Colorectal transit study	Constipation Severity Index	H & H
Anorectal function test	**Lab Work**	Serum Fe, ferritin
Colonoscopy or sigmoidoscopy	Alb, transthyretin	Gluc
	BUN, Creat	Na^+, K^+
		Ca^{++}, Mg^{++}
		Stool guaiac

INTERVENTION

OBJECTIVES

- **Atonic constipation ("lazy bowel"):** Stimulate peristalsis, provide bulk, and retain water in the feces.
- **Spastic constipation:** Undue distention and stimulation of the bowel should be prevented during exacerbations. After the patient is well, fiber should be increased.
- **TF constipation:** Check for obstruction (nausea, vomiting, distention, and dehydration). Record I & O, along with activity levels.
- **Pediatric constipation and encopresis:** Provide laxatives and lubricants initially, followed by improved fiber and fluid intakes.

SAMPLE NUTRITION CARE PROCESS STEPS

Altered GI Function—Constipation

Assessment Data: Fluid intake records (I & O), medication history and recent changes, stool patterns and frequency. No food allergies or intolerances noted. Limited fluid intake recently.

Nutrition Diagnosis (PES): Altered GI function related to very low fluid intake and use of constipating medications as evidenced by patient complaints of hard, dry, infrequent stools, low intake of fluids, and poor nutritional quality of life.

Intervention: Food-Nutrient Delivery: Incorporate more fluids into meals and nourishments, discussion of fluid tracking with staff or family as well as patient. Offer hot or caffeinated beverages, such as coffee or hot tea. Try prune juice mixed with bran and applesauce, especially for the elderly.

Education: Discuss role of fiber, fluid and physical activity in maintenance of normal bowel activity.

Counseling: Discuss food sources and ways to increase fiber content in recipes and meals.

Coordination of Care: Discuss need for stool softener with doctor or nursing staff. Avoid mineral oil that interferes with nutrient absorption.

Monitoring and Evaluation: Fluid intake records, stool patterns and frequency records. Review medication changes or addition of stool softener. Note fewer complaints of incidents of hard, dry, and infrequent stools or straining. Note more GI comfort and improved nutritional quality of life.

FOOD AND NUTRITION

- In general, it may be helpful to consume more fruits and vegetables and more servings from the bread/cereal group each day, especially whole grains, root vegetables such as carrots or potatoes, stewed dried fruit, and cabbage. Gradually increase fiber, maintain an adequate fluid intake, and exercise regularly.
- **Atonic constipation:** The diet should contain 20–35 g of fiber, with liberal use of whole grains, fruits, and vegetables. Adding a few carrots and whole-grain breads and cereal may be an easy solution. Use adequate fluid (30–35 mL/Kg).
- **Spastic constipation:** Decrease fiber during painful episodes. Then, increase use of prune juice, dried fruits, raw fruits and vegetables, nuts, and whole grains. If wheat allergy is a concern for a patient, do not promote use of bran.
- **TF constipation:** Use a fiber-containing formula if appropriate. Use adequate flushes.
- **Pediatric constipation and encopresis:** Add more fruits and vegetables to the diet; increase whole-grain fiber and fluid intake.

Common Drugs Used and Potential Side Effects

- There is limited evidence about efficacy of treatments used for constipation. Good evidence supports the use of polyethylene glycol and tegaserod. Moderate evidence supports the use of psyllium and lactulose. Limited-quality data exist for milk of magnesia, senna, bisacodyl, and stool softeners (Kamm et al, 2005; Ramkumar and Rao, 2005).
- Discourage overuse or dependence on laxatives and cathartics; see Table 7-10.
- Prescription medications that may cause constipation include opiates, anticholinergics, tricyclic antidepressants, calcium channel blockers, antiparkinsonian drugs, antipsychotics, diuretics and antihistamines.
- Over the counter drugs that can cause constipation include calcium-containing antacids, calcium supplements, iron supplements, NSAIDs, and antidiarrheal agents.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Flax, aloe (anthraquinones), fenugreek, and rhubarb have been recommended for constipation, but no clinical trials have proven efficacy.
- With use of bisacodyl, avoid aloe, cascara, senna, and yellow dock because of enhancing effects.
- A herbal tea (Smooth Move) increases bowel movements naturally (Bub et al, 2006).
- More studies on probiotic products are needed, but they have possibility.

TABLE 7-10 Medications for Constipation

Medication	Description
Bulking agents Psyllium (Metamucil, Effersyllium, Perdiem Fiber), Benefiber (with guar gum), methylcellulose (Citrucel), calcium polycarbophil (FiberCon), Fiberall, Fiber-Lax,Equilactin, Konsyl, Serutan	Fiber supplements that retain water and are safe choices. Take with plenty of water or juice (8 oz per teaspoonful). Results may require 1–4 teaspoons of the product.
Chloride channel activators Lubiprostone (Amitiza)	Activators increase intestinal motility and fluid to ease passage of stool. Not recommended for use longer than 12 months without doctor's evaluation.
Lubricant laxatives (Fleet Mineral Oil, Zymenol)	Lubricant laxatives coat the intestinal lining to allow easier passage of stool but may also interfere with absorption of calcium and fat-soluble vitamins.
Osmotic or Hyperosmolar laxatives (Cephulac, Fleet Phospho-Soda, Milk of Magnesia or MOM, Kristalose) or Lactulose (Chronulac) or Polyethylene Glycol 3350 (Miralax, Glycolax)	Osmoticsand saline laxatives draw fluid, cause bowel distention, and may be useful for idiopathic constipation. "The wetter the better." Take with plenty of fluid. Monitor for electrolyte imbalances. Use caution in diabetes. Miralax is safe and effective in pediatrics and pregnancy.
Prebiotics and probiotics	Use of probiotics such as yogurt with active cultures, lactobacilli and bifidobacteria, and prebiotics (nondigestible oligosaccharides) may be beneficial for gut integrity.
Stimulant laxatives Bisacodyl (Fleet Stimulant Laxative, Correctol, Dulcolax) Ex-Lax or Senokot (with senna) Herbal Authority Aloe Vera, Purge, Feen-a-Mint. Prunes (dried plums) are a natural stimulant laxative.	Stimulant laxatives irritate the intestine, causing bowel contractions. They can cause severe cramping, diarrhea, nausea, and electrolyte imbalances. Avoid using bisacodyl (Dulcolax) with dairy products; take with a high-fiber diet. Not recommended for daily or regular use as they can deplete vitamin D and calcium.
Stool softeners (Emollient laxatives) Docusate sodium (Colace, Fleet Sof-lax, Peri-Colace, Surfak)	Stool softeners are short-term solutions; they allow more water to penetrate stool and to facilitate elimination. Docusate sodium (Colace) should be taken with milk or juice. Long-term use can deplete electrolytes.
Tegaserod (Zelnorm)	Tegaserod was removed from the market in 2007.

See also: Drug Class Review on constipation Drugs at http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=constip, accessed August 11, 2009.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain that proper diet can produce relief but cannot cure the condition. A normal bowel routine is needed, but daily fecal evacuation is not needed by everyone.
- Specifically identify foods that have a laxative effect for the patient. Explain why fiber should be increased on a gradual basis only and that items such as prune juice may help. For every gram of cereal fiber, stool weight increases by 3–9 g. For some, a cup of hot prune juice may be useful.
- Have the patient drink 8–10 glasses of water daily, as permitted. Warm fluids are especially useful.
- Exercise may be beneficial in maintaining regularity, especially abdominal-strengthening exercises.
- Discuss foods that have caused constipation, flatus, and GI distress; offer relevant suggestions.
- Medical assistance is needed for diarrhea, bleeding, infection, or other changes in bowel habits.
- Bowel retraining programs may be helpful and may include increasing exercise, including more fiber in the diet, drinking more liquids, and setting aside 15 minutes to spend on the toilet after breakfast. Regular practices may help reestablish a healthy pattern.
- In a pediatric population, dietary changes, corn syrup, or both may resolve constipation in 25% of children. Laxatives such as milk of magnesia and polyethylene glycol are efficient and safe for almost all cases (Loening-Baucke, 2005).

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.
- Hand washing is extremely important after toileting.

For More Information

- Constipation
  http://digestive.niddk.nih.gov/ddiseases/pubs/constipation/
- Constipation Algorithm
  http://www.uwgi.org/guidelines/ch_05/AlgA.htm
- Emedicine
  http://www.emedicine.com/med/topic2833.htm
- International Foundation for Functional Gastrointestinal Disorders
  http://www.aboutconstipation.org/
- Med Info
  http://www.medinfo.co.uk/conditions/constipation.html
- Web MD—Chronic Constipation
  http://www.webmd.com/digestive-disorders/chronic-constipation-7/default.htm

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DIARRHEA, DYSENTERY, AND TRAVELER'S DIARRHEA

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Diarrhea (acute enteritis) is a symptom of many disorders in which there is usually an increased peristalsis with decreased transit time through the GI tract. In Table 7-11, etiologies and comments related to diarrhea are given. Reduced reabsorption of water and watery stools result; see Table 7-12.

Bacterial diarrhea is caused from food or water contaminated with *Campylobacter, Salmonella, Shigella, and E. coli.* Chronic diarrhea involves production of loose stools with or without increased frequency for more than a month. An individualized approach, knowledge of GI physiology, and awareness of the physiological effects of foods or medications are best used to design an effective dietary plan (Schiller, 2006). Dehydration is a common problem; watch for decreased skin turgor, dry mucous membranes, thirst, 2% weight loss or more, low BP, postural hypotension, increased BUN and hematocrit, and decreased urinary output.

Early childhood diarrhea can come from or lead to malnutrition. Diarrhea is the leading cause of death in children younger than 5 years of age, especially in developing countries. Early rehydration may prevent many deaths in high-risk infants if mothers seek medical attention and offer proper rehydration solutions as soon as diarrhea begins. Oral rehydration solutions (ORS) are well tolerated and shorten illness and decrease fluid losses. Diarrhea can be prevented by breastfeeding, by immunizing all children

TABLE 7-11 Diarrhea: Etiologies, Comments and Bristol Stool Chart

Diarrhea Type	Cause	Comments
Antibiotic-induced	*Clostridium difficile* is the gram-positive anaerobic bacterium most often responsible.	Clindamycin or cephalosporin often causes diarrhea.
Chronic diarrhea	Celiac disease, cow's milk allergy, bacterial and parasitic factors, cystic fibrosis, or post-infectious gastroenteritis	Manage the underlying condition and often the diarrhea resolves. Celiac disease or food allergy should be considered.
Dysentery	**Dysentery is from poor sanitation; causes range from contact with feces to contamination by houseflies.**	**Dysentery may involve diarrhea, with blood and mucus, intestinal rumbling, cramps, fever, and pus in stools.**
Functional	From irritation or stress	Often resolves on its own
Organic	From intestinal lesion	May require further medical evaluation
Osmotic	From carbohydrate intolerance	Lactose, fructose, or sorbitol malabsorption may be a cause
Secretory	From bacteria, viruses, bile acids, laxatives, or hormones	This is typically a more serious condition.
		Rotavirus and *Norwalk virus* commonly affect infants and school-aged children; vomiting and watery diarrhea may occur.
		Norwalk-like viruses may also affect the frail elderly.
Traveler's diarrhea (TD)	**TD is from contaminated food or water.**	**TD is usually caused by enterotoxic bacteria (*E. coli, Campylobacter, Shigella, Salmonella,* or *Yersinia*) or viruses or protozoa, *Giardia* is less common.**

TABLE 7-12 Bristol Stool Scale[a]

Type 1		Separate hard lumps, like nuts (hard to pass)
Type 2		Sausage-shaped but lumpy
Type 3		Like a sausage but with cracks on its surface
Type 4		Like a sausage or snake, smooth and soft
Type 5		Soft blobs with clear-cut edges (passed easily)
Type 6		Fluffy pieces with ragged edges, a mushy stool
Type 7		Watery, no solid pieces. **Entirely Liquid**

[a]This medical aid was designed to classify feces into seven groups, developed by Heaton and Lewis at the University of Bristol, and first published in the *Scandinavian Journal of Gastroenterology* in 1997. The form of the stool depends on the time it spends in the colon. For more information, see www.continence.org.au. Accessed August 1, 2009.

against measles, by keeping food safe and water clean, and by washing hands before touching food.

***Clostridium difficile* infection (CDI)** diarrhea can be caused by use of most antibiotics. There may be profuse watery diarrhea that may be foul smelling; abdominal pain, cramping, and tenderness; stools that may be guaiac positive and grossly bloody; and fever or WBC count of 12,000–20,000/μL. In severe cases, toxic megacolon, colonic perforation, peritonitis, hypovolemic shock, sepsis, and hemorrhage might occur. Symptoms may develop within a few days or even 6–10 weeks after antibiotic therapy is completed. Because *C. difficile* may be a normal bowel organism (especially in children), simply culturing the organism does not mean that diarrhea is caused by *C. difficile*. In mild cases, symptoms will usually resolve spontaneously once the causative antibiotic is withdrawn. More severe cases warrant therapy with vancomycin and metronidazole for 10 days. Tapered-dose oral vancomycin followed by a pulsed-dose regimen, probiotic approaches, restoration of the normal flora, immunological approaches, toxin-binding approaches, and serial therapy with vancomycin followed by rifaximin may be needed for recurrent infections (Johnson, 2009). See Section 15 for more information.

Dysentery involves severe diarrhea containing mucus or blood. Vomiting of blood occurs. If left untreated, it may be fatal. Oral rehydration therapy is needed, and medication to treat any parasitic or bacterial infection.

For **traveler's diarrhea (TD)**, high-risk destinations include most countries in Latin America, Africa, the Middle East, and Asia. Both cooked and uncooked foods are a concern if they have been improperly handled. Risky foods include raw or undercooked meat and seafood and raw fruits and vegetables. Tap water, ice, and unpasteurized milk and dairy products can be associated with increased risk. Daily doses of rifaximin (Xifaxan) appear to significantly decrease the incidence of TD from *E. coli*.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Diarrhea is a symptom rather than a disease. Identify the cause and whether or not there is a genetic origin.

Clinical/History

- Height
- Weight
- Weight changes
- BMI
- Diet history
- Number of stools daily
- Stool consistency (see Stool Chart)
- BP
- I & O; dehydration?
- Temperature
- Abdominal pain

Lab Work

- Na^+ (decreased)
- K^+, Cl^-
- Ca^{++}, Mg^{++}
- H & H
- Serum Fe, ferritin
- Lactose tolerance test
- Hydrogen breath test
- Stool culture such as for *C. difficile*
- BUN:Creat ratio
- Gluc
- Alb, transthyretin
- CRP
- Serum copper
- N balance

SAMPLE NUTRITION CARE PROCESS STEPS

Altered GI Function with Diarrhea

Assessment: Food diary, bowel patterns. Medical history or genetic etiology of diarrheal disorders.

Nutrition Diagnosis (PES): Altered GI function related to excessive intake of poorly absorbed CHOs as evidenced by frequent intake of apple juice and sorbitol-containing dietetic products with cramping and loose stools.

Intervention: Food and Nutrient Delivery: change diet as needed. Educate about impact of poorly absorbed CHO on bowel function.

Monitoring/Evaluation: Reports of less abdominal cramping and loose stools.

INTERVENTION

OBJECTIVES

- Determine cause and apply an appropriate treatment.
- Prevent or alleviate dehydration, electrolyte imbalances, anemia, weight loss, and hypoglycemia.
- Avoid refeeding syndrome. CPN may lead to reduction in nutrient intake and atrophy of the gut.
- Restore normal bowel motility. Alter stool consistency and quantity; up to 200 g of stool per day is normal.
- Avoid extremes in food and beverage temperatures, which may stimulate extra colonic activity.
- Correct intolerances for CHO and protein. Ensure adequate fat intake. Short-chain fatty acids enhance sodium reabsorption; include adequate fiber (American Dietetic Association, 2008).
- Probiotic foods may be useful, such as yogurt with live and active cultures.

FOOD AND NUTRITION

- It may be useful to hold food for 12 hours with use of IV fluids and electrolytes. Start oral fluids as soon as allowed. Use oral rehydration therapy, shown in Table 7-13, or prepared products such as CeraLyte.
- **Infants:** Use rehydration solutions if allowed. Breastfeeding may be continued, or return to lactose-containing formula when feasible. Cut back on use of sorbitol (as in apple juice).
- **Adults:** Start with broth, tea, toast, and gradually add foods to a normal diet as tolerance progresses. Three to four small meals may be better tolerated. Products such as Gatorade may be useful. Banana flakes may be a safe, cost-effective treatment for diarrhea in critically ill patients on TFs. Use products containing probiotics. For potassium, include bananas, orange juice, fruits, and vegetables in the diet. Cocoa beans contain a large amount of flavonoids; dark chocolate may offer mild relief from diarrhea (Schuier et al, 2005).
- If TF is used, check tube placement and medications, presence of bloody stools, and endoscopic exams. Treatment includes changing medications, decreasing rate of feeding, changing to a high formula, antibiotic therapy, or using antidiarrheal medications. A jejunal placement may be too far for some patients and may actually cause some diarrhea.
- Use CPN only for intractable diarrhea. Osmotic diarrhea abates with NPO. Short-chain fatty acids from high-fiber sources may be useful.
- Use multivitamin–mineral supplements to replace vitamins A and C, zinc, iron, and other nutrients.

Common Drugs Used and Potential Side Effects

- Every patient should receive a careful medication review. Magnesium-containing antacids, digoxin, broad-spectrum antibiotics, antifungal agents, colchicine, thiazide diuretics and other antihypertensives, Azulfidine, methotrexate and other anticancer agents, cholinergic stimulants, antiemetics such as metoclopramide, and laxatives such as mineral oil or methylcellulose may cause drug-induced diarrhea. Sorbitol may cause diarrhea; it is found in many medications. Megadoses of vitamin C (>1 g daily) may cause diarrhea.
- Antibiotics are used if shigellae or amoebae are causing the problem. Suggest use of probiotics with antibiotic therapy. Intestinal flora modifiers (e.g., *Lactobacillus acidophilus*, Lactinex, Bacid) also help recolonize normal intestinal flora. A common prescription is three to four packages every day for 3 days in adults. Modifiers may be mixed with water for tube-fed patients.
- Antidiarrheal drugs are used to slow peristalsis or thicken stools. Kaolin (Kaopectate) has no major side effects but

TABLE 7-13 UNICEF/WHO Oral Rehydration Therapy[a]

Ingredients	Original ORS	New Reduced Osmolarity ORS
Sodium chloride	3.5 g/L	2.6 g/L
Potassium chloride	1.5 g	1.5 g/L
Sodium	2.5 g sodium bicarbonate	2.9 g/L trisodium citrate
Glucose	20 g /L of clean drinking water	13.5 g/L of clean drinking water

[a]Where ORS is not available, home-prepared solutions may use 1 teaspoon of salt, 8 teaspoons of sugar, and 4 oz of orange juice mixed into 1 L of water. From WHO Rehydration Project. Accessed August 1, 2009, at http://www.rehydrate.org/ors/ort_how_it_works.htm.

is not useful with infants. Lomotil should be taken with food; it may cause bloating, constipation, dry mouth, swollen gums, dizziness, nausea, and vomiting. Avoid using it with alcohol. Psyllium ingestion reduces stool looseness.
- Cholestyramine (Questran) may be used for bile acid diarrhea. Nausea, belching, or constipation may result. Replace fat-soluble vitamins.
- Ciprofloxacin is used for TD; one dose is often sufficient. It is a quinolone-class antibiotic. Avoid milk or yogurt. Nausea is one side effect.
- Vancomycin is used for treating *C. difficile*. Anorexia, GI distress, diarrhea, and nausea may result.
- Daily doses of rifaximin (Xifaxan) may significantly decrease TD from *E. coli*. It is not known yet if this same treatment is effective against TD from *Salmonella* and other bacteria.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician. Apple, carrot, blackberry, carob, bilberry, and tea have been recommended, but no clinical trials have proven efficacy.
- Probiotic medications may be helpful: for children, *Lactobacillus* GG or Culturelle; and for adults, *Saccharomyces boulardii*. Probiotics in foods can help to maintain good bacteria in the GI tract of those taking antibiotics. Yogurt may help to reculture the GI tract; check labels for live and active cultures. Acidophilus milk is also useful.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Describe the effects of pectin as a thickening agent (as in apples and bananas), and inform about yogurt or acidophilus milk or other specific probiotic foods/supplements.
- Avoid sweetened carbonated beverages because their electrolyte content is low and osmolality is high. Caffeine, apple juice, and milk can aggravate diarrhea; omit until resolved.
- Limit fruit juice to 6 oz daily in children.
- Partially hydrolyzed guar gum (Benefiber) added to a diet ferments in the colon and produces short-chain fatty acids. This improves intestinal function, including colonic salt and water absorption (Alam et al, 2005). It may be a useful addition until diarrhea resolves.
- Newly formulated oral rehydration salts contain lower concentrations of glucose, and salt and zinc supplementation can drastically reduce the number of child deaths.
- Teach about the prevention and treatment of dehydration with appropriate fluids, breastfeeding of infants, and selective use of antibiotics to reduce the duration and severity of diarrheal episodes.
- Signs of dehydration include increased thirst, dark urine, light headedness, dry skin and less-frequent urination. Children may have no tears when crying, dry mouth and tongue, sunken abdomen or eyes.

Patient Education—Foodborne Illness

- The World Health Organization (2009) states the following facts:

  A total of 1.8 million people die every year from diarrheal diseases (including cholera); 90% are children under 5 who live in developing countries.

  Nearly all diarrheal disease is attributed to unsafe water supply, inadequate sanitation, and poor hygiene.

  Improved water supply reduces diarrhea morbidity by 21%, improved sanitation by 37.5%.

  Simply washing hands at critical times can reduce diarrheal cases by up to 35%. Improvement of drinking water quality, such as point of use disinfection, would lead to a reduction of diarrheal episodes by half.
- To prevent TD, follow these guidelines:

  Use safe, bottled water for drinking and brushing teeth.

  Wash hands before eating, using antiseptic gel or hand wipes.

  Avoid ice in drinks.

  Do not eat raw vegetables or salads, raw fruits, or unpasteurized dairy products.

  Avoid swimming in streams and lakes.

  Use only cooked foods and bottled beverages (e.g., water, juices, beer, etc.).

  Brush teeth with bottled water only.

  Use caution with fresh foods that may have been washed in contaminated water and foods prepared with unheated water (e.g., jello) or ice cubes made with contaminated water.

  If symptoms persist, medical attention should be sought.

For More Information

- CeraLyte
  http://www.ceraproductsinc.com/productline/ceralyte.html
- Centers for Disease Control and Prevention (CDC) Division of Parasitic Diseases
  http://www.cdc.gov/ncidod/dpd/
- Diarrhea
  http://digestive.niddk.nih.gov/ddiseases/pubs/diarrhea/
- Medicine Net—Diarrhea
  http://www.medicinenet.com/diarrhea/article.htm
- Diarrhea Algorithm
  http://www.uwgi.org/guidelines/ch_04/ch04.htm
- Giardiasis
  http://www.cdc.gov/ncidod/dpd/parasites/giardiasis/factsht_giardia.htm
- Rehydration Formula
  http://www.rehydrate.org/
- Travelers' Health (CDC)
  http://www.cdc.gov/travel/diarrhea.htm
- World Health Organization—Dysentery
  http://www.who.int/topics/dysentery/en/

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DIVERTICULAR DISEASES

NUTRITIONAL ACUITY RANKING: LEVEL 2–3

DEFINITIONS AND BACKGROUND

Diverticular disease results from formation of small pouches (diverticula) in the colon wall and lining due to chronic constipation. Diverticular disease occurs in Westernized countries because of low-fiber diets but is rare in societies that subscribe to high-fiber patterns. Lower GI bleeding from diverticulosis is a common reason for hospital admission, particularly in the elderly (Strate, 2005).

A high-fiber diet is the mainstay of management for diverticulosis (American Dietetic Association, 2008). Diverticulitis (inflammation) develops when bacteria or other irritants are trapped in the pouches, causing spasm and pain in the lower left side of the abdomen, as well as distention, nausea, vomiting, constipation or diarrhea, chills, and fever. Bowel cancer has been associated with the presence of diverticular disease.

Serotonin is widely distributed throughout the gut in both the enteric nerves and enterochromaffin cells, where they act as signal transducers and respond to bacterial or dietary substances with an inflammatory response and associated diarrheal symptoms respond well to 5-HT(3) receptor antagonists (Spiller, 2008). Probiotics and prebiotics are being tested for relief of pain and inflammation. Prebiotics such as guar gum are fermented into short chain fatty acids in the colon, where they stimulate water and sodium absorption. Probiotics are used to prevent or treat diarrhea; they are live organisms that eliminate toxins and inhibit proliferation of pathogens.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Altered motility is an important feature of the pathogenesis of diverticular disease, and serotonin (5-HT) release is a primary trigger of gut motility (Costedio et al, 2008).

Clinical/History
Height
Weight
BMI
Diet history
Obesity?
Physical inactivity?
Stool number, frequency
BP
Abdominal pain
Blood in stool?
Sigmoidoscopy

Abdominal ultrasound
CT scan
Barium enema

Lab Work
H & H
Serum Fe, ferritin
Transferrin, TIBC
CRP
Erythrocyte sedimentation rate (increased)
Alb, transthyretin
Na^+, K^+
Ca^{++}, Mg^{++}
WBC (increased)

INTERVENTION

OBJECTIVES

Diverticulitis (Inflamed State)

- Allow complete bowel rest to prevent perforation by avoiding the laxative effect of excess fiber.
- Eliminate food particles that accumulate in sacs as they may cause bacterial contamination.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Fiber Intake

Assessment: Dietary recall of food intake with calculation of average fiber intake <8 g based on 24-hour recall and food frequency.

Nutrition Diagnoses (PES): Inadequate fiber intake (NI 5.8.5) related to nutrition-related knowledge deficit as evidenced by inability to list five foods high in fiber and average daily fiber intake less than 3 g/d. Altered GI function (NC 1.4) related to diarrhea for 3 days, frequent pain, and increased flatus over 3 months.

Interventions: Food-Nutrient Delivery—ND 1.2—soft diet, low-fiber foods until pain subsides.

Education—E 2.2—current diet order for soft, low fiber. Education on gradual introduction of fiber to diet to goal daily amount of 25–35 g; adequate fluid intake; increasing physical activity if possible. Provide list of high-fiber foods for inclusion in the diet, discuss the importance of fiber in diverticulosis.

Counseling: Discuss foods to avoid when there is discomfort.

Monitoring and Evaluation: Reassess dietary intake of fiber at next visit in 3 months; goal is to increase intake to 25–35 g/d. Evaluate daily dietary fiber intake using food recall/diary. Fewer complaints of pain, flatus, or diarrhea.

- Prevent peritonitis and abscess.
- Correct any GI bleeding, hypoalbuminemia, or anemia.
- Each person differs in the amounts and types of foods they can eat; keep a food diary to identify any foods that may cause symptoms or discomfort.

Diverticulosis (Convalescent State)

- Improve stool quality and increase volume, mostly from fiber.
- Relieve intraluminal pressure; decrease the contractions of colonic circular smooth muscle.
- Distend the bowel wall to prevent development of high-pressure segments and inflammation.
- Elimination of specific foods is not necessary.

FOOD AND NUTRITION

Diverticulitis (Inflamed State)

- As treatment begins, use a soft diet with low fiber. Gradually add fiber as inflammation and pain decrease.
- Ensure adequate intake of protein and iron sources.
- A low-fat diet may be better tolerated.

Diverticulosis (Convalescent State)

- Diet should be high in fiber; 25–35 g/d is desirable. Whole grains, stewed or dried fruits, potato skins, raw carrots, or celery may be used; see Table 7-14. No evidence supports the common advice to avoid nuts and seeds (Strate et al, 2008). Eating nuts, corn, and popcorn does not increase the risk; in fact, nuts and popcorn may have a protective effect (Weisberger and Jamieson, 2009). Increase fiber gradually; avoid large excesses of fiber that may interfere with mineral absorption. Seeds in fruits or vegetables, and sunflower, pumpkin, caraway, poppy and sesame seeds do not enter, block, or irritate the diverticula and may be consumed.
- Adequate fluid is recommended.
- A vegetarian, plant-based diet is beneficial (Leitzmann, 2005) and a low-fat diet may reduce intracolonic pressure.

TABLE 7-14 How to Eat More Fiber

- To get adequate fiber in the diet, follow the U.S. Department of Agriculture Food Guidance System (MyPyramid), which recommends eating 2–4 servings of fruit, 3–5 servings of vegetables, and 6–11 servings of cereal and grain foods each day.
  1. Be smart by eating a whole-grain cereal that contains at least 5 g of fiber per serving. 1/2 cup of bran cereal contains 10 g of fiber.
  2. Beans, peas, and lentils contain 6–9 g per 1/2 cup portion. Add them to soups, stews, and salads.
  3. Eat raw vegetables as much as possible because cooking may reduce fiber content. A baked potato with skin, broccoli, shredded carrots, and cauliflower are great choices.
  4. Eat the peel on fruits (such as apples, pears), and vegetables (such as cucumbers) because much of the fiber is found in the skin.
  5. Eat fresh and dried fruits as snacks. Pears, apples, oranges, strawberries, other berries, prunes, bananas, and figs are all good choices.
  6. Read food labels. Select foods that contain fiber whenever possible.

Sources: U.S. Department of Agriculture Nutrient Database, Release 17, 2004; and Medline Plus. Dietary fiber. 2009. Accessed at http://www.nlm.nih.gov/medlineplus/dietaryfiber.html

Common Drugs Used and Potential Side Effects

- Antibiotics: For patients with severe and complicated diverticulitis, ampicillin, gentamicin, metronidazole, piperacillin, and tazobactam are used. Ciprofloxacin, metronidazole, and rifaximin are used for uncomplicated diverticular disease. Side effects may include nausea, vomiting, stomatitis, and other GI effects.
- There is not enough evidence to recommend the anti-inflammatory drug mesalamine or a polybacterial lysate for immunostimulation (Weisberger and Jamieson, 2009).
- Pain medicine may be needed when cramping or bloating is significant.
- Overusing laxatives may result in dependence on them.
- Probiotics seem to be beneficial.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Flax, wheat, wild yam, slippery elm, and chamomile have been recommended for this condition, but no clinical trials have proven efficacy.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient concerning dietary fiber:

  Some ingested plant material is not digested by GI enzymes, including cellulose, pectin, lignin, and hemicelluloses.

  Some dietary fibers (whole grains) resist intestinal disintegration, whereas others (fruits and vegetables) are more or less disintegrated. In general, increased fiber increases stool volume, frequency, and transit rate and decreases intracolonic pressure (American Dietetic Association, 2008).

  Increased stool volume and decreased intracolonic pressure improve transit time.

  Take a fiber product such as methylcellulose (Citrucel) or psyllium (Metamucil) one to three times a day with at least 8 oz of water.
- Instruct patient to chew slowly and to avoid constipation and straining.
- If flatulence is a problem, advise that approximately 6 weeks will be needed to allow bacterial flora to adapt to increased fiber intakes.
- Adequate physical activity is beneficial.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing diarrhea and related discomfort.
- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Diverticular Diseases
  http://emedicine.medscape.com/article/774922-overview
- International Foundation for Functional Gastrointestinal Disorders
  http://www.iffgd.org/
- Merck Manual—Diverticular disease
  http://www.merck.com/mmhe/sec09/ch128/ch128c.html
- NIDDK
  http://digestive.niddk.nih.gov/ddiseases/pubs/diverticulosis/
- Web MD
  http://www.webmd.com/digestive-disorders/diverticular-disease

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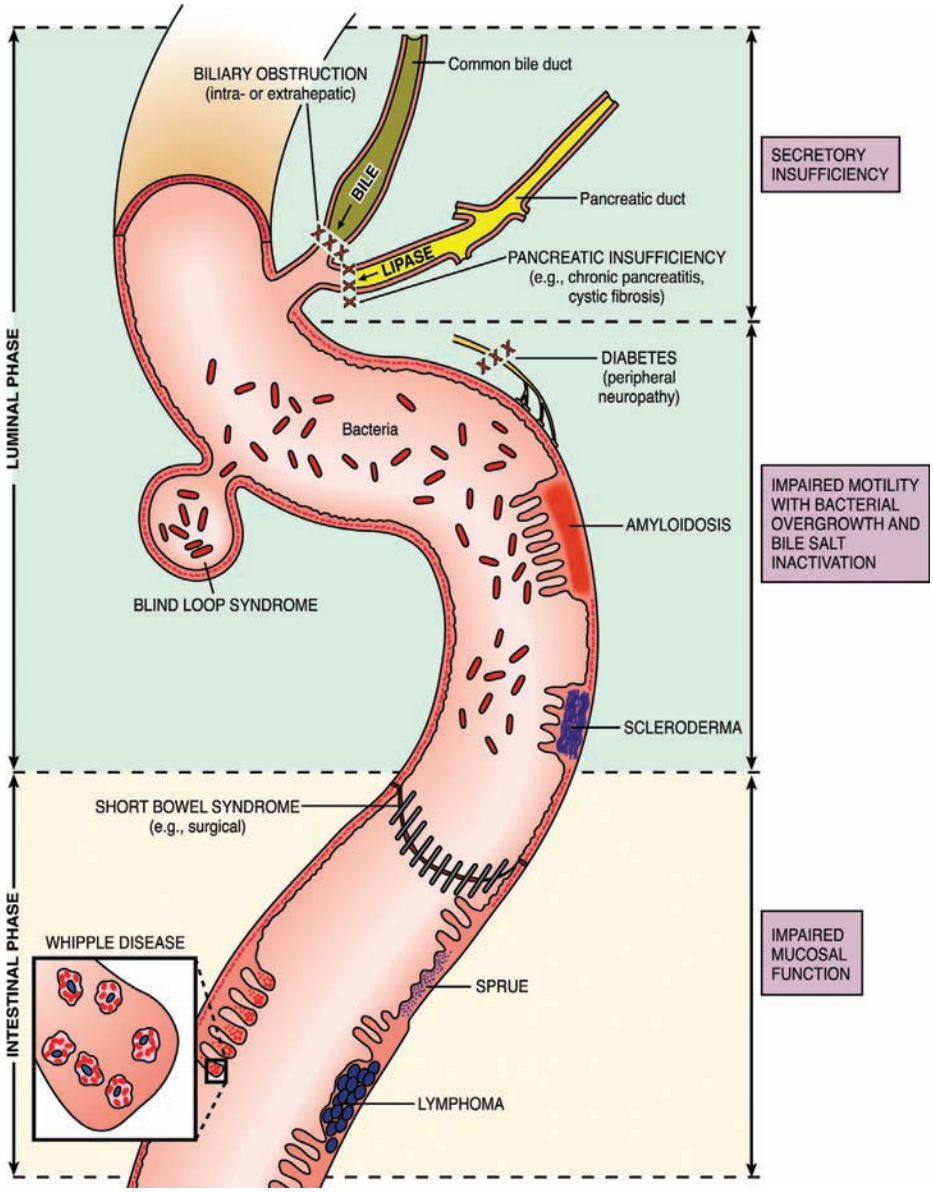
Strate LL, et al. Nut, corn, and popcorn consumption and the incidence of diverticular disease. *JAMA.* 300:907, 2008.

Weisberger L, Jamieson B. Clinical inquiries: How can you help prevent a recurrence of diverticulitis? *J Fam Pract.* 58:381, 2009.

FAT MALABSORPTION SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 3



Adapted from: Rubin E MD and Farber JL MD. *Pathology,* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

TABLE 7-15 Altered Stools and Related Disorders

Characteristic	Disorder
Yellow or silver color	Fat malabsorption
Pale, foamy, mushy, or floating	Pan malabsorption
Formed in morning	Diarrhea
Formed in evening	Bile salt malabsorption

TABLE 7-16 Fecal Fat Study

Fat absorption is tested by quantitative measurement of total fat in the stool.

Preparation:	Consume 100 g of long-chain triglycerides (LCT) over 3 days
Normal excretion:	Less than 7 g (5% of a 60–100 g intake)
Mild malabsorption:	7–25 g (defects in micelle formation)
Moderate malabsorption:	25–30 g (intestinal mucosal disease)
Severe malabsorption:	More than 40 g (massive ileal resection or pancreatic disease)

Adapted from Hermann-Zaidins M. Malabsorption. *J Am Diet Assoc.* 86:1171, 1986.

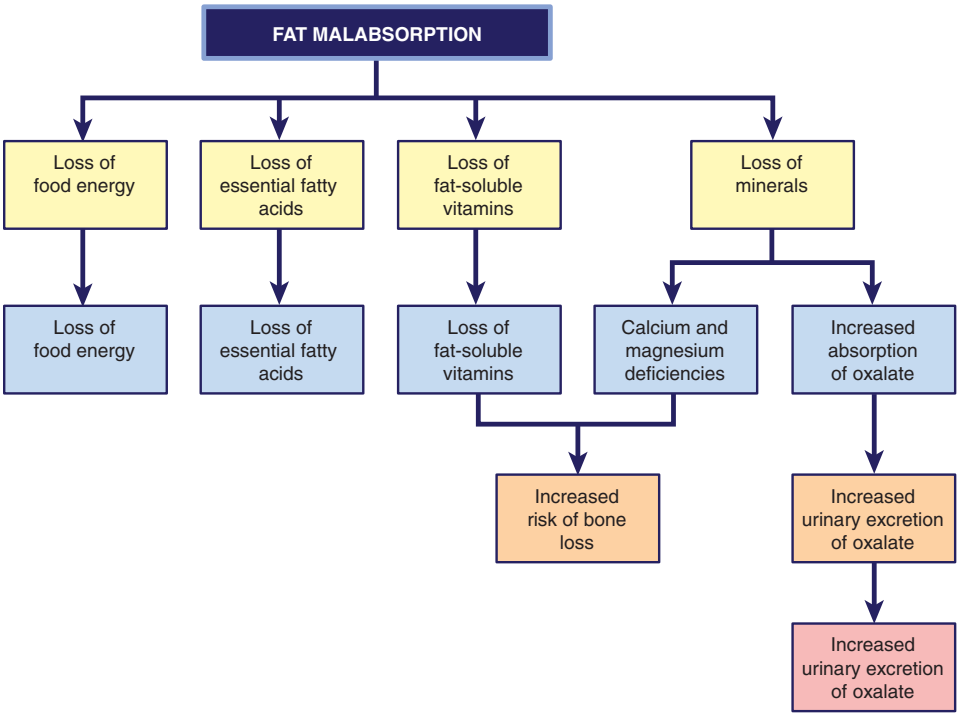
DEFINITIONS AND BACKGROUND

Fat malabsorption syndrome is caused by functional or organic causes. There may be fatigue, weight changes, steatorrhea, abdominal distention with cramps and gas, explosive diarrhea with foul-smelling stools, malnutrition and weight loss, and biochemical abnormalities. Exocrine pancreatic insufficiency is the principal cause. Other causes may include: postgastrectomy, blind loop syndrome, Crohn's disease, small bowel resection; cystic fibrosis, chronic pancreatitis, pancreatic cancer, pancreatectomy; biliary atresia or steatorrhea; CD, lipoprotein deficiency, or HIV infection.

The individual with malabsorption syndrome must be monitored for dehydration (dry tongue, mouth, and skin; increased thirst; low, concentrated urine output; weakness or dizziness when standing). Signs of nutrient depletion include nausea, vomiting, fissures at corner of mouth, fatigue, weakness, and dry, pluckable hair. Long-term nutritional monitoring is necessary after operations for morbid obesity, which can lead to fat malabsorption and vitamin deficiencies for vitamins A, D, and K (Malinowski, 2006). Malabsorption can have severe clinical consequences, including a decline in bone health.

To avoid nutritional deterioration, early screening for fat malabsorption should be recommended. Low total cholesterol (<120 mg/dL) or low serum carotene levels may be typical of fat malabsorption but are not necessarily diagnostic. Altered stools characterize different types of malabsorption, as shown in Table 7-15. In patients with bile acid malabsorption, a larger amount of bile acids is spilled into the colon, where the acids stimulate electrolyte and water secretion, which results in loose to watery stools (Westergaard, 2007). Patients with more severe bile acid malabsorption have both diarrhea and steatorrhea; these patients are best treated with a low-fat diet supplemented with MCTs (Westergaard, 2007). A fecal fat study, though now uncommon, may be ordered; see Table 7-16.

Enteric hyperoxaluria is the major risk factor for fat malabsorption, especially with IBD. In UC after restorative proctocolectomy, an increase of serum AA occurs with lower utilization for inflammatory processes, and reduced LDL cholesterol occurs as an index of malabsorption (Scarpa et al, 2008). Probiotics are being studied.



Adapted from: Merck Manual. Malabsorption syndromes. http://www.merck.com/media/mmpe/pdf/Figure_ 017-1.pdf. Website accessed 9/1/09.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Fat malabsorption is typically a symptom of an underlying disorder that may be either genetic or acquired.

Clinical/History	Lab Work
Height	Tryptophan load test for vitamin B_6
Weight	Fecal Ca^{++}
BMI	Acid steatocrit
Diet history	Malabsorption blood test
BP (may be low)	^{14}C-triolein breath test
Signs of dehydration?	Fecal fat study: 72-hour stool collection
Failure to thrive in children	Labeled carbon breath test
Perianal itching or soreness	Sudan stain test
Frequent, foul-smelling stools	Serum carotene, vitamin A (may be low)
Small intestine biopsy	D-xylose test (decreased excretion?)
CT scan or MRI	Serum vitamin E
Barium enema or x-rays	Schilling test for vitamin B_{12}
	Na^+, K^+, Cl^- (may be low from diarrhea)
	Ca^{++}, Mg^{++} (may be low)
	Chol (may be decreased)
	Trig
	H & H
	Serum Fe, ferritin
	Gluc
	Alb, transthyretin (may be low)
	CRP

INTERVENTION

OBJECTIVES

- Monitor for malabsorption of fat-soluble vitamins (A, D, E, and K). Long-term consequences of vitamin deficiency results if fat malabsorption is not corrected.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal Nutrient (FAT) Utilization

Assessment: I & O, abnormal nutritional labs for cholesterol, albumin, electrolytes. Fecal fat test results showing excesses of 20 g fat in stool; stools of yellowish hue. Abdominal pain.

Nutrition Diagnoses (PES): Abnormal fat utilization related to malabsorption as evidenced by fecal fat study showing fat in stool of >20 g/d, yellowish stools, and abdominal pain.

Interventions: Food and nutrient delivery: alter diet to include products containing MCT oil until condition can be resolved.

Education: Discuss how fat malabsorption affects metabolism and absorption of nutrients. Counseling: Inclusion of water-miscible forms of fat-soluble vitamins, more calcium and water-soluble vitamins.

Monitoring and Evaluation: Improvement in nutritional labs; fewer yellowish or abnormal stools. Fecal fat study showing less than 7 g fat. Less abdominal discomfort.

TABLE 7-17 Medium-Chain Triglycerides (MCTs)

- Many products now contain MCTs as the primary fat source.
- MCTs use portal (albumin-free fatty acids) rather than lymphatic system transport and absorption (using less lipase and bile).
- MCTs have an 8- to 10-carbon source of fat and are useful when longer chain fatty acids (16–18 carbons) cannot be efficiently digested or absorbed.
- MCTs have concentrated calories made from coconut oil for adjunct therapy.
- MCT oil has 230 kcal/30 mL (6–7 kcal/g). Use instead of vegetable oil in recipes.

- Prevent calcium oxalate stone formation, or correct where present.
- Correct all other nutrient deficiencies.
- Alleviate steatorrhea and reduce intake of fat sources that are not tolerated. MCTs are useful. See Table 7-17.

FOOD AND NUTRITION

- Initial treatment should consist of parenteral solutions or liquid formulas that contain MCT. MCTs alleviate steatorrhea in some cases; start with 20–60 g and increase gradually in an adult.
- For mild cases, oral feeding is preferred because it stimulates brush-border activity. For moderate-to-severe cases, tube feed if necessary (50 mL/hr full strength initially; advance gradually).
- Dietary fat may be limited to one egg and 4–6 oz of meat, poultry, or fish. Gradually check tolerance for long-chain triglycerides (LCTs) and work up to 30–40 g.
- Increase intake of protein, which may be in the form of skim milk, egg white, cereals, or legumes.
- Complex carbohydrates may be better tolerated than simple sugars. Lactose may not be tolerated.
- A multivitamin–mineral supplement may be necessary to offset fecal losses of nutrients, vitamins, and water in patients with malabsorption syndromes—especially zinc, folate, vitamin B_{12}, calcium, magnesium, iron, and fat-soluble vitamins (A, D, E, and K).
- Monitor or decrease dietary oxalate intake to prevent renal stones. Use of probiotics may be helpful.

Common Drugs Used and Potential Side Effects

- Antibiotics are used for bacterial overgrowth. Suggest use of yogurt with live and active cultures with antibiotic therapy. Intestinal flora modifiers (e.g., *Lactobacillus acidophilus*, Lactinex, Bacid) also help recolonize normal intestinal flora, perhaps three to four packages every day for 3 days in adults. They may be mixed with water for TFs.
- Antidiarrheals may be used, such as kaolin (Kaopectate). Cholestyramine may be needed for bile salt diarrhea; fat-soluble vitamins can be depleted.
- Cholylsarcosine (CS) is a semisynthetic bile salt replacement. If used properly, no side effects occur.
- Orlistat inhibits pancreatic lipase and blocks the absorption of 30% of ingested fat in patients seeking weight loss.

By its nature, it has the potential to create fat malabsorption syndrome, and use should be carefully monitored to assure that nutrient deficiencies do not occur.
- Pancreatic enzymes may be needed if there is pancreatic insufficiency.
- Patients with mild-to-moderate bile acid malabsorption present with watery diarrhea and generally respond very well to treatment with bile acid binders such as cholestyramine (Westergaard, 2007).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Caution the patient about a rapid consumption of MCTs. If they are consumed too rapidly, hyperosmolar diarrhea may result. Abdominal discomfort, flatulence, diarrhea, or steatorrhea may indicate continued malabsorption; the physician should be contacted.
- Encourage several small, frequent meals throughout the day, avoiding fluids and foods that promote diarrhea. Monitor I & O of fluids, along with the number, color, and consistency of stools.
- Remember that a source of essential fatty acids may be needed if MCTs are used with a low-fat diet.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to this individual who may be experiencing diarrhea and related discomfort.
- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Fat Malabsorption Syndrome
  http://www.merck.com/mrkshared/mmanual/section3/chapter30/30a.jsp
- E-medicine
  http://www.emedicine.com/PED/topic1356.htm
- Merck manual—malabsorption
  http://www.merck.com/mkgr/mmg/sec13/ch111/ch111a.jsp

FAT MALABSORPTION SYNDROME—CITED REFERENCES

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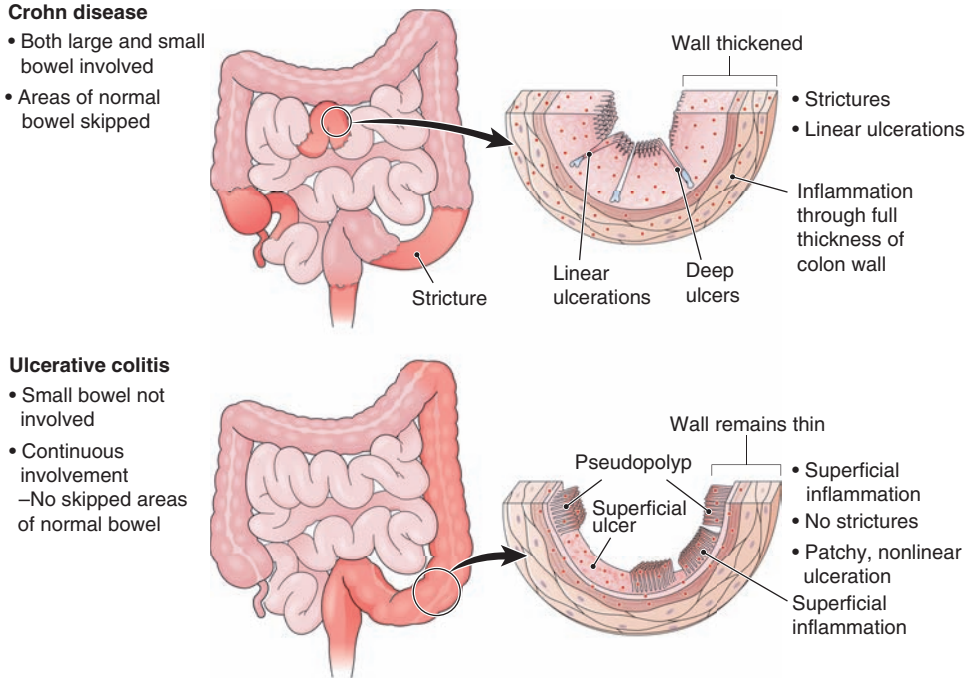
Scarpa M, et al. Restorative proctocolectomy for ulcerative colitis: impact on lipid metabolism and adipose tissue and serum fatty acids. *J Gastrointest Surg.* 12:279, 2008.

Westergaard A. Bile Acid malabsorption. *Curr Treat Options Gastroenterol.* 10:28, 2007.

INFLAMMATORY BOWEL DISEASE: CROHN'S DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 3–4



Adapted from: Thomas H. McConnell, *The Nature Of Disease Pathology for the Health Professions,* Philadelphia: Lippincott Williams & Wilkins, 2007.

DEFINITIONS AND BACKGROUND

Crohn's disease involves acute and chronic granulomatous, IBD with a cobblestone effect. Onset is generally between 15 and 30 years of age. Crohn's disease differs from UC by affecting mucosal tissue of GI tract from oral cavity to rectum. In 33%, only the ileum is involved; in 45%, both the ileum and the large intestine are affected.

As many as 1.4 million persons in the United States and 2.2 million persons in Europe suffer from with IBD. Macroscopic cobblestoning, segmental colitis, ileal stenosis and ulceration, perianal disease, and multiple granulomas in the small bowel or colon strongly suggest a diagnosis of CD. The causative antigen in IBD is the microflora in the intestinal lumen, facilitated by an impaired innate immune system; these provoke T helper 1 and T helper 17 responses in Crohn's disease and a T helper 2 response in UC, resulting in pro-inflammatory cytokines and interleukins (Festen et al, 2009). Increased numbers of mucosa-associated *E. coli* are observed (Willing et al, 2009). Patients with CD exhibit a profound systemic failure of the acute inflammatory response that results in markedly delayed clearance of bacteria from the tissues, leading to local chronic granulomatous inflammation and compensatory adaptive changes (Sewell et al, 2009).

Environmental triggers include smoking, northern geographic residence, allergic and autoimmune responses. Both passive and active smoke exposure in childhood predisposes children to IBD (Mahid et al, 2007). In addition, the pathways for amino acids, fatty acids, bile acids and AA are altered (Jansson et al, 2009).

The intestinal lumen decreases; peristalsis from food intake causes cramping pain, especially in the right lower quadrant. Only 25% of Crohn's disease cases present with the classic triad of abdominal pain, weight loss, and diarrhea (Beattie et al, 2006). Chronic watery diarrhea results from edema, bile salt malabsorption, bacterial overgrowth, and ulceration. Children may present with growth failure, inflammation, fever, pallor, and anemia. Stricture may precipitate bowel obstruction. Fever, weight loss, nausea, mouth sores, anal fissures, vomiting, abdominal pain, intestinal bleeding, arthritis, iritis or uveitis, conjunctivitis, jaundice, or pruritus may also be present. Elevated plasma tHcy is common. Nutritional therapy is an essential adjunctive treatment. In cases of short bowel syndrome (SBS), long-term CPN may be essential.

Anti-TNF alpha (anti-TNFα) therapies have been used. Complications associated with Crohn's disease include arthritis, skin problems, inflammation in the eyes or mouth, kidney stones, gallstones, or other diseases of the hepatobiliary system. If medical management fails, surgery may be indicated for continuous bleeding; recurrent ileus, abscesses, or fistulae. After total proctocolectomy, patients have less morbidity and are more likely to be weaned off all Crohn's-related medications (Fichera et al, 2005).

Persons with Crohn's disease are at increased risk for colon cancer, obstruction, anorectal fistulas, and abscesses. Chronic inflammatory processes cause overproduction of reactive oxygen and nitrogen species, overproduction or activation of key AA metabolites and cytokines/growth factors, and immunity system dysfunction (Yang et al, 2009). Researchers are looking at genetically altering *Bacteroides ovatus*, naturally in the gut, to secrete human growth factor KGF-2 protein when exposed to xylan sugar and to heal the damage caused by inflammation (Hamady et al, 2008).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Crohn's disease is associated with multiple mutations in the CARD15, the interleukin 23 receptor (IL23R) and autophagy-related 16-like 1 (ATG16L1) genes. The G allele of SNP rs2241880 confers strong risk for CD with a coding variant, threonine-to-alanine substitution at amino acid position 300 of the ATG16L1 protein (Grant et al, 2008).

Clinical/History

- Height
- Weight
- BMI
- Weight loss?
- Stunted growth?
- Diet history
- BP (fever?)
- Temperature
- Rectal bleeding
- Diarrhea
- Number of bowel movements, frequency
- Barium upper GI series with small bowel follow-through
- Abdominal CT
- Upper endoscopy
- Colonoscopy with biopsies

Lab Work

- WBC, ESR (increased)
- Crohn's disease activity factor (CDAI)
- CRP
- H & H
- Serum Fe, ferritin (decreased)
- Na^+, K^+
- Alb, transthyretin (low)
- Transferrin, TIBC
- N balance
- Serum tHcy
- Serum folate
- Serum B_{12} and Schilling test
- Ca^{++}, Mg^{++}
- Serum B_{12}
- Total protein
- BUN, Creat
- Serum carotene
- Serum zinc (decreased)
- TLC
- Serum Cu
- Hydrogen breath test

INTERVENTION

OBJECTIVES

- Replace fluid and electrolytes lost through diarrhea and vomiting. Lessen mechanical irritation and promote rest, especially with diarrhea.
- Replenish nutrient reserves; correct malabsorption or anemia. Poor nutritional status may be related to decreased intake from anorexia, nausea or vomiting, abdominal pain, restrictive diets, side effects of medications, protein losses from ulcerated mucosal lesions, blood loss or wound-healing requirements, bacterial overgrowth, and malabsorption.
- Monitor lactose and gluten intolerances, which may be present.
- Promote healing; rest bowel from offending agents. Provide foods that contain short-chain fatty acids and glutamine to promote healing.
- Prevent peritonitis, obstruction, renal calculi, and fistulas.

