic diseases (rheumatoid arthritis, systemic sclerosis)
 - Nonalcoholic steatohepatitis
 - Acute ischemic and chronic congestive hepatopathy
 - Parasitic, bacterial, fungal, and granulomatous liver diseases

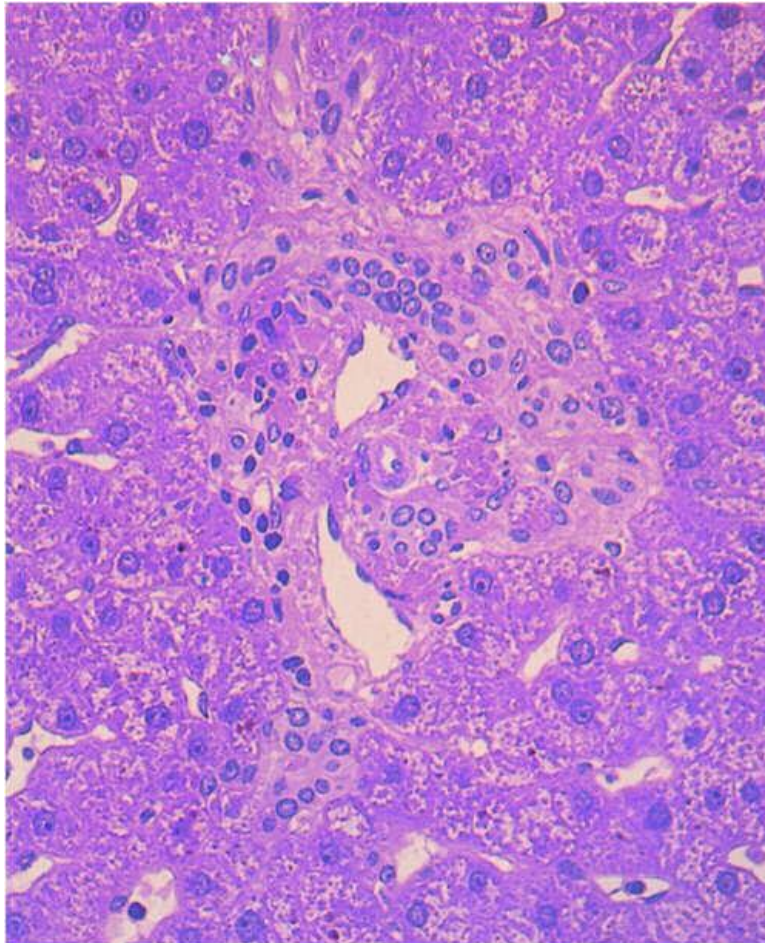
Normal Liver vs. Damaged Liver



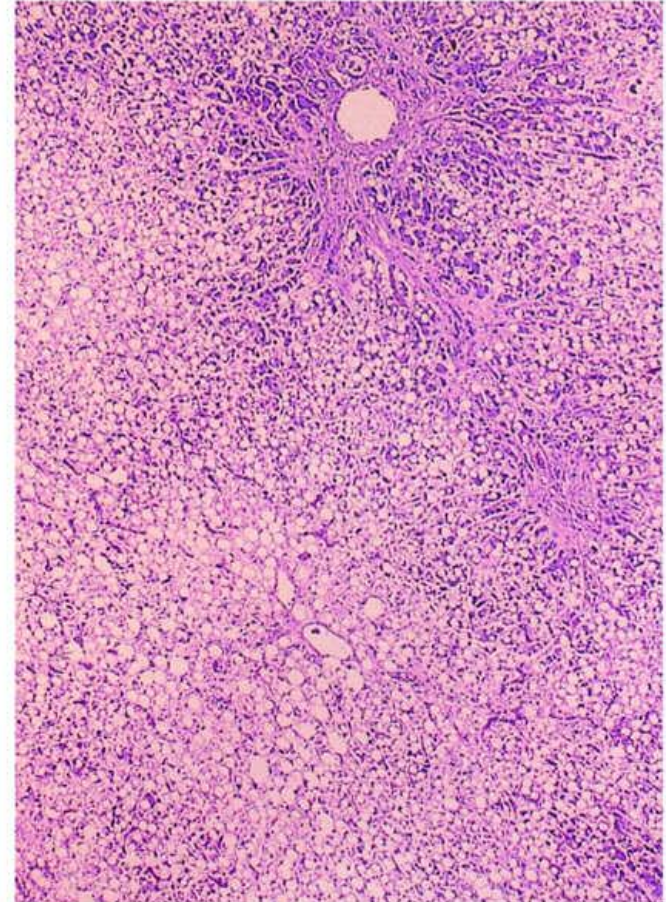
Complications of Excessive Alcohol Consumption Stem Largely from Excess Hydrogen and from Acetaldehyde



