

Gluc CRP	Secretin stimula- tion test	Fecal elastase-1 in stool
Serum carotene levels	Trypsin or chy- motrypsin in stool	Serum copper (Cu)
Ca <sup>++</sup> , Mg <sup>++</sup>		

## INTERVENTION



### OBJECTIVES

- Determine current level of care needed (Table 5-8). Effective treatment should allow a normal diet, symptom control, malabsorption correction, and attainment of a normal nutritional state and growth.
- Achieve or maintain desirable BMI. BMI percentile is a better evaluation than % ideal body weight (IBW) in CF patients; %IBW underestimates the severity of malnutrition in children with short stature and overestimates the severity of malnutrition in children with tall stature (Zhang and Lai, 2004).
- Correct anorexia from respiratory distress.
- Provide optimal amounts of protein for growth, development, and resistance to infection. Increase LBM if depleted. Spare protein by providing up to twice the normal amount of calories from CHO and fat as in usual diet plans. Stunting may require extra protein, as from TF (Geukers et al, 2005).
- Decrease electrolyte losses in vomiting and steatorrhea. Replace lost electrolytes.
- Achieve adequate enzyme replacement to bring about near-normal digestion. Reduce excessive nutrient losses from maldigestion and malabsorption.
- Provide essential fatty acids in a tolerated form. Reduce arachidonic acid use to lessen inflammatory cascade. Include omega-3 fatty acids and antioxidants such as selenium and vitamins C and E. Vitamin E may be especially important for improving cognitive function (Koscik et al, 2004).
- Promote adequate bone mass, as serum levels of vitamins D and K may be low (Grey et al, 2008).
- Correct edema, diarrhea, anemia, azotorrhea, and steatorrhea.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Poor Nutritional Quality of Life

**Assessment Data:** Low BMI, anorexia, polypharmacy, depression.

**Nutrition Diagnoses (PES):** Poor nutritional quality of life related to GI distress with meals and polypharmacy as evidenced by depression, low BMI and anorexia.

**Interventions:** Offer nutrient dense foods. Collaborate with medical team to address depression and polypharmacy. Assure intake of pancreatic enzymes with meals to reduce GI distress.

**Monitoring and Evaluation:** Improved appetite and intake; BMI closer to normal range; improved nutritional quality of life.

**TABLE 5-8 Nutritional Management for Cystic Fibrosis (CF)**

Routine care	Within desired BMI
Anticipatory guidance	At 90–95% desired BMI
Supportive intervention	At 85–90% desired BMI
Rehabilitative care	At 75–85% desired BMI
Resuscitative or palliative care	Below 75% desired BMI

- Prevent progressive pulmonary disease or complications such as glucose intolerance, intestinal obstruction, cirrhosis, and pancreatic or cardiac diseases.
- Improve lung function outcomes from better nutrition and fewer chronic infections (McPhail et al, 2008).



## FOOD AND NUTRITION

- Energy expenditure may be as high as 199% of predicted in CF patients. CF patients may need to be given 120–150% more calories than for age-matched and gender-matched controls; this may mean 3000–4000 kcal for teens.
- Design the plan for 45–65% CHO and 20–30% fat. For persons with acute disease, starch and fat will not be well tolerated unless adequate levels of pancreatic enzymes are provided. Calorie intake should be about 150 kcal/kg for children and 200 kcal/kg for infants. Specific interventions for increasing total energy intake in CF patients are the role of the dietitian (Powers et al, 2004). Many supplements are available at little or no cost to the patient.
- Manage glucose levels if CF diabetes mellitus (CFDM) develops. Intensive insulin therapy and CHO counting will be important.
- Protein should be 10–35% of total calories. This may translate into 4 g/kg for infants, 3 g/kg for children, 2 g/kg for teens, and 1.5 g/kg for adults.
- Increase fat:CHO ratio with respiratory distress. Special respiratory formulas may be useful during those times, or use of MCTs and safflower oil may be beneficial. Be sure to time intake according to the use and type of pancreatic enzymes.
- Encourage intake of omega-3 fatty acids (DHA and EPA), selenium, betaine, and choline to reduce inflammation and enhance immunity.
- Supplement the diet with two times the normal RDAs for fat-soluble vitamins A, D, and E (use water-miscible sources such as “ADEKS” brand).
- Replace vitamin K as needed; check levels regularly. Either 1 mg or 5 mg doses of vitamin K will help replenish low levels (Drury et al, 2008).
- Use extra riboflavin if there is cheilosis; include the other B-complex vitamins and vitamin C at recommended levels.
- Be sure that iron, zinc, copper, selenium intakes meet recommended levels.
- Use liberal amounts of salt to replace perspiration losses.
- Lactose intolerance is common. Omit milk during periods of diarrhea if lactose intolerance persists.
- Intolerance for gas-forming foods and concentrated sweets may occur; alter dietary plans accordingly.

- Soft foods may be useful if chewing causes fatigue.
- Fluid intake should be liberal unless contraindicated.
- Use of turmeric and cumin in foods may be beneficial for CF patients (Berger et al, 2005). Research is ongoing to determine overall practicality of uses.
- Infants can tolerate most formulas (may need 24 kcal/oz) and commercial products that include some MCT oil. Do not add pancreatic enzymes to formula because desired amounts may not be totally consumed or enzymes may block the opening of the nipple.
- Nocturnal TF may be appropriate with growth failure. With reflux, a gastrostomy feeding tube may be well tolerated (Oliver et al, 2004). PN is not recommended due to high risk of infection.

### Common Drugs Used and Potential Side Effects

- Growth hormone may be used to bring onset of puberty in prepubescent children who have CF (Vanderwel and Hardin, 2006).
- New aerosol treatments show promise. See Table 5-9 for alternative therapies.

### Herbs, Botanicals, and Supplements

- Interesting studies suggest that curcumin may directly stimulate CFTR  $\text{Cl}^-$  channels (Berger et al, 2005). Use of turmeric and cumin in foods served to this population may have therapeutic benefits.
- Dietary supplement use is prevalent among CF children. Identify use of nonprescribed supplements because of unknown effects on growth and development and the potential for adverse drug interactions (Ball et al, 2005).

- The individual with CF should work with the CF nutritionist to maintain a healthy diet before considering adding herbal therapies. Each label on any supplement should be read carefully; some ingredients that can be toxic to people with CF.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Diet must be periodically reevaluated to reflect growth and disease process.
- New foods may be introduced gradually.
- A behavioral and nutrition intervention can be used with children to enhance weight and height velocities (Powers et al, 2005).
- To liquefy secretions, adequate fluid intake should be ensured. Discuss signs of dehydration and how to prevent or correct.
- Bronchopulmonary drainage, three times daily, may be required. Plan meals to be 1 hour before or after therapy.
- Ensure that all foods and beverages are nutrient dense.
- As needed, discuss issues related to fertility (most males with CF are infertile, but females are not).
- In adults with CF, 40% have glucose intolerance. Discuss how to manage diabetes in those cases.
- Discuss reimbursement issues for TFs and pumps.
- Depression is common and should be adequately managed (Quittner et al, 2008). Hypnosis may be useful in reducing pain from frequent intravenous injections or other treatments.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

**TABLE 5-9 Medications Used in Cystic Fibrosis (CF) and Potential Side Effects**

Medication	Description and Side Effects
Aerosolized antibiotics	TOBI (tobramycin solution for inhalation) can be delivered in a more concentrated dose directly to the site of CF lung infections and is preservative free.
Antibiotics	Antibiotics are needed during infections. Monitor for magnesium depletion.
Azithromycin	Azithromycin is an antibiotic that is effective in people with CF whose lungs are chronically infected with the common <i>Pseudomonas aeruginosa</i> bacteria.
Bisphosphonates	Bisphosphonates may be used to increase bone density.
Bronchodilators	Bronchodilators are used to open breathing passages. Monitor for side effects.
Glutathione (GSH)	Buffered GSH has been tested in some CF patients. Nebulized buffered GSH may ameliorate CF disease; longer and larger studies of inhaled GSH are warranted (Bishop et al, 2005).
L-arginine	Oral L-arginine (200 mg) may reduce nitric oxide levels, which can be detrimental (Grasemann et al, 2005). More studies are needed.
Mucolytics	Mucolytics, such as potassium iodide, liquefy secretions.
Pancreatic enzymes (pancrelipase)	Pancreatic granules (Viokase or Cotazym) are used to help improve digestion/absorption. Enteric preparations (Pancreas) act in the duodenum, so give before meals; for nocturnal feedings, give before, during, and after feedings. Avoid mixing with milk or ice cream. If too much is given, anorexia and constipation may result. Return of a voracious appetite and increase in stool bulk suggest an inadequate dosage. Dosing should be based on stool tests for malabsorption.
Pulmozyme	A mucus-thinning drug shown to reduce the number of lung infections and improve lung function.
Ursodeoxycholic acid	Used for meconium ileus and liver disease associated with CF (Lamireau et al, 2004).

NOTE. The need to take up to 40–60 pills daily is common in CF.

### For More Information

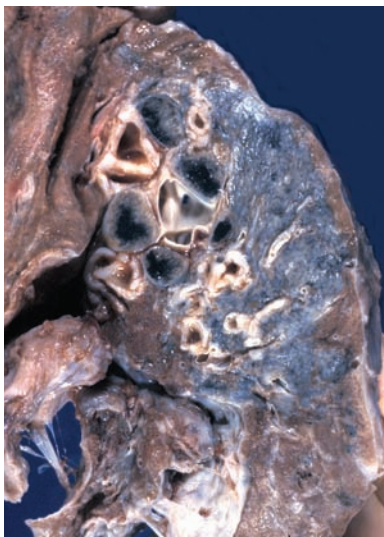
- Cystic Fibrosis  
<http://www.cysticfibrosis.com/>
- Cystic Fibrosis Foundation  
<http://www.cff.org/>
- Cystic Fibrosis Research  
<http://www.cfri.org/indexframes.htm>
- Medline  
<http://www.nlm.nih.gov/medlineplus/ency/article/000107.htm>
- NHLBI—Cystic Fibrosis  
[http://www.nhlbi.nih.gov/health/dci/Diseases/cf/cf\\_what.html](http://www.nhlbi.nih.gov/health/dci/Diseases/cf/cf_what.html)
- NIH  
<http://ghr.nlm.nih.gov/condition=cysticfibrosis>
- Nutrition for CF  
<http://www.nlm.nih.gov/medlineplus/ency/article/002437.htm>

### CYSTIC FIBROSIS—CITED REFERENCES

- Augarten A, et al. Systemic inflammatory mediators and cystic fibrosis genotype. *Clin Exp Med*. 4:99, 2004.
- Ball SD, et al. Dietary supplement use is prevalent among children with a chronic illness. *J Am Diet Assoc*. 105:78, 2005.
- Berger AL, et al. Curcumin stimulates CFTR Cl-channel activity. *J Biol Chem*. 280:5221–5226, 2005.
- Bishop C, et al. A pilot study of the effect of inhaled buffered reduced glutathione on the clinical status of patients with cystic fibrosis. *Chest*. 127:308, 2005.
- Dobson L, et al. Microalbuminuria as a screening tool in cystic fibrosis-related diabetes. *Pediatr Pulmonol*. 39:103, 2005.
- Drury D, et al. Efficacy of high dose phyloquinone in correcting vitamin K deficiency in cystic fibrosis. *J Cyst Fibros*. 7:457, 2008.
- Farrell PM, et al. Evidence on improved outcomes with early diagnosis of cystic fibrosis through neonatal screening: enough is enough! *J Pediatr*. 147:30S, 2005.
- Fischer R, et al. Lung disease severity, chronic inflammation, iron deficiency, and erythropoietin response in adults with cystic fibrosis. *Pediatr Pulmonol*. 42:1193, 2007.
- Geukers VG, et al. Short-term protein intake and stimulation of protein synthesis in stunted children with cystic fibrosis. *Am J Clin Nutr*. 81:605, 2005.
- Grasemann H, et al. Oral L-arginine supplementation in cystic fibrosis patients: a placebo-controlled study. *Eur Respir J*. 25:62, 2005.
- Grey V, et al. Prevalence of low bone mass and deficiencies of vitamins D and K in pediatric patients with cystic fibrosis from 3 Canadian centers. *Pediatrics*. 122:1014, 2008.
- Hart N, et al. Nutritional status is an important predictor of diaphragm strength in young patients with cystic fibrosis. *Am J Clin Nutr*. 80:1201, 2004.
- Hudson VM. New insights into the pathogenesis of cystic fibrosis: pivotal role of glutathione system dysfunction and implications for therapy. *Treat Respir Med*. 3:353, 2004.
- Innes SM, et al. Choline-related supplements improve abnormal plasma methionine-homocysteine metabolites and glutathione status in children with cystic fibrosis. *Am J Clin Nutr*. 85:702, 2007.
- Koscik RL, et al. Cognitive function of children with cystic fibrosis: deleterious effect of early malnutrition. *Pediatrics*. 113:1549, 2004.
- Lamireau T, et al. Epidemiology of liver disease in cystic fibrosis: a longitudinal study. *J Hepatol*. 41:920, 2004.
- Levy H, et al. Inflammatory markers of lung disease in adult patients with cystic fibrosis. *Pediatr Pulmonol*. 42:256, 2007.
- McPhail GL, et al. Improvements in lung function outcomes in children with cystic fibrosis are associated with better nutrition, fewer chronic pseudomonas aeruginosa infections, and dornase alfa use. *J Pediatr*. 153:752, 2008.
- Nick JA, Rodman DM. Manifestations of cystic fibrosis diagnosed in adulthood. *Curr Opin Pulm Med*. 11:513, 2005.
- Oliver MR, et al. Factors affecting clinical outcome in gastrostomy-fed children with cystic fibrosis. *Pediatr Pulmonol*. 37:324, 2004.
- Powers SW, et al. A comparison of food group variety between toddlers with and without cystic fibrosis. *J Hum Nutr Diet*. 17:523, 2004.
- Powers SW, et al. Randomized clinical trial of behavioral and nutrition treatment to improve energy intake and growth in toddlers and preschoolers with cystic fibrosis. *Pediatrics*. 116:1442, 2005.
- Quittner AL, et al. Prevalence and impact of depression in cystic fibrosis. *Curr Opin Pulm Med*. 14:582, 2008.
- Vanderwel M, Hardin DS. Growth hormone normalizes pubertal onset in children with cystic fibrosis. *J Pediatr Endocrinol Metab*. 19:237, 2006.
- Zhang Z, Lai HJ. Comparison of the use of body mass index percentiles and percentage of ideal body weight to screen for malnutrition in children with cystic fibrosis. *Am J Clin Nutr*. 80:982, 2004.

## INTERSTITIAL LUNG DISEASE

### NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: Cagle PT, MD. *Color Atlas and Text of Pulmonary Pathology*. Philadelphia: Lippincott Williams & Wilkins, 2005.



### DEFINITIONS AND BACKGROUND

Interstitial lung disease (ILD) is a general term that includes a variety of chronic lung disorders, sometimes also known as “interstitial pulmonary fibrosis.” In ILD, the lung tissue is damaged; the walls of the air sacs in the lung become inflamed; and, finally, scarring (fibrosis) occurs in the interstitium (tissue between the air sacs). The lung becomes stiff.

Although the histologic patterns of ILD in children and adults share similar features, important differences exist in etiology, clinical manifestations, and outcome (Young et al, 2008). Causes of adult ILD include environmental exposure to inorganic dust (such as silica) or organic dust (such as animal or bacterial proteins); exposure to gases, fumes, or poisons; or medical conditions such as sarcoidosis, scleroderma, rheumatic arthritis, and lupus. Agricultural workers also can be affected, with moldy hay causing allergic reactions in a disorder known as Farmer’s Lung.

Breathlessness during exercise and dry cough can be the first symptoms. Other symptoms vary in severity. Further testing is usually recommended to identify the specific type

of ILD a person has; some have known causes and some have unknown causes (idiopathic). The course of these diseases is unpredictable.

Some ILDs improve with medication if treated when inflammation occurs. Inflammation of these parts of the lung may heal or may lead to permanent scarring of the lung tissue. Fibrosis results in scarring and permanent loss of that tissue's ability to transport oxygen. The level of disability that a person experiences depends on the amount of scarring of the tissue. Oxygen may be needed; some patients need it all of the time, and others need it only during sleep and exercise.

A pulmonary rehabilitation program is often recommended for education, exercise conditioning, breathing retraining, energy-saving techniques, respiratory therapy, nutritional counseling, and psychosocial support. Lung transplantation has become an option for some patients.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Prevalence of ILDs is low in children. A total of 10–16% may be familial, with mutations in surfactant proteins or the SFTPC and ABCA3 gene (Nogee, 2006).

Clinical/History		Lab Work
Height, weight	Chest x-ray or CT scan	pCO <sub>2</sub> , pO <sub>2</sub>
Growth chart for height and weight	Pulmonary function test	Chol, Trig
BMI	Exercise function test	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>
Diet history	Clubbing of fingers	Alb
Fecal fat study	Dry cough	H & H
pH	Cyanosis	Serum Fe
Bronchoalveolar lavage (BAL)		WBC count
Lung biopsy		PT or INR
		Gluc
		Ca <sup>++</sup> , Mg <sup>++</sup>

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Underweight

**Assessment Data:** Poor dietary intake, underweight for height.

**Nutrition Diagnoses (PES):** Underweight (NC 3.1) related to inadequate energy intake as evidenced by low percentile height, weight 30%tile on growth chart and estimated intake approximately 500 calories below estimated needs.

**Interventions:** Enhance intake through adding protein and calories to casseroles and foods served. Educate about nutrient density and increasing calories. Counsel with tips about eating slowly and frequently.

**Monitoring and Evaluation:** Weight gain closer to desired BMI; improved intake of energy and nutrients.

## INTERVENTION



### OBJECTIVES

- Early identification and aggressive treatment are needed to lessen inflammation and prevent further lung damage.
- Remove the source of the problem, if known.
- Lessen the effect of complications.
- Maintain nutritional immunity as far as possible. Improve poor status.
- Provide nutritional repletion before surgery, if surgery is scheduled.



### FOOD AND NUTRITION

- If not contraindicated, offer 3–3.5 L fluid daily to liquefy secretions and to help lower temperature.
- A high-calorie, soft diet is recommended, especially if oxygen is used. Frequent, small meals may be beneficial.
- Discuss how to make mealtimes relaxed, especially if oxygen is required at the same time. Plan for longer mealtimes accordingly.
- A multivitamin–mineral supplement may be beneficial, especially for vitamins A, C, and E. Vitamin E reduces the extent of pulmonary damage in some types of ILD (Card et al, 2003).
- Ensure adequate potassium intake, as from fruits and juices.
- When possible, add more fiber to prevent constipation.
- TF at night may be beneficial if intake is poor during the day. A gastrostomy or transpyloric feeding tube may be desirable.

### Common Drugs Used and Potential Side Effects

- Oral prednisone or methylprednisone is frequently the first medication used to help decrease inflammation.
- Cyclophosphamide (Cytoxan) may be used if steroid therapy fails or if it is not possible. It reduces inflammation by killing some inflammatory cells and suppressing their function. Response to therapy may require up to 6 months or longer. In some cases, a combination of prednisone and cyclophosphamide is used with good results. Side effects include GI irritation, bladder inflammation, bone marrow suppression, infection, and blood disorders.
- Azathioprine (Imuran) is used if there are problems tolerating the side effects of the above medications. It is not as effective as cyclophosphamide, but side effects are more tolerable. Side effects include fever, skin rash, GI irritation, and blood disorders.
- Interferon has been tested in clinical trials with some promising results.

### Herbs, Botanicals, and Supplements

- Herbs and botanicals should not be used for ILD because there are no controlled trials to prove efficacy.





## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss how a balanced diet supports overall immunity and health status. Teach principles of the Food Guide Pyramid and the Dietary Guidelines.
- Teach how to incorporate antioxidants and related nutrients in the diet, especially if energy intake is low because of poor appetite.
- Influenza vaccine and pneumococcal pneumonia vaccine are both recommended for people with ILD.
- Rehabilitation and education programs may help some people. Local support groups have benefited people with ILD and their family members and friends.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- Children's Interstitial Lung Disease  
<http://emedicine.medscape.com/article/1003631-treatment>
- Interstitial Lung Disease Program  
<http://www.nationaljewish.org/a4.html>
- Medicine Net  
[http://www.medicinenet.com/interstitial\\_lung\\_disease/article.htm](http://www.medicinenet.com/interstitial_lung_disease/article.htm)

### INTERSTITIAL LUNG DISEASE—CITED REFERENCES

- Card JW, et al. Attenuation of amiodarone-induced pulmonary fibrosis by vitamin E is associated with suppression of transforming growth factor-beta1 gene expression but not prevention of mitochondrial dysfunction. *J Pharmacol Exp Ther*. 304:277, 2003.
- Nogee LM. Genetics of pediatric interstitial lung disease. 18:287, 2006.
- Young LR, et al. Usual interstitial pneumonia in an adolescent with ABCA3 mutations. *Chest*. 134:192, 2008.

# PNEUMONIA

## NUTRITIONAL ACUITY RANKING: LEVEL 1-2



### DEFINITIONS AND BACKGROUND

Pneumonia involves acute inflammation of the alveolar spaces of the lung. Lung tissue is consolidated as alveoli fill with exudate, usually after a cold or the flu. To protect against pneumonia, dental and oral health care are important. Dental plaque germs may be inhaled and may lead to onset of pneumonia; regular tooth brushing, flossing, and dental check-ups are recommended (El-Solh et al, 2004).

A productive cough that is painful and incessant (generally with green/yellow sputum that progresses to pink, brown, or rust color) may be indicative. Pneumonia may be classified as community acquired, hospital acquired, or atypical. Table 5-10 describes the common types of pneumonia. The most common form is community-acquired pneumococcal pneumonia. With treatment, most types of bacterial pneumonia can be cured within 1-2 weeks; viral pneumonia may last longer. Mycoplasmal pneumonia resolves in 4-6 weeks. Before antibiotics, pneumonia caused many deaths in elderly individuals; it now ranks sixth among causes of death in the United States.

People at high risk for pneumonia include the elderly; the very young; those with COPD, diabetes mellitus, congestive heart failure, sickle cell anemia, AIDS, or asthma; and people undergoing cancer therapy or organ transplantation. Nursing home residents have chronic medical conditions that gradually lead to “decompensation” in functional status, nutritional status, and pulmonary clearance. Elderly patients with low body weight and hypoalbuminemia are more likely to die from pneumonia than healthy patients.

Inflammation may cause low serum albumin levels in many pneumonia patients. GSH is the primary antioxidant that lines alveolar space; selenium, vitamins E and C may be

beneficial. However, supplementation alone will not prevent pneumonia in well-nourished older individuals (Merchant et al, 2004).

Enteral feeding provides nutrients for patients who require endotracheal tubes and mechanical ventilation. There is a presumed increase in the risk of ventilator-associated pneumonia (VAP) with TF, but this is not always true.

Pneumonia due to immune system suppression and membrane damage induced by oxidative stress suggest that sufficient fatty acid intake may be useful in the nutritional repletion of such patients with pneumonia. The American Dietetic Association previously recommended 3 MNT visits for persons who have pneumonia.



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Genes that have inflammatory molecules such as tumor necrosis factor, interleukin-10, and angiotensin-converting enzyme may play a role in susceptibility to pneumonia. Population studies are needed.

<b>Clinical/History</b>	BMI	Chills, fever
Height	I & O	(102–106°F),
Weight	Diet history	delirium
	BP	Pleuritic pain

TABLE 5-10 Types of Pneumonia

Type	Description
Allergic	From sensitivity to dust or pollen.
Aspiration	From swallowing a foreign substance. The gastric volume predisposing to aspiration is larger than 30 mL (Kalinowski and Kirsch, 2004). Just a few hours with “nothing by mouth” helps prepare for surgery because risk of aspiration is lower than previously believed. Ventilator-associated pneumonia, a common and serious complication in critically ill patients who require a ventilator, results from pneumonia occurring >48 hours after endotracheal intubation (Parker and Heyland, 2004). It is caused by microaspiration of contaminated oropharyngeal or gastrointestinal secretions into the airways (Parker and Heyland, 2004).
Bacterial	From bacteria normally present in mouth/throat. Quick onset with high fever and rapid breathing. Several bacteria may be relevant. <i>Streptococcus pneumoniae</i> causes about 25% of bacterial types. <i>Mycoplasma</i> causes walking pneumonia, notorious for sore throat and headache in addition to the usual symptoms; causes about 20% of all kinds of pneumonia. When pneumonia is due to pertussis (whooping cough), long coughing spells, turning blue from lack of air, and making a classic “whoop” sound when trying to take a breath will occur. <i>Haemophilus influenzae</i> type b (Hib) is America’s most common cause of bacterial meningitis; it is also an agent of pneumonia.
Chemical	From accidental inhalation of toxic fumes and chemicals, often in the workplace or when using cleaning agents such as bleach in a closed space.
Healthcare-associated pneumonia	Pneumonia in any patient who has been hospitalized in an acute care hospital for 2 or more days within the past 90 days; residents of a nursing home or long-term care facility; recipients of recent intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days; or patients who have attended a dialysis clinic.
Hypostatic	In bedridden persons, usually elderly individuals.
<i>Pneumocystis carinii</i> pneumonia	Caused by a fungus, primarily in AIDS patients.
Viral	More common; leads to about 50% of pneumonia cases. Symptoms appear more gradually; less severe than bacterial form. Wheezing is common in this type. Adenoviral infections often affect infants and young children. Other viruses that can cause pneumonia include rhinovirus, influenza, respiratory syncytial virus, and parainfluenza virus (croup).

Difficult, painful respirations	Tachypnea, tachycardia	pCO <sub>2</sub> , pO <sub>2</sub>
SOB, rales, rhonchi	Anorexia, malaise	Na <sup>+</sup> , K <sup>+</sup>
Bronchoscopy	Abdominal distention	Ca <sup>++</sup> , Mg <sup>++</sup>
Productive cough (purulent, green, or rust)	Anxiety, restlessness	Alb, transthyretin
Respiratory rate (increase)	Cyanosis of nail beds	CRP
Fatigue, weakness	<b>Lab Work</b>	H & H
	WBCs (increased)	Serum Fe, ferritin
		Transferrin
		Gluc
		BUN, Creat

## INTERVENTION



## OBJECTIVES

- Prevent or correct dehydration.
- Relieve breathing difficulty and discomfort. Oxygenate all tissues.
- Prevent weight loss from a hypermetabolic state.
- Support diet with adequate antioxidants and nutrient-dense foods.
- Avoid additional infections; prevent sepsis and multiple organ dysfunction syndrome.
- In convalescent stage, avoid constipation.



## FOOD AND NUTRITION

- If not contraindicated, offer 3 L or more of fluid daily to liquefy secretions and to help lower elevated temperature.
- Progress, as tolerated, to a high-calorie diet. If overweight, allow normal calorie intake for age and sex.
- Early enteral nutrition, properly administered, can decrease upper GI intolerance and nosocomial pneumonia (Kompan et al, 2004).
- Frequent, small meals and a soft diet may be tolerated better.
- A multivitamin–mineral supplement may be beneficial, especially including selenium and vitamins E and C. Vitamin A is needed to keep mucous membranes healthy.

## SAMPLE NUTRITION CARE PROCESS STEPS

## Inadequate Fluid Intake

**Assessment Data:** Dehydration, rapid breathing, poor skin turgor, I & O records.

**Nutrition Diagnoses (PES):** Inadequate fluid intake related to fever of 103°F, and pneumonia as evidenced by signs of dehydration and low I & O.

**Interventions:** Encourage intake of fluids at all meals and between meals as well. Keep water or a beverage on hand within easy reach.

**Monitoring and Evaluation:** Improved I & O records; reduction of fever; normal skin turgor.

- When possible, add more fiber to prevent constipation.
- Ensure adequate potassium intake, as from fruits and juices.

### Common Drugs Used and Potential Side Effects

- A 7-day course of low-dose hydrocortisone infusion speeds recovery from community-acquired pneumonia (CAP) and prevents complications due to sepsis (Confalonieri et al, 2005).
- Antibiotics, such as clarithromycin (Biaxin), are used in bacterial pneumonia. Nausea, diarrhea, and abdominal pain can occur.
- Telithromycin (Ketek) is used for the treatment of infections caused by bronchitis, bacterial sinusitis, and CAP.
- Analgesics are used to reduce pain and antipyretics are used to lessen fever.
- Cephalosporins are often useful for nursing home-acquired pneumonia (Muder et al, 2004). Ceftriaxone may be used against MRSA, *Enterococcus faecalis*, *Enterobacteriaceae*, and *Pseudomonas aeruginosa*.

### Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of herbs or botanicals, such as echinacea, honeysuckle, garlic, dandelion, astragalus, and baikal skullcap in pneumonia patients.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of diet and fluid intake in recovery.
- Loss of swallowing function can lead to dehydration, malnutrition, pneumonia, and reduced quality of life for senior citizens; increasing strength of head and neck musculature can be recommended (Ney et al, 2009). In hypostatic pneumonia, occupational or physical therapy may be beneficial.
- Fruit and vegetable juices add calories, fluid, and sometimes fiber to the diet and can be available at bedside.
- Routine immunizations are available against *Haemophilus influenzae* and pertussis beginning at 2 months of age; pertussis immunization is the “P” part of the routine DtaP or DTP.

- Vaccines are now also given against the pneumococcus organism (PCV), a common cause of bacterial pneumonia. Although the polysaccharide pneumococcal vaccine (PPV) does not prevent CAP, it might still improve outcomes in those who develop pneumonia (Johnstone et al, 2007).
- Flu vaccines are recommended for individuals with chronic illnesses such as heart and lung disorders, including asthma. Premature infants may need protection against respiratory syncytial virus (RSV). Individuals who have HIV infection may need protection against *Pneumocystis carinii*.
- Protect people who have pneumonia from others who have respiratory tract infections, such as the common cold.

### Patient Education—Foodborne Illness

- Careful food handling will be important.
- Hand washing is key as well, especially after sneezing and coughing and before eating.