SAMPLE NUTRITION CARE PROCESS STEPS

Food and Nutrition Knowledge Deficit

Assessment: Food diary, x-ray reports, biopsy.

Nutrition Diagnosis (PES): Food and nutrition-related knowledge deficit (NB 1.1) related to management of Crohn's as evidenced by new diagnosis and request for information from patient and family.

Altered GI function (NC 1.4) related to inflammation in newly diagnosed Crohn's disease as evidenced by diarrhea for 2 weeks; abdominal pain and cramping after meals.

Involuntary weight loss (NC 3.2) related to diarrhea and inability to consume and digest adequate daily kilocal as evidenced by weight loss of 15 lb in last 60 days.

Interventions: Food and Nutrient Delivery: ND 3.2.1 Multivitamin/mineral supplement. Clear liquids advancing to low residue diet prior to discharge.

Educate on low residue diet as needed, use of probiotic foods, and small frequent meals with lactose-free foods as needed.

Coordinate Care: Refer to gastroenterology dietitian specialist for outpatient follow-up.

Monitoring and Evaluation: Improved intake and type of foods; symptoms resolved or improved. Monitor Hgb, Alb, Na^+, K^+ for improvements while inpatient; weight for maintenance and eventual regain to usual body weight; bowel health with regard to decreased cramping and diarrhea after meals.

- Promote weight gain or prevent losses from exudates or inadequate intake.
- Prepare for surgery if necessary after failed medical management, obstruction, fistula, or peritonitis. A total colectomy or a right-sided ileocolectomy may be necessary.
- In a child, promote growth. Growth spurts follow sustained weight gain.
- Reduce inflammatory process; fish oil intake may reduce severity of symptoms.
- Monitor mineral and trace element levels carefully to ensure adequacy. Iron tends to be low. Antioxidant intake should be increased.
- Prevent or correct metabolic bone disease (e.g., osteopenia, arthropathies) caused by disease itself, nutrient malabsorption, side effects of medications, or lifestyle factors.
- Because tHcy production within the intestinal mucosa may contribute to the inflammatory response and endothelial cell dysfunction, treat folate and vitamin B_{12} deprivation as well as methylenetetrahydrofolate reductase (MTHFR) polymorphisms (Peyrin-Biroulet et al, 2007). Prevent thrombotic events.

FOOD AND NUTRITION

- For adult energy requirements, estimate needs according to current BMI. A low BMI (<15) may require 35–45 kcal/kg; a BMI of 15–19 may require 30–35 kcal/kg; a BMI of 20–29 may require 25–30 kcal/kg; and a high BMI (>30) may only require 15–25 kcal/kg. Estimate needs at the high end of normal for growth and repair in infants or children.
- With strictures or fistulas, use a low-fiber diet that is high in energy with a high protein content of 1–1.5 g/kg.
- For some patients, TF with added glutamine may be useful. Polymeric formulas are acceptable; elemental products are not required. Randomized controlled trials show that enteral nutrition is effective.
- Perioperative PN may reverse malnutrition and facilitate rehabilitation (Yao et al, 2005). If CPN is needed after total colectomy, use indirect calorimetry to estimate needs.
- A diet relatively high in fat may improve energy balance. Limit fat intake only if steatorrhea is present, in which case MCTs may be better tolerated. Omega-3 fatty acids may be indicated.
- Supplement the diet with multivitamins and minerals, especially thiamine, folacin, vitamin B_{12}, vitamin E, zinc, vitamin D, calcium, magnesium, and iron. Vitamins A and K should be given every other day. With resection greater than 200 cm, selenium may become deficient; monitor carefully.
- Reduce lactose intake if not tolerated. Check for wheat and gluten tolerances.
- Monitor progress carefully; patients may be finicky. Small, frequent meals may be better tolerated.

Common Drugs Used and Potential Side Effects

- Therapies for CD typically include aminosalicylates and antibiotics (for mild mucosal disease), nutritional therapy (including elemental or polymeric formulas), corticosteroids (for moderate disease), and infliximab for corticosteroid-resistant or fistulizing disease (Rufo and Bousvaros, 2006). Standard treatments with 5-aminosalicylic acid, antibiotics, corticosteroids, and immunosuppressives have serious and potentially adverse events (Krygier et al, 2009).
- Anti-inflammation drugs are first-line treatments, with mesalamine in 5-ASA agents (Asacol, Canasa, Pentasa) or sulfasalazine (Azulfidine). Side effects include nausea, vomiting, heartburn, diarrhea, headache, folate depletion.
- Antibiotics reduce bacterial overgrowth and vitamin K may be needed for fistulas. Ampicillin, sulfonamide, cephalosporin, tetracycline, or metronidazole are common choices. Metronidazole may be used when there is anal involvement; nausea, vomiting, anorexia, or diarrhea may occur.
- Anti-diarrheal agents are needed, such as diphenoxylate, loperamide, and codeine.
- Corticosteroids such as Budesonide (Entocort) are used in large doses at first. They are most effective with colon involvement. Patients will need a diet that provides sodium restriction, with extra protein, calcium, and potassium. Corticosteroids are more effective than enteral nutrition (Zachos et al, 2007).

- Anti-TNFα therapy is beneficial. Infliximab (Remicade) was the first treatment approved specifically to reduce inflammation in Crohn's disease. The anti–TNF alpha agents are effective in the induction and maintenance of remission in luminal and fistulizing Crohn's disease; early use with immunosuppressives may alter progression of the disease and prevent late complications (Krygier et al, 2009).
- Some neuroregulatory peptides act as endogenous immune factors with anti-inflammatory effects; selective peptide analogs are being developed as novel therapeutic strategies for IBD patients (Motilva et al, 2008).
- Retinoic acid and vitamin D are being studied for their possible effects in gut immunity and Crohn's disease (O'Sullivan, 2009).
- In MTHFR polymorphisms, use of L-methylfolate may be useful; Deplin is one prescription drug.

Herbs, Botanicals, and Supplements

- Use of complementary medicine is common in this population (Langhorst et al, 2005). Omega-3 fatty acids help to reduce symptoms of Crohn's disease. Alpha linoleic acid (ALA) works better at decreasing bowel inflammation than EPA and DHA. Fish oil supplements can cause side effects such as flatulence and diarrhea.
- Phytochemicals such as turmeric (curcumin), red pepper (capsaicin), cloves (eugenol), ginger (gingerol), cumin, anise, and fennel (anethol), basil and rosemary (ursolic acid), garlic (diallyl sulfide, *S*-allylmercaptocysteine, ajoene), and pomegranate (ellagic acid) can suppress some inflammatory pathways (Aggarwal and Shishodia, 2004).
- Probiotics may be beneficial; clinical studies are needed to verify the specific bacteria to be recommended. Studies have been performed with a variety of antioxidants, glutamine, short-chain fatty acids and prebiotics.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage patient to eat. Discuss fiber, fluid, and supplements.
- Periodic assistance or reevaluation by a qualified dietitian may be helpful. Alleviate fears associated with mealtimes.
- Ensure that sources of potassium are increased during periods of diarrhea.
- Instruct patient to chew foods well and avoid swallowing air.
- Bone density should be monitored yearly (Sylvester et al, 2007). Highlight calcium and vitamin D and discuss alternate sources when milk cannot be used. Promote exercise as well.
- Nocturnal TFs have been useful to regain weight or to promote growth. Total enteral nutrition with a liquid formula can suppress gut inflammation and induce remission in active Crohn's disease (Johnson et al, 2006; Newby et al, 2005).

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing diarrhea and related discomfort.
- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Colon Cancer Screening
  http://www.uwgi.org/guidelines/ch_08/ch08txt.htm
- Crohn's Disease
  http://digestive.niddk.nih.gov/ddiseases/pubs/crohns/
- Crohn's and Colitis Foundation of America
  http://www.ccfa.org/
- HealingWell Crohn's Disease Resource Center
  http://www.healingwell.com/ibd/
- National Association for Colitis and Crohn's Disease
  http://www.nacc.org.uk/content/home.asp
- Reach Out for Youth with Ileitis and Colitis
  http://www.reachoutforyouth.org/

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INFLAMMATORY BOWEL DISEASE: ULCERATIVE COLITIS

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

UC is a chronic inflammatory disease of the mucosa of the colon. Complications can include arthritis, severe skin rashes, endocarditis, cirrhosis, splenomegaly, and stomatitis. Patients with UC may have histological features such as microscopic inflammation of the ileum, histological gastritis, periappendiceal inflammation, patchiness, and relative rectal sparing at the time of diagnosis (North American Society for Pediatric Gastroenterology, 2007).

UC usually begins in the rectum or sigmoid colon. When UC affects only the rectum, it is called **ulcerative proctitis**. If the disease affects only the left side of the colon, it is called **distal colitis**; and if it permeates the entire colon, it is termed **pancolitis**. The condition is often a relentless, continuous lesion of the colon with some involvement of the terminal ileum. It does not affect full thickness of the intestine and never affects the small intestine. UC may be acute, mild, or chronic. **Indeterminate colitis** may be diagnosed with features of both UC and Crohn's disease; this is called **colitis of uncertain type or etiology** (Geboes et al, 2008).

The pathogenesis is complex and involves environmental, genetic, microbial, and immune factors. Onset of UC is usually between 15 and 35 years of age. There is a second, lesser peak of onset between ages 50 and 70 years. Intestinal microvascular ischemia usually precedes the onset of UC (Ibrahim et al, 2009). Blood in the stool is the most common symptom (Beattie et al, 2006) but children who present with UC may also have growth failure. Remissions occur, and there may be long periods between exacerbations. Flares often involve non-GI symptoms such as arthritis, uveitis, and ankylosing spondylitis. Because increased risk of colon cancer exists for the more extensive disease, patients with UC should be enrolled in a colonoscopy surveillance program after 8–10 years of disease duration (Rufo and Bousvaros, 2006).

Probiotic bacterial mixtures provide relief in mild-to-moderate UC by reducing the number of "bad" bacteria, reducing the amount of inflammation, increasing the mucus layer of the gut, and increasing the number of anti-inflammatory molecules in the intestine (Bibiolni et al, 2005). Researchers are looking at genetically altering *Bacteroides ovatus*, naturally in the gut, to secrete human growth factor KGF-2 protein when exposed to xylan sugar (Hamady et al, 2008). This may help to heal the damage caused by inflammation.

Both nutritional deficiencies and nutritional excesses impair GI responses and alter susceptibility to inflammation and other diseases. Enhancement of micronutrient nutrition is an important determinant of immunity and treatment of side effects in UC (Scrimgeour and Condlin, 2009).

Approximately 15% of patients with UC will develop a severe exacerbation requiring hospitalization (Doherty and Cheifiz, 2009). If medical management fails, surgical colectomy to remove the colon and rectum is curative. The ileocecal valve should be preserved where possible. Colectomy with creation of an ileal pouch anal anastomosis (J pouch) is standard for patients with severe or refractory colitis, resulting in an improved quality of life in most patients (Rufo and Bousvaros, 2006).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: IBD is associated with inheritance of several specific SNPs and gene variants that disrupt bacterial homeostasis mechanisms (Ferguson et al, 2007) and promote venous thrombosis (Bernstein et al, 2006). The ECM1 gene is found in UC (Jung and Hugit, 2009). Methionine synthase and MTHFR C677 T are also associated with extent of UC (Chen et al, 2008). Microflora in the intestinal lumen and an impaired innate immune system provoke a T helper 2 response, resulting in pro-inflammatory cytokines and interleukins (Festen et al, 2009). The IL-12-related cytokine, designated IL-23, establishes chronic inflammation and in the development of a Th cell subset producing IL-17, designated Th17 (Boniface et al, 2008; Shih and Targan, 2008).

Clinical/History		
Height	Temperature	Pus or mucus discharged between stools
Weight	Stool sample	
BMI	Bloody or explosive diarrhea	
Diet history	Crampy abdominal pain	Anemia, fatigue
		Anorexia

Biopsy
Sigmoidoscopy
Colonoscopy
Capsule endoscopy to rule out Crohn's

Lab Work

CRP
ESR
Perinuclear antineutrophilic cytoplasmic antibody (pANCA)
Anti–Saccharomyces cerevisiae antibody (ASCA)
Bilirubin
Chol, Trig
Na^+, K^+, Cl
BUN, Creat
Alb, RBP
Ca^{++}, Mg^{++}
Serum phosphorus, Alk phos
PT or INR
Transferrin, TIBC
N balance
Serum tHcy
Serum folate
Serum B_{12} and Schilling test
Genetic testing for ECM1 and MTHFR
H & H
Serum Fe, ferritin
Gluc
Complete blood count (CBC), ESR
WBC
Hydrogen breath test
Fecal fat study

INTERVENTION

OBJECTIVES

- In acute stages, allow the bowel to heal and use products that include short-chain fatty acids and glutamine to prevent a decline in nutritional status. Correct fluid and electrolyte imbalance.
- Develop an optimal regimen of pharmacologic therapies, nutritional management, psychologic support, and properly timed surgery (when necessary) to maintain disease remission, minimize disease and drug-induced adverse effects, and optimize growth and development (Rufo and Bousvaros, 2006).

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Nutrient Intake

Assessment Data: BMI 19, recent weight loss of 10 lb in 3 months, abdominal pain and bloody diarrhea at least once daily for 2 weeks. Diet hx reveals intolerance for high fiber fruits and vegetables and poor intake of protein-rich foods.

Nutrition Diagnoses (PES): Inadequate nutrient intake related to GI discomfort and maldigestion as evidenced by weight loss 10 lb in 3 months, intolerance of fruits and vegetables, poor intake of protein-rich foods.

Interventions: Food and nutrient delivery—change diet to low residue during flare. Educate about the role of diet in reducing inflammation; add omega-3 fatty acids and a multivitamin–mineral supplement once daily. Counsel about fiber-rich foods and when to avoid them (such as during times of GI distress). Discuss use of frequent small meals and intake of fruit and vegetable juices. Provide tips for adding protein and kilocals into the diet to regain lost weight. Discuss the option of using probiotic food sources.

Monitoring and Evaluation: Weight status; diet hx revealing improved intake from all food groups; fewer incidents of GI distress with use of the low residue diet, omega-3 fatty acid supplements, and appropriate medicines.

- Replenish depleted stores and correct poor nutritional status. Poor nutritional status may be related to decreased intake from anorexia, nausea or vomiting, abdominal pain, restrictive diets, side effects of medications, protein losses from ulcerated mucosal lesions, blood loss or wound-healing requirements, bacterial overgrowth, or malabsorption.
- Avoid further irritation of the bowel by managing fiber intake. Large fecal volume distends the bowel and could create obstruction. Correct for diarrhea, steatorrhea, obstruction, and related anemias.
- Provide sufficient dietary antioxidants and omega-3 fatty acids, which play a role in inflammatory processes. UC patients may be able to reduce steroid doses and achieve good symptom control by drinking a nutritionally balanced cocktail of fish oil, soluble fiber, and antioxidants daily.
- Prolonged use of corticosteroids, calcium and vitamin D deficiency, and a low BMI are some of the possible contributing factors to bone disease. Replenish as needed.
- Production of tHcy within the intestinal mucosa may contribute to the inflammatory response and endothelial cell dysfunction; treat folate and vitamin B_{12} deprivation as well as MTHFR polymorphisms, history of intestinal resection, or treatment with methotrexate (Peyrin-Biroulet et al, 2007). Prevent thrombotic events.

FOOD AND NUTRITION

- For adult energy requirements, estimate needs according to current BMI. A low BMI (<15) may require 35–45 kcal/kg; a BMI of 15–19 may require 30–35 kcal/kg; a BMI of 20–29 may require 25–30 kcal/kg; and a high BMI (>30) may only require 15–25 kcal/kg.
- Estimate needs at the high end of normal for growth and tissue repair in infants or children.
- To treat the condition in its acute state, a low-fiber diet is needed to minimize fecal volume. A nutritional supplement that contains fish oil, soluble fiber, and antioxidants reduces reliance on traditional therapies and lessens the need to start on corticosteroid therapy (Seidner et al, 2005).
- Persons with this condition often have lactose, wheat, or gluten intolerance. Alter diet accordingly.
- Dietary changes that have been suggested include use of less red meat, dairy products, artificial sweeteners, and caffeine. Controlled trials are needed to confirm efficacy of these changes.
- Diet should limit nuts, seeds, legumes, and coarse whole grains during a flare. Fresh fruits and vegetables may not be tolerated if they are highly fibrous; monitor carefully. A low-residue diet is useful during exacerbations.
- CPN is useful when needed, often for 2 weeks or longer during acute stages. CPN may be needed in long term if there is short-gut syndrome.
- As the patient progresses, a high-protein diet (1–1.5 g/kg) with high energy intake, given in six small feedings, is recommended. Protein may have to be restricted in patients with renal disease.
- Vitamin–mineral supplementation may be needed. Supplement the diet with multivitamins and minerals,

especially zinc, thiamin, folic acid, vitamin B_{12}, vitamin E, vitamin D, calcium, magnesium, and iron. Vitamins A and K can be given every other day. With resection greater than 200 cm, selenium may become deficient; monitor carefully.
- MCTs may be helpful. Use omega-3 fatty acid sources, such as salmon, mackerel, and tuna; supplements also may be beneficial.
- **After colectomy with ileostomy:** IV feeding should continue for 1–2 days. Diet should progress slowly to a low-fiber, high-protein regimen with high energy, vitamins, and minerals (especially sodium and potassium). The patient will need vitamin B_{12} injections and adequate fluid. CPN may be needed if progress is slow. Once liberalized, foods are added one at a time. Avoid gas-forming foods that may cause increased peristalsis; products to reduce flatulence may be helpful.

Common Drugs Used and Potential Side Effects

- The principal medical therapies used to induce disease remission in patients with UC are aminosalicylates for mild disease, corticosteroids for moderate disease, and cyclosporine for severe disease (Rufo and Bousvaros, 2006). Maintenance therapies that are used to prevent disease relapse include aminosalicylates, mercaptopurine, and azathioprine (Rufo and Bousvaros, 2006).
- Aminosalicylates (mesalamine, olsalazine, and sulfasalazine) contain 5-aminosalicylic acid (5-ASA) and reduce inflammation. Extra fluid intake is needed to avoid renal stone formation. anorexia, nausea, vomiting, and GI distress may occur. Folic acid supplements also may be required.
- Antibiotics such as metronidazole and ciprofloxacin reduce intestinal bacteria and directly suppress the intestine's immune system. They should be used with probiotics such as yogurt containing live and active cultures.
- Corticosteroids (prednisone, methylprednisolone, and budesonide) are used for patients with moderate-to-severe disease to reduce inflammation also. Although steroids can be quite effective for short-term control of acute episodes of colitis (flare-ups), they are not recommended for long-term use due to side effects. Negative nitrogen and calcium balances may result. Monitor the need for extra vitamins and minerals.
- Immunomodulatory medicines including azathioprine, 6-mercaptopurine (6-MP), 6-thioguanine (6-TG), and cyclosporine alter the immune cell interaction with the inflammatory process. They are used for patients when aminosalicylates and corticosteroids have been ineffective. Azathioprine and 6-MP may be useful in reducing dependence on corticosteroids and in maintaining remission in some patients. These medications take several months before their beneficial effects begin to work.
- Psyllium laxatives (Metamucil) can help with constipation and diarrhea. Long term use alters electrolytes. Flatulence or steatorrhea may occur.
- TNF-neutralizing agent infliximab (Remicade) appears to be an effective option for patients with UC that are not responding to conventional treatment.
- Therapies under investigation include tacrolimus and novel biologic drugs. However, there is the challenge of addressing potential safety issues, while more traditional drugs should be further developed to facilitate patient compliance (Pastroelli et al, 2009). The delivery system and frequency of medication administration should be resolved (Tindall, 2009).

Herbs, Botanicals, and Supplements

- Probiotics may be beneficial. Inulin and oligofructose have been suggested to increase the number of natural intestinal flora.
- Use of complementary medicine is common in this population (Langhorst et al, 2005). Further study is needed to evaluate whether glutathione or coenzyme Q10 affects prevention or treatment of IBD.
- Herbs and botanical supplements should not be used without discussing with the physician. Onion, cat's claw, boswellia, honeysuckle, peppermint, valerian, and tea have been recommended for this condition, but no clinical trials have proven efficacy.
- Omega-3 fatty acids may help to reduce symptoms of UC. ALA works better at decreasing bowel inflammation than EPA and DHA. Fish oil supplements may cause flatulence and diarrhea.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Ensure that the patient avoids foods that are known to cause diarrhea. Avoid extremes in food or beverage temperatures.
- Pleasant mealtimes are an important part of treatment. Frequent, small meals may increase the total nutritional intake. Discontinue eating 2–3 hours before bedtime.
- Avoid iced or carbonated beverages, which may stimulate peristalsis in times of discomfort.
- Instruct the patient to eat slowly and chew foods well. Discuss fears related to eating.
- Frequent counseling by a dietitian may be helpful.
- It has been noted that women with IBD tend to be childless or have fewer children. However, fears should be discussed, as adverse reproductive problems are not common (Mountifield et al, 2009).

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to this individual who may be experiencing diarrhea and related discomfort.
- Avoid excessive use of hand sanitizers, as a healthy gut contains some bacteria.
- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Crohn's and Colitis Foundation of America
  http://www.ccfa.org/research/info/aboutcd
  http://www.ccfa.org/info/diet?LMI=4.2

- FACSR
  http://www.fascrs.org/patients/conditions/ulcerative_colitis/
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
  http://digestive.niddk.nih.gov/ddiseases/pubs/colitis/index.htm
- National Institutes of Health—Ulcerative Colitis
  http://www.nlm.nih.gov/medlineplus/ulcerativecolitis.html

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INTESTINAL FISTULA

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

An intestinal fistula is an unwanted pathway from intestines to other organs (e.g., the bladder). External fistulas are between the small intestine and the outside (e.g., skin). Internal fistulas are between two internal organs. Most fistulas occur secondary to abdominal surgery, and a high proportion occurs in association with IBD, intestinal cancer, or trauma. In patients with a suspected fistula, CT followed by a colonoscopy helps to rule out malignancy.

Nutrition support may support spontaneous fistula closure. If spontaneous (nonoperative) closure does not occur in 5–6 weeks, it is unlikely to occur and an operation will be required (Osborn and Fischer, 2009).

GI cutaneous fistulas are among the more complex surgical conditions, with mortalities between 6% and 20%, and even up to 40% (Osborn and Fischer, 2009). Substantial morbidity is related to large fluid and electrolyte losses and metabolic disturbances; mortality is most often related to sepsis and malnutrition (Slater, 2009). Hypertriglyceridemia (266 mg/dL) is commonly observed with enterocutaneous fistulas; it is associated with sepsis, a high output small bowel fistula, nutrition by the parenteral route, and primary diseases with inflammatory etiology (Visschers et al, 2009).

Laparoscopic surgery may be a safe and effective procedure (Laurent et al, 2005), but surgery is performed only after 4–6 months of medical therapy. Fistuloclysis is a procedure in which nutrition is provided via an enteral feeding tube placed directly into the distal lumen of a high output fistula (Slater, 2009). The primary determinant of mortality after enterocutaneous fistula (ECF) repair is a failed operation leading to recurrence of the fistula, especially with IBD, fistula located in the small intestine, an interval of 36 weeks or longer between diagnosis and operation, and resection with stapled anastomosis (Brenner et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: A fistula is acquired in most cases.

Clinical/History		
Height	BMI	I & O
Weight	Diet history	Pain, tenderness
Weight loss	Temperature (Fever?)	Malaise
		CT scan

Fistulagram	Serum Fe, ferritin	RBP
Colonoscopy		BUN, Creat
Endosonography	Serum folate	Na^+, K^+, Cl^-
	N balance	Transferrin
Lab Work	Alb, transthyretin	Ca^{++}, Mg^{++}
H & H		

INTERVENTION

OBJECTIVES

- Promote rest and healing, minimize drainage from fistula, and prevent organ failure from sepsis.
- Monitor the type of dietary regimen according to the location of the fistula and surgical or medical treatment. Adjunctive steps following the operation usually include a gastrostomy and a catheter jejunostomy (Osborn and Fischer, 2009).
- Replace fluid and electrolyte imbalances.
- Decrease malnutrition and infections through aggressive nutritional support. Promote positive nitrogen balance. Additional surgery may be needed to drain infection.

FOOD AND NUTRITION

- A jejunostomy may help a duodenal fistula. A higher protein intake than usual may be needed. Use of an elemental formula diet also may be beneficial for an extended period of time to support GI tract recovery.
- Use CPN for jejunal fistulas. Monitor closely for hypertriglyceridemia.
- Progress to a low-residue, soft or normal diet as tolerated.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Parenteral Nutrition

Assessment Data: High output small bowel fistula, elevated triglycerides (>300 mg/dL), CPN for 10 days, 15 lb weight loss from Crohn's disease and poor intake over past year. BMI 19.

Nutrition Diagnoses (PES): Excessive intake from PN related to attempt to resolve high output small bowel fistula with CPN as evidenced by elevated triglycerides and use of CPN exceeding needs for 10 days.

Interventions: Calculation of current needs for protein, kilocals, and lipids to lower TG level without compromising fistula repair.

Monitoring and Evaluation: Weights, lab values (especially glucose and TG), gradual healing of fistula over 4–6 weeks without surgery, BMI improving slowly.

Common Drugs Used and Potential Side Effects

- Antibiotics commonly are used. Metronidazole and ciprofloxacin are useful.
- Infliximab has been used with some success for healing fistulas in IBD.
- Octreotide (somatostatin analog) inhibits endocrine/exocrine secretions and excessive GI motility. It is only used parenterally and can cause nausea, vomiting, abdominal pain, diarrhea, or flatulence.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Probiotic and prebiotic therapy may be useful.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Defined formula diets can help support spontaneous closure in approximately 4–6 weeks. If closure has not occurred, surgery is aided by better nutritional status.
- Instruct the patient regarding the fiber content of foods. Discuss how much to include during periods of flare-up and how to gradually increase fiber to achieve the goal set individually for that person.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to this individual.
- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- American Society of Colon and Rectal Surgeons
  http://www.fascrs.org/patients/conditions/anal_abscess_fistula/
- Anal fissure
  http://www.gicare.com/diseases/Anal-fissure.aspx
- Fistula
  http://www.nlm.nih.gov/medlineplus/ency/article/002365.htm

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INTESTINAL LYMPHANGIECTASIA

NUTRITIONAL ACUITY RANKING: LEVEL 3

DEFINITIONS AND BACKGROUND

Intestinal lymphangiectasia (IL) is a rare protein-losing enteropathy (PLE). It occurs most often in children, with 11 years as the average age of onset. IL can also occur secondary to conditions such as Crohn's disease, scleroderma, sclerosing mesenteritis, CD, lupus, lymphenteric fistula, constrictive pericarditis, or pancreatitis. IL is potentially fatal if not recognized and properly treated (McDonald and Bears, 2009). Increased intestinal lymphatic pressure with vessel dilatation occurs, discharging fluid into the bowel lumen. The fluid is then digested by intestinal enzymes and is reabsorbed. Massive fluid retention occurs from obstructed lymph vessels, especially in the abdomen and pleural cavities. While malabsorption and PLE occur, only marginal loss of protein occurs in most cases.

The main clinical features of this disorder include edema, fat malabsorption, lymphopenia, and hypoalbuminemia. Nausea, vomiting, nonbloody diarrhea, and abdominal pain result. Long-range problems may include lymphoma, osteomalacia or osteoporosis.

A low-fat diet associated with MCT supplementation is the cornerstone of medical management (Vignes and Bellanger, 2008). The need for dietary control appears to be permanent, because clinical and biochemical findings re-appear after low-fat diet withdrawal (Vignes and Bellanger, 2008).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Primary intestinal lymphangiectasia (PIL) is a rare disorder, generally diagnosed before 3 years of age; is it also called Waldman's disease (Vignes and Bellanger, 2008). Three genes, FLT4 (VEGFR3), FOXC2, and SOX18, cause varying forms of primary lymphedema (Finegold et al, 2008).

Clinical/History

- Height
- Weight
- BMI
- Weight loss, inability to gain weight
- Stunting?
- Diet history
- Fatigue
- Abdominal pain
- Steatorrhea
- Peripheral edema
- Diarrhea
- Chylous ascites?
- Double-contrast radiographs of the small bowel
- Ultrasound or CT scans
- Jejunal biopsy

Lab Work

- Fecal concentration of alpha-1 antitrypsin (>56 cc/d with diarrhea)
- Jejunal biopsy (dilated lymphatic lacteal vessels)
- Alb (decreased)
- Transthyretin
- Serum 25-OH-vitamin D and fat-soluble vitamins (low?)
- Trace metal deficiency
- Chol (normal or low)
- Trig
- Na^+, K^+
- H & H
- Serum Fe, ferritin
- Transferrin (decreased)
- TLC (Lymphocytopenia)
- Hypogammaglobulinemia
- Gluc
- Ca^{++} (low)
- Mg^{++}
- BUN, Creat

INTERVENTION

OBJECTIVES

- Identify and correct the underlying cause (e.g., constrictive pericarditis).
- Decrease symptoms and promote recovery. Minimize peripheral edema.
- Decrease intake of long-chain fatty acids because they form chylomicrons and stimulate lymphatic flow into the gut. Use MCTs because they are more water-soluble and can be absorbed through the portal vein instead of the lymphatic system.
- Meet all nutritional needs for age and sex. Monitor absorption of fat-soluble vitamins; ensure adequacy from dietary or supplemental sources.

FOOD AND NUTRITION

- Reduce intake of long-chain fatty acids. A formula or low-fat diet using a high concentration of MCTs is useful.
- Adequate protein and calories are needed, according to the individual's needs.
- Fat-soluble vitamins may be required in water-miscible form for adequate absorption.
- A calcium supplement may also be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Inappropriate Intake of Types of Fats

Assessment Data: Peripheral edema, weight loss, diarrhea, fecal alpha-1 antitrypsin >60 cc/d, low albumin (3.0 g/dL)

Nutrition Diagnoses (PES): Inappropriate intake of fatty acids (long-chain) related to intolerance of long-chain fatty acids and IL as evidenced by diarrhea, weight loss, altered labs (albumin, alpha-1 antitrypsin).

Interventions: Food-Nutrient Delivery—reduce intake of long-chain fatty acids and use MCTs instead. Education—teach diet principles. Counseling about the diet for managing the life-long condition.

Monitoring and Evaluation: Improvements in edema, lab values, and diarrhea after following the diet and taking appropriate medication.

Common Drugs Used and Potential Side Effects

- Octreotide (Sandostatin) may be used. It is a potent inhibitor of growth hormone, glucagons, and insulin; it also suppresses gastrin, motilin, secretin, and pancreatic polypeptide.
- Over-the-counter remedies (bulking agents, drugs to control diarrhea) may be useful.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Supplements of vitamins and calcium are generally prescribed.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of fat in digestion, along with the need for MCT oils in the daily diet.
- Discuss fat-soluble vitamins and their sources in the diet.
- To minimize peripheral edema, elevation of extremities above the head may be recommended to decrease cellulitis and lymphangitis. In addition, use of a recliner or elastic support stockings is suggested.
- It is not helpful to reduce salt intake or to use diuretics in this condition.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Intestinal Lymphangiectasia
  http://www.emedicine.com/med/topic1178.htm
- Medscape
  http://emedicine.medscape.com/article/1086917-overview
- Merck Manual
  http://www.merck.com/mmhe/sec09/ch125/ch125f.html

INTESTINAL LYMPHANGIECTASIA—CITED REFERENCES

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McDonald KQ, Bears CM. A preterm infant with intestinal lymphangiectasia: a diagnostic dilemma. *Neonatal Netw.* 28:29, 2009.
Vignes S, Bellanger J. Primary intestinal lymphangiectasia (Waldmann's disease). *Orphanet J Rare Dis.* 3:5, 2008.

INTESTINAL TRANSPLANTATION

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

Intestinal transplantation (ITx) represents a difficult life-saving intervention reserved for patients with irreversible intestinal failure (Varga et al, 2009). Intestinal failure leads to the inability to maintain protein energy, fluid, electrolyte, or micronutrient balance due to GI disease when on a normal diet. Transplantation is an effective therapy for the treatment of patients with end-stage intestine failure who cannot tolerate PN (Grant et al, 2005). The procedure should be considered before the development of PN failure (Matarese et al, 2007). PN-associated liver disease, recurrent catheter-related sepsis, and threatened loss of central venous access are the major reasons for transplantation. SBS, cancers, radiation enteritis, trauma, Crohn's disease, or other major intestinal diseases may also warrant ITx.

Transplantation of the small bowel restores quality of life for recipients who have functioning grafts. ITxs should be considered early in intestinal failure patients who develop liver injury to prevent irreversible liver disease that would mandate a simultaneous liver transplantation (Fryer, 2005). Medicare pays for ITx in patients who fail PN therapy in specific cases. Living-related donor transplantation is an option if a potential donor is available. The donor should have no history of liver disease or major intestinal pathology.

The carbohydrate and amino acid absorptive capacity of the transplanted intestine normalize within the first several months whereas fat absorption is impaired for several months. Morbidity and mortality following ITx are greater than that following liver or kidney transplantation, but long-term survival is improving. Infectious enteritis can occur in recipients after ITx; viral agents are the cause in most cases (Ziring et al, 2005). With newer immune-suppressive protocols, 1-year graft and patient survival rates have improved. Surgery and high-dose immunosuppression must be managed. Radiation to the small bowel before transplanting the organ, then administration of donor's bone marrow stem cells may reduce organ rejection. A loop ileostomy is often created for future endoscopy. Patients with ITx will require regular expert follow-up care and careful attention.

A serious complication of ITx is jejunal graft (JG) damage (Varga et al, 2009). Induction therapy, combined with advancements on surgical technique and clinical management, has improved patient and graft survival (Vianna and Mangus, 2009). An organized multidisciplinary approach is recommended for long-term follow-up.

Early and progressive enteral feeding using a complex polymeric formula is safe and effective after successful transplantation, eventually followed by unrestricted oral diet (Matarese et al, 2007). This modality of treatment is an alternative to PN, especially for those patients who have a poor quality of life as a result of PN (Middleton, 2007).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Transplantation is a surgical procedure for a variety of conditions, some of which may be genetic.

Clinical/History

- Height
- Dry weight, present weight
- BMI
- Diet history
- BP
- I & O
- Temperature
- Wireless capsule endoscopy (CE)
- Colonoscopy
- CT scan to monitor for fistula or obstruction
- Doppler ultrasonography
- Liver biopsy

Lab Work

- Alb, transthyretin
- CRP
- CBC count
- Coagulation profile
- Human leukocyte antigen (HLA) status
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- H & H
- Serum Fe, ferritin
- Serum folacin
- BUN, Creat
- Glomerular filtration rate (GFR)
- WBC, TLC
- Gluc
- Chol, Trig
- N balance
- Alk phos, phosphorus
- AST, ALT
- Bilirubin
- Serum pH and lactate

INTERVENTION

OBJECTIVES

- Timely nutrition assessment and intervention may improve outcomes. Recovery of normal motility and absorptive capacity are the goals after surgery.
- Prevent infection and promote wound healing.
- Replenish lost nutrient stores as malnutrition compromises posttransplantation survival.
- Meet metabolic demands and support recovery.
- Control complications. Diarrhea and high stomal output are common problems and can lead to nutrient deficits, especially electrolytes.
- Supplement enteral feedings with MCTs for several months posttransplantation.
- Supplement the diet with IV fats and fat-soluble vitamins (vitamin D, E, A, and K) until the intestinal lymphatics are reestablished.

SAMPLE NUTRITION CARE PROCESS STEPS

Altered GI Function

Assessment Data: Recent ITx following failure to tolerate PN; hx Crohn's disease; unintentional weight loss over past year.

Nutrition Diagnoses (PES): Altered GI function related to intestinal surgery as evidenced by intolerance of oral, enteral and PN with poor nutrition quality of life.

Interventions: Food and nutrient delivery—clear liquids, progressing slowly to enteral feedings that contain MCT. Progress gradually to oral diet. Educate about the transition period from enteral to oral diet. Counsel about potential problems, such as diarrhea, electrolyte depletion, vitamin and mineral deficiencies; teach about products or medications that may be needed.

Monitoring and Evaluation: Tolerance of enteral feedings post surgery. Resolution of diarrhea, high stomal output, abnormal labs. Achievement of desirable body weight range.

FOOD AND NUTRITION

- When GI function is re-established, as indicated by decreasing G-tube returns and increasing gas and enteric contents in the ileostomy, a diet can be initiated. Advance as tolerated to provide full nutritional support. Monitor fluid status and adjust as needed.
- Daily intake of protein should be appropriate for age and sex; 1.5 g/kg while on steroids may be recommended. Calories should be calculated as 30–35 kcal/kg.
- Control CHO intake and encourage use of whole grains, vegetables, and fruits in cases of hyperglycemia.
- To avoid the development of chylous ascites, a no-fat or low-fat diet can be initially used. Gradually increase fat intake to 25–30% of total kilocal.
- Daily intake of sodium should be 2–4 g until the drug regimen is reduced. Adjust potassium levels as needed.
- Daily intake of calcium should be 1–1.5 times the DRI levels to offset poor absorption. Children especially need adequate calcium for growth. Daily intake of phosphorus should be equal to calcium intake.
- Supplement diet with vitamin D, magnesium, and thiamin if needed.
- Reduce gastric irritants as necessary if GI distress or reflux occurs.
- The special diet may be discontinued when drug therapy is reduced to maintenance levels. Encourage exercise and a weight control plan thereafter.

Common Drugs Used and Potential Side Effects

- See Table 7-18.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Grapefruit juice decreases drug metabolism in the gut (via P-450–CYP3A4 inhibition). One glass can affect medications up to 24 hours later. Avoid taking with cyclosporine and tacrolimus.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Indicate which foods are sources of protein, calcium, and other key nutrients in the diet.

TABLE 7-18 Medications Used after Intestinal Transplantation

Education	Description
Antibiotics	Broad-spectrum intravenous antibiotics are administered for about 1 week after the transplant.
Antiviral prophylaxis	Ganciclovir and/or cytomegalovirus (CMV) immunoglobulin (CytoGam) may be used.
Corticosteroids (prednisone or Solu-Cortef)	Corticosteroids such as prednisone and Solu-Cortef are used for immunosuppression. Side effects include increased catabolism of proteins, negative nitrogen balance, hyperphagia, ulcers, decreased glucose tolerance, sodium retention, fluid retention, and impaired calcium absorption and osteoporosis. Cushing's syndrome, obesity, muscle wasting, and increased gastric secretion may result. A higher protein intake and lower intake of simple CHOs may be needed.
Cyclosporine	Cyclosporine does not retain sodium as much as corticosteroids do. Intravenous doses are more effective than oral doses. Nausea, vomiting, and diarrhea are common side effects. Hyperlipidemia, hypertension, and hyperkalemia may also occur; decrease sodium and potassium as necessary. Elevated glucose and lipids may occur. The drug is also nephrotoxic; a controlled renal diet may be beneficial. Taking omega-3 fatty acids during cyclosporine therapy may reduce the toxic side effects (such as high blood pressure and kidney damage) associated with this medication in transplantation patients.
Immunosuppressants	Immunosuppressants such as muromonab (Orthoclone OKT3) and antithymocyte globulin (ATG) are less nephrotoxic than cyclosporine but can cause nausea, anorexia, diarrhea, and vomiting. Monitor carefully. Fever and stomatitis also may occur; alter diet as needed.
Induction Therapy	Monoclonal (alemtuzumab, basiliximab, daclizumab) or polyclonal (Thymoglobulin) antibody preparations are often administered intraoperatively or preoperatively.
Probiotics, Prebiotics	Although research is still preliminary, the use of probiotics and prebiotics may become common practice in the future.
Prostaglandin E_1	Prostaglandin E_1 is given to improve the small bowel microcirculation.
Tacrolimus (Prograf, FK506)	Tacrolimus suppresses T-cell immunity; it is 100 times more potent than cyclosporine, thus requiring smaller doses. Side effects include GI distress, nausea, vomiting, hyperkalemia, and hyperglycemia.

- If the patient does not drink milk or use dairy products, discuss other sources of calcium.
- Alcohol should be avoided unless permitted by the doctor.
- Discuss control of hyperglycemia when appropriate. Discuss long-term problems such as obesity and dyslipidemia.
- Encourage moderation in diet; promote adequate exercise.
- Financial constraints can be a burden. Home care has evolved, but many disciplines are needed to keep everything in order.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing graft–host rejection.
- Prevent infections from foodborne illness; patients who have undergone transplantation may be prone to increased risk more than other individuals.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Intestinal Transplantation
  http://www.emedicine.com/ped/topic2845.htm
- Intestinal Transplant Centers
  http://www.intestinaltransplant.org/centres.htm
- Intestinal Transplant Registry
  http://www.intestinaltransplant.org/

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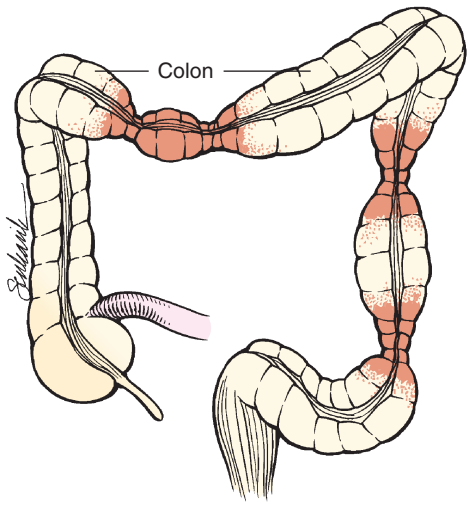
Vianna RM, Mangus RS. Present prospects and future perspectives of intestinal and multivisceral transplantation. *Curr Opin Clin Nutr Metab Care.* 12:281, 2009.

Ziring D, et al. Infectious enteritis after intestinal transplantation: incidence, timing, and outcome. *Transplantation.* 79:702, 2005.

IRRITABLE BOWEL SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 3



DEFINITIONS AND BACKGROUND

Irritable bowel syndrome (IBS) is a functional GI disorder affecting up to 3–15% of the general population in western countries (Camilleri and Andresen, 2009). The pathophysiology of IBS involves disturbances of the brain–gut axis (Camilleri and Andresen, 2009). Stimulated 5-HT3 receptors promote intestinal motility, secretion, and sensation. Approximately one in ten patients with IBS believe their IBS began with an infectious illness (Spiller and Garsed, 2009). Prospective studies have shown that 3–36% of enteric infections lead to persistent new IBS symptoms; bacterial enteritis, protozoan, and helminth infections are often followed by prolonged postinfective IBS (Spiller and Garsed, 2009). Rome III Diagnostic criteria for IBS include at least 3 months of continuous or recurrent symptoms of abdominal pain or discomfort relieved with defecation or associated with change in frequency of stool or changed consistency of stool. Individuals with IBS often have had a prolonged initial illness, a toxic infecting bacterial strain, history of smoking, mucosal markers of inflammation, female gender, depression and adverse life events in the preceding 3 months (Spiller and Garsed, 2009).

Because 95% of the body's serotonin is located in the GI tract, it is important to note that people with IBS have diminished receptor activity. There are several subgroups of IBS: alternating bowel habits (IBS-A), constipation-predominant IBS (IBS-C), and diarrhea-predominant IBS (IBS-D). Signs that IBS patients require medical attention include anemia, fever, persistent diarrhea, rectal bleeding, weight loss, and nocturnal symptoms. In addition, a family history of IBD, CD, or colorectal cancer requires further diagnostic work-up.

Up to 65% of IBS patients attribute their symptoms to food allergies (Zar et al, 2005). However, this finding has not been confirmed (Brant et al, 2009). Some IBS sufferers may have a mild form of CD and should be tested for gluten intolerance. Others may have lactose maldigestion. A food diary is useful to note symptoms and specific foods or practices that cause problems.

Therapies focus on specific GI dysfunctions (e.g., constipation, diarrhea, pain), and medications should only be used when nonprescription remedies do not work or when symptoms are severe. In some IBS patients, there is *H. pylori* infection; appropriate antibiotics may relieve symptoms. Therapies focus on nerve–gut communication dysfunction and antibiotics. Probiotics are another important therapy. Symptoms may also be improved by diets supplemented with probiotics such as hydrolyzed guar gum (Hadley and Gaardner, 2005) or bifidobacteria. Where carbohydrate malabsorption (lactose, fructose, or sorbitol) occurs, restrict the offending sugar(s). Overall, there is a reduction in quality of life (Brant et al, 2009). The American Dietetic Association has recommended three MNT visits for patients who have IBS.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Genetic modeling has identified a genetic link for IBS in twin studies (Lembo et al, 2007). However, the heritable components are poorly understood (Camilleri and Andresen, 2009).

Clinical/History

- Height
- Weight
- Weight changes
- BMI
- Diet history
- Family history of GI disorders
- Recurrent abdominal pain or discomfort
- Change in frequency of stool
- Change in form of stool
- Belching, flatulence, heartburn
- Nausea
- Mucus in stool?
- Polycystic ovarian syndrome?
- Urgency for defecation
- Lower GI x-rays
- Colonoscopy or sigmoidoscopy

Lab Work

- tTG testing to rule out CD
- Hydrogen breath test for lactose tolerance
- CRP
- H & H
- Serum Fe, ferritin
- Alb, transthyretin
- Gluc
- Na^+, K^+
- Ca^{++}, Mg^{++}

INTERVENTION

OBJECTIVES

- Encourage regular eating patterns, regular bowel hygiene, adequate rest, and relaxation.

SAMPLE NUTRITION CARE PROCESS STEPS

Undesirable Food Choices

Assessment: Diet history with analysis of fiber, caffeine, fluid, and CHOs; family history and medication/food allergies; bowel patterns.

Nutrition Diagnosis (PES): Undesirable food choices related to excessive intake of colas and coffee as evidenced by abdominal pain each day, symptoms of IBS-D, and poor nutritional quality of life.

Intervention: Educate about better fluid choices, probiotics, prebiotics, fiber, reduction in gas-forming foods.

Monitoring and Evaluation: Dietary and fluid intake changes; changes in abdominal pain, bowel patterns, frequency of diarrhea. Improved enjoyment of meals and a variety of foods and beverages.

- Avoid constipation by increasing physical activity and consuming adequate fluids and fiber (American Dietetic Association, 2008).
- Monitor for food intolerances to gluten in wheat, rye, barley; chocolate; lactose or milk products; caffeine; alcohol. Omit offending agents.
- Individualize diet management to the patient's symptoms. Alleviate pain, symptoms, and flatulence. Production of colonic gas, especially hydrogen, may be uncomfortable.
- Improve nutritional quality of life.

FOOD AND NUTRITION

- Fiber is marginally beneficial; insoluble fiber may worsen symptoms but soluble fiber may help to alleviate constipation (Heizer et al, 2009). In acute phases, a low-fiber diet may be better tolerated. As treatment progresses, use adequate but not excessive fiber and ensure adequate fluid intake (30–35 mL/kg).
- Avoid high-fat foods, which may increase cholecystokinin release. Avoid high sugar intake, which increases osmolarity.
- Liberal amounts of fruits and vegetables are useful. Omit gas-forming oligosaccharides (beans, barley, Brussels sprouts, cabbage, nuts, figs, and soybeans), if not tolerated. Limit or omit spicy foods if poorly tolerated.
- A modified exclusion diet and stepwise reintroduction of foods or trials of eliminating classes of food may be useful (Heizer et al, 2009). Omit milk products if lactose is not tolerated; add calcium in other forms.
- In patients with CD, omit gluten and supplement with B-complex vitamins if needed.

Common Drugs Used and Potential Side Effects

- 5-HT is a key modulator of GI sensorimotor function and this has led to the development of 5-HT(3) antagonists and 5-HT(4) agonists; alosetron (Lotronex), cilansetron, and tegaserod are all effective in the treatment of IBS (Ford et al, 2009). If constipation results, a lower dose may be needed (Krause et al, 2007).
- Antidepressants, novel selective anticholinergics, alpha-adrenergic agonists, opioid agents, cholecystokinin-antagonists, neurokinin-antagonists, somatostatin receptor agonists, corticotropin releasing factor antagonists, chloride channel activators, guanylate cyclase-c agonists, melatonin, atypical benzodiazepines, antibiotics, immune modulators are under study (Camilleri and Andresen, 2009). Many of these have mixed reviews at the present time.
- Methylcellulose (Metamucil) and other bulking agents always must be taken with large amounts of water. Generally, 1 tablespoon is sufficient per day. Increased peristalsis occurs.

Herbs, Botanicals, and Supplements

- Daily use of peppermint oil is effective in relieving IBS symptoms (Hadley and Gaardner, 2005; Heizer et al, 2009).
- The usefulness of probiotics in the form of foods such as live-culture yogurt and buttermilk for IBS symptoms is not established (Heizer et al, 2009). Two double-blind, randomized, placebo-controlled studies identified that *Bifidobacterium infantis* provides relief from IBS (Brenner and Chey, 2009; Spiller, 2005). Yogurt and other cultured foods may not be as effective as specific probiotics with high concentrations of microbes, but they are good sources of nutrients.
- To prevent intestinal gas from forming, try Beano and other products available on the market.
- Twice-weekly Acupuncture/Moxabustion treatment improves average daily abdominal pain and discomfort (Anastasi et al, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Slowly increase dietary fiber by 2–3 g/d to prevent discomfort and to promote soft, painless stools. Large servings of bran may aggravate IBS; assess individually.
- IBS does not harm the intestines and does not lead to cancer; it is unrelated to IBD.
- Drink six to eight glasses of water or fluids per day. Avoid carbonated beverages, chewing gum, or eating quickly, which may promote gas production.
- Regular times for bowel evacuation should be planned.
- Ensure the patient has adequate food intake and is not afraid to eat because of potential pain.
- A food diary may help to identify any food sensitivities.
- Large meals can cause cramping and diarrhea; smaller meals or smaller portions eaten more often may help IBS symptoms. Meals that are low in fat and high in carbohydrates such as pasta, rice, whole-grain breads and cereals (except with CD), fruits, and vegetables may help.
- Refer the patient for stress management. Patients with rapidly cycling symptoms may need further counseling and support (Tillisch et al, 2005). Cognitive behavioral therapy is beneficial.

- Family members may need to flexible and help to create regularity in the home. Avoid disorganization, overscheduling, lack of planning.
- Regular exercise is important, such as walking, swimming, or yoga. Adequate sleep is important as well.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing diarrhea and related discomfort.

For More Information

- American College of Gastroenterology—IBS
  http://www.acg.gi.org/patients/ibsrelief/
- E-Medicine Health
  http://www.emedicinehealth.com/irritable_bowel_syndrome/article_em.htm
- IBS Treatment Matrix
  http://www.acg.gi.org/patients/ibsrelief/treatmentmatrix/index.asp
- International Foundation for Functional GI Disorders
  http://www.iffgd.org/
- Living with IBS
  http://www.iamibs.org/
- Mayo Clinic
  http://www.mayoclinic.com/health/irritable-bowel-syndrome/DS00106
- National Digestive Diseases Information Clearinghouse
  http://digestive.niddk.nih.gov/ddiseases/pubs/ibs_ez/
  http://digestive.niddk.nih.gov/ddiseases/pubs/ibs_ez/IBS.pdf
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LACTOSE MALDIGESTION

NUTRITIONAL ACUITY RANKING: LEVEL 2–3

DEFINITIONS AND BACKGROUND

Lactose is a disaccharide (glucose-1 galactose) found in milk. If lactase enzyme is missing, lactose passes into the colon, where it is fermented to gases and organic acids by colonic bacteria, resulting in bloating, cramping, nausea, or diarrhea.

A limited proportion of the human adult population retains intestinal lactase-phlorizin hydrolase (LPH) activity during adulthood, the lactase persistence phenotype (Robayo-Torres and Nichols, 2007). Lactase persistence is an autosomal-dominant trait that is common in European-derived populations (Smith et al, 2009). A staggering 4000 million people cannot digest lactose properly (Campbell et al, 2005). As many as 75% of all African American, Jewish, Native American, and Mexican American adults and 90% of Asian American adults are lactose intolerant. A total of 95% of all adults have adult-type hypolactasia (ATH) and have difficulty digesting milk sugar (Robayo-Torres and Nichols, 2007). Table 7-19 describes the various types of this condition.

CD can lead to lactase deficiency (Ojetti et al, 2005). An increased prevalence of lactose intolerance is also seen in patients with IBS. In addition, fructose intolerance may be unrecognized, and testing should clarify which disaccharide

TABLE 7-19 Types of Lactose Maldigestion

Type	Description	Incidence
Congenital, primary, or genetic	Rare, present at birth	Low incidence in children
Lactase "nonpersistence" or hypolactasia	Lactase decline, often to about 10% of neonatal values	Occurs after weaning; more common in adults
Secondary or acquired	From gastrointestinal disease, food allergy, antibiotics, or intestinal trauma	May occur in children after diarrhea or giardiasis. Common with HIV infection, inflammatory bowel disease.

is poorly tolerated. Children with suspected lactose intolerance can be assessed clinically by dietary lactose elimination or by tests including noninvasive hydrogen breath testing or invasive intestinal biopsy determination of lactase (and other disaccharidase) concentrations. (Heyman and Committee on Nutrition, 2006).