Microscopic Appearance of (A) a Normal Liver and (B) Acute Fatty Liver



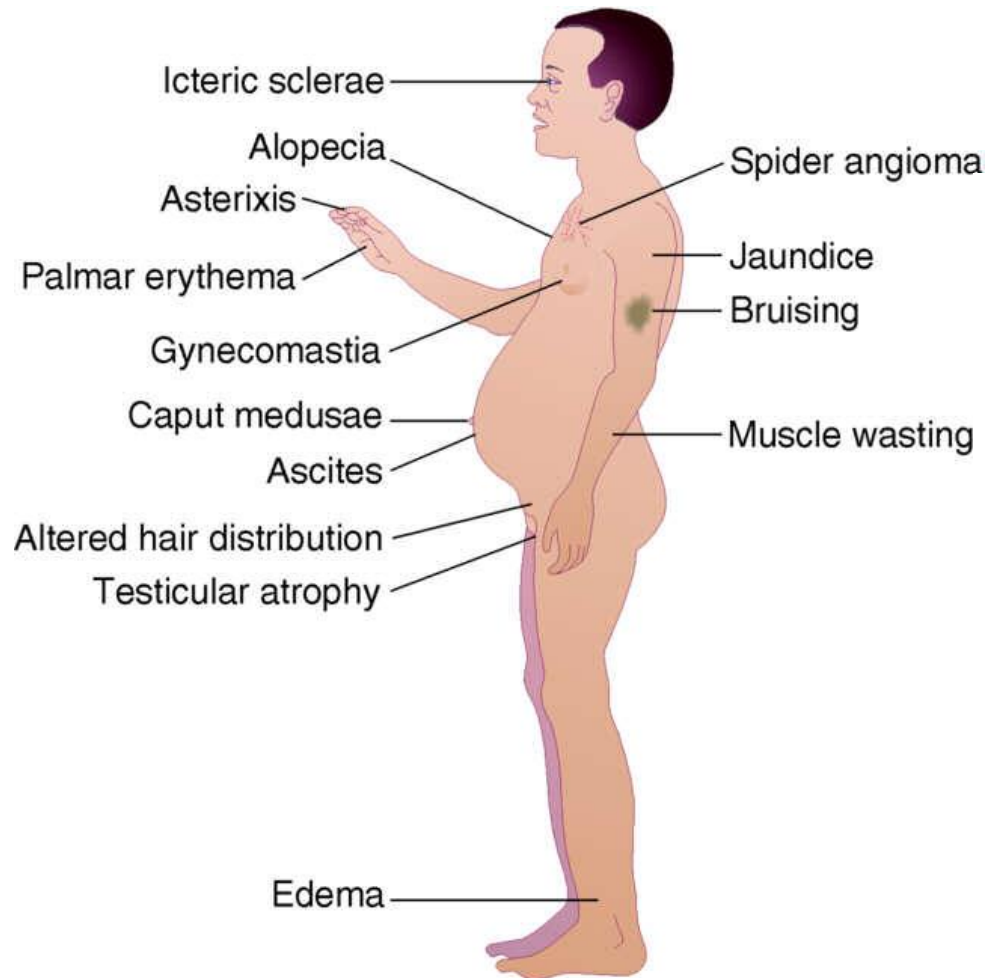
A



B


(From Kanel G, Korula J. Atlas of Liver Pathology. W.B. Saunders, 1992.)