### For More Information

- American Lung Association—Pneumonia  
<http://www.lungusa.org/diseases/lungpneumoni.html>
- KidsHealth  
[http://kidshealth.org/kid/ill\\_injure/sick/pneumonia.html](http://kidshealth.org/kid/ill_injure/sick/pneumonia.html)
- Medicine Net  
<http://www.medicinenet.com/pneumonia/article.htm>

### PNEUMONIA—CITED REFERENCES

- Confalonieri M, et al. Hydrocortisone infusion for severe community-acquired pneumonia: a preliminary randomized study. *Am J Respir Crit Care Med*. 171:242, 2005.
- El-Solh AA, et al. Colonization of dental plaques: a reservoir of respiratory pathogens for hospital-acquired pneumonia in institutionalized elders. *Chest*. 126:1575, 2004.
- Johnstone J. Effect of pneumococcal vaccination in hospitalized adults with community-acquired pneumonia. *Arch Intern Med*. 167:1938, 2007.
- Kalinowski CP, Kirsch JR. Strategies for prophylaxis and treatment for aspiration. *Best Pract Res Clin Anaesthesiol*. 18:719, 2004.
- Kompan L, et al. Is early enteral nutrition a risk factor for gastric intolerance and pneumonia? *Clin Nutr*. 23:527, 2004.
- Merchant AT, et al. Vitamin intake is not associated with community-acquired pneumonia in U.S. men. *J Nutr*. 134:439, 2004.
- Muder RR, et al. Nursing home-acquired pneumonia: an emergency department treatment algorithm. *Curr Med Res Opin*. 20:1309, 2004.
- Ney DM, et al. Senescent swallowing: impact, strategies, and interventions. *Nutr Clin Pract*. 24:395, 2009.
- Parker CM, Heyland DK. Aspiration and the risk of ventilator-associated pneumonia. *Nutr Clin Pract*. 19:597, 2004.

## PULMONARY EMBOLISM

### NUTRITIONAL ACUITY RANKING: LEVEL 1–2



### DEFINITIONS AND BACKGROUND

A pulmonary embolism is caused by a partial or complete occlusion of a pulmonary artery from a blood clot from another part of the body that has found its way to the lung. The condition can be life-threatening. Sudden, sharp substernal pain, SOB, cyanosis, pallor, faintness, fever, hypoten-

sion, and wheezing can occur, sometimes followed by right heart failure. Approximately 10% of patients suffer some form of tissue death or pulmonary infarction.

Venous thrombosis most often starts in the calf veins and moves on to the lung. Thrombosis in the veins is triggered by venostasis, hypercoagulability, and vessel wall inflammation (the Virchow triad).

Common causes include recent surgery, fractures, immobility, burns, obesity, chemotherapy, old age, heart failure, polycythemia, ulcerative colitis, homocystinemia, and even pregnancy. It is actually one of the primary concerns during pregnancy (Stone and Morris, 2005). Hormone replacement therapy (HRT) is no longer recommended for women after menopause because of the increased risk for pulmonary embolism (Hillman et al, 2004).

Massive pulmonary embolism causes hypotension, with a systolic arterial pressure less than 90 mm Hg; mortality ranges from 30% to 60%. Nonmassive pulmonary embolism presents with systolic arterial pressure greater than or equal to 90 mm Hg and is much more common. Oxygen therapy is always initiated, and fibrinolytic therapy is the primary mode of treatment.

Interesting studies have been conducted to evaluate the role of diet on embolism. In the Iowa cohort study of older women, greater intake of alcohol was associated with a lower risk of incident thromboembolism; no associations were seen with “Western” or “Prudent” dietary patterns, fruit, vegetables, dairy, meat, refined grains, whole grains, regular soda, vitamins E, vitamin B<sub>6</sub>, vitamin B<sub>12</sub>, folate, omega-3 fatty acids, or saturated fat (Lutsey et al, 2009). In the Longitudinal Investigation of Thromboembolism Etiology study, greater intake of fish, fruit, and vegetables were noted as beneficial (Steffen et al, 2007). Clearly, more research is needed.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Hereditary factors that may produce a hypercoagulable state include Antithrombin III deficiency, Protein C or Protein S deficiency, Factor V Leiden, plasminogen or fibrinogen abnormalities.

Homocysteine, factor VIII and von Willebrand factor levels are risks for embolism and they are influenced by dietary intake (Steffen et al, 2007).

Clinical/History	Arterial blood gases	Low oxygen saturation (hypoxia)
Height	Diaphoresis	Perfusion scans
Weight	Temperature (fever)	CT pulmonary angiography
BMI	I & O	Cyanosis
Sudden SOB	Sharp chest pain	
Hemoptysis with pink, foamy mucus	ECG	<b>Lab Work</b>
Tachypnea (respiratory rate >16/min)	Echocardiography	Alb,
Tachycardia (heart rate >100/min)	Doppler ultrasound	transferrin
	Cardiac murmur	Na <sup>+</sup> , K <sup>+</sup>
	Palpitations	Ca <sup>++</sup> , Mg <sup>++</sup>
		PT or INR

WBCs  
(increased)  
Troponin  
(high?)

H & H, Serum Fe  
Elevated BNP?  
Liver function tests

Homocysteine  
Folic acid and B<sub>12</sub> levels

## INTERVENTION



### OBJECTIVES

- Prevent right-sided heart failure, atelectasis, and bleeding.
- Stabilize PT and INR if warfarin (Coumadin) is used.
- Maintain lung function through higher antioxidant intake.
- Normalize body temperature where there is fever.
- Replenish nutrients depleted by respiratory distress.
- Oxygenate tissues.
- Eliminate edema when present.



## FOOD AND NUTRITION

- Use a regular or high-calorie diet; use a low-sodium diet for patients with edema.
- Increase fluid intake as tolerated.
- Control vitamin K in diet when PT cannot be stabilized.
- Small meals may be needed.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Involuntary Weight Loss

**Assessment Data:** Poor oral intake and weight loss of 15 lb in 6 months; swallowing difficulty; decreased appetite and frequent coughing during meals.

**Nutrition Diagnoses (PES):** Involuntary weight loss related to poor oral intake, coughing at meals and swallowing difficulty as evidenced by 15 lb weight loss in 6 months.

### Interventions:

ND 4.5 Patient to use oxygen via nasal cannula during meals to assist with breathing and swallowing and to improve energy levels.

E2.2 Basic education tips on managing discomfort related to SOB by using—five to six small meals throughout the day that are easily prepared; consume main meal early in the day; drink fluids between meals; add protein and calories into meal items.

RC2.2 Refer to local meals on wheels program for home-delivered meals 5 days per week.

**Monitoring and Evaluation:** Improvement in oral intake and weight status; fewer complaints of fatigue at mealtime; better appetite.



- Provide sufficient antioxidants such as vitamins C and E and selenium. A diet including more plant foods, alcohol, and fish and less red and processed meat may be suggested (Lutsey et al, 2009; Steffen et al, 2007).

### Common Drugs Used and Potential Side Effects

- Rivaroxaban is a novel oral direct factor Xa inhibitor for prophylaxis in total knee and hip replacements, with few side effects and low potential for drug–food interactions (Chen and Lam, 2009). Liver enzymes should be checked during use.
- Heparin slows down clot progression and reduces risk of further clots. Warfarin (Coumadin) increases clotting times by thinning the blood. If problems in stabilizing the PT exist, controlled vitamin K may be needed. Use stable amounts of green leafy vegetables and fish.
- Fibrinolytic therapy is a primary treatment. Alteplase is generally infused over several hours. Tissue plasminogen activator (tPA) is also available for thrombolysis.
- Estrogen-containing contraceptives and hormone replacements may promote an embolism in susceptible women. Close medical monitoring is advised.

### Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of herbs or botanicals in pulmonary embolism.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain sources of vitamin K in the diet. Therapy often continues for 3–6 months.
- Individuals on long airline flights should try to obtain some physical activity to prevent embolism.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

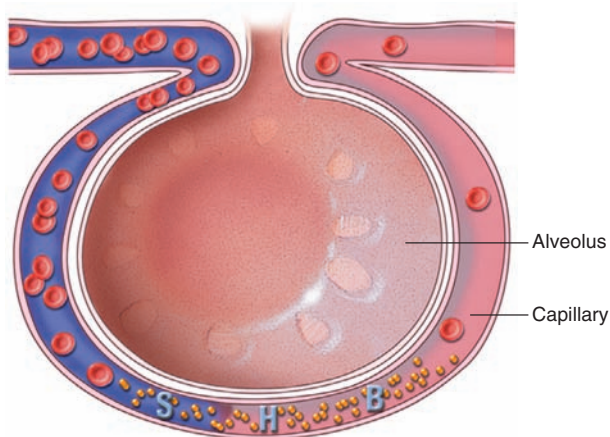
- E-medicine  
<http://www.emedicine.com/EMERG/topic490.htm>
- Mayo Clinic  
<http://www.mayoclinic.com/health/pulmonary-embolism/DS00429/>
- Web MD  
<http://www.webmd.com/a-to-z-guides/pulmonary-embolism-topic-overview>

### PULMONARY EMBOLISM—CITED REFERENCES

- Chen T, Lam S. Rivaroxaban: an oral direct factor xa inhibitor for the prevention of thromboembolism. *Cardiol Rev.* 17:192, 2009.
- Hillman JJ, et al. The impact of the Women's Health Initiative on hormone replacement therapy in a Medicaid program. *J Womens Health (Larchmt).* 13:986, 2004.
- Lutsey PL, et al. Diet and incident venous thromboembolism: the Iowa Women's Health Study. *Am Heart J.* 157:1081, 2009.
- Steffen LM, et al. Greater fish, fruit, and vegetable intakes are related to lower incidence of venous thromboembolism: the Longitudinal Investigation of Thromboembolism Etiology. *Circulation.* 117:188, 2007.
- Stone SE, Morris TA. Pulmonary embolism during and after pregnancy. *Crit Care Med.* 33:294S, 2005.

## RESPIRATORY DISTRESS SYNDROME

### NUTRITIONAL ACUITY RANKING: LEVEL 3–4



Phase 1. Injury reduces normal blood flow to the lungs. Platelets aggregate and release histamine (H), serotonin (S), and bradykinin (B).

Asset provided by Anatomical Chart Co.



### DEFINITIONS AND BACKGROUND

RDS may occur as part of systematic inflammatory response syndrome (SIRS), affecting approximately 70% of patients in the ICU. Acute respiratory distress syndrome (ARDS) develops within 24–48 hours in patients who have sepsis or who are critically ill, in shock, or severely injured. Other causes include infectious pneumonia, aspiration of food into the lung, several blood transfusions, pulmonary embolism, chest injury, burns, near drowning, cardiopulmonary bypass surgery, pancreatitis, overdose of drugs such as heroin, methadone, or aspirin.

ARDS has three pathologic stages: exudative, proliferative, and fibrotic. Patients often have pulmonary edema but have normal left atrial and pulmonary venous pressures.

In infants, RDS occurs in premature or low birth weight babies as hyaline membrane disease. Such babies are often born to mothers who have diabetes. Surfactant treatment may be of significant benefit in newborn infants with respiratory compromise (Finer, 2004).

One of the most common causes of ARDS in adults is sepsis. Here, a high-fat diet or formula with EPA may be beneficial. In ARDS, an overwhelming inflammatory response damages the endothelial-alveolar units, reducing oxygen diffusion and increasing pulmonary workload (Singer and Shapiro, 2009). Specialized enteral formulas may be beneficial adjunctive therapy by reducing lung inflammation and improving oxygenation (Malik and Zaloga, 2010; Priestley and Helfaer, 2004).

Indirect calorimetry (IC) accurately estimates a patient's energy expenditure; this helps the health care team when there is weaning failure.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** ARDS may have a relationship with tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin-b (IL-b), interleukin 10 (IL-10), and soluble intercellular adhesion molecule 1 (sICAM-1).

Clinical/History	Lab Work	
Height	Complete	Transferrin
Weight	blood count	pCO <sub>2</sub> , pO <sub>2</sub>
BMI	(CBC)	Transthyretin
Growth profile	Low blood	Na <sup>+</sup> , K <sup>+</sup>
Diet history	pH (acidic)	Ca <sup>++</sup> , Mg <sup>++</sup>
IC	H & H	Serum
I & O	Serum Fe,	phosphorus
BP	ferritin	BUN, Creat
Temperature		
RQ		

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Excessive Enteral Nutrition

**Assessment Data:** Ventilator dependency for acute respiratory distress, ICU admission, inability to consume oral food and beverages, IC indicates energy needs as 1400 kcal/d; current TF order for 2000 kcal

**Nutrition Diagnoses (PES):** Excessive enteral nutrition related to overfeeding with order for 2000 kcal as evidenced by IC results suggesting 1400 kcal as sufficient.

**Interventions:** Nutrition prescription should change to match energy needs. Educate nutrition support team about results of IC (1400 kcal vs. current order for 2000 kcal). Suggest immunomodulating diet (IMD) formula supplemented with fructooligosaccharides (FO).

**Monitoring and Evaluation:** Improved arterial blood gases; enteral nutrition formula tolerated; able to gradually wean from ventilator.

## INTERVENTION



### OBJECTIVES

- Identify the cause and remove the ongoing insult. Promote rapid recovery and oxygenation of tissues; support ventilator management.
- Prevent relapse. Avoid secondary insults through aggressive immune surveillance, complete nutrition, and adequate oxygen delivery.
- Counteract side effects of medications as ordered.
- Replace essential fatty acids, carnitine, and other nutrients.
- Prevent malnutrition, which depresses CNS output for ventilatory drive. Starvation decreases the desire to breathe, causing an abnormal breathing pattern, pneumonia, and atelectasis. Muscle mass (including diaphragm) varies with body weight, and refeeding may take 2–3 weeks.
- Prevent overfeeding (hepatic dysfunction, fatty liver, and CO<sub>2</sub> overproduction) and underfeeding (morbidity, mortality, and decreased response to therapy). Avoid refeeding syndrome.
- Prevent fluid overload.
- Support lung function, which is found to be better with higher antioxidant intake levels (Singer et al, 2006).
- An IMD that is supplemented with FO improves the outcome of medical ICU patients with SIRS/sepsis and ARDS (Marik and Zaloga, 2010).



### FOOD AND NUTRITION

- Provide parenteral fluids and oxygen as needed.
- Progress, when possible, to oral feedings. Use TPN only if GI tract is nonfunctional. TPN-induced changes in CO<sub>2</sub> production occur if overfed (Plurad et al, 2009).
- For calories, use 30–35 kcal/kg. Nonprotein calories should come from 50% glucose and 50% lipid.
- Increased fat may be required to normalize the RQ. Fat also adds extra energy intake and palatability to the diet.
- Ensure adequate provision of EFA. Low linoleic acid status in critically ill RDS infants may require IVs with a fat emulsion added.
- Increase intake of omega-3 fatty acids, especially EPA and GLA (Singer et al, 2006). Enteral administration of fish oil, antioxidants and arginine improves oxygenation and clinical outcomes (Singer and Shapiro, 2009). Provide vitamins C and E and selenium at slightly higher than RDA levels, and fat-soluble vitamins in water-miscible form if necessary.
- Inositol supplementation promotes survival of premature infants with RDS (Howlett and Ohlsson, 2003).

### Common Drugs Used and Potential Side Effects

- Heparin or warfarin (Coumadin) may be used as a blood thinner.

- Ventilator-dependent surgical patients receiving oxandrolone have prolonged courses of mechanical ventilation; oxandrolone may enhance collagen deposition and fibrosis in the later stages of ARDS and thus delay recovery (Bulger et al, 2004).

## Herbs, Botanicals, and Supplements

- Use of n-3 PUFA targets the inflammatory response in ARDS (Singer and Shapiro, 2009).



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of fat intake on respiratory requirements. Fat decreases CO<sub>2</sub> production.
- Small, frequent feedings may be beneficial.
- Tight glucose control is needed.
- Prone positioning, especially for meals, is recommended.

## Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

## For More Information

- Acute Respiratory Distress Clinical Network  
<http://www.ardsnet.org/>
- ARDS Support  
<http://www.ards.org/>
- Medscape  
<http://emedicine.medscape.com/article/803573-overview>
- Respiratory Distress Syndrome  
[http://www.nlm.nih.gov/health/dci/Diseases/Ards/Ards\\_WhatIs.html](http://www.nlm.nih.gov/health/dci/Diseases/Ards/Ards_WhatIs.html)

## RESPIRATORY DISTRESS SYNDROME—CITED REFERENCES

- Bulger EM, et al. Oxandrolone does not improve outcome of ventilator dependent surgical patients. *Ann Surg*. 240:472, 2004.
- Finer NN. Surfactant use for neonatal lung injury: beyond respiratory distress syndrome. *Paediatr Respir Rev*. 5:S289, 2004.
- Howlette A, Ohlsson A. Inositol for respiratory distress syndrome in preterm infants. *Cochrane Database Syst Rev*. 4:CD000366, 2003.
- Marik PE, Zaloga GP. Immunonutrition in high-risk surgical patients: a systematic review and analysis of the literature. *JPEN J Parenter Enteral Nutr*. 34:378, 2010.
- Plurad D, et al. A 6-year review of total parenteral nutrition use and association with late-onset acute respiratory distress syndrome among ventilated trauma victims. *Injury*. 40:511, 2009.
- Priestley MA, Helfaer MA. Approaches in the management of acute respiratory failure in children. *Curr Opin Pediatr*. 16:293, 2004.
- Singer P, et al. Benefit of an enteral diet enriched with eicosapentaenoic acid and gamma-linolenic acid in ventilated patients with acute lung injury. *Crit Care Med*. 34:1033, 2006.
- Singer P, Shapiro H. Enteral omega-3 in acute respiratory distress syndrome. *Curr Opin Clin Nutr Metab Care*. 12:123, 2009.

# RESPIRATORY FAILURE AND VENTILATOR DEPENDENCY

## NUTRITIONAL ACUITY RANKING: LEVEL 4



Adapted from: Springhouse. *Lippincott's Visual Encyclopedia of Clinical Skills*. Philadelphia: Wolters Kluwer Health, 2009.



## DEFINITIONS AND BACKGROUND

RF involves ineffective gas exchange across the lungs by the respiratory system. Arterial blood gases should be used to determine the presence of RF and Table 5-11 lists common causes. Acute respiratory failure (ARF) involves sudden

absence of respirations, with confusion or unresponsiveness and failure of pulmonary gas exchange mechanism. Chronic pulmonary disease or an acute injury can cause ARF, which requires mechanical ventilation.

**TABLE 5-11 Causes of Respiratory Failure**

Symptom	Cause
Airway obstruction	Chronic bronchitis, emphysema, bronchiectasis, cystic fibrosis, asthma, bronchiolitis, inhaled particles, subglottic stenosis, tumor, laryngeal edema
Poor breathing	Obesity, sleep apnea, drug intoxication, trauma, hypothyroidism
Neuromuscular disease	Myasthenia gravis, muscular dystrophy, polio, Guillain-Barré syndrome, botulism, polymyositis, stroke, amyotrophic lateral sclerosis, spinal cord injury
Abnormality of lung tissue	Acute respiratory distress, drug reaction, pulmonary fibrosis, fibrosing alveolitis, widespread tumors, radiation therapy, sarcoidosis, burns
Abnormality of chest wall	Kyphoscoliosis, chest wound

Mechanical ventilation can be delivered with a plastic tube inserted through the nose or mouth into the trachea. A tracheostomy is safer and more comfortable for long-term ventilation for either pure oxygen or a mixture of oxygen and air.

Anabolic and catabolic hormones, muscle work, and nutritional status affect skeletal muscle mass and muscle strength. Substrate plus muscle work help to stimulate protein synthesis. Randomized controlled trials comparing early aggressive use of enteral nutrition with delayed, less-aggressive use of enteral nutrition suggest that providing early, aggressive enteral nutrition promotes improved clinical outcomes (Stapleton et al, 2007). In starvation, respiratory muscles are catabolized to meet energy needs; refeeding helps ventilatory response. Enteral feedings started within 24–48 hours may reduce length of time on a ventilator.

Daily screening of ventilator patients is recommended, followed by trials of spontaneous breathing. The process of weaning takes a few days and requires proper refeeding. Table 5-12 identifies ventilator-dependency feeding stages. The length of ventilator dependency time relates to energy and CHO intake. Aggressive immune surveillance, nutritional support, and fluid management are critical (Michaels, 2004).

Older patients are more at risk for RF and may be harder to wean (Sevransky and Haponik, 2003). Attention must be paid to factors such as electrolytes, infections, anemia, heart failure, medications, or hypothyroidism (Datta and Scalise, 2004). Use of an evidence-based nutrition support protocol improves the likelihood of meeting nutritional requirements (Mackenzie et al, 2005). Patients with RF often have 30% or higher increase in oxygen requirements. Too much oxygen can be damaging, though, so careful monitoring is needed. Lung function is found to be better with higher antioxidant levels.

**TABLE 5-12 Ventilatory-Dependency Feeding Stages**

Stage	Objectives and Actions
Intubation/ Acute Phase	Replenish muscle glycogen stores and reverse catabolism. Enteral nutrition by day 3; parenteral by day 7 if GI tract not functioning.
Prewaning	Maintain positive nitrogen balance, improve visceral protein stores, improve lean body mass, and promote weight gain. Evaluate albumin or prealbumin levels.
Weaning 1–4 weeks	Provide energy substrates to cover needs of respiratory muscles that are working harder; minimize CO <sub>2</sub> production. Be careful not to overfeed. Check prealbumin levels and monitor fatigue. Get a Speech evaluation for swallowing. Assess for gastrostomy if needed.
Rehabilitation	Maintain nutrient needs despite anorexia or dysphagia; support anabolism. Maintain enteral nutrition at night until oral intake meets needs. If aspiration risk remains, continue gastrostomy.

Sources: Delmore BA. Levine's framework in long-term ventilated patients during the weaning course. *Nurs Sci Q*. 19:247, 2006.  
Matarese LE, Gottschlich M. Contemporary nutrition support practice: a clinical guide. St Louis: Elsevier, 2003:398–399.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Mutations in the ABCA3 transporter have been associated with childhood respiratory disease; there is a role for surfactant, a mixture of phospholipids, cholesterol, and hydrophobic proteins (Fitzgerald et al, 2007; Shulenin et al, 2004).

Clinical/History	Temperature (fever?)	H & H Serum Fe, ferritin
Height	Forced vital capacity (FVC)	Na <sup>+</sup> , K <sup>+</sup>
Weight	Skinfold thickness	Ca <sup>++</sup> , Mg <sup>++</sup>
BMI		WBC (elevated)
Resting energy expenditure (REE) from IC	<b>Lab Work</b>	Chol, Trig
Diet history	Hypophos- phatemia (can cause ARF)	Transferrin
I & O	Gluc	Thyroid tests
PaO <sub>2</sub> < 60 mm Hg	Urinary Gluc	CRP
PaCO <sub>2</sub> > 50 mm Hg	Transthyretin (decreased)	pH (acidemia below 7.4, alkalemia above 7.4)
Respiratory rate	TLC (decreased)	
RQ		
BP		

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Excessive CHO Intake

**Assessment Data:** IC indicates need for 1675 kcals daily. TF provides 65% CHO, 20% lipid, 15% protein; 2300 kcals total. ARF with ventilator dependency post motor vehicle accident. BMI 24. Serum glucose levels 350, 250, 301 on 3 days.

**Nutrition Diagnoses (PES):** Excessive CHO intake related to high CHO and energy content of TF formula as evidenced by ventilator dependency, elevated CO<sub>2</sub> levels, and inability to wean.

**Interventions:** Evaluate enteral needs and select TF product that has less CHO. Calculate energy, protein, and fluid needs with a new product and provide rate and amount using continuous drip equal to 1675 kcals daily. Monitor use of insulin and adjust according to serum glucose levels.

**Monitoring and Evaluation:** Gradual weaning from ventilator dependency. Improved blood gases and lower CO<sub>2</sub> production. Weight maintenance; BMI remaining at 24. Serum glucose within acceptable range.



**INTERVENTION****OBJECTIVES**

- Promote normalized nutritional intake despite hypermetabolic status of the patient and the prohibition of oral intake due to endotracheal tubes.
- Oxygenate tissues and relieve breathlessness; decrease CO<sub>2</sub> production.
- Monitor sensations of hunger when patients are unable to communicate their hunger and thirst.
- Prevent respiratory muscle dysfunction by ensuring that the patient is properly nourished.
- Provide intensive metabolic support with insulin therapy, an appropriate blood glucose target, nutrition risk assessment, early or combined enteral nutrition and PN, and close nutritional monitoring (Mechanick and Chiolerio, 2008).
- Counteract hypotension caused by positive-pressure ventilation, acidosis, or both.
- Provide nutritional substrates that will maintain surfactant production and LBM. Achieve or maintain weight; note that not all patients are malnourished.
- Prevent atelectasis, pulmonary infection, sepsis, glucose or lipid intolerance, multiple organ dysfunction syndrome, and aspiration.
- Alleviate GI complications, which are a concern with mechanical ventilation. Hypomotility and diarrhea are common.
- Protocol-driven weaning reduces use of mechanical ventilation (Dries et al, 2004; Graham and Kirbey, 2006). Adjust goals as appropriate.
- Maintain flexible approaches to patient requirements. Nutritional supplements containing selenium, vitamins, and antioxidants may provide needed support to shift from catabolic to anabolic, reduce free radicals, and quiet inflammation (Meltzer and Moitra, 2008).

**FOOD AND NUTRITION**

- Begin nourishing the patient as soon as possible to wean the patient from the ventilator. Start a TF of low osmolality slowly to avoid gastric retention or diarrhea. Advance gradually and use continuous administrations unless contraindicated. Do not add blue food coloring to feedings to detect aspirate in tracheal secretions (Kattelman et al, 2006).
- Ambulatory adults need about 30 kcal/kg daily. In ICU, the goal of 20–25 kcal/kg is sought; if it cannot be met, then combined enteral and PN should be considered to reduce the risk of complications and longer length of stay (Scurlock and Mechanick, 2008).
- Increased needs occur from labored breathing; monitor using IC. Use of specialty products such as Pulmocare or Respalor may be recommended, but they are not always necessary. Include 2% of total fat as essential fatty acids with some omega-3 fatty acids.
- Provide 1.2–1.5 g protein/kg/d (Mechanick and Chiolerio, 2008).
- While hypermetabolism and malnutrition are common, there is no need for supplemental PN to increase

caloric delivery in the early phase of critical illness (Stapleton et al, 2007). Monitor TPN carefully for complications such as pneumonia, refeeding syndrome from high-calorie loading, and increased CO<sub>2</sub> production.

- Patients with pulmonary edema should have their sodium intake reduced if needed. Include adequate protein in the diet to prevent additional fluid retention from lowered colloidal osmotic pressure.
- Supplement diet with a multivitamin supplement. Include antioxidant-rich foods for vitamins E, selenium, carotenoids, and vitamin C. Phosphorus and magnesium may be needed if stores are depleted.

**Common Drugs Used and Potential Side Effects**

- Bronchodilators, antibiotics, diuretics, or corticosteroids may be needed. Monitor side effects.
- For diarrhea, treatment depends on the cause. For *Clostridium difficile* infection, antibacterial therapy should be discontinued, if possible, and treatment with oral metronidazole should be initiated (Mutlu et al, 2003).

**Herbs, Botanicals, and Supplements**

- No clinical trials have proven efficacy for use of herbs or botanicals in RF.

**NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT**

- A daily calorie count may be needed to assess the patient's nutritional status.
- The greatest danger in using enteral nutrition is aspiration. Low-osmolality products are essential, as well as elevation of the head of the bed.
- Discuss early satiety, bloating, fatigue, dyspnea as related to food or TF intake.
- Delivery of enteral nutrition in patients receiving mechanical ventilation is interrupted by practices required for the care of these patients (O'Meara et al, 2008). Discharge planning for the ventilator patient to return home is ideal.

**Patient Education—Foodborne Illness**

- Careful food handling will be important. Hand washing is key as well.

**For More Information**

- Merck Manual—Respiratory Failure  
<http://www.merck.com/mmhe/sec04/ch055/ch055a.html>
- Medicine Net  
<http://www.medterms.com/script/main/art.asp?articlekey=10698>
- Respiratory Failure  
<http://www.med-help.net/AcuteRespiratoryFailure.html>

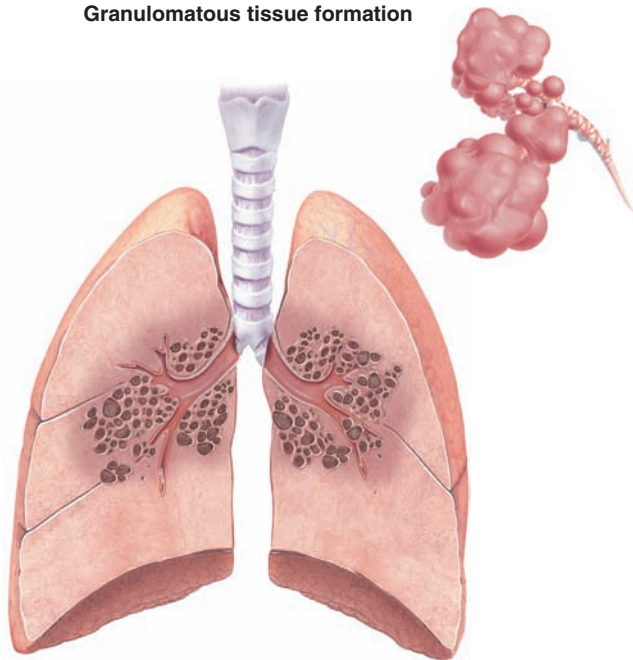
## RESPIRATORY FAILURE AND VENTILATOR DEPENDENCY—CITED REFERENCES

- Datta D, Scalise P. Hypothyroidism and failure to wean in patients receiving prolonged mechanical ventilation at a regional weaning center. *Chest*. 126:1307, 2004.
- Delmore BA. Levine's framework in long-term ventilated patients during the weaning course. *Nurs Sci Q*. 19:247, 2006.
- Dries DJ, et al. Protocol-driven ventilator weaning reduces use of mechanical ventilation, rate of early reintubation, and ventilator-associated pneumonia. *J Trauma*. 56:943, 2004.
- Fitzgerald ML, et al. ABCA3 inactivation in mice causes respiratory failure, loss of pulmonary surfactant, and depletion of lung phosphatidylglycerol. *J Lipid Res*. 48:621, 2007.
- Graham AS, Kirby AL. Ventilator management protocols in pediatrics. *Respir Care Clin N Am*. 12:389, 2006.
- Kattelman K, et al. Preliminary evidence for a medical nutrition therapy protocol: enteral feedings for critically ill patients. *J Am Diet Assoc*. 106:1226, 2006.
- Mackenzie SL, et al. Implementation of a nutrition support protocol increases the proportion of mechanically ventilated patients reaching enteral nutrition targets in the adult intensive care unit. *JPEN J Parenter Enteral Nutr*. 29(2):74, 2005.
- Matarese L, Gottschlich M. *Contemporary nutrition support practice: a clinical guide*, 2nd ed. St Louis: Elsevier, 2003:398–400.
- Mechanick JL, Chioloro R. Special commentary: a call for intensive metabolic support. *Curr Opin Clin Nutr Metab Care*. 11:666, 2008.
- Meltzer JS, Moitra VK. The nutritional and metabolic support of heart failure in the intensive care unit. *Curr Opin Clin Nutr Metab Care*. 11:140, 2008.
- Michaels AJ. Management of post traumatic respiratory failure. *Crit Care Clin*. 20:83, 2004.
- Mutlu GM, et al. Prevention and treatment of gastrointestinal complications in patients on mechanical ventilation. *Am J Respir Med*. 2:395, 2003.
- O'Meara D, et al. Evaluation of delivery of enteral nutrition in critically ill patients receiving mechanical ventilation. *Am J Crit Care*. 17:53, 2008.
- Scurlock C, Mechanick JL. Early nutrition support in the intensive care unit: a US perspective. *Curr Opin Clin Nutr Metab Care*. 11:152, 2008.
- Sevransky JE, Haponik EF. Respiratory failure in elderly patients. *Clin Geriatr Med*. 19:205, 2003.
- Shulenin S, et al. ABCA3 gene mutations in newborns with fatal surfactant deficiency. *N Engl J Med*. 350:1296, 2004.
- Stapleton RD, et al. Feeding critically ill patients: what is the optimal amount of energy? *Crit Care Med*. 35:535S, 2007.