Lactose maldigesters can consume up to 1 cup (8 oz) of milk without experiencing symptoms; tolerance can be improved by consuming the milk with a meal, choosing yogurt or hard cheeses, or using products that aid in the digestion of lactose such as lactase supplements or lactose-reduced milks (Byers and Savaiano, 2005). Symptoms caused by lactose maldigestion need not hinder ingestion of a diet rich in dairy products that supplies around 1500 mg calcium daily (i.e., 2 cups of milk, 1 cup of yogurt, and several ounces of cheese). Bloating, abdominal pain, diarrhea, and overall symptom severity are often tolerable.

Pregnant women, children and teens who are lactose intolerant should be given appropriate counseling about intake of calcium from nonlactose sources. Individuals who are self-described as lactose intolerant may restrict dairy and calcium intake and are at greater risk of osteoporosis and bone fractures. The American Dietetic Association recommends at least three MNT visits for adults who have lactose maldigestion.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Congenital lactase deficiency (CLD) occurs because of an SNP upstream from the lactase (C-13910 > T) gene (Ingram 2009).

Clinical/History

Height
Weight
BMI
Weight changes
Lactose challenge test
Hydrogen breath test (3–5 hours)
Stool acidity test

Lab Work

Alb, transthyretin
BUN, Creat
Ca^{++} (better absorption can occur over time)
Mg^{++}
Alk phos
Gluc
H & H
Serum Fe, ferritin
Na^{+}, K^{+}

INTERVENTION

OBJECTIVES

- Manage pain and discomfort related to lactose ingestion that is caused by flatulence and bloating or diarrhea. Control lactose intake, which comprises 10% of the carbohydrate found in the American diet.
- Regular consumption of milk by lactase-deficient persons may improve colonic tolerance. Check for actual tolerance by monitoring intake.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Cho (Lactose) Intake

Assessment: Food diary and records with analysis of grams of lactose consumed on average day, stool frequency, and GI complaint reports.

Nutrition Diagnosis (PES): Excessive CHO (lactose) intake related to poor food choices as evidenced by frequent use of liquid dairy products, ice cream and milkshakes with resulting abdominal pain and diarrhea.

Intervention: Counseling on lactose-restricted diet, use of Lact-Aid and lactose-reduced products in meals and in cooking.

Monitoring and Evaluation: Food records and reports of GI distress or diarrhea after diet alterations have been implemented consistently for at least 7 days.

- Offer calcium and riboflavin from other foods and sources besides lactose-containing dairy products.

FOOD AND NUTRITION

- Most people can tolerate up to 6 g of lactose, which is found in ½ cup (4 oz) of fluid milk. If small amounts are gradually added over approximately 3 months, most adults can ultimately adapt to 12 g lactose, equal to one 8-oz glass of regular milk.
- Dairy foods contain approximately 1–8% lactose by weight (milk, 4–5%; yogurt, 4%; ice cream, 3–4%; milk chocolate, 8%; cottage cheese, 1–2%). Because symptoms are related to dose, consume no more than 8 oz of lactose-containing milk at a time after other foods have been consumed to slow down transit time. See Table 7-20.
- Provide lactase enzyme supplements (e.g., Lactaid, Lactrase, Dairy Ease) 30 minutes before the consumption of a lactose-containing product. Two capsules provide enough lactase to hydrolyze the lactose in an 8-oz glass of whole milk. Lactose-hydrolyzed milk is generally tolerated. Note that not all preparations are equally effective.
- Persons on a lactose-free diet can use lactate, casein (curds), lactalbumin, and calcium. If the patient is highly sensitive, check labels of foods for fillers, whey protein, milk, whey solids, and milk solids; most people can tolerate small amounts in mashed potatoes, breads, and medications. Processed cheese or cheese foods may have nonfat dry milk solids; use in moderate amounts.
- Fermented products (buttermilk, natural or aged cheese, yogurt with live and active cultures, cottage cheese, or sour cream) may be better tolerated than milk by many individuals.
- Frozen yogurt has little or no lactase activity. It may be tolerated in small amounts.
- For infants with the condition, try milk-free formulas; gradually introduce foods that contain milk or lactose to test for tolerance. Distinguish milk allergy (usually early in life) from lactose maldigestion (more often in adults).
- Secondary lactase deficiency results from injury to the small intestine that occurs with severe diarrheal illness, CD, Crohn's disease, or chemotherapy; this is more common in infancy.

TABLE 7-20 Lactose and Substitutes in Common Foods

Food and Portion	Portion	Lactose (g)
Evaporated milk	1 cup	24
Sweetened condensed milk	½ cup	20
Milk, reduced fat	1 cup	11–14
Acidophilus Milk	1 cup	11
Yogurt, whole milk	1 cup	10–12
Buttermilk	1 cup	10
Ice cream	½ cup	6
Yogurt, plain, low fat	1 cup	5
Sour cream or light cream	½ cup	4
Cottage cheese	½ cup	3 creamed, 2 dry
Cheese, hard	1 oz	1–2
Swiss cheese	1 oz	1
Cream cheese	1 oz	1
Butter or margarine	1 tsp	Trace

Recipe Substitutes for Dairy Products

- 1 cup whole milk = mix ½ cup water with ½ cup nondairy cream or use 1 cup soy or rice milk
- 1 cup skim milk = ¼ cup nondairy cream plus ¾ cup water
- 1 cup evaporated milk = 1 cup nondairy cream or soy milk
- 1 cup buttermilk = ½ cup nondairy cream plus ½ cup water + 1 tbsp lemon juice or vinegar
- 1 cup whipped cream = 1 cup nondairy whipped topping
- 1 tbsp cream cheese = 1 tbsp mayonnaise
- ½ cup cottage cheese = ½ cup tofu
- 1 tbsp butter = 1 tbsp milk-free margarine or 1 tbsp vegetable oil
- 1 cup sour cream = ¼ cup cornstarch in ¾ cup water plus ¼ cup vinegar

Common Drugs Used and Potential Side Effects

- Many drugs contain lactose but seldom over 500 mg; most should be well tolerated. For example, some types of birth pills, as well as tablets for stomach acid and gas may contain lactose.
- Antidiarrheal agents such as loperamide (Imodium A-D) help reduce symptoms of lactose intolerance.

Herbs, Botanicals, and Supplements

- Probiotics (such as lactobacilli and bifidobacteria) and prebiotics (nondigestible oligosaccharides) assist in alleviating lactose intolerance and improving calcium absorption (Doron and Gorbach, 2006).
- Take calcium supplements containing calcium carbonate with meals because stomach acid enhances absorption. Calcium citrate can be taken with meals or on an empty stomach. Take calcium in doses of 500 mg several times a day rather than all at once.
- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Identify foods that are lactose free and foods that are lactose-free sources of calcium.
- Reading labels is helpful. Store-bought cookies, cakes, bread, baked goods, cereals, instant potatoes, soups, margarine, lunch meat, salad dressings, pancakes, biscuits, nondairy creamers or whipped toppings, and candy may contain lactose.
- The patient must read labels, looking for and avoiding "milk," "lactose," butter, cheese, cream, milk solids, powdered milk, and whey.
- Home-cooked meals and lactose-free recipes are useful. Recipes are available for use of lactose-free formulas in products such as meat loaf. Advise that heating of milk does not change lactose.
- If dairy products are eliminated, other dietary sources of calcium or calcium supplements need to be provided, especially for infants, children, teens (Heyman and Committee on Nutrition, 2006), and pregnant women. Calcium-fortified soy milk, broccoli, leafy greens, canned salmon, almonds, oranges, certain kinds of tofu and soy milk, and calcium-fortified breads and juices can be used.
- Kosher foods are often acceptable if they are pareve (nonmilk, nonmeat).
- Discuss how to use LactAid drops to allow the enzyme to hydrolyze the lactose. Five to 15 drops per quart of milk will reduce lactose by 70–99%.
- Drink milk with meals rather than alone to decrease symptoms.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing diarrhea and related discomfort.

For More Information

- Dairy-Ease
  http://www.dairyease.com/benefits/
- Lactose-Free Diet
  http://www.gicare.com/pated/edtgs05.htm
- Lactose Intolerance
  http://digestive.niddk.nih.gov/ddiseases/pubs/lactoseintolerance/
- Lactose Intolerance Glossary
  http://www.medicinenet.com/lactose_intolerance/glossary.htm
- Lactose Diet
  http://www.cpmc.org/advanced/pediatrics/patients/topics/lactosefree.html
- LactAid (McNeil Consumer Products)
  http://www.lactaid.com/
- LactAid recipes
  http://lactaid.allrecipes.com/
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
  http://digestive.niddk.nih.gov/ddiseases/pubs/lactoseintolerance_ez/#food

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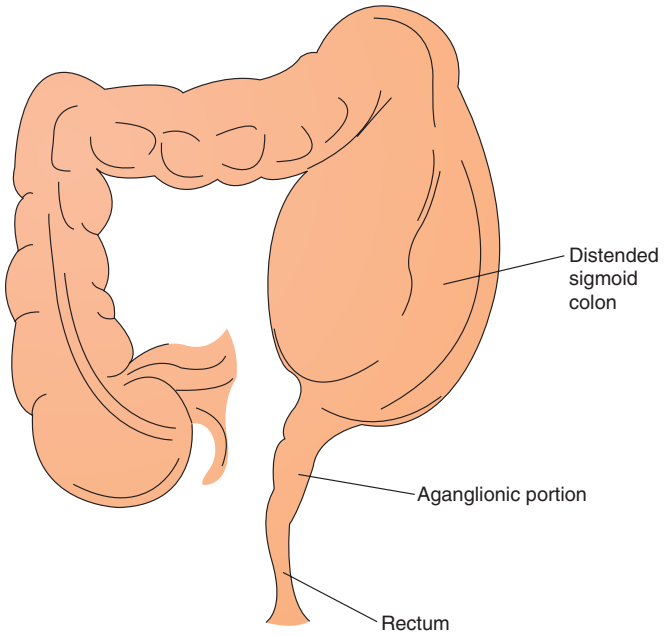
MEGACOLON

NUTRITIONAL ACUITY RANKING: LEVEL 3



Adapted from: Pillitteri, A. *Maternal and Child Nursing,* 4th ed. Philadelphia: Lippincott, Williams & Wilkins, 2003.

DEFINITIONS AND BACKGROUND

Megacolon may come in three forms: acute (before age 1), chronic (after age 10), or toxic. Acquired megacolon is associated with chronic constipation. The enlarged bowel results from an abnormal colonic dilatation often reaching 8–10 cm in diameter. It may occur in elderly persons who have a long history of elimination problems created by laxative abuse or constipation. Persons with diabetes, hypothyroidism, scleroderma, spinal cord injury, Parkinson's, multiple sclerosis, electrolyte imbalances, tumor, strictures, and other conditions may be affected. Worldwide, infection with *Trypanosoma cruzi* (Chagas disease) is the major cause of megacolon.

Normal urges to defecate are affected by physical activity, neurological status, chemical/drug use, and bowel condition. Normal reflexes are needed for muscular and sphincter control. Family physicians must be alert for the presence of uncommon but serious megacolon (Biggs and Dery, 2006). Signs and symptoms of megacolon involve abdominal distention, flatus, absence of stool, smearing or bowel incontinence, nausea, anorexia, fatigue, and headache. Note that the colon provides reabsorption of water and electrolytes as well as elimination of waste and regulation of bacterial homeostasis; motility is crucial for these roles.

The ENS is the intrinsic segment of the GI tract that controls essential functions such as motility, secretion, and blood flow; it is organized into neurons and glial cells that are distributed throughout the entire length of the gut wall (Burns and Pachnis, 2009). Enteric nervous system stem cells (ENSSCs) provide potential tools to replenish absent ganglia in the congenital form of megacolon, Hirschsprung's disease (Metzger et al, 2009). IBD is associated with a host of intestinal disease–related complications including toxic megacolon (Scherer, 2009). Subtotal colectomy with ileostomy remains the procedure of choice (Ausch et al, 2006; Gladman et al, 2005). Colectomy is complicated by considerable morbidity, while rates have improved over the past several decades (Teeuwen et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Hirschsprung's disease is caused by a single gene mutation of the *RET* proto-oncogene on band 10q11.2. Megacolon can occur in multiple endocrine neoplasia type 2A (MEN 2A) or 2B (MEN 2B).

Clinical/History
Height
Weight
BMI
Diet history
BP
I & O
Stool pattern
Stool consistency
Endoscopy
Barium x-rays
Abdominal girth

Lab Work
BUN, Creat
Gluc
H & H
Serum Fe, ferritin
Na^+, K^+, Cl^-
Ca^{++}, Mg^{++}
Thyroid function
Stool guaiac

INTERVENTION

OBJECTIVES

- Prevent complications such as lung atelectasis from distention, sepsis, ulceration with hemorrhage or perforation, or sigmoid volvulus.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Fluid Intake

Assessment Data: Height, Weight, BMI normal. Hx chronic constipation and Parkinson's disease. Recent identification of megacolon. Minimal intake of fluids without prompting. Signs of early dementia.

Nutrition Diagnoses (PES): Inadequate fluid intake related to cognitive changes and forgetting to drink as evidenced by I & O records with chronic constipation and megacolon.

Interventions: Increase fluid intake to meet estimated needs (30 mL/kg). Offer beverages every 2 hours. Educate staff and family about cueing to drink and offering beverages often. Review effects of laxatives, stool softeners, and other prescribed medications on constipation.

Monitoring and Evaluation: Improvement in signs of chronic constipation; relief from megacolon. Improved fluid intake to meet estimated needs.

- Empty the bowel as needed, with osmotic laxatives, enemas, suppositories, cathartics, or digital disimpaction.
- Normalize bowel function as much as possible. Evaluate bowel pattern by history and at present, including drug use and laxative abuse. Exclude underlying cause if possible, such as calcium channel antagonists, narcotics, anticholinergics.
- Identify and correct any nutrient deficiencies, electrolyte imbalances, or protein-energy malnutrition.
- Assure adequate hydration.

FOOD AND NUTRITION

- Use adequate fluid and fiber, pending status, and other conditions (such as heart failure). Prune juice added to hot cereal may help normalize bowel function. If raw fruits and vegetables are not tolerated at first, add over time. For some people, excessive bran will not be tolerated.
- Avoid excesses of refined foods and concentrated sweets to the exclusion of desirable foods.
- Fiber-rich TF may be needed for selected patients.

Common Drugs Used and Potential Side Effects

- Osmotic agents, such as magnesium salts, sorbitol, or lactulose are often used; some may increase flatulence.
- Stimulant laxatives (senna and bisacodyl) may decrease the ability of the colon to evacuate; they are not as helpful in this condition.
- Suppositories and stool softeners may be used or may have been used excessively. Monitor specific medications accordingly and their side effects.
- Anticholinergics, opiates, and antidepressants may increase or aggravate constipation. Limit use.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Many patients take natural herbal laxatives that contain cascara.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of exercise in maintaining normal bowel function.
- Discuss the role of fluid and fiber in bowel regularity (American Dietetic Association, 2008). For example, drinking a cup of hot prune juice may be effective for some patients.
- Include probiotic foods such as yogurt with live and active cultures, lactobacilli and bifidobacteria, and prebiotics (nondigestible oligosaccharides) for gut integrity.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Acquired Megacolon
  http://www.emedicine.com/med/byname/megacolon-chronic.htm
- NIH—Megacolon
  http://www.nlm.nih.gov/medlineplus/ency/article/000248.htm

MEGACOLON—CITED REFERENCES

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Ausch C, et al. Aetiology and surgical management of toxic megacolon. *Colorectal Dis.* 8:195, 2006.

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Burns AJ, Pachnis V. Development of the enteric nervous system: bringing together cells, signals and genes. *Neurogastroenterol Motil.* 21:100, 2009.

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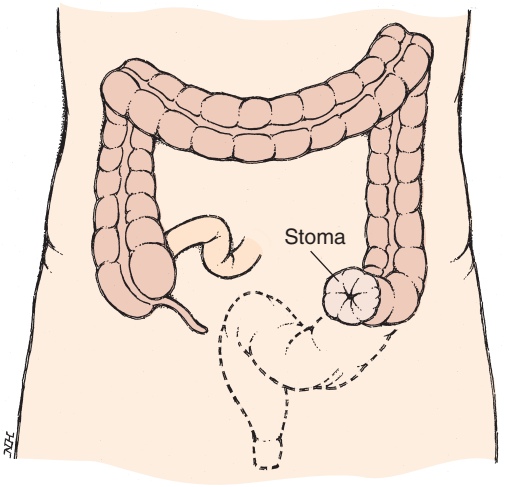
OSTOMY: COLOSTOMY

NUTRITIONAL ACUITY RANKING: LEVEL 2–3



Adapted from: Neil O. Hardy. Wesport, CT. *Stedman's Medical Dictionary,* 27th ed. Baltimore: Lippincott Williams & Wilkins, 2000, p. 383.

DEFINITIONS AND BACKGROUND

The colon functions primarily to absorb water and sodium and to excrete potassium and bicarbonate. A colostomy is an artificial outlet for intestinal wastes created surgically by bringing a portion of the colon through the abdominal wall, resulting in a stoma. Colostomy can be permanent or temporary. It may be indicated for intestinal cancer, diverticulitis, perforated bowel, radiation enteritis, obstruction, and Hirschsprung's disease. It may also be indicated for spinal cord–injured patients where bowel management (bowel care and defecation time) averages 6 hours/wk prior to stoma formation, but decreases to 1.5 hours/wk after a left colostomy, thus improving quality of life (Munck et al, 2009).

Abdominoperineal resection, with iliac colostomy, remains a standard treatment (Portier et al, 2005). With proper patient selection, laparoscopic colorectal surgery can be performed. Video-assisted, double barreled wet colostomy is minimally invasive. Table 7-21 lists the common types of colostomies.

Colostomy output is generally more formed than ileostomy output. Some colostomates can "irrigate," using a procedure similar to an enema to clean stool directly out of the colon through the stoma. This requires special irrigation appliances: an irrigation bag and a connecting tube (or catheter), a stoma cone, and an irrigation sleeve. A special lubricant is sometimes used on the stoma in preparation for irrigation. Following irrigation, some colostomates can use a stoma cap, a one- or two-piece system that simply covers and protects the stoma. This procedure is usually done to avoid the need to wear an appliance.

TABLE 7-21 Common Types of Colostomies

Colostomy Type	Description
Temporary Colostomy	Allows the lower portion of the colon to rest or heal. It may have one or two openings (if two, one will discharge only mucus).
Permanent Colostomy	Usually involves the loss of part of the colon, most commonly the rectum. The end of the remaining portion of the colon is brought out to the abdominal wall to form the stoma.
Sigmoid or Descending Colostomy	The most common type of ostomy surgery, in which the end of the descending or sigmoid colon is brought to the surface of the abdomen. It is usually located on the lower left side of the abdomen.
Transverse Colostomy	The surgical opening created in the transverse colon resulting in one or two openings. It is located in the upper abdomen, middle or right side.
Loop Colostomy	Usually created in the transverse colon. This is one stoma with two openings; one discharges stool, the second discharges mucus.
Ascending Colostomy	A relatively rare opening in the ascending portion of the colon. It is located on the right side of the abdomen.

Source: United Ostomy Association, Inc. What is ostomy? at http://www.uoa.org/ostomy_main.htm, accessed March 31, 2005.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Colostomy is a surgical procedure, occasionally used for a congenital condition.

Clinical/History
- Height
- Weight
- BMI
- Diet history
- I & O, hydration status
- Diarrhea or constipation
- Renal stones?

Lab Work
- H & H
- Serum Fe, ferritin
- Na^+, K^+
- Gluc
- Alb, transthyretin
- Ca^{++}, Mg^{++}
- Chol, Trig
- Transferrin, TIBC
- Serum B_{12}
- Serum folate
- PT or INR

INTERVENTION

OBJECTIVES

- To avoid major problems of blockage, increased flatulence, and problems with certain foods, preoperative teaching and postoperative follow-up must include food selection guidance.

SAMPLE NUTRITION CARE PROCESS STEPS

Poor Nutritional Quality of Life—Ostomy

Assessment: I & O records or food diary; weight fluctuations. Complaints of abdominal discomfort and unplanned ostomy output of large quantity.

Nutrition Diagnosis (PES): Poor nutritional quality of life related to fear and frequent ostomy output as evidenced by weight fluctuations and reports of GI discomfort after meals.

Intervention: Educate about how to meet estimated nutritional needs using tolerated foods; how to track GI symptoms and food tolerances using a food diary.

Monitoring and Evaluation: Improved I & O records; stabilized weight. Fewer or no GI distress following meals when foods poorly tolerated are omitted from the diet. Ostomy output more predictable and normal.

- Speed wound healing and recovery.
- Correct weight loss or malnutrition from GI blood loss, anemia, protein malabsorption, steatorrhea.
- Prevent watery or unscheduled bowel movements. Correct or prevent dehydration.
- Individualize the diet: Eat regularly, avoid odor-causing foods, and monitor food preferences. Normalize nutritional quality of life as much as possible.
- Avoid infection and skin irritants.

FOOD AND NUTRITION

- Early oral feeding in the patients undergoing colorectostomy is feasible, safe, and associated with reduced postoperative discomfort; it can accelerate the return of bowel function and improve rehabilitation (Zhou et al, 2006). Most people can resume oral diet 2 days after surgery.
- Progress from a liquid to a low-residue diet. To speed healing, the formula or diet should also be high in protein, energy, vitamins, and minerals.
- Diet should provide normal or increased salt intake. One to two quarts of fluid, taken between meals, should be ingested daily.
- Gradually introduce new foods; if done slowly, offending foods can be identified and obstruction can be controlled or prevented. Foods that may not be tolerated include:
  1. **Foods that may cause loose stools or diarrhea:** apple juice, prune juice, dried beans, chocolate, green beans, raw fruits and raw vegetables, fried foods, highly spiced foods, broccoli, and leafy green vegetables.
  2. **Gas or odor-causing foods:** alcohol (beer), beans, onions, cabbage, broccoli, cauliflower, Brussels sprouts, fish, eggs, asparagus, and garlic. Fresh parsley is a natural deodorizer.
  3. Foods that cause **urinary odor** include asparagus and seafood.
  4. Beets and red gelatin can cause **abnormal stool coloration**.
  5. Avoid **high fiber foods:** granola, bean sprouts, bamboo shoots, bran, whole-kernel corn, mushrooms, celery, nuts, pineapple, popcorn, coleslaw, apple skins, seeds, or coconut.
  6. Foods **most often avoided** because of an ostomy include fresh fruits and vegetables such as cabbage, beans, onions, hot dogs, sausages and other meats with casings.
- Foods that thicken stool include applesauce, bananas, marshmallows, rice, pasta, peanut butter, tapioca, and yogurt.
- Progress to a high-fiber diet with short-fibered foods.
- If calcium oxalate stones develop after the colostomy, diet should provide a high-fluid intake. Restrict intake of oxalates from spinach, rhubarb, wild greens, coffee and tea, and chocolate.
- Signs of blockage include: almost constant spurt of highly watery stool, bloating, cramping, swelling around stoma, strong odor of stool, nausea, vomiting, and pain. Avoid eating solid food, and do not take any laxatives or stool softeners. Drink a hot, caffeinated beverage; try a hot bath; gently massage the abdomen; and apply a pouch that has a larger opening.

Common Drugs Used and Potential Side Effects

- Probiotics and prebiotics may be useful for return of gut immunity.
- Polyethylene glycol solution may be used as an alternative fluid regimen for colostomy irrigation.
- Patients with electrolyte concerns should be carefully monitored if sodium phosphate preparations (Osmoprep, Visicol) are used for colonscopy preparation (Lichtenstein, 2009).
- Prednisone: Restrict excessive sodium intake. Monitor nitrogen, calcium, and potassium losses when used over a long period of time.
- Lomotil is a stool thickener and deodorizer; use plenty of fluids.
- Bulk-forming agents such psyllium (Metamucil) may be useful. Increased peristalsis occurs.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Approximately 6 weeks are required to acclimate the bowel to new procedures of irrigation. Enemas are used to wash the bowel up to the ileocecal valve (with 1000 mL of tap water). Constipation can occur with dehydration; therefore, adequate intake of fluid and fiber is important.
- Use of a commercial deodorant in the colostomy bag is preferred for eliminating highly flavored or nutrient-dense foods.

- Instruct the patient to eat slowly, chew foods well, and avoid swallowing air.
- Irrigations should not be performed when there is vomiting or diarrhea. Working with an enterostomal therapist can be helpful for more suggestions.
- Regular mealtimes should be encouraged.
- Reassurance is needed, without misleading the patient. Some colostomies are permanent (Riansuwan et al, 2009). Quality of life may decrease after the procedure (Yau et al, 2009).
- If a temporary colostomy is performed, a patient may be evaluated for a reversal of the colostomy where the remaining colon and rectum must be examined for normalcy.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing diarrhea and related discomfort.
- Proper ostomy care requires careful and constant hand washing.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Colostomy—Medicine Net
  http://www.medicinenet.com/colostomy_a_patients_perspective/article.htm
- Colostomy Guide
  http://www.cancer.org/docroot/CRI/content/CRI_2_6x_Colostomy.asp
- Medline Plus
  http://www.nlm.nih.gov/MEDLINEPLUS/ency/article/002942.htm
- United Ostomy Association (UOA)
  http://www.uoa.org/ostomy_main.htm

OSTOMY: COLOSTOMY—CITED REFERENCES

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Portier G, et al. Use of malone antegrade continence enema in patients with perineal colostomy after rectal resection. *Dis Colon Rectum.* 48:499, 2005.

Riansuwan W, et al. Nonreversal of Hartmann's procedure for diverticulitis: derivation of a scoring system to predict nonreversal. *Dis Colon Rectum.* 52:1400, 2009.

Yau T, et al. Longitudinal assessment of quality of life in rectal cancer patients with or without stomas following primary resection. *Dis Colon Rectum.* 52:669, 2009.

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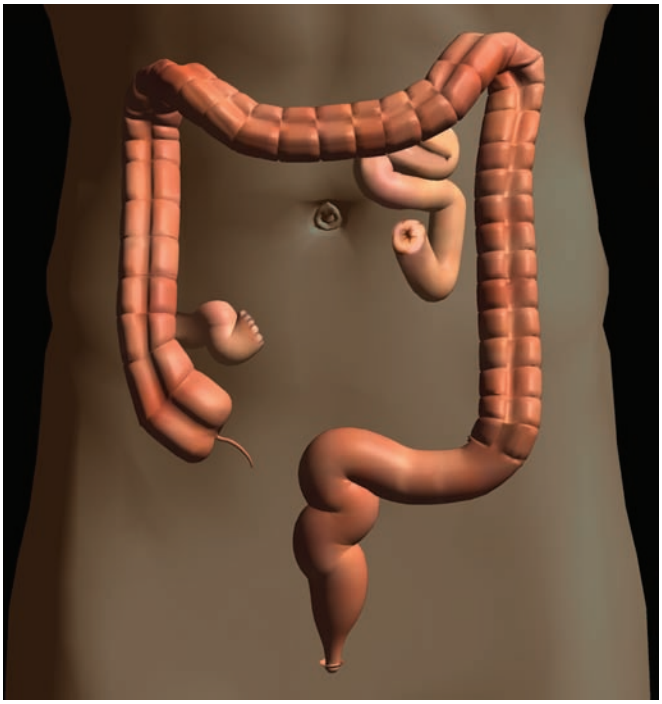
OSTOMY: ILEOSTOMY

NUTRITIONAL ACUITY RANKING: LEVEL 3



DEFINITIONS AND BACKGROUND

Used to treat intractable cases of ulcerative disease, Crohn's disease, polyposis, and colon cancer, an ileostomy is a surgical procedure (stoma/opening formation) that brings the ileum through the abdominal wall. It may be temporary or permanent. This procedure causes a decrease in fat, bile acid, and vitamin B_{12} absorption, as well as greater losses of sodium and potassium.

Patients will be incontinent of gas and stool. Ideally, the ileocecal valve can be kept to decrease bacterial influx into the small intestine. Of these patients, 50–70% will have recurrent disease. Effective ostomy management is important and involves establishment of an effective system for managing altered dietary and fluid intake, maintaining fluid and electrolyte balance, and preventing food blockage (Doughty, 2005).

Subtotal colectomy with ileostomy is a safe and effective treatment for patients requiring urgent surgery (Hyman et al, 2005). Interval ileal pouch–anal anastomosis reconstruction without a stoma after colectomy equals the more traditional protocol in terms of clinical outcome but yields lower hospital costs and probably a shorter length of hospital stay (Swenson et al, 2005). Table 7-22 describes the procedures for ileostomy.

TABLE 7-22 Ileostomy Procedures

Procedure	Description
Ileoanal Anastomosis	This is now the most common alternative to the conventional ileostomy. Technically, it is not an ostomy since there is no stoma. In this procedure, the colon and most of the rectum are surgically removed, and an internal pouch is formed out of the terminal portion of the ileum. An opening at the bottom of this pouch is attached to the anus such that the existing anal sphincter muscles can be used for continence. This procedure should only be performed on patients with ulcerative colitis or familial polyposis and who have not previously lost their rectum or anus. It is also called J-pouch, pull-thru, endorectal pullthrough, pelvic pouch, or a combination of these terms.
Continent Ileostomy	This surgical variation of the ileostomy is also called a **Kock pouch**. A reservoir pouch is created inside the abdomen with a portion of the terminal ileum. A valve is constructed in the pouch, and a stoma is brought through the abdominal wall. A catheter or tube is inserted into the pouch several times a day to drain feces from the reservoir. This procedure has generally been replaced in popularity by the ileoanal pouch. A modified version of this procedure called the Barnett continent ileal reservoir is performed at a limited number of facilities.

Source: United Ostomy Association, Inc. What is ostomy? at http://www.uoa.org/ostomy_main.htm, accessed March 31, 2005.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Ileostomy is a surgical procedure, occasionally used for a congenital condition.

Clinical/History	Lab Work	
Height	H & H	Transferrin
Weight	Serum Fe, ferritin	TIBC
BMI	BUN, Creat	Gluc
Diet history	Na^+, K^+	WBC
Stool (occult blood)	Alb, transthyretin	Ca^{++}, Mg^{++}
BP		Serum B_{12}
		Schilling test if needed
		Serum folate

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Fluid Intake

Assessment Data: I & O, weight changes, signs of dehydration, high output from ileostomy.

Nutrition Diagnoses (PES): Inadequate fluid intake related to losses from ileostomy and insufficient intake as evidenced by I & O records (intake 1200 mL, output 1600 mL/d).

Interventions: Food and nutrient delivery: Calculate needs, provide extra liquids with meals, offer a beverage every few hours. Educate: Discuss the need for extra fluids when ileostomal losses are increased, during febrile periods, when there is vomiting.

Monitoring and Evaluation: I & O records, weight records, fewer signs of dehydration (poor skin turgor, sunken eyeballs, etc).

INTERVENTION

OBJECTIVES

- Modify the diet to counteract malabsorption of nutrients secondary to diarrhea, protein, and fluid losses, negative nitrogen balance from nutrient loss, and anorexia.
- Correct any anemia caused by inadequate intake or blood losses.
- Counteract weakness and muscle cramping from potassium losses.
- Provide increased energy intake during periods of fever or infection.
- Replenish calcium to reverse losses caused by steatorrhea and bone density loss if steroid therapy is used.
- Prevent gallstones, renal oxalate stones, bacterial overgrowth, and fatty acid malabsorption.

FOOD AND NUTRITION

- **Preoperatively:** Fiber and lactose intolerances are common; alter diet accordingly. With strictures, avoid popcorn, nuts, seeds, mushrooms, celery, fruit skins, and vegetable skins. Have the patient chew thoroughly.
- **Postoperatively:** Provide a high-energy, high-protein diet for wound healing that is low in excess insoluble fiber. Avoid the high-fiber foods suggested for preoperative care for about 4 weeks. Pectin in apples and oligosaccharides in oatmeal may be beneficial to add back first. Spinach or parsley are natural intestinal deodorizers, but beware of excesses of oxalate-rich foods.
- The patient needs an adequate intake of protein (provided by low-fat sources such as lean meats and egg white), vitamin B_{12} (provided by liver, fish, and eggs), folacin, calcium, magnesium, iron, sodium, vitamin C, and potassium.
- Diet should provide an adequate amount of fluids, especially in hot weather. Add salt if needed.
- Apple juice has chemoprotective properties for human colon cells. Intake of apple juice results in bioavailable polyphenols in the gut lumen, which could contribute

to reduced genotoxicity, enhanced antigenotoxicity and favorable modulation of GSTT2 gene expression (Veeriah et al, 2008).
- Since obesity can cause more discomfort, a weight management plan may be useful.

Common Drugs Used and Potential Side Effects

- Corticosteroids: With prednisone, restrict excessive sodium intake and monitor nitrogen and calcium losses. The corticosteroid budesonide (Entocort) may be administered to increase the absorptive capacity of the intestinal mucosa in patients with ileostomies (Ecker et al, 2005). Side effects may include increased appetite and weight gain, hypokalemia, and elevated CRP.
- Lomotil is a stool thickener and deodorizer. Plenty of fluids should be used.
- Probiotics and prebiotics may be useful to help with recovery of gut immunity. Research is underway for the use of a special form of *Bacteroides ovatus* (Hamady et al, 2008).
- Psyllium (Metamucil) is used as a bulk-forming agent. Increased peristalsis will occur.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Use of fish oil may be beneficial during periods of inflammation.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain which foods are common sources of the needed nutrients in a diet or suggest supplementation with multivitamins and minerals. Encourage use of apple juice and other protective foods as tolerated.
- Monitor individual tolerance to offending foods such as gas-forming or fried foods, highly seasoned foods, nuts, raisins, and pineapple. Foods that may cause rapid intestinal transit or discomfort may include dried fruits, prune juice, fresh strawberries or peaches, coconut, nuts, seeds, cabbage, celery, bamboo shoots, corn, and milk.
- An enterostomal therapist may be of assistance.
- Discuss replacement of fluid and sodium, especially during hot weather.
- Eating before bedtime should be avoided to lessen discomfort.
- More than half of patients operated with proctocolectomy will need surgical intervention within 20 years; the failure rate is more than 10% and complications must be addressed (Wasmuth et al, 2009).

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual who may be experiencing diarrhea and related discomfort.
- Proper ostomy care requires careful and constant hand washing.
- If home TF or PN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Continent Ileostomy
  http://www.medicinenet.com/caring_for_a_continent_ileostomy/article.htm
- Ileostomy
  http://digestive.niddk.nih.gov/ddiseases/pubs/ileostomy/
- Ilesotomy Guide—American Cancer Society
  http://www.cancer.org/docroot/CRI/content/CRI_2_6x_Ileostomy.asp
- Jackson Gastroenterology
  http://www.gicare.com/pated/ecdgs11.htm
- Mayo Clinic—Ostomy
  http://www.mayoclinic.com/health/ostomy/SA00072
- United Ostomy Association
  http://www.uoa.org/ostomy_facts_ileostomy.htm

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Hyman NH, et al. Urgent subtotal colectomy for severe inflammatory bowel disease. *Dis Colon Rectum.* 48:70, 2005.
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PERITONITIS

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

In peritonitis, inflammation of the peritoneal cavity due to infiltration of intestinal contents occurs. Contents from such conditions as ruptured appendix, gastric or intestinal perforation such as in diverticulitis, trauma, fistula, anastomotic leaks, or failed peritoneal dialysis (PD) may initiate the problem.

Bacterial peritonitis is a major cause of morbidity in pediatric PD patients and can lead to catheter removal, hospitalizations, peritoneal membrane dysfunction, and sepsis (Chand et al, 2009). Spontaneous bacterial peritonitis is also common in patients with cirrhosis and ascites because of bacterial translocation. The diagnostic evaluation of ascites involves an assessment of its cause by determining the serum-ascites albumin gradient and the exclusion spontaneous bacterial peritonitis (Hou and Sanyal, 2009). Peritonitis in liver transplant patients may suggest organ rejection (Macedo et al, 2005). Prophylactic antibiotics and omentectomy at catheter insertion are useful (Chand, 2009). Aggressive nutritional supplementation pre- and postoperatively is suggested (De Frietas et al, 2008). It is important to avoid refeeding syndrome.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Peritonitis is the result of infection and not genetic origins.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Abdominal pain, tenderness
- Abdominal rigidity (washboard appearance)
- Fever?
- BP
- I & O
- Ileus
- Sepsis?
- X-rays
- Paracentesis (>250 polymorphonucleate cells)
- Laparoscopy

Lab Work

- H & H
- Serum Fe, ferritin
- WBC
- BUN, Creat
- Gluc
- Alb, transthyretin
- CRP
- Na^+, K^+
- Ca^{++}, Mg^{++}
- TLC

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Energy Intake

Assessment Data: I & O records, weight, severe abdominal pain, intake <25% in past 24 hours, fever 102°F.

Nutrition Diagnoses (PES): Inadequate energy intake related to inability to eat orally and abdominal pain as evidenced by intake <25% in past day and fever demands about 21% higher energy needs than normal.

Interventions: IV fluids with dextrose until able to eat orally. Monitor for readiness to eat again, based on drop in fever, return of bowel sounds, tolerance for soft or bland foods.

Monitoring and Evaluation: Gradual return to oral diet with improved I & O records; weight returning to usual. No fever or signs of abdominal pain or peritonitis No signs of refeeding.

INTERVENTION

OBJECTIVES

- Correct fluid/electrolyte imbalances when present. Vigorous IV rehydration is needed.
- Provide bowel rest and recovery. PN may be better than NG feeding but further studies into dualenteral nutrition and PN are needed (De Frietas et al, 2008).
- Improve nutritional status, especially if patient has been malnourished over a period of time or if there is anorexia or ileus.
- Avoid high risk for refeeding syndrome (De Frietas et al, 2008).

FOOD AND NUTRITION

- Patient generally is NPO with IV feedings for at least 24 hours. Progress as tolerated to a soft or general diet appropriate for the condition that caused the peritonitis originally.
- Increase protein intake to correct catabolic state. Increase calories because basal energy expenditure is generally elevated by 10–17%.

Common Drugs Used and Potential Side Effects

- Ciprofloxacin is effective in the treatment. Other antibiotics may be used for bacterial peritonitis. Monitor for specific side effects.
- Probiotics may be useful; research continues in this area.
- PPIs suppress gastric acid secretion, allowing bacterial colonization of the upper GI tract, this may predispose to bacterial overgrowth, translocation and peritonitis (Bajaj et al, 2009). Research is needed in this area.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- With patients on CAPD, diet may need to be altered similar to the diet typical for renal patients.
- Discuss diet appropriate for the illness of origin (such as diabetes, hypertension, toxemia, or renal disease).

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Mayo Clinic
  http://www.mayoclinic.com/health/peritonitis/DS00990/DSECTION%3Dcauses
- NIH—Peritonitis
  http://www.nlm.nih.gov/medlineplus/ency/article/001335.htm
- Peritonitis
  http://www.nlm.nih.gov/medlineplus/ency/article/001335.htm

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SHORT BOWEL SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

SBS is the predominant cause of intestinal failure, with a high degree of morbidity and mortality. Intestinal failure involves reduction of gut mass below the minimal amount needed for nutrient absorption. SBS involves surgical resection of a portion of the small bowel, compromising the absorptive surface and resulting in malabsorption (especially if more than 50% of the small intestine has been removed). Malnutrition from maldigestion, malabsorption diarrhea, and steatorrhea may result.

A bowel resection that results in SBS may be due to Crohn's disease, intestinal cancer, scleroderma, or fistula in adults; or it may be due to necrotizing enterocolitis, intestinal atresia, or mesentery artery occlusion in infants. If only 30% of the small intestine remains in an adult (or <30 cm in infants), the resulting malabsorption may be life threatening. Problems are significant when more than 70% of the bowel is resected, unless the terminal ileum and ileocecal valve remain. Every attempt should be made to keep the ileocecal valve to prevent contamination of the small intestine. See Table 7-23 regarding the implications of bowel resections.

Normal bowel length is about 600 cm. SBS generally leaves less than 150 cm of small intestine. A total of 100 cm is necessary to completely absorb bile salts; 50–70 cm of jejunum–ileum maintains minimal intestinal autonomy. The minimal length of functional bowel needed for enteral feeding is over 100 cm in the absence of an intact colon and over 60 cm in continuity with the colon.

Patients with SBS require long-term PN. After massive intestinal resection, the intestine undergoes adaptation, and nutritional autonomy may be obtained if the adaptive process is supported by both nutritive and nonnutritive factors (Weale et al, 2005). Length of small intestine remaining after resection is the best predictor of the final success in terminating PN. Home PN may cost between $400+ a day; most insurance companies cover up to $1 million, about 10 years of reimbursement.

Aggressive nutritional intervention is necessary for resolution of nutritional deficits and recovery of health. Early oral feeding after colorectal surgery is safe. Bowel adaptation takes about 1 year. Assessment must include oral intake, stool and urine output, serum electrolytes and visceral proteins, and body weight (DiBaise et al, 2006). Diarrhea and high stomal output are common problems and can lead to dehydration; hypokalemia; deficiencies of calcium, magnesium, and zinc; carbohydrate and lactose malabsorption; protein malabsorption; renal oxalate stone formation; cholesterol biliary stones; gastric acid hypersecretion; vitamin B_{12} or iron deficiency; fat-soluble vitamin deficiency; and diarrhea. See Table 7-24 for malabsorption concerns in SBS.

Patient education and motivation are key factors in successful weaning from PN, where possible (DiBaise et al, 2006).

TABLE 7-23 Implications of Bowel Resections

Loss of jejunum. Ileum undergoes hyperplasia; length and absorption per centimeter increases.

Loss of ileum. This is more serious than loss of jejunum because vitamin B_{12} and bile salts will be reabsorbed poorly as a result. The ileocecal valve keeps colonic bacteria out of the small intestine and regulates chyme flow. Some colonic adaptation occurs during the next 2–5 years.

Loss of colon. Overall, with removal of the colon versus the small intestine, fewer malabsorption problems occur. Loss of electrolyte and water-absorbing capacity occurs, as well as loss of salvage absorption of CHO and other nutrients. Most oxalate absorption occurs here.

In short bowel syndrome (SBS), short-chain triglycerides have a precursor in pectin; oligosaccharides increase O_2 uptake in the colon, thereby maintaining gut integrity. Early refeeding (i.e., free fatty acids, sugars, proteins) stimulates mucosal growth. Hyperphagia can increase enterocyte production. Adaptation requires adequate nutrition, intraluminal nutrients, and bile and pancreatic secretions. Enhancers of the adaptive process seem to include gastrin, glutamine, growth hormone, insulin, short-chain fatty acids, fats, some dietary fibers, cholecystokinin (CCK), glucagon, insulin-like growth factor I, neurotensin, and glucose.

TABLE 7-24 Malabsorption Concerns in Short Bowel Syndrome

Bacterial overgrowth, if ileocecal valve is absent
Decreased bile acid concentration (with loss of ileum and bacterial overgrowth)
Decreased surface area and lessened fluid reabsorption
Dumping syndrome from rapid transit and reduced bowel length
Gastric acid oversecretion with resulting damage to duodenal cells and altered pH, thus affecting pancreatic enzyme and bile activity
Maladaptive remaining small bowel, especially from original bowel disease, lactase deficiency
Pancreatic enzyme activity loss from duodenum, with malnutrition

The use of trophic substances may increase the absorptive function of the remaining gut. Glucagon-like peptide 2 (GLP-2) has been shown to improve intestinal absorption in SBS patients; it reduces fecal weight and enables SBS patients to maintain their intestinal fluid and electrolyte absorption at lower oral intakes (Jeppesen et al, 2009). Current guidelines, however, suggest that the use of growth hormone, glutamine and GLP-2 should not be recommended in patients with short bowel syndrome (Van Gossum et al, 2009).

Serial transverse enteroplasty (STEP) procedure is a safe way to lengthen the small bowel in patients with SBS (Ehrlich et al, 2007). Surgical bowel lengthening should be considered in any chronically PN-dependent patient when there is substantial bowel dilation, regardless of remnant bowel length (Thompson and Sudan, 2008). ITx may also restore quality of life by recovery of normal motility and absorptive capacity. While high-dose immunosuppression is a potential problem, advances in transplantation hold promise as an alternative to intestinal failure and chronic dependence on CPN.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: SBS is a surgical procedure.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Weight changes
- I & O
- Steatorrhea
- Stool output
- Dehydration?
- Urinary output
- DEXA bone density scan

Lab Work

- H & H
- Serum Fe, ferritin
- Transferrin
- CRP, ESR
- D-xylose absorption
- Serum gastrin (increased)
- Ca^{++}, Mg^{++}
- 25-hydroxyvitamin D level
- Alk phos
- Na^{+} (serum, urine, stool)
- K^{+} (serum, urine, stool)
- Fecal nitrogen
- GTT
- Gluc
- RBP
- N balance
- Serum oxalate
- Alb, transthyretin
- Barium follow-through (BaFT) examination
- Schilling test
- Serum B_{12}
- Serum amylase, lipase
- Serum copper
- Bile acid breath test
- Lactose tolerance test
- Hydrogen breath test
- Fecal fat test

INTERVENTION

OBJECTIVES

- Determine the location and amount of the intestine that was resected to predict likelihood of diarrhea, malabsorption, and malnutrition (see Table 7-24). Provide nutrient replacements, dependent on area of resection (proximal jejunum—calcium, iron, magnesium, protein, CHO, and fat; terminal ileum—bile acids and intrinsic factor-bound vitamin B_{12}).
- Manage post surgical phases. The first phase lasts 1–3 months, massive diarrhea and limited absorption; the second phase: 4–12 months where weight gain begins and absorption improves. In the last phase between 13 and 24 months, maximal adaptation occurs, with possible discontinuation of PN when fluid is >7 L daily and when energy intake is sufficient for desired weight goal.
- Support hyperphagia, in which the amount of protein absorbed increases relative to the amount of remnant small bowel length. Try weaning off PN without trophic factors. If this is unsuccessful, a trophic factors should be attempted (Steiger et al, 2006).
- Prevent and correct fluid and electrolyte imbalances and dehydration. Oral intake could aggravate already massive losses of fluid (3–10 L/d is common). Immediately postoperatively, NPO with IV fluid replacement is likely. Eventually switch to oral intake as tolerated.
- Use remaining bowel surface and maximize efficacy. Prevent atrophy of small bowel mucosa, catheter sepsis, metabolic bone disease, and liver disease with long-term use of CPN. Carefully monitor for signs and symptoms of these problems and manage accordingly.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Parenteral Infusion

Assessment Data: Calculations of energy needs showing PN infusion at a rate exceeding goal by 25%; hypophosphatemia, hypokalemia and signs of refeeding syndrome.

Nutrition Diagnoses (PES): Excessive parenteral infusion (NI 2.4) related to order for CPN solution that exceeds estimated needs by 25% (200 kcal/d) as evidenced by signs of refeeding syndrome with hypophosphatemia and hypokalemia.

Interventions: Reduction in CPN infusion to meet rather than exceed requirements Intervention code.

Monitoring and Evaluation: Consistent monitoring of labs until resolution of refeeding syndrome; tolerance of CPN solution with no further complications.

- Correct symptoms of deficiency and malabsorption, when possible, for vitamins B_{12}, A, D, E, and K and for the minerals zinc, potassium, and magnesium.
- Minimize weight loss (approximately 10 lb monthly until adaptation occurs). Maximize energy intake from fats and carbohydrates without worsening the diarrhea.
- Omit lactose if not tolerated; provide adequate calcium replacements.
- Decrease oxalate from the diet to reduce renal stone formation. Excess bile in the colon from decreased ileal absorption enhances absorption of free oxalate (normally only 10–15% is absorbed).
- Control or prevent gallstone formation (increased risk of two to three times normal), anemia, PLE, peptic ulcer from increased gastric acid secretion, and liver disease (often from home CPN). Parenteral fish-oil lipid or olive-oil emulsions are now more available.
- Allow remaining intestine to compensate over time by hypertrophy of villi and increased diameter. Home CPN is expensive but can be lifesaving for months or years. Transitional feedings may be needed over several months from PN to oral diet. For care of colostomy or ileostomy, see appropriate entries.

FOOD AND NUTRITION

- **First Postoperative Phase:** IV nutrition or CPN may be appropriate immediately before and approximately 5 days after surgery to allow rest. Determine whether the patient has problems with bloating. The first phase involves extensive diarrhea greater than 2 L daily; advance CPN slowly to avoid refeeding syndrome. At the end of this time, if diarrhea continues to be greater than 2 L, CPN may be lifelong.
- **Second Postoperative Phase:** Diarrhea is lessened and intestinal adaptation begins; CPN may be slowly reduced and polymeric TF started at a slow, continuous rate according to stomal output or stool output. Provide 40–60 kcal/kg and 1.2–1.5 g protein/kg. Providing patients with enteral nutrition, glutamine, dietary fiber, and r-hGH during bowel rehabilitation therapy allows weaning from CPN. If weight loss is greater than 1 kg/wk, CPN may need to be restarted.
- Nocturnal enteral rehydration is an intervention using ORS through PEG tubes at night; this allows for earlier discontinuation of CPN and improved fluid absorption.
- **Third Postoperative Phase:** Complete bowel adaptation begins as TF is tolerated and oral diet is slowly resumed (from 2 months to 1 year). Six small feedings that are high CHO and low fat may be tolerated (60% CHO, 20% protein, 20% fat, with a limit on MCT of 40 g/d). With no colon, the jejunostomy feeding may need to be 40–50% CHO, 20% protein, 30–40% fat. PN reductions can be made by either decreasing the days of PN infusion per week or decreasing the PN infusion volume equally across all days of the week.
- Supplemental zinc, potassium, liquid magnesium, oral calcium (600–1000 mg), manganese, iron, vitamin C, selenium, B-complex vitamins (especially folic acid), and other nutrients may be needed. Determine needs based on site of resection and signs of malnutrition.
- Monitor for needs for vitamins A, E, and D; use water-miscible forms. Patients with SBS are depleted in diet-derived carotenoids despite oral and IV multivitamin supplementation; reduction of PN lipid infusion may improve serum alpha-tocopherol concentrations (Luo et al, 2009). With antibiotic use, the patient will need extra vitamin K.
- Lactose-restricted and oxalate-restricted diets may be needed for an extended period of time. Rhubarb, spinach, beets, cocoa and chocolate, sweet potatoes, strawberries, celery, and peanuts are high-oxalate foods. Nuts and nut butters, berries, Concord grapes, sweet potatoes and potatoes, and most vegetables have smaller amounts.
- Omit alcoholic beverages and caffeine unless physician permits small quantities.
- Taking fluids between instead of with meals may be helpful to reduce dumping. Restrict at first to 1500 mL; progress as tolerated.
- With osmotic diarrhea, a reduction in simple carbohydrates and an increase in complex carbohydrates may be needed. Sorbitol, mannitol, and xylitol are usually poorly absorbed.
- Restricted foods such as lactose may be attempted and added back to the diet if they are tolerated. Bowel adaptation occurs over time and may eventually lead the patient back to an unrestricted diet.

Common Drugs Used and Potential Side Effects (see Table 7-25)

- **Note: Most oral medications are poorly tolerated in this condition!** Accelerated intestinal luminal transit time causes a reduction in absorption of certain antimicrobial agents, digoxin, hydrochlorothiazide, cyclosporine, cimetidine, mesalazine (5-aminosalicylic acid), oral contraceptives, and levothyroxine.
- Dietary glutamine (GLN) or oral antibiotics (ABX) can blunt gut barrier dysfunction (Tian et al, 2009). Antibacterial drugs, antimotility drugs, antidiarrheal agents, H_2 antagonists and PPIs, pancreatic enzymes, somatostatin analogs, antimicrobials, and trophic factors have been quite helpful in addition to nutritional therapy (Matarese and Steiger, 2006).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Probiotics and foods such as acidophilus or yogurt are useful aids in bowel adaptation phases. Not all probiotic bacteria have similar therapeutic effects. Research is available for probiotic supplementation for children with SBS (Wallace, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Importance of nutrition and supplementation must be discussed to prevent or correct malnutrition and

TABLE 7-25 Medications Used in Short Bowel Syndrome

Medication	Description
Antibiotics	Tetracycline, Flagyl, Septra, or Cipro may be needed for bacterial overgrowth. Monitor hydrogen breath tests (especially with blind loop).
Antidiarrheals	Antidiarrheals such as Lomotil, Imodium, and codeine are useful. Liquid preparations often are better tolerated. If dehydration occurs, use oral rehydration therapy but not sports drinks, which do not have the adequate electrolyte replacements.
Bile salt replacements	Cholylsarcosine (CS) is a semisynthetic bile salt that may be useful in bile salt replacement therapy of short bowel syndrome (Furst et al, 2005).
Calcium supplements	Oral calcium supplements (OsCal or Tums four times daily) often are used to bind oxalate excesses and to decrease diarrhea. Do not take with a bulk-forming laxative or with an iron supplement. Increase water intake.
Cholestyramine	Cholestyramine may be used for choleraic diarrhea when less than 100 cm is resected and when the colon is in continuity; prevent excessive use. Take before meals. Nausea, vomiting, or constipation may occur.
Cimetidine or omeprazole	Cimetidine and omeprazole may be needed to decrease gastric hypersecretion; parenteral administration may be needed. Serum gastrin levels should be monitored. A dose two to three times higher than normal may be needed because of gastric hypersecretion; lack of sufficient time with intestinal mucosa leads to insufficient absorption. Vitamin B_{12} absorption decreases with use of these medications.
Clonidine	Clonidine can effectively reduce intestinal fluid and electrolyte losses and should be considered in patients with short bowel syndrome.
Growth hormone	Growth hormone increases water/sodium transport. It is useful in combination with glutamine and a modified diet, but results have been mixed.
Minerals	Liquid potassium and intravenous or intramuscular magnesium may be needed.
Pancreatic enzymes	Pancrelipase may improve fat and protein absorption after jejunal resection; results are variable.
Peptides	Glucagon-like peptide-2 (GLP-2) is an enteroendocrine peptide that is released in response to luminal nutrients; it seems to help support the adaptive response to resection (Martin et al, 2005).
Probiotics and prebiotics (synbiotics)	This type of therapy might be a potent modulator of intestinal flora and a promising strategy to treat short bowel patients with enterocolitis.
Vitamins	Vitamins that are chewable or in liquid form may be better tolerated. Parenteral vitamin B_{12} may be necessary. Add extra fat-soluble vitamins A, E, D, and K if deficiency occurs. Do not use vitamin C in excessively large quantities; some links with oxalate stones have been noted.

malabsorption. After adaptation, significant energy intakes will be needed to maintain desired weight.