Clinical Manifestations of Cirrhosis



EXTERNAL SYMPTOMS

Factors That Affect Interpretation of Objective Nutrition Assessment Tests in Patients with End-Stage Renal Disease

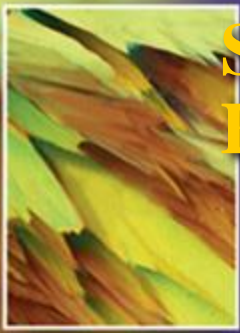
- 
- n Body weight
 - 3-methylhistidine excretion
 - n Anthropometric measurements
 - Visceral protein levels
 - n Creatinine-height index
 - Immune function tests
 - n Nitrogen balance studies



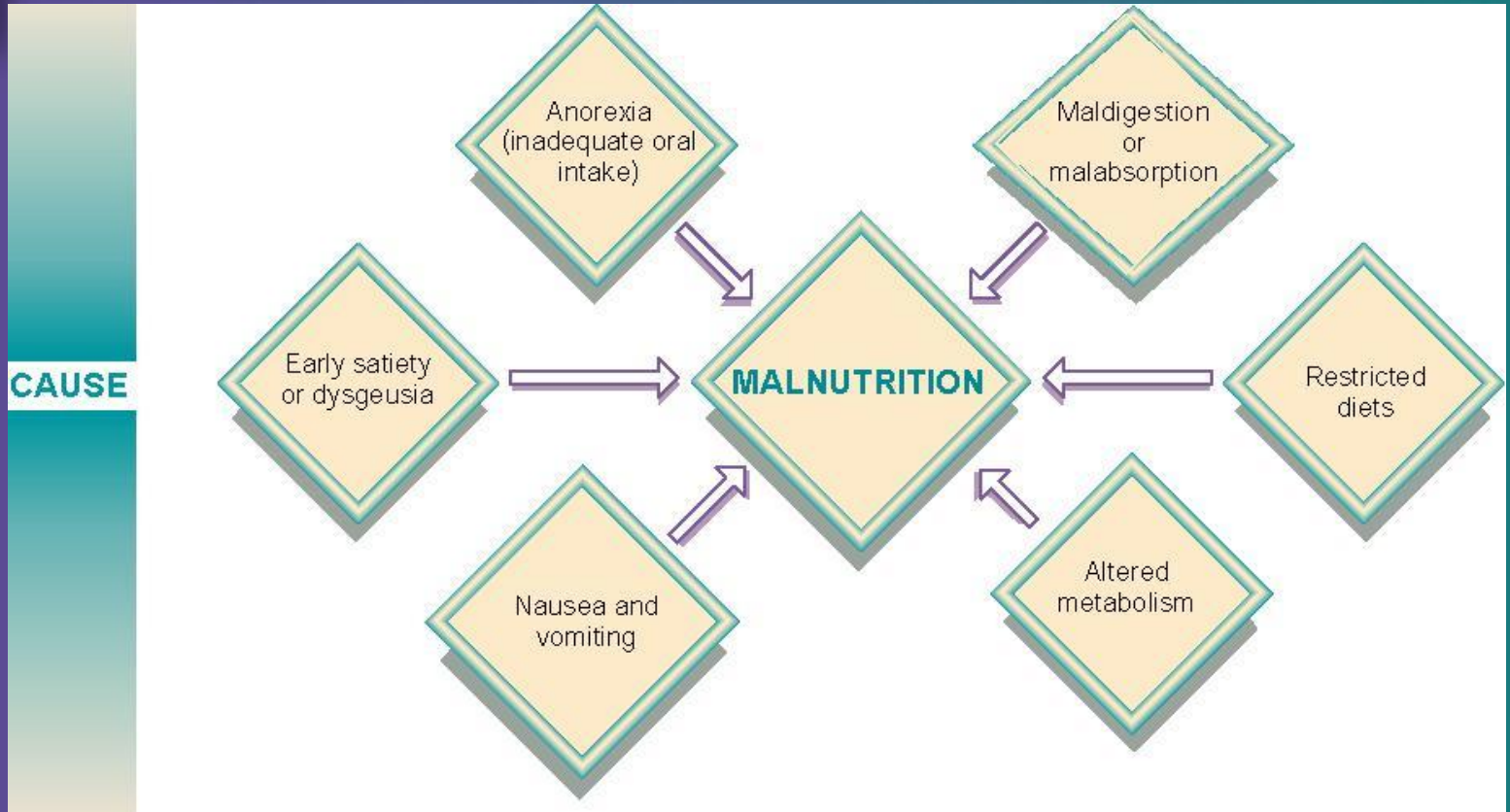
SGA Parameters for Nutritional Evaluation of Liver Transplant Candidates