## SARCOIDOSIS

### NUTRITIONAL ACUITY RANKING: LEVEL 1–2

#### Granulomatous tissue formation



Asset provided by Anatomical Chart Co.



### DEFINITIONS AND BACKGROUND

Sarcoidosis is a disease of undetermined origin with tiny patches of inflammation (granulomas) occurring in almost any organ. Pulmonary effects are most common. It develops most often between ages 20 years and 40 years, more often among women than men, and more commonly among Swedes, Danes, and African Americans. Sarcoidosis is more common among nonsmokers than among smokers.

In children, renal impairment of sarcoidosis usually is caused by either hypercalcemia leading to nephrocalcinosis or interstitial nephritis with or without granulomata (Thumfart et al, 2005). Lofgren's syndrome is a classic set of signs and symptoms involving fever, enlarged lymph nodes, arthritis in the ankles, or erythema nodosum. Overall, prognosis is good for most cases, and most sarcoidosis subsides on its own within 3 years. In 10% of cases, the condition becomes chronic. Sarcoidosis leads to organ damage in about one third of the people diagnosed; the lungs, heart, or brain may be affected.

Hypercalcemia can occur in patients with granulomatous disorders such as sarcoidosis, often related to high serum 1,25-dihydroxyvitamin D (OHD) concentrations (Falk et al, 2007). Endogenous antioxidant defense is significantly reduced, and oxidative stress underlies the pathology of this disease (Boots et al, 2009).



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Sarcoidosis CD4+ T cells are primarily responsible for the systemic responses. The 1-alpha-hydroxylase gene affects alveolar macrophages and disturbances in calcium metabolism.

<b>Clinical/History</b>	BMI	BP
Height	Weight loss?	Fever
Weight	Diet history	

Tender red lumps on shins or ankles	PH cor pulmonale	Alkaline phosphatase (Alk phos)
Lupus pernio (painful sores on face)	Clubbing of fingers, hypoxemia	Nitrogen (N) balance
Granulomas	Iritis, glaucoma, blindness	Transferrin
Enlarged liver or spleen	Chest pain, even heart failure	Globulin
Uveitis	Chest x-ray	(increase common)
SOB, cough	Biopsy	Serum Ca <sup>++</sup>
Night sweats	Gallium scan	(increased)
Bone or joint pain	TB test (rule out tuberculosis)	Ca <sup>++</sup> in urine
Anorexia, weakness, aching joints	Pulmonary function tests	(increased?)
Abdominal pain, lymphadenopathy	<b>Lab Work</b>	Serum vitamin D <sub>3</sub>
Bone cysts in hands and feet	H & H (anemia common)	Na <sup>+</sup> , K <sup>+</sup>
	Serum Fe, ferritin	Mg <sup>++</sup>
	Alb (decreased)	Uric acid
	CRP (elevated)	(increased)
		PO <sub>4</sub>
		Kveim test
		Erythrocyte sedimentation rate (ESR)

## INTERVENTION



### OBJECTIVES

- Reduce heart failure, BX, and related problems.
- Correct weight loss, anorexia, fever, and abdominal pain.
- Improve ability to breathe and eat normally.
- Prevent further deterioration of organ functions with any and all affected organ systems.
- Prevent or correct fluid retention.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Imbalance of Nutrients

**Assessment Data:** Altered lab values, elevated uric acid, and ESR; uveitis, wheezing, coughing, Dx of sarcoidosis; diet hx showing minimal intake of vitamins and minerals.

**Nutrition Diagnoses (PES):** Imbalance of nutrients related to minimal intake of omega-3 fatty acids, vitamins, and minerals as evidenced by chronic inflammation and altered labs (uric acid, ESR, CRP).

**Interventions:** Modify dietary intake to increase antioxidants from fruits, vegetables, whole grains, and nuts as well as omega-3 fatty acids from salmon/tuna/sardines. Provide multivitamin—mineral supplement. Educate about the natural role of diet in reducing inflammation.

**Monitoring and Evaluation:** Improved balance of nutrients from diet and supplementation; normalized labs including CRP, ESR, uric acid.

- High levels of calcium may accumulate in the blood and urine. Monitor for related nausea, anorexia, vomiting, thirst, excessive urination, or renal failure.



## FOOD AND NUTRITION

- Restrict salt if necessary for heart failure or for use of corticosteroids. A 2- to 3-g sodium diet may be beneficial.
- Use a diet containing adequate to high potassium (unless medications are used).
- Patients might benefit from antioxidants such as quercetin (Boots et al, 2009). More fruits and vegetables should be consumed.

## Common Drugs Used and Potential Side Effects

- Prednisone is used to suppress severe symptoms such as SOB. Watch electrolytes, nitrogen balance, and other changes. Treatment may require several years.
- Methotrexate works best for treating sarcoidosis that affects lungs, eyes, skin, or joints. Folic acid depletion can occur (Low et al, 2008).
- Calcium-chelating agents may be used if hypercalcemia persists.
- Sarcoid granulomatous interstitial nephritis may respond to infliximab therapy (Thumfart et al, 2005). The drug seems to work against elevated TNF.
- For pain or fever, nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen may help.

## Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of herbs or botanicals in sarcoidosis.



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- If the patient is using steroids, antacids could also be taken to reduce GI side effects. Check with the doctor.
- Discuss the role of diet in maintaining immunocompetence and in improving tolerance for other therapies.
- Follow regarding calcium and vitamin D supplements to avoid prolonged hypercalcemia and hypercalciuria. If needed, avoid intake of fish oils and excessive sun exposure.

## Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

## For More Information

- Mayo Clinic—Sarcoidosis  
<http://www.mayoclinic.com/health/sarcoidosis/ds00251>
- National Heart, Lung, and Blood Institute—Sarcoidosis  
<http://www.nhlbi.nih.gov/health/public/lung/other/sarcoidosis/index.htm>

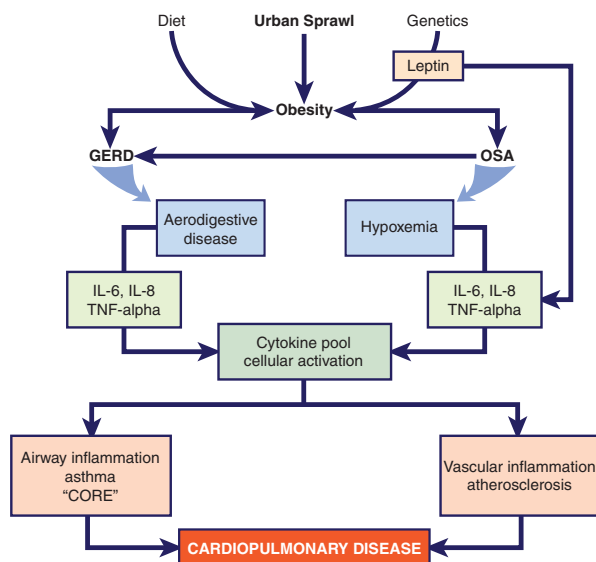
- National Sarcoidosis Resources Center  
<http://www.nsrc-global.net/>
- Sarcoidosis Center  
<http://www.sarcoidcenter.com/>
- Sarcoidosis Family Aid and Research Foundation Hotline  
<http://www.medicinenet.com/sarcoidosis/page10.htm>
- Sarcoidosis Research Institute  
<http://www.sarcoidcenter.com/sricontents.htm>

## SARCOIDOSIS—CITED REFERENCES

- Boots AW, et al. Antioxidant status associated with inflammation in sarcoidosis: a potential role for antioxidants. *Respir Med.* 103:364, 2009.
- Falk S, et al. Hypercalcemia as a result of sarcoidosis with normal serum concentrations of vitamin D. *Med Sci Monit.* 13:113, 2007.
- Low PS, et al. Discovery and development of folic-acid-based receptor targeting for imaging and therapy of cancer and inflammatory diseases. *Acc Chem Res.* 41:120, 2008.
- Thumfart J, et al. Isolated sarcoid granulomatous interstitial nephritis responding to infliximab therapy. *Am J Kidney Dis.* 45:411, 2005.

# SLEEP APNEA

## NUTRITIONAL ACUITY RANKING: LEVEL 2–3



Obstructive sleep apnea (OSA) can lead to chronic disease, such as cardiopulmonary disease.



## DEFINITIONS AND BACKGROUND

Approximately 4% of middle-aged men and 2% of middle-aged women suffer from obstructive sleep apnea (OSA). OSA affects 12–18 million Americans and is associated with irritability, excessive daytime sleepiness, an inability to concentrate, depression, morning headaches, and decreased job performance in adults. Untreated sleep apnea also can increase an individual's risk of heart attack, high BP, diabetes, stroke, and automobile accidents. OSA is often undiagnosed and is a major contributing factor in the development of essential hypertension.

Sleep apnea occurs in both genders and in all ages, weights, and ethnicities. Certain risk factors are associated with a higher incidence, such as excess weight or obesity (BMI >25); family history of sleep apnea; male sex; large neck (greater than 17 inches in men, greater than 16 inches in women); recessed chin; physical abnormality in the nose, throat, or upper airway structure; older age; smoking; use of alcohol or sleeping pills; ethnicity (African Americans, Pacific Islanders, and Hispanics seem to be at an increased risk); and snoring.

Sleep apnea may develop in any patient who has an endocrine disorder or is receiving certain hormonal therapies. Increases in habitual sleep duration is associated with elevations in CRP and IL-6 while reduced sleep duration is associated with elevated TNF $\alpha$  levels; activation of pro-inflammatory pathways may represent a mechanism by which extreme sleep habits affect health (Patel et al, 2009). IL-6, TNF $\alpha$ , and insulin levels are elevated in sleep apnea independently of obesity; visceral fat is the primary parameter linked with sleep apnea (Vgontzas, 2008).

Effective assessment and management of OSA may lead to a reduction in insulin resistance and hypertension as well as other markers of vascular risk in patients with metabolic syndrome (Yee et al, 2004). Untreated severe OSA results in elevated CRP levels and cardiovascular risks. Clinicians should be aware. Both atherosclerosis and OSA are associated with endothelial dysfunction; increased CRP, interleukin-6, fibrinogen, and plasminogen activator inhibitor; and reduced fibrinolytic activity. OSA has also been associated with enhanced platelet activity and aggregation and leukocyte adhesion on endothelial cells (Parish and Somers, 2004).

Obstructive sleep-disordered (OSD) breathing is common in children (3–12% of children snore); mild sleep apnea affects 1–10% of children (Chan et al, 2004). Risk factors of children who are more at risk for OSA are physical abnormalities of the face or skull, cerebral palsy, muscular dystrophy, Down syndrome, sickle cell disease, obesity, and mouth breathing. Consequences of untreated OSA include failure to thrive, enuresis, attention-deficit disorder, behavior problems, poor academic performance, and cardiopulmonary disease (Chan et al, 2004). Sleep deprivation and sleep apnea may even be related to some sudden infant death syndrome (SIDS) cases; upper airway obstruction and depressed arousability from sleep may contribute (Franco et al, 2004).

Treatment includes the use of continuous positive airway pressure (CPAP), weight loss in obese children, or adenotonsillectomy. Use of a CPAP device can be worn while sleeping. This device works to keep the airway open by continuously blowing air through the nasal passages at a high pressure. CPAP may help medically treated patients with heart failure and other cardiovascular conditions. In addition, some dental appliances may reposition the tongue or lower jaw so that the airway remains open while the patient sleeps, thus preventing the apnea. Surgical treatments may



also be done, such as septoplasty, tonsillectomy, uvulopalatopharyngoplasty (UPPP, also known as UP3), and laser-assisted uvulopalatopharyngoplasty (LAUP).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** IL-6, TNF $\alpha$ , and insulin levels are elevated in sleep apnea.

Clinical/History	Apnea–hypopnea index (AHI)	H & H (anemia common)
Height	Respiratory disturbance index (RDI)	Serum Fe, ferritin
Weight	Epworth Sleepiness Scale (ESS)	CRP
BMI		Homocysteine
Abdominal adiposity?		Alb, transthyretin
PCOS?		Ca <sup>++</sup> , Mg <sup>++</sup>
Diet history		Na <sup>+</sup> , K <sup>+</sup>
BP		pCO <sub>2</sub> , pO <sub>2</sub>
Chest x-ray	<b>Lab Work</b>	
Polysomnography (sleep study)	Gluc	
Hypopnea (less than normal breath)	Serum insulin	
	CBC	

## INTERVENTION



### OBJECTIVES

- If obese, weight loss will be beneficial. Obesity is associated with comorbidities such as PH, hypoventilation, and sleep apnea that may lead to disability or death (Poirier et al, 2009).
- In children with sleep apnea and failure to thrive, medical or surgical treatments may help to alleviate the problem so catch-up growth can occur. These children may have reduced upper airway muscle tone, evident mostly during REM sleep stages (Eckert et al, 2009).
- Lessen insulin resistance, where possible.
- Manage other medical and health complications that are present in the individual; cardiovascular disease, hypertension, or metabolic syndrome may coexist with OSA.



### FOOD AND NUTRITION

- Lower energy intake to promote weight loss of 1–1.5 lb weekly if possible.
- Alter diet plan if needed to manage diabetes, sickle cell anemia, or other underlying conditions.
- The DASH diet or a calorie-controlled diet may be useful.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Obesity and Metabolic Syndrome

**Assessment Data:** Divorced male, age 45, BMI 31, chronic hypertension, central adiposity, hx of sleep apnea with snoring, HDL 40, LDL 210, diet hx shows eating away from home six to eight times weekly, no cooking skills or interest, drinks two to three alcoholic beverages daily.

**Nutrition Diagnoses (PES):** Obesity related to excessive intake of energy-dense foods as evidenced by BMI 31, hypertension, sleep apnea, and central adiposity (waist 46").

**Interventions:** Educate about simpler meals that are nutrient-dense and less energy-dense, lower in fat and alcohol; good choices from grocery store or dining at restaurants. Materials from Cooperative Extension or Health Department on "eating for one." Referral go to Sleep Disorder Clinic if interested.

**Monitoring and Evaluation:** Meal and food choices; improvement in BMI, BP, lipids; less problem with snoring and sleep apnea. Participation in Sleep Disorder Clinic evaluation.

## Common Drugs Used and Potential Side Effects

- OSA can be induced, unmasked, or exacerbated by the effects of sedative, analgesic, and anesthetic agents (Jain and Dhand, 2004). Sleeping agents are not generally recommended.
- Treatment of depression or mood disorders may be needed. In patients who are on chronic neuroleptic drugs for schizophrenia, weight management will be very important.

## Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of herbs or botanicals in sleep apnea.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Typically, patients diagnosed with sleep apnea are advised to avoid tobacco, alcohol, sedatives, and medications that relax the airway and/or reduce respiratory function.
- Regular exercise and weight reduction can help some patients with mild or moderate sleep apnea minimize their symptoms.
- Sleep apnea sufferers are advised to avoid sleeping on their back, if possible. Using pillows and other devices that help the patient sleep in a side position may help.
- The relationship of OSA with hypertension, stroke, and cardiovascular disease should be discussed.
- Help with obesity if needed, especially central adiposity (Schwartz et al, 2008).
- Patients who have a cough, OSA, rhinosinusitis, and esophageal reflux clustered together can be categorized as having CORE syndrome (Arter et al, 2004).

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

#### For More Information

- Narcolepsy Network  
<http://www.websciences.org/narnet/>
- National Sleep Foundation  
<http://www.sleepfoundation.org/>
- Sleep Apnea Association  
<http://www.sleepapnea.org/>

### SLEEP APNEA—CITED REFERENCES

- Arter JL, et al. Obstructive sleep apnea, inflammation, and cardiopulmonary disease. *Front Biosci.* 9:2892, 2004.
- Chan J, et al. Obstructive sleep apnea in children. *Am Fam Physician.* 69:1147, 2004.

- Eckert DJ, et al. The influence of obstructive sleep apnea and gender on genioglossus activity during rapid eye movement sleep. *Chest.* 135:954, 2009.
- Franco P, et al. Decreased arousals among healthy infants after short-term sleep deprivation. *Pediatrics.* 114:192, 2004.
- Jain SS, Dhand R. Perioperative treatment of patients with obstructive sleep apnea. *Curr Opin Pulm Med.* 10:482, 2004.
- Parish JM, Somers VK. Obstructive sleep apnea and cardiovascular disease. *Mayo Clin Proc.* 79:1036, 2004.
- Patel SR, et al. Sleep duration and biomarkers of inflammation. *Sleep.* 32:200, 2009.
- Poirier P, et al. Cardiovascular evaluation and management of severely obese patients undergoing surgery. A Science Advisory From the American Heart Association [epub ahead of print June 15, 2009.]. *Circulation.* 120:86, 2009.
- Schwartz AR, et al. Obesity and obstructive sleep apnea: pathogenic mechanisms and therapeutic approaches. *Proc Am Thorac Soc.* 5:185, 2008.
- Vgontzas AN. Does obesity play a major role in the pathogenesis of sleep apnoea and its associated manifestations via inflammation, visceral adiposity, and insulin resistance? *Arch Physiol Biochem.* 114:211, 2008.
- Yee B, et al. Neuroendocrine changes in sleep apnea. *Curr Opin Pulm Med.* 10:475, 2004.

## THORACIC EMPYEMA

### NUTRITIONAL ACUITY RANKING: LEVEL 2



#### DEFINITIONS AND BACKGROUND

Thoracic empyema involves accumulation of pus in the pleural cavity, sometimes as a complication of pneumonia. Complications may include septic shock, multiple organ failure, cardiac insufficiency, and end-stage renal failure. A chest tube may be placed (thoracentesis) to drain the infection.

In diaphragmatic injury, empyema is a rare but serious complication that can lead to prolonged hospital or ICU lengths of stay; gastric trauma is often associated (Bramparas et al, 2009). Use of prophylactic antibiotics may be prescribed.

An increase in the incidence of thoracic empyema in children has been noted, and the causative pathogen is often unknown (Sagiani et al, 2005). *Staphylococcus aureus* is a common micro-organism isolated from the bacterial cultures, as is *Mycobacterium tuberculosis* (Ozel et al, 2004). With an increasing incidence of *S. aureus*, particularly MRSA, the use of video-assisted thoracoscopy (VATS) results in a decreased duration of fever and length of hospitalization (Schultz et al, 2004).



#### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Most empyema is from pneumonia or trauma. The noted virulence of invasive pneumococcal disease (IPD) after the initiation of vaccine has led to speculation about antibiotic resistance in some individuals.

Clinical/History	Productive cough I & O Dyspnea, orthopnea Constant localized chest pain Tachycardia, tachypnea CT scan Ultrasound Tachycardia?	Lab Work Alb, transthyretin H & H Serum Fe Gluc Na <sup>+</sup> , K <sup>+</sup> Ca <sup>++</sup> , Mg <sup>++</sup> pO <sub>2</sub> (often decreased) pCO <sub>2</sub> Transferrin CRP
Height		
Weight		
BMI		
Weight loss?		
Anorexia, fatigue		
Diet history		
BP		
Temperature (fever?)		
Pleural examination		

#### SAMPLE NUTRITION CARE PROCESS STEPS

##### Inadequate Oral Food and Beverage Intake

**Assessment Data:** Chronic cough and chest pain, fatigue and anorexia with weight loss of 12 lb in past month. Fever 102°F for past 3 days.

**Nutrition Diagnoses (PES):** Inadequate oral food and beverage intake related to anorexia, fever, tachycardia, chronic cough and chest pain from thoracic empyema as evidenced by weight loss of 12 lb in past month.

**Interventions:** Educate about simple, nutrient and energy-dense meals and snacks. Counsel about ways to lessen fatigue with meal-time preparation. Coordinate care with home-delivered meals or shopping assistance when discharged.

**Monitoring and Evaluation:** Improved oral food and beverage intake as per patient food diary and weight gain of 5 lb in 3 weeks after returning home. Fewer complaints of anorexia or poor nutrition quality of life.

**INTERVENTION****OBJECTIVES**

- Lessen fatigue; promote improved well-being.
- Reduce fever. Prevent sepsis, organ failure, and other complications.
- Correct weight loss.
- Control and reduce anorexia.
- Support the capacity for wound healing if surgery is needed.

**FOOD AND NUTRITION**

- Provide diet as ordered. Patient may need high-calorie/high-protein foods served at frequent intervals.
- Two or more liters of fluid may be needed daily, unless contraindicated.
- Meals should be served in an attractive manner to stimulate appetite.
- A multivitamin–mineral supplement may be useful.

**Common Drugs Used and Potential Side Effects**

- Antibiotics such as streptokinase are common (Cameron and Davies, 2004). Monitor side effects accordingly.
- Monitor effects of other medications as prescribed.

**Herbs, Botanicals, and Supplements**

- No clinical trials have proven efficacy for use of herbs or botanicals in thoracic empyema.

**NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT**

- Discuss the role of nutrition in illness and recovery, especially as it relates to immunocompetence.
- With family, discuss signs to observe for future problems or relapses.

**Patient Education—Foodborne Illness**

- Careful food handling will be important. Hand washing is key as well.

**For More Information**

- Empyema  
<http://emedicine.medscape.com/article/355892-overview>
- NIH—Empyema  
<http://www.nlm.nih.gov/MEDLINEPLUS/ency/article/000123.htm>
- Thoracic Empyema  
<http://www.encyclopedia.com/html/e1/empyema.asp>

**THORACIC EMPYEMA—CITED REFERENCES**

- Bramparas G, et al. Risk factors for empyema after diaphragmatic injury: results of a National Trauma Databank analysis. *J Trauma*. 66:1672, 2009.
- Cameron R, Davies HR. Intra-pleural fibrinolytic therapy versus conservative management in the treatment of parapneumonic effusions and empyema. *Cochrane Database Syst Rev*. 2:CD002312, 2004.
- Ozel SK, et al. Conservative treatment of postpneumonic thoracic empyema in children. *Surg Today*. 34:1002, 2004.
- Saglan S, et al. Empyema: the use of broad range 16 S rDNA PCR for pathogen detection. *Arch Dis Child*. 90:70, 2005.
- Schultz KD, et al. The changing face of pleural empyemas in children: epidemiology and management. *Pediatrics*. 113:1735, 2004.

# TRANSPLANTATION, LUNG

**NUTRITIONAL ACUITY RANKING: LEVEL 3–4****DEFINITIONS AND BACKGROUND**

Lung transplantation (LTX) is an accepted treatment for end-stage pulmonary parenchymal and vascular diseases. LTX is a well-tolerated, effective therapy for RF with interstitial lung disease, CF or COPD. The International Society for Heart and Lung Transplantation and the Cystic Fibrosis Foundation have uniform guidelines for transplantation candidate selection. Over 13,000 LTXs have occurred worldwide (Tynan and Hasse, 2004).

Proper nutrition plays a key role in preparing for LTX. Therefore, the LTX dietitian plays an important role and meets with the patient for an initial interview. Weight and weight history, foods typically eaten, and appetite are reviewed. Being at ideal body weight range for height helps assure good physical condition for pretransplantation pulmonary rehabilitation and for the transplantation itself. Certain patients with advanced pulmonary disease are unable to eat enough to maintain ideal body weight because of

increased metabolic demands and breathlessness with eating. In such situations, it may be recommended that a percutaneous endoscopic gastrostomy (PEG) feeding tube be placed.

Proper nutrition is critical to maximize the chances of a successful transplantation. Occasionally, listing for transplantation will be delayed until the patient's nutritional status improves. LBM depletion may be associated with more severe hypoxemia, reduced walking distance, and a higher mortality. Both undernutrition and obesity should be carefully managed before surgery. Diabetes is a common problem after LTX in CF patients even though quality of life is dramatically improved (Hadjiliadis, 2007).

As with other types of transplantations, graft–host resistance and sepsis are the major concerns after LTX. Infections are the most common cause of morbidity and mortality in LTX recipients. Immunosuppressive therapy with glucocorticoids contributes to protein degradation.

Nitrogen balance after LTX is negative because of high glucocorticoid requirements; aggressive nutritional intervention and increased nitrogen intake are needed to reduce protein losses in these patients. Chronic infection (bronchiolitis obliterans syndrome) is the most common cause of death after transplantation (Quattrucci et al, 2005).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Organ transplant researchers are increasingly using microarrays to identify specific patterns of gene expression that predict and characterize acute and chronic rejection. Increased expression of genes involved in inflammation, apoptosis, and T-cell activation and proliferation may play a role in organ rejection (Lande et al, 2007).

Clinical/History	Lab Work	
Height	Alb,	BUN, Creat
Weight	transferrin	Na <sup>+</sup> , K <sup>+</sup>
BMI	CRP	Ca <sup>++</sup> , Mg <sup>++</sup>
Weight changes	Transferrin	PO <sub>4</sub>
Diet history	Chol, Trig	AST, ALT
RQ	H & H	Lactate
Ventilator	Serum Fe,	TLC
support	ferritin	CRP
I & O	Gluc	pCO <sub>2</sub> , pO <sub>2</sub>

## INTERVENTION



### OBJECTIVES

#### Preoperative

- Because nutritional depletion in LTX candidates is highly prevalent, it should be precisely assessed both before and after LTX. Attempts should be made to increase LBM and reverse cachexia and vitamin and mineral deficiencies before LTX.
- Prepare for a surgical procedure. Most patients will require sodium or fluid restrictions; monitor serum potassium as well.
- Allow for mild weight loss with a planned diet if the patient is obese and has time to do this.

#### Postoperative

- Prevent infection, surgical complications, organ rejection, and organ failure.
- Promote wound healing.
- Support ideal body weight and LBM maintenance.
- Reduce protein losses, support nitrogen balance, and correct hypoalbuminemia.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Inadequate Protein Intake

**Assessment Data:** BMI 18, recent weight loss of 20 lb, lung failure with planned Tx surgery, low serum albumin.

**Nutrition Diagnoses (PES):** Inadequate protein intake related to loss of LBM and insufficient oral intake as evidenced by albumin 2.1 and diet history showing low meat and milk consumption.

**Interventions:** Enhance meals by adding dry milk powder to recipes such as mashed potatoes and casseroles; offer puddings, eggnog and oral supplements between meals; add protein powder scoops to milkshakes or soups. Educate patient and family about the importance of protein for maintaining LBM and wound healing.

**Monitoring and Evaluation:** Improved intake of protein-rich foods. Successful wound healing after surgery. Improvement in weight and BMI over several months.

- Prevent aspiration.
- Wean from ventilator or oxygen when possible.
- Treat comorbid conditions such as cardiovascular disease (CVD), osteoporosis, dyslipidemia, diabetes, hyperglycemia, metabolic syndrome, and hyperkalemia (Tynan and Hasse, 2004).



## FOOD AND NUTRITION

#### Preoperative

- Prepare patient nutritionally to alleviate malnutrition in advance (Inouye et al, 2004). Home enteral or PN may be useful.
- Promote adequate intake of kcal (25–30 kcal/kg) and protein (1 g/kg body weight).
- Manage coexisting problems such as diabetes, heart disease, and hypertension with an appropriate diet such as the DASH diet.

#### Postoperative

- Return to oral intake by 48–72 hours postoperatively, when possible. Limit simple CHO when there are signs of hyperglycemia (Tynan and Hasse, 2004).
- Promote adequate intake of kcal (30–35 kcal/kg) and protein of 1.3–1.5 g/kg body weight (Tynan and Hasse, 2004). Use high nitrogen TF when needed, but do not overfeed, and monitor for needed changes in electrolytes according to lab values. Discontinue TF when intake meets >60% of estimated needs (Tynan and Hasse, 2004).
- Parenteral solutions may be used if the gut is nonfunctioning (Tynan and Hasse, 2004).
- Calorie-dense options should be considered if fluid restriction is required. Use caution with high-caloric loads because of RQ; maintain sufficient fat intake to prevent excess CO<sub>2</sub> production from a high-CHO intake.
- Restrict sodium and potassium if needed to improve cardiac or renal status.



**TABLE 5-13 Medications Used for Lung Transplant Patients**

Medication	Description
Azathioprine (Imuran)	May cause leukopenia, thrombocytopenia, oral and esophageal sores, macrocytic anemia, pancreatitis, vomiting, diarrhea, and other side effects that are complex. 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 (such as prednisone, hydrocortisone)	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	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 also may 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.
Immunosuppressants	Less nephrotoxic than cyclosporine but can cause nausea, anorexia, diarrhea, and vomiting. Monitor carefully. Fever (muromonab [Orthoclone OKT3] and stomatitis also may occur; alter diet as needed and antithymocyte globulin).
Diuretics	Diuretics such as furosemide may cause hypokalemia. Low-sodium/low-calorie diets may be indicated. If spironolactone is used, it spares potassium.
Tacrolimus (Prograf, FK506)	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. Tacrolimus therapy has aided in success of lung transplantation and has become the primary immunosuppressant agent used (Fan et al, 2009; Garrity and Mehra, 2004). A low-potassium diet may be needed to prevent cardiac arrhythmia (Tynan and Hasse, 2004).

- Reduce energy intake and increase activity if weight gain or diabetes occurs after long-term corticosteroid use (Tynan and Hasse, 2004).
- Prevent osteoporosis by using adequate calcium and vitamin D. Provide sufficient magnesium and vitamins to heal and promote adequate nutritional status.