- Provide recipes or food-preparation tips to support the specific dietary regimen and to evaluate tolerances over time.
- Progression in diet is allowed when the small intestine adapts over several months. Use yogurt with live and active cultures.
- Discuss the need for free water.
- The use of home CPN is beneficial in many cases. Judicious use of home CPN in this setting requires careful clinical assessment on a patient-by-patient basis (Hoda et al, 2005). A supportive attitude from family and caregivers is essential.
- There are potential benefits of a multidisciplinary intestinal rehabilitation program. Interventions can help decrease incidence and economic impact of intestinal failure.
- Exercise, such as resistance training, can help regenerate lean body mass.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- E-medicine
  http://www.emedicine.com/ped/topic2088.htm
- Short Bowel Syndrome
  http://digestive.niddk.nih.gov/ddiseases/pubs/shortbowel/
- Short Bowel Syndrome—Pediatrics
  http://depts.washington.edu/growing/Assess/SBS.htm

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Weale AR, et al. Intestinal adaptation after massive intestinal resection. *Postgrad Med J.* 81:178, 2005.

TROPICAL SPRUE

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

Tropical sprue is an acquired disorder that presents with chronic diarrhea, anorexia, weight loss, megaloblastic anemia (folic acid deficiency), light-colored stools, diarrhea, weight loss, pallor, sore tongue (vitamin B_{12} deficiency), and easy bruising (vitamin K deficiency). Etiology is often bacterial, viral, or parasitic infection or toxins in spoiled food. *Giardia intestinalis* is a common culprit. Tropical sprue may occur after traveling to the Caribbean, southern India, and Southeast Asia.

Tropical sprue causes progressive villus atrophy in the small intestine, similar to CD ("nontropical" sprue). Villi of the small intestinal mucosa become blunted, or obliterated. Treatment of tropical sprue includes folic acid and vitamin B_{12} replacement. However, even prolonged treatment fails to resolve malabsorption. While tropical sprue is less common than it was decades ago (Nath, 2005), sporadic tropical sprue is still an important cause of malabsorption in adults and in children in South Asia (Ramakrishna, 2007).

Biomarker (13)C-sucrose breath test (SBT) to measure enterocyte sucrase activity can be used as a marker of small intestinal villus integrity and function (Ritchie et al, 2009). Enhanced magnification endoscopy can help identify patchy areas of partial mucosal atrophy, potentially reducing the need for blind biopsies (Lo et al, 2007).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: An infectious cause is suspected in tropical sprue.

Clinical/History

Height
Weight
BMI
Diet history
Weight loss?
I & O; dehydration?
Malabsorption, especially for xylose
Glossitis
Cramps, nausea
Gas, indigestion
Paleness
Biopsy
Enhanced magnification endoscopy (EME)

Lab Work

Gluc
SBT
Na^+, K^+
H & H, Serum Fe, ferritin (decreased)
Alb, transthyretin (decreased)
Serum B_{12} (decreased)
Serum folate (decreased)
Ca^{++} (decreased)
Mg^{++}
PT or INR (altered)

INTERVENTION

OBJECTIVES

- Provide fluids and electrolytes.
- Differentiate between tropical sprue and CD. Control diarrhea, which leads to malabsorption and malnutrition over time.
- Improve or correct folic acid, vitamin B_{12}, and vitamin K deficiencies. Treat tropical sprue with folate and vitamin B_{12} cures the macrocytic anemia and the accompanying glossitis.
- Avoid fatty foods that can cause oily, foul-smelling stools.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function

Assessment Data: Diarrhea, fever, macrocytic anemia, biopsy showing flattened villi and diagnosis of tropical sprue.

Nutrition Diagnoses (PES): Abnormal GI function related to infectious process as evidenced by diarrhea, macrocytic anemia, and flattened villi on biopsy.

Interventions: Provide folic acid and vitamin B_{12} in supplemental form along with antibiotic therapy; extra fluids, sodium and potassium as needed. Progress to low-fat diet.

Monitoring and Evaluation: Improvement in macrocytic anemia; resolution of diarrhea.

FOOD AND NUTRITION

- Use a regular diet with supplements of vitamin B_{12} and folic acid. Good sources of folacin include liver, kidney, yeast, leafy greens, lean beef, and eggs. Good sources of vitamin B_{12} include meat, poultry, fish, dairy products, and eggs.
- Avoid meals with fatty foods.
- Diet should provide sufficient amounts of energy intake, protein, calcium, iron, and vitamins.
- Extra fluid may be needed for dehydration.
- No gluten restriction is needed.

Common Drugs Used and Potential Side Effects

- Tetracycline may be used; do not take within 2 hours of a calcium-containing supplement or meal as calcium makes the drug less effective.
- Vitamin supplements are required, especially for vitamin B_{12}, folate, and vitamin K. Calcium may also be needed. Avoid excesses of any single nutrient for an extended time period.
- Suggest use of probiotics such as yogurt with live and active cultures during antibiotic therapy. Intestinal flora modifiers (e.g., *Lactobacillus acidophilus*, Lactinex, Bacid) also help recolonize normal intestinal flora in people on antibiotics. A common order is three to four packages every day for 3 days in adults. They may be mixed with water for TFs.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain to the patient which foods are good sources of folic acid and vitamin B_{12}.
- Describe good sources of protein and calories from the diet.
- Describe how to follow a low-fat diet and how to use MCT oil in cooking.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- NIH—Tropical Sprue
  http://www.nlm.nih.gov/MEDLINEPLUS/ency/article/000275.htm
- Tropical Sprue
  http://www.intelihealth.com/IH/ihtIH/WSIHW000/9339/10902.html

TROPICAL SPRUE—CITED REFERENCES

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Nath SK. Tropical sprue. *Curr Gastroenterol Rep.* 7:343, 2005.
Ramakrishna BS. Tropical malabsorption. *Postgrad Med J.* 82:779, 2007.
Ritchie BK, et al. 13 C-sucrose breath test: novel use of a noninvasive biomarker of environmental gut health. *Pediatrics.* 124:620, 2009.

WHIPPLE'S DISEASE (INTESTINAL LIPODYSTROPHY)

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

Whipple's disease is a chronic systemic inflammatory disease caused by *Tropheryma whipplei*. The hallmark of Whipple's disease is invasion of the intestinal mucosa with macrophages incompetent to degrade *T. whipplei* (Moos et al, 2010). This condition presents with weight loss, arthralgias, and diarrhea that involves infiltration of the small intestine with glycoprotein-laden macrophages in varying body tissues (Muller et al, 2005). Endocarditis and heart murmur are common. Infection may spread to the central nervous system, which may lead to loss of memory, confusion, or disturbed gait. Treatment is antibiotic therapy.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Decreased production of Interleukin (IL)-12, IL-2 and Interferon (IFN)-g accompanied by an increased secretion of IL-4 may predispose patients for an infection with *T. whipplei* (Deriban and Marth, 2006).

Clinical/History

Height
Weight
BMI
Weight loss?
Wasting?
Diet history
Fever
I & O
Malabsorption, cholestasis
Gray to brown skin pigmentation
Lymphadenopathy
Confusion, memory loss
Edema?
Duodenal biopsy; villous blunting
Skin biopsy

Lab Work

Alk phos
Na^+, K^+
Ca^{++}, Mg^{++}
Alb (decreased)
CRP (elevated?)
Acid-Schiff (PAS)-positive foamy macrophages
T whipplei DNA—polymerase chain reaction (PCR) assays
H & H (decreased)
Serum Fe, ferritin
Transferrin

INTERVENTION

OBJECTIVES

- Reduce fever and inflammatory processes.
- Correct malnutrition and malabsorption.
- Correct anemia, iron overloading, or hypoproteinemia when present.
- Prevent or correct dehydration and electrolyte imbalances.

FOOD AND NUTRITION

- Use a high-protein/high-calorie diet appropriate for the patient's age and sex.
- Ensure that the diet includes sufficient vitamins and minerals, especially for vitamin D and calcium, when steatorrhea is a problem. Vitamins A, B-complex, and K may also be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Weight Loss

Assessment Data: I & O, weight loss, BMI, fever; low albumin, elevated CRP

Nutrition Diagnoses (PES): Involuntary weight loss (NC-3.2) related to fever and infection as evidenced by BMI below usual range and inflammatory process with elevated CRP.

Interventions: Offer frequent small meals and snacks with nutrient dense, calorie-rich foods. Educate about the role of nutrition in maintaining healthy immune system.

Monitoring and Evaluation: Weight gain back to usual weight; no further fever or weight loss. Maintain adequate hydration.

- Provide adequate fluid intake to reduce fever and replenish tissues.
- If edema is a problem, control excess sodium.

Common Drugs Used and Potential Side Effects

- Antibiotic treatment is mandatory and leads to a rapid clinical improvement and remission in most patients: 2-week parenteral cephalosporins followed by long-term therapy with trimethoprim-sulfamethoxazole. Trimethoprim-sulfamethoxazole (Bactrim) combinations may have numerous side effects; monitor closely for anorexia, nausea, vomiting, and diarrhea.

Herbs, Botanicals, and Supplements

- Use probiotic or prebiotic foods when possible to replenish "good" gut bacteria.
- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss inclusion of high-quality proteins in the diet. Frequent snacks may be beneficial if large meals are not tolerated.
- Provide lists for nutrient-dense foods rich in specific and needed nutrients (e.g., iron, calcium, etc).

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- NIH—Medline
  http://www.nlm.nih.gov/medlineplus/ency/article/000209.htm
- Whipple's Disease
  http://www.whipplesdisease.net/
- Whipple's Disease Info
  http://www.whipplesdisease.info/

WHIPPLE'S DISEASE—CITED REFERENCES

Deriban G, Marth T. Current concepts of immunopathogenesis, diagnosis and therapy in Whipple's disease. *Curr Med Chem.* 13:2921, 2006.

Moos V. Impaired immune functions of monocytes and macrophages in Whipple's disease [published online ahead of print 4 August 2009]. *Gastroenterology.* 138:210, 2010.

Muller SA, et al. Deadly carousel or difficult interpretation of new diagnostic tools for Whipple's disease: case report and review of the literature. *Infection.* 33:39, 2005.

Rectal Disorders

FECAL INCONTINENCE

NUTRITIONAL ACUITY RANKING: LEVEL 1

DEFINITIONS AND BACKGROUND

Fecal incontinence is the inability to control bowel movements, and stool may leak from the rectum unexpectedly. More than 5.5 million Americans have fecal incontinence, affecting both children and adults, especially seniors. Fecal incontinence is more common in women than in men (Novi and Mulvihill, 2005); see Table 7-26. Fecal incontinence affects up to 20% of community-dwelling adults and more than 50% of nursing home residents, and is one of the major risk factors for elderly persons in the nursing home (Leung and Rao, 2009).

Bowel training helps some people relearn how to control their bowels. In some cases, it involves strengthening muscles; in others, it means training the bowels to empty at a specific time of day. Biofeedback and sacral nerve stimulation may be useful in refractory patients and should be considered before colostomy (Leung and Rao, 2009; Jarrett et al, 2005). Biofeedback helps strengthen and coordinate the muscles. Kegel exercises may be used to strengthen the muscles in the pelvic floor, including those involved in controlling stool. This condition is often correlated with the presence of stroke and diabetes, or use of certain psychoactive medications (Quander et al, 2005). Treatment depends on the cause and severity of fecal incontinence; it may include dietary changes, medication, bowel training, and/or surgery. Surgery may be needed if fecal incontinence occurs in severe cases where injury has taken place. In patients with functional bowel disorders not responding to maximal medical treatment, bowel lavage or biofeedback therapy, can nowadays be treated by sacral nerve neuromodulation (Govaert et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Clinical/History		
Height	BMI	BP
Weight	Diet history	History of diarrhea
	I & O	

TABLE 7-26 Fecal Incontinence: Causes and Comments

Cause	Comments
Constipation	One of the most common causes of fecal incontinence, constipation causes large, hard stools to become lodged in the rectum. Watery stool can then leak out around the hardened stool. Constipation also causes the muscles of the rectum to stretch, which weakens the muscles so they cannot hold stool in the rectum long enough for a person to reach a bathroom. In children, early toilet training or some developmental disorders can cause or aggravate constipation. Consult the doctor for specific management techniques.
Damage to the anal sphincter muscles	Fecal incontinence can be caused by injury to one or both of the ring-like muscles at the end of the rectum called the anal internal and/or external sphincters. The sphincters keep stool inside. When damaged, the muscles are not strong enough to do their job, and stool can leak out. In women, the damage often happens when giving birth. The risk of injury is greatest if the doctor uses forceps to help deliver the baby or does an episiotomy, which is a cut in the vaginal area to prevent it from tearing during birth. Hemorrhoid surgery can damage the sphincters as well.
Damage to the nerves of the anal sphincter muscles or the rectum	Fecal incontinence can also be caused by damage to the nerves that control the anal sphincters or to the nerves that sense stool in the rectum. If the nerves that control the sphincters are injured, the muscle does not work properly, and incontinence can occur. If the sensory nerves are damaged, they do not sense that stool is in the rectum. The individual will not feel the need to use the bathroom until stool has leaked out. Nerve damage can be caused by childbirth, a long-term habit of straining to pass stool, stroke, and diseases that affect the nerves, such as diabetes and multiple sclerosis.
Loss of storage capacity in the rectum	Normally, the rectum stretches to hold stool until a person can get to a bathroom. But rectal surgery, radiation treatment, and inflammatory bowel disease can cause scarring that makes the walls of the rectum stiff and less elastic. The rectum then cannot stretch as much and cannot hold stool, and fecal incontinence results. Inflammatory bowel disease also can make rectal walls very irritated and thereby unable to contain stool.
Diarrhea	Diarrhea, or loose stool, is more difficult to control than solid stool that is formed. Even people who do not have fecal incontinence can have an accident when they have diarrhea.
Pelvic floor dysfunction	Abnormalities of the pelvic floor can lead to fecal incontinence. Examples of some abnormalities are decreased perception of rectal sensation, decreased anal canal pressures, decreased squeeze pressure of the anal canal, impaired anal sensation, a dropping down of the rectum (rectal prolapse), protrusion of the rectum through the vagina (rectocele), and/or generalized weakness and sagging of the pelvic floor. Often the cause of pelvic floor dysfunction is childbirth, and incontinence does not show up until the midforties or later (Bharucha et al, 2005).

Constipation	**Lab Work**	Stool (occult blood)
Anal manometry	H & H	Na^+, K^+
Anorectal ultrasonography	Serum Fe, ferritin	Ca^{++}, Mg^{++}
Proctography	BUN	
Proctosigmoidoscopy	Transferrin, TIBC	
Anal electromyography tests for nerve damage	PT	
	Alb, transthyretin	

INTERVENTION

OBJECTIVES

- Timely assessment and intervention are needed to manage constipation and incontinence of stool. Maintain a food diary to determine when incontinence occurs and to identify changes that may be helpful.
- Establish a bowel training regimen to develop a regular pattern of bowel movements; some people train themselves to have bowel movements at specific times during the day, such as after every meal.
- Identify and treat underlying causes, such as diet- or medication-induced diarrhea, constipation, and fecal impaction (Leung and Rao, 2009).
- Prepare for surgery if needed.

FOOD AND NUTRITION

- Daily intake of protein should be appropriate for age and sex. Calories should be calculated as 30–35 kcal/kg.
- Foods that may make the problem worse are drinks containing caffeine, such as coffee, tea, and chocolate, which relax the internal anal sphincter muscle. Other foods that have been implicated are cured or smoked meats such as sausage, ham, or turkey; spicy foods; alcohol; dairy products such as milk, cheese, and ice cream; fruits such as apples, peaches, or pears; fatty and greasy foods; sweeteners, such as sorbitol, xylitol, mannitol, and fructose, which are found in diet drinks, sugarless gum and candy, chocolate, and fruit juices.
- Serve smaller meals more frequently.
- Since liquid helps move food through the digestive system, drink half an hour before or after meals.
- Foods that contain soluble, digestible fiber slow the emptying of the bowels. Bananas, rice, tapioca, bread, potatoes, applesauce, cheese, smooth peanut butter, yogurt, pasta, and oatmeal may be helpful.
- High-fiber foods will add bulk and make stool easier to control. Fiber is found in fruits, vegetables, and grains. 20–30 g of fiber a day is needed; add it slowly.
- Too much fiber all at once can cause bloating, gas, or even diarrhea. If fiber intake makes diarrhea worse, cut back to two servings each of fruits and vegetables and remove skins and seeds. Increase fluid intake when fiber intake is increased to prevent fecal obstruction.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Physical Activity

Assessment Data: I & O, constipation and fecal incontinence, bedridden with no transfer from bed to chair; no walking.

Nutrition Diagnoses (PES): Inadequate physical activity related to nonambulatory status as evidenced by being bedridden for 5 months after hip replacement leading to fecal incontinence.

Interventions: Ensure adequate fluid and fiber intake; prune juice once daily as tolerated. Coordinate with physical therapy to enhance strengthening exercises; with nursing to offer bowel training.

Monitoring and Evaluation: Improvement in fecal continence; resident more compliant with bed exercises for strengthening lean body mass.

Common Drugs Used and Potential Side Effects

- If diarrhea is causing the incontinence, medication may help. Sometimes doctors recommend using bulk laxatives to help people develop a more regular bowel pattern. Antidiarrheal medicines, such as loperamide or diphenoxylate, may be used to slow down the bowel activity.
- Use of probiotic or prebiotic therapy is under study for this condition.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The key to a successful bowel management program rests in tailoring the type of enema, medication, and diet to the specific type of colon (Bischoff et al, 2009).
- The skin around the anus is delicate and sensitive. Constipation and diarrhea or contact between skin and stool can cause pain or itching. To relieve discomfort, wash the area with water, but not soap, after a bowel movement; wash in the shower with lukewarm water or use a sitz bath. Premoistened, alcohol-free towelettes are a better choice than toilet paper. Let the area air dry after washing. Use a moisture-barrier cream. Wear loose clothing, absorbent pads and special undergarments (Leung and Rao, 2009).
- Ensure that the patient adequately exercises, rests, and maintains regular bowel habits.
- Indicate which foods are sources of fiber and other key nutrients in the diet. See Table 7-27.

TABLE 7-27 Fiber Content of Common Foods

Fruits	Amount	Grams of Fiber
Figs	2 dried	4.6
Pear	1 pear	4.0
Raspberries	1/2 cup	4.0
Strawberries, raw	1 cup, sliced	3.8
Apple, large with skin	1 apple	3.7
Apple, medium	1 apple	3.3
Prunes, dried	5 prunes	3.0
Orange	1 orange	3.1
Banana	1 banana	2.8
Tangerine	1 medium	1.9
Peach	1 medium	1.8
Peaches, canned	1/2 cup	1.3
Raisins	1 miniature box (14 g)	0.6
Vegetables		
Peas, split, cooked	1/2 cup	8.1
Lentils, cooked	1/2 cup`	7.8
Beans, kidney, canned	1/2 cup	4.5
Lima beans, cooked	1/2 cup	4.5
Black eyed peas	1/2 cup	4.0
Peas, green, canned	1/2 cup	3.5
Cauliflower	1 cup raw	2.5
Spinach, cooked	1/2 cup	2.2
Cabbage, raw	1 cup	2.0
Brussels sprouts	1/2 cup	2.0
Squash, acorn	1 cup raw	2.1
Carrots, raw	1/2 cup	1.8
Potatoes, boiled	1/2 cup	1.6
Zucchini, raw	1 cup	1.4
Broccoli, raw	1/2 cup	1.3
Celery, raw	1/2 cup	1.0
Lettuce, iceberg	1 cup shredded	0.8

Fruits	Amount	Grams of Fiber
Grains		
All Bran	1/2 cup	9.6
Raisin bran	1 cup	7.5
Shredded wheat	2 biscuits	5.0
Wheat bran flakes	3/4 cup	4.6
Rice, brown, cooked	1 cup	3.5
Total cereal	3/4 cup	3.4
Oatmeal, cooked	3/4 cup	3.0
Wheatena, cooked	1 packet	3.0
Oat bran muffin	One muffin	2.6
Bread, whole wheat	1 slice	1.9
Bread, whole grain (check label)	1 slice	1.7–2
Rye crisp wafer	1 wafer	1.7
Crackers, graham	2 squares	0.4
Bread, white wheat	1 slice	0.6
Other		
Nuts, mixed, dry roast	1 oz	2.6
Apple pie, sliced	1 slice	1.9
Chocolate cake, sliced	1 slice	1.8
Yellow cake, sliced	1 slice	0.2

Source: U.S. Department of Agriculture Nutrient Database for Standard Reference, Release 14 http://www.nal.usda.gov/fnic/foodcomp/Data/SR14/sr14.html, accessed September 1, 2009.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Mayo Clinic
  http://www.mayoclinic.com/health/fecal-incontinence/DS00477
- Medicine Net
  http://www.medicinenet.com/fecal_incontinence/article.htm
- NIDDK—Fecal incontinence
  http://digestive.niddk.nih.gov/ddiseases/pubs/fecalincontinence/

FECAL INCONTINENCE—CITED REFERENCES

Bharucha AE, et al. Relationship between symptoms and disordered continence mechanisms in women with idiopathic faecal incontinence. *Gut.* 54:546, 2005.

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Govaert B, et al. Neuromodulation for functional bowel disorders. *Best Pract Res Clin Gastrointest.* 23:545, 2009.

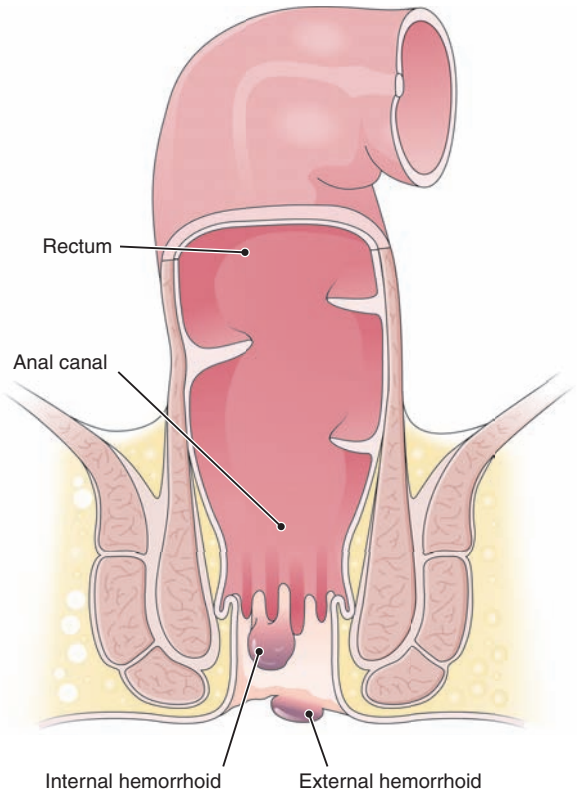
Jarrett ME, et al. Sacral nerve stimulation for fecal incontinence following surgery for rectal prolapse repair: a multicenter study. *Dis Colon Rectum.* 48:1243, 2005.

Novi JM, Mulvihill BH. Fecal incontinence in women: a review of evaluation and management. *Obstet Gynecol Surv.* 60:261, 2005.

Quander CR, et al. Prevalence of and factors associated with fecal incontinence in a large community study of older individuals. *Am J Gastroenterol.* 100:905, 2005.

HEMORRHOIDS

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: Thomas H. McConnell, *The Nature Of Disease Pathology for the Health Professions,* Philadelphia: Lippincott Williams & Wilkins, 2007.

DEFINITIONS AND BACKGROUND

Chronic constipation is believed to be the main cause of hemorrhoids. The disorder is common in Americans (50% of Americans older than 50 years of age will have suffered at least once, especially if obese). Causes include increased abdominal pressure secondary to straining during bowel movements, heavy lifting, childbirth, and benign prostatic hypertrophy.

Internal hemorrhoids are normal anatomical structures and rarely are painful (they may only bleed). External hemorrhoids are usually from excessive diarrhea or from constipation; they are tender, painful, bluish, localized swellings of varicose veins at the anal margin. Bleeding, pain, soiling, and prolapse are the classic symptoms in hemorrhoid disease (Johannsson et al, 2005). Although some patients fear that rectal bleeding signifies colorectal cancer, most patients with the primary diagnosis of symptomatic hemorrhoids do not need further investigative procedures (Tang et al, 2005).

Techniques that fix the cushions back in position can be performed in outpatients with reasonable success rates. Sclerotherapy—Used to treat varicose veins, in this procedure a chemical solution is injected into the vein, which causes the hemorrhoid to collapse. Surgery should be aimed at symptomatic hemorrhoids. Stapled hemorrhoidectomy is safe and effective for acute thrombosed hemorrhoids (Wong et al, 2009). Patients have reduced pain, shorter length of stay, and earlier return to work (Chung et al, 2005). Transanal hemorrhoidal dearterialization is also a potential treatment option for second-degree and third-degree hemorrhoids (Giordano et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Hemorrhoids are not likely to have a genetic origin.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- I & O
- Anal itching
- Bright red blood on toilet tissue
- Pain during bowel movements
- One or more hard tender lumps near the anus
- Anoscope or sigmoidoscopy
- History of diarrhea
- Constipation

Lab Work

- H & H
- Serum Fe, ferritin
- BUN
- Transferrin, TIBC
- Alb, transthyretin
- Stool (occult blood)
- Na^{+}, K^{+}
- Ca^{++}, Mg^{++}

INTERVENTION

OBJECTIVES

- Provide comfort. Prevent prolapse and thrombosis.
- Avoid constipation, infection, and anemia.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Fiber Intake

Assessment Data: I & O and diet history revealing low fiber intake (<10 g daily).

Nutrition Diagnoses (PES): Inadequate fiber intake related to poor dietary intake as evidenced by recurrent hemorrhoids.

Interventions: Food and Nutrient Delivery—provide high fiber foods and extra fluids. Education—high fiber diet and how to assure adequate hydration; increasing physical activity such as walking.

Monitoring and Evaluation: Fewer complaints of hemorroids, straining during deification at stool, dry stool consistency.

- Reduce possible irritation from excessive amounts of fiber. Avoid irritants, such as laxatives.
- After surgery, reduce irritation while patient heals. Promote rapid healing. Prevent future recurrence.

FOOD AND NUTRITION

- Diet should be low in fiber only when the patient is in pain. Otherwise, a high-fiber diet (25–35 g) should be used.
- Fluids should be increased to 8–10 glasses daily.
- After surgery, a low-fiber/soft diet should be used until full recovery occurs. Eventually, adequate fiber (25–35 g) should be taken.
- Omit lactose and highly seasoned foods only if not tolerated by the individual.

Common Drugs Used and Potential Side Effects

- Hemorrhoid creams with lidocaine, over-the-counter, can reduce pain.
- Laxatives and enemas may have caused faulty bowel function. Avoid use unless prescribed by the doctor.
- Troxerutin and carbazochrome are used as a combination therapy. Monitor for any untoward side effects.
- Lubrication with glycerin suppositories may help to reduce symptoms.
- Medicated suppositories such as Anusol HC (contains hydrocortisone) may help to decrease inflammation. Limit steroid-containing medications to less than 2 weeks of continuous use to avoid atrophy of anal tissues.
- Psyllium laxatives (Metamucil) can help with constipation; long-term use alters electrolytes. Flatulence or steatorrhea may occur.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Comfrey, plantain, butcher's broom, horse chestnut, and witch hazel have been recommended for this condition, but no clinical trials have proven efficacy.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Ensure that the patient adequately exercises, rests, and maintains regular bowel habits. Limit the time sitting on the toilet. Avoid straining.
- Teach the patient about the role of fiber in the diet.
- Persistent or recurrent bleeding requires medical attention, especially to monitor vitamin K, iron, and B-complex vitamin levels and to prevent additional losses.
- It is important to keep the anal skin area dry.
- Over-the-counter products may aggravate an allergic response.
- Warm sitz baths may help to reduce symptoms.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Ano-Rectal Algorithm
  http://www.uwgi.org/guidelines/ch_10/ch10.htm
- Hemorrhoids
  http://digestive.niddk.nih.gov/ddiseases/pubs/hemorrhoids/
- Web MD
  http://www.webmd.com/a-to-z-guides/hemorrhoids-topic-overview

HEMORRHOIDS—CITED REFERENCES

Chung CC, et al. Stapled hemorrhoidopexy vs. harmonic scalpel hemorrhoidectomy: a randomized trial. *Dis Colon Rectum.* 48:1213, 2005.

Giordano P, et al. Transanal hemorrhoidal dearterialization: a systematic review. *Dis Colon Rectum.* 52:1665, 2009.

Johannsson HO, et al. Bowel habits in hemorrhoid patients and normal subjects. *Am J Gastroenterol.* 100:401, 2005.

Tang T, et al. An approach to haemorrhoids. *Colorectal Dis.* 7:143, 2005.

Wong JC, et al. Stapled technique for acute thrombosed hemorrhoids: a randomized, controlled trial with long-term results. *Dis Colon Rectum.* 51:397, 2009.

PROCTITIS

NUTRITIONAL ACUITY RANKING: LEVEL 1

DEFINITIONS AND BACKGROUND

Proctitis is inflammation of the lining of the rectal mucosa and may be acute or chronic. Proctitis may be a side effect of medical treatments such as radiation therapy or antibiotics. It may also be caused by UC, rectal injury, bacterial infection, allergies, malfunction of the nerves in the rectum, Crohn's disease, or sexually transmitted diseases.

Proctitis is most common in UC patients at diagnosis, and younger patients are more likely than older patients to have extensive disease (Tarrant et al, 2008). For this form of proctitis, 5-aminosalicyclic acid (5-ASA) or corticosteroids may

be applied directly to the area or taken in a pill form. Ileal pouch rectal anastomosis (IPRA) preserves bowel continuity and provides a good function (Kariv et al, 2009). There may also be a role for appendectomy in ulcerative proctitis (Bolin et al, 2009).

Antibiotics are used for proctitis caused by bacterial infection. Lymphogranuloma venereum (LGV) L2b proctitis may cause proctitis in HIV-positive men who have sex with men (MSM); doxycycline is the drug of choice (White, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Clinical/History

- Height
- Weight
- BMI
- Diet history
- I & O
- BP
- Bowel habits
- Abdominal pain
- Rectal bleeding
- Mucus in stool
- Left-sided abdominal pain
- Frequent or continuous urge to defecate
- Constipation
- Rectal fullness
- Proctoscopy
- Sigmoidoscopy

Lab Work

- Stool (occult blood)
- HPV testing
- H & H
- Serum Fe, ferritin
- Gluc
- BUN, Creat
- Transferrin, TIBC
- Alb, transthyretin
- CRP
- Na^+, K^+
- Ca^{++}, Mg^{++}
- Serum folate, vitamin B_{12}

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Mineral (Iron) Intake

Assessment Data: I & O, abdominal pain, mucus and blood in stool, constipation, poor oral intake, medical diagnosis of anemia, labs indicative of anemia (low H & H, ferritin, Fe).

Nutrition Diagnoses (PES): Inadequate mineral (iron) intake related to blood loss and rectal bleeding in proctitis as evidenced by labs (low Fe, ferritin, H & H).

Interventions: Food and Nutrient Delivery—enhance food choices with iron-rich food choices. Educate about food choices that are rich in iron; discuss heme and nonheme sources. Counseling about use of medications and dietary changes that may be needed with abdominal pain.

Monitoring and Evaluation: Improvement in serum labs, less abdominal pain, fewer incidents of rectal bleeding.

INTERVENTION

OBJECTIVES

- Manage symptoms and alleviate pain. Prepare for surgery if needed.
- Reduce inflammation and promote healing.
- Correct anemia from blood loss where present.

FOOD AND NUTRITION

- Diet therapy depends on the cause of proctitis. Identify appropriate etiology and review entries in this text for dietary management.
- Some patients find that avoidance of caffeine, red meat, dairy products, and artificial sweeteners can be beneficial.
- Use of omega-3 fatty acids may be helpful to reduce inflammation.

Common Drugs Used and Potential Side Effects

- Ulcerative proctitis patients with frequent relapses may need a longer duration of topical therapy. Prolonged oral mesalazine treatment period protects against the proximal spread of rectal inflammation (Pica et al, 2004).
- 5-ASA or corticosteroids may be needed if IBD is the cause. Monitor for numerous side effects.
- Antibiotics are the best treatment for proctitis caused by a specific bacterial infection. When proctitis is caused by use of an antibiotic that destroys normal intestinal bacteria, a doctor may prescribe metronidazole (Flagyl) or vancomycin (Vancocin), which should destroy the harmful bacteria.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Teach the patient about the role of fiber in the diet.
- It is important to keep the anal skin area dry. Over-the-counter products may aggravate an allergic response.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the individual.
- If home TF or CPN is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Ano-Rectal Algorithm
  http://www.uwgi.org/guidelines/ch_10/ch10.htm

- Mayo Clinic—Proctitis
  http://www.mayoclinic.com/health/proctitis/DS00705
- Proctitis
  http://digestive.niddk.nih.gov/ddiseases/pubs/proctitis/

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S E C T I O N 8

Hepatic, Pancreatic, and Biliary Disorders

CHIEF ASSESSMENT FACTORS

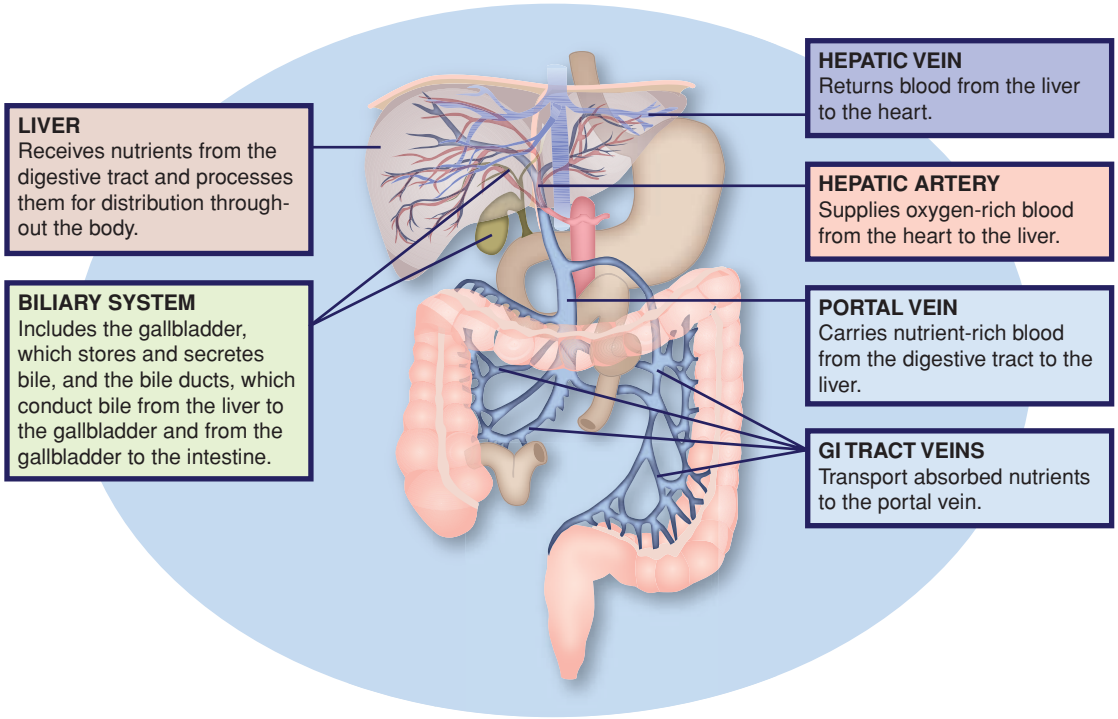
Clinical Factors

- Abnormal Liver MRI, Ultrasound, or Biopsy
- Abdominal or Radiating Pain
- Anorexia, Malaise, Fatigue
- Ascites, Large Abdominal Girth, Pot Belly
- Brownish spots or blemishes on the skin
- Darkened urine
- Depression or mood swings
- Diabetes, Hyperglycemia, Hypoglycemia
- Diarrhea, Steatorrhea
- Edema in feet or ankles
- Encephalopathy?
- Flushed facial appearance
- Gastrointestinal (GI) Bleeding
- Hepatomegaly or Shrunken Liver (in cirrhosis)
- Itchy skin
- Hypoglycemia
- Jaundice
- Light colored stools
- Malnutrition, Subjective Global Assessment (SGA) Score
- Offensive body odor
- Red, swollen or itchy eyes
- Unusual Weight Loss or Gain
- Varices
- Vomiting, Nausea

Laboratory Assessment

A "liver panel" usually includes tests for alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, and alkaline phosphatase. Liver Function is best measured by the prothrombin time (PT), international normalized ratio (INR) and albumin (see below).

- Bilirubin (total or indirect) is released from destroyed red blood cells and passed on to the liver where it is excreted through bile.
- Bleeding/clotting times: PT, INR measure how long it takes blood to form a clot in seconds; a normal PT/INR indicates that a normal amount of blood-clotting protein is available.



- Blood product transfusions.
- Cholestasis tests: Serum alkaline phosphatase (Alk Phos), Gamma-glutamyltransferase (GGT). High alk phos level occurs when there is a blockage of flow in the biliary tract or a buildup of pressure in the liver, often from a gallstone.
- Family history of liver diseases.
- Glucose levels: poorly controlled diabetes can lead to fatty liver.
- Intake of medications, vitamins, herbs, drugs, and alcohol.
- Liver enzymes: ALT (SGPT), AST (SGOT). The AST level is not as helpful as the ALT level for checking the liver.
- Neutrophils: role in inflammation, formation of pus, and destruction of bacteria; check ANC regularly while on interferon treatment. Total white blood cell (WBC) count includes neutrophils plus the four other types (eosinophils, basophils, monocytes, and lymphocytes).
- Pancreatic enzyme levels.
- Serum proteins: PT, INR, albumin, globulin, mitochondrial antibodies, antinuclear and smooth muscle antibodies. A normal INR is 1.0. Low levels of total protein (TP) may indicate impaired liver function.
- Serum ammonia.
- Subjective global assessment (SGA:) evaluation of protein-energy malnutrition (PEM) based on evidence of edema, ascites, muscle wasting, subcutaneous fat loss, decreased functional capacity, and GI symptoms of diarrhea, nausea, and vomiting.
- Specific markers: serum ferritin, ceruloplasmin, alpha-fetoprotein (tumor marker), alpha-1 antitrypsin.
- Thyroid tests: thyroid stimulating hormone (TSH) is produced by the pituitary gland in the brain and causes the thyroid gland to produce thyroid (T4 and T3). TSH may be high if using interferon treatments.
- Viral hepatitis tests (hepatitis A, B, and C serologies).

BACKGROUND: HEPATOBILIARY DISORDERS

Nutrition and the liver are interrelated since everything is refined and detoxified by the liver. The liver is the largest organ in the body, and it performs many complex and essential functions (see Table 8-1). As the body's internal chemical power plant, one cannot live without a liver. Mild elevations in liver chemistries such as ALT and AST can reveal serious underlying conditions, such as viral hepatitis, alcohol use, medication use, steatosis, fatty liver disease, nonalcoholic steatohepatitis (NASH), cirrhosis, or more chronic health conditions, such as diabetes, heart disease, or thyroid disease (Giboney, 2005). Serotonin is a mediator of several hepatic functions; in the diseased liver, it can promote hepatic fibrosis and steatohepatitis (Lesurtel et al, 2008).

Jaundice is a yellowish discoloration of the skin, mucous membranes, and some body fluids from accumulation of bile or bilirubin. Jaundice may be classified as *hemolytic* (from excessive red blood cell destruction), *hepatic* (from an immature liver or from damage), or *obstructive* (from obstructed biliary ducts or gallstones). In neonatal jaundice, there is a somewhat normal pattern of hyperbilirubinemia that is generally not detrimental. In obstructive jaundice, no bile pigment

TABLE 8-1 Liver, Gallbladder, and Pancreatic Functions

Liver

The largest single organ of the body; it is the central biochemical organ of the body. Functionally, it:

1. Converts galactose and fructose to glucose; makes glycogen; degrades glycogen upon demand.
2. Converts proteins into glucose; synthesizes albumin, globulin, fibrinogen, prothrombin, and transferrin; removes nitrogenous wastes (ammonia); provides transamination; synthesizes purines and pyrimidines; forms amines by decarboxylation.
3. Synthesizes triglycerides; forms very low–density lipoproteins (VLDLs); oxidizes fatty acids for energy and ketones.
4. Synthesizes cholesterol from acetate; makes high-density lipoproteins (HDLs).
5. Stores vitamins A, D, E, and K and some vitamin B_{12} and C.
6. Hydroxylates vitamin D for renal activation; activates folic acid to tetrahydrofolic acid (THFA).
7. Stores minerals (e.g., iron, copper, zinc, magnesium).
8. Detoxifies drugs.
9. Produces bile.

Gallbladder

Stores bile, which helps counteract stomach acidity and aids in fat digestion through emulsification.

Pancreas

1. Produces pancreatic juice when stimulated by secretin. Pancreatic juice contains bicarbonate, which helps neutralize acid chyme.
2. Secretes insulin and glucagon hormones.
3. Secretes metabolic/digestive enzymes involved in protein, carbohydrate, and fat metabolism. Pancreatic secretion has gastric, cephalic, and intestinal phases. The islets secrete insulin (b) and glucagon (a). The acini secrete lipase, amylase, trypsin, chymotrypsin, ribonuclease, and carboxypolypeptidase. The pancreas secretes enzymes (trypsin, lipase, and amylase) into the collecting duct as stimulated by cholecystokinin (also called pancreozymin), which is produced by the duodenum.

is present; stools become pale and clay colored, indicating fat maldigestion or malabsorption. Obstructive jaundice leads to bacterial translocation by disruption of the gut barrier, intestinal microecology, and impaired host immune defense. Anorexia is common in obstructive jaundice; biliary drainage improves appetite. Oral administration of an arginine, omega-3 fatty acids, glutamine, and an RNA-supplemented enteral diet for immunonutrition may be useful.

Nonalcoholic fatty liver disease is accumulation of fat in the liver of people who drink little or no alcohol. It may have only symptoms of fatigue, or it may progress to hepatosteatosis (NASH) or liver failure. NASH may occur in obese individuals and may have an inflammatory origin.

Oxidative stress contributes to hepatic cell damage when there is an excess of reactive species derived from oxygen (ROS) and nitrogen (RNS), or a defect of antioxidant molecules (Leung and Chan, 2009). This cell damage aggravates alcoholic liver disease, chronic viral hepatitis, autoimmune liver diseases and nonalcoholic steatohepatitis. Antioxidants in S-adenosylmethionine (SAMe), vitamin E, polyenylphosphatidylcholine, or silymarin have a protective effect on the liver and continue to be studied.

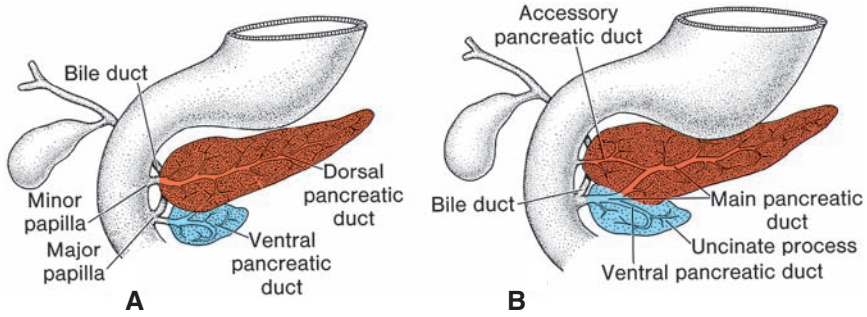
Malnutrition is often a problem in liver diseases. Oral nutritional supplements and tube feeding (TF) can be used to supplement intake when needed. TF improves nutritional status and liver function, reduces the rate of complications, and prolongs survival in cirrhosis and in acute liver failure (ALF). Long-term PN still promotes hepatobiliary dysfunction and steatosis and is used less often.

Use herbs and alternative therapies with caution. Use of glycyrrhizin, phyllanthin, silibinin, milk thistle, and sho-saiko to show merit. However, some herbs are hepatotoxic and should be avoided; this includes comfrey, chaparral, germander, and Chinese herbal mixtures.

BACKGROUND: PANCREATIC AND GALLBLADDER DISORDERS

In the United States, acute pancreatitis, chronic pancreatitis, and pancreatic cancer are the most common pancreatic disease states. Pancreatic cancer is responsible for nearly 30,000 deaths annually. Excessive consumption of alcohol is the major risk factor for pancreatic disease, and smoking causes pancreatic cancer (Lowenfels et al, 2005). To reduce the burden of pancreatic disease, focus on the control of three lifestyle factors: smoking, drinking, and obesity.

Bile is the greenish-yellow fluid made of bile salts and waste products such as bile pigments. Bile flows through small bile ducts inside the liver, then into the common bile duct outside of the liver. Some flows directly to the duodenum, and the rest is stored in the gallbladder. After a meal, the gallbladder releases some bile into the small intestine to help digest fats. Gallbladder disease is also strongly associated with obesity.



For More Information

- Abnormal Liver Function Test Algorithm
  http://www.uwgi.org/guidelines/ch_09/ch09txt.htm
- American Association for the Study of Liver Diseases
  http://www.aasld.org/
- American Liver Foundation
  http://www.liverfoundation.org/
- Centers for Disease Control and Prevention (CDC)—Fast Statistics
  http://www. cdc.gov/nchs/fastats/liverdis.htm
- National Digestive Diseases Information Clearinghouse
  http://digestive.niddk.nih.gov/

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LIVER DISORDERS

ALCOHOLIC LIVER DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 3

DEFINITIONS AND BACKGROUND

Alcohol is a hepatotoxin and is ulcerogenic, especially to the esophagus and other organs. Alcohol cannot be stored and is used preferentially over other energy fuels.

Alcoholic liver disease (ALD) is a major cause of illness and death. ALD affects about 2 million people in the United States. Signs and symptoms of alcoholism include restlessness, agitation, spider angiomas on the face or back or belly, insomnia, anorexia, weight loss, GI cramping, malnutrition, delirium tremens, and hand tremors. In men, altered hair distribution and gynecomastia may occur. Understanding alcohol addiction is key to treating ALD, since abstinence leads to improvement in all forms of alcoholic liver damage (Lucey, 2009). Section 4 addresses alcohol addiction. Table 8-2 lists stages and effects of alcoholism. Given the benefit of drug treatment, it is important to identify patients at risk of early mortality from alcoholic hepatitis using tools such as the Maddrey Discriminant Function, the Model of End-Stage Liver Disease score, and the Glasgow Alcoholic Hepatitis score (Maher, 2007).

Alcoholics may replace as much as one third of their daily energy requirements from alcohol. As a result, they are malnourished. Either they eat poorly or alcohol metabolism prevents them from properly absorbing, digesting, and using nutrients, particularly vitamin A (Plauth et al, 2006). Classic effects of malnutrition from alcoholism include Wernicke's encephalopathy, Korsakoff's psychosis, muscle wasting, weight loss, and liver disease.

Most tissues of the body contain enzymes capable of ethanol metabolism, but significant activity occurs only in

TABLE 8-2 Stages of Alcoholism-Related Effects

Stage	Condition	Effects
I.	Fatty liver (steatosis)	Reversible. Acetaldehyde promotes hepatic fat accumulation. Hepatomegaly, hypertriglyceridemia, hypoalbuminemia, cytochrome P-450 2E1 induction, free radical generation, lipid peroxidation, and increased transcription of proinflammatory mediators, including TNF-alpha, occur.
II.	Alcholic hepatitis	Fibrosis begins. Fever with tachycardia; liver enlargement is mild, and tenderness can occur.
III.	Cirrhosis	Not reversible. Diffuse necrosis and regeneration of fibrous tissue leading to loss of normal hepatic function.
IV.	Encephalopathy or Coma	May lead to death if not treated. Impaired mentation, altered neuromuscular function, and altered consciousness.

the liver and stomach (Lieber, 2005). Alcohol dehydrogenase is made with zinc. Alcohol decreases absorption of fats, fat-soluble vitamins, thiamin, folic acid, vitamin B_{12}, and zinc. Nicotine adenine dinucleotide (NADH) is significant in alcohol metabolism by reduction of pyruvate and promotion of steatosis.

Adequate nutrition is critical and should be provided by TF if necessary (Maher, 2007). A prompt decline in serum bilirubin within 1 week indicates a favorable response to therapy; nonresponders have a 6-month mortality rate of 50% or higher (Maher, 2007).

Plasma homocysteine levels are altered in actively drinking patients, causing brain atrophy and withdrawal seizures (Bleich et al, 2005). Methionine needs to be activated to *S*-adenosylmethionine (SAM); this metabolism is impaired in liver disease. Folate deficiency accentuates abnormal methionine metabolism, lipid oxidation, and liver injury (Halsted et al, 2002; Schalinske and Nieman, 2005). SAM, betaine, and folate decrease oxidative stress by upregulation of glutathione and interleukin-10 and downregulation of tumor necrosis factor-alpha, TNF-a (Purohit et al, 2008). No benefit has been found in randomized, placebo-controlled clinical trials of colchicine, S-adenosylmethionine (SAMe), or phosphatidylcholine (Lucey, 2009). Betaine may attenuate ALD by increasing synthesis of SAM and glutathione, decreasing homocysteine (tHcy) levels (Song et al, 2008). More research is indicated.

Alcohol-induced liver injury is an immunological response of the liver; neutrophils damage liver cells through cytotoxicity (Leevy and Elbeshbeshy, 2005). Men and women metabolize alcohol differently. It takes less time and lower doses of alcohol exposure to cause liver damage in females than in males. Community-dwelling heavy drinkers who are not in alcoholism treatment have dose-related gray matter volume losses (Cardenas et al, 2005).

Treatment strategies for ALD include lifestyle changes for abstinence from alcohol consumption. Nutrition therapy and medications are also important. Serious alcoholic hepatitis has a mortality record of up to 50%. If necessary liver transplantation may be life-saving.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The dopamine (DR2) receptor promotes effects of alcohol. People with a genetic deficit of beta-endorphin peptide are susceptible (Manzardo et al, 2005; Zalewska-Kaszubska and Czarnecka, 2005). The dopaminergic mesolimbic system activates the endogenous mu and delta opioid receptors; mu receptor polymorphisms may be associated with ethanol dependence (Job et al, 2007). Polymorphisms in cytochrome P450 2E1 (CYP2E1), the major microsomal ethanol metabolizing enzyme, can alter detoxification of alcohol by glutathione-S-transferases M1 (GSTM1) and gamma-aminobutyric acid receptor gamma2 (Khan et al, 2009).

Clinical/History

- Height
- Weight
- Body mass index (BMI)
- Usual body weight (UBW)
- Diet history
- Blood pressure (BP)
- Intake and output (I & O)
- Food intolerances, taste aversions
- Anorexia, nausea, vomiting, diarrhea
- Scurvy—ecchymoses, hemorrhagic gingivitis, perifollicular hemorrhages
- Leg edema, poor wound healing
- CT scan or ultrasound of abdomen
- Liver biopsy
- Ascites (mild, moderate, or severe)
- Fatigue
- CAGE test (Cut down, Annoyed, Guilty, Eye opener)
- Alcohol Use Disorders Identification Test (AUDIT)
- Dual-energy x-ray absorptiometry (DEXA) bone scan

Lab Work

- Glucose (increased or decreased)
- Glucose tolerance test (sensitive and reliable)
- AST (increased)
- ALT (normal or only mildly elevated)
- INR
- Bilirubin (often elevated)
- Serum ammonia (may be elevated)
- Blood urea nitrogen (BUN) (low?)
- Albumin or transthyretin (low?)
- C-reactive protein (CRP)
- Triglycerides (increased?)
- Cholesterol (increased or decreased)
- WBC count
- Serum B_{12} and folate
- Plasma homocysteine (high?)
- Na^+ (hyponatremia?)
- K^+
- Hemoglobin and hematocrit (decreased)
- Serum Fe, ferritin
- Transferrin
- Uric acid (UA, increased)
- Globulin
- Alk phos (mildly elevated)
- Mg^{++} (decreased)
- Ca^{++}
- Serum phosphorus (decreased)

INTERVENTION

OBJECTIVES

- Remove alcohol to allow the disabled liver to function more effectively while protecting it from metabolic stress. Avoid alcohol in miscellaneous products, such as vinegar, sauces, and cough syrup.
- Improve health of liver so it can synthesize albumin and other serum proteins. Help liver tissue regenerate; replenish plasma proteins that are lost. Improve skeletal muscle synthesis.
- Prevent hypoglycemia from blocked gluconeogenesis. Correct metabolic syndrome, hyperglycemia, hypertension, or hypertriglyceridemia.
- Repair damage from fatty liver and diminished bile salt synthesis.
- Repair neural damage from malnutrition and malabsorption.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Alcohol Intake

Assessment Data: Dietary intake records; low protein and energy intake for age/gender. Intake of one fifth of vodka per day to the exclusion of most meals.