- n History
- n Physical
- n Existing conditions
- n Nutritional rating (based on results of above parameters)

Severe Malnutrition and Ascites in a Man with End-Stage Renal Disease



Malnutrition in Liver Disease—Cause



Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Jeanette M. Hasse and Laura E. Matarese, 2002.

Malnutrition in Liver Disease—Pathophysiology

PATHOPHYSIOLOGY

NUTRITION ASSESSMENT

Serial monitoring of body weight and anthropometry

Dietary intake

Subjective global assessment

POSSIBLE CLINICAL FINDINGS

Abnormal liver function tests

Jaundice

Ascites and edema

Hepatic encephalopathy

Portal hypertension and varices

Altered amino acid levels

Vitamin/mineral deficits

Glucose intolerance or fasting hypoglycemia

Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Jeanette M. Hasse and Laura E. Matarese, 2002.



Malnutrition in Liver Disease—Medical and Nutritional Management

MEDICAL MANAGEMENT

- Diuretic therapy
- Medication for encephalopathy (e.g., lactulose, neomycin)
- Management of portal hypertensive bleeding (e.g., pharmacologic therapy, shunts, banding)
- Monitoring of blood glucose

NUTRITIONAL MANAGEMENT

- Increased energy intake via small, frequent meals
- Sodium restriction for fluid retention
- Fluid restriction for hyponatremia
- Carbohydrate-controlled diets for hyperglycemia
- Vitamin and mineral supplements
- Oral liquid supplements or enteral (tube) feeding (consider BCAA formulas for encephalopathy)

Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Jeanette M. Hasse and Laura E. Matarese, 2002.



Vitamin/Mineral Deficits in Severe Hepatic Failure

- Vitamin A
- Vitamin D
- Vitamin E
- Vitamin K
- Vitamin B₆
- Vitamin B₁₂
- Folate
- Niacin
- Thiamin
- Zinc
- Magnesium
- Iron
- Potassium
- Phosphorus



Four Stages of Hepatic Encephalopathy

Stage Symptom

- I** Mild confusion, agitation, irritability, sleep disturbance, decreased attention
- II** Lethargy, disorientation, inappropriate behavior, drowsiness
- III** Somnolence but arousable, incomprehensible speech, confusion, aggression when awake
- IV** Coma



End-Stage Liver Disease

Hepatic Encephalopathy

1. Consider major causes of encephalopathy

- GI bleeding
- Fluid and electrolyte abnormalities
- Uremia
- Use of sedatives
- Hypo- or hyperglycemia
- Alcohol withdrawal
- Constipation
- Acidosis



End-Stage Liver Disease

Hepatic Encephalopathy—cont'd

2. Treat underlying cause.
3. Treat with medications.
 - Lactulose
 - Neomycin
4. Ensure adequate diet is consumed.



End-Stage Liver Disease

- *Energy*: 30 to 35 kcal/kg dry weight

BEE x 1.2 to 1.5, depending on degree of malnutrition

- *Fat*: 25% to 40% of kcal

May try MCT if steatorrhea is present; with severe case, try fat restriction and discontinue if diarrhea does not improve