### Common Drugs Used and Potential Side Effects

- Using tacrolimus as primary immunosuppressant for lung transplant recipient results in comparable survival and reduction in acute rejection episodes when compared with cyclosporine (Fan et al, 2009). See Table 5-13 for more information.

### Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of herbs or botanicals after LTX.



#### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss appropriate calorie and protein levels. Protein helps to heal after surgery.
- Drink plenty of water until restriction is prescribed.
- Decreased saturated fat and cholesterol intakes may be useful to decrease cardiac risks and to prevent unwanted weight gain, which is common. Read food labels and monitor portions carefully. Choose condiments such as mustard rather than mayonnaise or salad dressing. Choose

healthy cooking methods. Instead of frying, try baking, grilling, broiling, or steaming foods; instead of oil, use nonstick, fat-free spray or sauces.

- Adequate fiber (from fresh fruits, vegetables, and whole grains) is important.
- A gradual return to activity will be important.
- Eat a minimum amount of salt, processed foods, and snacks. Use herbs and spices to add flavor instead of salt.
- Add calcium by eating calcium-rich foods, such as low-fat dairy products and green, leafy vegetables, or by using calcium supplements.
- Avoid alcohol and do not use drugs that are not prescribed.

### Patient Education—Foodborne Illness

- Preventing infection is very important after transplantation surgery. Hand washing is critically important.
- Careful food handling will be important.

### For More Information

- Cystic Fibrosis—Transplantation  
<http://www.cff.org/treatments/LungTransplantation/>
- Fast Facts about Transplants  
<http://www.ustransplant.org/csr/current/fastfacts.aspx>
- International Society for Heart and Lung Transplantation  
<http://www.ishlt.org/>
- Lung Transplantation  
<http://www.nlm.nih.gov/medlineplus/lungtransplantation.html>
- Organ Procurement and Transplantation Network  
<http://www.optn.org/>
- Transplant Terms  
<http://www.transplantliving.org/Community/glossary.aspx>
- Trans Web  
<http://www.transweb.org/>

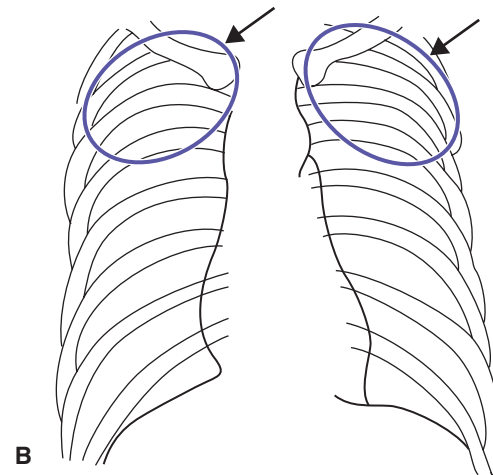
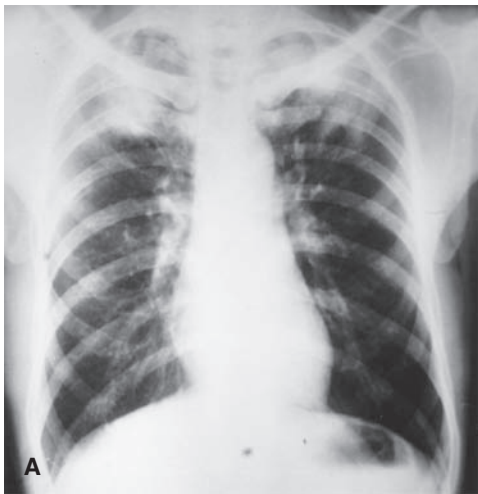
## TRANSPLANTATION, LUNG—CITED REFERENCES

- Fan Y, et al. Tacrolimus versus cyclosporine for adult lung transplant recipients: a meta-analysis. *Transplant Proc.* 41:1821, 2009.
- Garrity ER Jr, Mehra MR. An update on clinical outcomes in heart and lung transplantation. *Transplantation.* 77:S68, 2004.
- Hadjiliadis D. Special considerations for patients with cystic fibrosis undergoing lung transplantation. *Chest.* 131:1224, 2007.

- Inouye Y, et al. Benefits of home parenteral nutrition before lung transplantation: report of a case. *Surg Today.* 34:525, 2004.
- Lande JD, et al. Novel insights into lung transplant rejection by microarray analysis. *Proc Am Thorac Soc.* 4:44, 2007.
- Quattrucci S, et al. Lung transplantation for cystic fibrosis: 6-year follow-up. *J Cyst Fibros.* 4:107, 2005.
- Tynan C, Hasse JM. Current nutrition practices in adult lung transplantation. *Nutr Clin Pract.* 19:587, 2004.

# TUBERCULOSIS

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2



**A** is an X-ray of tubercular lungs. **B** shows the presence of TB by circled areas.

Adapted from: Engleberg NC, Dermody T, DiRita V. *Schaecter's Mechanisms of Microbial Disease*, 4th ed. Baltimore: Lippincott Williams & Wilkins, 2007.



## DEFINITIONS AND BACKGROUND

TB is caused by a tubercle bacillus (*Mycobacterium tuberculosis*) invading the lungs and setting up an inflammatory process. Healing occurs with a calcification of the tubercular cavity. TB causes loss of appetite, constant fatigue, tissue wasting, exhaustion, hemoptysis, cough lasting 3 weeks or longer with occasional blood-tinged sputum, fever or chills, profuse night sweats, and weight loss. The acute form resembles pneumonia; the chronic form causes low-grade fever.

Nearly one third of the world's population is infected with *M. tuberculosis* (Pai et al, 2006). More than 9 million new cases were reported in 2007, many of them in Africa. An increase in TB in the United States may be related to inadequate compliance with prescribed drug therapy or to recently acquired or reactivated latent infections. Among U.S. born citizens, non-Hispanic African American, Mexican Americans, and individuals living in poverty have the highest risk for TB (Bennett et al, 2008). Immunocompromised persons are more vulnerable to the effects of TB, especially those persons who have HIV infection. Hypermetabolism appears to play a role in the wasting process in

patients infected with both HIV and TB. HIV infection is associated with a significant downregulation of whole-body protein flux, adding to the nutritional decline if TB is also present (Paton et al, 2003).

Vitamin D signaling within macrophages enables them to respond to and kill *Mycobacterium tuberculosis* organisms (Bikle, 2008; Shapira et al, 2009). This is an intracrine–autocrine–paracrine system for vitamin D that is just being recognized (Adams et al, 2007).

Active TB begins in the lungs but often spreads through the bloodstream as extrapulmonary TB. Fatigue, abdominal tenderness, painful urination, headache, SOB, arthritis-like symptoms, kidney damage, and pain in the spine and bones can occur. TB meningitis is a very dangerous complication, especially for the elderly.

Many TB patients have early, unplanned readmission and often need assistance with activities of daily living. They may have drug complications, the need to use a nonstandard drug regimen, and other illnesses. With a high prevalence of malnutrition, a relatively low utilization rate of nutritional services, and the potential effect of adverse reactions to therapeutic drugs, careful attention is needed for this patient population.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** It is suspected that TB has connections with allergy. There are higher levels of specific IgE, interleukin (IL)-6, and interferon (IFN) gamma to different inhalant allergens in TB patients; successful treatment lowers these levels (Ellertsen, 2009). In addition, polymorphisms in the gene that encodes the vitamin D receptor (VDR) influence host response to *Mycobacterium tuberculosis* (Roth et al, 2004).

Bioconversion of 25-hydroxyvitamin D<sub>3</sub> (25D<sub>3</sub>) into bioactive 1,25D<sub>3</sub>, leading to VDR activation and antimicrobial activity against intracellular TB (Krutzik et al, 2008).

<b>Clinical/History</b>	Biopsy or sputum test for <i>M. tuberculosis</i>	Lymphopenia? H & H
Height		Serum Fe, ferritin
Weight	Temperature, fever or chills	Normocytic anemia?
BMI	Night sweats	Serum pyridoxine
Diet history	Anorexia	N balance
BP	Spinal tap for polymerase chain reaction (PCR)	Chol (decreased)
Mantoux skin test	I & O	Na <sup>+</sup> , K <sup>+</sup>
QuantiFERON®-TB Gold test (QFT-G)	<b>Lab Work</b>	Ca <sup>++</sup> , Mg <sup>++</sup>
T-SPOT® TB test	Alb, transthyretin	Serum folate
Chest x-rays (irregular white areas on dark background)	CRP	Transferrin
Bronchoscopy	RBP	BUN, Creat
Blood-tinged sputum	TLC	Liver function tests (from medication use)

## INTERVENTION



### OBJECTIVES

- Maintain or prevent losses in weight. Reduce fever. The basal metabolic rate is 20–30% above normal to counteract fever of 102°F or higher.
- Normalize serum calcium and vitamin D<sub>3</sub> levels; either hypocalcemia or hypercalcemia may occur.
- TB often coincides with nutritional deficiencies; micronutrient supplementation may improve the outcome in patients undergoing TB treatment (Villamor et al, 2008).

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Involuntary Weight Loss

**Assessment Data:** Analysis of estimated oral intake below estimated needs.

**Nutrition Diagnosis (PES):** Involuntary weight loss related to insufficient intake and frequent coughing spells, medication-related GI symptoms as evidenced by 15-lb weight loss since TB diagnosis months ago.

**Intervention:** Food and nutrient delivery with careful timing of meals and snacks in relation to medication administration and coughing episodes. Small, frequent meals and oral supplements.

**Monitoring and Evaluation:** Monitor and evaluate changes in intake and weight; tolerance for medications; and nutritional quality of life.

- Replace nutrient losses from lung hemorrhage, if present.
- Promote healing of the cavity.
- Counteract neuritis from isoniazid (INH) therapy, when used.
- Stimulate appetite, which is generally poor.
- Prevent dehydration.
- Prevent lung inflammation, infections, and complications.



### FOOD AND NUTRITION

- Use a well-balanced diet containing liberal amounts of protein and adequate calories. It may be useful to calculate needs as 35–45 kcal/kg if weight loss has been significant.
- Use adequate fluids (35 cc/kg) unless otherwise contraindicated.
- Add more omega-3 fatty acids; they may improve food intake, restore normal eating patterns, and prevent body weight loss (Ramos et al, 2004).
- Ensure that the diet provides sufficient levels of calcium and vitamin D.
- Iron and vitamin C are needed for proper hemoglobin formation and wound healing.
- B-complex vitamins, especially vitamin B<sub>6</sub>, are needed to counteract INH therapy.
- Use supplemental vitamin A as carotene as it is poorly converted.
- Alcohol should not be used as a calorie replacement or appetite enhancer.

### Common Drugs Used and Potential Side Effects

- Current anti-TB chemotherapies, although effective, are associated with side effects and are limited in treating drug-resistant strands (Shapira et al, 2009).
- See Table 5-14 for more drug therapies.

**TABLE 5-14 Medications Used for Tuberculosis (TB)**

Medication	Description
Aminosalicylic acid	Interferes with vitamin B <sub>12</sub> and folate absorption. Nausea and vomiting are common.
Chemotherapy	Chemotherapy can increase serum calcium levels.
Ethionamide (Trecator-SC)	Requires a vitamin B <sub>6</sub> supplement. It may cause anorexia, metallic taste, nausea, vomiting, diarrhea, weight loss, and hypoglycemia.
Ethambutol (Myambutol)	May cause GI distress, nausea, or anorexia. It should not be used longer than 2 months because it can harm the eyes.
Immunotherapy	According to the Centers for Disease Control and Prevention (2004): TB disease is a potential adverse reaction from treatment with tumor necrosis factor-alpha (TNF- $\alpha$ ) antagonists infliximab (Remicade), etanercept (Enbrel), and adalimumab (Humira). These products block TNF- $\alpha$ , an inflammatory cytokine, and are approved for treating rheumatoid arthritis and other selected autoimmune diseases. Blocking TNF- $\alpha$ can allow TB disease to emerge from latent <i>Mycobacterium tuberculosis</i> infection. Health care providers should take steps to prevent TB in immunocompromised patients and remain vigilant for TB as a cause of unexplained fever.
Isoniazid (INH)	May cause neuritis by depleting vitamin B <sub>6</sub> ; usual dose is 300 mg INH with 50 mg pyridoxine. Bad taste can be disguised in pureed fruit or jam to make it palatable, especially for pediatric patients. Niacin, calcium, and vitamin B <sub>12</sub> are also depleted. Nausea, jaundice, vomiting, stomach cramping, and dry mouth are common. INH must be taken for 9 months to eradicate the condition completely.
Pyrazinamide (PZA)	May cause anorexia, nausea, and vomiting. It can be hepatotoxic.
Rifampin (Rifadin, Rimactane)	Has side effects such as anorexia and GI distress.
Streptomycin	One of the first drugs used to treat TB. It is given by injection. Use of longer than 3 months can affect balance and hearing.

NOTE. Therapy always involves two or more drugs because of the long-term treatment period required.

## Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of eucalyptus, echinacea, garlic, licorice, honeysuckle, or forsythia in TB management.
- Deficiencies of multiple micronutrients (MMN) are common in developing countries or where TB is common; outcomes are better using MMN than when providing just one to two micronutrients (Allen et al, 2009). Vitamins D, E, and selenium are supplements that should be highlighted.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Add protein powders or nonfat dry milk to beverages, casseroles, soups, and desserts to increase protein and calcium intake, unless contraindicated for other medical reasons.
- Encourage preparation of small, appetizing meals. Plan rest periods before and after meals.
- Discuss tips for managing anxiety related to weight loss, night sweats, loss of strength, high fever, and abnormal chest x-rays.
- Discuss communicability of TB. Family members and those living in proximity should have x-rays and other tests. About 5% of exposures result in TB within 1 year; others may be dormant until another condition sets in such as HIV infection, diabetes, or leukemia.
- Promote adequate rehabilitation if the patient is an alcoholic.
- Promote as much quality of life as possible; this is often overlooked (Marra et al, 2004).

- A TB vaccine is available. The BCG (bacille Calmette-Guérin) vaccine for TB disease is not widely used in the United States, but it is often used in other countries where TB is common.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Foodservice employees who are exposed to those at risk for active TB should be tested regularly. People are at risk and may need to be tested if they:
  - Have symptoms of active TB disease
  - Have been exposed to someone (family member, friend, or coworker) who has active TB
  - Have HIV infection, diabetes, or chronic kidney failure
  - Take steroids or other immune-suppressing drugs for chronic medical conditions
  - Live or work in a homeless shelter, prison, hospital, nursing home, or other group setting
  - Have recently moved from a region with active TB (Africa, Asia, the Caribbean, Eastern Europe, and Latin America).
- When preparing food:
  - Separate raw meat from cooked or ready-to-eat foods. Do not use the same chopping board or the same knife for preparing raw meat and cooked or ready-to-eat foods.
  - Do not handle either raw or cooked foods without washing hands in between.
  - Do not place cooked meat back on the same plate or surface it was on before it was cooked.
  - All foods from poultry should be cooked thoroughly, including eggs. Egg yolks should not be runny or liquid. Because influenza viruses are destroyed by heat,



the cooking temperature for poultry meat should reach 70°C (158°F).

- Wash egg shells in soapy water before handling and cooking, and wash hands afterwards.
- Do not use raw or soft-boiled eggs in foods that will not be cooked.
- After handling raw poultry or eggs, wash hands and all surfaces and utensils thoroughly with soap and water.
- Do not eat uncooked or undercooked poultry or poultry products, including food with uncooked poultry blood.

### For More Information

- CDC  
<http://www.cdc.gov/tb/links/default.htm>
- JAMA—Patient page for TB  
<http://jama.ama-assn.org/cgi/reprint/300/4/464.pdf>
- Joint HIV/TB Interventions  
<http://www.who.int/hiv/topics/tb/tuberculosis/en/>
- Lung Association of Canada  
[http://www.lung.ca/diseases-maladies/tuberculosis-tuberculose\\_e.php](http://www.lung.ca/diseases-maladies/tuberculosis-tuberculose_e.php)
- National Tuberculosis Curriculum Consortium  
<http://ntcc.ucsd.edu/>
- National TB Center  
<http://www.nationaltbcenter.edu/>
- NIH—Medline  
<http://www.nlm.nih.gov/medlineplus/tuberculosis.html>
- Travelers Health Website  
<http://www.cdc.gov/travel>
- World Health Organization  
<http://www.who.int/tb/en/>

## TUBERCULOSIS—CITED REFERENCES

- Adams JS, et al. Vitamin D in defense of the human immune response. *Ann NY Acad Sci.* 1117:94, 2007.
- Allen LH, et al. Provision of multiple rather than two or fewer micronutrients more effectively improves growth and other outcomes in micronutrient-deficient children and adults. *J Nutr.* 139:1022, 2009.
- Bennett DE, et al. Prevalence of tuberculosis infection in the United States population: the national health and nutrition examination survey, 1999–2000. *Am J Respir Crit Care Med.* 177:348, 2008.
- Bikle DD. Vitamin D and the immune system: role in protection against bacterial infection. *Curr Opin Nephrol Hypertens.* 17:348, 2008.
- Centers for Disease Control and Prevention. Tuberculosis associated with blocking agents against tumor necrosis factor- $\alpha$ —California, 2002–2003. *MMWR Morb Mortal Wkly Rep.* 53:683, 2004.
- Ellertsen LJ. Allergic sensitization in tuberculosis patients at the time of diagnosis and following chemotherapy. *BMC Infect Dis.* 9:100, 2009.
- Krutzik SR, et al. IL-15 links TLR2/1-induced macrophage differentiation to the vitamin D-dependent antimicrobial pathway. *J Immunol.* 181:7115, 2008.
- Marra CA, et al. Factors influencing quality of life in patients with active tuberculosis. *Health Qual Life Outcomes.* 2:58, 2004.
- Pai M, et al. New tools and emerging technologies for the diagnosis of tuberculosis. Part I. Latent tuberculosis. *Expert Rev Mol Diagn.* 6:413, 2006.
- Paton NI, et al. Effects of tuberculosis and HIV infection on whole-body protein metabolism during feeding, measured by the [15 N]glycine method. *Am J Clin Nutr.* 78:319, 2003.
- Ramos EJ, et al. Effects of omega-3 fatty acid supplementation on tumor-bearing rats. *J Am Coll Surg.* 199:716, 2004.
- Roth DE, et al. Association between vitamin D receptor gene polymorphisms and response to treatment of pulmonary tuberculosis. *J Infect Dis.* 190: 920, 2004.
- Shapira Y, et al. Mycobacterium tuberculosis, autoimmunity, and vitamin D [epub ahead of print Jun 20., 2009]. *Clin Rev Allergy Immunol.* 38:169, 2010.
- Villamor E, et al. A trial of the effect of micronutrient supplementation on treatment outcome, T cell counts, morbidity, and mortality in adults with pulmonary tuberculosis. *J Infect Dis.* 197:1499, 2008.

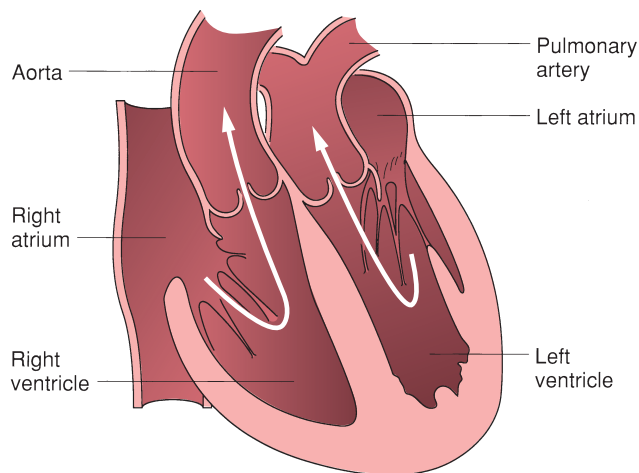


# Cardiovascular Disorders

## CHIEF ASSESSMENT FACTORS

- Age: Males  $\geq 45$  Years of Age and Females  $\geq 55$  Years of Age
- Alcohol Use (none, moderate, excessive)
- Angiograms, ECG, Echocardiograms
- Ascites, Edema
- Blood Pressure
- Cardiogenic Shock: Low Systolic Blood Pressure (BP), Cool and Moist Skin, Decreased Urinary Output, Pulmonary Edema, Tachycardia, Weak Pulse
- Chest Pain
- Cholesterol and Lipid Profiles (higher HDL is protective, small dense LDL is atherogenic)
- Contraceptive Use or Menopause
- C-Reactive Protein (CRP) and CoQ10 serum levels
- Decreased Cardiac Output: Arrhythmias, Fatigue, Labored Respirations, Pallor, Rales, Vertigo
- Diabetes
- Dietary Pattern with High Saturated Fat Intake
- Electrolyte Balance
- Exercise Patterns
- Family Hx (Use ATP III Guidelines) or Sibling Cardiovascular Disease
- Herbs or Botanical Product Use
- Homocysteinemia and Genetic Alleles Predisposing to Heart Diseases
- International Normalized Ratio (INR) Coagulation Index
- Lactic Acid Dehydrogenase (LDH), Creatine Phosphokinase (CPK) Levels
- Medications
- Obesity
- Smoking and Tobacco Use
- Type A Personality, Stressful Lifestyle
- Serum Vitamin D<sub>3</sub> levels
- Xanthomas

## OVERVIEW: DIET IN HEART DISEASE



Adapted from: Michael W. Mulholland, Ronald V. Maier et al. *Greenfield's Surgery Scientific Principles And Practice*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.

## LIPIDS

Cardiovascular disease (CVD) includes hypertension, coronary heart disease (CHD), heart failure (HF), congenital heart defects, and stroke; CHD accounts for thousands of deaths annually. CVD accounts for almost 50% of all deaths in industrialized nations. Estimates for the year 2006 are that 80,000,000 people in the United States have one or more forms of CVD (American Heart Association, 2009). Despite a dramatic decline in mortality over the past three decades, CHD remains a leading cause of death and disability. Death rates for women have not declined as much as those for men.

Classic cardiovascular risk factors are common but largely undertreated and undercontrolled in many regions of the world (Bhatt et al, 2006). Seventy percent of CVD can be prevented or delayed with dietary choices and lifestyle modifications (Forman and Bulwer, 2006). There are 12 modifiable dietary, lifestyle, and metabolic risk factors: high blood glucose, low-density lipoprotein (LDL) cholesterol (Chol), and blood pressure (BP); overweight-obesity; high dietary trans fatty acids and salt; low dietary polyunsaturated fatty acids, omega-3 fatty acids (seafood), and fruits and vegetables; physical inactivity; alcohol use; and tobacco smoking (Danaei et al, 2009). Of these factors, tobacco smoking and high BP are responsible for the most causes of death in the United States.

Many patients with classic CVD risk factors can achieve risk-reduction goals without medications within 3 months after initiating therapeutic lifestyle changes (TLCs). TLC includes exercise training, nutrition counseling, and other appropriate lifestyle interventions based on several well-established behavior change models. The benefits of primary prevention of CVD are greatest for people who have multiple risk factors. Secondary prevention is beneficial for high-risk and low-risk patients. The Adult Treatment Panel (ATP III) report provides scientific evidence for dyslipidemia management. While dyslipidemia with small dense LDL molecules is atherogenic, dietary Chol is only one of

many factors to play a role in the etiology of heart disease. Chol is readily made from acetate in all animal tissues and has many roles in the body. In children and teens, widespread Chol screening is not warranted, except where there is early cardiovascular morbidity and mortality in immediate family members. There is a strong, independent relationship of vitamin D<sub>3</sub> [25(OH)D] deficiency (levels <20 ng/mL) with prevalent CVD in a large sample of the U.S. adult population; this has implications for both angina and myocardial infarction (MI, Kendrick et al, 2009). Other nutrients also play a role. Studies show a link between intake of fruit, vegetables, and whole grains and protection against CHD due to fiber, vitamin, mineral, and phytochemical content. Folate, vitamins B<sub>6</sub>, B<sub>12</sub>, E, and C, flavonoids, phytoestrogens, and a wholesome total dietary pattern may be protective.

In a specific analysis of "low-fat" diets, there were no significant effects on the incidence of stroke and CHD, but there were small reductions in LDL and total Chol (TC) levels, diastolic BP, and factor VIIc levels (Howard et al, 2006). There are indirect benefits of a diet with a lower intake of saturated and trans fats, higher intake of vegetable and fruits, use of specific types of fats including fish oils, as well as fish, and perhaps energy restriction (Anderson, 2006). Epidemiologic data suggest that omega-3 fatty acids derived from fish oil reduce CVD (Marik and Valon, 2009).

An Elderly Dietary Index (EDI) is useful for assessing risk factors for CVD in older adults (Kourlaba et al, 2009). Because kidney disease is a risk factor for mortality and CVD in older adults, elevated cystatin C and albuminuria are independent, graded risk factors for CVD and mortality (Rifkin et al, 2009).

Nutrition counseling should receive high priority, both in medical training and in patient care for both men and women (Krummel, 2008). The American Dietetic Association estimates cost savings per cardiovascular case to be nearly \$2500 annually with nutrition counseling, thereby reducing the need for many medications. Key components of counseling include: (1) reduced caloric intake; (2) reduced total fat, saturated fat, trans fat, and Chol with proportional increases in monounsaturated, omega-3, and omega-6 fatty acids; (3) increased dietary fiber, fruit, and vegetables; (4) increased micronutrients (e.g., folate and vitamins B<sub>6</sub> and B<sub>12</sub>); (5) increased plant protein in lieu of animal protein; (6) reduced portions of highly processed foods; (7) adopting a Mediterranean dietary pattern; (8) adding physical activity; and smoking cessation (Forman and Bulwer, 2006). See Table 6-1 regarding evidence for dietary recommendations in heart disease.

The Women's Nutrition Intervention Study used a low-fat eating plan that serves as a model for implementing a long-term dietary intervention in clinical practice (Hoy et al, 2009). Table 6-2 lists other influential factors on diet and its relationship to heart disease. In the future, the development of functional foods that contain ingredients that have preventive benefits will be important. Products that contain green tea with Epigallocatechin-3-gallate (EGCG), omega-3 fatty acids, folate, vitamins C and E, flavonoids such as quercetin, eritadenine in mushrooms will be popular (Ferguson, 2009). Table 6-3 provides a list of commonly used herbs and botanical products in heart disease.



**TABLE 6-1 Levels of Best Evidence in Dietary Recommendations for Heart Disease<sup>a</sup>**

Dietary Recommendation	Evidence Level	Dietary Recommendation	Evidence Level
<i>Antioxidants</i>		<i>Omega-3 Fatty Acids</i>	
Supplemental beta-carotene (60–200 mg/d) does not decrease the risk for cardiovascular death or nonfatal myocardial infarction (MI) in primary and secondary prevention patients.	I	Approximately 1 g/d of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) from a supplement or fish decreases the risk of death from cardiac events in patients with heart disease.	II
Supplemental vitamin E, given in both natural and synthetic forms, in doses of 30–600 mg/d or 400–800 IU/d, alone or in combination with other antioxidants, is not harmful but has not been shown to decrease the risk for cardiovascular death or MI.	II	Regular consumption of an average of two servings of fatty fish per week (about 3.5 oz per serving; high in EPA and DHA) is associated with a 30–40% reduced risk of death from cardiac events.	II
Supplemental vitamin E (100–1200 IU/d) alone or in combination with other antioxidants has not been shown to have a favorable or unfavorable effect on serum lipids.	II	Increased plasma levels and adipose tissue and cholesterol ester concentrations of alpha linolenic acid, EPA, and DHA have been associated with reduced risk of mortality.	II
Supplemental vitamin C (50–1000 mg/d) in combination with other antioxidants (vitamin E, beta-carotene, selenium) has not been shown to have any effect on cardiovascular death or MI.	II	<i>Nuts</i>	
Supplemental beta-carotene (60–120 mg/d) is associated with an increase in all-cause mortality and cardiovascular death in patients at increased risk for lung cancer.	II	Consumption of 50–113 g (1/2 to 1 cup) of nuts daily with a diet low in saturated fat may decrease total cholesterol by 4–21% and LDL cholesterol by 6–29% when weight is not gained.	II
Supplemental vitamins C and E, beta-carotene, and selenium should not be taken with simvastatin–niacin drug combinations because this may lower HDL cholesterol, a beneficial subfraction of HDL cholesterol.	II	Consumption of 5 oz of nuts per week is associated with reduced risk of CVD.	II
<i>Hypertension</i>		<i>Soy Protein</i>	
Consuming a diet rich in fruits and vegetables and low-fat dairy products and low in sodium and saturated fat will decrease blood pressure. Reductions have been 4–12 mm Hg in systolic and 1–3 mm Hg in diastolic blood pressure. This dietary pattern is enhanced by weight loss and increased physical activity.	I	Studies varied greatly in their estimation of the effect of diets low in saturated fat and cholesterol containing ~26–50 g of soyprotein either as food or as a soy supplement, with 0–165 mg of isoflavones. Studies of individuals with normal or elevated total cholesterol >200 mg/dL and individuals with diabetes varied, showing either 0–20% lower serum total cholesterol; 0–22% lower triglycerides; 4–24% lower LDL cholesterol.	II
<i>Fiber</i>		Diets containing up to 30 g of soy protein (as supplements) per day are well tolerated.	II
Consuming diets high in total fiber (17–30 g/d) and soluble fiber (7–13 g/d) as part of a diet low in saturated fat and cholesterol can further reduced total cholesterol by 2–3% and LDL by up to 7%.	I	<i>Statins, Stanols, and Sterols</i>	
Diets high in total dietary fiber (>25 g/d) are associated with decreased risk for coronary heart disease (CHD) and Cardiovascular disease (CVD).	II	Plant sterols and stanols are potent hypocholesterolemic agents. Daily consumption of 2–3 g (through margarine, low-fat yogurt, orange juice, breads, and cereals) lowers total cholesterol concentrations in a dose-dependent manner without changing HDL cholesterol or triacylglycerol concentrations.	I
<i>LDL Cholesterol Reduction</i>		For patients receiving statin therapy, plant stanols further reduce LDL and total cholesterol.	I
A diet consisting of 25–35% total fat, <7% saturated and trans fat, and <200 mg dietary cholesterol lowers serum total and LDL cholesterol 9–16% and decreases the risk of CHD.	I	The total and LDL cholesterol-lowering effects of stanols and sterols are evident even when sterols and stanols are consumed as part of a cholesterol-lowering diet.	I
Isocalorically replacing saturated fatty acids with MUFA and PUFA is associated with reductions in LDL cholesterol.	I	Sterols lower total cholesterol by 6–11% and LDL cholesterol by 7–15%. Stanols lower total cholesterol by 4–10% and LDL cholesterol by 7–14%.	II
		An intake of 2–3 g of plant sterols and stanols per day generally appears to be safe.	II

Adapted from: American Dietetic Association, Evidence analysis library. Web site accessed July 1, 2009, at <http://www.adaevidencelibrary.org/>. Key: Level I evidence = strong evidence from randomized controlled trials; level II evidence = moderate evidence.