Nutrition Diagnosis (PES): Excessive alcohol intake >25–30 g/d related to daily consumption above this level as evidenced by alcohol-induced liver injury, elevated LFTs and ascites.

Intervention:

Food and Nutrient Delivery: ND 1.1 General Healthful diet (avoid alcohol); ND 3.1.4 Modified food—increased calorie/protein intake.

Education: E 1.3 Survival Information. Educate about nutrient-dense foods and the role that alcohol plays in liver damage. E-1.1 Present concise and clear educational material with nutritional tips for patients with liver disease.

Counseling: C 2.5 Social support—avoid social outings with alcohol present.

Coordination of Care: RC 1.4 Referral to community agencies/programs. RC-1.3 Refer to social worker for alcohol rehabilitation. C-2.9 Relapse prevention by explaining the pros of following diet and medications as recommended, as the importance of maintain sober.

Monitoring and Evaluation: Track food intake through food diary or history. Coordinate care for rehabilitation program. Follow-up on intake of energy, protein and nutrients after omission of alcohol.

- Correct fluid and electrolyte imbalances, nutritional deficits such as iron deficiency anemia from chronic blood loss in varices, ulcers, and vomiting.
- Be honest and direct in approach. Gently confront conflicting information when stated by the patient.

FOOD AND NUTRITION

- Avoid alcohol to allow the liver to begin heal (DiCecco and Francisco-Ziller, 2006).
- Malnourished alcoholics should consume a diet rich in carbohydrate and protein, preferentially via the oral or enteral route. Provide protein as 1.5 g/kg body weight if malnourished. Plan sufficient carbohydrates and fat to spare protein, but monitor for hyperglycemia or dyslipidemia.
- In hypertensive patients, a Dietary Approaches to Stop Hypertension (DASH) diet may be planned that provides a sufficient mixture of nutrients without excessive kilocalories. All fasting or very low–calorie diets should be avoided.
- Include a mix of fat from omega-3 (fish oils), omega-6 fatty acids, and medium-chain fatty acids.
- Micronutrient deficiencies require supplementation. Supplement the diet with B-complex vitamins, but supplemental vitamins A and D may not be well tolerated. Oral diet should provide adequate amounts of vitamins C, E, and K, phosphorus, potassium, selenium, magnesium, zinc, and calcium.
- Provide small frequent meals to prevent hypoglycemia, resulting from limited glycogen storage.
- Monitor iron intake to avoid excesses from diet or supplements, especially if there is the possibility of iron storage disease.
- Make meals appealing to stimulate the appetite.
- If TF is needed, avoid glutamine-enriched formulas which may increase ammonia levels.

Common Drugs Used and Potential Side Effects

- Corticosteroids have become the standard of care in patients with severe alcoholic hepatitis (Lucey, 2009). Methylprednisolone improves the ability to produce albumin and to normalize PT and bilirubin levels. Side effects may include negative nitrogen balance, hypocalemia, or hyperglycemia.
- Pharmacotherapy for alcoholism with naltrexone, acamprosate, topiramate, and baclofen is exciting (Lucey, 2009). Naltrexone is more effective in some individuals than in others (Rubio et al, 2005).
- Disulfiram (Antabuse) is given with patient's consent. It causes the patient to vomit after ingesting alcohol and can be dangerous.
- Beta-blockers (propranolol, nadolol) or octreotide (Sandostatin) may be used to reduce portal hypertension when varices occur.
- Insulin may be necessary; do not mix with alcohol. Alcohol intake may cause severe hypoglycemia in patients taking insulin (Pedersen-Bjergaard et al, 2005). Metformin should be avoided in patients with liver disease.

Herbs, Botanicals, and Supplements

- Antioxidants are increasingly used. Agents involved in methionine metabolism such as SAM and betaine have shown efficacy in liver disease. Milk thistle (*Silybum marianum*) may have some therapeutic effect as well. *Curcuma longa* (turmeric) and *Glycyrrhiza glabra* (licorice) are being evaluated. Tea polyphenols, especially green tea, may alleviate liver damage (Zhang et al, 2005).
- Herbs and botanical supplements should not be used without discussing with the physician. Chaparral is especially toxic to the liver and should be avoided; severe hepatitis or liver failure may result. Aloe vera should be avoided orally.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient on the sources of necessary nutrients in the diet and use of the prescribed multivitamins. Help patient in the planning and preparing of appetizing, nutrient-dense meals.
- Explain that alcohol is metabolized readily by the liver but cannot be used for muscular activity or energy production. Chemical addiction is a disease; self-help programs and follow-up can reduce dependency.
- General multivitamin–mineral supplementation may improve a poor appetite.

- Obesity, diabetes, and hyperinsulinemia play a role in the development of hepatic steatosis; weight loss remains a critical part of protecting the liver against damage.
- Identify sources of assistance for persons who need help with meal preparation or with access to meals.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Alcoholics Anonymous (AA) World Services
  http://www.alcoholics-anonymous.org/
- Alcoholic Hepatitis
  http://www.emedicine.com/med/topic101.htm
- International Society for Biomedical Research on Alcoholism
  http://www.isbra.com/
- National Council on Alcoholism and Drug Dependence
  http://www.ncadd.org/
- National Institute on Alcohol Abuse and Alcoholism (NIAAA)
  http://www.niaaa.nih.gov/
- International Research Society on Alcoholism
  http://www.rsoa.org/
- Substance Abuse and Mental Health Administration (DHHS)
  http://www.samhsa.gov/

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ASCITES AND CHYLOUS ASCITES

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Ascites is defined as a distended abdomen due to pathological fluid in the peritoneal cavity. The development of ascites indicates a pathological imbalance between the production and resorption of intraperitoneal fluid; appearance and composition vary based on the underlying pathophysiology (Rochling and Zetterman, 2009). Ascites develops in decompensated cirrhosis, cardiac failure, or renal insufficiency. Portal hypertensive gastropathy (PHG) causes upper gastrointestinal bleeding in advanced cases. Liver transplantation may be the only way to improve survival in refractory ascites (Sandhu and Sanyal, 2005).

Although weight is not used for nutritional assessment here, it does help determine fluid balance. The goal of diuretic therapy in ascites is to promote weight loss of 1–3 kg/d. Nutrient depletion can occur if left untreated; fat, proteins, fat-soluble vitamins, and electrolytes may be lost. An oral diet devoid of long-chain triglycerides (LCTs) but that includes medium-chain triglycerides (MCTs) may be used in mild cases.

Management of ascites from decompensated liver disease focus on low-sodium diets and diuretics, supplemented by paracentesis or transvenous intrahepatic portosystemic shunts (Rochling and Zetterman, 2009). While paracentesis improves patient comfort and reduces intra-abdominal pressure and secondary renal dysfunction, it also carries risk for spontaneous bacterial peritonitis (SBP) or renal failure (Sargent, 2006). Bacterial contamination of ascites fluid leading to SBP is caused by bacterial translocation with subsequent bacteremia; proton pump inhibitors (PPIs) suppress gastric acid secretion, and possibly should be avoided in this population (Bajaj et al, 2009).

Chylous ascites is a rare form of ascites, resulting from increased hydrostatic pressure and lymphatic blockade. Accumulation of LCT-dense chyle occurs in the peritoneum. Chyle leaks are a rare complication following abdominal surgery, trauma, cancer, or fistula. Although the incidence of chyle

leak post surgery is low (1–4%), this complication can present significant challenges (Smoke and Delegge, 2008). Any source of large fluid volume losses, lymph vessel obstruction, or leakage may cause chylous effusions in the peritoneal cavities. Most chylous effusions heal spontaneously. Early introduction of enteral feeding may encourage chyle leaks (Malik et al, 2007), whereas total parenteral nutrition along with somatostatin can relieve the symptoms rapidly (Huang et al, 2004).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: No specific genetic causes are clear in cases of ascites.

Clinical/History	Lab Work	
Height	Serum ascites-albumin gradient (>1.1 g/dL = portal hypertension)	ALT
Weight		AST
Dry weight or estimated dry weight		H & H (high in hemochromatosis)
BMI	Alb (decreased)	Serum Fe, ferritin
Diet history	Transthyretin	TIBC,% saturation
BP	CRP	Gluc
I & O	Na^+, K^+	Chol
Temperature	Ca^{++}, Mg^{++}	Trig
Ascites, mild to severe	BUN, creatinine (Creat)	
Ultrasonography		

INTERVENTION

OBJECTIVES

- Reduce fluid retention, usually by diuretics. Mild ascites may present with fluid excess of 3–5 kg; moderate ascites may present with excess of 7–9 kg; and severe ascites may present with excess of 14–15 kg above usual weight.
- Prevent electrolyte imbalances.
- Prevent further pain, fatigue, loss of lean body mass (LBM), and anorexia.
- If possible, prevent hepatorenal syndrome, which can occur in patients with severe liver disease. If severe, it may require transplantation. Prepare for surgery, especially nutritionally (Hasse, 2006).
- Individualize diet as needs change.
- For **chylous ascites**, treat the underlying cause to decrease production of the chylous fluid. Malnutrition is a common result if left untreated; essential fatty acid deficiency must be avoided. Fluid and electrolyte replacement may be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Sodium Intake-Ascites

Assessment Data: Dietary intake records.

Nutrition Diagnosis (PES): Excessive sodium intake related to presence of ascites and portal hypertension as evidenced by paracentesis of 7–8 kg over 24 hours.

Intervention: Food and Nutrient Delivery—manage sodium intake. Educate about sodium sources and requirements. Counsel about preferred foods that are high in sodium and ways to alter intake that are acceptable; how to shop, dine out, travel.

Monitoring and Evaluation: Track food intake through food diary. Follow-up on intake of sodium and alleviation of ascites.

FOOD AND NUTRITION

- Energy needs are often as high as 1.5 times normal, and protein needs are often 1.5 g/kg of body weight (Hasse and Matarese, 2008). Smaller, more frequent meals are often better tolerated.
- If TF or central parenteral nutrition (CPN) is needed, use nutrient-dense formula but not glutamine-enriched formula; glutamine may increase ammonia production. While no high-quality data are available to prove that enteral nutrition is of benefit (Koretz, 2007), malnutrition should be addressed.
- Ensure that intake of vitamins and minerals is adequate. Water-soluble forms of vitamins may be needed; zinc and magnesium may be needed since levels are often low after diuretic therapy (Hasse and Matarese, 2008). Monitor for signs of malnutrition.
- Fluid restriction may be necessary (1–1.5 L/d), with two thirds with meals and one third for thirst/medicines.
- Restrict patient's intake of sodium to 2 g/d (Hasse and Matarese, 2008).
- Often, patients take spironolactone (Aldactone) or have renal insufficiency, which may increase potassium retention. Diet should be altered in potassium if serum levels so indicate. Other diuretics may cause potassium losses.
- For **chylous ascites**, a low-fat diet or enteral feeding is needed with MCTs as the preferred fat source; the addition of essential fatty acids (EFAs) will be needed. Adequate protein and calories are also needed since there may be significant losses. If oral diet fails, CPN may be needed (Assumpcao et al, 2008). Water-miscible forms of fat-soluble vitamins may be needed, along with extra fluid and electrolytes.

Common Drugs Used and Potential Side Effects

- Diuretics are the most important treatment (Rosner et al, 2006). Furosemide (Lasix) is not very effective. Check whether the specific drug retains or spares potassium; spironolactone spares potassium.
- Albumin replacement, while costly, may help to maintain oncotic pressure.
- Somatostatin analogs have been demonstrated to be effective (Huang et al, 2004).
- With bacterial peritonitis, antibiotic therapy is needed. Monitor for specific side effects. PPIs increase enteric bacterial colonization, overgrowth, and translocation (Campbell et al, 2008).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Milk thistle may have some therapeutic effects in liver disease, but no controlled trials have shown efficacy for ascites at this time.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient concerning good sources of key nutrients to include and which nutrients to limit. Instruct patient to follow high-energy, high-protein diet to prevent wasting.
- Ensure that the patient follows a 2-g, low-sodium diet. Explain which foods have hidden sources of sodium, and share recipes if needed.
- For chylous ascites, treatment is generally managed through a hospital stay.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Ascites
  http://www.nlm.nih.gov/medlineplus/ency/article/000286.htm
- Chylous Ascites
  http://emedicine.medscape.com/article/185777-overview
- Medicine Net
  http://www.medicinenet.com/ascites/article.htm

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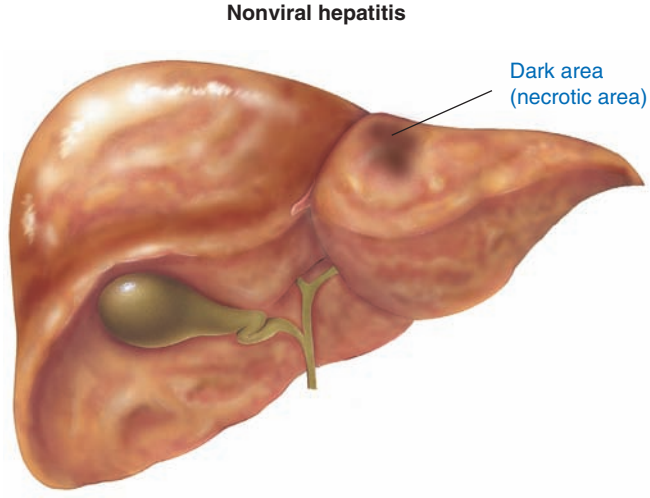
HEPATITIS

NUTRITIONAL ACUITY RANKING: LEVEL 2



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DEFINITIONS AND BACKGROUND

Hepatitis is defined as liver inflammation resulting from alcohol use, toxic materials (carbon tetrachloride), or viral infection (transmitted in food, liquids, or blood transfusions). There is also an autoimmune hepatitis and a NASH.

Acute viral hepatitis is a widespread inflammation of the liver and is caused by hepatitis viruses A, B, C, D, or E. Hepatitis causes nausea, fever, liver tenderness and enlargement, jaundice, pale stools, and anorexia. The first stage of viral hepatitis is preicteric/prodromal (with flu-like symptoms); the second stage is icteric (with jaundice, dark urine, and light stools). The third stage is posticteric/convalescent.

With chronic active hepatitis, inflamed liver cells continue for years, which is usually an autoimmune response. Metabolic diseases, such as Wilson's disease, hemochromatosis, and alpha-1 antitrypsin deficiency, and use of some drugs, such as methyldopa, nitrofurantoin, papaverine, dantrolene, clometacine, and ticrynafen, can cause chronic hepatitis (Hasse and Matarese, 2008). American Indian and Alaska Native (AI/AN) people suffer disproportionately from infectious diseases including **Hepatitis A virus** (**HAV**) and **Hepatitis B virus** (**HBV**); childhood immunizations have reduced these disease disparities (Singleton et al, 2009).

HAV, which is transmitted by fecal–oral route, comprises approximately 50% of hepatitis cases. **HBV** is considered a sexually transmitted disease; 20% of those affected develop

TABLE 8-3 Hepatitis Symptoms, Transmission, and Treatment

Type	Incubation	Symptoms	Transmission	Treatment
Hepatitis A Infectious HAV	30 days	Flu-like illness, jaundice, nausea, fatigue, abdominal pain, anorexia, diarrhea, fever	Ingestion of items contaminated with infected feces, drinking water or ice contaminated with raw sewage, eating fruits, vegetables, or uncooked food contaminated during handling. Risk factors: overseas travel, anal sex, IV drug use, living in poor sanitation.	Immunoglobulin 2–3 months before or 2 weeks after exposure. Vaccine is available.
Hepatitis B Serum HBV	Can survive 7 days outside of the body	Flu-like illness, jaundice, nausea, fatigue, vomiting, fever, often no symptoms	Contact with contaminated body fluids, exposure to sharp instruments that contain contaminated blood, human bites, blood transfusion before 1975. Risk factors: IV drug use, multiple sex partners, travel or work in developing countries, transfusion before 1975.	Interferon alfa and lamivudine. HBV immunoglobulin (HBIG) within 14 days of exposure. There are safe and effective vaccines. Ribavirin is under study.
Hepatitis C HCV	Average 7–9 weeks; can live 28 weeks	Often no symptoms until liver damage occurs, flu-like illness, fatigue, nausea headaches, abdominal pain	Blood-to-blood contact, especially IV drug use and shared needles. Exposure to items with contaminated blood, such as needles (tattoo, body piercing, acupuncture), razors, nail files, toothbrushes, scissors, tampons. Sexually transmitted disease with rashes or sores. Blood transfusions before July 1992. At risk: IV drug use, had a blood transfusion or organ transplantation before July 1992, snorts cocaine. Widespread—affects 4 million people. Silent. Leading cause of cirrhosis in the United States.	Interferon or combination drug treatments. Liver transplantation for end-stage. There are no vaccines. Treatment takes minimum of 1 year. Ribavirin is under study.
Hepatitis D HDV	Occurs only with HBV infection, cannot survive on own	Flu-like illness, jaundice, nausea, fatigue, vomiting, fever, often no symptoms	Sexual contact with HBV-infected person. Exposure to sharp instruments contaminated with HBV. At risk: IV drug use.	Interferon alpha for chronic cases. Vaccination against HBV provides protection against type D.
Hepatitis E HEV	2–9 weeks	Malaise, loss of appetite, abdominal pain, joint pain, fever	Fecal transmission, often through contaminated water. At risk: pregnant women, those who travel in developing countries.	No specific treatment.

some form of chronic liver disease. Table 8-3 provides symptoms and treatments for the many forms of hepatitis, including incomplete viral forms of **Hepatitis D and E**. Hepatitis E virus has been reported to result in chronic hepatitis in transplant patients.

Hepatitis C virus (HCV) is a complex and challenging medical condition. Nearly 20% of Americans test positive for HCV. Many persons with HCV develop chronic disease; 25% of cases lead to cirrhosis, liver cancer, or liver failure that requires transplantation. Recently, the gene marker that predicts response to HCV therapy has been identified; this is significant because about half of cases respond poorly to treatment. Regular coffee consumption is associated with lower rates of disease progression (Freedman et al, 2009) and may actually reduce onset of hepatocellular cancer (Inouye et al, 2005).

A deranged metabolic status and alcohol intake may trigger induction and progression of chronic HCV liver disease; variable intakes of carbohydrates, lipids, polyunsaturated fatty acids, iron, zinc, vitamin A, niacin, and alcohol are factors as are genotype, age, BMI, steatosis, and fibrosis (Loguercio et al, 2008). In the Hepatitis C Antiviral Long-Term Treatment Against Cirrhosis (HALT-C) trial, insulin resistance, histologic features of fatty liver disease, and weight change promoted poor outcomes; improvement in weight may modify disease progression (Everhart et al, 2009). HCV-related cirrhosis can lead to decompensation, end-stage liver failure, and death (Bruno et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: How the hepatitis virus DNA evolves and changes is of great interest to researchers. Since the identification of the hepatitis viruses over the past decades, the understanding of host innate and adaptive immune responses has increased significantly (Rehermann, 2009). Drug-induced liver injury may stem from concomitant hepatic diseases, age, and poor health status; polymorphisms of CYP liver enzymes,

phenotyping and genotyping studies may also be needed (Tarantino et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- I & O
- Diet history
- Right upper abdominal pain
- Jaundice
- Temperature, fever
- Severe nausea, vomiting
- Dark urine
- Joint pain

Lab Work

- IgM antibody for HAV
- Hepatitis B surface antibody (*Anti-HBs or* core antibody (*Anti-HBc*)
- Hepatitis B e antigen (*HBeAg*)
- Hepatitis C Antibody (HCV Ab, anti-HCV)
- Hepatitis C viral load test (or PCR test)
- Bilirubin (increased)
- AST (increased)
- ALT
- Alk phos (increased)
- Chol
- Lactate dehydrogenase (LDH) (increased)
- Alb, transthyretin
- Globulin
- CRP
- Absolute neutrophil count (ANC)
- BUN
- Serum ammonia
- Lipase
- Amylase (increased)
- PT or INR
- Gluc
- Transferrin (increased in acute stage)
- H & H, ferritin
- WBC
- GGT
- Na^+, K^+
- Ca^{++}, Mg^{++}

INTERVENTION

OBJECTIVES

- Promote liver regeneration and rest. Prevent further injury.
- Prevent or correct weight loss, which often results from poor appetite, nausea, and vomiting.
- Spare protein by providing a diet high in carbohydrates.
- Force fluids to prevent dehydration, unless contraindicated.
- Encourage intake of coffee and antioxidant foods.
- Prevent the spread to others by hand washing and safe hygiene practices.

FOOD AND NUTRITION

- For patients with all forms of hepatitis, provide a complete and balanced diet. If nutritional support is necessary, consider TF. Nocturnal feedings may be beneficial.
- Use CPN only if necessary because of ileus or obstruction, and avoid products containing glutamine.
- Progress to a diet of small, frequent feedings of regular or soft foods.
- Diet should provide 30–35 kcal/kg body weight. Provide sufficient carbohydrate to replenish liver stores of glycogen; include 50–55% total energy as carbohydrate.
- Intake of protein should be 1–1.2 g/kg body weight for acute hepatitis. Well-nourished or chronic active hepatitis patients may need levels that just meet dietary reference intake (DRI).

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Bioactive Substance Intake

Assessment: Weight and diet history; abnormal liver tests. Intake <25% for past 3–4 weeks. Diet history reveals low intake of fruits, vegetables, whole grains, and coffee.

Nutrition Diagnosis (PES): Inadequate bioactive substance intake related to loss of appetite as evidenced by low intake of nutrient-dense foods and coffee.

Intervention: Food-Nutrient Delivery: high-protein, high-calorie diet in frequent small meals. Educate about the role of bioactive substances in protecting the liver; encourage use of coffee as tolerated.

Monitoring and Evaluation: Evaluate diet history and weight. Assess for improvement in appetite and intake of foods rich in bioactive substances.

- Fat intake should be moderate to liberal, depending on tolerance. Cut back if diarrhea or other signs of malabsorption occur.
- Supplement diet a multivitamin supplement with B-complex vitamins (especially thiamin, folate, and vitamin B_{12}), vitamin K (to normalize bleeding tendency), vitamin C, and zinc for anorexia and to improve encephalopathy. Monitor for excesses of iron and vitamin A, which may not be well tolerated in a supplemental form.
- Extra fluid should be encouraged, unless contraindicated. Encourage coffee intake (Freedman et al, 2009).

Common Drugs Used and Potential Side Effects

- A combined HAV and HBV vaccine, Twinrix, is available in many parts of the world. Hepatitis B vaccine is found singly as Engerix-B, Recombivax, or Cornvax. Because of vaccinations, the incidence of HAV and HBV has declined, especially among children.
- Two formulations of interferon (standard interferon (IFN) and pegylated IFN) can be used for sustained response to HBV and five nucleoside analogues (lamivudine, adefovir, entecavir, telbivudine, and tenofovir) for treatment-maintained response (Buster et al, 2008). Interferon (Intron-A) can lead to dry mouth, stomatitis, nausea, vomiting, and calcium depletion. The oral antiviral entecavir (Baraclude) is more effective than lamivudine (Dienstag et al, 2007) or adefovir (Leung et al, 2009).
- The gold standard for new patients with chronic HCV is the combination of pegylated interferon (Pegasys or PEG-Intron) and ribavirin (Copegus, Rebetol); individualizing dose and duration improves the sustained virologic response (Camma et al, 2005; Degertekin and Lok, 2009; McHutchison et al, 2009). There may be hemolytic side effects, depression, and weight or lipid changes. Etanercept as adjuvant therapy to interferon and ribavirin improves response and has decreased adverse effects (Zein et al, 2005).
- Steroids may cause sodium retention, nitrogen depletion, or hyperglycemia.

- Monitor for idiosyncratic drug-induced liver injury (DILI;) acute HCV may be present. DILI is caused by a single prescription medication in most cases and by multiple agents or dietary supplements in the remaining cases (Chalasani et al, 2008).

Herbs, Botanicals, and Supplements

- Chaparral and kava kava are especially toxic to the liver and should be avoided.
- Avoid excessive fat-soluble vitamin intake (vitamins A and D). Vitamin A toxicity is possible in compromised liver function; monitor all supplements carefully. Beta-carotene is much less toxic and may offer reasonable antioxidant protection.
- *Silybum marianum* (milk thistle) has been shown to have clinical applications in the treatment of toxic hepatitis and viral hepatitis via its antioxidant properties.
- Oregano, sage, peppermint, garden thyme, lemon balm, clove, allspice, and cinnamon as well as the Chinese medicinal herbs *Cinnamomi cortex* and *Scutellariae radix* contain very high concentrations of antioxidants. In a normal diet, intake of herbs contributes significantly to the total intake of plant antioxidants and can be an even better source of dietary antioxidants than other foods.
- Herbs and botanical supplements should not be used without discussing with the physician. Carrot, schisandra, dandelion, Indian almond, and licorice have been recommended but need research.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help patient make attractive, appetizing meals. Encourage frequent, small meals.
- Educate patient about how to increase calorie, protein, and vitamin intakes. Discuss pros and cons of supplemental products.
- Encourage coffee intake as well as intake of other antioxidant-rich foods.
- Ensure patient abstains from alcohol and drugs that are hepatotoxic. DILI may occur with antibiotics, acetominophen, CNS agents, herbal or dietary supplements.
- All children should receive Hepatitis B vaccinations.
- The NIH recommends that all immigrants be screened and treated for HBV when they move to the United States to prevent liver failure or hepatic carcinoma (NIH, 2008).
- In health care employment, follow standard precautions: handle needles and other sharps safely; report every needlestick on the job; get vaccinated against hepatitis B.
- Consider the risks of HCV before getting a tattoo or body piercing; infection is possible if the tools have someone else's blood on them, or if the artist or piercer does not follow practices such as washing hands and using disposable gloves.

Patient Education—Food Safety

- HAV is usually spread by putting something in the mouth that is contaminated by the stool of another person with hepatitis A. It is usually spread through household contact with an infected person, sharing utensils that are contaminated, eating or drinking contaminated food or water, touching a contaminated surface and then the mouth.
- Teach safe personal hygiene in regard to hand washing and use of disinfectants, especially when traveling overseas. Boil water or drink bottled water in areas where there is a risk for HAV contamination. Eat cooked foods and fruits that you can peel and avoid eating vegetables or fruits that could have been washed with contaminated water, such as lettuce. Avoid eating raw or steamed shellfish, such as oysters, that live in contaminated waters.
- Discuss other principles of food safety and personal hygiene. To protect against HCV, do not share items such as razors, toothbrushes, and other personal health items that might have had blood on them. Do not inject street drugs.

For More Information

- Centers for Disease Control and Prevention (CDC) Information
  http://www.cdc.gov/ncidod/diseases/hepatitis/
- Department of Veterans Affairs
  http://www.hepatitis.va.gov/vahep?page=basics-00–00
- Hepatitis Information Network
  http://www.hepnet.com/
- Hepatitis Information
  http://www.hepatitis.org
- Hepatitis B Foundation
  http://www.hepb.org/
- Hepatitis C Central
  http://www.hepatitis-central.com/
- Hepatitis Foundation International
  http://www.hepatitisfoundation.org/
- National HCV Prison Coalition
  http://www.hcvinprison.org/cms/index.php
- NIDDK—Hepatitis
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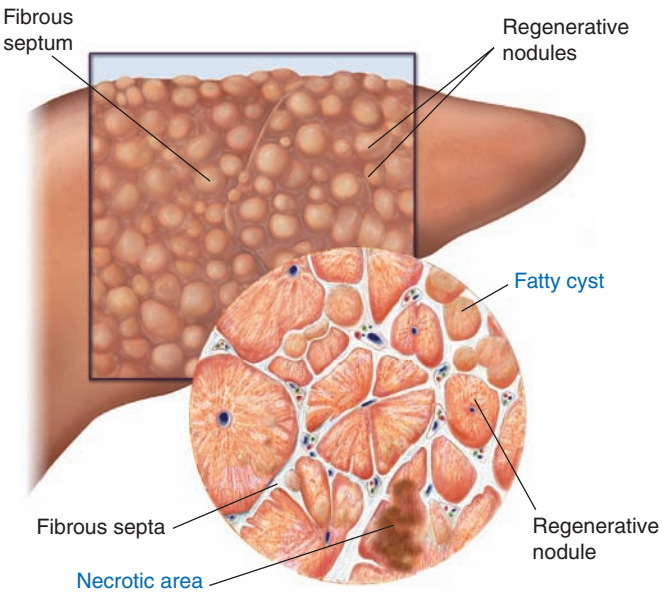
HEPATIC CIRRHOSIS

NUTRITIONAL ACUITY RANKING: LEVEL 2–3



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DEFINITIONS AND BACKGROUND

Cirrhosis is caused by chronic degeneration of the parenchymal liver cells and thickening of the surrounding tissue; the liver slowly deteriorates and malfunctions due to chronic injury. Alcoholism and hepatitis C are the most common causes; alcoholic cirrhosis is known as Laennec's cirrhosis. Cirrhosis may also result from biliary stenosis, hepatitis B-D, obesity with nonalcoholic fatty liver disease (NAFLD), autoimmune hepatitis, prolonged exposure to toxic chemicals, and inherited diseases such as glycogen storage disease, cystic fibrosis, alpha-1 antitrypsin deficiency, hemochromatosis, Wilson disease, or galactosemia.

Malnutrition plays a significant role in the pathogenesis of liver injury and should be carefully managed. Cirrhosis is a disease of accelerated use of alternative fuel (such as fat) since liver stores of glycogen tend to be depleted after an overnight fast. About 50% of energy kilocalories should be consumed from carbohydrate to minimize use of fat stores or protein for energy. Glucose intolerance, insulin resistance, and higher circulating glucagon may cause early satiety, hypophagia, and depleted nutrient stores. There is a high incidence of muscle wasting, weight loss, and malnutrition with cirrhosis. Table 8-4 list the related forms of malnutrition.

Exercise and protein-rich nutrition at the early stage of liver cirrhosis can help to maintain or increase muscular volume (Kotoh et al, 2005). Nutritionally depleted patients need extra attention. Both enteral and parenteral nutritional support can improve the general nutrition condition; EN has fewer complications (Zhang et al, 2005). Cirrhosis related to CPN may be rapidly reversible after isolated intestinal transplantation (Fiel et al, 2009).

Plasma aromatic amino acid (AAA) concentrations (phenylalanine, tyrosine, and tryptophan) tend to increase from rapid muscle proteolysis and decreased synthesis of proteins. Branched-chain amino acids (BCAAs) leucine, isoleucine, and valine are then imbalanced; the low BCAA to AAA ratio contributes to hepatic encephalopathy. When AAAs are high, BCAAs are more limited in cerebral uptake. A higher BCAA intake helps to improve cognitive status (Bianchi et al, 2005; Charlton, 2006). Long-term BCAA supplementation is associated with decreased frequency of hepatic failure and overall complication frequency, along with improved nutritional status (Charlton, 2006).

Severe cirrhosis may lead to decreased serum lipids. Omega-3 fatty acids in enteral supplementation completely protect the liver; IV sources provide only partial protection (Alwayn et al, 2005).

Liver damage from cirrhosis cannot be reversed, but treatment can stop or delay further progression and reduce complications. Participants who consume a diet high in protein are at a higher risk of hospitalization or death due to cirrhosis; those who report a diet high in carbohydrates are at a lower risk after adjusting for daily consumption of protein, carbohydrate, fat, tea or coffee, and alcohol, gender, race, age, educational attainment, U.S. geographical region, diabetes, and BMI ratio (Ioannou et al, 2009).

Treatment will depend on the cause of cirrhosis and any related complications. Serum ammonia levels may be high. Fermentable fiber or lactulose may be used for management of cirrhosis. Antibiotic therapy works to prevent infections, including SBP in cirrhosis (Saab et al, 2009).

Portal hypertension causes increased collateral flow, often with varices in parts of the GI tract. These veins become distended, may bleed, and cause pain. Esophageal varices are the most serious complication of cirrhosis. **Splenorenal shunting** with a trans-jugular intrahepatic portosystemic shunt

TABLE 8-4 Causes of Malnutrition in Cirrhosis

Decreased Oral Intake

- Anorexia
- Ascites
- Altered mental status or encephalopathy
- Delayed gastric emptying
- Early satiety
- Inadequate diet or strict limits on protein, fluid and salt
- Medications causing GI distress or taste changes
- Nausea
- Restrictive diets or NPO for several days

Maldigestion and Malabsorption

- Accelerated intestinal transit
- Accelerated protein breakdown
- Anemia from impaired GI and liver function
- Bacterial overgrowth
- Biliary flow changes
- Choline depletion and betaine
- Decreased hepatic production and storage of nutrients
- Decreased fat absorption
- Diuresis, paracentesis, and micronutrient losses
- Increased rate of gluconeogenesis
- Increased urinary and fecal losses
- Lactulose use
- Pancreatic insufficiency
- Villi damaged by alcohol
- Vomiting

Adapted from Caly WR, et al. Different degrees of malnutrition and immunological alterations according to the aetiology of cirrhosis: a prospective and sequential study. *Nutr J.* 2:10, 2003.
Stockslager JL, et al. *Nutrition made incredibly easy.* Baltimore: Lippincott Williams & Wilkins, 2003, p. 242.

(TIPS) is performed when the portal vein is obstructed. Portacaval blood flow is diverted from the liver by anastomosing the portal vein to the inferior vena cava. The shunt procedure is positive in most cases. Liver transplantation is needed when encephalopathy, ascites, or bleeding varices are uncontrollable.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Autoimmune hepatitis is caused by the body's immune system attacking liver cells and causing inflammation, damage, and cirrhosis; 70% of those with autoimmune hepatitis are female. A cirrhosis-risk score is being developed to assess host genetics and polymorphisms.

Clinical/History	Lab Work	
Height	MELD score for severity (INR for clotting, bilirubin, creatinine)	Alb, transthyretin (not valid in cirrhosis)
Weight	PT (prolonged); INR	ALT
BMI	BUN	AST (increased)
Weight loss?	Globulin	Bilirubin (increased)
I & O	Somatomedin C	Alk phos (increased)
Diet history	TP	WBC
Bowel changes	UA	Trig (increased)
Bleeding hemorrhoids	Gluc (increased or decreased)	Chol (low?)
Confusion	Na^+, K^+	LDH (increased)
Loss of libido	Ca^{++}, Mg^{++}	Copper, ceruloplasmin (increased)
Jaundice	Transferrin	Folate
Ascites	H & H (decreased)	GGT
Spider-like blood vessels on skin	Serum ammonia (elevated?)	
Itching		
Fatigue		
BP (elevated?)		
Easy bruising or bleeding		
CT scan		
Liver biopsy		

INTERVENTION

OBJECTIVES

- Slow the progression of scar tissue and support residual liver function.
- Provide supportive treatment for ascites, edema, muscle wasting, weight loss, esophageal varices, and portal hypertension.
- Monitor steatorrhea; offer suggestions for managing.
- Correct nutritional deficiencies, which are common. Tube feed if needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Oral Food and Beverage Intake

Assessment Data: Dietary intake records indicating poor appetite and intake of <50% at most meals; diagnosis of NASH; mild weight loss 5% in past 3 months; mild jaundice.

Nutrition Diagnosis (PES): Inadequate oral food and beverage related to anorexia as evidenced by intake only about 50% and weight loss of × lb in past 3 months (5% UBW).

Intervention: Food and Nutrient Delivery—offer smaller meals and snacks every few hours while awake; increase nutrient density and quality of foods chosen. Educate about the role of good nutrition in liver health and recovery. Assure no intake of alcohol.

Monitoring and Evaluation: Improvement in appetite, reduced nausea or side effects of liver disease. Gradual return of usual weight.

- Monitor closely for signs of hepatic encephalopathy such as drowsiness or confusion.
- Provide adequate glucose for brain metabolism but beware of glucose intolerance, especially with alcoholic cirrhosis.
- Prevent long-term bone disease, hyperkalemia or hypokalemia, hyponatremia, renal failure, and anemia. If hepatorenal failure occurs, hemodialysis may be necessary.
- Avoid hepatic insults from alcohol, drugs, vitamins and herbal products. Consult the physician first.

FOOD AND NUTRITION

- Increased energy is needed. Calculate energy at 50–75% above usual requirements if malabsorption is present or if repletion is needed. Use ideal or estimated dry weight.
- Diet should provide 1–1.5 g of high-quality protein/kg body weight with adequate carbohydrates to spare protein. Meat has a high level of AAAs; vegetable proteins or casein may be better tolerated.
- Fat is a preferred fuel in cirrhosis. Omega-3 fatty acids should be included (Alwayn et al, 2005). Malabsorption occurs from diminished lipase output; decrease LCTs in steatorrhea. Carefully monitor use of MCTs because they may cause diarrhea or acidosis.
- Supplement diet with B-complex vitamins, vitamins C and K, zinc, and magnesium through foods or supplements. Monitor need for vitamins A and D; do not use excesses in liver disease. Liquid form may be needed for patients with esophageal varices.
- TF can be used with cirrhosis or esophageal varices (Hasse and Matarese, 2008). PN is safe and improves mental state in patients with cirrhosis in whom enteral nutrition is insufficient or impossible (Plauth et al, 2009). Glutamine is not generally recommended in liver disease.
- Avoid alcoholic beverages.
- Control total carbohydrate intake with signs of hyperglycemia or with diabetes.
- Low sodium intake (2–4 g) is recommended with ascites.
- Decrease fluid if there is hyponatremia.
- Enhance nutrient density of food choices if malnourished.

Common Drugs Used and Potential Side Effects

- See Table 8-5.

Herbs, Botanicals, and Supplements

- People with liver disease must be particularly careful because the liver processes almost everything consumed. Herbs and botanical supplements should not be used without discussing with the physician. Some herbal tea preparations may be harmful (such as Comfrey tea) and should be avoided. Chaparral is an herbal product that may contribute to severe hepatitis or liver failure (Stickel et al, 2005). Mistletoe, kava kava, European barberry, and germander should also be avoided.
- Antioxidants, such as vitamin E and selenium, might help in treating cirrhosis. Supplements of vitamins A, C, E, selenium, methionine, and co-enzyme Q10 provide no specific benefit; fresh fruits, vegetables, and whole grains should be consumed instead.
- Betaine (10 g twice daily) reduces elevated homocysteine levels and might be helpful in treating alcohol-induced cirrhosis.
- Bupleurum (*Bupleurum chinese*) is a Chinese herb with anti-inflammatory properties that may lower risk of liver cancer in people with cirrhosis.
- Licorice root (*Glycyrrhiza glabra*) contains glycyrrhizin. Stronger neominophagen C (SNMC) is a Japanese preparation that contains 0.2% glycyrrhizin, 0.1% cysteine, and 2% glyceine that has anti-inflammatory or cytoprotective drug. Do not take licorice with high blood pressure, pregnancy, steroid use, digoxin (Lanoxin), diuretics, or anticoagulants (warfarin/Coumadin).
- Milk thistle (*S. marianum*) has been shown to have clinical applications in the treatment of cirrhosis through its antioxidative and anti-inflammatory effects. A total of 420 mg/d may protect the liver from damage caused by viruses, toxins, alcohol, and drugs such as acetaminophen, but milk thistle does not reduce mortality.
- SAM (*S*-adenosylmethionine 1200–1600 mg/d) may be effective in alcoholic cirrhosis. People with liver disease have low levels of SAMe, and glutathione. Avoid use with prescription antidepressants.
- Zinc deficiency has been implicated in the pathogenesis of liver diseases. Supplementation with zinc may have therapeutic benefits.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Diet may be an important and potentially modifiable determinant of liver disease (Ioannou et al, 2009). Higher CHO and lower protein intake may be a reasonable goal.
- A better appetite at certain meals may be common; breakfast or another meal may be better tolerated. Some patients sleep late with a sleep reversal pattern.
- Discuss use of nutrient-dense foods and higher intakes of vegetable and dairy sources of protein.
- Dietary intake must be adjusted according to the changing status of the patient. Large meals increase portal pressure; recommend use of smaller meals throughout the day.
- Avoid skipping meals. Discuss proper menu planning.
- Avoid high doses of vitamins A and D, which may be toxic to the diseased liver. It is also not clear whether vitamin K supplementation is either safe or useful in cirrhosis. Vitamin E may be beneficial for its antioxidant properties; encourage dietary intake.

Patient Education—Foodborne Illness

- Avoid raw shellfish, which may contain *Vibrio vulnificus* which is dangerous to people with cirrhosis.

TABLE 8-5 Medications Used in Cirrhosis

Medication	Description
Antibiotics: tetracycline (Achromycin V), ampicillin (Polycillin), trimethoprim-sulfamethoxazole (Bactrim, Septra)	Antibiotics kill the bacteria that cause infections, a common complication of cirrhosis. Possible side effects include nausea and vomiting, poor appetite, diarrhea, sore mouth or tongue, and increased sensitivity to sunlight (tetracycline).
Antiviral medications: interferon-alpha (Alferon N, Roferon-A, Intron A), ribavirin (Virazole), lamivudine (Epivir, Epivir-HBV), baraclude (Entecavir)	These may be used if viral hepatitis B or C is the cause of cirrhosis. Treatment usually lasts for 4 months. Ribavirin is an oral antiviral agent that is given twice a day. Lamivudine is used to treat hepatitis B infection. Sometimes lamivudine is combined with interferon. Side effects may include severe GI pain, feeling of fullness, nausea, tingling, burning, numbness, or pain in the hands, arms, feet, or legs.
Anti-inflammatory medications (corticosteroids): prednisone, azathioprine (Imuran)	Corticosteroids reduce liver inflammation and prevent the progression of cirrhosis. High doses given long term are associated with an increase in serious side effects. Lower doses of prednisone may be used when combined with azathioprine. Possible side effects include: hypertension, glucose intolerance, and bone thinning.
Antihypertensives (beta-blockers): atenolol (Tenormin), metoprolol (Lopressor), nadolol (Corgard), propranolol (Inderal), timolol (Blocadren)	Beta-blockers are used to reduce venous blood pressure in the abdomen (portal hypertension) to reduce the risk of esophageal variceal bleeding and other complications. Possible side effects associated with beta-blocker use include: drowsiness, dizziness, cold sensitivity, and sleep disorders.
Diuretics: "Loop" diuretics: bumetanide (Bumex), furosemide (Lasix); thiazide diuretics: hydrochlorothiazide (HydroDIURIL, Esidrix), chlorothiazide (Diuril); potassium-sparing diuretics: amiloride (Midamor), triamterene (Dyrenium)	Diuretics are used to treat the buildup of excess fluid in the body that occurs with cirrhosis (as well as other diseases). These drugs act on the kidneys to increase urine output, which reduces the amount of fluid in the bloodstream. This can help to reduce portal vein hypertension and help alleviate some of the symptoms of cirrhosis, such as fluid accumulation in the abdomen and legs. Possible side effects associated with diuretic use include: loss of appetite, nausea and vomiting, dizziness, headache, lethargy, and altered blood potassium level.
Insulin	If insulin is needed, monitor carefully for hypoglycemic episodes.
Laxatives: beta-galactosidofructose (lactulose [Cephulac] and kristalose [Chronulac])	In cirrhosis, laxatives such as beta-galactosidofructose (lactulose) can help to absorb or bind toxins, such as ammonia, in the intestine and remove them from the body. Possible side effects associated with laxative use include: diarrhea, abdominal cramping, flatulence and bloating, dehydration, and weakness. Take with food or milk.
Metal Chelating Agents: penicillamine (Cuprimine, Depen), trientine (Syprine), deferoxamine (Desferal)	Metal chelating agents draw toxic metals from the bloodstream so that the body can excrete them. Chelating agents are used to rid the body of excess copper in Wilson's disease or excess iron in hemochromatosis. Both of these rare inherited diseases can produce liver damage resulting in cirrhosis. Possible side effects include: fever; joint pain; lesions on the face, neck, scalp, and/or trunk; rash, hives, or itching; swollen glands; sores or white spots on lips or in mouth; cyanosis; blurred vision; convulsions; wheezing or fast breathing; tachycardia; flushing of skin; nausea, vomiting or diarrhea; and blood in the urine.
Vitamin K: phytonadione (AquaMEPHYTON, Mephyton)	Bleeding abnormalities are common in cirrhosis. Vitamin K helps prevent excessive bleeding. Possible side effects include: flushing of the face and unusual taste.

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- CDC—Cirrhosis
  http://www.cdc.gov/nchs/fastats/liverdis.htm
- Cirrhosis Diet
  http://digestive-system.emedtv.com/cirrhosis/cirrhosis-diet.html
- Hepatic Foundation International
  http://www.hepfi.org/
- Hep Net
  http://www.hepcnet.net/nutritionandcirhossis.html
- Medicine Net—Cirrhosis
  http://www.medicinenet.com/cirrhosis/article.htm
- Milk Thistle
  http://nccam.nih.gov/health/milkthistle/ataglance.htm
- National Institutes of Health—Cirrhosis
  http://www.nlm.nih.gov/medlineplus/cirrhosis.html
- University of Maryland
  http://www.umm.edu/altmed/articles/cirrhosis-000037.htm
- Veterans Administration
  http://www.hepatitis.va.gov/vahep?page=diet-03–00
- WebMD—Cirrhosis
  http://www.webmd.com/digestive-disorders/cirrhosis-liver

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Saab S, et al. Oral antibiotic prophylaxis reduces spontaneous bacterial peritonitis occurrence and improves short-term survival in cirrhosis: a meta-analysis. *Am J Gastroenterol.* 104:993, 2009.

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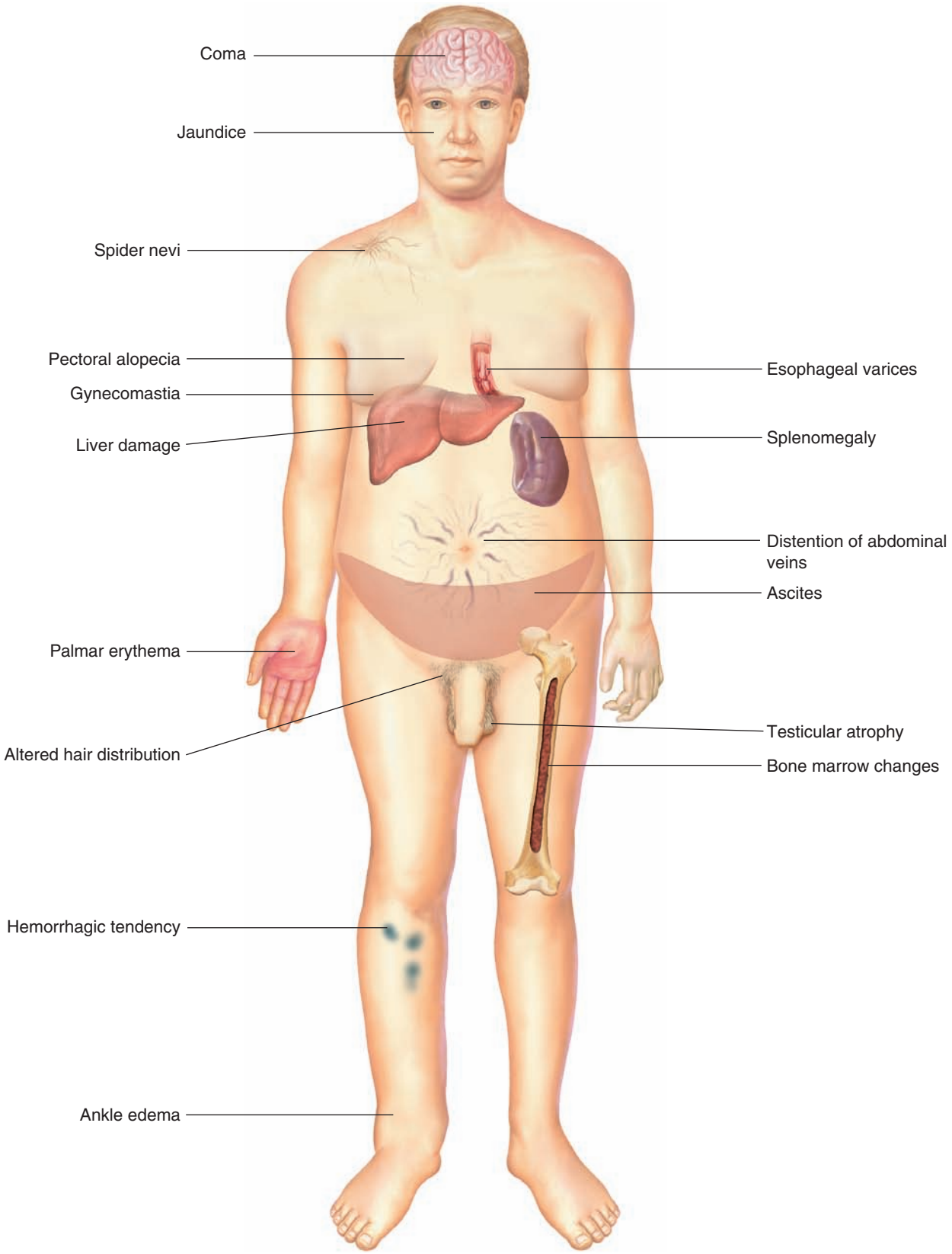
HEPATIC FAILURE, ENCEPHALOPATHY, AND COMA

NUTRITIONAL ACUITY RANKING: LEVEL 3–4



Asset provided by Anatomical Chart Co.

DEFINITIONS AND BACKGROUND

Hepatic failure is common in critical illnesses. Acetaminophen overdose is the leading cause of the acute form. Hallmarks include coagulopathy, usually an INR of 1.5 or more, and encephalopathy. Typical nutrition assessment measures may not reflect the severity of malnutrition because ascites can mask loss of LBM. Blood levels of lactate appear to be good markers for predicting which patients can be managed medically and which need a transplantation (MacQuillan et al, 2005). If hepatorenal syndrome occurs, hemodialysis may be needed; creatinine is not useful here but glomerular filtration rate (GFR) is an important measure.

Hepatic encephalopathy (HE) is a clinical complication caused by portosystemic venous shunting, with or without intrinsic liver disease (Munoz, 2008). HE can be precipitated by GI bleeding, abnormal electrolytes, renal failure, infection, diuretic therapy, use of sedatives or medications that affect the central nervous system, and constipation. HE is estimated to occur in 30–45% of patients with liver cirrhosis and in 10–50% of patients with portosystemic shunts (Eroglu and Byrne, 2009). Patients with HE present with the onset of mental status changes ranging from subtle psychologic abnormalities to profound coma (Munoz, 2008). See Table 8-6 for stages of HE. Acute forms may be reversible; chronic forms may worsen or lead to coma.

Brain glutamine, a byproduct of ammonia detoxification, is elevated in HE (Rama Rao et al, 2005). Causes of hyperammonemia include GI bleed, muscle catabolism, infection, dehydration, noncompliance with lactulose/neomycin, and constipation. The basis of neurotoxicity from ammonia, gamma-aminobutyric acid (GABA), or other agents is not clear. Astrocytes are the most abundant cell type in the brain; they buffer extracellular K(+), regulate neurotransmitter release, form the blood–brain barrier, release growth factors, and regulate the brain immune response (Gee and Keller, 2005). Acute exposure of the astrocytes to ammonia results in alkalinization, with calcium-dependent glutamate release and dysfunction (Rose et al, 2005).

Encephalopathy is usually not caused by altered protein in the diet (Shawcross and Jalan, 2005).

Protein restriction is only necessary in rare, refractory encephalopathy. Patients who have been given a portacaval shunt (TIPS) may benefit from mild protein restriction; nutritional status improves after the shunt.

Decreased dopamine and BCAAs occur in HE; increased AAAs and serotonin also occur. Nevertheless, the use of BCAA solutions is not fully supported by the literature.

Measuring nutritional status in HE can be a challenge. Subjective global assessment and other techniques are not very effective. Measuring handgrip strength may be useful in undernourished patients (Alvares da Silva and Reverbel da Silveira, 2005). Because oxidative stress is a possible trigger in the progression of chronic liver disease, antioxidants and omega-3 polyunsaturated fatty acids may be useful. In addition, because zinc improves taste and immune function, supplementation may improve neurological symptoms and nutrition (Grungrieff and Reinhold, 2005).

Minimal hepatic encephalopathy (MHE) is the mild cognitive impairment commonly seen in patients who have cirrhosis, but it often goes undiagnosed (Stewart and Smith, 2007). It is important to identify signs and symptoms that require medical attention. Commonly associated disorders include energy production deficiencies (hypoglycemia), coagulation abnormalities, immune system dysfunctions, cerebral edema, or **hepatic coma** (Cochran and Losek, 2009). Treatment of HE involves correction of sepsis, gastrointestinal bleeding, and electrolyte imbalance (Sundaram and Shaikh, 2009). Lactulose may be used.

Fischer's ratio between BCAAs and AAAs correlates with the degree of HE; the lower Fischer's ratio, the higher the grade of HE (Koivusalo et al, 2008). Some procedures, such as albumin dialysis, may be used; plasma levels of neuroactive amino acids, methionine, glutamine, glutamate, histidine, and taurine are lowered as a result (Koivusalo et al., 2008).

Signs of impending coma include irritability, change in mentation; disorientation to time and place; asterixis or involuntary jerky movements of the hands; constructional apraxia (inability to draw simple diagrams;) difficulty with writing; ascites, edema; fetor hepaticus (sweet, musty odor of the breath); and GI or esophageal bleeding. Coma patients have increased intracranial pressure and brain edema with a poor prognosis without liver transplantation. Clearly, much more research is needed to resolve these life-threatening disorders.