- *Protein*: 1 to 1.5 g/kg dry wt depending on degree of malnutrition, malabsorption, metabolic stress



End-Stage Liver Disease—cont'd

- May try BCAA formulas for >grade 2 encephalopathy
- CHO: high intake of both complex and simple carbohydrates
- Vitamin and mineral supplements
- Electrolytes: restrict sodium with edema or ascites (2-4 g/day)
- Fluid: restrict fluid if hyponatremia is present



Amino Acids Commonly Altered in Liver Disease

- Aromatic amino acids—serum levels increased
 - Tyrosine
 - Phenylalanine*
 - Free tryptophan*
- Branched-chain amino acids—serum levels decreased
 - Valine*
 - Leucine*
 - Isoleucine*
- Other amino acids—serum levels increased
 - Methionine*
 - Glutamine
 - Asparagine
 - Histidine*

* Denotes essential amino acids



Medications Commonly Used after Liver Transplantation

- n Azathioprine
- n Antithymocyte globulin
- n Basiliximab
- n Cyclosporine
- n Daclizumab
- n Glucocorticoids
- Muromonab-CD3
- Mycophenolate mofetil
- Sirolimus
- Tacrolimus
- 15-deoxysperagualin



Nutrition Care Guidelines for Liver Transplantation

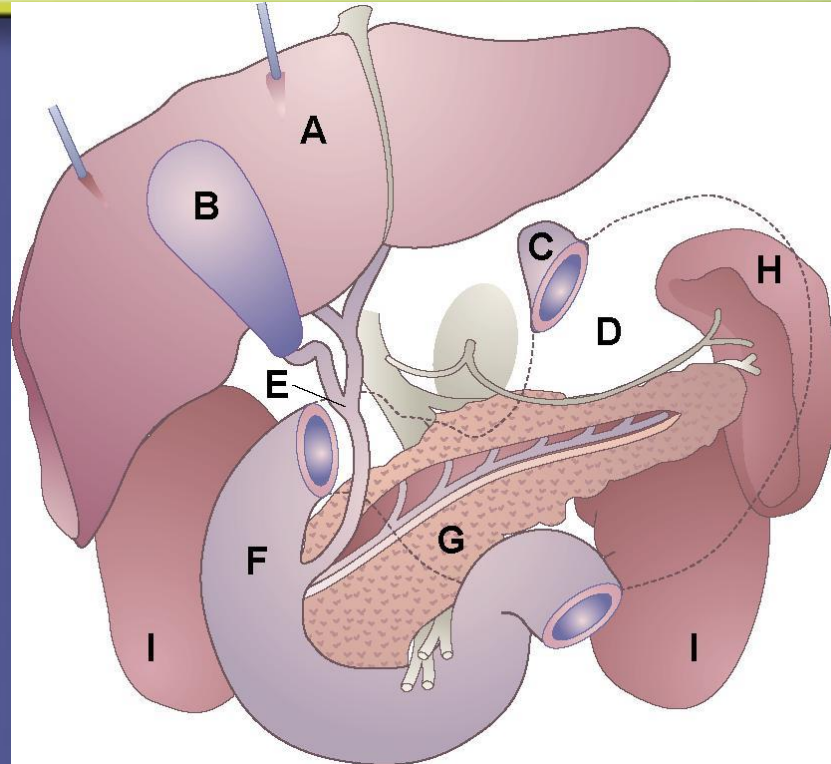
- n Pretransplantation
 - Calories
 - Protein
 - Fat
 - Carbohydrate
 - Sodium
 - Fluid
 - Calcium
 - Vitamins
- n Immediate posttransplantation
- n Long-term posttransplantation



Liver Transplantation—Diet

- Nutrition support: pre- and posttransplant
- Long-term preventive nutrition to optimize health and to avoid or minimize
 - Excessive weight gain
 - Hyperlipidemia
 - Hyperglycemia
 - Hypertension
 - Osteopenia

Relationship of Organs of the Upper Abdomen



A, Liver (retracted upward); **B**, gallbladder; **C**, esophageal opening of the stomach; **D**, stomach (shown in dotted outline); **E**, common bile duct; **F**, duodenum; **G**, pancreas and pancreatic duct; **H**, spleen; **I**, kidneys.