**TABLE 6-2 Key Influences and Factors Related to Heart Disease**

Influence	Description
Alcohol	Moderate red wine consumption may be associated with desirable changes in HDL cholesterol. The “French paradox” suggests that wine intake and type of fat consumed are protective.
Alpha linolenic acid (ALA)	ALA in flaxseed, walnuts, and canola oil may protect against sudden cardiac death and cardiac arrhythmias.
Aspirin and salicylates	Aspirin (usually 80 mg/d) and other salicylates inhibit production of enzymes that influence platelet release and aggregation, vasoconstriction, and vasodilation. Salicylates have analgesic, antipyretic, and anti-inflammatory properties. They occur naturally in many foods, including herbs, spices, fruits, and tomatoes.
Apolipoprotein (Apo) E phenotype	ApoE genotype modifies the serum lipid response to changes in dietary fat and cholesterol intake. Inherited hypercholesterolemias are common disorders characterized by elevated LDL cholesterol levels and premature coronary heart disease.
Carbohydrate	High glycemic index foods should be studied further for their effects on heart disease.
C-reactive protein (CRP)	Inflammation is important in atherosclerosis. CRP is one of the acute phase proteins that increase during systemic inflammation. Dietary/lifestyle factors that decrease CRP levels are: weight loss, alpha linolenic acid, vegetarian diet, and moderate alcohol intake.
Cholesterol, total serum	The NHLBI promotes <200 mg/dL as desirable; 200–239 mg/dL is borderline high; >240 mg/dL is high. Age, lifestyle habits (smoking high BMI), and high serum cholesterol levels are consistently associated with CHD mortality. The ATP III report found that even older persons with established CHD can show benefit from LDL-lowering therapy.
Cholesterol, HDL	Low levels of HDL cholesterol are an independent risk factor for cardiovascular death; HDL <40 mg/dL is low and not desirable; >60 mg/dL is high and better. In women, changes in HDL cholesterol and triglyceride levels are good predictors of coronary risk.
Cholesterol, LDL (see Table 6-1)	Initiate therapeutic lifestyle changes (TLC) if LDL is above goal; this is a primary target of therapy. LDL <100 mg/dL is optimal; 100–129 mg/dL is near-optimal; 130–159 mg/dL is borderline high; 160–189 mg/dL is high; and >190 mg/dL is very high. Statins can help lower LDL levels.
Copper	Elevated serum copper levels are strong pro-oxidants. Supplementation is not recommended; the relationship of copper and zinc is complex and excesses are not desirable.
Dairy products	Low fat dairy products provide a major source of vitamins and minerals.
Diabetes	Elevated systolic and diastolic blood pressure, high serum cholesterol level, high body mass index, presence of diabetes, and smoking status are key risks for CHD. Replace saturated fat with monounsaturated fat.
Eggs	Limit intake to no more than 1 whole egg daily, especially with diabetes.
Erythrocyte sedimentation rate (ESR)	ESR is a marker of inflammation but whether this signifies an independent marker for heart disease remains to be seen.
Estrogen	High plasma triglycerides are an independent risk factor for CHD. Hormone replacement therapy may protect younger more than older women.
Exercise	Low fitness in adolescents and adults is common in the U.S. population and is associated with an increased prevalence of CVD. Increased exercise is associated with a lower waist circumference and higher HDL cholesterol levels. Inactive individuals benefit by even slightly increasing activity, such as walking.
Fats, monounsaturated (MUFA) (see Table 6-1)	Nuts are a good source of MUFA. Substitution of extra virgin olive oil for saturated fats will not only decrease SFA but provide phytochemicals.
Fats, saturated (SFA)	SFA is more important than total cholesterol intake in affecting total and LDL cholesterol levels, and risk of CHD, in women especially.
Fiber and whole grains (see Table 6-1)	Whole grains and high fiber from cereals, vegetables, and fruits are protective against CHD. Fiber may protect against CHD by lowering blood cholesterol (soluble fibers), attenuating blood triglyceride levels (mostly soluble fibers), decreasing hypertension (all fibers), and normalizing postprandial blood glucose levels (all fibers). Total fiber is important.
Flavonoids	There are over 6000 so far. Chocolate decreases blood pressure and enhances blood flow. Dark chocolate also improves insulin sensitivity. Soy isolate protein and green tea tend to be helpful in lowering CVD risks. Flavonoids in red wine, grape juice, grapefruit, tea, onions, apples, cloves, licorice, and sage are beneficial.
Folic acid	Diets that are low in folate and carotenoids (beta-carotene, lutein, zeaxanthin) contribute to increased coronary risk mortality.
Glycemic load, high	High glycemic load due to high intake of refined carbohydrates is positively related to CAD risk, independent of other known risk factors.
Homocysteine (tHcy)	Elevated tHcy levels are associated with increased risk of cardiac disease, stroke, and peripheral artery disease. Therapy with folic acid and vitamins B <sub>6</sub> and B <sub>12</sub> can reduce plasma tHcy levels. Fortified breakfast cereals that contain 200 µg folic acid are useful. The Dietary Approaches to Stop Hypertension (DASH) diet also reduces tHcy; the diet includes high quantities of fruits, vegetables, low-fat dairy products, whole grains, poultry, fish, and nuts.

(continued)

**TABLE 6-2 Key Influences and Factors Related to Heart Disease (continued)**

Influence	Description
Iron	High heme iron intake may increase risks of coronary heart disease (CHD) while anemia is damaging to the heart. A carefully balanced iron intake is a safe recommendation.
Mediterranean diet	A Mediterranean diet is protective. Mediterranean diets have a healthier balance between omega-3 and omega-6 fatty acids. The Mediterranean diet does not include much meat (high omega-6 fatty acids;) it emphasizes whole grains, fresh fruits and vegetables, fish, olive oil, garlic, and wine.
Metabolic syndrome	Atherogenic dyslipidemia is characterized by three lipid abnormalities: elevated triglycerides, small LDL particles, and reduced HDL cholesterol. Dyslipidemia, elevated blood pressure, impaired glucose tolerance, and central obesity comprise the metabolic syndrome. Prevention includes (1) correcting overweight by reducing energy density of the diet and (2) improving insulin sensitivity and associated metabolic abnormalities through a reduction of dietary saturated fat, partially replaced with MUFA and PUFA. Mild-to-moderate alcohol intake is protective while excessive intake is detrimental.
Methionine	Found in red meats, methionine may contribute to fatty plaque buildup by its relationship to elevated homocysteine.
Nicotinic acid	Niacin has a potent effect on high-density lipoprotein (HDL) cholesterol levels. Data on cardiovascular event rate reductions are limited.
Nuts and seeds (see Table 6-1)	Nuts and seeds provide monounsaturated fat, natural vitamin E, magnesium, and other heart-healthy nutrients. Walnuts and almonds are especially effective for reducing lipids.
Obesity	Individuals who are obese in middle age have a higher risk of hospitalization and mortality from CHD. A major goal of dietary prevention and treatment is to attain and maintain weight within a healthy body weight range. Decreasing excess calories, reducing total fat intake, adding fiber, reducing excess intake of refined carbohydrates, and increasing exercise can help achieve this goal.
Omega-3 fatty acids (see Table 6-1)	Both eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) in foods and supplements decrease production of inflammatory mediators. Omega-3 s may reduce CHD events. Japan has the lowest heart disease rate in the world; fish is a common part of that diet. RBC <sup>15</sup> N is a biomarker of EPA and DHA intake; it is rapid and inexpensive (O'Brien et al, 2009).
Oral health	Poor oral health may be a risk factor for CHD; improvement of periodontal status may influence the related systemic inflammation.
Phytosterols and stanols (see Table 6-1)	Plant stanol or sterol esters are phytosterols found in plant foods such as corn, soy, and other vegetable oils. Plant stanol esters block intestinal absorption of dietary and biliary cholesterol. Fat-soluble vitamins are not significantly affected. Sunflower kernels, pistachio nuts, sesame seeds and wheat germ are good sources.
Plasma lipoprotein (a) [Lp(a)]	Elevated plasma Lp(a) is an independent risk factor of heart disease. Ethnicity-related differences in Lp(a) levels exist.
Quercetin and antioxidants from fruits and vegetables	Vegetables, citrus fruits, seeds, olive oil, tea, and spices are antioxidant foods to include in the diet. Fruits are more protective than vegetables. Quercetin (in apples, onions) protects against CHD and hypertension. Pomegranate can lower cholesterol synthesis and spices and herbs can suppress inflammatory pathways.
Smoking	Both active smoking and secondary exposure are associated with the progression of atherosclerotic heart disease. Smoking is of greater concern among persons who also have diabetes mellitus and hypertension, or who drink a lot of alcohol (Cullen et al, 2009).
Soy protein (see Table 6-1)	The FDA permits labeling of products high in soy protein as helpful in lowering heart disease risk; products must contain at least 6.25 g per serving. Tofu contains 13 g of soy protein in one 4-oz serving; one soy burger contains 10–12 g of protein; 1/4 cup of soy nuts and 1/2 cup of tempeh contain 19 g of protein each.
Trans fatty acids (TFAs)	TFAs are strongly associated with systemic inflammation in patients with heart disease. Food labels must list the amount of TFAs.
Triglycerides	High levels of triglycerides are an independent risk factor of cardiovascular death. A low-fat diet can help.
Vitamin C (see Table 6-1)	Vitamin C has a role in cholesterol metabolism and affects levels of LDL cholesterol. Men, smokers, elderly individuals, and persons with diabetes or hypertension tend to have lower levels of serum ascorbic acid and higher risks for heart disease.
Vitamin E (see Table 6-1)	Alpha-tocopherol decreases lipid peroxidation and platelet aggregation and functions as a potent anti-inflammatory agent. Prospective human clinical trials with alpha-tocopherol have not shown effectiveness in lowering CHD risk.
Vitamin K	Vitamin K influences vascular health. Use darker greens or red leaf lettuce to increase vitamins A and K.

Cullen MW, et al. No interaction of body mass index and smoking on diabetes mellitus risk in elderly women. *Prev Med.* 48:74, 2009.

**TABLE 6-3 Commonly Used Herbs and Botanical Products in Heart Disease**

Chromium	Chromium is sometimes used for dyslipidemia. Do not use excesses of chromium with insulin or hypoglycemic agents because chromium may lower glucose levels excessively.
Coenzyme Q10 (CoQ10; ubiquinone)	Statins block production of farnesyl pyrophosphate, an intermediate in the synthesis of ubiquinone. Because CoQ10 and statins share a similar pathway, they can be taken simultaneously (Mabuchi et al, 2005; Strey et al, 2005). While there is insufficient evidence to prove the role of CoQ10 deficiency in statin-associated myopathy, there are no specific risks for this supplement. Do not take with gemfibrozil, tricyclic antidepressants, or warfarin.
L-arginine	Arginine appears to reduce endothelin, a protein that causes blood vessel constriction and is found in high amounts in HF patients; avoid use after an MI (Schulman et al, 2006).
Danshen	Used for ischemic heart disease. Avoid large amounts with warfarin, aspirin, and other antiplatelet drugs as it can increase risk of bleeding or bruising.
Fenugreek	This product may improve serum lipid levels slightly. Do not take with diuretics.
Garlic	Garlic may have short-term effects on blood lipids. Avoid use in large amounts with warfarin, aspirin, and other antiplatelet drugs because of increased risks of bleeding or bruising. It may also increase insulin levels with hypoglycemic results; monitor carefully in patients with diabetes.
Grapefruit	Grapefruit juice decreases drug metabolism in the gut (via P-450-CYP3A4 inhibition) and can affect medications up to 24 hours later. Consistency of use is more important than total quantity. Avoid taking with alprazolam, buspirone, cisapride, cyclosporine, statins, tacrolimus, and other cardiac drugs.
Guggul; Gugulipid	This yellowish resin from mukul myrrh tree is used in Indian Ayurveda medicine. It lowers low-density lipoprotein (LDL) and increases high-density lipoprotein (HDL) because of its plant sterols; it also stimulates the thyroid, is an anti-inflammatory, and works like an antioxidant. Gugulipid is the safest form, but a high dose is needed. Gastrointestinal discomfort may occur. Do not take with Inderal or Cardizem, and do not use during pregnancy or lactation.
Hawthorn	This is used for heart failure in Germany. Hawthorn should not be taken with digoxin, angiotensin-converting enzyme (ACE) inhibitors, and other cardiovascular drugs.
Niacin (nicotinic acid)	Do not take with statins, antidiabetic medications, or carbamazepine because of potentially serious risks of myopathy and altered glucose control.
Omega-3 fatty acids	Some studies support the role of DHA and EPA in preventing heart failure, but not with a high-fat diet (Shah et al, 2009). Fish oil capsules can cause hypervitaminosis A and D if taken in large doses. Avoid use in pregnant or lactating women. Avoid taking with warfarin, aspirin, and other antiplatelet medications because of the risk of increased bruising or bleeding.
Psyllium (metamucil)	Used to lower total and LDL cholesterol levels; evidence is slim.
Vitamin C and E, beta-carotene, and selenium	Do not take with simvastatin–niacin drug combinations because the combination of these antioxidants may lower HDL2 cholesterol, a beneficial subfraction of HDL cholesterol.
Vitamin E	Do not take with warfarin because of the possibility of increased bleeding. Avoid doses greater than 400 IU/d.

**REFERENCES**

- Mabuchi H, et al. Reduction of serum ubiquinol-10 and ubiquinone-10 levels by atorvastatin in hypercholesterolemic patients. *J Atheroscler Thromb*. 12:111, 2005.
- Schulman SP, et al. L-arginine therapy in acute myocardial infarction: the Vascular Interaction with Age in Myocardial Infarction (VINTAGE MI) randomized clinical trial. *JAMA*. 295:58, 2006.
- Shah KB, et al. The cardioprotective effects of fish oil during pressure overload are blocked by high fat intake. role of cardiac phospholipid remodeling [published online ahead of print Jul 13, 2009]. *Hypertension*. 54:65, 2009.
- Strey CH, et al. Endothelium-ameliorating effects of statin therapy and coenzyme Q10 reductions in chronic heart failure. *Atherosclerosis*. 179:201, 2005.

**SODIUM AND OTHER MINERALS**

A majority of Americans over age 60 have high BP with a shortened life expectancy. High BP affects over 73,600,000 in the United States (American Heart Association, 2009). Hypertension increases the risk for coronary artery disease (CAD), MI, stroke, renal failure, and HF. Careful attention to hypertension is essential in all ages and both sexes. African Americans and Hispanics of Caribbean descent tend to have a high prevalence of hypertension. Vitamin D plays a role in heart health (Martins et al, 2007). Increasing sun exposure or using a supplement may be indicated (Scragg et al, 2007). There are also many benefits in reducing sodium intake while increasing intakes of potassium, calcium, magnesium, and whole grains. Table 6-4 highlights key nutrients (folic acid, potassium, magnesium, and calcium) and provides heart healthy food choices based on the Dietary Approaches to Stop Hypertension (DASH) diet principles.

Excessive alcohol intake is one of the major causes of magnesium loss from various tissues, including the heart; mag-

nesium loss may represent a predisposing factor to the onset of alcohol-induced pathologies including stroke and cardiomyopathy (Romani, 2008). Rich sources of magnesium include nuts and seeds, soybeans, tofu, chocolate, dark-green vegetables, legumes, yogurt, wheat germ and dairy products. Although supplement use can help meet DRI levels for calcium, vitamin C and magnesium, supplementation may not work for potassium and may be too high for some other nutrients (Burnett-Hartman et al, 2009). In 2004, The Institute of Medicine of the National Academy of Sciences issued recommendations for intake of water and electrolytes that include the following suggestions:

- **CHLORIDE:** 2300 mg daily for adults to replace losses in perspiration.
- **POTASSIUM:** 4700 mg is needed to lower BP and reduce the risk of kidney stones and bone loss for most adults. No upper limit is set. African Americans may benefit. Natural sources are best.
- **SODIUM:** 1500 mg for adults aged 19–50; 1300 mg for adults aged 50–70; 1200 mg for adults aged 71 and over.



**TABLE 6-4 Key Sources of Folate, Potassium, Calcium, and Magnesium and the DASH Diet Principles**

<b>Folate Sources</b>	<b>Micrograms (μg)</b>	<b>Folate Sources</b>	<b>Micrograms (μg)</b>
Breakfast cereals fortified with 100% of the daily value (DV), 3/4 cup	400	Avocado, raw, all varieties, sliced, 1/2 cup sliced	45
Beef liver, cooked, braised, 3 oz	185	Peanuts, all types, dry roasted, 1 ounce	40
Cowpeas (blackeyes), immature, cooked, boiled, 1/2 cup	105	Lettuce, romaine, shredded, 1/2 cup	40
Breakfast cereals, fortified with 25% of the DV, 3/4 cup	100	Wheat germ, crude, 2 tablespoons	40
Spinach, frozen, cooked, boiled, 1/2 cup	100	Tomato juice, canned, 6 oz	35
Great Northern beans, boiled, 1/2 cup	90	Orange juice, chilled, includes concentrate, 3/4 cup	35
Asparagus, boiled, 4 spears	85	Turnip greens, frozen, cooked, boiled, 1/2 cup	30
Rice, white, long-grain, parboiled, enriched, cooked, 1/2 cup	65	Orange, all commercial varieties, fresh, 1 small	30
Vegetarian baked beans, canned, 1 cup	60	Bread, white, 1 slice	25
Spinach, raw, 1 cup	60	Bread, whole wheat, 1 slice	25
Green peas, frozen, boiled, 1/2 cup	50	Egg, whole, raw, fresh, 1 large	25
Broccoli, chopped, frozen, cooked, 1/2 cup	50	Cantaloupe, raw, 1/4 medium	25
Egg noodles, cooked, enriched, 1/2 cup	50	Papaya, raw, 1/2 cup cubes	25
Broccoli, raw, 2 spears (each 5-in long)	45	Banana, raw, 1 medium	20

From: U.S. Department of Agriculture, Agricultural Research Service. USDA national nutrient database for standard reference, Release 16. 2003. Nutrient Data Laboratory Home Page, accessed July 7, 2009, at [http://www.nal.usda.gov/fnic/cgi-bin/nut\\_search.pl](http://www.nal.usda.gov/fnic/cgi-bin/nut_search.pl).

<b>Potassium Sources</b>	<b>Milligrams (mg)</b>	<b>Potassium Sources</b>	<b>Milligrams (mg)</b>
Apricots, 3 medium	272	Orange juice, 8 oz	473
Artichoke, 1 cup, raw	644	Papaya, 1 whole	781
Avocado, Jerusalem, 1 medium	976	Potato, baked with skin, medium	1081
Banana, 1 cup	537	Pumpkin, 1 cup, cooked	564
Beans, canned white, 1 cup	1189	Prunes (dried plums), 1 cup, stewed	796
Beet greens, boiled, 1/2 cup	653	Prune juice, 1 cup	707
Broccoli, 1 cup chopped	457	Raisins, 1/3 cup	362
Cantaloupe, 1 cup	427	Refried beans, canned, 1 cup	673
Grapefruit juice, sweetened, 1 cup	405	Spinach, 1 cup, cooked	574
Halibut, cooked, 1/2 fillet	916	Sweet potato, canned, 1 cup	796
Kidney beans, 1 cup	713	Tomato, 1 medium	426
Kiwifruit, 1 medium	252	Tomato juice, 6 oz	417
Lima beans, cooked, 1 cup	955	Tomato puree, 1/2 cup	1328
Milk, 1 cup, skim	382	Tomato sauce, 1 cup	940
Milk, 1 cup, chocolate	425	Tropical trail mix, 1 cup	993
Milkshake, 16 oz, vanilla	579	Vegetable juice cocktail, 1 cup, canned	467
Nectarine, 1 medium	273	Winter squash, 1 cup	896
Orange, 1 medium	237	Yogurt, 8 oz, low fat	443

For other sources. Web site accessed July 7, 2009, at <http://www.nal.usda.gov/fnic/foodcomp/Data/SR17/wtrank/sr17w306.pdf>.

<b>Calcium Sources</b>	<b>Milligrams (mg)</b>	<b>Calcium Sources</b>	<b>Milligrams (mg)</b>
Broccoli, cooked, 1 cup	156	Collards, 1 cup, cooked	266
Cheddar cheese, 1 oz	204	Egg nog, 1 cup	330
Cheese sauce, 1 cup	756	Enchilada with cheese, 1	324
Cheese, Swiss, 1 oz	224	Milk, canned evaporated, 1 cup	742
Clam chowder, New England, 1 cup	186	Milk, fluid, 1%, 1 cup	290
Milk, fluid, chocolate, low fat, 1 cup	288	Tofu made with calcium, 1/4 block	164

(continued)

**TABLE 6-4 Key Sources of Folate, Potassium, Calcium, and Magnesium and the DASH Diet Principles (continued)**

Calcium Sources	Milligrams (mg)	Calcium Sources	Milligrams (mg)
Milkshake, thick, vanilla, 11 oz	457	Total brand cereal, 3/4 cup	1104
Molasses, blackstrap, 1 tablespoon	172	Sardines, canned with bones, 3 oz	325
Pudding, chocolate, 4 oz, ready to serve	102	Spinach, canned, 1 cup	272
Ricotta cheese, part skim, 1 cup	669	Turnip greens, frozen, cooked, 1 cup	249
Soybeans, green, cooked, 1 cup	261	Yogurt, low fat with fruit, 1 cup	345

For a more specific list, Web site accessed July 7, 2009, at <http://www.nal.usda.gov/fnic/foodcomp/Data/SR17/wtrank/sr17w301.pdf>.

Magnesium Sources	Milligrams (mg)	Magnesium Sources	Milligrams (mg)
Barley, pearled, raw 1 cup	158	Plantain, raw, 1 medium	66
Beans, canned white, 1 cup	134	Seeds, pumpkin or squash seed kernels, 1 oz (142 seeds)	151
Broccoli, cooked, 1 cup	33	Soybeans, mature, cooked, 1 cup	148
Cereal, All-Bran, 1/2 cup	109	Spinach, cooked, 1 cup	163
Chocolate candy, semisweet, 1 cup	193	Tomato paste, 1 cup	110
Halibut, cooked, 1/2 fillet	170	Trail mix, with chocolate chips, nuts, seeds, 1 cup	235
Nuts, Brazil, 6-8	107	Whole-grain wheat flour, 1 cup	166
Oat bran, 1 cup	221		
Okra, cooked, 1 cup	94		

For a more specific list, Web site accessed July 7, 2009, at <http://www.nal.usda.gov/fnic/foodcomp/Data/SR17/wtrank/sr17w304.pdf>.

DASH Diet Principles	Food
Vegetables, choose 4-5 servings daily	Carrots, sweet potatoes, pumpkin, winter squash Green leafy vegetables (broccoli, kale, cabbage, etc.), green beans Tomato salsas, 6-oz servings of tomato juice or other vegetable juices
Fruits, choose 4-5 servings daily	Fresh fruits, including apples, bananas, cantaloupe, melons, berries Red or black grapes; grape juice (1 cup per day) Grapefruit, especially pink (40% more beta-carotene) Dried fruits, especially apricots, dates, prunes Pomegranates and other antioxidant juices (blueberry juice, red wine, orange juice, cranberry juice, green tea)
Protein-rich foods, choose 2 or less	Lean chicken and turkey breast Salmon and other fish Meats that are lean or have fat trimmed away
Low-fat dairy, 2-3 servings daily	Skim milk and yogurt (8 oz) Low-fat cheeses (1-1/2 oz per serving)
Low-fat foods	Tomato sauces with pasta Homemade pizza with low-fat toppings (chicken, vegetables, low-fat cheese)
Grains, choose 7-8 servings daily	Oatmeal, shredded wheat; high-fiber, low-sugar cereals Baked whole-wheat chips and tortillas Whole-grain breads and pastas, wheat germ
Nuts, seeds, and dry beans, 4-5 servings per week	Peanuts, walnuts, almonds, pistachios, other nuts in moderation Pumpkin seeds, sunflower seeds, sesame seeds Bean and chickpea dishes and dips
Oils, 2-3 servings	Olive oil and canola oil substituted for other oils Salad dressings and dips with nonfat sour cream or homemade yogurt

**NOTE:** Most of these foods are recommended in both the DASH and Mediterranean diets.

For more information, see Web site accessed July 7, 2009, at [http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new\\_dash.pdf](http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash.pdf).

Highly active people may need more. If sodium sensitive, intake may need to be lower. Upper limit (UL) is set at 2.3 g sodium/d; over 95% of the American public consumes sodium above the UL level on a regular basis.

- **WATER:** 91 oz daily for women, 125 oz daily for men; more in hot climates or with physical activity. Drinking fluids with and between meals according to thirst is usually sufficient, although seniors may lose their thirst sensation. Beverages provide 80% of daily intake, 20% comes from food.

#### For More Information

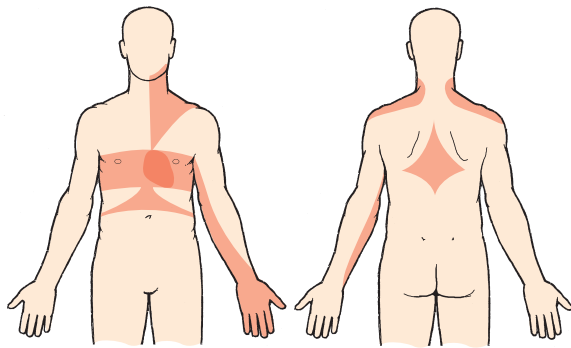
- American Association of Cardiovascular Pulmonary Rehabilitation <http://www.aacvpr.org/>
- American Heart Association <http://www.americanheart.org/>

#### LIPIDS—CITED REFERENCES

- American Heart Association. Accessed June 7, 2009, at <http://www.americanheart.org>.
- Anderson C. Dietary modification and CVD prevention: a matter of fat. *JAMA*. 295:693, 2006.
- Bhatt DL, et al. International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. *JAMA*. 295:180, 2006.
- Burnett-Hartman AN, et al. Supplement use contributes to meeting recommended dietary intakes for calcium, magnesium, and vitamin C in four ethnicities of middle-aged and older Americans: the Multi-Ethnic Study of Atherosclerosis. *J Am Diet Assoc*. 109:422, 2009.
- Danaei G, et al. The preventable causes of death in the United States: comparative risk assessment of dietary, lifestyle, and metabolic risk factors. *PLoS Med*. 6(4):e1000058, 2009.
- Ferguson LR. Nutrigenomics approaches to functional foods. *J Am Diet Assoc*. 109:452, 2009.
- Forman D, Bulwer BE. Cardiovascular disease: optimal approaches to risk factor modification of diet and lifestyle. *Curr Treat Options Cardiovasc Med*. 8:47, 2006.
- Howard BV, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*. 295:655, 2006.
- Hoy MK, et al. Implementing a low-fat eating plan in the Women's Intervention Nutrition Study. *J Am Diet Assoc*. 109:688, 2009.
- Kendrick J, et al. 25-Hydroxyvitamin D deficiency is independently associated with cardiovascular disease in the Third National Health and Nutrition Examination Survey. *Atherosclerosis*. 205:255, 2009.
- Kourlaba G, et al. Development of a diet index for older adults and its relation to cardiovascular disease risk factors: the Elderly Dietary Index. *J Am Diet Assoc*. 109:1022, 2009.
- Krummel D. Nutrition in cardiovascular disease. In: Mahan K, Escott-Stump S, eds. *Krause's food, nutrition, and diet therapy*. 12th ed. Philadelphia, PA: WB Saunders, 2008.
- Marik PE, Varon J. Omega-3 dietary supplements and the risk of cardiovascular events: a systematic review. *Clin Cardiol*. 32:365, 2009.
- Martins D, et al. Prevalence of cardiovascular risk factors and the serum levels of 25-hydroxyvitamin D in the United States: data from the Third National Health and Nutrition Examination Survey. *Arch Intern Med*. 167:1159, 2007.
- Rifkin DE, et al. Albuminuria, impaired kidney function and cardiovascular outcomes or mortality in the elderly [published online ahead of print 2009]. *Nephrol Dial Transplant*. 25:1560, 2010.
- Romani AM. Magnesium homeostasis and alcohol consumption. *Magnes Res*. 21:197, 2008.
- Scragg R, et al. Serum 25-hydroxyvitamin D, ethnicity, and blood pressure in the Third National Health and Nutrition Examination Survey. *Am J Hypertens*. 20:713, 2007.

## ANGINA PECTORIS

### NUTRITIONAL ACUITY RANKING: LEVEL 1



#### DEFINITIONS AND BACKGROUND

Angina pectoris involves retrosternal chest pain or discomfort from decreased blood flow to the myocardium from decreased oxygen supply (often during exertion). Traditional risk factors include tobacco use, hypertension, diabetes mellitus, dyslipidemia, obesity, sedentary lifestyle, atherogenic diet; high-sensitivity C-reactive protein (hsCRP), lipoprotein (a), and elevated homocysteine (tHcy). Angina can also occur from anemia, hyperthyroidism, aortic stenosis, or vasospasm. In hypertrophic cardiomyopathy (HCM), an area of abnormally thick heart muscle impairs the heart's

pumping action and causes angina during or shortly after exercise.

Stable (classic) angina occurs after exertion and is relieved by rest and vasodilation; it lasts 3–5 minutes. Intractable (progressive) angina causes chronic chest pain that is not relieved by medical treatment. Variant angina is a mixed condition. If diagnosed early, the chance of living longer than 10–12 years is at least 50%. A very low-fat diet (i.e., 10% fat calories) has a substantial impact (Griel and Kris-Etherton, 2006). In addition, vitamin D provision through sun exposure or supplementation may be indicated when serum levels are below 20 ng/L (Kendrick et al, 2009).

An “ABCDE” approach is effective: “A” for antiplatelet therapy, anticoagulation, angiotensin-converting enzyme (ACE) inhibition, and angiotensin receptor blockade; “B” for beta-blockade and BP control; “C” for Chol treatment and cigarette-smoking cessation; “D” for diabetes management and diet; and “E” for exercise (Gluckman et al, 2005). Management of factors leading to the metabolic syndrome is also recommended (see Table 6-5). Cardiac rehabilitation helps to improve aerobic exercise capacity, physical functioning, and mental depression. Invasive treatments for chronic stable angina are only needed in a small number of patients (Kirwan et al, 2005). Currently, use of percutaneous coronary intervention (PCI) improves quality of life by relieving angina in patients with stable CAD (Brar and Stone, 2009).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** There are shared genetic pathways for angina pectoris (AP) and CHD death among both sexes; AP is important as a risk factor for CHD death (Zdravkovic et al, 2007).