TABLE 8-6 Stages of Hepatic Encephalopathy—West Haven Classification

Grade 0	Minimal hepatic encephalopathy. Lack of detectable changes in personality or behavior. Minimal changes in memory, concentration, intellectual function, and coordination. Asterixis is absent.
Grade 1	Trivial lack of awareness. Shortened attention span. Impaired addition or subtraction. Hypersomnia, insomnia, or inversion of sleep pattern. Euphoria, depression, or irritability. Mild confusion. Slowing of ability to perform mental tasks. Asterixis can be detected.
Grade 2	Lethargy or apathy. Disorientation. Inappropriate behavior. Slurred speech. Obvious asterixis. Drowsiness, lethargy, gross deficits in ability to perform mental tasks, obvious personality changes, inappropriate behavior, and intermittent disorientation, usually regarding time.
Grade 3	Somnolent but can be aroused, unable to perform mental tasks, disorientation about time and place, marked confusion, amnesia, occasional fits of rage, present but incomprehensible speech.
Grade 4	Coma with or without response to painful stimuli.

Adapted from: Emedicine. Accessed September 5, 2009, at http://www.emedicine.com/med/topic3185.htm.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: HE is generally acquired.

Clinical/History

Height
Weight
Euvolemic (dry) weight
BMI
Diet history
I & O
BP
Muscle stiffness or rigidity
Changes in mentation or personality
Daytime sleepiness
Decreased self-care
Dysfunctional movements, agitation
Flapping tremor (positive Babinski reflex)
Jaundice
Ascites
Early satiety?
Musty odor of breath and urine
Electroencephalogram (EEG)
Handgrip strength
Acute Physiology and Chronic Health Evaluation (APACHE II) score

Lab Work

Serum lactate levels
BUN (decreased)
Creatinine (not valid?)
Bilirubin (increased)
Alk phos (increased)
AST (increased)
Tumor necrosis factor (elevated)
ALT, GGT
Ca^{++}, Mg^{++}
H & H (decreased)
Serum iron, ferritin
Na^{+}, K^{+}
Chol, Trig
UA
Ammonia
Alb (decreased)
Transthyretin
Nitrogen (N) balance
CRP
PT or INR
Transferrin
Gluc (decreased)
Actin-free Gc globulin (Af-Gc)
Plasma isoleucine, leucine, valine
Plasma tryptophan, phenylalanine, tyrosine
Fischer's ratio
Serum insulin, epinephrine
Thyroxine

SAMPLE NUTRITION CARE PROCESS STEPS

Underweight and Altered Nutritional Lab Values

Assessment Data: Dietary intake records; temporal wasting; low weight and BMI of 17; loss of LBM in arms and legs; ascites; confusion and signs of impending coma. Altered LFTs and albumin 2.1 g/dL.

Nutrition Diagnoses (PES):

NC 3.1 Underweight related to decreased appetite prior to admission as evidenced by 90% DBW, BMI 17.

NC 2.2 Altered nutrition related lab value related to liver dysfunction as evidenced by elevated ALT, ALP, AST, NH3, albumin 2.7 g/dL.

Interventions:

Food and Nutrient Delivery: ND 1.2 Modify, distribution type or amount of food and nutrients within meals or specified time (recommend diet change to 2 g sodium, 60 g protein, and six small meals per day; focus on lower animal proteins)

Education: E 1.1 Purpose of nutrition education

Counseling: C 2.2 Goal setting(improve lab values with change)

Coordination of Care: RC 1.1 Team meeting

Monitoring and Evaluation: Track food intake (food diary or history); improvement in albumin or other lab values. Improvement in weight and BMI.

INTERVENTION

OBJECTIVES

- Treat specific causes and prevent multiple organ system failure. Stop any GI bleeding; offer life support if comatose.
- Provide nutrition support to promote regeneration of liver tissue. Support respiratory, neurological, GI, circulatory systems while the liver regenerates.
- Avoid skeletal muscle catabolism from inadequate oral intake, severely restricted diets or nothing by mouth (NPO) status.
- Decrease ammonia and toxin production. Normalize serum amino acid patterns.
- Avoid daytime or nocturnal fasting by using frequent meals and late evening snacks.
- Prevent hypokalemia, sepsis, starvation, and acute crises.
- Reduce circulating amines and lessen shunting of blood around the liver. Control hemorrhage and blood loss into the gut.
- Correct anemia, zinc, and other deficiencies such as magnesium, thiamin, and folate (see Table 8-7).
- Prevent progression to hepatic cancer and improve quality of life.

FOOD AND NUTRITION

Follow Practice Parameters of the American College of Gastroenterology (Blei and Cordoba, 2009):

- **Acute encephalopathy:** Withhold oral intake for 24–48 hours, and provide intravenous glucose until improvement is noted. Start TF if patient appears unable to eat after this period. Protein intake begins at a dose of 0.5 g/kg/d; progress to 1–1.5 g/kg/d.
- **Chronic encephalopathy:** Focus protein intake on dairy products and vegetable-based diets. Consider oral BCAAs for individuals intolerant of all protein.
- **Problematic encephalopathy:** Consider lactulose, neomycin, oral zinc, and surgical shunts.
- With coma, use TF with 0.5–0.6 g protein/kg body weight; advance to 1–1.5 g/kg euvolemic weight. Higher intake of BCAAs and glutamine-enriched products are not usually beneficial.
- Glucose is needed to reduce likelihood or presence of hypoglycemia. Start feeding slowly to prevent refeeding syndrome; then to progress to desired level of intake in the malnourished patient. It is prudent to start with

TABLE 8-7 Nutrient Relationships in Hepatic Failure and Hepatic Encephalopathy

Increased sodium and fluid	Edema; fluid retention
Decreased protein	Swollen belly (ascites) from decreased albumin production
Decreased protein and fat with malabsorption	Somnolence, euphoria, asterixis, coma
Decreased vitamin A	Increased respiratory infections
Decreased vitamins C and K	Hemorrhage; scurvy
Decreased magnesium, niacin, thiamin	Hallucinations, delirium, bei-beri, pellagra
Decreased B-complex vitamins, iron, and protein	Glossitis, anemias
Decreased thiamin	Amnesia, confabulation, Korsakoff's psychosis
Decreased niacin	Memory loss
Decreased folacin	Degeneration of spinal cord
Decreased vitamin K	Muscle weakness
Decreased magnesium	Marked anxiety, hyperirritability, confusion, seizures, tremor
Decreased zinc	Poor taste acuity, impaired wound healing

15–20 kcal/kg and progress as tolerated over several days.

- For the patient who is not comatose, diet should provide moderate-to-high levels of protein (Shawcross and Jalan, 2005). Protein restriction has been discontinued in most cases.
- Use enteral nutrition to correct protein–energy malnutrition. A calorie-dense product is desirable. A nasogastric tube placement may be better tolerated when there is ascites.
- To minimize muscle catabolism, diet should provide extra energy from carbohydrates and fats. Use 30 kcal/kg to maintain and 35 kcal/kg body weight to replete tissue; calculate needs using indirect calorimetry whenever possible. Fats should be 30–35% of kilocalories, using MCT if needed.
- When necessary, administer PN with 50% of energy as nonprotein kilocalories. Because PN does not use the gut, where bacteria may otherwise produce ammonia, parenteral protein is well tolerated and may be given as 1.0–1.5 g/kg. Parenteral solutions have risks of infection and metabolic complications.
- Ensure adequate intake of fluids and electrolytes as monitoring determines. Often, sodium is limited to aid diuresis. Restrict fluid only with dilutional hyponatremia (usually 1000–1500 mL).
- Vitamin–mineral supplements may be needed for niacin, thiamin, folate, phosphate, zinc, calcium, and magnesium.
- Monitor fat-soluble vitamin intake (vitamins A, D, E, and K) carefully and avoid excesses. Avoid copper and manganese at this time, and do not give iron supplements randomly.
- If oral diet is tolerated, use a bedtime snack to avoid hypoglycemia. Small meals and snacks throughout the day may increase intake; oral liquid supplements can be made readily available. Avoid severe restrictions of protein, sodium, fluid, fiber. Liquids are often better tolerated than bulky meals.

Common Drugs Used and Potential Side Effects

- Drug-induced ALF accounts for approximately 20% of ALF in children and a higher percentage of ALF in adults; the most common cause of drug-induced ALF in children is acetaminophen (Murray et al, 2008). N-acetylcysteine is effective in ALF caused by acetaminophen overdose, with better results related to how soon it is given (Khashab et al, 2007).
- For other treatments of HE, see Table 8-8.

Herbs, Botanicals, and Supplements

- Healthy enterocytes can degrade peptides and amino acids and use ammonia via glutamate, glutamine, citrulline, and urea synthesis (Bergen and Wu, 2009). Probiotic, CO_2-producing lactobacilli are useful for enhancing gut microbial metabolism in HE (Bergen and Wu, 2009; Bongaerts et al, 2005). Other treatments using prebiotics and probiotics are under study; see Table 8-9.
- Avoid high doses of vitamins A and D, which may be toxic to the diseased liver.
- Herbs and botanical supplements should not be used without discussing with physician. For example, chaparral use can lead to liver failure. Kava kava and many other products should also be avoided in this population. *Silybum marianum* (milk thistle) is not proven to have a therapeutic role in liver failure.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Hospitalization is usually required; discuss symptoms that require immediate medical attention.
- Dietary intake must be adjusted according to the changing status of the patient. Large meals increase portal pressure; use smaller meals more frequently.
- Milk and eggs tend to produce less ammonia than meats or poultry.
- Discuss the importance of refraining from use of alcoholic beverages.
- A better appetite at certain meals may be common. Identify if breakfast or another meal is best tolerated. Some patients sleep late and have a sleep reversal pattern.
- Discuss proper menu planning. Avoid skipping meals.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

TABLE 8-8 Medications Used for Hepatic Encephalopathy

Medication	Description
Antibiotics: Neomycin	Orally administered antibiotics kill some of the bacteria present within the intestines that produce the dangerous toxins. Be careful not to miss doses. Adverse side effects are common.
Rifaximin	Rifaximin is a nonabsorbed antibiotic with a broad spectrum of activity against aerobic and anaerobic Gram-positive and Gram-negative organisms. It has a better safety and tolerability profile than that of lactulose and possibly neomycin.
Cholestyramine or ursodeoxycholic acid	For itching.
Dietary supplements	Vitamin D and calcium may be needed if osteopenia occurs. Fat-soluble excesses should be avoided since the liver is damaged.
Laxatives: Lactulose (Chronulac, Duphalac, Cholac Syrup, Constulose)	Lactulose is a synthetic sugar used to treat constipation. It is broken down in the colon into products that pull water out from the body and into the colon to soften stools. It also removes ammonia. One or two bowel movements a day are needed. Take lactulose with juice. It may cause abdominal bloating or gas. Be careful not to miss doses, but avoid excesses which can cause diarrhea.
Zinc sulfate or acetate	RNA oxidation and an increase of free intracellular zinc is a consequence of astrocyte swelling and ROS/RNOS production. RNA oxidation may impair postsynaptic protein synthesis, which is critically involved in learning and memory consolidation. Zinc supplementation is recommended.
Medications to avoid	Certain medications can increase the brain's sensitivity to ammonia and other toxins and should not be taken: sedative drugs (Valium, Ativan, Xanax), pain medications (Darvocet, codeine, Vicodin, Percocet, Demerol), antinausea agents (Phenergan, Compazine), antihistamines (Benadryl)

TABLE 8-9 Prebiotics, Probiotics, and Healthy Foods Shopping List[a]

Grains	Beans and Peas (canned/dried)	Oils
Whole grain breads[b] (rye,[c] barley,[c] wheat,[c] oat,[b] buckwheat[b]) Pasta,[b] whole grain[b] Bulgur,[b] wheat berries[c] Polenta, cornmeal Tortillas Flours,[b] whole grain (pastry)[b] Rice, brown Oats Wild rice Exotic grains (spelt, quinoa) Cereals, prepared whole grain Barley,[b] pearled[c]	Beans:[b] black, pinto, garbanzo, kidney lima, soy, small red, small white, cannellini, Black eyed peas, exotics Lentils:[b] black, red, brown, French Split peas (yellow, green) Edamame (soy beans)	Olive Canola or vegetable Peanut Sesame Walnut Exotic

Baking	Nuts and Seeds	Dairy and Cold Case
Flour, whole grain Jam or jelly Syrup Honey Sugar Baking soda/powder Tapioca Vanilla Yeast Chocolate Corn Starch Baking mixes Carob	Almonds[b] Cashews Coconuts, fresh Flaxseed[b] Hazelnuts Macadamias Peanuts Pecans Pine nuts Pistachios Poppy seeds Pumpkin seeds Sesame seeds Sunflower seeds Walnuts Tahini (ground sesame seeds) Nut butters from the above	Pesto Salsa Yogurt[d] Yogurt smoothies[d] Kefir[d] Cottage cheese[b] (check for live cultures or[d] prebiotic inulin)[b] Skim Milk Acidophilus milk[d] Cheese Eggs Dips Spreads Tofu[e] Miso (soy paste)[e]

(continued)

TABLE 8-9 Prebiotics, Probiotics, and Healthy Foods Shopping List[a] *(continued)*

Beverages	Condiments	Meat, Poultry, Fish, Other
Coffee	Vinegar (apple cider, balsamic, red wine, malt)[e]	Chicken
Tea	Horseradish	Turkey
Chocolate or cocoa	Mustard	Beef
Beer[e]	Mayonnaise	Pork
Wine[e]	Catsup	Lamb
Soymilk	Worcestershire[e]	Fish
Nut milk	Soy sauce/Tamari[e]	Exotics: bison, ostrich, etc.
Rice milk	Chutney	Tofu[e]
Kombucha (tea/live cultures)[e]	Salsa	Tempeh (soy beans)[e]
	Chile oil or sauce	Seitan (wheat gluten)
	Wasabi	Natto (fermented beans)[e]
		Soy turkey, soy lunchmeat, etc.

Fermented/Pickled[e]	Snacks	Freezer Items	Deli
Pickled cucumbers	Popcorn	Vegetables	Bean salads
Olives	Dips made from beans, vegetables	Fruits	Grain salads
Pickled beets	Crackers with whole grain	Waffles	Vegetable salads
Kimchi (fermented cabbage)	Chips, whole grain		
Sauerkraut	Snack bars[b] (check ingredients for whole grains,[b] inulin,[b] probiotics[d])		

Vegetables

Artichokes[c]
Asparagus[c]
Avocados
Bamboo shoots
Beans, green or waxed
Beans, lima (unshelled)
Beets
Bok choy
Broccoli
Broccoli rabe
Brussel sprouts
Burdock[c]
Cabbage (red, green, Chinese)
Cauliflower
Carrots
Celery
Celery root
Chestnuts
Chicory[c]
Corn (in husks)
Cucumbers
Daikon radish
Dandelion greens[c]
Eggplant
Endive
Fennel
Fiddleheads
Garlic[c]
Ginger root
Greens (spinach[c], chard, leafy greens etc.)
Horseradish
Jerusalem artichoke[c]
Jicama[c]
Kale
Kohlrabi
Leeks[c]
Lettuce, iceberg
Lettuce, leaf
Lettuce (dandelion greens[c], endive, watercress)
Mushrooms
Okra
Onions[c]
Onions, dry[c]
Onions, green[c]
Palm hearts
Parsnips
Peas (unshelled)
Peppers, chili
Peppers, bell
Potatoes
Potatoes, sweet, yams
Pumpkin
Radishes
Rhubarb

Fruits

Apples
Apricots
Asian pears
Bananas[c]
Berries (raspberry, blackberry, strawberry, gooseberry, elderberry, red currants, exotics)
Cactus pears
Cherries
Coconut, fresh
Cranberries
Currants
Dates

Vegetables (continued)

Rutabagas
Salsify[c]
Seaweed, edible
Shallots[c]
Snow peas
Sprouts, bean, alfalfa, etc
Squash, summer varieties
Squash, winter varieties
Taro
Tomatillo
Tomatoes
Turnips
Watercress

Yacon[c]
Figs
Gooseberries
Grapefruit
Grapes
Guava
Jujubee
Kiwi
Kumquat
Lemon
Lime
Mango
Melon, musk
Nectarines
Oranges
Papaya
Passion fruit
Peaches
Pears
Persimmon
Pineapple
Plantain
Plums, pluot, plumcot
Pomegranate
Pommelo
Raisins
Star fruit
Quince
Watermelon

Herbs and Spices

Allspice
Anise
Basil
Black Pepper
Caraway
Chili
Cilantro
Cinnamon
Clove
Coriander
Cumin
Dill
Fennel
Ginger
Mace
Marjoram
Mint
Nutmeg
Oregano
Parsley
Rosemary
Sage
Savory
Tarragon
Thyme
Turmeric
Vanilla

NOTE—read labels: Strain. What probiotic is inside? *Lactobacillus casei Shirota, Lactobacillus acidophilus, Bifidobacteriumlactis, Saccharomyces cereviase boullardii.* CFU (Colony Forming Units). How many live microorganisms are in each serving? When does it expire? Packaging should ensure an effective level of live bacteria through the "best by" or expiration date. Suggested serving size. How much do I take? Health benefits. What can this product do for me? Proper storage conditions. Where do I keep it to ensure maximum survival of the probiotic? Corporate contact information. Who makes this product? Where to do I go for more information?
From: International Scientific Association for Probiotics and Prebiotics, http://www.ISAPP.net.
Adapted from: Gut Insight © 2009 Gut Insight: probiotics and prebiotics for digestive health and well-being by Jo Ann Tatum Hattner, MPH, RD, with Susan Anderes, MLIS. San Francisco: Hattner Nutrition, 2009. Used with permission.
Other resources: U.S. Probiotics, http://www.usprobiotics.org/.

[a]Seventy percent of the body's immunity is in the gut. There are 300–1000 species of bacteria, 100 trillion in the gut (about 3 lb). Alcohol, smoking, stress, poor bowel hygiene, aging, intestinal infections, antibiotics, and a poor diet can affect intestinal microbiota. The normal levels of lactobacilli, bifidobacteria, and other "good bacteria" may be decreased. Imbalanced flora may lead to abnormal GI function, such as constipation, diarrhea, flares of inflammatory bowel disease or irritable bowel syndrome, other pancreatic or abdominal inflammations, allergic responses, and an impaired immune system. Choosing foods wisely can improve gut health.
[b]Prebiotic potentials.
[c]Prebiotic stars.*
[d]Probiotics.
[e]Fermented foods.

For More Information

- Hepatic Encephalopathy
  http://www.nlm.nih.gov/medlineplus/ency/article/000302.htm
- Medline
  http://www.nlm.nih.gov/medlineplus/ency/article/000302.htm

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LIVER TRANSPLANTATION

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

Liver transplantation (LT) is now a viable alternative for patients with end-stage hepatic failure due to cirrhosis, viral hepatitis, chronic active liver disease, alpha-1 antitrypsin deficiency, primary sclerosing cirrhosis, cholangiocarcinoma, hemochromatosis, autoimmune hepatitis, Budd–Chiari syndrome, hepatoma, primary biliary cirrhosis, or cystic fibrosis.

Nutritional depletion occurs in this population before surgery. Muscle wasting, cachexia, and decreased fat stores are common. Supportive care for all patients with ALF includes adequate enteral nutrition, aggressive screening and treatment of infection, prophylactic broad-spectrum antibiotics, and antifungal agents (Hay, 2004).

Patients are screened carefully for other underlying conditions; many will not be suitable for transplantation. Alcohol is a major contributor to cirrhosis and the need for transplantation. NASH, obesity, diabetes, and hyperinsulinism play a role in cirrhosis and the need for transplantation. Older age, higher BMI, diabetes and HTN are associated with poor outcomes (Malik et al, 2009).

Symptoms and signs leading to the need for transplantation include ascites, jaundice, edema, CNS dysfunction, and cachexia. In living donor liver transplantation (LDLT), a healthy, living person donates a portion of his or her liver to another person.

Preoperative and early postoperative nutrition may speed recovery, lessen time in the intensive care unit, and promote fewer infections. Subjective global assessment (SGA) is often useful because lab work varies so much in liver disease. SGA includes physical signs and symptoms, dietary changes and intolerances, medical/surgical history, GI symptoms and complaints, history of weight loss, and functional capacity. Transthyretin may be a reliable test when the inflammatory process resolves.

Patients with end-stage liver disease are prone to develop osteopenia and osteoporosis, and additional bone loss may occur with the use of immunosuppression agents after transplant. Bone loss occurs early after liver transplant and leads to postoperative fractures, especially with low bone mass. Calcium and vitamin D intake must be a priority.

In general, enteral nutrition is effective in maintaining nutritional status after transplantation. Nutritional supplementation after LT quickly restores protein synthesis. PN may be needed (Plauth et al, 2009). Patients with LT regain a normal life within months of surgery but have a lifetime of immunosuppressive treatment. Intravenous fish oil lipid emulsion may be useful.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Respiratory chain disorders may present with neonatal ALF, hepatic steatohepatitis, cholestasis, cirrhosis with chronic liver failure and the need for LT; molecular defects (mutations in nuclear genes such as SCO1, BCS1L, POLG, DGUOK, and MPV17 and the deletion or rearrangement of mitochondrial DNA) have been identified (Lee and Sokol, 2007). Methylmalonic acidemia with complete mutase deficiency (mut(0) type) is an inborn error of metabolism with high mortality and morbidity; LT may be a solution (Chen et al, 2009).

Clinical/History	Lab Work	
Height	Serum lactate levels	Serum Fe, ferritin
Weight—usual	BUN, Creat	Transferrin
Present weight	Alb, Transthyretin	BUN, creatinine
BMI	N balance	Chol, Trig
SGA with diet history	Na^+, K^+	Carotenoids
Cachexia?	CRP (elevated?)	AST, ALT
Edema	Alk phos	GGT
Nausea and vomiting	Bilirubin	Gluc
I & O	Amino acid profiles	PT or INR
BP	Serum ammonia	
Ascites?	Cerebrospinal fluid (CSF)	
Early satiety?	Ca^{++}, Mg^{++}	
Jaundice	H & H	
CNS dysfunction?		
DEXA		

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Intake From Enteral Nutrition

Assessment Data: I & O records showing long periods without enteral infusion for testing, lab work and nursing shift changes. Dry weight beginning to drop below desired range.

Nutrition Diagnosis (PES): Inadequate intake of enteral nutrition (NI-2.3) related to infusion being held for >3 hours daily as evidenced by intake at 70% of expected protein and kilocalories.

Intervention: Food and Nutrient Delivery—recalculation of TF formula to provide appropriate protein and kilocalories over 21 versus 24 hours daily. Education of nursing staff about necessary change in the TF order; coordination of care.

Monitoring and Evaluation: Track intake of enteral infusion; identify if weight is stable and labs are normal. Determine if current intake meets estimated needs; continue therapy. Evaluate every other day until discharge.

INTERVENTION

OBJECTIVES

Pretransplantation

- Correct malnutrition; lessen edema and ascites.
- Treat hyponatremia and electrolyte imbalances, depending on medications and renal function.
- Prevent or correct catabolic wasting of muscle mass from increased hormonal levels of insulin, glucagon, epinephrine, or cortisol.
- Provide nutritional support in an appropriate mode of feeding to provide a normalized nitrogen balance and other normalized laboratory values. Consider nausea, vomiting, anorexia, diarrhea.
- Correct fat malabsorption, with or without steatorrhea and diarrhea.
- Normalize blood glucose levels and prevent hypoglycemia; diabetes is common.
- Correct abnormal amino acid metabolism and neural accumulation of amino acids that are precursors for dopamine, serotonin, and norepinephrine. Normalize serum ammonia.

Posttransplantation

- Promote normalized protein synthesis in the liver for albumin, globulins, clotting factors. Monitoring nutritional parameters will not be a simple process; many usual measurements are not useful markers of nutritional decline (Shahid et al, 2005).
- Prevent or correct hyperglycemia, fasting hypoglycemia, and abnormal glucagon storage. Diabetes is a common complication from use of prednisone, cyclosporine, and tacrolimus.
- Prevent hypophosphatemia and refeeding syndrome.
- Support wound healing.
- Prevent infection and rejection, the most common complications. Portal venous complications have been well documented and can lead to graft failure (Woo et al, 2007).
- Manage long-term hypercholesterolemia, hypertension, obesity, osteopenia, or bone fractures.
- The role of probiotics in LT is unclear (Jenkins et al, 2005).

FOOD AND NUTRITION

Pretransplantation

- Energy should be 35–45 kcal/kg dry weight for malnourished patients and 30–35 kcal/kg dry weight to maintain weight (Hasse, 2005). Use sufficient carbohydrate and fat to spare protein and meet energy needs. Monitor closely for hyperglycemia.
- Protein needs will vary: 0.8–1.0 g/kg dry weight in compensated liver disease. Calculate 1.5–2.0 g/kg dry weight in decompensated liver disease and 0.6–1.0 g/kg dry weight for HE.
- Modify for fluid, sodium, potassium, and other electrolytes depending on lab values and renal status. Restrict

TABLE 8-10 Post–Liver or Pancreatic Transplant Nutrition Guidelines

Nutrient	LIVER Short-Term	LIVER Long-Term	Pancreas Short-Term	Pancreas Long-Term
Calories	20–30% above normal or measure through indirect calorimetry; increase for weight gain	Maintenance: 30–35 kcal/kg depending on activity level	20–30% above normal or measure through indirect calorimetry; increase for weight gain	Maintenance: 30–35 kcal/kg depending on activity level
Protein	1.3–2 g/kg/d	1 g/kg/d	1.3–2 g/kg/d	1 g/kg/d
Carbohydrate	50–70% of calories	50–70% of calories; restrict simple sugars	45–55% of kcals; use CHO counting	45–55% of kcals; use CHO counting
Fat	30% of calories; up to 50% with severe hyperglycemia	<30% of total calories; <10% saturated fats	25–35% of kcals, depending on lipid levels Up to 50% of calories with severe hyperglycemia	25–35% of total calories; <10% saturated fats
Calcium	800–1200 mg/d	1–1.5 g/d (consider the need for vitamin D supplements)	800–1200 mg/d	1000–1500 mg/d (consider the need for vitamin D supplements)
Sodium	2–4 g/d	3–4 g/d	2–4 g/d	3–4 g/d
Magnesium and phosphorus	Encourage intake of foods high in these nutrients	Encourage intake of foods high in these nutrients; supplement as needed	Encourage intake of foods high in these nutrients; supplement as needed	Encourage intake of foods high in these nutrients; supplement as needed
Potassium	Supplement or restrict based on serum levels	Supplement or restrict based on serum levels	Supplement or restrict based on serum levels	Supplement or restrict based on serum levels
Other vitamins and minerals	Multivitamin–mineral: supplement to DRI or RDA levels	Adequate vitamin D will be needed to prevent deficiency; multivitamin–mineral: supplement to DRI or RDA levels	Multivitamin–mineral: supplement to RDA levels	Multivitamin–mineral: supplement to RDA levels

Source: Hasse J, Matarese L. Medical nutrition therapy for liver, biliary system, and exocrine pancreas disorders. In: Mahan L, Escott-Stump S, eds. *Krause's food nutrition and diet therapy*. 12th ed. St Louis: Elsevier, 2008.

sodium to 2- to 4-g and limit fluid to 1000- to 1500-mL with edema.
- Use a low-volume TF if needed, diluting the concentration. Avoid glutamine-enriched solutions with elevated serum ammonia levels. Consider use of BCAA-enriched formulas.
- Vitamins and minerals should be given at levels that meet but not exceed recommended daily allowances. Fat malabsorption is common; use water-miscible forms of vitamins A, D, E, and K with steatorrhea. B-complex vitamins may be depleted. Stores of calcium, magnesium, potassium, phosphorus, manganese, copper, and zinc are often low and should be repleted.

Post-transplantation

- See Table 8-10.

Common Drugs Used and Potential Side Effects

- See Table 8-11.

Herbs, Botanicals, and Supplements

- Use of probiotics may be quite helpful in this population. A synbiotic composition in an enteral feeding, consisting of one lactic acid bacteria (LAB) and one fiber, greatly reduces incidence of postoperative bacterial infections (Rayes et al, 2005).
- Chaparral should be avoided after transplantation. Herbs and botanical supplements should not be used without discussing with physician. St. John's wort interferes with the metabolism of cyclosporine and should not be used.
- In one study, 50% of LT patients admitted to using vitamins after surgery, and 19% used herbal remedies combined with vitamins, mostly silymarin (Neff et al, 2004).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of diet in wound healing, graft retention, and improvement in health status.
- Provide patient or family with recipes for no-added-salt and sugar-free foods as needed.
- Obesity can occur unless energy intake is controlled over the long term.
- Discuss sources of foods that contain calcium, magnesium, and other desirable nutrients. Individualize to patient preferences and needs.
- Discuss the need for alcohol rehabilitation, family counseling, or other available services. Ethanol (ETOH) abuse affects such key nutrients as niacin, folate, vitamin B_{12}, zinc, phosphorus, and magnesium.

TABLE 8-11 Medications Used after Liver Transplantation

Medication	Description
Analgesics	Analgesics are used to reduce pain. Long-term use may affect such nutrients as vitamin C and folacin; monitor carefully for each specific medication.
Azathioprine (Imuran)	Azathioprine may cause anorexia, leukopenia, thrombocytopenia, oral and esophageal sores, macrocytic anemia, pancreatitis, vomiting, diarrhea, and altered taste acuity. Folate supplementation and other dietary modifications (liquid or soft diet, use of oral supplements, and flavor enhancements) may be needed. The drug works by lowering the number of T cells; it is often prescribed along with prednisone for conventional immunosuppression.
Corticosteroids (prednisone or Solu-Cortef)	Corticosteroids such as prednisone and Solu-Cortef are used for immunosuppression. Side effects include increased catabolism of proteins, negative nitrogen balance, hyperphagia, ulcers, decreased glucose tolerance, sodium retention, fluid retention, and impaired calcium absorption and osteoporosis. Cushing's syndrome, obesity, muscle wasting, and increased gastric secretion may result. A higher protein intake and lower intake of carbohydrate and sodium may be needed.
Cyclosporine	Cyclosporine does not retain sodium as much as corticosteroids do. Intravenous doses are more effective than oral doses. Nausea, vomiting, and diarrhea are common side effects. Hyperlipidemia, hyperglycemia, and hyperkalemia may also occur; decrease fat intake as well as sodium and potassium if necessary. Magnesium may need to be replaced. The drug is also nephrotoxic; a controlled renal diet may be beneficial. Taking omega-3 fatty acids during cyclosporine therapy may reduce toxic side effects (such as high blood pressure and kidney damage) associated with this medication in transplantation patients. Avoid use with St. John's wort.
Diuretics	Diuretics such as furosemide (Lasix) may cause hypokalemia. Aldactone actually spares potassium; monitor drug changes closely. In general, avoid use with fenugreek, yohimbe, and ginkgo.
Immunosuppressants	Immunosuppressants such as muromonab (Orthoclone OKT3) and antithymocyte globulin (ATG) are less nephrotoxic than cyclosporine but can cause nausea, anorexia, diarrhea, and vomiting. Monitor carefully. Fever and stomatitis also may occur; alter diet as needed.
Insulin	Insulin may be necessary during periods of hyperglycemia. Monitor for hypoglycemic symptoms during use.
Mycophenolate mofetil	Diarrhea is common. Extra fluids will be needed.
Muromonab (Orthoclone OKT3)	This drug can lead to nausea, vomiting, diarrhea, and anorexia, and meal adjustments may be needed.
Tacrolimus (Prograf, FK506)	Tacrolimus suppresses T-cell immunity; it is 100 times more potent than cyclosporine, thus requiring smaller doses. Side effects include GI distress, nausea, vomiting, hyperkalemia, and hyperglycemia; adjust diet accordingly by reducing carbohydrate or elevating potassium intake.

- Maintain a good diet and physical activity to support bone density.

Patient Education—Foodborne Illness

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the transplantation individual who may be experiencing graft–host rejection.
- Prevent infections from foodborne illness; patients who have undergone transplantation may be prone to increased risk more than other individuals.

For More Information

- Medicine Net
  http://www.medicinenet.com/liver_transplant/article.htm
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)–Liver Transplant
  http://digestive.niddk.nih.gov/ddiseases/pubs/livertransplant/
- United Network for Organ Sharing
  http://www.unos.org/
- USC Liver Transplant Guide
  http://www.surgery.usc.edu/divisions/hep/patientguide/

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PANCREATIC DISORDERS

PANCREATITIS, ACUTE

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

Acute pancreatitis is an inflammatory, clinical syndrome defined by a discrete episode of abdominal pain and elevations in serum enzyme levels (Gramlich and Taft, 2007). The exocrine pancreas secretes proteolytic, lipolytic, and amylolytic enzymes for nutrient digestion in the intestines. AP is initiated inside acinar cells by premature activation of digestive enzymes in the pancreas instead of the duodenum. Seventy-five percent to 85% of all pancreatic episodes are considered mild and self-limiting and do not require intervention with nutrition support (Gramlich and Taft, 2007). In the unfortunate 15–25% who need nutrition support, preventing multiple organ failure is important.

AP is common in men between the ages of 35 and 45 years, primarily from alcohol abuse or secondarily from gallstones (cholelithiasis). It is often difficult to differentiate between pancreatitis and acute cholecystitis; the correct diagnosis is important because treatments are very different. Other causes of AP include end-stage renal disease, lupus, biliary tract disease, abdominal trauma, certain dyslipidemias (especially triglycerides >1000 mg/dL), AIDS, and pancreatic cancer.

Symptoms of AP include sudden, severe abdominal pain, nausea, vomiting, and diarrhea. Complications include sepsis, acute renal failure, hypovolemia, circulatory shock, and pancreatic necrosis. Abdominal pain can be constant and disabling, causing some patients to become addicted to pain medications. About 25% of persons with AP go on to have chronic pancreatitis. Surgery for AP may include necrosectomy, pancreaticoduodenectomy, or sphincterotomy.

Oxygen free radical–mediated tissue damage is well established in the pathogenesis of AP. Cytokines involved in the systemic inflammatory response in AP include lipid mediators (prostanoids, thromboxanes, and leukotrienes) generated from arachidonic acid. Reactive oxygen species mediate inflammatory cytokine expression and apoptosis of pancreatic acinar cells (Parks et al, 2009). Omega-3 fatty acids DHA and alpha-linolenic acid (ALA), suppress the expression of inflammatory cytokines (IL-1beta, IL-6) and inhibit the activation of transcription factor activator protein-1 in cerulein-stimulated pancreatic acinar cells (Parks et al, 2009). Heat shock proteins (HSPs) that inhabit almost all subcellular locations and cellular membranes play a major role in the protection of cells against stressful and injury-inciting stimuli, including acinar cell injury in AP (Dudeja et al, 2009).

The role of the gut in maintaining immune system integrity is widely recognized. Therefore, nutrition support by the enteral route is preferred (Cao et al, 2008). Nasojejunal feeding tube and a low-molecular diet provide clear advantages compared to parenteral nutrition, such as fewer infectious complications, shorter length of hospital stay, lower cost, and less need for surgery (McClave et al, 2006; Meier and Beglinger, 2006; Petrov et al, 2008). Parenteral nutrition (PN) is used for short term when full nutritional requirements cannot be met enterally so that body composition is preserved (Chandrasegaram et al, 2005).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Evaluation for a susceptible genotype is important (Balog et al, 2005). This is true for pancreatic carcinoma more than pancreatitis per se.

Clinical/History	Lab Work	
Height	CRP (used to measure severity in AP)	Mg^{++} (decreased)
Weight	Procalcitonin (elevated?)	LDH (>700)
BMI (obese?)	Lipase (>110; more sensitive than amylase)	ALT (elevated)
Diet history	Amylase (>250)	AST (>250)
BP (low)	K^{+} (decreased)	WBC (>10,000 cells/mm^3)
Rapid heart rate	Na^{+} (decreased)	Alb (low)
Left upper quadrant abdominal pain	PT or INR	Partial pressure of carbon dioxide (pCO_2) (increased)
Nausea, vomiting	Bilirubin (elevated)	Partial pressure of oxygen (pO_2) (decreased)
Temperature	Ca^{++} (decreased)	BUN
Chvostek's sign	Gluc (increased, >200)	H & H
Steatorrhea or oily stools	Chol (total decreased)	Serum folate
Multiple Organ System Score (MOSS)	LDL cholesterol (elevated?)	Alk phos (increased)
CT scan showing interstitial pancreatic edema	Trig (increased)	CT scan for necrosis
Magnetic resonance imaging (MRI)		Ultrasound
		Fecal fat study

SAMPLE NUTRITION CARE PROCESS STEPS

Impaired Nutrient Utilization

Assessment Data: Patient statement of not taking enzyme medication, reports of abdominal pain, diarrhea after eating, vomiting.

Nutrition Diagnosis (PES): Impaired nutrient utilization related to pancreatic enzyme deficiency as evidenced by abdominal pain and steatorrhea and report of medication noncompliance.

Intervention: Interventions would address the appropriate dose and frequency of medication intake.

Monitoring and Evaluation: Plan would include asking patient time of next clinic visit. Evaluate change in steatorrhea and improvement in enzyme medication compliance.

INTERVENTION

OBJECTIVES

- Reduce pain. Achieve pancreatic rest simultaneously with gut use.
- Use enteral nutrition to reduce the systemic inflammatory response syndrome (Gramlich and Taft, 2007). Failure to use the GI tract in AP may exacerbate disease severity (McClave et al, 2006).
- Avoid pancreatic irritants, especially alcohol and caffeine. Monitor for increased need for pancreatic enzymes with the use of TF.
- Avoid overfeeding.
- Correct fluid and electrolyte imbalances and malnutrition. Acid–base imbalance is common with nasogastric suctioning, fistula losses, renal failure, nausea, and vomiting.
- Reduce fever; prevent shock and hypovolemia, hypermetabolism, sepsis, and compression of the stomach or colon. Avoid cardiovascular, pulmonary, hematological, renal, neurological, or metabolic complications with organ failure. Extensive necrosis and infection are associated with the development of organ failure (Garg et al, 2005).
- Use CPN if abdominal pain is refractory. CPN use can promote positive nitrogen balance (Chandrasegaram et al, 2005).

FOOD AND NUTRITION

- Postpyloric TF is often well tolerated (Niv et al, 2009). Products containing MCT are useful for TF, especially when there is steatorrhea. Omega-3 fatty acids are also helpful (Lasztity et al, 2005). Transition to jejunostomy can be considered when pain is refractory, using a standard formula and needle catheter jejunostomy.
- Progress to a diet given in six daily feedings used with pancreatic enzymes for all meals and snacks.
- Alcoholic beverages and nicotine are prohibited. Limit gastric stimulants, such as peppermint and black pepper, if not tolerated.
- Diet should include adequate amounts of vitamin C, B-complex vitamins, and folic acid for water-soluble vitamin needs. Vitamin B_{12} deficiency can occur because intrinsic factor is prevented from binding with vitamin B_{12}. Use fat-soluble vitamins in water-miscible form.
- Antioxidants including selenium may be needed (Musil et al, 2005). Adequate calcium, magnesium, and zinc supplementation should also be provided.

Common Drugs Used and Potential Side Effects

- Many drugs can trigger acute pancreatitis: furosemide (Lasix), azathioprine, dideoxyinosine, DDI (used for treating AIDS), 6-mercaptopurine, 6-MP (an immunosuppressant drug), angiotensin-converting enzyme (ACE) inhibitors, dapsone, acetaminophen, estrogens, methyldopa, nitrofurantoin, steroids, thiazides, cimetidine, erythromycin, salicylates, sulfonamides, and tetracyclines.
- The most common cause of death in AP patients is infection with enteric bacteria, but there is no convincing evidence for routine administration of prophylactic antibiotics (Wittau et al, 2008).
- See Table 8-12 for guidance on medications.

Herbs, Botanicals, and Supplements

- Antioxidants have protective properties that can be beneficial. See Table 8-13.
- Other herbs and botanical supplements should not be used without discussing with physician as many have hepatotoxic properties.
- Probiotics have had mixed results; they may actually promote higher mortality in patients with AP (Besselink et al, 2008).

TABLE 8-12 Medications Used in Pancreatitis

Medication	Description	Acute	Chronic
Antibiotics	Antibiotics may be needed to manage necrosis and systemic complications.	X	X
Bile salts	Bile salts or water-miscible forms of fat-soluble vitamins may be needed.	X	X
Diuretics	Diuretics such as acetazolamide (Diamox) may be needed to control fluid retention. Nausea, vomiting, and diarrhea may result.	X	X
H_2-receptor antagonists (cimetidine, ranitidine)	Cimetidine may deplete vitamin B_{12}, especially among the elderly. Histamine H_2-receptor antagonists or proton pump inhibitors can improve fat malabsorption and steatorrhea.	—	X
Insulin	Insulin may be necessary. Monitor for hypoglycemia during use.	X	X
Octreotide	Octreotide may have a beneficial role in the management of AP.	X	—
Opiates	Opiates may be prescribed for pain.	X	—
Painkillers	Pain control requires the use of morphine-like drugs (pethidine, morphine, and diamorphine), which have the risk of addiction, particularly if their use is not controlled.	—	X
Pancreatic enzymes	30,000 IU per meal may be needed to reduce steatorrhea to less than 20 g/d. Enteric coating is necessary to prevent destruction by enzymes. Take enteric-coated enzymes with cimetidine, food, or antacids. Capsules or tablets should be swallowed whole.	X	X

TABLE 8-13 Antioxidants and Sources[a]

Vitamins	Sources or Comments
• Vitamin A as beta carotene protects vegetables and fruits from solar radiation damage.	See beta-carotene (below). Supplements are not recommended for general use.
• Vitamin C (ascorbic acid) is a reducing agent.	Citrus fruits (oranges, sweet limes, grapefruit, tangerines), green peppers, broccoli, black currants, strawberries, blueberries, raw cabbage. Destroyed by long-term storage or cooking.
• Vitamin E (alpha-tocopherol) protects lipid membranes. It also protects glutathione peroxidase (GPX-4). Some tocotrienol isomers also have antioxidant properties.	Wheat germ, nuts, seeds, whole grains, green leafy vegetables, vegetable oil, fish oil. GPX4 is the only molecule that efficiently reduces lipid hydroperoxides within the cell membranes.
Vitamin Cofactors and Minerals	
• Coenzyme Q10	Ubiquinol is made in the body and is poorly absorbed from the gut
• Manganese	Part of the superoxide dismutase (SOD) enzyme in mitochondria
• Copper	Part of the superoxide dismutase (SOD) enzyme in the cytosol and extracellular fluid
• Zinc	Part of the superoxide dismutase (SOD) enzyme in the cytosol and extracellular fluid
• Iodide	Iodized salt and seafood
• Selenium and Glutathione	Liver contains a large amount as part of detoxification system. Glutathione is made from amino acids, but diet does not control its production. Acetylcysteine is a sulfur-containing amino acid that may increase glutathione levels.
Carotenoid Terpenoids	
• Alpha-carotene	Less active than beta-carotene
• Astaxanthin	Found naturally in red algae and animals higher in the marine food chain. It is a red pigment familiarly recognized in crustacean shells and salmon flesh/roe.
• Beta-carotene	Butternut squash, carrots, orange bell peppers, pumpkins, broccoli, cantaloupe, peaches, apricots and sweet potatoes
• Canthaxanthin	Edible mushrooms, crustaceans, fish such as carp.
• Lutein	Spinach, red peppers. Destroyed by long-term storage or cooking.
• Lycopene	Ripe red tomatoes, tomato sauces, watermelon
• Zeaxanthin	Yellow corn
Flavonoid Polyphenolics	
• Flavones:	Tea, coffee, soy, fruit, olive oil, red wine, chocolate, cinnamon, oregano
• Apigenin	Parsley, celery, dandelion. Potent inhibitor of CYP2C9, which metabolizes drugs.
• Luteolin	Celery, thyme, green pepper, chamomile tea
• Tangeritin	Tangerine and citrus peels. May help lower cholesterol or prevent Parkinson's disease.
• Flavonols:	The flavonols (kaempferol, quercetin, and myricetin) may reduce the risk of pancreatic cancer.
• Isorhamnetin	Quercetin 3-O-methyltransferase uses S-adenosyl methionine and quercetin to produce S-adenosylhomocysteine and isorhamnetin.
• Kaempferol	Tea, broccoli, grapefruit, brussel sprouts, applies
• Myricetin	Walnuts, grapes, berries, fruits, vegetables, herbs
• Proanthocyanidins, or condensed tannins	Sorghum bran, cocoa powder, and cinnamon are rich sources of procyanidins, found in many fruits and some vegetables
• Quercetin	Apples, onions, beans
• Rutin	Citrus rinds, buckwheat, asparagus, cranberries. Rutin as ferulic acid can help lower cholesterol levels.
• Flavanones:	
• Eriodictyol	From Yerba Santa plant; a glycoside found in rose hips
• Hesperetin	Found in citrus fruits. Metabolizes to hesperidin.
• Naringenin	Grapefruit and citrus fruits. Metabolizes from naringin. Lowers blood lipids, has anticancer activity, inhibits CYP3A4 and CUP1A2 enzymes.
• Flavanols and their polymers:	
• Catechin, gallocatechin and gallate esters	Strawberries, green and black teas
• Epicatechin, epigallocatechin	Cocoa, dark chocolate. Green tea. Improves blood flow.
• Theaflavin	Black tea
• Thearubigins	Formed during fermentation of tea leaves.
• Isoflavone phytoestrogens	Soy, peanuts, other members of the Fabaceae family
• Daidzein	Soybeans, tofu, textured vegetable protein
• Genistein	Soybeans, tofu, textured vegetable protein
• Glycitein	Soy food products

(continued)

TABLE 8-13 Antioxidants and Sources[a] *(continued)*

Vitamins	Sources or Comments
• Stilbenoids:	
• Resveratrol	Skins of dark-colored grapes, and concentrated in red wine
• Pterostilbene	Blueberries and grapes. Methoxylated analogue of resveratrol but not found in wine. May function like metformin to lower blood glucose; more research is needed.
• Anthocyanidins	
• Cyanidin	Grapes, bilberry, blackberry, blueberry, cherry, cranberry, elderberry, hawthorn, loganberry, acai berry and raspberry; apples, red cabbage, and plums.
• Delphinidin	Blue-red grapes, pomegranate, cranberries.
• Malvidin	Red wine
• Pelargonidin	Ripe raspberries, blueberries, blackberries, plums, cranberries, pomegranates.
• Peonidin	Raw cranberries; blueberries, plums, grapes, cherries. Not found in frozen fruits; raw only.
• Petunidin	Chokeberries, muscadine grapes
Phenolic Acids and Their Esters	
• Chicoric acid—a caffeic acid derivative	Found only in Echinacea purpurea.
• Chlorogenic acid—produced from esterification of caffeic acid.	High concentration in coffee (more concentrated in robusta than arabica beans), blueberries and tomatoes.
• Cinnamic acid and ferulic acid	Seeds of plants such as in brown rice, whole wheat and oats, as well as in coffee, apple, artichoke, peanut, orange and pineapple.
• Ellagic acid	Raspberry, strawberry. In ester form in red wine tannins.
• Ellagitannins—hydrolyzable tannin polymer	Formed when ellagic acid, a polyphenol monomer, esterifies and binds with the hydroxyl group of a polyol carbohydrate such as glucose
• Gallic acid	Found in gallnuts, sumac, witch hazel, tea leaves, oak bark, and many other plants
• Gallotannins—hydrolyzable tannin polymer	Formed when gallic acid, a polyphenol monomer, esterifies and binds with the hydroxyl group of a polyol carbohydrate such as glucose.
• Rosmarinic acid	High concentration in rosemary, oregano, lemon balm, sage, and marjoram.
• Salicylic acid	Found in most vegetables, fruits, and herbs; but most abundantly in the bark of willow trees (in aspirin).
Other Nonflavonoid Phenolics	
• Curcumin	Low bioavailability, because, much of it is excreted through glucuronidation. Chemopreventive.
• Eugenol	Oil of cloves. May be toxic if used in undiluted essential oils.
• Flavoglycosides	Gingko
• Flavonolignans—silymarin	Flavonolignans extracted from milk thistle.
• Xanthones—mangosteen and derivatives	Mangostin may only be found in inedible shell
Other Organic Antioxidants	
• Bilirubin, a breakdown product of blood	Possibly a significant antioxidant
• Cannabidiol, THC and synthetic cannabinoids	Cannabidiol is protective against glutamate neurotoxicity; a potent cerebral antioxidant.
• Citric acid	Lemons, limes, other citrus fruits. Part of citric acid cycle, so found in all living things.
• Lignan—antioxidant and phytoestrogen	Oats, flax seeds, pumpkin seeds, sesame seeds, rye, soybeans, broccoli, beans, and some berries.
• N-Acetylcysteine—water soluble	Augments glutathione reserves in the body to protect hepatocytes in the liver from acetaminophen toxicity. Precursor in the formation of the antioxidant glutathione in the body with antioxidant effects to reduce free radicals.
• Oxalic acid	Rhubarb, buckwheat, star fruit, black pepper, parsley, poppy seed, spinach, chard, beets, cocoa, chocolate, most nuts, berries, beans.
• Phytic acid (inositol, phytate)	Storage form of phosphorus in bran and seeds. Sesame seeds, pinto beans, Brazil nuts, peanuts, soybeans, linseed.
• R-α-Lipoic acid—fat and water soluble	Lipoic acid is found in almost all foods; slightly more in kidney, heart, liver, spinach, broccoli, and yeast extract.
• Uric acid	In humans, uric acid accounts for roughly half the antioxidant ability of plasma
Other Substances	
• Melatonin, a hormone	Easily crosses the blood-brain barrier. Once oxidized, it cannot be reduced to its former state.

[a]Antioxidants prevent or slow down oxidation where free radicals damage cells; they are often reducing agents. While supplements are available from many health food stores and nutraceutical companies, clinical trials have not found them to be useful and some may be harmful in elderly or vulnerable populations. See also, Table 1–23 and Table 2–2.

Sources:
1. Antioxidants. Accessed September 15, 2009, at http://en.wikipedia.org/wiki/List_of_antioxidants_in_food.
2. Nothlings U, et al. Flavonols and pancreatic cancer risk. *Am J Epid* 166:924, 2007.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient to watch for signs and symptoms of diabetes, tetany, peritonitis, acute respiratory distress syndrome, and pleural effusion. These patients are best managed by a multidisciplinary team approach, especially in pediatrics (Stringer et al, 2005).
- Discuss omission of alcohol and gas-forming foods.
- Discuss tips for handling nausea and vomiting (e.g., dry meals, taking liquids a few hours before or after meals, use of ice chips, sipping beverages, asking physician about available antiemetics).
- If home enteral nutrition is needed, teach appropriate management methods.
- Teach use of a low-fat, high-protein, high-calorie oral diet when and if appropriate.
- Discuss the use of foods rich in antioxidants.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- American Gastroenterological Association
  http://www.gastro.org/clinicalRes/brochures/pancreatitis.html
- Childhood Pancreatitis
  http://www.aafp.org/afp/990501ap/2507.html
- Medscape
  http://emedicine.medscape.com/article/775867-overview
- Merck—Pancreatitis
  http://www.merck.com/mmhe/sec09/ch124/ch124b.html

PANCREATITIS, ACUTE—CITED REFERENCES

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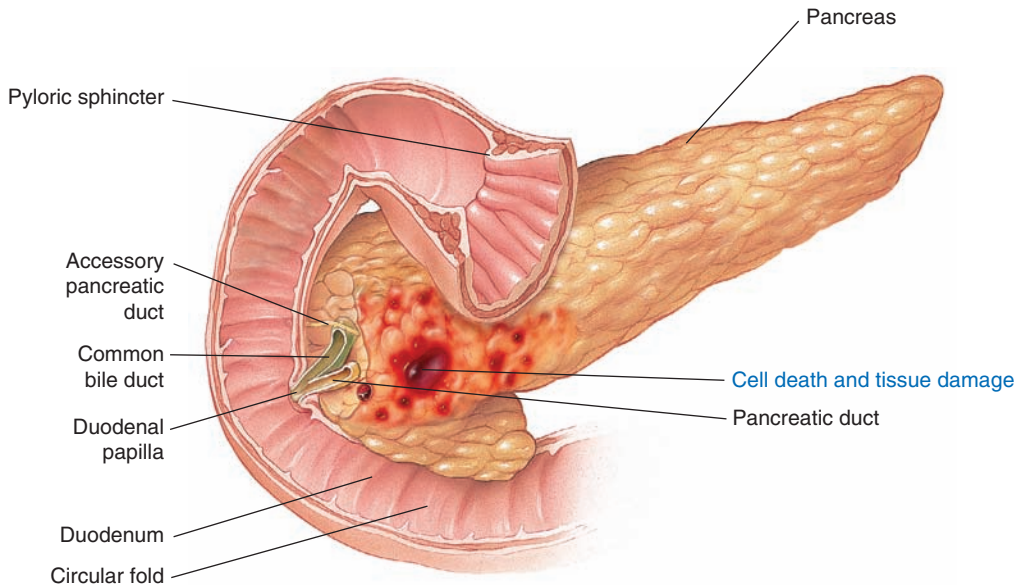
PANCREATITIS, CHRONIC

NUTRITIONAL ACUITY RANKING: LEVEL 3



Asset provided by Anatomical Chart Co.

DEFINITIONS AND BACKGROUND

Chronic pancreatitis (CP) is an inflammatory disorder that results in permanent impairment of the pancreas. CP involves edema, fat necrosis, cellular exudate, fibrosis, and decreased enzymatic processes with abdominal pain, nausea, vomiting, and diarrhea. CP typically develops in people between the ages of 30 and 40, and can be caused by hereditary disorders of the pancreas, cystic fibrosis, hypercalcemia, excessive use of alcohol, hyperparathyroidism, hyperlipidemia, or lipase deficiency. The autoimmune forms of CP have been identified through elevated IgG4 levels. Approximately 5% of persons with acute pancreatitis go on to have CP.