Courtesy The Cleveland Clinic Foundation, Cleveland, Ohio, 2002.



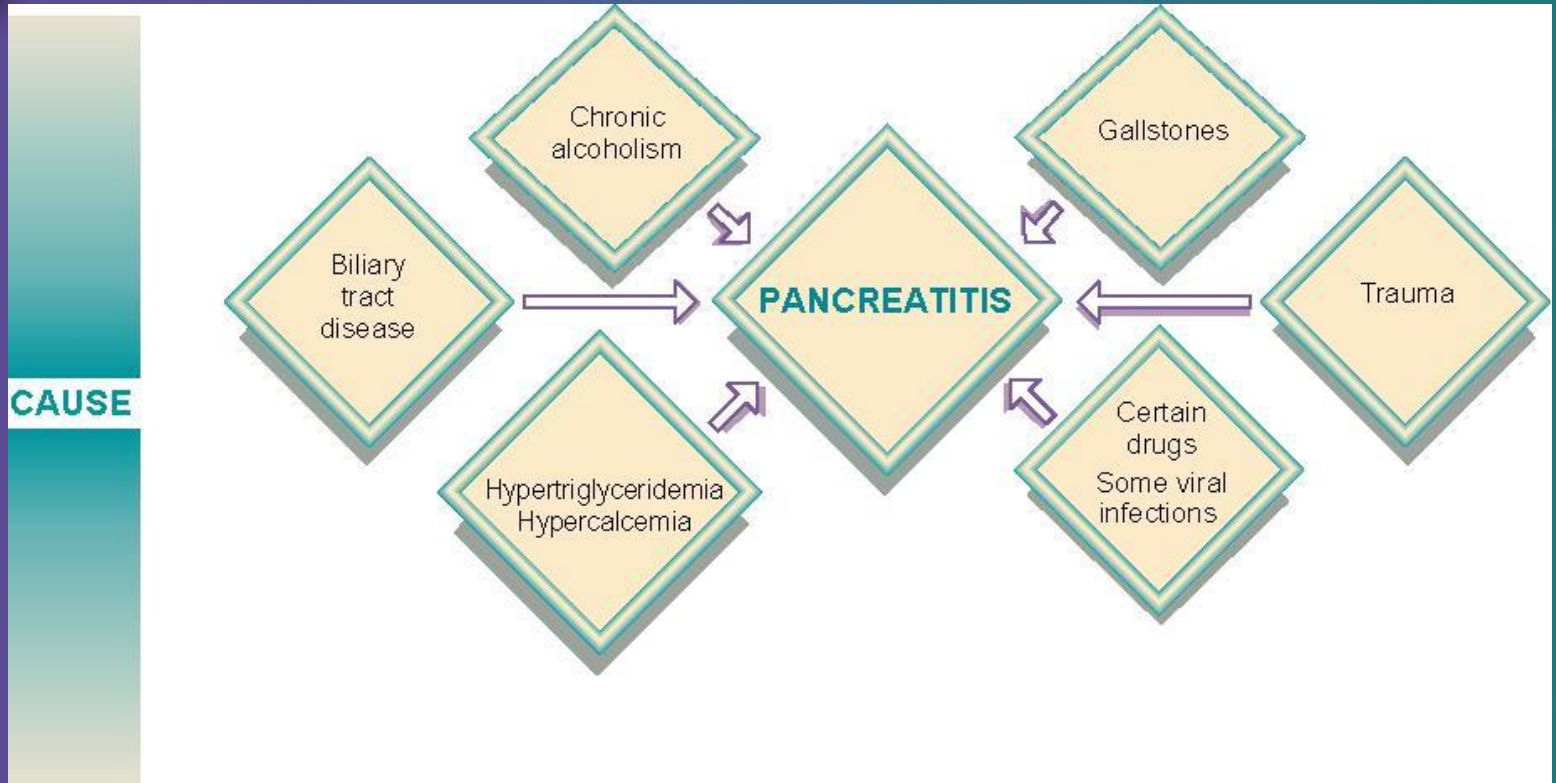
Some Tests of Pancreatic Function

TABLE 31-8

Some Tests of Pancreatic Function

TEST	SIGNIFICANCE
Secretin stimulation test	Measures pancreatic secretion, particularly bicarbonate, in response to secretin stimulation