<b>Clinical/History</b>	Electrocardiogram (ECG)	CoQ10 serum levels
Height	Radionucleotide imaging	Serum folate
Weight	Exercise stress test	Glucose (Gluc)
Body mass index (BMI)	Coronary angiography	Hemoglobin and hematocrit (H & H)
Waist circumference	<b>Lab Work</b>	Serum Fe, ferritin
Recent weight changes (e.g., gain)	Chol—LDL, HDL, total	Total iron-binding capacity (TIBC)
Diet history	Advanced lipid testing—	Aspartate aminotransferase (AST)
Chest pain	lipoprotein particle size	Alanine aminotransferase (ALT)
Shortness of breath	Triglycerides (TGs)	Transferrin
Sweating, nausea, vertigo	Lactate dehydrogenase (LDH)	Na <sup>+</sup> , K <sup>+</sup>
Ache in neck or jaw, earache	tHcy	Ca <sup>++</sup> , Mg <sup>++</sup>
Numbness or burning sensations	C-reactive protein (CRP)	Alkaline phosphatase (Alk phos)
Pulse (NL = 60–100 beats/min)		
BP		
Intake and output (I & O)		

**TABLE 6-5 Signs of the Metabolic Syndrome (Any Three of the Following)**

Risk Factor	Defining Level
Abdominal obesity <sup>a</sup>	Waist circumference <sup>b</sup>
Men	>102 cm (>40 in)
Women	>88 cm (>35 in)
Triglycerides	≥150 mg/dL
HDL cholesterol	
Men	<40 mg/dL
Women	<50 mg/dL
Blood pressure	≥130/≥85 mm Hg
Fasting glucose	≥110 mg/dL

From: National Heart, Lung, and Blood Institute. Web site accessed July 7, 2009, at <http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.htm#Step1>.

<sup>a</sup>Overweight and obesity are associated with insulin resistance and the metabolic syndrome. Abdominal obesity is more highly correlated with the metabolic risk factors than is an elevated BMI. Simple measure of waist circumference is recommended to identify the body weight component.

<sup>b</sup>Some male patients can develop multiple metabolic risk factors when the waist circumference is only marginally increased, e.g., 94–102 cm (37–39 in). Such patients may have a strong genetic contribution to insulin resistance. They should benefit from changes in life habits.

## INTERVENTION



### OBJECTIVES

- Relieve chest pain. Improve circulation to the heart.
- Increase activity as tolerated. Gradually increase exercise, especially through programs in cardiac rehabilitation.
- Maintain adequate rest periods.
- Maintain weight or lose weight if obese. A conventional dietetic intervention with weight loss helps to reduce pain frequency.
- Reduce symptoms of the metabolic syndrome where present (see Table 6-5).
- Avoid constipation with straining.
- Control BP and lower elevated serum Chol.



## FOOD AND NUTRITION

- Small, frequent feedings rather than three large meals are indicated.
- Increase fiber as tolerated; include an adequate fluid intake. Increase intake of fruits.
- Restrict saturated fats, dietary Chol, and sodium as necessary according to the individual profile. A very low-fat diet can be quite effective (Griel and Kris-Etherton, 2006).
- Limit stimulants such as caffeine to less than 5 cups of coffee or the equivalent daily. Energy drinks may contain 50–500 mg of caffeine.
- Promote calorie control if overweight; modify by age and sex.
- If tHcy levels are high, add more foods with folic acid, vitamins B<sub>6</sub> and B<sub>12</sub> to the diet.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Overweight

**Assessment Data:** BMI >90 percentile for age; complaints of heartburn and chest pain; diet hx shows intake of energy about 600 kcals extra per day and BMI 28.

**Nutrition Diagnoses (PES):** Overweight related to excessive energy intake as evidenced by BMI of 28 and limited physical activity.

**Interventions:** Education about the role of weight and heart disease, including angina. Counseling about desirable energy intake for age and activity level; ways to gradually increase physical activity.

**Monitoring and Evaluation:** BMI closer to desirable range; tolerance of physical activity; adequate drug therapy for angina.



- A Mediterranean diet that is rich in alpha linolenic acid (ALA) is effective (Estruch et al, 2006). It is prudent to increase intake of olive, soybean, and canola oils, seeds and nuts, including walnuts, almonds, macadamias, pecans, peanuts, and pistachios. Walnuts contain ALA; almonds are a good source of vitamin E. Nuts also contain flavonoids, phenols, phytosterols, saponins, elegiac acid, folic acid, magnesium, copper, potassium, and fiber. Pistachios, sunflower kernels, sesame seeds, and wheat germ are highest in phytosterols; use often.
- Beta-carotene supplements actually seem to increase angina. Dietary sources of carotenoids are a healthier choice.

### Common Drugs Used and Potential Side Effects

- Relief comes from use of tricyclic agents, beta-blockers, statins, or ACE inhibitors (Bugiardini and Bairey Mertz, 2005). Isosorbide (Isordil or Imdur) may cause nausea, vomiting, or dizziness; take on an empty stomach. Nadolol (Corgard) is a beta-blocker; it may cause weakness. Disopyramide (Norpace) may cause abdominal pain, nausea, or constipation.
- Antiplatelet therapy or anticoagulation therapy will be prescribed. Monitor for vitamin K intake with warfarin.
- Calcium channel blockers (verapamil [Calan], nifedipine, or diltiazem [Cardizem]) are used to dilate coronary arteries and slow down nerve impulses through the heart, thereby increasing blood flow. Nausea, edema, or constipation may be side effects. Take on an empty stomach. These drugs may also cause HF or dizziness; avoid taking with aloe, buckthorn bark and berry, cascara, and senna leaf. With nifedipine (Procardia), nausea, weakness, dizziness, and flatulence may occur; take after meals.

### Herbs, Botanicals, and Supplements

- The patient should not take herbals and botanicals without discussing with the physician. See Table 6-3.



#### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The patient will require stress management, activity, and education about proper eating habits.

- Discuss the role of nutrition in maintenance of wellness and in CVD. Discuss in particular: fiber, total fat intake, potassium and sodium, calcium and other nutrients, and caffeine.
- Discuss the importance of weight control in reduction of cardiovascular risks.
- Elevate the head of the bed 30–45° for greater comfort.
- Unstable angina is dangerous and should be treated as a potential emergency; new, worsening, or persistent chest discomfort should be evaluated in a hospital emergency department or “chest pain unit” and monitored carefully for acute MI (heart attack), severe cardiac arrhythmia, or cardiac arrest leading to sudden death. Dyspnea is a key factor and should be addressed (Arnold et al, 2009).

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

#### For More Information

- American Heart Association—Angina  
<http://www.americanheart.org/presenter.jhtml?identifier=4472>
- Medicine Net—angina  
<http://www.medicinenet.com/angina/article.htm>
- NHLBI—angina  
[http://www.nhlbi.nih.gov/health/dci/Diseases/Angina/Angina\\_WhatIs.html](http://www.nhlbi.nih.gov/health/dci/Diseases/Angina/Angina_WhatIs.html)

### ANGINA PECTORIS—CITED REFERENCES

- Arnold SV, et al. The impact of dyspnea on health-related quality of life in patients with coronary artery disease: results from the PREMIER registry. *Am Heart J*. 157:1042, 2009.
- Brar SS, Stone GW. Advances in percutaneous coronary intervention. *Curr Cardiol Rep*. 11:245, 2009.
- Bugiardini R, Bairey Mertz CN. Angina with “normal” coronary arteries: a changing philosophy. *JAMA*. 293:477, 2005.
- Estruch R, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. *Ann Intern Med*. 145:1, 2006.
- Gluckman TJ, et al. A simplified approach to the management of non-ST-segment elevation acute coronary syndromes. *JAMA*. 293:349, 2005.
- Griel AE, Kris-Etherton PM. Beyond saturated fat: the importance of the dietary fatty acid profile on cardiovascular disease. *Nutr Rev*. 64:257, 2006.
- Kirwan BA, et al. Treatment of angina pectoris: associations with symptom severity. *Int J Cardiol*. 98:299, 2005.
- Zdravkovic S, et al. Genetic influences on angina pectoris and its impact on coronary heart disease. *Eur J Hum Genet*. 15:872, 2007.

## ARTERITIS

### NUTRITIONAL ACUITY RANKING: LEVEL 1



#### DEFINITIONS AND BACKGROUND

Arteritis involves inflammation of artery walls with decreased blood flow. **Cranial arteritis** (temporal or giant-cell arteritis) yields chronically inflamed temporal arteries

with a thickening of the lining and a reduction in blood flow; this condition is linked to polymyalgia rheumatica (PMR). Women older than 55 years of age are twice as likely to have the condition compared with other people. The greatest danger is permanent blindness or stroke.

**Buerger's disease** involves an arteritis that causes limb pain and numbness. **Periarteritis nodosa** is an autoimmune disease that can affect any artery in the body. A rare form, **Takayasu's arteritis**, affects the mesenteric artery and creates local ischemia; IL-8 may be involved. Patients with **giant cell arteritis** (GCA) are at risk for developing extra-cranial large vessel inflammation. GCA is a medical emergency with neuro-ophthalmic complications and permanent vision loss in up to a fifth of patients (Borg and Dasgupta, 2009).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Tumor necrosis factor appears to influence susceptibility and interleukin (IL)-1 receptor antagonist seems to play a role in the pathogenesis. Other genetic markers are being studied.

<b>Clinical/History</b>	Jaw pain	CoQ10 serum levels
Height	Muscular aches	Na <sup>+</sup> , K <sup>+</sup>
Weight	Abnormal arterial biopsy	Ca <sup>++</sup> , Mg <sup>++</sup>
BMI	(necrotizing vasculitis with	Gluc
Diet history	granulomatous proliferation)	Albumin (Alb) or transthyretin
BP	Ultrasonography	Creatinine kinase (CK)
Temperature (mild fever?)		Transferrin
Severe, throbbing headache (temples, back of the head)	<b>Lab Work</b>	Chol—total, HDL, LDL
Red, swollen, painful temporal artery	White blood cell count (WBC) (increased)	Trig
Anorexia, weight loss	Erythrocyte sedimentation rate (ESR) >50 mm/h	H & H (often decreased)
Scalp tenderness	CRP	Serum Fe, ferritin
Dysphagia		tHcy level
Hearing problems or vision changes		Serum folate and B <sub>12</sub>

## INTERVENTION



### OBJECTIVES

- Prevent stroke and blindness, which are potential complications.
- Reduce inflammation.
- Promote increased blood flow through the affected vessels.
- Modify intake according to requirements and coexisting problems such as hypertension.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Dysphagia

**Assessment Data:** Weight loss, dysphagia and jaw pain, throbbing headache.

**Nutrition Diagnoses (PES):** Dysphagia related to arteritis with jaw pain as evidenced by weight loss, complaints of difficulty swallowing solids, choking on thin liquids.

**Interventions:** Education about blending foods and simplifying meal planning to increase intake. Swallowing evaluation for determination of appropriate textures and thickening of liquids. Counseling on positioning at mealtime to reduce likelihood of aspiration.

**Monitoring and Evaluation:** Improved intake for meals and snacks; fewer complaints of difficulty swallowing. No further weight loss.



## FOOD AND NUTRITION

- Follow usual diet, with increased calories if patient is underweight or decreased calories if the patient is obese.
- Reduce excess sodium and total fat intake; monitor regularly. Increase intake of fruits.
- Patient may need to include carnitine in the diet. Although not yet proven, it may be reasonable to include in the diet more sources of vitamins E, B<sub>6</sub>, and B<sub>12</sub>, riboflavin, and folic acid or to use a multivitamin supplement that includes sufficient amounts.
- With steroids, decreased sodium intake with higher potassium intake may be needed. Adequate to high protein may also be necessary. Monitor for glucose intolerance.
- Omega-3 fatty acids may be used to reduce inflammation. Include salmon, tuna, sardines, mackerel, walnuts, and related foods.
- Treat with supplements of folic acid, vitamins B<sub>6</sub> and B<sub>12</sub> to reduce elevated tHcy concentrations.

## Common Drugs Used and Potential Side Effects

- The mainstay of therapy remains corticosteroids (Borg and Dasgupta, 2009). High-dose prednisone successfully controls the inflammatory process. Side effects include elevated glucose and decreased nitrogen levels, especially with long-term use.
- Methotrexate is being evaluated but more research is needed.
- Low-dose aspirin is sometimes recommended.

## Herbs, Botanicals, and Supplements

- The patient should not take herbals or botanicals without discussing with the physician. See Table 6-3.



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of nutrition in the maintenance of health for CVD.

- Discuss the effects of medications on nutritional status and appetite.
- If dysphagia is present, discuss ways to alter texture or liquid consistencies.
- With long-term use of prednisone, monitor for osteoporosis, cataracts, easy bruising, elevated glucose levels.

#### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

#### For More Information

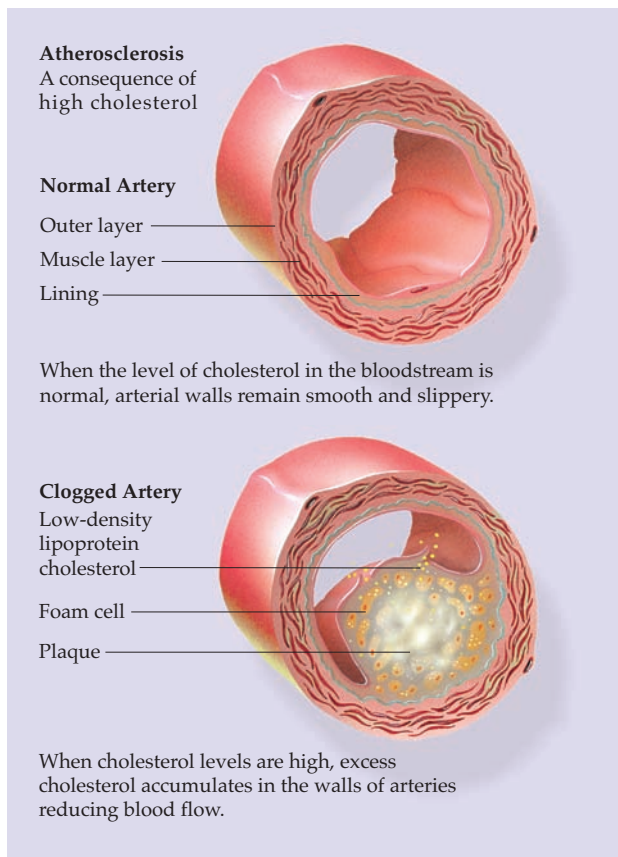
- American Autoimmune Association  
<http://www.aarda.org/>
- Emedicine: Temporal arteritis  
<http://emedicine.medscape.com/article/809492-overview>
- Giant Cell Arteritis  
<http://www.mayoclinic.com/health/giant-cell-arteritis/DS00440>
- Takayasu Arteritis Foundation  
<http://www.takayasu.org/>

#### ARTERITIS—CITED REFERENCE

Borg FA, Dasgupta B. Treatment and outcomes of large vessel arteritis. *Baillieres Best Pract Res Clin Rheumatol*. 23:325, 2009.

## ATHEROSCLEROSIS, CORONARY ARTERY DISEASE, DYSLIPIDEMIA

### NUTRITIONAL ACUITY RANKING: LEVEL 3



lations occur; the heart, brain, and leg arteries are most often affected. Metabolic syndrome is prevalent (see Table 6-5). Obesity leads to a proinflammatory and prothrombotic state that potentiates atherosclerosis (Moller and Kaufman, 2005). Vascular lipid accumulation and inflammation are hallmarks of atherosclerosis. As precursors to prostaglandins, thromboxanes, leukotrienes, lipoxins, and resolvins, essential fatty acids (EFAs) have significant clinical implications in hypertension, CHD and atherosclerosis (Das, 2006). The initial insult in adipose inflammation is mediated by macrophage recruitment and endogenous activation of Toll-like receptors, perpetuated through chemokine secretion, adipose retention of macrophages, and elaboration of pro-inflammatory adipocytokines (Shah et al, 2009). Then, paracrine and endocrine adipose inflammatory events induce a local and systemic inflammatory, insulin-resistant state promoting dyslipidemia and CVD (Shah et al, 2009).

**CAD** occurs when the coronary arteries that supply blood to the heart muscle become hardened and narrowed due to the buildup of plaque on the inner walls or lining of the arteries. Blood flow to the heart is reduced as plaque narrows the coronary arteries and diminishes oxygen supply to the heart muscle. CAD is common and is most often caused by smoking, high BP, low HDL Chol, family history of early CHD, and age (see Tables 6-1 and 6-2 for a long list of dietary and other relevant factors). Obesity, diabetes, and insulin resistance are also risks. Elevated plasma tHcy is an independent risk factor and a strong predictor of mortality in patients with CAD (Ntaios et al, 2010). Angioplasty is safe and as effective as bypass surgery for treatment of CAD.

**Ischemia** is a local and temporary deficiency of blood caused by obstruction, as from thrombosis. People with ischemic heart disease benefit from diets high in monounsaturated



#### DEFINITIONS AND BACKGROUND

**Atherosclerosis** involves progressive narrowing of the arterial tree, giving rise to collateral vessels. Fat-deposit accumu-

fatty acids (MUFA), omega-3 fatty acids, whole grains, vitamin E, wine, vegetables, and fruits.

**Dyslipidemia** involves hypertriglyceridemia and low levels of high-density lipoprotein Cholesterol. These imbalances in individual lipid components contribute to the increased risk of CAD. Serum lipid reductions decrease CAD risk; for each 1% reduction in serum Cholesterol, there is a 2% reduction in CAD risk.

Cholesterol screening is recommended, even for older adults. When treated, risks decrease significantly; therefore, early nutritional intervention is beneficial. In addition to BMI, use waist circumference or waist-to-hip ratio (WHR) to assess obesity and CVD risk since BMI alone is not a good predictor of CVD risk in persons over 65 years of age (ADA Evidence Analysis Library, 2009). In addition, correction of dyslipidemia in patients after coronary artery bypass graft (CABG) surgery prevents progression of atherosclerosis.

A therapeutic lifestyle modification program is effective. Nutrition and physical activity interventions have the potential to dramatically reduce the risks. Dietary counseling and education by a registered dietitian are associated with improved diet-related outcomes, and sufficient time to consult with a dietitian should be planned in cardiac rehabilitation (Locklin Holmes et al, 2005). Referral to a registered dietitian for medical nutrition therapy (MNT) is recommended whenever an individual has an abnormal lipid profile or CHD, with a planned initial visit lasting from 45–90 minutes and at least two to six planned follow-up visits of 30–60 minutes each (ADA Evidence Analysis Library, 2009).



## ASSESSMENT, MONITORING, AND EVALUATION



## CLINICAL INDICATORS

**Genetic Markers:** Lipid alterations have a polygenic basis. Family history is the most significant independent risk factor for CAD. In the Multi-Ethnic Study of Atherosclerosis (MESA) the lipid-related HMG-CoA reductase (HMGCR) variations differed greatly among ethnicities (Chen et al, 2009). Another study identified 30 distinct loci with lipoprotein concentrations variants (Kathiresan et al, 2009). ApoE and lipoprotein lipase (LPL) genes have been known for some time. In addition, elevated plasma tHcy can result from genetic errors, including methylenetetrahydrofolate reductase (MTHFR) C677T and A1298C polymorphisms (Freitas et al, 2008). Genetic testing is needed in high-risk population groups (Olthoff and Verhoef, 2005).

<b>Clinical/History</b>	Waist circumference >102 cm (40 in) in men and >88 cm (35 in) in women	Diet history BP Pancreatitis? Xanthomas?
Height		
Weight		
BMI		

## Lab Work

CRP	LDL Cholesterol: Goal <100 mg/dL	Serum B <sub>6</sub>
CoQ10 serum levels	Advanced lipid testing—small particle size	H & H
Trig: Goal below 150 mg/dL	tHcy: serum and urinary	Serum Fe
TC (often increased)	Serum and urinary folate	Na <sup>+</sup> , K <sup>+</sup>
HDL: Goal >40 mg/dL in men and >50 mg/dL in women	Serum and urinary B <sub>12</sub>	Ca <sup>++</sup> , Mg <sup>++</sup>
		Gluc
		AST, ALT
		Serum copper

## INTERVENTION



## OBJECTIVES

- Use a team approach to support the best possible outcomes: doctors, nurses, dietitians, other therapists.
- Improve LDL and HDL Cholesterol levels to prevent formation of new lesions. Lower elevated Cholesterol levels (>200 mg/dL) and Triglyceride levels (>200 mg/dL) using the TLC diet from the National Heart, Lung, and Blood Institute.
- Treat elevated TGs if over 150 mg/dL. Intensify weight management and increase physical activity. If TGs are ≥200 mg/dL after LDL goal is reached, set a secondary goal for non-HDL Cholesterol (total cholesterol minus HDL) 30 mg/dL higher than LDL goal.
- Treat metabolic syndrome. Address underlying causes (overweight/obesity and physical inactivity).

## SAMPLE NUTRITION CARE PROCESS STEPS

### Dyslipidemia

**Assessment Data:** Food frequency recall and intake records; computer nutrient analysis.

**Nutrition Diagnosis (PES):** Inappropriate intake of food fats related to food and nutrition-related knowledge deficit as evidenced by daily consumption of bacon, sausage, butter and ice cream, saturated fat intake of 15% of kilocal, TC 220 mg/dL, and LDL Cholesterol of 165 mg/dL and HDL Cholesterol of 30 mg/dL.

**Intervention:** Food and Nutrient Delivery: Clarify current diet order to include no fried foods, bacon, sausage, and creams/custards. Provide Cheerios or oatmeal for breakfast to help further lower Cholesterol. Add plant sterols or stanols.

Education: identify alternate sources of fats from meals and snacks that are more desirable.

**Monitoring and Evaluation:** Repeat lab values after 3–6 months; dietary recall. Goal = TC below 200 mg/dL.



- Initiate and maintain weight loss if overweight. Obesity with a high waist circumference is especially important to correct in both men and women.
- Moderate carbohydrate restriction and weight control can improve dyslipidemia.
- Correct elevated levels of tHcy ( $>6 \mu\text{mol/L}$ ).
- Use more flavonoids, phytochemicals, soy products, and fruits and vegetables.
- Treat hypertension; use aspirin for CAD patients to reduce prothrombotic state if appropriate.
- Monitor for effects of MTHFR genotype (Ilhan et al, 2008). This may be useful in predicting the development of premature coronary artery disease, especially in hypertensive adolescents (Koo et al, 2008) and those who have type 1 diabetes (Wiltshire et al, 2008).



## FOOD AND NUTRITION

- There is no “one size fits all” guideline; combine diet with exercise and other lifestyle changes. The Mediterranean diet tends to be quite acceptable to most people and works well in lowering coronary risk factors; it encourages use of olive oil, red wine, fish, fruits, and vegetables.
  - The Evidence Analysis Library of the American Dietetic Association recommends the “Therapeutic Lifestyle” diet consisting of 25–35% total fat,  $<7\%$  saturated and trans fat, and  $<200 \text{ mg}$  dietary Chol (NHLBI, 2009). To keep fat at about 3 g/100 kcal, examples include:
- | 25% of kilocal    | 30% of kilocal    | 35% of kilocal    |
|-------------------|-------------------|-------------------|
| 28 g in 1000 kcal | 33 g in 1000 kcal | 39 g in 1000 kcal |
| 33 g in 1200 kcal | 40 g in 1200 kcal | 47 g in 1200 kcal |
| 42 g in 1500 kcal | 50 g in 1500 kcal | 59 g in 1500 kcal |
| 50 g in 1800 kcal | 60 g in 1800 kcal | 70 g in 1800 kcal |
| 56 g in 2000 kcal | 67 g in 2000 kcal | 78 g in 2000 kcal |
| 67 g in 2400 kcal | 80 g in 2400 kcal | 93 g in 2400 kcal |
- Use isocaloric replacement of saturated fatty acids (SFA) with MUFA and polyunsaturated fatty acids (PUFA); include olive oil and canola oil in cooking and salad dressings.
  - A diet rich in fruits, vegetables, low-fat dairy products, and low in sodium and saturated fat can decrease BP, an effect that is enhanced by weight loss and increased physical activity.
  - Consume  $1/2$  cup of nuts daily or 5 oz per week with a diet low in saturated fat and Chol to decrease TC. Nuts contain flavonoids, phenols, sterols, saponins, elegendic acid, folic acid, magnesium, copper, potassium, and fiber. Almonds are a very good source of vitamin E; walnuts contain ALA.
  - Consume antioxidants from dietary sources. Vitamin E foods include asparagus, spinach, wheat germ, nuts, salad oils, and creamy salad dressings. Vitamin C foods should be consumed in amounts that meet DRI levels. Supplemental antioxidants alone or in combination with other antioxidants may act as pro-oxidants and have no protection for CVD events (ADA Evidence Analysis Library, 2009).
  - Use flavonoids from tea, blueberries, yellow onions, red wine, grape juice, apples, cocoa, dark chocolate, products such as CocoaVia. Pomegranate increases antioxidant consumption and lowers LDL Chol (Arias and Ramon-Laca, 2005). Cinnamon, cloves, licorice, and sage are recommended as well.
  - Consume a diet high in total fiber (17–30 g/d) and soluble fiber (7–13 g/d) as part of a diet low in saturated fat and Chol. Soluble fiber may include oatmeal, high fiber cereal, prunes, oat bran, corn bran, apples, and legumes as good sources. Risk factors associated with BP, lipoprotein subclasses and particle sizes, insulin resistance, postprandial glucose, fatal and non-fatal MI, or stroke are decreased as dietary fiber intake increases (ADA Evidence Analysis Library, 2009).
  - Consume 2–3 g of plant sterols and stanols (through margarine, low-fat yogurt, orange juice, breads, and cereals) daily to lower total and LDL Chol, even with statins. Stanol-containing margarines may be consumed for at least 3 weeks before reassessment. Consume one extra carotenoid-rich fruit or vegetable per day to maintain plasma carotenoid levels when consuming sterol-enriched spreads.
  - Pistachios, sunflower kernels, sesame seeds, and wheat germ are high in natural phytoosterols; use often.
  - Intake of approximately 1 g/d of EPA and DHA from a supplement or fish may decrease the risk of death from cardiac events in patients with heart disease. Regular consumption of an average of two servings of fatty fish per week (about 3.5 oz per serving) reduces risk of death from cardiac events. However, a low-to-moderate fat diet is also needed (Shah et al, 2009).
  - Diets containing soy are well tolerated but soy will produce varied results based on initial Chol level and conditions such as diabetes.
  - Use fewer animal proteins and more legumes or vegetable protein sources. Fish and shellfish may be used three to four times weekly, especially sources rich in omega-3 fatty acids. Remove chicken skin before cooking or just before serving. Lean beef and chicken are considered to be comparable.
  - Trans fatty acids should be avoided; read labels.
  - Provide chromium, copper, vitamin K in recommended amounts.
  - A diet rich in folic acid, vitamins B<sub>6</sub> and B<sub>12</sub>, betaine and choline might benefit cardiovascular health through a tHcy-lowering effect (da Costa et al, 2005; Olthof and Verhoef, 2005). Betaine is widely distributed in animals and plants, especially seafood, wheat germ, bran, and spinach; intake from foods is estimated at 0.5–2 g/d (Olthof and Verhoef, 2005).
  - The DASH diet is useful; see Table 6-4. This diet will include 9–12 servings of fruits and vegetables, 2–3 servings of low-fat dairy products, less than 2.3 g sodium, weight loss if necessary, and increased physical activity with moderate intensity three times per week (ADA Evidence Analysis Library, 2009).

## Common Drugs Used and Potential Side Effects

- Reduced progression of atherosclerosis is associated with intensive statin treatment which reduces both atherogenic lipoproteins and CRP (Nissen et al, 2005). Try “Diet First, Then Drugs”: see Table 6-6.
- If anticoagulants (warfarin) are used, limit vitamin K-containing foods to 1 per day. Foods high in vitamin K include mayonnaise, canola and soybean oils, Brussels sprouts, collards, endive, spinach, watercress, red bibb lettuce, cabbage, broccoli, kale, and parsley.
- Aspirin may decrease serum ferritin by increasing occult blood loss if used over a long time.
- Digitalis and digoxin (Lanoxin) require the patient to avoid excessive amounts of vitamin D, natural licorice, fiber, and potassium. Take these drugs 30 minutes before meals. Do not take with herbal teas, Siberian ginseng, milkweed, hawthorn, guar gum, or St. John’s wort.
- Gemfibrozil (Lopid) is used for elevated TGs when there is a risk of pancreatitis; taste changes or abdominal pain may occur. Probucol (Lorelco) may cause vomiting or anorexia.

- Statins reduce Chol production by the liver. They may lower coenzyme Q10 to the point of deficiency. Simvastatin may cause constipation; fluvastatin may cause nausea and abdominal cramps; pravastatin can elevate AST and ALT levels or cause nausea, vomiting, and diarrhea. Interestingly, statins support bone health. Increase omega-3 fatty acids and reduce the omega-6 to omega-3 ratio to allow statins to work more effectively.
- Thiazides, propranolol, estrogens, and oral contraceptives may increase lipid levels or may lower folate levels. Monitor for specific side effects.
- Colesevelam HCl (WelChol) can be used with statins; it is not absorbed into the bloodstream and has few side effects.

## Herbs, Botanicals, and Supplements

- In one small clinical trial, red yeast rice and TLC decreased LDL Chol level without increasing CPK levels; this may be a treatment option for dyslipidemic patients who cannot tolerate statin therapy (Becker et al, 2009).