Very heavy alcohol consumption and smoking are independent risks for CP (Yaday et al, 2009). Chronic alcohol abuse is the cause in 70% of adult cases, proportional to the dose and duration of alcohol consumption. The factors determining which alcoholic will develop alcoholic CP involve genetic and dietary factors or pancreatic injury from trauma, gallstones, and viruses. There is a gradual decrease in antioxidant enzyme expression in the pancreatic cells in CP and many patients will progress to having diabetes or pancreatic cancer.

Analysis of pancreatograms and textural changes of the parenchyma help diagnose CP (Kwon and Brugge, 2005). To avoid nutritional deterioration, screen early for fat malabsorption from decreased lipase levels. Steatorrhea occurs when fecal fat >7 g/dL is noted and over 90% of pancreatic enzyme secretion is lost. Weight loss is common, as is pain after intake of foods containing fat and protein. Jaundice, hypoalbuminemia, pancreatic pseudocysts or calcification, and splenic vein thrombosis may also occur.

Abstinence from alcohol, dietary modifications, use of oral supplements, and pancreatic enzyme supplementation will suffice in most patients (Meier et al, 2006). Enteral nutrition may be necessary for those in whom weight loss continues. Long-term use of a jejunostomy feeding may be needed (Stanga et al, 2005). PN is very seldom needed and is not as beneficial.

Oxidative stress occurs when there is an imbalance between generation of reactive oxygen species and inadequate antioxidant defense systems, causing cell damage either directly or through altering signaling pathways. Antioxidant supplementation relieves pain and reduces levels of oxidative stress (Bhardwaj et al, 2009; Verlaan et al, 2006). Patients may benefit from supplementation of vitamins A, C, E, selenium, and carotenoids. Episodes of inflammation and duct obstruction cause disabling pain (Gabbrielli et al, 2005). Some patients become dependent on pain medications. Supportive treatments, inhibition of gastric acid secretion, nerve blocks, reduction of oxidative stress, and endoscopic and surgical treatments are used.

Surgery, such distal pancreatectomy, achieves pain relief and better quality of life. The first successful autoislet cell transplants in patients who have severe CP were performed recently by surgeons at the University of Arizona Medical Center. This procedure avoids surgically caused diabetes.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Tropical calcific pancreatitis (TCP) is an idiopathic pancreatitis prevalent in Asia; trypsin inhibitor (SPINK1) N34S variant partially explains the genetic susceptibility (Sundaresan et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Diet history
- SGA evaluation
- Alcohol intake
- Smoking history
- Left upper quadrant abdominal pain
- Vomiting
- Anorexia, nausea
- Steatorrhea
- Temperature (fever?)
- I & O
- Chvostek's sign
- CT scan or MRI
- Endoscopic ultrasound
- Exploratory laparotomy
- Endoscopic retrograde cholangiopancreatography (ERCP)
- See Table 8-3 also

Lab Work

- Bicarbonate levels (decreased; sensitivity 95)
- Secretin stimulation test
- IgG4 level, ESR, rheumatoid factor, ANA
- Thiobarbituric acid-reactive substances [TBARS] for oxidative stress
- Ferric-reducing ability of plasma [FRAP] for antioxidant status
- Fecal elastase (<200 μg/g)
- Sudan staining of feces
- Fecal fat test (24 hours on 100 g fat diet)
- CRP and WBC (elevated)
- K^+, Na^+, Ca^{++} (decreased)
- Gluc (often increased)
- Lipase and Amylase (elevated?)
- Serum trypsinogen (low)
- Chol (LDL up, or total decreased)
- Trig (increased)
- Mg^{++} (decreased)
- LDH (>700)
- AST (>250)
- WBC (>200)
- Bilirubin and Alk phos (increased)
- pCO_2 (increased) and pO_2 (decreased)
- Alb, transthyretin
- BUN
- H & H
- Serum folate

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Bioactive Substance Intake

Assessment Data: Dietary intake records, Dx of CP with frequent abdominal pain.

Nutrition Diagnosis (PES): Inadequate bioactive substance intake related to low intake of antioxidant-rich foods and beverages as evidenced by frequent bouts of abdominal pain with CP.

Intervention: Food and Nutrient Delivery—encourage intake of foods rich in antixoidants (see Tables 8-13 and 8-14).

Monitoring and Evaluation: Track food intake (food diary or history) to assess increased intake of foods rich in antioxidants and fewer episodes of abdominal pain, hospitalization, and use of analgesics.

TABLE 8-14 Oxygen Radical Absorbance Capacity (ORAC) Rating of Foods[a]

	USDA Data on Foods with High ORAC Scores				
Food	100 g Serving	Household Equivalent	Antioxidant capacity (TE) per 100 g	Other Common Unit	TE per Unit
Cinnamon, ground	100 g	14 Tbsp	264,543	1 Tbsp	19,110
Turmeric, ground	100 g	14 Tbsp	159,277	1 Tbsp	11,377
Sorghum, bran, black	100 g	1/3 cup	100,800	—	—
Cumin seed	100 g	14 Tbsp	76,800	1 Tbsp	5486
Parsley, dried flakes	100 g	4.7 cups	74,349	1/4 cup	3955
Basil, dried	100 g	4.7 cups	67,553	1/4 cup	3594
Curry powder	100 g	14 Tbsp	48,504	1 Tbsp	3465
Dutched chocolate powder	100 g	1 cup	40,200	1/4 cup	10,050
Sage, fresh	100 g	4.7 cups	32,004	1/4 cup	1702
Cloves, ground	100 g	14 Tbsp	31,446	1 Tbsp	2246
Mustard seed	100 g	8.75 Tbsp	29,257	1 Tbsp	3344
Pepper, black	100 g	14 Tsbp	27,618	1 Tbsp	1950
Marjoram	100 g	4.7 cups	27,297	1/4 cup	1452
Rice, brown	100 g	1/2 cup	24,287	—	—
Chili powder	100 g	14 Tbsp	23,636	1 Tbsp	1688
Dark chocolate candy	100 g	3.5 oz	21,800	1 oz	6229
Oregano powder, dried	100 g	4.7 cups	20,019	1/4 cup	1065
Semi-sweet chocolate morsels	100 g	1/2 cup	18,053	1/4 cup	9027
Pecans	100 g	7/8 cup	17,940	1/4 cup	5119
Paprika	100 g	14 Tbsp	17,919	1 Tbsp	1280
Tarragon, fresh	100 g	4.7 cups	15,542	1/4 cup	827
Ginger root, raw	100 g	1 cup	14,840	1/4 cup	3710
Elderberries, raw	100 g	1 cup	14,697	1/2 cup	7349
Peppermint, fresh	100 g	4.7 cups	13,978	1/4 cup	744
Oregano, fresh	100 g	4.7 cups	13,970	1/4 cup	743
Small Red Beans, dried	100 g	1/2 cup	13,727	—	—
Walnuts, English	100 g	7/8 cup	13,541	1/2 cup	3869
Wild blueberry	100 g	1 cup	13,427	1/2 cup	6713
Red kidney bean, dried	100 g	1/2 cup	13,259	—	—
Pinto beans	100 g	1/2 cup	11,864	—	—
Hazelnuts or filberts	100 g	3/4 cup	9645	—	—
Pears, dried	100 g	1/2 cup	9496	1/4 cup	4748
Blueberry, cultivated	100 g	1 cup	9019	1/2 cup	4510
Cranberry, whole	100 g	1 cup	8983	1/2 cup	4492
Pistachios	100 g	4 oz	7983	1 oz	1995
Artichoke hearts, cooked	100 g	1 cup	7904	1/2 cup	2635
Plums, black	100 g	1⅓ medium	7581	—	—
Prunes	100 g	1/2 cup	7291	—	—
Lentils, raw	100 g	1/2 cup	7282	—	—
Milk chocolate candy	100 g	3.5 oz	7203	1 oz	2058
Dried apples (to 40% moisture)	100 g	1/2 cup	6681	—	—
Garlic powder	100 g	14 Tbsp	6665	1 Tbsp	476
Chocolate syrup	100 g	1/3 cup	6330	—	—
Baby food, fruit, peaches	100 g	3.5 oz	6257	—	—
Raspberries	100 g	2/3 cup	6058	—	—

(continued)

TABLE 8-14 Oxygen Radical Absorbance Capacity (ORAC) Rating of Foods[a] *(continued)*

	USDA Data on Foods with High ORAC Scores				
Food	**100 g Serving**	**Household Equivalent**	**Antioxidant capacity (TE) per 100 g**	**Other Common Unit**	**TE per Unit**
Strawberries	100 g	1 cup	5938	1/2 cup	2969
Red Delicious apple	100 g	1 apple	5900	—	—
Blackberry, cultivated	100 g	3/4 cup	5775	—	—
Soybean seeds, raw, mature	100 g	1/2 cup	5764	—	—
Onion powder	100 g	14 Tbsp	5735	1 Tbsp	410
Granny Smith apple	100 g	1 apple	5381	—	—
Garlic, raw	100 g	21 cloves	5436	1 clove	259
Cilantro (coriander leaves), raw	100 g	4.7 cups	5141		
Sweet cherries	100 g	10 cherries	4873	—	—
Baby food, fruit, apple and blueberry, Junior	100 g	3.5 oz	4822	—	—
Black plum	100 g	1⅓ medium	4844	—	—
Russet potato	100 g	4 oz	4649	—	—
Almonds	100 g	3/4 cup	4454	1/4 cup	1485
Dill weed, fresh	100 g	4.7 cups	4392	1/2 cup	234
Black-eyed peas, cowpeas	100 g	1/2 cup	4343	—	—
Peaches, dried (40% moisture)	100 g	1/2 cup	4222	—	—
Black beans, dried	100 g	1/2 cup	4181	—	—
Plum	100 g	1⅓ medium	4118	—	—
Gala apple	100 g	1 small	3903	—	—
Red table wine	100 g	3.5 oz	3873	—	—
Peanut butter, smooth, with salt	100 g	1/2 cup	3432	2 Tbsp	858
Currants, red, raw	100 g	2/3 cup	3387	1/3 cup	1694
Figs, dry	100 g	1/2 cup	3383	—	—
Peanuts, all types, raw	100 g	2/3 cup	3166	1/3 cup	1583
Raisins, seedless	100 g	2/3 cup	3037	1/3 cup	1519
Pear, raw	100 g	2/3 medium	2941	—	—
Blueberry juice	100 g	3.5 oz	2906	—	—
Lettuce, red leaf, raw	100 g	1¾ cup	2380	—	—
Concord grape juice	100 g	3.5 oz	2377	—	—
Cornflakes cereal, crumbs	100 g	1 cup	2359	1/2 cup	1180
Pomegranate juice	100 g	3.5 oz	2341	—	—
Oats, quick, plain, dry	100 g	1¼ cup	2308	—	—
Ready to eat cereal, granola with raisins	100 g	3.5 oz	2294	1 oz	655
Red cabbage, raw	100 g	7/8 cup	2252	1/2 cup	643
Ready to eat cereal, toasted oatmeal squares	100 g	3.5 oz	2143	1 oz	612
Sweet potato, baked in skin	100 g	2/3 medium	2115	—	—
Chives, raw	100 g	4.7 cups	2094	1/2 cup	111
Prune juice, canned	100 g	3.5 oz	2036	—	—
Cashew nuts	100 g	4 oz	1948	1 oz	487
Orange, raw, navel	100 g	1 average	1819	—	—
Red grape juice	100 g	3.5 oz	1788	—	—
Radishes, raw	100 g	11 radishes	1736	3 radishes	473
Macdamia nuts	100 g	3/4 cup	1695	1/2 cup	565

(continued)

TABLE 8-14 Oxygen Radical Absorbance Capacity (ORAC) Rating of Foods[a] *(continued)*

	USDA Data on Foods with High ORAC Scores				
Food	**100 g Serving**	**Household Equivalent**	**Antioxidant capacity (TE) per 100 g**	**Other Common Unit**	**TE per Unit**
Tangerines or mandarin oranges	100 g	1/3 cup	1620	—	—
Spinach, frozen	100 g	3/4 cup cooked	1515	—	—
Onions, red, raw	100 g	2/3 cup	1521	1/3 cup	761
Cranberry-grape juice	100 g	3.5 oz	1480	—	—
Butterhead lettuce, raw	100 g	1¾ cup	1423	—	—
Chocolate milk, fluid, commercial, low fat	100 g	3.5 oz	1263	8 oz	2887
Grapes, red, raw	100 g	2/3 cup	1260	—	—
Tea, brewed green	100 g	3.5 oz	1253	—	—
Lemon juice, raw	100 g	3.5 oz	1225	1 oz	350
Onions, yellow, raw	100 g	2/3 cup	1220	1/3 cup	610
Olive oil, extra virgin	100 g	7 Tbsp	1150	1 Tbsp	164
Onions, raw	100 g	2/3 cup	1034	1/3 cup	517
Sweet pepper, orange	100 g	2/3 cup	984	1/3 cup	492
Mangos, raw	100 g	1 cup	982	1/2 cup	491
Sweet pepper, yellow	100 g	2/3 cup	964	1/3 cup	482
Romaine lettuce raw	100 g	1¾ cup	963	—	—
Eggplant, raw	100 g	7/8 cup	933	1/2 cup	266
Sweet pepper, green	100 g	2/3 cup	923	1/3 cup	462
Kiwi fruit, raw	100 g	1 kiwi	882	1/2 kiwi	441
Cranberry juice blend, red	100 g	3.5 oz	865	—	—
Lime juice, raw	100 g	3.5 oz	823	1 oz	235
White table wine	100 g	3.5 oz	392	—	—

[a]Oxygen radical absorbance capacity (ORAC) is a method of measuring antioxidant capacities in biological samples. Values are expressed as the sum of the lipid soluble (e.g., carotenoid) and water-soluble (e.g., phenolic) antioxidant fractions (i.e., "total ORAC") reported as in micromoles Trolox equivalents (TE) per 100 g sample. Foods rich in antioxidants have an ORAC rating of 1000 per 100 g.

For household measurements:

- 100 g = 3.5 oz dry ingredients
- 3 tsp = 1 Tbsp.
- 48 tsp = 16 Tbsp = 1 cup.
- 1 Tbsp fresh herbs = ½ teaspoon, dried, crushed
- 1 oz = 4½ Tablespoons allspice, cinnamon, curry, paprika or dry mustard
  or 4 Tablespoons cloves or prepared mustard
  or 3½ Tablespoons nutmeg or pepper
  or 3 Tablespoons sage, cream of tartar or cornstarch
  or 2 Tablespoons salt or any liquid
- 1 pound = 2 cups liquid
  or 4 cups flour
  or 10 eggs without shells
  or 4 cups grated cabbage, cranberries, coffee or chopped celery
  or 3 cups corn meal
  or 2 cups uncooked rice
  or 2¾ cups raisins or dried currants

Sources:

Nutrient Data Laboratory, Agriculture Research Service, U.S. Department of Agriculture, Oxygen radical absorbance capacity (ORAC) of Selected Foods—2007. Accessed September 15, 2009, at http://www.ars.usda.gov/SP2UserFiles/Place/12354500/Data/ORAC/ORAC07.pdf.
Berry Health Fact Sheets. Accessed September 22, 2009, at http://berryhealth.fst.oregonstate.edu/health_healing/fact_sheets/index.htm.
Natural antioxidants. Accessed September 15, 2009, at http://naturalantioxidants.org/.

INTERVENTION

OBJECTIVES

- Decrease pain from oxidative stress. Abstain from alcohol and smoking.
- Correct fluid and electrolyte imbalances and malnutrition; avoid overfeeding. Acid–base imbalance is common with nasogastric suctioning, fistula losses, renal failure, nausea, and vomiting.
- Provide optimal nutrition support for weight gain as weight loss is common in the late course of CP (Stanga et al, 2005).
- Alleviate steatorrhea; decrease number of stools per day if there is diarrhea.
- Avoid or control complications (cardiovascular, pulmonary, hematological, renal, neurological, or metabolic); prevent multiple organ dysfunction.
- With diabetes, it may be better to have glucose elevated slightly (200 mg/dL) than to allow prolonged hypoglycemia to occur.
- If tube fed, monitor for abdominal pain or discomfort, and offer pain medication as needed. Administer pancreatic enzymes with meals or TF (Dominguez-Munoz et al, 2005; Stanga et al, 2005). CPN may be needed for resistant cases where pain does not subside.

FOOD AND NUTRITION

- If tolerated, use a diet with low-to-moderate fat (0.7–1 g/kg) and high carbohydrates; calculate needs accordingly. Diet should be low in fiber with six small meals a day. Include adequate amounts of antioxidants (Tables 8-13 and 8-14), minerals (calcium, magnesium, selenium, and zinc), fat-soluble vitamins (A, D, E, and K) in water-miscible form, and water-soluble vitamins.
- High-energy, standard feeding by enteral pump is desirable for those who need it. Estimate needs at 35 kcals/kg, 1–1.5 g protein per kilogram. Jejunal feeding is more beneficial than gastric placement. Needle catheter jejunostomy may be used safely (Stanga et al, 2005), but not with ascites.
- Treat steatorrhea to minimize symptoms of the underlying disease and to promote weight retention or gain. MCTs and pancreatic replacement therapy combat maldigestion and malabsorption (Stanga et al, 2005). Monitor for hyperglycemia with a high-carbohydrate diet.
- When CPN is needed (in about 1% of cases), estimate needs according to similar parameters as for oral diet. The benefits of BCAAs and glutamine are not clear. If intravenous lipids are used, do not use more than 1.5 g/kg for adults. Provide no more than 5 mg/kg/min of glucose.
- Alcoholic beverages are absolutely prohibited.

Common Drugs Used and Potential Side Effects

- See Table 8-12 for guidance on medications.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Antioxidant therapy with green tea polyphenols and gene therapy with superoxide dismutase can markedly attenuate disease (Dryden et al, 2005).
- The combination of pre- and probiotics (synbiotics) have mixed results in this population. In the acute form of pancreatitis, probiotic use may actually increase mortality (Besselink et al, 2008).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient to watch for signs and symptoms of diabetes, tetany, peritonitis, acute respiratory distress syndrome, and pleural effusion.
- Discuss omission of alcohol from the typical diet.
- Discuss tips for handling nausea and vomiting (e.g., dry meals, taking liquids a few hours before or after meals, use of ice chips, sipping beverages, asking physician about available antiemetics, etc.). Dietary counseling has been found to be as effective as dietary supplementation for managing CP (Singh et al, 2008). Gas-forming foods may need to be omitted. Teach high-calorie, high-protein, low-fat diet rich in antioxidant foods with a frequent small meal pattern.
- To prevent onset of pancreatic cancer, avoiding tobacco smoking.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Medline Plus
  http://www.nlm.nih.gov/medlineplus/ency/article/000221.htm
- Merck Manual—Chronic Pancreatitis
  http://www.merck.com/mmpe/sec02/ch015/ch015c.html#sec02-ch015-ch015c-901

PANCREATITIS, CHRONIC—CITED REFERENCES

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Verlaan M, et al. Assessment of oxidative stress in chronic pancreatitis patients. *World J Gastroenterol.* 12:5705, 2006.
Yaday D, et al. Alcohol consumption, cigarette smoking, and the risk of recurrent acute and chronic pancreatitis. *Arch Int Med.* 169:1035, 2009.

PANCREATIC INSUFFICIENCY

NUTRITIONAL ACUITY RANKING: LEVEL 2–3

DEFINITIONS AND BACKGROUND

Pancreatic insufficiency is caused by the inability of the exocrine pancreas to secrete digestive enzymes such as lipase. Cystic fibrosis (CF), type 1 (autoimmune) diabetes, Shwachman-Diamond Syndrome (SDS), pancreatic cancer, or pancreatitis will often lead to pancreatic insufficiency. Other disorders of the gastrointestinal tract, such as celiac disease, inflammatory bowel disease, Zollinger–Ellison syndrome or gastric resection can either mimic or cause pancreatic exocrine Insufficiency (Keller et al, 2009).

Lipase is the key enzyme for breaking down triglycerides. Patients often have mild-to-moderate fat malabsorption. In CF, there may be recurrent problems with fatty acid abnormalities or pancreatitis (DeBoeck et al, 2005). Fecal elastase (FE) 1 levels indicate lipase activity; assessment should be used to verify pancreatic status (Cohen et al, 2005). A fecal fat or Steatocrit test may identify the problem.

Stool trypsin tests or low levels of trypsinogen are used to determine whether sufficient amounts of this pancreatic enzyme are reaching the intestines to metabolize proteins. Because the overt clinical symptoms of pancreatic exocrine insufficiency are steatorrhea and maldigestion (Keller et al, 2009), oral pancreatic enzyme supplements should be properly administered to ensure adequate gastric mixing with the food.

Nonendocrine pancreatic disease is a critical factor for development of diabetes; this "type 3c" affects over 8% of the general diabetic patient population (Hardt et al, 2008). There is a continuous interstitial matrix connection between the endocrine and exocrine pancreas; fibrosis and inflammation cause dysfunctional insulino-acinar-ductal-incretin gut hormone axis, resulting in pancreatic insufficiency and glucagon-like peptide deficiency and diabetes (Hayden et al, 2008).

The delivery of sufficient enzyme concentrations into the duodenal lumen simultaneously with meals can reduce nutrient malabsorption, improve the symptoms of steatorrhea, and in some cases alleviate pain (Ferrone et al, 2007). The best duodenal pH permits optimal efficacy of these extracts and the buffered, enteric-coated enzymes are most useful (Brady et al, 2006; Pezzilli, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Diagnosis of chronic pancreatic insufficiency is difficult in early stages; eventually, genetic testing may prove to be helpful. Hereditary pancreatitis involves a cationic trypsingen gene (PRSS1) gene, where treatment involves attempting to inactivate that gene before it causes pancreatic insufficiency. CF patients will have the CFTR gene that is associated with pancreatic insufficiency.

Clinical/History

- Height
- Weight
- BMI
- Weight loss?
- Malabsorption
- Steatorrhea
- Diet history
- I & O
- Pale, bulky stools
- Abdominal bloating
- Stool weight
- CT scan or MRI
- ERCP

Lab Work

- Amylase (increased)
- Lipase
- Trypsinogen
- CRP
- Serum carotene
- FE 1
- Ca^{++}, Mg^{++}
- Trig, Chol
- PT or INR
- Bicarbonate
- Gluc
- Na^{+}, K^{+}
- Alk phos
- H & H

INTERVENTION

OBJECTIVES

- Maintain or achieve BMI >19.
- Correct fatty acid abnormalities, maldigestion, diarrhea, and steatorrhea. Prevent EFA deficiency.
- Provide adequate energy intake while lowering intake of fats.
- Provide fat-soluble vitamins A-D-E-K and zinc with malabsorption. Prevent overload of iron.
- Attenuate intestinal inflammation (Pezzilli, 2009). Prevent or manage type 2 diabetes.

SAMPLE NUTRITION CARE PROCESS STEPS

Malnutrition

Assessment Data: Dietary intake records; weight loss; steatorrhea; pale, bulky, foul stools.

Nutrition Diagnosis (PES): Malnutrition related to inability to digest nutrients as evidenced by steatorrhea and unintentional weight loss.

Intervention: Food and Nutrient Delivery—provide high kilocalories, high protein foods with low fat content. Include adequate pancreatic enzymes with every meal. Educate about the role of the pancreas in nutrient absorption. Counsel about ways to enjoy meals that are low fat while nutrient dense. Discuss the need for water-miscible forms of vitamins A, D E, and K if needed.

Monitoring and Evaluation: Track food intake (food diary or history). Evaluate for signs of malnutrition. Review lab test results. Monitor for improvement in weight, decrease in steatorrhea and pale, bulky stools.

FOOD AND NUTRITION

- Encourage high-calorie, high-protein intake to maintain body weight. Dose pancreatic enzyme replacement adequately to minimize fat malabsorption.
- Use MCTs since they do not require lipase. They may be taken with simple sugars, jelly, jams and in mixed dishes.
- Increase use of omega-3 fatty acids from tuna, mackerel, salmon, and other fatty fish, as well as flaxseed. Probiotics may be considered.
- If diabetes is present, emphasize regular mealtimes and carbohydrate spacing to prevent hyperglycemia.
- Use tender meats and low-fiber fruits and vegetables.
- Alcoholic beverages are prohibited.
- Tube feed in severe cases of malnutrition. Night feeding may be a reasonable option.

Common Drugs Used and Potential Side Effects

- Pancreatic extract preparations contain pancreatin and pancrelipase; both contain principally amylase, protease, and lipase. Pancreatic enzymes (Cotazym, Creon) should be taken with food. Take enteric-coated tablets with cimetidine or antacids. Administration of lipase 25,000–40,000 units/meal is given by using pH-sensitive pancrelipase microspheres, along with dosage increases and compliance checks (Ferrone et al, 2007).
- Fat-soluble vitamins should be taken in water-miscible form, with the pancreatic enzymes.

Herbs, Botanicals, and Supplements

- New approaches may include the use of probiotics to reduce inflammation (Pezzilli, 2009).
- Lipase enzymes derived from microbial species show promise; plant-based enzymes, such as bromelain from pineapple, also serve as effective digestive aids in the breakdown of proteins (Roxas, 2008).
- Herbs and botanical supplements should not be used without discussing with physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient in the role of the pancreas in digestion.
- Discuss how pancreatic enzymes should be taken with meals or afterward for best results.
- Discuss appropriate measures for recovery and control.
- Share menu planning tips for a high protein, low fat diet and how to include desired nutrients.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Cystic Fibrosis Foundation
  http://www.cff.org/
- Food and Drug Administration (FDA)
  http://www.fda.gov/cder/drug/infopage/pancreatic_drugs/default.htm
- Lab tests on line
  http://www.labtestsonline.org/understanding/conditions/pancreatic_insuf.html
- National Pancreas Foundation
  http://www.pancreasfoundation.org/
- Pancreas
  http://www.pancreas.org/
- Recipes, Low fat
  http://www.pancreasfoundation.org/live/recipes.shtml

PANCREATIC INSUFFICIENCY—CITED REFERENCES

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Keller J, et al. Tests of pancreatic exocrine function—clinical significance in pancreatic and non-pancreatic disorders. *Best Pract Res Clin Gastroenterol.* 23:r425, 2009.

Pezzilli R. Chronic pancreatitis: maldigestion, intestinal ecology and intestinal inflammation. *World J Gastroenterol.* 15:1673, 2009.

Roxas M. The role of enzyme supplementation in digestive disorders. *Altern Med Rev.* 13:307, 2008.

PANCREATIC TRANSPLANTATION

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

Pancreatic islet cell transplantation is a treatment alternative for patients with type 1 diabetes who experience hypoglycemic unawareness despite maximal care (Onaca et al, 2007). Islet transplant can restore pancreatic endocrine function, endogenous insulin secretion, and insulin independence in more than 80% of patients, and recover the metabolism of glucose, protein, and lipids (Bertuzzi et al, 2006). It helps to decrease nephropathy, early or mild retinopathy, and neuropathy.

Pancreas transplantation alone (PTA) involves transplanting a pancreas from a cadaver to a patient whose kidneys have not been damaged by diabetes. Simultaneous pancreas and kidney (SPK) transplantation involves kidney and pancreas being transplanted at the same time from a cadaver, often leading to freedom from both dialysis and insulin dependency. Pancreas after kidney (PAK) surgery is another option, as is islet cell or isolated beta-cell transplantation. Pancreatic islet cells are highly sensitive to hypoxia, which contributes to poor islet yield, inflammatory events, and cellular death during the early posttransplantation period (Lau et al, 2009). Pancreatic transplantation is major surgery, with risk of bleeding, infection, and reactions to anesthesia. Antirejection medicines, which have many side effects, have to be taken for a long time.

Whole pancreas transplantation is associated with a significant risk of surgical and postoperative complications (Meloche, 2007). Islet cell transplantation has fewer side effects but does not yield an ideal level of glucose control. Persistent graft function results in improved glucose control and avoidance of hypoglycemic events (Onaca et al, 2007). Improved control of glycated HbA1c, reduced risk of recurrent hypoglycemia and of diabetic complications are benefits of islet cell transplantation, irrespective of the status of insulin independence (Bertuzzi et al, 2006).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Pancreatic transplantation is needed for type 1 diabetes, which has a genetic origin (American Diabetes Association, 2009).

Clinical/History	I & O	H & H
Height	BP	Serum Fe
Weight, dry	Temperature	Serum amylase
BMI		Urinary amylase (only if bladder drained)
Diet history	**Lab Work**	
Weight changes and goals	Gluc	Na^+, K^+
	BUN, Creat	Ca^{++}, Mg^{++}

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Oral Food and Beverage Intake

Assessment Data: Dietary intake records pretransplantation indicate poor oral intake of both food and beverages; status posttransplant, 21 days on enteral nutrition; wishes to try some liquids and soft foods.

Nutrition Diagnosis (PES): Inadequate oral food and beverage related to inability to be fed orally posttransplant as evidenced by 14 days on EN and now asking to attempt oral intake.

Intervention: Food and Nutrient Delivery—offer one to two small items at mealtimes or every few hours while awake; gradually increase food intake as tolerated. Educate about the role of nutrition in liver health and recovery; discuss ways to progress orally and wean from enteral nutrition.

Monitoring and Evaluation: Improved oral appetite. No nausea or unplanned side effects after surgery Gradual return of usual weight and tolerance of oral intake closer to desired level.

INTERVENTION

OBJECTIVES

- **Preoperatively:** Meet nutritional needs; improve visceral protein stores; maintain lean tissue. Nutritional management of candidates requires management of renal function, and treatment of obesity in advance as a BMI >27 kg/m^2 may delay wound healing.
- **Postoperatively:** Support graft survival. Promote wound healing. Improve or maintain nutritional status.
- **Long Term:** Weaned off CPN within the first year to achieve optimal nutritional status. Prevent weight gain. Prevent complications such as gastroparesis, hypertension, hyperlipidemia, hyperglycemia, and osteoporosis. Attain near-normal blood glucose control and normal hemoglobin A1c levels without risks of severe hypoglycemia (Meloche, 2007).

FOOD AND NUTRITION

- **Preoperatively:** Meet nutritional needs; improve visceral protein stores; maintain lean tissue.
- **Postoperatively:** Control carbohydrates if there is diabetes or hyperglycemia. Manage diet carefully to prevent hypoglycemia in patients who take insulin.

Common Drugs Used and Potential Side Effects

- See Table 8-15.

TABLE 8-15 Medications Used after Pancreatic Transplantation

Medication	Description
Analgesics	Analgesics are used to reduce pain. Long-term use may affect such nutrients as vitamin C and folacin; monitor carefully for each specific medication.
Azathioprine (Imuran)	Azathioprine may cause leukopenia, thrombocytopenia, oral and esophageal sores, macrocytic anemia, pancreatitis, vomiting, diarrhea, and other complex side effects. Folate supplementation and other dietary modifications (liquid or soft diet, use of oral supplements) may be needed. The drug works by lowering the number of T cells; it is often prescribed along with prednisone for conventional immunosuppression.
Corticosteroids (prednisone or Solu-Cortef)	Corticosteroids such as prednisone and Solu-Cortef are used for immunosuppression. Side effects include increased catabolism of proteins, negative nitrogen balance, hyperphagia, ulcers, decreased glucose tolerance, sodium retention, fluid retention, and impaired calcium absorption and osteoporosis. Cushing's syndrome, obesity, muscle wasting, and increased gastric secretion may result. A higher protein intake and lower intake of carbohydrate and sodium may be needed.
Cyclosporine	Cyclosporine does not retain sodium as much as corticosteroids do. Intravenous doses are more effective than oral doses. Nausea, vomiting, and diarrhea are common side effects. Hyperlipidemia, hyperglycemia, and hyperkalemia may also occur; decrease fat intake as well as sodium and potassium if necessary. Magnesium may need to be replaced. The drug is also nephrotoxic; a controlled renal diet may be beneficial. Taking omega-3 fatty acids during cyclosporine therapy may reduce toxic side effects (such as high blood pressure and kidney damage) associated with this medication in transplantation patients (Tsipas and Morphake, 2003). Avoid use with St. John's wort.
Diuretics	Diuretics such as furosemide (Lasix) may cause hypokalemia. Aldactone actually spares potassium; monitor drug changes closely. In general, avoid use with fenugreek, yohimbe, and ginkgo.
Immunosuppressants	Immunosuppressants such as muromonab (Orthoclone OKT3) and antithymocyte globulin (ATG) are less nephrotoxic than cyclosporine but can cause nausea, anorexia, diarrhea, and vomiting. Monitor carefully. Fever and stomatitis also may occur; alter diet as needed.
Insulin	Insulin may be necessary during periods of hyperglycemia. Monitor for hypoglycemic symptoms during use; teach patient self-management tips.
Pancreatic Enzymes	Pancreatic enzymes may be needed if pancreatitis occurs again after transplantation.
Tacrolimus (Prograf, FK506)	Tacrolimus suppresses T-cell immunity; it is 100 times more potent than cyclosporine, thus requiring smaller doses. Side effects include GI distress, nausea, vomiting, hyperkalemia, and hyperglycemia; adjust diet accordingly by controlling carbohydrate and enhancing potassium intake.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- St. John's wort interferes with the metabolism of cyclosporine and should not be used after transplantation.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage activity to prevent excessive weight gain; 14–30 lb may be a common gain.
- Discuss surgical stress. Encourage positive protein balance to promote anabolism and wound healing.
- Over the long term, follow a low-cholesterol and low–saturated fatty acid dietary plan.
- Sudden abdominal pain, fever, and increased amylase and glucose can occur and are signs of pancreatitis even after transplantation. Report these warning signs immediately to the physician.
- Problems after transplantation include diabetic complications, bone loss, and failure of the pancreas graft. A multidisciplinary team is required to maximize long-term quality of life.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- E-medicine
  http://emedicine.medscape.com/article/429408-overview
- Insulin
  http://www.insulin-free.org/
- Mayo Clinic—Pancreatic Transplant
  http://www.mayoclinic.com/health/pancreas-transplant/DA00047
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
  http://diabetes.niddk.nih.gov/dm/pubs/pancreaticislet/
- NIH—Genetics of Diabetes
  http://www.ncbi.nlm.nih.gov/bookshelf/br.fcgi?book=diabetes&part=A987
- University of Minnesota—Diabetes Institute
  http://www.diabetes.umn.edu/diabinst/video/index.html
- USC Pancreatic Transplant Program
  http://www.pancreastransplant.org/

PANCREATIC TRANSPLANTATION—CITED REFERENCES

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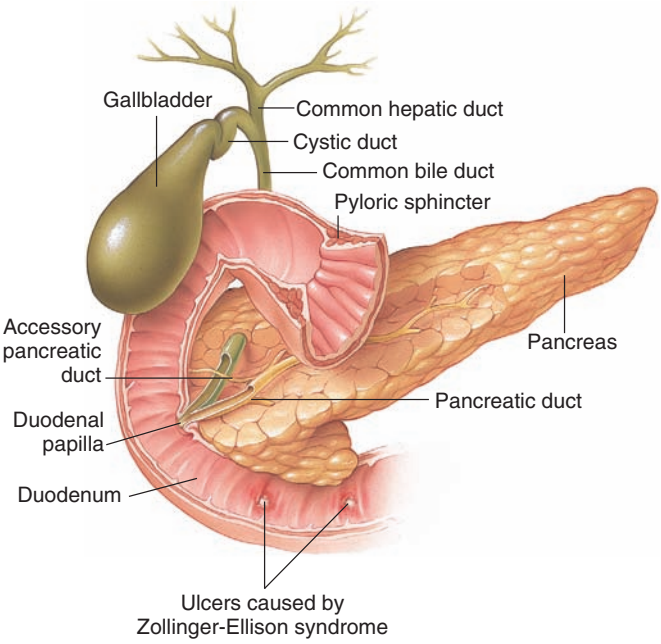
Onaca N, et al. Pancreatic islet cell transplantation: update and new developments. *Nutr Clin Pract.* 22:485, 2007.

ZOLLINGER–ELLISON SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 3



Asset provided by Anatomical Chart Co.

DEFINITIONS AND BACKGROUND

Zollinger–Ellison syndrome (ZES) is a severe disease with ulceratogenic tumor (gastrinoma) of the delta-cells of the pancreatic islets of Langerhans; the cells produce gastrin. Almost every patient with ZES has marked gastric acid hypersecretion and ulcerations in the esophagus, stomach, duodenum, and jejunum. It is more common among 30- to 50-year-old men. ZES occurs in less than 1% of all patients with duodenal ulcers.

Gastrinomas producing ZES are the most frequent symptomatic, malignant pancreatic endocrine tumors (Gibril and Jensen, 2005). They frequently are accompanied by secretory diarrhea. Interestingly, insulin production is often increased in the beta-cells. Of all cases, 60% occur in males; two thirds of cases are malignant. Widespread metastasis indicates a poor prognosis.

Gastric carcinoid tumors in patients with longstanding ZES may be symptomatic and aggressive and may metastasize to the liver; they require long-term medical treatment. Curing gastrinoma or appropriately inhibiting gastric acid hypersecretion in ZES patients prevents death and favors long-term survival (Quatrini et al, 2005).

Resection of localized gastrinomas often does not require extended surgical resection and is associated with excellent long-term outcomes (Mortellaro et al, 2009). Total gastrectomy is reserved for patients with extensive tumor involvement of the gastric wall or for patients with emergency bleeding.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The disease is more common in people who have an inherited condition called multiple endocrine neoplasia type 1 (MEN1). Evaluate for family history of hyperparathyroidism, nephrolithiasis, or gastrinoma.

Clinical/History		Lab Work
Height	Heartburn	N balance
Weight	Diarrhea or malabsorption	Na^+
BMI	BP	Ca^{++} (usually increased)
Weight loss?	Negative *H pylori* testing	Alb
Diet history	Upper GI endoscopy	H & H
I & O	Somatostatin receptor scintigraphy (SRS)	K^+ (decreased)
Burning abdominal pain	Gastrin radioimmunoassay	Mg^{++}
Nausea, vomiting	CT Scan	BUN
Severe esophageal reflux		Serum insulin
		Serum gastrin
		Trig, Chol
		Gluc

INTERVENTION

OBJECTIVES

- Relieve stomach acid to lessen ulcer symptoms, usually with medications. Surgery may be needed to remove tumors.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function

Assessment Data: Dietary intake records, diarrhea, steatorrhea, weight loss, nausea and vomiting.

Nutrition Diagnosis (PES): Abnormal GI function related to excessive gastrin production as evidenced by gastrinoma with weight loss, nausea & vomiting, diarrhea and steatorrhea.

Intervention: Food and Nutrient Delivery—provide easily tolerated foods, low acid.

Monitoring and Evaluation: Track food intake (food diary or history).

- Overcome malabsorption and lessen diarrhea.
- Decrease problems with dysphagia and reflux.
- Prevent complications such as esophageal stricture or abdominal perforation.
- Manage or reduce cancer, which can occur in 25% of cases.

FOOD AND NUTRITION

- Modify fiber, seasonings, and textures as necessary. Decrease intake of acidic foods if not tolerated (such as citrus juices, tomato products).
- Alter feeding modality to TF such as jejunostomy placement as needed.

Common Drugs Used and Potential Side Effects

- PPIs are used for the treatment of acid-related disorders. Use of these medications can prevent the need for surgery (Hirschowitz et al, 2005). Take omeprazole (Prilosec), esomeprazole (Nexium), lansoprazole (Prevacid), or pantoprazole (Protonix) before meals. Iron and vitamin B_{12} levels may become depleted. Rabeprazole (Acifex) has been tested and has fewer side effects than some other PPIs (Baldwin and Keam, 2009).
- Chemotherapy may be needed if surgery to remove the tumors is not feasible. Streptozotocin (Zanosar), 5-fluorouracil (Adrucil) or doxorubicin (Doxil) may be used. Monitor for side effects.
- Octreotide or interferon may also be used.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain which modifications of fiber in the diet are appropriate.
- Explain how malabsorption compromises nutritional status. Discuss limiting fat intake, as appropriate for the patient.
- Lower acid foods may be better tolerated; discuss ways to adjust recipes or good choices.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- **NIDDK**
  http://digestive.niddk.nih.gov/ddiseases/pubs/zollinger/

ZOLLINGER–ELLISON SYNDROME—CITED REFERENCES

Baldwin CM, Keam SJ. Rabeprazole: a review of its use in the management of gastric acid-related diseases in adults. *Drugs.* 69:1373, 2009.

Gibril F, Jensen JT. Advances in evaluation and management of gastrinoma in patients with Zollinger-Ellison syndrome. *Curr Gastroenterol Rep.* 7:114, 2005.

Hirschowitz BI, et al. Clinical outcome using lansoprazole in acid hypersecretors with and without Zollinger-Ellison syndrome: a 13-year prospective study. *Clin Gastroenterol Hepatol.* 3:39, 2005.

Mortellaro VE, et al. Long-term results of a selective surgical approach to management of Zollinger-Ellison syndrome in patients with MEN-1. *Am Surg.* 75:730, 2009.

Quatrini M, et al. Follow-up study of patients with Zollinger-Ellison syndrome in the period 1966–2002: effects of surgical and medical treatments on long-term survival. *J Clin Gastroenterol.* 39:376, 2005.

BILIARY DISORDERS

BILIARY CIRRHOSIS

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Biliary cirrhosis, also called cholangiolitic hepatitis or obstructive jaundice, occurs in two to five cases per 100,000 worldwide. Symptoms include pruritus, jaundice, and portal hypertension. Biliary atresia is the result of an inflammatory process that affects the intrahepatic and extrahepatic bile ducts, leading to fibrosis and obliteration of the biliary tract with development of biliary cirrhosis in infants (Tang et al, 2005).

Primary biliary cirrhosis (PBC) is characterized by progressive destruction of intrahepatic bile ducts; it primarily affects middle-aged women. Autoimmune factors may trigger the disease in genetically susceptible hosts; genetic susceptibility is a predisposing factor. Environmental factors (infection, chemicals, smoking) may also be relevant. Environmental xenobioticsmay aggravate genetic weaknesses in mechanisms of immune regulation, and subsequent immunopathology (Gershwin and Mackay, 2008). PBC that is associated with other autoimmune diseases such as Sjogren's syndrome, scleroderma, Raynaud's phenomenon, or CREST syndrome is regarded as an organ-specific autoimmune disease. Osteoporosis is prevalent in this population (Guanabens et al, 2005) and celiac disease is also commonly found (Duggan and Duggan, 2005).

Homocysteine has been proposed to be involved (Ebrahimkhani et al, 2005). In symptomatic patients, advanced age, elevated serum bilirubin levels, and decreased serum albumin levels lead to shortened survival. PBC slowly progresses and may lead to liver failure where transplantation is the only effective therapy.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: There is a role for HLA to determine PBC susceptibility (Invernizzi et al, 2008). Vitamin D receptor polymorphisms are also being studied.

Clinical/History	Lab Work	
Height	Antimitochondrial antibodies (AMA)	Transferrin
Weight		Tissue transglutaminase (for celiac disease)
BMI	Bilirubin (increased)	Globulin
Diet history	Alk phos (increased)	Gluc
Jaundice	ALT/AST	Ca^{++}, Mg^{++}
Portal hypertension?	Alb, transthyretin	Na^{+}, K^{+}
Pruritus	PT (decreased)	Chol (increased)
Xanthomas	Serum homocysteine	Ceruloplasmin
Cholangiography		
Liver biopsy		

INTERVENTION

OBJECTIVES

- Correct diarrhea, steatorrhea, malnutrition, and osteomalacia. Prevention of bone density loss is important, as vitamin D deficiency is common.
- Limit or control symptoms. Prevent progression to end-stage liver disease when possible.
- Prevent or correct zinc and vitamin deficiencies.

SAMPLE NUTRITION CARE PROCESS STEPS

Altered Nutrition-Related Labs

Assessment Data: Dx biliary cirrhosis with presence of AMA, elevated bilirubin and alk phos.

Nutrition Diagnosis (PES): Altered nutrition-related labs related to biliary cirrhosis with cholestasis as evidenced by AMA, elevated bilirubin and alk phos levels.

Intervention: Food and nutrient delivery—use antioxidant-rich foods, supplement with fat-soluble vitamins.

Monitoring and Evaluation: Track lab values.

- Manage related disorders (such as Sjögren's syndrome or celiac disease).

FOOD AND NUTRITION

- Increase vitamin D and calcium intake to protect against osteopenia. Vitamin K may also play an important role in protection of bone health and may be supplemented (Plaza and Lamson, 2005). Use water-miscible sources of vitamins A, D, E, and K with steatorrhea.
- Reduce cholesterol and saturated fats in hypercholesterolemia.
- Ensure adequate intake of zinc from diet.
- To lower elevated homocysteine levels, which seem to aggravate hepatic fibrogenesis, be sure the diet contains adequate amounts of folic acid and vitamins B_6 and B_{12} (Ebrahimkhani et al, 2005).
- If the patient also has celiac disease, omit gluten from the diet (e.g., wheat, rye, barley). Initiation of a gluten-free diet may help to resolve iron deficiency anemia, pruritus, and elevated serum liver biochemistries.
- Control carbohydrate intake with hyperglycemia.

Common Drugs Used and Potential Side Effects

- Prolonged administration of ursodeoxycholic acid (UDCA) in patients with PBC is associated with survival benefit and delay of LT. Ursodeoxycholic acid (UDCA) is the only currently known medication that can slow the disease progression. When UDCA is ineffective, cholestyramine is the treatment of choice; it decreases bile acids but can cause belching or constipation.
- Budesonide is a glucocorticoid that may help when given with UDCA to improve liver status (Rautiainen et al, 2005). Weight gain and increased appetite often result.
- Bezafibrate, a hypolipidemic drug, has been shown to benefit patients with PBC in some studies (Akbar et al, 2005).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- High doses of vitamin E (tocopherol) will elevate transaminases.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of bile salts in fat and fat-soluble vitamin absorption. If supplements are used, water-miscible forms may be needed.
- Protection of bone mineral density will be important. Discuss the role of medications, calcium, and vitamin D on bone health (Plaza and Lamson, 2005).

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Mayo Clinic—Primary Biliary Cirrhosis
  http://www.mayoclinic.com/health/primary-biliary-cirrhosis/DS00604
- Medicine Net
  http://www.medicinenet.com/primary_biliary_cirrhosis/article.htm

BILIARY CIRRHOSIS—CITED REFERENCES

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Gershwin ME, Mackay IR. The causes of primary biliary cirrhosis: convenient and inconvenient truths. *Hepatology.* 47:737, 2008.

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Plaza SM, Lamson DW. Vitamin K2 in bone metabolism and osteoporosis. *Altern Med Rev.* 10:24, 2005.

Rautiainen H, et al. Budesonide combined with UDCA to improve liver histology in primary biliary cirrhosis: a three-year randomized trial. *Hepatology.* 41:747, 2005.

Tang ST, et al. Diagnosis and treatment of biliary atresia: a retrospective study. *Hepatobiliary Pancreat Dis Int.* 4:108, 2005.

CHOLESTASIS

NUTRITIONAL ACUITY RANKING: LEVEL 2–3

DEFINITIONS AND BACKGROUND

Cholestasis involves reduced bile flow in any liver disease with bilirubin over 2.0 mg/dL. Disturbance of the flow of bile leads to intracellular retention of biliary constituents. Hepatic causes of cholestasis include viral hepatitis, ALD, hemochromatosis, and autoimmune hepatitis. Biliary causes include primary sclerosing cholangitis (in which the intrahepatic and/or extrahepatic bile ducts undergo inflammation and fibrosis), choledocholithiasis, PBC, and biliary atresia. It can also occur with inflammatory bowel disease (Huang and Lichtenstein, 2005). Prolonged PN may be needed in the absence of GI tract stimulation.

Cholestasis interferes with excretion of the bile salts required for emulsification and absorption of dietary fat. Reduced bile secretion impairs micelle formation, which is needed for digestion of fat by pancreatic enzymes. Vitamin and mineral deficiencies and alterations are common, especially if cholestasis is significant. Zinc, magnesium, and calcium may be deficient because they are albumin-bound and the liver is not working properly. Deficiency of fat-soluble vitamins A, D, E, and K may occur. Of particular concern is vitamin E, which circulates in the blood almost exclusively attached to the lipoprotein fractions.

In chronic cholestasis with biliary obstruction, hyperlipidemia and accumulation of copper result, and manganese can accumulate in the brain; avoid overfeeding with copper or manganese. Hepatic copper overload in CPN patients occurs through chronic cholestasis in CPN-associated liver disease regardless of duration (Blaszyck et al, 2005).

Chronic CPN may induce fatty liver and inflammation, especially in patients with short bowel syndrome. Deficiency of choline in parenteral solutions has been proposed as the mechanism for liver disease. With CPN, cholestatic jaundice may occur from a lack of enteral nutrition and failure of biliary stimulation. In patients receiving home PN, prevalence of liver disease increases with duration.

Signs and symptoms of cholestasis can include glossitis from B-complex vitamin deficiency, protein and iron deficiency, hemorrhagic tendencies due to vitamin C or K inadequacy, and flatulence. Patients with steatorrhea may benefit from a low-fat diet or from use of MCTs. Intrahepatic cholestatic syndromes cause a decrease in bile flow with no overt bile duct obstruction; bile constituents accumulate in the liver and blood.

Ursodeoxycholic acid and adequate nutritional support are the usual treatments, with LT being performed in severe cases only (Huang and Lichtenstein, 2005). Depending on the cause (such as medication effects, postoperative jaundice, sepsis, CPN, or acalculous cholecystitis), treatment includes removal of offending drugs, supportive care, broad-spectrum antibiotic agents with drainage of infected fluids, CPN adjustment including cycling and limiting carbohydrates, and cholecystectomy.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Because ATP-binding cassette (ABC) transporters are important for normal bile secretion, hereditary and acquired ABC transporter defects play a central role in the pathogenesis of cholestasis (Trauner et al, 2007).

Clinical/History

Height
Weight
BMI
Diet history
Ascites
Edema
I & O
Jaundice
Pale stools
Fatty yellow deposits in skins
Eas bleeding
Small, spider-like blood vessels visible on skin
Nausea
Flatulence

Lab Work

Chol, Trig (increased)
PT (prolonged)
AST, ALT (increased)
Bilirubin (increased, >2 mg/dL)
Somatomedin C
Alk phos (increased)
Gamma-glutamyl transpeptidase (very high)
Serum carotene (increased or decreased)
Bun, Creat
H & H
Serum Fe
Alb, transthyretin
Globulin
Amylase, lipase
Serum manganese
Serum zinc
Na^+, K^+
Ca^{++}, Mg^{++}

INTERVENTION

OBJECTIVES

- Promote return of normal liver function and bile flow.
- Treat fat malabsorption and deficiency of any additional nutrients.
- Correct steatorrhea, GI bleeding, and copper overloading when present.
- Prevent or correct for liver failure, osteomalacia, or osteoporosis.
- Correct nutrient excesses (e.g., manganese).
- Prepare for surgery when indicated.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive CPN Infusion

Assessment Data: Intake records indicating CPN solution exceeding estimated kilocalories requirements.

Nutrition Diagnosis (PES): Excessive CPN solution related to exceeding calculated needs as evidenced by cholestasis.

Intervention: Food and Nutrient Delivery—change CPN solution to meet and not exceed kilocalories needs.

Monitoring and Evaluation: Track CPN intake according to estimated needs; evaluate labs, weight, liver function.

FOOD AND NUTRITION

- In chronic cases, 10–20% added kilocalories may be needed. Infants need more kilocalories, and adults tend to use CHO poorly. In acute stages, use IV glucose to prevent hypoglycemia and protein catabolism.
- In acute stages, infants will need 1.0–1.5 g/kg protein. Children, teens, and adults need 0.5–1.2 g protein/kg; highlight BCAAs sources. In chronic cases, use 3 g protein/d for infants and 1–1.5 g protein/kg in adults.
- Supplement with vitamins and minerals, especially fat-soluble vitamins. Vitamin D and calcium will be needed if osteopenia is present. Zinc and selenium may be needed.
- Small, frequent feedings and snacks may be better tolerated than large meals.
- Use enteral nutrition (where possible), if CPN has caused cholestasis. If CPN is required, early use of cyclic CPN may be useful. Avoid excesses of copper (Blaszyck et al, 2005) in the solutions.

Common Drugs Used and Potential Side Effects

- Ursodeoxycholic acid slows disease progression and should be used in relatively high doses as 20–30 mg/kg/d (Huang and Lichtenstein, 2005).
- Treat pruritus with bile acid–binding exchange resins such as cholestyramine or colestipol (Huang and Lichtenstein, 2005). Use with a low-fat diet and increase fluids and fiber. Constipation, nausea, or vomiting may be a side effect.
- Water-miscible forms of fat-soluble vitamins A, D, E, and K may be needed in cholestasis. Sample amounts of vitamin A may be given at 25,000–50,000 IU/d as Aquasol A; vitamin D may be given as 12,000–50,000 IU/d over a month; and vitamin E may be given as 10–25 IU/kg/d. Once nutrient stores are repleted and cholestasis is resolved, the supplementation can stop.
- Medications known to cause cholestasis include estrogens and anabolic steroids, chlorpromazine, erythromycin, and oxypenicillins.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Cholestatic liver injury may occur from some herbal remedies, such as greater celandine, glycyrrhizin, chaparral.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of fat in normal metabolic processes; simplify explanation in correlation to absorption of fat-soluble vitamins and other nutrients affected by the liver.
- Discuss ways to increase satiety from the diet with appetizing recipes.
- Discuss use of over-the-counter (OTC) vitamin and mineral supplements, especially regarding possible toxicity if taken in large doses with liver disease.