Glucose tolerance test	Assesses endocrine function of the pancreas by measuring insulin response to a glucose load
72-hr stool fat test	Assesses exocrine function of the pancreas by measuring fat absorption that reflects pancreatic lipase secretion

Pancreatic Disorders—Cause



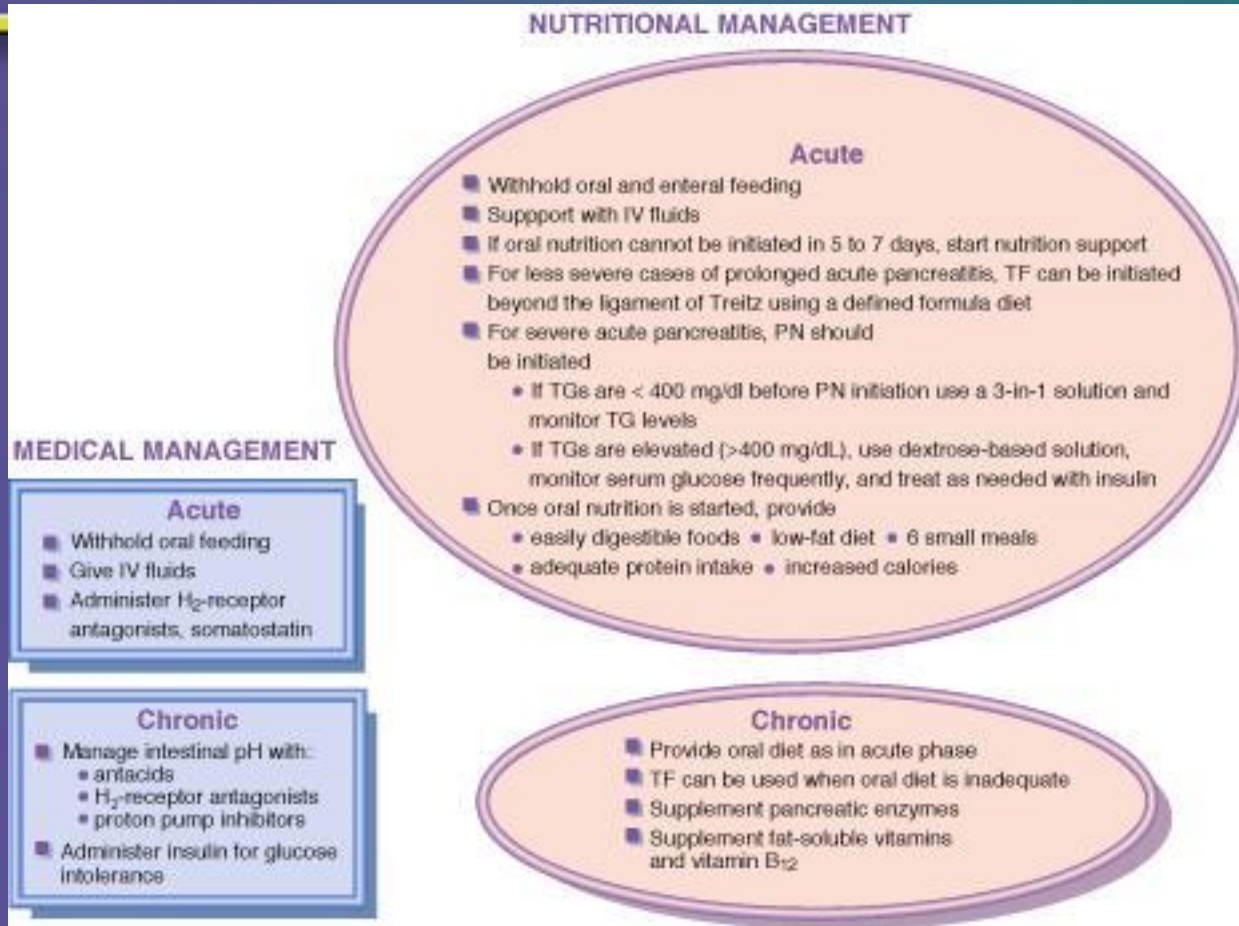
Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000.