**TABLE 6-6** Drugs Affecting Lipoprotein Metabolism

Drug Class	Agents and Daily Doses	Lipid/Lipoprotein Effects	Side Effects and Comments	Contraindications
Statins—HMG-CoA reductase inhibitors	Lovastatin (20–80 mg)	LDL-C, ↓18–55%	Muscle pain and tenderness; myopathy	Absolute:
	Pravastatin (20–40 mg)	HDL-C, →5–15%		Active or chronic liver disease
	Simvastatin (20–80 mg)	TG, ↓7–30%	Severe cases: rhabdomyolysis and release of myoglobin into the bloodstream	Relative:
	Fluvastatin (20–80 mg)		Increased liver enzymes	Concomitant use of certain drugs <sup>a</sup>
	Atorvastatin (10–80 mg)			
Bile acid sequestrant	Cerivastatin (0.4–0.8 mg)	LDL-C, ↓15–30%	GI distress	Absolute:
	Cholestyramine (4–16 g)	HDL-C, →3–5%	Constipation; use more fiber	Dysbetalipoproteinemia
	Colestipol (5–20 g)	TG, No change or increase	Decreased absorption of other drugs	TG >400 mg/dL
	Colesevelam (2.6–3.8 g)		Add folate and fat-soluble vitamins; mix with liquids	Relative: TG >200 mg/dL
Nicotinic acid (Nico-Bid, Nico-400)	Immediate-release (crystalline) nicotinic acid (1.5–3 g), extended-release nicotinic acid (Niaspan) (1–2 g), sustained-release nicotinic acid (1–2 g)	LDL-C, ↓5–25% HDL-C, →15–35% TG, ↓20–50%	Flushing Hyperglycemia Hyperuricemia (or gout) Upper GI distress Hepatotoxicity or altered LFTs Vomiting, diarrhea	Absolute: Chronic liver disease Severe gout Relative: Diabetes Hyperuricemia Peptic ulcer disease
Fibric acids	Gemfibrozil (600 mg BID)	LDL-C, ↓5–20% (may be increased in patients with high TG)	Dyspepsia	Absolute:
	Fenofibrate (200 mg)		Gallstones	Severe renal disease
	Clofibrate (1000 mg BID)	HDL-C, →10–20% TG, ↓20–50%	Myopathy Weight gain Diarrhea, nausea	Severe hepatic disease

HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; LFT, liver function test; GI, gastrointestinal.

Adapted from National Heart, Lung, and Blood Institute. Web site accessed July 7, 2009, at <http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3full.pdf>.

<sup>a</sup>Cyclosporine, macrolide antibiotics, various antifungal agents, and cytochrome P-450 inhibitors (fibrates and niacin should be used with appropriate caution).

- Folic acid lowers plasma tHcy by 25% maximally, because 5-methyltetrahydrofolate is a methyl donor in the remethylation of tHcy to methionine (Olthof and Verhoef, 2005). In the NORVIT trial, researchers concluded that folic acid supplements could cause more harm than good if given with B<sub>12</sub> supplements (Bonaa et al, 2006). However, betaine given in high doses (6 g/d and higher) acutely reduces increased tHcy after methionine loading by up to 50%, whereas folic acid has no effect (Olthof and Verhoef, 2005). This fact may have played a role in the outcome of recent folic acid trials where Albert et al (2008) and Ebbing (2008) and their colleagues reported that long-term studies for lowering tHcy with folic acid, B<sub>12</sub> and B<sub>6</sub> supplements failed to prevent cardiac events.
- Herbs and botanicals such as angelica, hawthorn, canola, cinchona, valerian, willow, grape, pigweed, and chicory have proven efficacy. Discuss herbs and botanicals with the physician; see Table 6-3.
- Encourage reading of food labels, including how to identify various ingredients such as “free, low, reduced” Chol.
- Aerobic exercise, weight loss, smoking cessation, and lifestyle changes are needed. Provide ideas, coping skills, motivational factors, and environmental barriers. Cardiac Rehabilitation programs that use a mind–body approach are useful.
- For individuals have MTHFR genetic allele, it is prudent to discuss dietary or supplemental measures (Klerk et al, 2002; Koo et al, 2008; Wiltshire et al, 2008). Sources of vitamin B<sub>6</sub> include eggs, meats, fish, vegetables, yeast, whole-wheat grains, and milk. Sources of vitamin B<sub>12</sub> include liver, meat, eggs, dairy products, and fish. See Table 6-4 for good sources of folate.
- Discuss low-fat cooking methods, such as baking, broiling, flame cooking, grilling, marinating, poaching, roasting, smoking, or steaming.
- Olestra, a fat substitute, decreases absorption of dietary fat. Use in moderate amounts to prevent diarrhea.
- Routine periodic fasting has been shown to have merit in reducing risk of CAD (Horne et al, 2008).
- Intensive lifestyle changes maintained for 5 years or longer may be needed. A strict low fat diet can help reverse atherosclerosis. Check serum lipids at least annually.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the roles of heredity, exercise, and lifestyle habits. BP, Chol, obesity, and diabetes are affected by dietary patterns; some control is possible.
- Adherence with the 2005 Dietary Guidelines for Americans (DGA) is significantly associated with slower atherosclerosis progression and less metabolic syndrome (Fogli-Cawley et al, 2007; Imamura et al, 2009).
- There is no Chol in foods of plant origin. Encourage use of a plant-based diet.
- Explain which foods are sources of saturated fats and trans fatty acids. Identify foods that are sources of polyunsaturated fats and monounsaturated fats (olive and peanut oils). An easy first step is changing to skim milk products instead of whole milk.
- Diets low in fat have different tastes and textures. If one changes diet too quickly, the diet may seem dry and unpalatable. Suggest changing gradually. Teach new ideas for moistening foods without adding excess fat (e.g., using applesauce instead of oil in some baked goods). Provide lists of resources such as cookbooks, newsletters, product samples, or coupons.
- Describe food sources of saturated MUFAs and PUFAs and Chol; discuss olive, soybean, walnut, and peanut oil uses. Help the patient make suitable substitutions. Although egg yolks contain Chol, they can be planned into the diet three to four times weekly.
- Use of red wine appears to be protective, but more studies are needed to verify effects on hemodynamics (Karatzis et al, 2009).
- Fish should be included several times weekly. Omega-3 fatty acids are found in fatter fish such as salmon, herring, tuna, mackerel, and other seafood.
- Sources of soluble fiber (guar gum, pectin) include apples, legumes, oat and corn brans. Include other whole-grain foods for insoluble fiber as both types of fiber are beneficial.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- American Heart Association—Atherosclerosis  
<http://www.americanheart.org/presenter.jhtml?identifier=4440>
- Dyslipidemia  
<http://www.merck.com/mmpe/sec12/ch159/ch159b.html>
- NCEP Guidelines  
<http://www.nhlbi.nih.gov/about/ncep/index.htm>
- NHLBI—ATP Guidelines, 2004 update  
<http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3upd04.htm>
- Your LDL and You  
<http://nhlbisupport.com/chd1/treatment.htm>

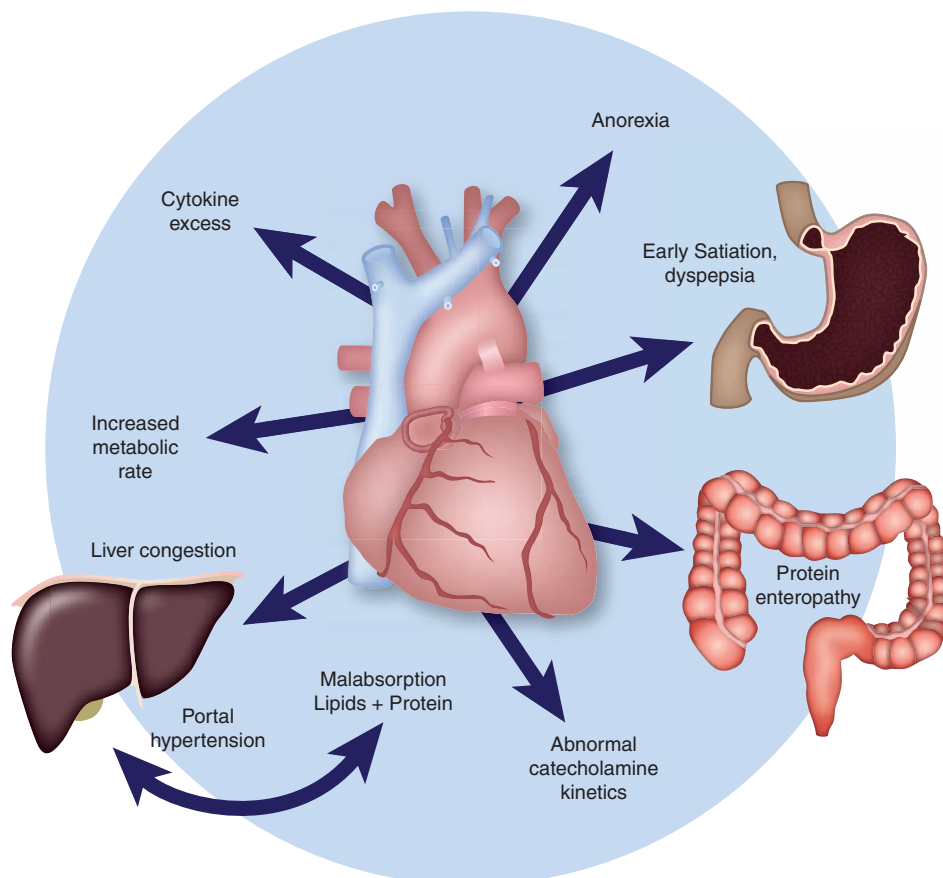
### ATHEROSCLEROSIS, CORONARY ARTERY DISEASE, DYSLIPIDEMIA—CITED REFERENCES

- ADA Evidence Analysis Library. American Dietetic Association. Chicago, IL. Accessed November 7, 2009, at <http://www.adaevidencelibrary.com/topic.cfm?cat=3015>.
- Albert CM, et al. Effect of folic acid and B vitamins on risk of cardiovascular events and total mortality among women at high risk for cardiovascular disease: a randomized trial. *JAMA*. 299:2027, 2008.
- Arias BL, Ramon-Laca L. Pharmacological properties of citrus and their ancient and medieval uses in the Mediterranean region. *J Ethnopharmacol*. 97:89, 2005.
- Becker DJ, et al. Red yeast rice for dyslipidemia in statin-intolerant patients. *Ann Intern Med*. 150:830, 2009.
- Bonaa KH, et al. Homocysteine lowering and cardiovascular events after acute myocardial infarction. *New Engl J Med*. 354:1578, 2006.
- Chen YC, et al. The HMG-CoA reductase gene and lipid and lipoprotein levels: the Multi-Ethnic Study of Atherosclerosis [published online ahead of print Jun 25, 2009]. *Lipids*. 44:733, 2009.
- da Costa KA, et al. Choline deficiency in mice and humans is associated with increased plasma homocysteine concentration after a methionine load. *Am J Clin Nutr*. 81:440, 2005.

- Das UN. Essential fatty acids—a review. *Curr Pharm Biotechnol*. 7:467, 2006.
- Fogli-Cawley JJ, et al. The 2005 Dietary guidelines for Americans and risk of the metabolic syndrome. *Am J Clin Nutr*. 86:1193, 2007.
- Freitas AI, et al. Methylenetetrahydrofolate reductase gene, homocysteine and coronary artery disease: the A1298 C polymorphism does matter. *Thromb Res*. 122:648, 2008.
- Horne BD, et al. Usefulness of routine periodic fasting to lower risk of coronary artery disease in patients undergoing coronary angiography. *Am J Cardiol*. 102:814, 2008.
- Ilhan N, et al. The 677 C/T MTHFR polymorphism is associated with essential hypertension, coronary artery disease, and higher homocysteine levels. *Arch Med Res*. 39:125, 2008.
- Imamura F, et al. Adherence to 2005 Dietary guidelines for Americans is associated with a reduced progression of coronary artery atherosclerosis in women with established coronary artery disease. *Am J Clin Nutr*. 90:193, 2009.
- Kathiresan S, et al. Common variants at 30 loci contribute to polygenic dyslipidemia. *Nat Genet*. 41:56, 2009.
- Karatzi K, et al. Red wine, arterial stiffness and central hemodynamics. *Curr Pharm Des*. 15:321, 2009.
- Klerk M, et al. MTHFR 677 C→T polymorphism and risk of coronary heart disease: a meta-analysis. *JAMA*. 288:2023, 2002.
- Koo HS, et al. Methylenetetrahydrofolate reductase tt genotype as a predictor of cardiovascular risk in hypertensive adolescents. *Pediatr Cardiol*. 29:136, 2008.
- Locklin Holmes A, et al. Dietitian services are associated with improved patient outcomes and the MEDFICTS dietary assessment questionnaire is a suitable outcome measure in cardiac rehabilitation. *J Am Diet Assoc*. 105:1533, 2005.
- Moller DE, Kaufman KD. Metabolic syndrome: a clinical and molecular perspective. *Annu Rev Med*. 56:45, 2005.
- Ntaios G et al. Iatrogenic hyperhomocysteinemia in patients with metabolic syndrome: A systematic review and metaanalysis. [published online ahead of print August 19, 2010]. *Atherosclerosis*.
- Nissen SE, et al. Statin therapy, LDL cholesterol, C-reactive protein, and coronary artery disease. *N Engl J Med*. 352:29, 2005.
- Olthof MR, Verhoef P. Effects of betaine intake on plasma homocysteine concentrations and consequences for health. *Curr Drug Metab*. 6:15, 2005.
- Shah KB, et al. The Cardioprotective effects of fish oil during pressure overload are blocked by high fat intake. role of cardiac phospholipid remodeling [published online ahead of print Jul 13, 2009]. *Hypertension*. 54:605, 2009.
- Wiltshire EJ, et al. Methylenetetrahydrofolate reductase and methionine synthase reductase gene polymorphisms and protection from microvascular complications in adolescents with type 1 diabetes. *Pediatr Diabetes*. 9:348, 2008.

## CARDIAC CACHEXIA

### NUTRITIONAL ACUITY RANKING: LEVEL 4







## DEFINITIONS AND BACKGROUND

Cardiac cachexia is concurrent with HF of such severity that patients cannot eat adequately to maintain weight. It involves a loss of more than 10% of lean body mass and can clinically be defined as a body weight loss of 7.5% of previous dry body weight over 6 months or longer. The condition usually follows HF (moderate to severe), with some valvular heart disease. Nutritional insults generally affect the heart muscle severely, and the insult may be significant. While the pathophysiological alterations leading to cardiac cachexia are unclear, metabolic, neurohormonal and immune abnormalities may play a role. Cachectic HF patients show raised plasma levels of epinephrine, norepinephrine, cortisol, renin, and aldosterone. Patients with cardiac cachexia suffer from a general loss of fat tissue, lean tissue, and bone tissue. Lower, rather than higher, Chol levels are associated with poor clinical outcome in patients with chronic HF.

Chronic HF (CHF) is increasingly recognized as a multi-system disease with alterations in intestinal morphology, permeability, and absorption that lead to chronic inflammation (Sandek et al, 2009). The wasting associated with chronic HF is an independent predictor of mortality. A loss of body weight or skeletal muscle mass is common in older persons and precedes a poor outcome; starvation results in a loss of body fat and nonfat mass due to inadequate intake of protein and energy and sarcopenia is associated with a reduction in muscle mass and strength (Thomas, 2007). Cardiac cachexia as a terminal stage of chronic HF carries a poor prognosis (von Haehling et al, 2009).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Ghrelin (the growth-hormone-releasing peptide in the stomach) may play a role in cachexia; more studies are needed. Tumor necrosis factor may be involved (Anker et al, 2004). In addition, adiponectin seems to have a role in the wasting process.

#### Clinical/History

Height	Steatorrhea or diarrhea	Gluc
Weight, current	Loss of muscle mass	Fecal fat (in steatorrhea)
Dry weight	Supraclavicular and temporal muscle wasting	H & H
BMI		Serum Fe, ferritin
Waist circumference; ascites?		Liver function tests
Anorexia, weight loss?	<b>Lab Work</b>	Thyroid-stimulating hormone (TSH)
Edema	Chol—total (low?)	Total lymphocyte count (TLC)
BP	Trig	Serum insulin
Fatigue	CRP (elevated?)	Alb, transthyretin
Shortness of breath (dyspnea)	CoQ10 serum levels	Serum thiamin

## SAMPLE NUTRITION CARE PROCESS STEPS

### Unintentional Weight Loss

**Assessment Data:** BMI <18, HF and shortness of breath, weight loss of 20 lb in 12 months.

**Nutrition Diagnoses (PES):** Unintentional weight loss related to low oral intake as evidenced by weight loss of 20 lb in past year and BMI 17.5.

**Interventions:** Education about ways to enhance energy and nutrient density in meals and snacks.

**Monitoring and Evaluation:** BMI now >19 in 3 months; better intake of energy-dense foods and beverages.

Transferrin  
Na<sup>+</sup>, K<sup>+</sup>  
Ca<sup>++</sup>, Mg<sup>++</sup>  
tHcy

Serum folate and vitamin B<sub>12</sub>  
Blood urea nitrogen (BUN)

## INTERVENTION



### OBJECTIVES

- Improve hypoxic state and heart functioning.
- Correct malnutrition, wasting, malabsorption, and steatorrhea. Patients with HF have breathing difficulties, fatigue, nausea, loss of appetite, early feeling of fullness, or ascites that tend to decrease intake.
- Optimize heart function through balance of medications, antioxidants, fluids, and electrolytes.
- Reduce impact of inflammatory cytokines. Meet hypermetabolic state with adequate energy intake.
- Prevent infection or sepsis, especially if tracheostomy is required.
- Provide gradual repletion to prevent overloading in a severely depleted patient.
- Treat constipation or diarrhea as necessary.



## FOOD AND NUTRITION

- Energy needs may be calculated as high as 50% above basic needs.
- Protein should be estimated at a rate of 1.0–1.5 g/kg, increasing or decreasing depending on renal or hepatic status.
- Offer tube feeding (TF) or parenteral nutrition if appropriate. Sometimes, TFs are not well tolerated because of access to the thoracic cavity and reduced blood flow to the gastrointestinal (GI) tract. High-calorie, low-volume products have a high density of calories; they are appropriate for persons with a fluid limitation but must be monitored with renal or hepatic insufficiency.
- Provide small, frequent meals to prevent overloading with high glucose or fat. A diet high in saturated fat may actually be beneficial (Berthiaume et al, 2010).
- Provide as many preferred foods as feasible to improve appetite and intake.

- Antioxidants may benefit. Nutrient-dense foods containing omega-3 fatty acids can be safely recommended. Use more foods such as fish, fruits, cinnamon, cocoa, green tea, berries, nuts and foods that contain flavonols. Use the DASH diet as much as possible (Levitan et al, 2009).
- Sodium may need to be restricted to 1–2 g daily. In older patients, a more lenient 4–6 g may suffice. Modify potassium intake as appropriate. The DASH diet is useful.
- Diet may need to be high in folate, magnesium, zinc, iron (depending on serum levels), vitamins E, B<sub>6</sub>, and B<sub>12</sub>. Thiamin should be included to alleviate cardiac beri-beri, which is common.

### Common Drugs Used and Potential Side Effects

- Therapeutic approaches include appetite stimulants such as megestrol acetate, medroxyprogesterone acetate, and cannabinoids (von Haehling et al, 2009).

### Herbs, Botanicals, and Supplements

- Discuss herbs and botanicals with the physician. See Table 6-3.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Balance medications, fluid, and electrolytes carefully.
- Supplements may be beneficial between meals to improve total calorie intake (e.g., sherbet shakes).

- The importance of diet in cardiovascular health should be addressed, but rapid weight loss should be prevented. Patients with low BMI are at higher risk after cardiac surgery than obese patients.
- Exercise, with supervised guidance, can be beneficial to rebuild lean body mass.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

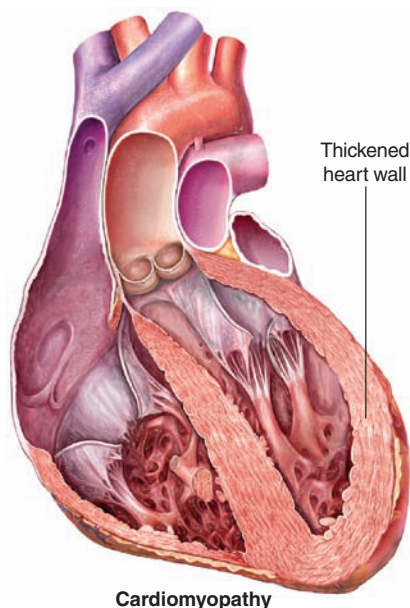
- Cardiac Cachexia  
<http://www.heartfailure.org/>
- Heart Hope  
<http://www.hearthope.com/>
- Merck Manual—Heart Failure  
<http://www.merck.com/mmpe/print/sec07/ch074/ch074a.html>

### CARDIAC CACHEXIA—CITED REFERENCES

- Anker SD, Berthiaume JM, et al. The myocardial contractile response to physiological stress improves with high saturated fat feeding in heart failure. *Am J Physiol Heart Circ Physiol*. 299:410, 2010.
- Hunt SA, et al. 2009 Focused update incorporated into the ACC/AHA 2005 Guidelines for the diagnosis and management of heart failure in adults. *J Am Coll Cardiol*. 53:e1, 2009.
- Levitan EB, et al. Relation of consistency with the dietary approaches to stop hypertension diet and incidence of heart failure in men aged 45 to 79 years. *Am J Cardiol*. 104:1416, 2009.
- Sandek A, et al. Nutrition in heart failure: an update. *Curr Opin Clin Nutr Metab Care*. 12:384, 2009.
- Thomas DR. Loss of skeletal muscle mass in aging: Examining the relationship of starvation, sarcopenia and cachexia. *Clin Nutr*. 26:389, 2007.
- von Haehling S, et al. Cardiac cachexia: a systematic overview. *Pharmacol Ther*. 121:227, 2009.

## CARDIOMYOPATHIES

### NUTRITIONAL ACUITY RANKING: LEVEL 3



Asset provided by Anatomical Chart Co.



### DEFINITIONS AND BACKGROUND

Cardiomyopathies may be caused by many known diseases or have no specific known cause. They are progressive disorders that impair the structure or function of the muscular wall of the lower chambers of the heart. Echocardiography is useful in demonstrating cardiac abnormalities.

In **restrictive cardiomyopathy**, the heart chambers are unable to properly fill with blood because of stiffness in the heart. Amyloidosis or scleroderma are common causes.

**Dilated congestive cardiomyopathy (DCC)** is most commonly caused by CHD in the United States. DCC may also occur from a viral infection such as coxsackievirus B, from diabetes or thyroid disease, or from excessive alcohol, cocaine, or antidepressant use. Rarely, pregnancy or rheumatoid arthritis can also trigger DCC. Because prenatal dilated cardiomyopathy (DCM) can also be the presenting sign of cblC (cobalamin C deficiency) inborn errors of metabolism should be considered in view of the possible impact on treatment and future reproductive options (De Bie et al, 2009).

The first symptoms of DCC are shortness of breath on exertion and easy fatigability; sometimes, a fever and flu-like

symptoms occur if triggered by a virus. Calcium and potassium irregularities have been noted (Olson et al, 2005). Remaining heart muscle stretches to compensate for lost pumping action, and when the stretching no longer compensates, DCC occurs. Blood may pool in the swollen heart, and clots may form on the chamber walls. Seventy percent of patients with DCC die within 5 years of the beginning of their symptoms, and the prognosis worsens as the walls become thinner and the heart valves begin to leak. Because of this, DCC is the most common cause for heart transplantation.

**HCM** may occur as a birth defect or as a result of acromegaly (excessive growth hormone in the blood), a pheochromocytoma, or a neurofibromatosis. Glycogen storage disease may also present with some cardiomyopathy (Arad et al, 2005). Thickening of the heart wall causes high BP, pulmonary hypertension, and chronic shortness of breath. Faintness, chest pain, irregular heartbeats and palpitations, and HF with dyspnea will occur. Hyperhomocysteinemia chelates copper and impairs copper-dependent enzymes; therefore, copper supplementation may be needed (Hughes et al, 2008).

HCM is a possible cause of sudden death; it is largely confined to young people but can occur suddenly at any stage of life. Most patients with mild hypertrophy are at low risk. Treatment options for patients with obstructive HCM include medical therapy, pacemaker insertion, percutaneous transluminal septal myocardial ablation, mitral valve replacement, and surgical resection of the obstructing muscle. Nonsurgical septal reduction therapy is also an effective therapy for symptomatic patients with obstructive HCM. A heart transplant may be indicated.



## ASSESSMENT, MONITORING, AND EVALUATION



## CLINICAL INDICATORS

**Genetic Markers:** About 90% of cases of HCM are familial with mutations in the MYBPC3 gene, encoding cardiac myosin-binding protein C (van Dijk et al, 2009). Genetic factors also may be responsible for half of DCM cases.

### Clinical/History

Height  
Weight, current  
Dry weight  
BMI  
Waist circumference  
Diet history  
Heart murmur  
Atrial fibrillation?  
BP (normal or low)  
Echocardiography

### ECG

Cardiac catheterization  
Temperature  
Dyspnea  
Fatigue and poor exercise tolerance  
Ascites or edema

### Lab Work

Chol—total, HDL, LDL  
Trig

### CRP

CoQ10 serum levels  
Prothrombin time (PT)  
INR  
Na<sup>+</sup>, K<sup>+</sup>  
Ca<sup>++</sup>, Mg<sup>++</sup>  
Gluc  
H & H  
Serum Fe, ferritin  
Serum copper  
Serum insulin

Alb,  
transferrin  
Transferrin  
BUN, Creat

Low serum  
vitamin D  
or rickets?  
Urinary methylmalonic acid

Homocystinuria?  
Serum tHcy  
Serum folate  
and B<sub>12</sub>

## INTERVENTION



## OBJECTIVES

- Improve hypoxic state and heart functioning.
- Correct malnutrition, malabsorption, and steatorrhea.
- Growth failure is a significant clinical problem of children with cardiomyopathy; nearly one third of these children manifest some degree of growth failure (Miller et al, 2007).
- Optimize heart function through balance of medications, fluids, and electrolytes.
- Meet hypermetabolic state with adequate calories.
- Provide gradual repletion to prevent overloading in a severely depleted patient.
- Treat constipation or diarrhea as necessary.
- Prepare for surgery, if planned.



## FOOD AND NUTRITION

- Energy may be calculated as much as 50% above usual needs. Optimal intake of macronutrients and antioxidants that can protect against free radical damage are needed (Miller et al, 2007).
- Protein should be calculated at a rate of 1.0–1.5 g/kg, increasing or decreasing depending on renal or hepatic status.
- Provide small, frequent meals to prevent overloading with high glucose or with rapid fat infusion. Provide as

## SAMPLE NUTRITION CARE PROCESS STEPS

### Excessive Intake of Fluids

**Assessment Data:** Dietary intake records; I & O records. Presence of 1+ pitting edema. Signs of cardiac failure with lung crackles.

**Nutrition Diagnosis (PES):** Excessive intake of fluids related to food choices and preference for primarily soups and liquids on most days of the week as evidenced by edema, low BP and easy fatigue.

**Intervention:** Education and counseling about fluid limits using a food diary, fluid calculation chart, I & O records.

**Monitoring and Evaluation:** Weight maintenance versus fluctuations; improved intake records and food diaries reflecting successful management of fluid intake. Fewer symptoms of HF and improved breathing.

many preferred foods as feasible to improve appetite and intake.

- Follow TLC diet interventions. Pistachios, sunflower kernels, sesame seeds, and wheat germ are high in phytosterols; use often.
- The DASH diet is useful. Diet may need to be high in calcium and potassium (Olson et al, 2005); folate, magnesium, copper, zinc, and iron may also be needed, depending on serum levels. Increasing vitamins E, B<sub>6</sub>, and B<sub>12</sub> may be beneficial. Thiamin should also be included because of likelihood of cardiac beriberi.
- Sodium may need to be restricted to 2–4 g daily; modify potassium intake as appropriate for serum levels.
- Offer TF or parenteral nutrition if appropriate. Sometimes, TFs are not well tolerated because of proximity to the thoracic cavity and because of reduced blood flow to the GI tract. High-calorie, low-volume products are useful for their high density of calories. They are appropriate for persons with a fluid limitation but must be monitored in patients with renal or hepatic difficulty.

### Common Drugs Used and Potential Side Effects

- Anticoagulant therapy is needed to prevent clots from causing heart attacks, strokes, and other problems. With warfarin (Coumadin), use a controlled amount of vitamin K; check TF products and supplements. Limit foods high in vitamin K to 1 serving per day. Foods high in vitamin K include mayonnaise, canola and soybean oils, Brussels sprouts, collards, endive, spinach, watercress, red bibb lettuce, cabbage, broccoli, kale, and parsley.
- Blood-thinning medications: Omega-3 fatty acids may increase the blood-thinning effects of aspirin or warfarin. While the combination of aspirin and omega-3 fatty acids may actually be helpful under certain circumstances (such as heart disease), they should only be taken together under the guidance and supervision of a health care provider. Be wary of using supplements containing vitamins A and C with these drugs; side effects may be detrimental. Vitamin E should not be taken with warfarin because of the possibility of increased bleeding; avoid doses greater than 400 IU/d. Avoid taking with dong quai, fenugreek, feverfew, excessive garlic, ginger, ginkgo, and ginseng because of their effects.
- Beta-blockers and calcium channel blockers may be used to reduce the force of heart contractions.
- Diuretics: Side effects may include potassium depletion; review types used and alter diet accordingly. Some diuretics spare calcium and protect bone health.
- Digoxin: Monitor potassium intake or depletion carefully, especially when combining with diuretics. Avoid excessive intakes of fiber and wheat bran. Avoid use with hawthorn, milkweed, guar gum, and St. John's wort.
- Insulin may be needed if patient has diabetes or becomes hyperglycemic. Alter mealtimes accordingly.