- LT works best in a well-nourished patient. Promote good tolerance and intake.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Characteristics of Liver Disease
  http://www.umm.edu/liver/common.htm
- Merck Manual—Cholestasis
  http://www.merck.com/mmhe/sec10/ch135/ch135c.html

CHOLESTATIC LIVER DISEASE—CITED REFERENCES

Blaszyck H, et al. Hepatic copper in patients receiving long-term total parenteral nutrition. *J Clin Gastroenterol.* 39:318, 2005.
Huang CS, Lichtenstein DR. Treatment of biliary problems in inflammatory bowel disease. *Curr Treat Options Gastroenterol.* 8:117, 2005.
Trauner M, et al. MDR3 (ABCB4) defects: a paradigm for the genetics of adult cholestatic syndromes. *Semin Liver Dis.* 27:77, 2007.

GALLBLADDER DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

The **gallbladder**, located under the liver, collects and stores bile, which is made up of bile salts, electrolytes, bilirubin, cholesterol, and other fats. Bile helps the small intestine digest fats and remove waste products, especially through bilirubin. It passes from the liver's bile duct into the duodenum through the common bile duct. Bile contains 85–95% water; electrolytes (sodium, potassium, chloride, bicarbonate, calcium, magnesium); bile acids and bilirubin; lecithin, cholesterol and protein. Loss of bile can cause malabsorption and maldigestion of fat, electrolyte imbalances, and poor excretion of drugs or heavy metals.

Cholelithiasis is defined as the presence of gallstones. In developed countries, at least 10% of white adults harbor cholesterol gallstones, especially after age 65. Women have twice the risk (Shaffer, 2005). There is also prevalence among persons who have hepatitis C, diabetes, obesity, pregnancy, use of estrogens, insulin, oral contraceptives, cholestyramine (Bini and McGready, 2005; Ko et al, 2005). The western diet that is high in kilocalories, fat, and refined carbohydrate is also a factor. Being Hispanic or Native American predisposes to gallbladder disease, as does sickle cell anemia and some other genetic traits.

Gallstones are a hepatobiliary disorder due to biochemical imbalances in the gallbladder bile (Uppal et al, 2008). Some gallbladders can concentrate bile normally but cannot acidify it. The result is that calcium may be less soluble in bile and precipitates out. Increasing consumption of magnesium (Mg^{++}) appears to decrease the risk of symptomatic gallstones in men (Ko, 2008). Magnesium deficiency can cause dyslipidemia and insulin hypersecretion, which may facilitate gallstone formation (Tsai et al, 2008).

Gallstones contain primarily cholesterol, bilirubin, and calcium salts, formed into either cholesterol or pigment stones. Symptoms include steady pain in the upper abdomen that increases rapidly and lasts from 30 minutes to several hours, pain in the back between the shoulder blades or under the right shoulder, nausea, vomiting, abdominal bloating, intolerance for fatty foods, belching, and indigestion.

Gallstones also form if the gallbladder does not contract completely or often enough to empty bile; this can also occur after eating too little, after periods of starvation, or fasting. Rapid weight loss or crash dieting is to be avoided. Preventive measures include a controlled weight loss rate, reduction of the length of overnight fast, inclusion of a small amount of fat in the diet, and eating foods rich in magnesium (nuts, vegetable protein, beans, and soy).

Cholecystitis is inflammation of the gallbladder. Rather than a single clinical entity, cholecystitis is a class of related disease states with different causes, degrees of severity, clinical courses, and management strategies. Gallstones with low-grade inflammation, scarring, and thickening are common triggers. If the gallbladder is removed, fat absorption still occurs, but it is less efficient because bile is not as concentrated.

Endoscopic stent placement in the gallbladder is effective for patients with gallbladder disease who are poor surgical candidates (Conway et al, 2005). However, surgery is needed for most cases. There are over 700,000 cholecystectomies performed annually in the United States alone (Shaffer, 2005).

Laparoscopic cholecystectomy (LC) reduces the length of hospital stay and can be performed on patients who are morbidly obese (Simopoulos et al, 2005). Extracorporeal shock wave lithotripsy (ESWL) is also effective.

Gallbladder cancer is not common but is more prevalent in women who have had gallstones for many years. Jaundice, pain above the stomach, lumps in the abdomen, and fever should be addressed. Gallbladder cancer is usually associated with late diagnosis, unsatisfactory treatment, and poor prognosis.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Alagille syndrome is a genetic disorder with mutations in the JAG1 and NOTCH2 genes where there are too few bile ducts to function properly;

this occurs in one in 70,000 people. Activation of nuclear receptor liver X receptor (LXR) sensitizes mice to lithogenic diet-induced gallbladder cholesterol crystallization; studies are needed in humans (Uppal et al, 2008).

Clinical/History

Height
Weight
BMI
Diet history
Intolerance for fatty foods?
WBC
Jaundice
Nausea, vomiting
I & O
Temperature
CT scan or endoscopic ultrasound
Magnetic resonance cholangiography (MRC)
Hepatobiliary iminodiacetic acid (HIDA) scan (cholescintigraphy)
Endoscopic retrograde cholangiopancreatography (ERCP)
Cholecochoscopy

Lab Work

Mg^{++} (low?)
Alk phos (ALP), elevated?
Bilirubin (increased)
AST, ALT (elevated?)
Amylase, Lipase (increased?)
Alb, transthyretin
Chol
Trig (elevated?)
H & H
Na^{+}, K^{+}
Ca^{++}

INTERVENTION

OBJECTIVES

- Lose excess weight, if needed but avoid fasting for rapid weight loss, which can lead to gallstones.
- Limit foods that cause pain or flatulence.
- For the patient with cholelithiasis, overcome fat malabsorption caused by obstruction and prevent stagnation in a sluggish gallbladder. Decreased bile secretion, bile stasis, bacteria, hormones, or fungi may be a problem; bacterial overgrowth alters bile acids so that they can no longer emulsify fats.
- Prevent biliary obstruction, cancer, or pancreatitis.
- Provide fat-soluble vitamins (ADEK) with signs of steatorrhea.
- Include a high magnesium food source daily.
- Ascorbic acid affects the catabolism of cholesterol to bile acids and the development of gallbladder disease; supplement the diet if needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Fat Intake

Assessment Data: Dietary intake records indicating intake of fried foods at most every meal; dining at fast food restaurants six to seven times weekly; abdominal and back pain, nausea, vomiting for 3 days.

Nutrition Diagnosis (PES): Excessive fat intake related to extensive use of fried foods and fast food choices as evidenced by diet history and signs of cholecystitis.

Intervention: Food and Nutrient Delivery—offer lower fat meals; prep for surgery. Educate about the role of the gallbladder in fat metabolism. Counsel about postsurgical diet (low fat, frequent small meals perhaps better tolerated) and gradual return to a general diet that contains more nutrient-dense and lower fat options. Discuss the role of minerals such as magnesium in maintaining GB health.

Monitoring and Evaluation: Track food intake (food diary or history). Evaluate for resolution of abdominal pain, nausea and vomiting after surgery; tolerance for diet and use of foods that are more nutrient-dense and lower in fat.

FOOD AND NUTRITION

- In **acute cholecystitis**, NPO or a low-fat diet may be needed. Progress to a diet with fewer condiments and gas-forming vegetables, which cause distention, increased peristalsis, and irritation.
- In **chronic cholecystitis**, use a fat/calorie-controlled diet to promote drainage of the gallbladder without excessive pain. Patient should consume adequate amounts of CHO, especially pectin, which binds excess bile acids.
- In **cholelithiasis**, encourage a diet that is high fiber, low in calories (as needed).
- Assure an adequate dietary intake of magnesium from nuts, bran, halibut, pollack, spinach, black or lima or white beans.
- Fat-soluble vitamins A-D-E-K may need to in water-miscible form.
- Increase dietary intake of sources of vitamin C such as citrus fruits and juices. Use supplemental forms if needed.

Common Drugs Used and Potential Side Effects

- Ursodiol (Actigall, Urso) is made from bile acid help dissolve small cholesterol gallstones over months or years. Take with food or milk. Ursodiol can lead to metallic taste, abdominal pain, mild diarrhea, or vomiting.
- The potent cholesterol absorption inhibitor ezetimibe reduces biliary cholesterol content and may be a promising strategy for preventing or treating cholesterol gallstones (Wang et al, 2008).
- Ursodeoxycholic acid decreases cholesterol saturation of bile and gallstone incidence during weight loss and may help to prevent gallstone formation. Orlistat is another option.
- Antibiotics may be used to counteract infection. Evaluate the need to take with food, milk or other liquids.
- If analgesics (Demerol, meperidine) are used to relieve pain, side effects such as nausea, vomiting, constipation, and GI distress can occur.
- Oral contraceptives and estrogens increase the risk of gallstones, especially after prolonged use. Orlistat has also been shown to cause gallstones for some patients. Thiazide diuretics have also been linked with gallstones (Leitzmann et al, 2005).

Herbs, Botanicals, and Supplements

- Herbal medicine such as turmeric and oregon grape may reduce gallbladder inflammation and relieve liver congestion.

- Herbs and botanical supplements should not be used without discussing with physician. Celandine, peppermint, couch grass, and goldenrod have been recommended for gallbladder disease, but no clinical trials have proven efficacy at this time.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- After a cholecystectomy, fat intake should be limited for several months to allow the liver to compensate for the gallbladder's absence. Fats should be introduced gradually; excessive amounts at one meal should be avoided. Use more unrefined carbohydrates as well.
- If diarrhea persists after surgery, try using antidiarrheal medications, such as loperamide (Imodium) and a high-fiber diet for more bulk.
- Avoid fasting and rapid weight loss schemes.
- People who have had their gallbladders removed should have their cholesterol levels checked periodically. To prevent new gallstones from forming, maintain a healthy weight.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- American College of Surgeons—Cholecystectomy
  http://www.facs.org/public_info/operation/cholesys.pdf
- Bile Duct Diseases
  http://www.nlm.nih.gov/medlineplus/bileductdiseases.html
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)—Gallstones
  http://digestive.niddk.nih.gov/ddiseases/pubs/gallstones/index.htm

GALLBLADDER DISEASE—CITED REFERENCES

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Shaffer EA. Epidemiology and risk factors for gallstone disease: has the paradigm changed in the 21st century? *Curr Gastroenterol Rep.* 7:132, 2005.

Simopoulos C, et al. Laparoscopic cholecystectomy in obese patients. *Obes Surg.* 15:243, 2005.

Tsai CJ, et al. Long-term effect of magnesium consumption on the risk of symptomatic gallstone disease among men. *Am J Gastroenterol.* 103:375, 2008.

Uppal H, et al. Activation of liver X receptor sensitizes mice to gallbladder cholesterol crystallization. *Hepatology.* 47:1331, 2008.

Wang HH, et al. Effect of ezetimibe on the prevention and dissolution of cholesterol gallstones. *Gastroenterology.* 134:2101, 2008.

SECTION 9

Endocrine Disorders

CHIEF ASSESSMENT FACTORS

Warning Signs of Type 1 Diabetes

- Drowsiness, Lethargy, Blurred Vision
- Extreme Thirst
- Frequent Urination
- Fruity, Sweet, Wine-Like Odor on Breath
- Glucose or Ketones in Urine
- Heavy, Labored Breathing
- Increased Appetite
- Sudden Weight Loss
- Stupor, Unconsciousness

Risk Factors for Type 2 Diabetes

- Age ≥45 Years
- Ethnicity—African American, Hispanic American, Native American, Asian American, Pacific Islander
- Family History (Parents or Siblings with DM)
- Habitually Sedentary
- High-Density Lipoprotein Cholesterol Level ≤35 mg/dL and Triglyceride Level ≥250 mg/dL
- History of Gestational Diabetes Mellitus (GDM) or Women Delivering Babies Weighing >9 lb
- History of Impaired Fasting Glucose (IFG) or Impaired Glucose Tolerance (IGT)
- Hypertension (Blood Pressure ≥140/90 mm Hg)
- Obesity with Body Mass Index ≥25 kg/m^2
- Polycystic ovarian syndrome, other conditions associated with insulin resistance
- Vascular disease

Assessment in Diabetes

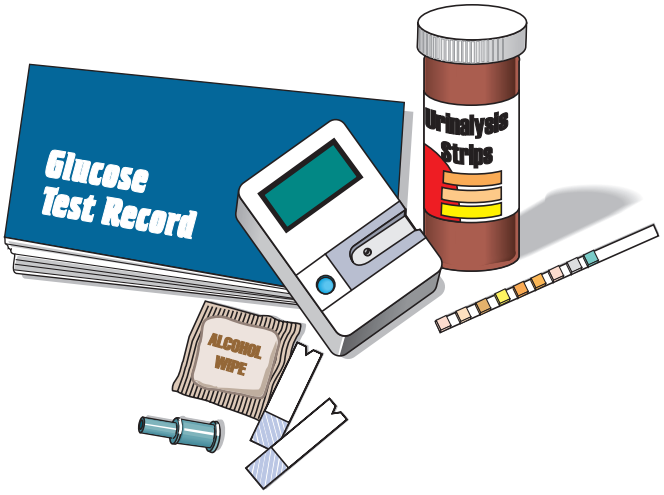
- Assessment for Mood Disorder
- Contraception; Reproductive and Sexual History
    - Current Treatment: Medications, Meal Plan, Results of Glucose Monitoring, Patients' Use of Data
    - Eating Patterns, Nutritional Status, and Weight History; Growth and Development in Children and Adolescents
        - Exercise History
        - Family History of Diabetes and Other Endocrine Disorders
        - Frequency, Severity, and Cause of Acute Complications Such as Ketoacidosis and Hypoglycemia

- History and Treatment of Other Conditions, Including Endocrine and Eating Disorders
- Infections: Skin, Foot, Dental, and Genitourinary Tract (Prior and Current)
- Laboratory Tests, Symptoms and Special Examination Results Related to the Diagnosis of Diabetes
- Lifestyle, Cultural, Psychosocial, Educational, and Economic Factors That Influence the Management of Diabetes
- Other Medications That May Affect Blood Glucose Levels
- Previous Treatment Programs (Nutrition and Diabetes Self-Management Education, Attitudes, and Health Beliefs)
- Prior HbA1c Records
- Risk Factors for Atherosclerosis: Smoking, Hypertension, Obesity, Dyslipidemia, and Family History
- Symptoms of Celiac Disease in Type 1 Diabetic Patients
- Symptoms and Treatment of Chronic Eye; Kidney; Nerve; Genitourinary (Including Sexual), Bladder, and Gastrointestinal Function; Heart; Peripheral Vascular; Foot; and Cerebrovascular Complications Associated with Diabetes
- Tobacco, Alcohol, and/or Controlled Substance Use

Assessment in Other Endocrine Conditions

- Adult Changes in Size of Head, Hands, Feet
- Altered Consciousness; Numbness, Tingling, or Paresthesia
- Anorexia, Nausea, Abdominal Pain, Malabsorption, Gastroparesis
- Bone Pain
- Decreased Libido, Erectile Dysfunction
- Diagnosis of Thyroid Disease or Other Endocrine Disorder
- Dysuria
- Goiter; Exophthalmos; Intolerance to Heat or Cold
- Headache, Seizures, Syncope
- Hormone Imbalances (Excess or Deficiency)
- Hormone Therapy: Anabolic Hormones—Growth Hormones, Androgens, Sex Hormones; Catabolic Hormones—Stress Hormones (Causing Gluconeogenesis from Protein) Such as Catecholamines (Epinephrine, Norepinephrine), Glucocorticoids (Cortisone, Cortisol), or Glucagon
- Hyperglycemia, Hypoglycemia
- Postural Hypotension, Weakness
- Pruritus, Dryness of Skin or Hair
- Shortness of Breath, Hoarseness
- Weight Changes

OVERVIEW OF DIABETES MELLITUS



Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels. Diabetes affects 24 million Americans; 54 million are estimated to have prediabetes, and over 200,000 individuals die each year of related complications (CDC, 2009; CMS, 2009).

Diabetes is divided into distinct types: type 1, type 2, gestational diabetes mellitus (GDM), and other types (see Table 9-1). Type 2 diabetes (T2DM), composing 90% of diabetes cases, is affected by both genetic and environmental factors. Types of diabetes affecting children and teens are listed in Table 9-2.

Adiposity-associated inflammation and insulin resistance are strongly implicated in the development of T2DM as well as the metabolic syndrome (Shah et al, 2008). Activation of nuclear transcription factor-B has been linked with a variety of inflammatory diseases, including diabetes. Antioxidant spices, herbs, and omega-3 fatty acids help to suppress inflammatory pathways.

Amino acids are also important; they support cell signaling, gene expression, and hormone synthesis. While physiological concentrations of amino acids and their metabolites (e.g., nitric oxide, polyamines, glutathione, taurine, thyroid hormones, and serotonin) are required, elevated levels of their products (e.g., ammonia, homocysteine, and asymmetric dimethylarginine) contribute to oxidative stress (Wu, 2009). Dietary supplementation with one or a mixture of arginine, cysteine, glutamine, leucine, proline, and tryptophan may be beneficial for ameliorating health problems including fetal growth restriction, neonatal morbidity and mortality, obesity, diabetes, cardiovascular disease (CVD), the metabolic syndrome, and infertility (Wu, 2009).

Medical nutrition therapy (MNT) replaced the term "diet therapy" years ago. There is no single "diabetic" or "ADA" diet. The recommended diet is an individualized nutrition prescription, based on assessment findings and treatment

TABLE 9-1 Etiologic Classification of Diabetes Mellitus

I. Type 1 diabetes (results from auto-immune beta-cell destruction, usually leading to absolute insulin deficiency).

II. Type 2 diabetes (results from a relative deficiency or insulin resistance).

III. Other specific types of diabetes due to other causes, for example, genetic defects in beta-cell function, genetic defects in insulin action, diseases of the exocrine pancreas (such as cystic fibrosis), or drug- or chemical-induced diabetes (such as in the treatment of AIDS or after organ transplantation).
   A. Genetic defects of beta-cell function: Maturity-onset diabetes of youth (MODY)
   B. Genetic defects in insulin action: Leprechaunism, Rabson–Mendenhall syndrome, Lipoatrophic diabetes
   C. Diseases of the exocrine pancreas: Pancreatitis, Trauma/pancreatectomy, Neoplasia, Cystic fibrosis, Hemochromatosis, Fibrocalculous pancreatopathy
   D. Endocrinopathies: Acromegaly, Cushing's syndrome, Glucagonoma, Pheochromocytoma, Hyperthyroidism, Somatostatinoma, Aldosteronoma
   E. Drug- or chemical-induced: Nicotinic acid, Glucocorticoids, Thyroid hormone, Beta-adrenergic agonists, Thiazides, Dilantin, Alpha-interferon
   F. Infections: Congenital rubella, Cytomegalovirus
   G. Other genetic syndromes sometimes associated with diabetes: Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Friedreich's ataxia, Huntington's chorea, Myotonic dystrophy, Porphyria, Prader-Willi syndrome

IV. Gestational diabetes mellitus (GDM) is diabetes first diagnosed during pregnancy.

Adapted from American Diabetes Association. Diagnosis and classification of diabetes mellitus. Web site accessed September 20, 2009, at http://care.diabetesjournals.org/content/29/suppl_1/s43.full; American Diabetes Association. Standards of Medical Care in Diabetes–2009.

TABLE 9-2 Types of Diabetes in Children and Teens

Type 1 Diabetes (Immune-Mediated)

- Usually not obese; often recent weight loss.
- Short duration of symptoms (thirst and frequent urination).
- Presence of ketones at diagnosis, with about 35% presenting with ketoacidosis.
- Often a honeymoon period after blood sugars are in control during which the need for insulin diminishes for awhile. Low dose of long-acting insulin, such as glargine (Lantus), may prolong the honeymoon phase.
- Ultimate complete destruction of the insulin-producing cells needing exogenous insulin for survival.
- Ongoing risk of ketoacidosis.
- Only about 5% with a family history (in first- or second-degree relatives) of diabetes.

Type 2 Diabetes (Insulin-Resistant)

- Usually overweight at diagnosis; little or no weight loss.
- Usually have sugar in the urine but no ketones.
- As many as 30% will have some ketones in the urine at diagnosis.
- About 5% will have ketoacidosis at diagnosis.
- Often little or no thirst and minimal increased urination.
- Strong family history of diabetes.
- 45–80% have at least one parent with diabetes.
- Diabetes may span many generations of family members.
- 74–100% have a first- or second-degree relative with diabetes.
- Typically from African, Hispanic, Asian, or American Indian origin.
- Disorders likely to cause insulin resistance are common.
- About 90% of children with type 2 diabetes have dark shiny patches on the skin (acanthosis nigricans), which are most often found between the fingers and between the toes, on the back of the neck (dirty neck), and in axillary creases.
- Polycystic ovarian syndrome (PCOS).

Maturity-Onset Diabetes of the Young (MODY)

- Rare form of diabetes; several varieties exist.
- Early age of onset (ages 9–25 years) with autosomal dominant inheritability.
- Results from defects to insulin-producing cells caused by a genetic defect in the pancreatic beta-cell function.
- Symptoms run the gamut from mild elevation in blood sugar to a severe disturbance.
- MODY can occur in all ethnic groups.
- Gene abnormalities are rare and can only be identified through testing that is currently available only in research laboratories.
- Not usually obese.
- Environmental stressors, such as illness or puberty, may unmask the genetically limited insulin secretory reserve of patients with undiagnosed MODY.
- Unlike type 1 diabetes, MODY does not cause polyuria, thirst, or extreme hunger. The primary goal is euglycemia.
- Fasting insulin and C-peptide levels are usually normal or elevated slightly.

Sources: American Diabetes Association, Web site accessed September 20, 2009, at http://spectrum.diabetesjournals.org/content/18/4/249.full; Children with Diabetes Web site accessed September 20, 2009, at http://www.childrenwithdiabetes.com/clinic/mody.htm

goals. MNT considers usual eating habits and other lifestyle factors. Strategic patient counseling should identify benefits of the plan, self-efficacy, obstacles, and lifestyle changes the patient is willing to make; these interventions will help reach desired outcomes. Using open-ended discussion and counseling encourages the client to assess and determine a realistic self-management plan. MNT should be offered in phases, according to patient comprehension and readiness.

Screening for diabetes is outlined in Table 9-3. The nutrition care process include assessment, nutrition diagnosis, intervention, followed by monitoring and evaluation. Monitoring of blood glucose, medications, physical activity, education, behavior modification, and evaluation of cardiovascular and renal status are all relevant. In addition, prevention of complications is essential; see Table 9-4.

A diabetes educator is defined as a health professional who has mastered the core knowledge and skills, communication, counseling, and education. The credentials of certified diabetes educators (CDE) designate professionals who have at least 1000 hours of direct diabetes teaching and who have successfully completed an examination. An advanced credential has been developed for registered dietitians (RDs), nurses, and pharmacists who have advanced degrees and meet experiential requirements; this credential is known as BC-ADM.

TABLE 9-3 Evaluation for Diabetes

Screen for prediabetes and diabetes in high-risk, asymptomatic, undiagnosed adults and children. An oral glucose tolerance test (OGTT) may be considered in patients with impaired fasting glucose (IFG) to better define the risk of diabetes. Testing for diabetes should be considered in all individuals at age 45 years and above, particularly in those with a BMI of 25 kg/m^2, and if normal, testing should be repeated at 3-year intervals. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss plus one of the following:

1. Symptoms of diabetes and a casual plasma glucose of 200 mg/dL. Casual is defined as any time of day without regard to time since last meal.
2. A fasting plasma glucose (FPG) test, a 2-hour OGTT (75-g glucose load), or both are appropriate. FPG ≥126 mg/dL. Fasting is defined as no caloric intake for at least 8 hours.
3. Two-hour plasma glucose of 200 mg/dL during an OGTT. The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

American Diabetes Association. Standards of Medical Care in Diabetes–2009. Web site accessed September 22, 2009, at http://care.diabetesjournals.org/content/32/Supplement_1/S13.full

TABLE 9-4 Potential Complications of Diabetes

ACUTE

Hyperglycemia	Symptoms include polyphagia, polydipsia, polyuria, dehydration, weight loss, weakness, muscle wasting, recurrent or persistent infections, hypovolemia, ketonemia, glycosuria, blurred or changed vision, fatigue, muscle cramps, and dry mouth. Blood glucose >250 mg/dL should be evaluated; monitor blood or urine ketones to check for diabetic ketoacidosis (DKA). Three forms of hyperglycemia may be noted in the hospitalized patient: (1) someone with a history of diagnosed diabetes; (2) previously unrecognized diabetes; (3) hospital-related fasting blood glucose of 126 mg/dL or random blood glucose of >200 mg/dL that reverts to normal after discharge.
DKA or nonketotic hyperosmolar state	The stress of illness, trauma, and/or surgery frequently aggravates glycemic control and may precipitate DKA or nonketotic hyperosmolar state. A vomiting illness accompanied by ketosis may indicate DKA, a life-threatening condition that requires immediate medical care to prevent complications and death (American Diabetes Association, 2009). Cerebral edema can occur. Early consultation with an endocrinologist is needed.
Hypoglycemia	Symptoms include shakiness, confusion, diplopia, irritability, hunger, weakness, headache, rapid and shallow breathing, numbness of mouth/lips/tongue, pulse normal or abnormal, convulsions, lack of coordination, dizziness, staggering gait, pallor, slurred speech, tingling, diaphoresis, nausea, sweating, tremors, and nightmares. Treat when blood glucose level is <70 mg/dL with 15 g of liquid or fat-free sources of glucose or carbohydrates (CHO); wait 15 minutes and retest, then treat with another 15 g of CHO if still <70 mg/dL. Evaluate blood glucose after 1 hour. Continue to monitor until next meal. Adding protein to carbohydrate does not affect the glycemic response and does not prevent subsequent hypoglycemia, but adding fat may retard and then prolong the acute glycemic response (American Diabetes Association, 2009). In rare, severe hypoglycemia (when the individual requires the assistance of another person and cannot be treated with oral carbohydrate), emergency glucagon kits may be used; these require a prescription and they can expire over time (American Diabetes Association, 2009).
Dawn phenomenon	The dawn phenomenon is the end result of a combination of natural body changes that occur during the sleep cycle. Between 3:00 a.m. and 8:00 a.m., the body naturally starts to increase the amounts of counter-regulatory hormones (growth hormone, cortisol, and catecholamines) which work against insulin's action to drop blood sugars. These cause a blood sugar levels to rise in the morning.
Somogyi effect	"Rebound hyperglycemia" leads to high blood sugar levels in the morning preceded by an episode of asymptomatic hypoglycemia. When blood sugar drops too low in the middle of the night, the body counters by releasing hormones to raise the sugar levels. Too much insulin earlier or not enough of a bedtime snack may be the problem.
Acute illness or infection	Any condition leading to deterioration in glycemic control necessitates more frequent monitoring of blood glucose and urine or blood ketones (American Diabetes Association, 2009). Aggressive glycemic management with insulin is needed with severe acute illness (American Diabetes Association, 2009). The risk for DKA is higher during this time; test blood glucose, drink adequate amounts of fluid, ingest CHO (50 g CHO every 3–4 hours) if blood glucose levels are low, and adjust medications to keep glucose in desired range and to prevent starvation ketosis. Marked hyperglycemia requires temporary adjustment of the treatment program and, if accompanied by ketosis, frequent interaction with the diabetes care team (American Diabetes Association, 2009). Hospitalization may be needed. In a pediatric case, cerebral edema may occur; referral to a pediatric endocrinologist is important.
Vaccines	Annually provide an influenza vaccine to all diabetic patients ≥6 months of age; provide at least one lifetime pneumococcal vaccine for adults with diabetes (American Diabetes Association, 2009). Persons who have diabetes tend to be at higher than normal risk for bacterial forms of pneumonia.
Critical illness	In critically ill patients, blood glucose levels should be kept as close to 110 mg/dL (6.1 μmol/L) as possible and generally <180 mg/dL (10 μmol/L); intravenous insulin is usually needed (American Diabetes Association, 2009). Scheduled prandial insulin doses should be given in relation to meals and should be adjusted according to point-of-care glucose levels; traditional sliding-scale insulin regimens may be ineffective and are not useful (American Diabetes Association, 2009).

(continued)

TABLE 9-4 Potential Complications of Diabetes *(continued)*

INTERMEDIATE	
Children with T1DM	Children's growth and development may be impaired with diabetes; adequate protein and energy must be provided. Children may be picky eaters.
	Blood glucose goals are less strict. Target range for normal blood glucose (90–130 mg/dL before eating for 13–19 year olds; 90–180 mg/dL for 6–12 year olds; and 100–180 mg/dL for 0–5 year olds) may be altered according to the health care provider's evaluation. Near-normalization of blood glucose levels is seldom attainable in children and adolescents after the honeymoon (remission) period (American Diabetes Association, 2009). The A1c goals also vary in children.
	Most children under 6 or 7 years of age have "hypoglycemic unawareness;" young children lack the cognitive capacity to recognize and respond to hypoglycemic symptoms (American Diabetes Association, 2009).
	Children with diabetes differ from adults in many ways, including insulin sensitivity related to sexual maturity, physical growth, ability to provide self-care, vulnerability to hypoglycemia, differing family dynamics, developmental stages, and physiological differences (American Diabetes Association, 2009). Work with the family and school to plan for emergencies such as low or high blood glucose levels.
Children with T2DM	Distinction between type 1 and type 2 diabetes in children can be difficult because autoantigens and ketosis may be present in a substantial number of patients (American Diabetes Association, 2009).
	Children with T2DM are usually overweight or obese and present with glycosuria with or without ketonuria, absent or mild polyuria and polydipsia, and little or no weight loss. Up to 33% have ketonuria at diagnosis; some may have ketoacidosis without any associated stress, illness, or infection.
Preconception	All women with diabetes and childbearing potential should be educated about the need for good glucose control before pregnancy, the need for family planning, and need for early treatment for diabetic retinopathy, nephropathy, neuropathy, and cardiovascular disease (American Diabetes Association, 2009). A1c levels should be normal or as close to normal as possible (<1% above the upper limits of normal) in an individual before conception is attempted (American Diabetes Association, 2009).
Pregnancy and diabetes	Infant mortality rates are approximately twice as high for babies born to women with uncontrolled diabetes compared with babies born to women without diabetes. To reduce the risk of fetal malformations and maternal or fetal complications, pregnant women and women planning to become pregnant require excellent blood glucose control. These women need to be seen frequently by a multidisciplinary team. Specialized laboratory and diagnostic tests may be needed. Women with diabetes must be trained in self-monitoring of blood glucose (SMBG).
	Nutrition recommendations for women with preexisting diabetes must be based on a thorough assessment. Monitoring blood glucose levels, blood or urine ketones, appetite, and weight gain is needed to develop an individualized nutrition prescription and to make adjustments to the meal plan.
	Plasma glucose should be maintained at 65–100 mg/dL before meals and in fasting; 110–135 mg/dL 1 hour after meals; and <120 mg/dL 2 hours after meals.
	Use an extra 300 kcal daily during the second and third trimester; 1 g/kg/d or an additional 25 g/d of protein; 175 g/d of CHO; 600 μg folic acid. Added iron and calcium may also be required.
Gestational diabetes	See the entry later in this chapter.
Lactation	Extra protein, calcium, and folic acid will be needed. Monitoring of blood glucose is recommended, but hypoglycemia should be avoided. Breastfeeding lowers blood glucose and may require women to eat a CHO-containing snack either before or during breastfeeding. Extra 330–400 kcal meets the needs of most lactating mothers.
Older adults	Unexplained weight loss should be viewed as a symptom of a problem. Diabetes is an important health condition for the aging population; at least 20% of patients over the age of 65 years have diabetes. These patients tend to have higher rates of premature death, functional disability, and coexisting illnesses such as hypertension, heart disease, and stroke than those without diabetes (American Diabetes Association, 2009). New-onset diabetes in adults over the age of 50 may signal underlying pancreatic cancer, which should be investigated.
	Strict diets in long-term care are not warranted and may lead to dehydration and malnutrition; specialized diabetic diets are not required, and a balanced diet with consistent timing of CHO is to be recommended. Modify medications, rather than diet, as needed. Lower hypertension gradually; implement the DASH diet whenever possible. A multivitamin–mineral supplement may be beneficial for this population.
CHRONIC	
A1c levels	Achieve and maintain normal blood glucose (BG) and lipids. Intensive therapy (IT) helps reduce onset or progression of vascular problems. Maintain hemoglobin A1c levels <7% (A1c level of 7 = BG 170).
	For evaluation, note that A1c of 5% = BG 100; 6% = BG 135; 7% = BG 170; 8% = BG 205; 9% = BG 240; 10% = BG 275; 11% = BG 310.
Microvascular	Retinopathy, ocular abnormalities, nephropathy, neuropathy (sensory or motor conditions, which may lead to ulceration or even limb amputation, orthostatic hypotension, intractable nausea and vomiting, and diabetic gastroenteropathy), diabetic cystopathy, and chronic diarrhea.

(continued)

TABLE 9-4 Potential Complications of Diabetes *(continued)*

CHRONIC	
Retinopathy	Retinopathy from diabetes accounts for 12,000–24,000 cases of blindness each year. Optimal glycemic and blood pressure control can substantially reduce the risk and progression of diabetic retinopathy (American Diabetes Association, 2009).
	In the presence of proliferative diabetic retinopathy (PDR) or severe nonproliferative diabetic retinopathy (NPDR), vigorous aerobic or resistance exercise may be contraindicated because of the risk of triggering vitreous hemorrhage or retinal detachment (American Diabetes Association, 2009).
	Pregnancy in T1DM may aggravate retinopathy; laser photocoagulation surgery can minimize this risk (American Diabetes Association, 2009).
Microalbuminuria and nephropathy	Early signs of nephropathy include hyperfiltration, renal hypertrophy, and microalbuminuria (loss of 30–300 μg/mg creatinine in the urine). Protein losses at a rate of >300 μg/mg creatinine may indicate advancement toward chronic kidney disease (CKD). To reduce the risk and/or slow the progression of nephropathy, optimize glucose and blood pressure control (American Diabetes Association, 2009). Nephropathy is preceded by microalbuminuria for several years; onset of end-stage renal disease is about 5 years after onset of microalbuminuria. For diabetic nephropathy, liberalize CHO intake and control insulin levels accordingly.
	Controlled protein intake will slow down progression of nephropathy. Adults need <0.8–1 g protein/kg daily (American Diabetes Association, 2009). Protein restriction is of benefit in slowing the progression of albuminuria, glomerular filtration rate (GFR) decline, and occurrence of end-stage renal disease (ESRD). Protein restriction should be considered in patients whose nephropathy seems to be progressing despite optimal glucose and blood pressure control and use of angiotensin-converting enzyme (ACE) inhibitors and/or angiotensin receptor blockers (ARBs) (American Diabetes Association, 2009).
	Intensive diabetes management with the goal of achieving near normoglycemia has been shown to delay the onset of microalbuminuria (American Diabetes Association, 2009). Refer to a physician experienced in the care of diabetic renal disease when the estimated GFR has fallen to <60 mL/min or if difficulties occur in the management of hypertension or hyperkalemia (American Diabetes Association, 2009). ACE inhibitors are almost always prescribed to decrease progression.
	Phosphorus should be controlled at 8–12 mg/kg/d. Some people may need phosphate binders and calcium supplements.
End-stage renal disease	Diabetes is the leading cause of CKD, especially among blacks, Mexican Americans, and Native Americans. Diabetic nephropathy occurs in 20–40% of patients with diabetes and is the single leading cause of ESRD (American Diabetes Association, 2009). Creatinine and GFR should be assessed annually in this population. Microalbuminuria (30–299 mg/24 h) and macroalbuminuria (≥300 mg/24 h) should be carefully monitored (American Diabetes Association, 2009).
	While physical activity can acutely increase urinary protein excretion, there is no evidence that vigorous exercise increases the rate of progression of diabetic kidney disease (American Diabetes Association, 2009).
Autonomic neuropathy	About 70% of persons who have diabetes have some degree of neuropathy, including impaired sensation in the hands and feet, slowed digestion, or carpal tunnel syndrome. Autonomic neuropathy can decrease cardiac responsiveness to exercise, causing postural hypotension, impaired thermoregulation, impaired skin blood flow and sweating, impaired night vision, impaired thirst, risk of dehydration, and gastroparesis with unpredictable food delivery (American Diabetes Association, 2009). Neuropathy may be delayed with careful blood glucose management. Weight loss may be beneficial for obese persons.
Skin and joints	Decreased pain sensation in the extremities may increase risk of skin breakdown and infection and joint destruction; it may be best to encourage nonweight-bearing activities such as swimming, bicycling, or arm exercises (American Diabetes Association, 2009).
Genitourinary	Diabetic autonomic neuropathy is associated with recurrent genitourinary tract disturbances or bladder and/or sexual dysfunction.
Amputations	Lower extremity amputations are painful and disabling; prevention is desirable. Amputation and foot ulcers are the most common consequences of diabetic neuropathy and the major causes of morbidity and disability in people with diabetes (American Diabetes Association, 2009).
Cardiac neuropathy	Major clinical manifestations of cardiac neuropathy in diabetes include resting tachycardia, exercise intolerance, orthostatic hypotension, constipation, gastroparesis, erectile dysfunction, sudomotor dysfunction, impaired neurovascular function, "brittle diabetes," and hypoglycemic autonomic failure (American Diabetes Association, 2009). Evaluate cardiac status before starting an exercise program.
Gastrointestinal (GI) neuropathy	GI disturbances (esophageal enteropathy, gastroparesis, constipation, diarrhea, and fecal incontinence) are common, and any section of the GI tract may be affected. Gastroparesis should be suspected in individuals with erratic glucose control (American Diabetes Association, 2009).

(continued)

TABLE 9-4 Potential Complications of Diabetes *(continued)*

Macrovascular	
Coronary artery disease and arterial vascular disease	CVD is a major cause of mortality and also a major contributor to morbidity in diabetics. T2DM is an independent risk factor for macrovascular disease (American Diabetes Association, 2009). Other risk factors include insulin resistance, hyperglycemia, hyper tension, dyslipidemia, and smoking. Smoking is related to the premature development of microvascular complications of diabetes and may have a role in the development of T2DM (American Diabetes Association, 2009). Advise all patients not to smoke; include smoking cessation as a routine component of care (American Diabetes Association, 2009).
Dyslipidemia	Lifestyle modification focusing on the reduction of saturated fat and cholesterol intake, weight loss (if indicated), and increased physical activity has been shown to improve the lipid profile in patients with diabetes (American Diabetes Association, 2009).
	Guidelines promote low-density lipoprotein (LDL) cholesterol levels of <100 mg/dL as optimal for all patients and lowering of triglyceride levels when >150 mg/dL. Statins may be helpful. Elevate high-density lipoprotein (HDL) to levels >50 mg/dL. If HDL is <50 mg/dL and LDL is between 100 and 129 mg/dL, a fibric acid derivative or niacin might be used. Niacin raises HDL but can significantly increase blood glucose at high doses (American Diabetes Association, 2009). Use aspirin therapy (75–162 mg/d) as a secondary prevention strategy in those with diabetes with a history of CVD (American Diabetes Association, 2009).
	Tailor nutrition interventions according to each patient's age, type of diabetes, pharmacological treatment, lipid levels, and other medical conditions. Focus on the reduction of saturated fat, cholesterol, and trans fat intake (American Diabetes Association, 2009). Monitoring of homocysteine levels and related actions should be taken. Adequate insulin therapy often returns lipids to normal in T1DM. Elevated levels in T2DM require strict management; limit saturated fat to 7–10% of total calories.
	The rate of heart disease is about two to four times higher in adults who have diabetes; it is the leading cause of diabetes-related deaths. Since people with diabetes tend to have high triglyceride and low HDL levels, omega-3 fatty acids from DHA and EPA (fish and marine oils) can help.
Hypertension	Over 70% of adults with diabetes also have hypertension. Control of hypertension in diabetes has been linked to reduction in the progression of both microvascular and macrovascular disease. In T1DM, hypertension is often the result of underlying nephropathy, whereas in T2DM, it may be present as part of the metabolic syndrome (American Diabetes Association, 2009). Patients with systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg should receive drug therapy in addition to lifestyle and behavioral therapy (American Diabetes Association, 2009).
	Blood pressure should be measured at every routine diabetes visit; patients with diabetes should be treated to reach a systolic blood pressure <130 mm Hg and a diastolic blood pressure <80 mm Hg (American Diabetes Association, 2009).
	In patients with T1DM with hypertension and any degree of albuminuria, ACE inhibitors have been shown to delay the progression of nephropathy. With T2DM, hypertension, and microalbuminuria, ACE inhibitors and ARBs have been shown to delay the progression to macroalbuminuria (American Diabetes Association, 2009).
	Modest weight loss is helpful. Prevent hypokalemia, which can blunt insulin release. Control glucose; limit alcohol and smoking; and add exercise. Use the DASH diet because it increases intake of calcium, magnesium, and potassium while lowering alcohol and excess weight.
Hypertension in pregnancy	Pregnancy-induced hypertension is two to four times more common in women with T1DM than in the general population. Screen for microalbuminemia as a strong predictor of pre-eclampsia.
	In pregnant patients with diabetes and chronic hypertension, blood pressure target goals are 110–129/65–79 mm Hg; avoid lower blood pressure because fetal growth may be impaired (American Diabetes Association, 2009). ACE inhibitors and ARBs are contraindicated during pregnancy (American Diabetes Association, 2009).
Stroke	Stroke risk is two to four times higher among persons with diabetes, and high blood pressure should be carefully controlled.
Catabolic illness	Catabolic illness (such as HIV infection, AIDS, or cancer) changes body compartments, with increased extracellular fluid and shrinkage of body fat and cell mass. Monitor unexplained weight losses carefully, especially 10% or more of usual weight.
	Standard tube feedings are usually well tolerated in persons with diabetes (50% CHO or lower); monitor fluid status, weight, plasma glucose and electrolytes, and acid–base balance. Overfeeding is to be avoided; start with 25–35 kcal/kg of body weight. Protein needs may be 1 g/kg up to 1.5 g/kg in stressed individuals, and pressure ulcers may require a higher level of protein and calories than normal for healing. Addition of insulin may be needed. Oral glucose-lowering medications need to be adjusted to achieve adequate control of glycemia.

Additional data from: American Diabetes Association, 2009; Centers for Disease Control and Prevention, 2009.

TABLE 9-5 Key Concepts in Diabetes Management

Glucose Control

- Improved glycemic control benefits people with either type 1 or type 2 diabetes.
- For every 1% reduction in results of A1c blood tests (e.g., from 8.0% to 7.0%), the risk of developing microvascular diabetic complications (eye, kidney, and nerve disease) drops by 40%.
- A1c is the primary target for glycemic control. More stringent glycemic goals (i.e., a usual A1c, <6%) may further reduce complications at the cost of increased risk of hypoglycemia. Less-intensive glycemic goals may be indicated in patients with severe or frequent hypoglycemia.
- Postprandial glucose may be targeted if A1c goals are not met despite reaching preprandial glucose goals.
- Goals should be individualized, and lower goals may be reasonable based on benefit–risk assessment.
- Certain populations (children, pregnant women, and elderly) require special considerations. See guidelines:

	Plasma Blood Glucose Goal			
Values by Age (years)	**Pre-prandial (mg/dL)**	**Bedtime/ Overnight**	**A1c**	**Rationale**
Toddlers and preschoolers age (0–6)	100–180	110–200	<8.5% (but >7.5%)	High risk and vulnerability to hypoglycemia
School age (7–12)	90–180	100–180	<8%	Risks of hypoglycemia and relatively low risk of complications prior to puberty
Adolescents and young adults (13–19)	90–130	90–150	<7%	Risk of severe hypoglycemia; developmental and psychological issues; a lower goal <7% is reasonable if achieved without excessive hypoglycemia
Adults (20+)	90–130		<7%	Peak post-prandial <180 mg/dL 1–2 hours after beginning of meal

Blood Pressure Control

- Goal: blood pressure <130/80 mm Hg.
- Restriction of sodium to 2400 mg/d assists in the control of hypertension.
- Blood pressure control can reduce cardiovascular disease (heart disease and stroke) by approximately 33–50% and can reduce microvascular disease (eye, kidney, and nerve disease) by approximately 33%.
- In general, for every 10-mm Hg reduction in systolic blood pressure, the risk for any complication related to diabetes is reduced by 12%.

Control of Blood Lipids

- Improved control of cholesterol or blood lipids (e.g., high-density lipoprotein [HDL], low-density lipoprotein [LDL], and triglycerides) can reduce cardiovascular complications by 20–50%.
- Goals: Total cholesterol minus HDL should be ≤130 mg/dL; LDL <100 mg/dL; Triglycerides<150 mg/dL; HDL<40 mg/dL in men, >50 mg/dL in women.

Preventive Care Practices for Eyes and Feet

- Detecting and treating diabetic eye disease with laser therapy can reduce the development of severe vision loss by an estimated 50–60%.
- Comprehensive foot care programs can reduce amputation rates by 45–85%.

Preventive Care for Kidneys (Prerenal Failure)

- Detecting and treating early diabetic kidney disease by lowering blood pressure can reduce the decline in kidney function by 30–70%. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are most effective in reducing the decline in kidney function.
- With protein intakes greater than 20% of energy intake, there is an association with increased albumin excretion rate.
- Once albuminuria is present, reduce protein to 0.8–1.0 g/kg/d with microalbuminuria and to 0.8 g/kg/d with macroalbuminuria. There may be a benefit to lowering phosphorus intake to 500–1000 mg/d.
- There is evidence for a benefit on renal function, glucose, lipids, and blood pressure from weight-maintaining diets.

Adapted from: National Diabetes Guidelines, Web site accessed September 20, 2009, at http://guidelines.gov/Compare/comparison.aspx?file=DIABETES_NUTRITION1.inc#t3general

Collaborative, multidisciplinary teams are best suited to provide such care for people with chronic conditions such as diabetes and to empower patients' performance of appropriate self-management (American Diabetes Association, 2009). MNT provided by RDs to patients with type 1 and T2DM has been shown to improve glycemic outcomes. Community-based education that incorporates Social Cognitive Theory and Stages of Change Theory, with three group sessions focused on meal planning with cooking demonstrations, is effective; the education successfully promotes use of herbs in place of salt, use of olive or canola oils, use of artificial sweeteners in baking, knowledge of diabetes and nutrition, and self-efficacy (Chapman-Novakofski and Karduck, 2005). Table 9-5 offers more tips for diabetes management.

It is also important to address culturally specific eating habits. For example, a recent study in South Dakota found that a diet patterned after the historical hunter–gatherer type diet (with the 25% of energy supplied from protein), provided better blood glucose control and lower the circulating insulin levels in Northern Plains Indians with T2DM. The same may be true for other high-risk groups.

For More Information

- American Association of Diabetes Educators
  http://www.aadenet.org/
- American Diabetes Association
  http://www.diabetes.org/
- American Diabetes Association Youth Zone
  http://www.diabetes.org/youthzone/youth-zone.jsp
- Calorie and Nutrient Information
  http://www.calorieking.com
- Canadian Diabetes Association
  http://www.diabetes.ca/
- Centers for Disease Control and Prevention (CDC) Diabetes Public Health Resources
  http://www.cdc.gov/diabetes/index.htm
- CDC Division of Diabetes
  http://www.cdc.gov/diabetes
- Centers for Medicare and Medicaid Services Diabetes Guidelines
  http://www.cms.hhs.gov/DiabetesScreening/
- Children with Diabetes
  http://www.childrenwithdiabetes.com
- Diabetes Care and Dietetic Practice Group
  http://www.dce.org/
- Diabetic Gourmet Magazine
  http://diabeticgourmet.com/dgarchiv2.shtml
- Diabetes Research Institute Foundation
  http://www.drinet.org/
- International Diabetes Federation
  http://www.idf.org
- Joslin Diabetes Center, Boston, MA
  http://www.joslin.org/
- Juvenile Diabetes Research Foundation International
  http://www.jdrf.org
- Low Literacy Information
  http://www.learningaboutdiabetes.com
- National Diabetes Education Program
  http://www.ndep.nih.gov/
- National Guideline Clearinghouse – Diabetes
  http://guidelines.gov/summary/summary.aspx?doc_id=12816&nbr=006618
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
  http://www.diabetes.niddk.nih.gov/
- Park Nicollet–International Diabetes Center
  http://www.Parknicollet.com/diabetes
- School Guidelines
  http://www.ndep.nih.gov/diabetes/pubs/Youth_NDEPSchoolGuide.pdf
- Taking Control of Your Diabetes
  http://www.tcoyd.org

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CDC (Centers for Disease Control and Prevention). National diabetes fact sheet. Web site accessed September 21, 2009, at http://www.cdc.gov/diabetes/pubs/general.htm#impaired

Chapman-Novakofski K, Karduck J. Improvement in knowledge, social cognitive theory variables, and movement through stages of change after a community-based diabetes education program. *J Am Diet Assoc.* 105:1613, 2005.

CMS (Centers for Medicare and Medicaid Services). Diabetes Screening. Web site accessed September 20, 2009, at http://www.cms.hhs.gov/DiabetesScreening/

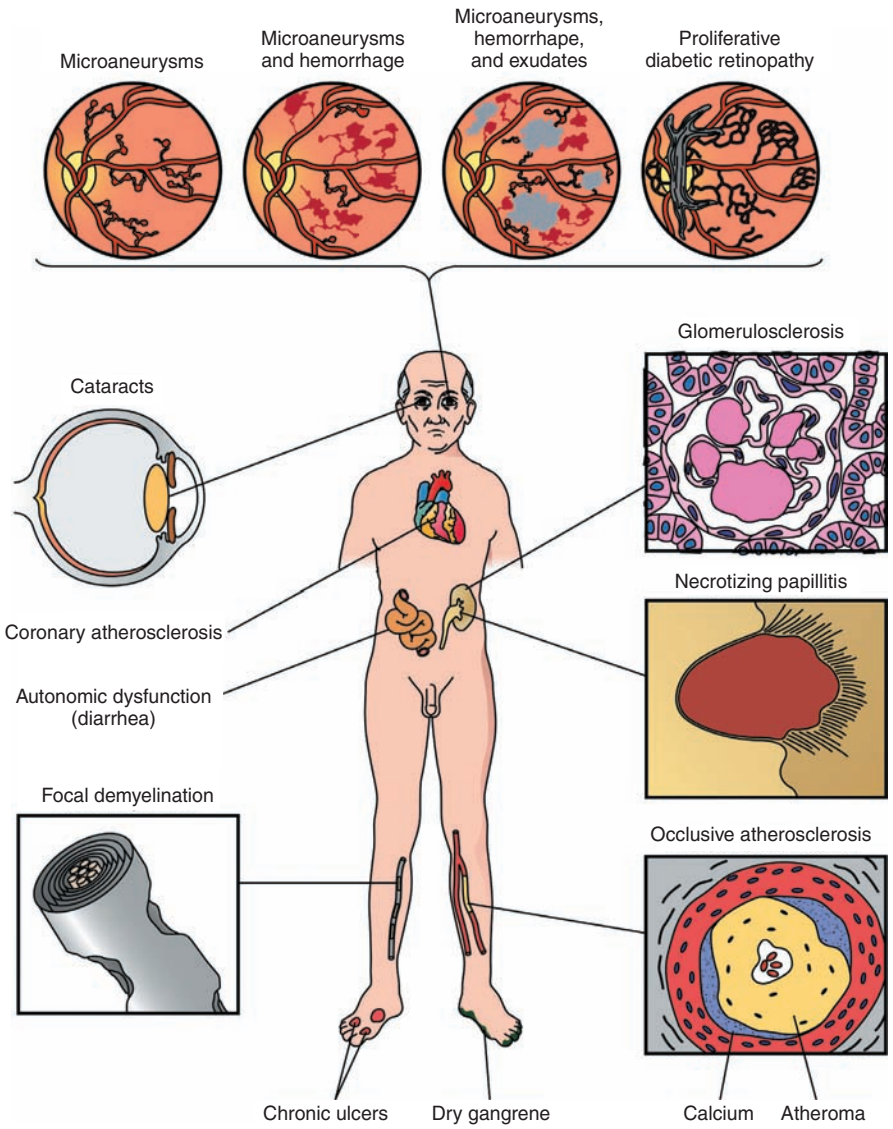
Shah A, et al. Adipose inflammation, insulin resistance, and cardiovascular disease. *JPEN J Parenter Enteral Nutr.* 32:638, 2008.

Wu G. Amino acids: metabolism, functions, and nutrition. *Amino Acids.* 37:1, 2009.

DIABETES MELLITUS, COMPLICATIONS, AND RELATED CONDITIONS

TYPE 1 DIABETES MELLITUS

NUTRITIONAL ACUITY RANKING: LEVEL 4



Adapted from: Raphael Rubin, David S. Strayer, *Rubin's Pathology: Clinicopathologic Foundations of Medicine,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

DEFINITIONS AND BACKGROUND

Type 1 diabetes mellitus (T1DM) is absolute insulin deficiency with total failure to produce insulin. Previously used terms include "type I," "insulin-dependent diabetes mellitus (IDDM)," "juvenile," "brittle," or "ketosis-prone" diabetes. T1DM involves autoimmune destruction of the pancreatic beta-cells (the islets of Langerhans). Onset often follows viral infection such as mumps. It usually starts in children or young adults, affecting 10% of cases of diabetes; beta-cell damage is often more severe in patients diagnosed before puberty.

Signs and symptoms of diabetes include polyuria (frequent urination, including frequent bedwetting in otherwise trained children), polydipsia (excessive thirst), polyphagia (extreme hunger), weakness, fatigue, irritability, and sudden weight loss. A fasting plasma glucose (FPG) test or an oral glucose tolerance test (OGTT) can be used. FPG is the preferred method; level of 126 mg/dL or higher is considered to be diabetes. With the OGTT, blood glucose is measured after fasting and 2 hours after drinking a glucose beverage; results between 140 and 199 mg/dL indicate prediabetes, and a level of 200 mg/dL represents diabetes. Hemoglobin A1c (HbA1c) test is not recommended for diagnosis; finger-prick tests are also not valid.

Serious complications begin earlier than previously thought. Glucose control really matters, as proven by the