Pancreatic Disorders—Pathophysiology



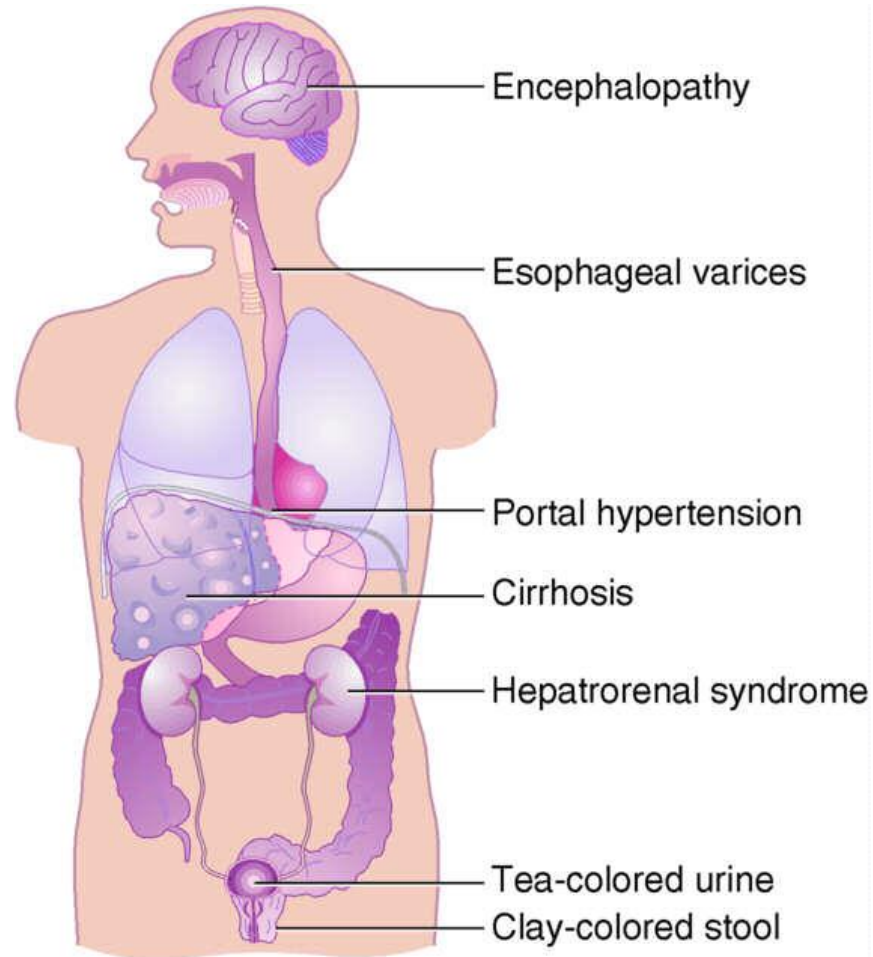
Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000.

Pancreatic Disorders—Medical and Nutritional Management



Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Jeanette M. Hasse and Laura E. Matarese, 2002.

Clinical Manifestations of Cirrhosis



INTERNAL SYMPTOMS



Summary

- n Liver disorders—role of liver is so crucial to overall health, its destruction is quite serious
- n Goals—support maintenance of as much normal liver function as possible
- n Transplantation, if needed