### Herbs, Botanicals, and Supplements

- Table grape powder contains important phytochemicals; a diet with grape powder may reduce cardiac oxidative damage, increase cardiac glutathione, lower BP, improve cardiac function, reduce systemic inflammation, reduce cardiac hypertrophy and fibrosis (Seymour et al, 2008).
- Discuss herbs and botanicals with the physician. See Table 6-3. CoQ10 may be protective.
- Ephedra and adderol have been associated with cardiomyopathy and should be avoided.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Adequate rest is essential. Avoidance of stress is also important.
- Balance medications, fluid, and electrolytes carefully.
- Supplements may be beneficial between meals to improve total calorie intake (e.g., sherbet shakes).
- The importance of diet in cardiovascular health should be addressed. A moderate calorie restriction may be beneficial.
- Because of the high risk for sudden death in HCM patients, they should be advised against participation in highly competitive sports.
- For inherited forms of HCM, genetic counseling may be beneficial if planning a family.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- Cardiomyopathy  
<http://www.americanheart.org/presenter.jhtml?identifier=4468>
- Cardiomyopathy in the Young  
<http://www.cardiomyopathy.org/Cardiomyopathy-in-the-Young.html>
- Hypertrophic cardiomyopathy  
<http://www.cardiomyopathy.org/>

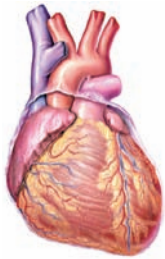
### CARDIOMYOPATHIES—CITED REFERENCES

- Arad M, et al. Glycogen storage diseases presenting as hypertrophic cardiomyopathy. *N Engl J Med*. 352:362, 2005.
- De Bie I, et al. Fetal dilated cardiomyopathy: an unsuspected presentation of methylmalonic aciduria and hyperhomocystinuria, cblC type. *Prenat Diagn*. 29:266, 2009.
- Hughes WM, et al. Role of copper and homocysteine in pressure overload heart failure. *Cardiovasc Toxicol*. 8:137, 2008.
- Miller TL, et al. Nutrition in Pediatric Cardiomyopathy. *Prog Pediatr Cardiol*. 24:59, 2007.
- Olson TM, et al. Sodium channel mutations and susceptibility to heart failure and atrial fibrillation. *JAMA*. 293:447, 2005.
- Seymour EM, et al. Chronic intake of a phytochemical-enriched diet reduces cardiac fibrosis and diastolic dysfunction caused by prolonged salt-sensitive hypertension. *J Gerontol A Biol Sci Med Sci*. 63:1034, 2008.
- van Dijk SJ, et al. Cardiac myosin-binding protein C mutations and hypertrophic cardiomyopathy: haploinsufficiency, deranged phosphorylation, and cardiomyocyte dysfunction. *Circulation*. 119:1473, 2009.



# HEART FAILURE

## NUTRITIONAL ACUITY RANKING: LEVEL 3



### • Heart Failure

Carbohydrates maintain sodium and fluid balance. A carbohydrate deficiency promotes loss of sodium and water, which can adversely affect blood pressure and cardiac function if not corrected.



### DEFINITIONS AND BACKGROUND

HF is the leading cause of CVD and related death, with nearly 5 million cases in the United States. HF results in reduced heart pumping efficiency in the lower two chambers, with less blood circulating to body tissues, congestion in lungs or body circulation, ankle swelling, abdominal pain, ascites, hepatic congestion, jugular vein distention, and breathing difficulty. The ability of mitochondria to oxidatively synthesize ATP from ADP and inorganic phosphate is compromised in the myocardium of HF patients.

Left ventricular failure will cause shortness of breath and fatigue; right ventricular failure causes peripheral and abdominal fluid accumulation. HF is a common diagnosis in hospitalized patients and four stages have been identified (Hunt et al, 2005):

- Stage A has mild symptoms and no limitation on physical activity.
- Stage B shows structural heart disease but no signs or symptoms of HF.
- Stage C demonstrates signs and symptoms of structural HF.
- Stage D shows refractory HF requiring specialized interventions.

HF can be caused by CHD, previous heart attack, history of cardiomyopathy, lung disease such as chronic obstructive pulmonary disease (COPD), severe anemia, excessive alcohol consumption, or low thyroid function. Male sex, lower education, physical inactivity, cigarette smoking, overweight, diabetes, hypertension, valvular heart disease, and CHD are all independent risk factors. The leading cause of HF in Western countries is ischemic heart disease. Therefore, aggressive therapy to halt progression of coronary atherosclerosis can have a major impact on controlling or curing HF.

B-type natriuretic peptide (BNP) is secreted by ventricles when pressure goes up in the heart. CoQ10 has been identified as a factor to consider. Patients with HF have low plasma levels of CoQ10, an essential cofactor for mitochondrial electron transport and myocardial energy supply (Molyneux et al, 2008). Low plasma TC concentrations have also been associated with higher mortality in HF; the relationship between CoQ10 and LDL-C levels may contribute to this association (Molyneux et al, 2008).

Decreased renal flow is common; BUN may be increased. Early adaptations to mild HF show susceptibility to sodium excess. Evidence suggests that advanced HF is a multifactor-

ial metabolic syndrome that can lead to cardiac cachexia, which then carries a very poor prognosis. The mechanisms underlying this association are poorly understood. Inflammatory cytokines may play a pathogenic role. HF is associated with elevated levels of angiotensin II in the blood, causing vessel contraction and high BP in addition to muscle wasting. Studies are reviewing the role of other factors.

Dairy nutrients (calcium, potassium and magnesium) have a BP lowering effect, as shown by studies showing the effectiveness of the DASH diet (Levitan et al, 2009). Low calcium increases intracellular calcium concentrations, thereby increasing 1,25-dihydroxyvitamin D(3) and parathyroid hormone (PTH), causing calcium influx into vascular smooth muscle cells and greater vascular resistance (Kris-Etherton et al, 2009). Dairy peptides may act as ACE inhibitors, thereby inhibiting the renin-angiotensin system with consequent vasodilation (Kris-Etherton et al, 2009). While sodium restriction and diuretics are basic treatments, treatment may also include implantation of a pacemaker or even cardiac transplantation. Joint efforts of cardiologists, endocrinologists, immunologists, and registered dietitians are required to develop effective therapeutic strategies. Referral to a registered dietitian is needed for MNT. For a person with HF, one planned initial visit and at least one to three planned follow-up visits can lead to improved dietary pattern and quality of life, decreased edema and fatigue, more optimal pharmacological management, and fewer hospitalizations (American Dietetic Association, 2009).



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Nuclear factor kappa B (NF-kappaB) is chronically activated in cardiac myocytes; the NFKB1 -94 insertion/deletion ATTG polymorphism is associated with DCM (Zhou et al, 2009).

Clinical/History	Skin, cyanotic or pale	Pulmonary
Height	Abnormal	edema, rales,
Weight	breath	dyspnea (left-sided HF)
BMI	sounds	Glomerular
Waist-hip ratio	Increased heart	filtration
Weight changes?	rate; pulse	rate (GFR)
Diet history	>80	Oliguria
BP	beats/min	Confusion,
Shortness of breath	Temperature	impaired
Dry, hacking cough or wheezing	Pitting edema, fatigue (right-sided HF)	thinking
		Chest x-ray
		Echocardiography

ECG	Partial pressure of oxygen (pO <sub>2</sub> )	Ca <sup>++</sup> , Mg <sup>++</sup>
Cardiac catheterization	Specific gravity (increased)	Gluc
<b>Lab Work</b>	Chol—total, HDL, LDL	Serum zinc
CRP	Trig	Alk phos
Serum CoQ10	H & H	Alb, transthyretin
BNP or NT-pro-BNP levels	Serum Fe, ferritin	BUN, Creat
Uric acid	tHcy	PT or INR
Oximetry	Serum folate	LDH
Partial pressure of carbon dioxide (pCO <sub>2</sub> )	Serum B <sub>12</sub>	(increased)
	Na <sup>+</sup> , K <sup>+</sup>	Nitrogen (N) balance
		AST, ALT

## INTERVENTION



### OBJECTIVES

- Lessen demands on the heart and restore hemodynamic stability.
- Prevent cardiogenic shock, thromboembolism, and renal failure.
- Maintain BP <140/90 mm Hg in all patients or <130/80 mm Hg in those with diabetes or chronic kidney disease (Khan et al, 2009).
- Eliminate or reduce edema.
- Avoid distention and elevation of diaphragm, which reduces vital capacity. Avoid overfeeding in cachexic patients to prevent refeeding syndrome.
- Attain desirable BMI and WHR to decrease oxygen requirements and tissue nutrient demands. Replenish lean body mass (LBM) when needed.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Herb and Drug–Nutrient Interactions

**Assessment Data:** Food frequency recall and intake records; medical prescription for warfarin. Taking large doses of vitamin K supplement randomly, and St John's wort (300 mg BID) for "the blues."

**Nutrition Diagnosis (PES):** Excessive bioactive substance intake (N1–4.2) related to knowledge deficit as evidenced by subtherapeutic INR of 1.2.

**Intervention:** Education about supplement-drug interaction and effects of vitamin K while using warfarin. Counseling to discontinue use of St. John's wort and consider psychological counseling. Coordinate care and referral for counseling.

**Monitoring and Evaluation:** Repeat INR weekly until therapeutic dose is achieved. Assess compliance with discontinuation of St John's wort and random use of vitamin K supplements. Assess understanding of risks of use of herbs or supplements while taking warfarin.

- Prevent or correct cardiac cachexia, low BP, listlessness, weak pulse from potassium-depleting diuretics, anorexia, nausea and vomiting, and sepsis.
- Correct nutrient deficits. Assure adequate intake of dairy products, as tolerated (Kris-Etherton et al, 2009).
- Because anemia is associated with an increased risk of mortality in both systolic and diastolic HF, strategies to increase hemoglobin levels should be used when needed (Groenveld et al, 2008).
- Encourage use of omega-3 fatty acids with a low-to-moderate fat intake (Shah et al, 2009).
- Prevent pressure ulcers from reduced activity levels and poor circulation.
- Promote use of the DASH diet and whole grains (Levitan et al, 2009; Nettleton et al, 2009). In addition, promote weight reduction, physical activity, restriction of dietary sodium, and excessive alcohol intake as methods for lowering BP.



## FOOD AND NUTRITION

Evidence-based recommendations for Registered Dietitians (RDs) to follow in providing nutrition treatment for people with HF include (American Dietetic Association, 2009):

- Appropriate daily intake of protein for clinically stable patients; HF patients have significantly higher protein needs than those without HF.
- Fluid intake between 48 and 64 oz/d, depending on fatigue or shortness of breath.
- Sodium intake less than 2 g/d to improve clinical symptoms and quality of life.
- Consume folate through food and/or a combination of B<sub>6</sub>, B<sub>12</sub>, and folate supplementation.
- Promote the DASH diet, with adequate potassium, calcium, and magnesium (Gums, 2004). Table 6-7 lists the sodium content of common foods. Table 6-8 provides some alternative tips for lowering sodium in the diet. For TF, use a low-sodium product and increase volume gradually.
- With total parenteral nutrition, ensure adequate intake of all micronutrients as well as macronutrients.
- Provide antioxidants, such as vitamin E, at DRI levels; there is no evidence that more is better. It is safe to consume more pomegranate, blueberry, and grape products.
- If patient is obese, a calorie-controlled diet can be recommended. A vegan pattern may be helpful with five to six small meals daily.
- Limit caffeine only if needed. The evidence is not definitive in this area (Ahmed et al, 2009).
- Beans, cabbage, onions, cauliflower, and Brussels sprouts may cause heartburn or flatulence; avoid if needed.
- Whole grains cut the risk for HF while eggs and high-fat dairy products contribute to it, according to the ARIC study (Nettleton et al, 2009). Add soluble fiber to the diet from apples or oat bran.
- Pistachios, sunflower kernels, sesame seeds, and wheat germ are high in phytosterols; use often.
- Thiamin levels tend to be low. Cardiovascular problems may be associated with beri-beri.
- Limit alcohol intake; for women, 1 drink per day and for men, 2 drinks or less a day.

**TABLE 6-7 Sodium Content of Typical Food Items**

Food Item	Milligrams (mg)	Food Item	Milligrams (mg)
<b>Meat, poultry, and fish</b>		<b>Dairy products</b>	
Sirloin steak (3 oz)	53	Butter, salted (1 tbsp)	116
Baked salmon (3 oz)	55	Milk (1 cup)	122
Chicken breast (3 oz)	64	Sour cream (1 cup)	123
Ground beef patty (4 oz)	87	Margarine (1 tbsp)	134
Chicken leg, fried (2.5 oz)	194	Chocolate pudding (1 cup)	180
Tuna, canned (3 oz)	468	Baked custard (1 cup)	209
Hot dog (1)	504	Buttermilk (1 cup)	257
Salami (2 slices)	607	Parmesan cheese (1/4 cup)	465
Fast food hamburger (4 oz)	763	Cheddar cheese (1 cup)	701
Corned beef (3 oz)	802	Cottage cheese, creamy (1 cup)	911
Ham, canned (3 oz)	908	Cheese sauce, prepared from recipe (1 cup)	1198
Fast foods, shrimp, breaded and fried (6–8 shrimp)	1446	<b>Snacks, drinks, condiments, desserts</b>	
Submarine sandwich (one 6" roll, cold cuts)	1651	Orange juice (1 cup)	2
Smoked salmon (3 oz)	1700	Peanuts, unsalted (1 cup)	22
<b>Soups, Vegetables, Fruit</b>		Chocolate fudge (1 oz)	54
Apple (1)	0	Diet cola, with Saccharin	75
Banana (1)	1	Club soda (12 oz)	78
Mixed vegetables, frozen (1 cup)	64	Potato chips (10)	94
Mixed vegetables, canned (1 cup)	243	Mustard (1 tbsp)	129
Chicken noodle soup, canned (1 cup)	1106	Ketchup (1 tbsp)	156 (1 cup)
Tomato sauce, canned (1 cup)	1482	Hard pretzel (1)	258
Sauerkraut (1 cup)	1560	Shortbread cookies (2)	300
<b>Breads and grains</b>		Apple pie (1 slice)	476
Wheat bread (1 slice)	106	Peanuts, salted (1 cup)	626
Oatmeal, cooked (1 cup)	2	Vegetable juice (1 cup)	883
Italian bread (1 slice)	176	Dill pickle (1)	928
Bagel (1)	245	Pretzel twists (10)	966
English muffin (1)	378	Pie crust, 1 shell	976
Bread crumbs (1 cup)	3180	Beef bouillon (1 packet)	1019

**REFERENCE**

USDA Nutrient Database. Web site accessed July 7, 2009, at <http://www.nal.usda.gov/fnic/foodcomp/Data/SR14/wtrank/sr14w307.pdf>.

**TABLE 6-8 Tips for Lowering Sodium in the Diet**

Choose More Often	Choose Less Often
<ul style="list-style-type: none"> <li>Fresh, plain frozen, or canned “with no salt added” vegetables</li> <li>Fresh poultry, fish, and lean meat, rather than canned or processed types</li> <li>Rice, pasta, and hot cereals cooked without salt. Cut back on instant or flavored rice, pasta, and cereal mixes, which usually have added salt.</li> <li>“Convenience” foods that are lower in sodium. Cut back on frozen dinners, pizza, packaged mixes, canned soups or broths, and salad dressings; these often have a lot of sodium</li> <li>Canned foods, such as tuna, drained and rinsed to remove some sodium</li> <li>Low- or reduced-sodium or no salt added versions of foods</li> <li>Ready-to-eat breakfast cereals that are lower in sodium, such as shredded wheat</li> <li>Rinse canned beans before using</li> <li>Herbs, spices, and salt-free seasoning blends in cooking and at the table. To make a spice blend, mix together 1 tablespoon each: ground cumin, onion powder, ground celery seed, ground basil, ground marjoram, ground oregano, ground thyme, ground coriander, crushed rosemary, garlic powder and paprika. One teaspoon contains 10 mg sodium and 46 mg potassium</li> </ul>	<ul style="list-style-type: none"> <li>Hogmaws, ribs, and chitterlings</li> <li>Smoked or cured meats like bacon, bologna, hot dogs, ham, corned beef, luncheon meats, and sausage</li> <li>Canned fish like tuna, salmon, sardines, and mackerel</li> <li>Buttermilk</li> <li>Most cheese spreads and cheeses</li> <li>Salty chips, nuts, pretzels, and pork rinds</li> <li>Some cold (ready to eat) cereals highest in sodium, instant hot cereals</li> <li>Quick cooking rice and instant noodles, boxed mixes like rice, scalloped potatoes, macaroni and cheese, and some frozen dinners, pot pies, and pizza</li> <li>Regular canned vegetables</li> <li>Pickled foods such as herring, pickles, relish, olives, and sauerkraut</li> <li>Regular canned soups, instant soups</li> <li>Butter, fatback, and salt pork</li> <li>Soy sauce, steak sauce, salad dressing, ketchup, barbecue sauce, garlic salt, onion salt, seasoned salts like lemon pepper, bouillon cubes, meat tenderizer, and monosodium glutamate (MSG)</li> </ul>

From: National Heart, Lung, and Blood Institute. Reduce salt and sodium in your diet. Accessed July 7, 2009, at <http://www.nhlbi.nih.gov/hbp/prevent/sodium/sodium.htm>.

- The following Figure provides a suggested interdisciplinary nutrition care plan:

<b>INTERDISCIPLINARY NUTRITION CARE PLAN</b> <b>Congestive Heart Failure (CHF)</b>	
<b>Client Name:</b> _____ <b>#:</b> _____ <b>Initiated by:</b> _____ <b>Date:</b> _____	
<div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>SCREEN</b>            Nutrition Screen diagnosis: CHF            Signed: _____ Date: _____         </div> <div style="text-align: center; font-size: 2em; color: blue; margin: 5px 0;">↓</div> <div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>ASSESS</b> <i>(Check any/all)</i>  <b>Shortness of breath (SOB) while</b>  <input type="checkbox"/> Eating      <input type="checkbox"/> Performing ADLs  <b>Weight/BMI</b>  <input type="checkbox"/> BMI &lt;20 (High Risk)  <input type="checkbox"/> BMI &lt;27  <input type="checkbox"/> Fluctuations ≥ 3–5 lb/wk  <b>Hydration status</b>  <input type="checkbox"/> Edema      <input type="checkbox"/> 1+      <input type="checkbox"/> 2+      <input type="checkbox"/> 3+  <input type="checkbox"/> Fluid restriction  <b>Exercise tolerance</b>  <input type="checkbox"/> Fatigue      <input type="checkbox"/> Restlessness  <input type="checkbox"/> Medications  <input type="checkbox"/> Pre- or postsurgery  <b>Poor Oral Intake Symptoms</b>  <input type="checkbox"/> Complex diet order  <input type="checkbox"/> Nausea/vomiting  <input type="checkbox"/> Poor appetite/early satiety  <input type="checkbox"/> Problems chewing/swallowing  <input type="checkbox"/> Depression/anxiety  <input type="checkbox"/> GI distress  <input type="checkbox"/> Anorexia            Signed: _____ Date: _____         </div> <div style="text-align: center; font-size: 1.5em; color: blue; margin: 5px 0;">↓</div> <div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>HIGH-RISK INTERVENTIONS</b> <i>(Check any/all)</i>  <input type="checkbox"/> <b>Eating Well With CHF</b> provided and explained  <input type="checkbox"/> <b>Food Record</b> provided and explained  <input type="checkbox"/> <b>How to read labels and track sodium intake</b> stressed  <b>Obtain Dr. orders as needed:</b>  <input type="checkbox"/> RD referral for home visit(s)  <input type="checkbox"/> Monitor weight q: _____  <input type="checkbox"/> Monitor I &amp; O q: _____  <input type="checkbox"/> Multiple vitamin/mineral supplement  <input type="checkbox"/> BID/TID supplements  <input type="checkbox"/> <b>Other:</b> _____            (See notes for documentation.)            Signed: _____ Date: _____         </div> <div style="text-align: center; font-size: 1.5em; color: blue; margin: 5px 0;">↓</div> <div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>ASSESS RESPONSE</b> <i>(Check any/all)</i>            SOB while  <input type="checkbox"/> Eating      <input type="checkbox"/> Performing ADLs  <input type="checkbox"/> Weight fluctuations  <input type="checkbox"/> Exercise tolerance declining  <input type="checkbox"/> Fatigue increasing  <b>Hydration status</b>  <input type="checkbox"/> Edema      <input type="checkbox"/> Dehydration  <input type="checkbox"/> <b>Other:</b> _____            (See notes for documentation.)            Signed: _____ Date: _____         </div> <div style="text-align: center; font-size: 1.5em; color: blue; margin: 5px 0;">↓</div> <div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>OUTCOMES NOT ACHIEVED</b>            Reassess/evaluate need for EN/PN (refer to Tube Feeding Nutrition Care Plan). Document on Nutrition Variance Tracking form.         </div>	<div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>GOALS</b> <i>(Check any/all):</i>  <input type="checkbox"/> Maintain or improve nutritional status in _____ (goal time).  <input type="checkbox"/> Eat meals/snacks without experiencing shortness of breath (SOB) in _____ (goal time).  <input type="checkbox"/> Perform Activities of Daily Living (ADLs) with minimal SOB in _____ (goal time).         </div> <div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>MODERATE RISK INTERVENTIONS</b> <i>(Check any/all)</i>  <input type="checkbox"/> <b>Eating Well With CHF</b> provided and explained  <input type="checkbox"/> <b>Food Record</b> provided and explained  <input type="checkbox"/> <b>How to read labels for sodium content</b> explained and encouraged  <b>Obtain Dr. orders as needed:</b>  <input type="checkbox"/> RD chart consult  <input type="checkbox"/> Monitor weight q: _____  <input type="checkbox"/> BID/TID supplements  <input type="checkbox"/> <b>Other:</b> _____            (See notes for documentation.)            Signed: _____ Date: _____         </div> <div style="text-align: center; font-size: 1.5em; color: blue; margin: 5px 0;">↓</div> <div style="border: 1px solid black; padding: 5px; margin-bottom: 10px;"> <b>OUTCOMES ACHIEVED</b>  <input type="checkbox"/> SOB decreased  <input type="checkbox"/> Weight stabilized or improved  <input type="checkbox"/> Exercise tolerance maintained or improved  <input type="checkbox"/> Hydration status maintained or improved  <input type="checkbox"/> Nutritional status maintained or improved  <input type="checkbox"/> <b>Other:</b> _____            (See notes for documentation.)  <input type="checkbox"/> Repeat Nutrition Risk Screen in _____ days            Signed: _____ Date: _____         </div>

## Common Drugs Used and Potential Side Effects

- Most patients with HF require combination therapy that includes a diuretic, an ACE inhibitor, an angiotensin II receptor blocker, beta-blockers, aldosterone antagonists, specialized implantable pacemakers. Once BP is controlled, acetylsalicylic acid therapy should be considered (Kahn et al, 2009).
- Nesiritide, a recombinant form of BNP may be used with acute HF (Waldo et al, 2008). Because obesity is associated with lower BNP levels in healthy individuals and patients with chronic congestive HF, use caution for interpretation of BNP levels (Krauser et al, 2005).
- Teach how to manage vitamin K intake if on warfarin. See Table 6-9.

**TABLE 6-9 Medications Used in Heart Failure**

Medication	Description	Medication	Description
<b>Angiotensin-converting enzyme (ACE) inhibitors</b>	ACE inhibitors block angiotensin II and decrease aldosterone output, thereby decreasing sodium and water retention. Monitor for hyperkalemia, nausea, vomiting, dizziness, and abdominal pain.	<b>Adrenergic system blockers</b>	Beta-adrenergic blockers reduce cardiac output in competing for available receptor sites; they decrease sympathetic stimulation of the heart. At first use, they may cause fatigue and fluid retention.
Benazepril ( <i>Lotensin</i> )		<b>Beta-blockers</b>	
Captopril ( <i>Capoten</i> )		Atenolol ( <i>Tenormin</i> )	
Enalapril ( <i>Vasotec</i> )		Bisoprolol ( <i>Zebeta</i> )	
Fosinopril ( <i>Monopril</i> )		Carvedilol ( <i>Coreg</i> )	
Lisinopril ( <i>Zestril/Prinivil</i> )		Metoprolol ( <i>Toprol XL</i> )	
Moexipril ( <i>Univasc</i> )		Propranolol ( <i>Inderal</i> )	
Perindopril ( <i>Aceon</i> )		<b>Cardiac glycosides</b>	Digitalis can deplete potassium, especially when taken with furosemide. Beware of excesses of wheat bran, which can decrease serum drug levels. Anorexia or nausea may occur.
Quinapril ( <i>Accupril</i> )		<b>Digitalis</b>	
Ramipril ( <i>Altace</i> )		Lanoxin (Digoxin)	
Trandolapril ( <i>Mavik</i> )			
<b>Angiotensin receptor blockers</b>	Common adverse effects include hypotension and dizziness, a concern when there is also blurred vision or worsening renal function.	<b>Diuretics</b>	Avoid use with fenugreek, ginkgo, and yohimbe. Some diuretics spare calcium and protect bone health.
Candesartan ( <i>Atacand</i> )		Hydrochlorothiazide ( <i>Microzide</i> );	
Eprosartan ( <i>Teveten</i> )		Eplerenone ( <i>Inspira</i> ); Furosemide ( <i>Lasix</i> ); Spironolactone ( <i>Aldactone</i> )	
Irbesartan ( <i>Avapro</i> )		<b>Loop diuretics</b>	Most loop diuretics deplete potassium and magnesium; calcium levels also decline. Glucose tolerance may be decreased; anorexia, nausea, or vomiting may occur. Use a low-sodium diet. Avoid use with Nonsteroidal anti-inflammatory drugs (NSAIDs).
Losartan ( <i>Cozaar</i> )		Bumetanide, Furosemide ( <i>Lasix</i> ), Torsemide	
Olmesartan ( <i>Benicar</i> )			
Telmisartan ( <i>Micardis</i> )			
Valsartan ( <i>Diovan</i> )			
<b>Aldosterone blockers</b>		<b>Thiazide diuretics</b>	Most thiazide diuretics deplete potassium, which must be replaced, either orally or by medication. Hyperkalemia can occur with use of potassium-sparing diuretics if lab values are not carefully monitored. Use potassium-rich foods and juices such as orange juice, bananas, potatoes. Monitor for signs of dehydration.
Eplerenone, Spironolactone		Hydrochlorothiazide ( <i>Microzide</i> )	
<b>Anticoagulants</b>	Consume foods high in vitamin K no more than once per day.	Chlorthalidone	
Warfarin (Coumadin)		Indapamide	
	Foods with high vitamin K content include: spinach, broccoli, Brussels sprouts, green raw cabbage, turnip greens, mustard greens, collard greens, parsley, green scallions, lettuce, endive, watercress, cucumber peels, kale, canola oil, soybean oil, raw chives, green onions, seaweed, green peas, liver, Swiss chard.	Metolazone	
		<b>Potassium-sparing diuretics</b>	No extra K <sup>+</sup> is needed.
	Avoid taking with dong quai, fenugreek, feverfew, ginger, ginkgo, ginseng, excesses of ginger, aniseed, celery, cranberry juice, dandelion, licorice, onion, passion flower, or willow bark. Coenzyme Q10, green tea, goldenseal, St. John's wort, and yarrow will decrease the effectiveness of warfarin.	Amiloride	
		Spironolactone ( <i>Aldactone</i> )	
		Triamterene ( <i>Dyrenium</i> )	
		<b>Salt substitutes</b>	Salt substitutes generally contain KCl. Use could lead to hyperkalemia if potassium-sparing diuretics are part of treatment.
		<b>Statins</b>	
			Statins may be used if the underlying problem is related to coronary artery disease or dyslipidemia.

## REFERENCE

- Hunt et al, 2005. Cleveland Clinic, website accessed 10/11/10 at: <http://www.clevelandclinic.org/health/health-info/docs/1800/1822.asp?index=8121>
- Hunt SA, et al. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). *J Am Coll Cardiol*. 46:e-1, 2005.



## Herbs, Botanicals, and Supplements

- The patient should not take herbals and botanicals without discussing with the physician. It is important to stress that no supplement or diet can cure HF. Taurine, hawthorn, magnesium, and other supplements have not been documented as effective at this time. See Tables 6-3 and 6-9 for more details.
- CoQ10 deficiency is detrimental to the long-term prognosis of HF (Molyneux et al, 2008). Supplementation of both CoQ10 and creatine may prove to be useful in HF; more controlled trials are needed.
- Supplementation with eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) from fish oil may prevent HF through lowering proinflammatory fatty acid arachidonic acid and urine thromboxane B<sub>2</sub>; high-fat diets (60% fat) block these effects in the chronically stressed myocardium.
- Pomegranate (*Punica granatum*) berries, grape and other antioxidant-rich juices can be safely consumed.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Identify the stage of readiness for change in the patient. The most difficult lifestyle changes include smoking cessation, weight loss, and restriction of dietary sodium (Paul and Sneed, 2004). Supervised nutrition intervention is of great importance (Colin Ramirez et al, 2004).
- Help patient plan fluid intake; usually 75% with meals and 25% with medications or between meals.
- A congested feeling may cause a poor appetite. Offer small, appetizing, frequent snacks, or meals. Use high-calorie, low-volume supplements to increase nutrient density when needed.
- Never force the patient to eat; rest before and after meals. Naps are good for heart health. Even bed rest may be required. To reduce congestion in the lungs, the patient's upper body should be elevated. For most patients, resting in an armchair is better than lying in bed. Relaxing and contracting leg muscles are important to prevent blood clots. As the patient improves, progressively more activity will be recommended.
- HF is associated with sleep apnea, in which tissues at the back of the throat periodically collapse and become blocked, causing the sleeper to gasp for air. Sleep apnea is associated with poorer survival in patients with HF. A continuous positive airway pressure (CPAP) device appears to improve ejection fraction; see Section 5.
- Teach label reading and tips for easy meal preparation. Choose items that state: sodium free, very low sodium, low sodium, reduced sodium, light in sodium, or unsalted. Avoid excessive use of canned soups, cured or smoked meats, and commercial sauces. Many frozen dinners are high in sodium; choose healthier brands.
- Check the water supply for use of softening agents. Monitor sodium-containing medications, toothpastes, and mouthwashes. Discuss spices and seasonings as salt alternatives.
- Freeze small meal portions to simplify meal preparation. Refer to local congregate meal programs or inquire about home-delivered meals if needed; many will provide low-sodium meals upon request.
- Implantation of circulatory assist devices as a permanent alternative to heart transplantation (destination therapy) is used with patients with HF who are not responding to medications. The HeartMate Left Ventricular Assist System is FDA-approved.

## Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

## For More Information

- Heart Failure  
<http://www.hearthope.com/learn.html>
- National Heart, Lung, and Blood Institute  
<http://www.nhlbi.nih.gov/hbp/hbp/effect/heart.htm>  
[http://www.nhlbi.nih.gov/hbp/prevent/h\\_eating/h\\_eating.htm](http://www.nhlbi.nih.gov/hbp/prevent/h_eating/h_eating.htm)
- Spices and Seasonings  
<http://www.nhlbi.nih.gov/hbp/prevent/sodium/flavor.htm>

## HEART FAILURE—CITED REFERENCES

- Ahmed HN, et al. Coffee consumption and risk of heart failure in men: an analysis from the Cohort of Swedish Men. *Am Heart J*. 158:667, 2009.
- American Dietetic Association. Heart Failure Evidence-Based Nutrition Practice Guideline. Accessed July 13, 2009, at <http://www.adaevidencelibrary.com/topic.cfm?cat=3249>.
- Colin Ramirez E, et al. Effects of a nutritional intervention on body composition, clinical status, and quality of life in patients with heart failure. *Nutrition*. 20:890, 2004.
- Groenveld HF, et al. Anemia and mortality in heart failure patients: a systematic review and meta-analysis. *J Am Coll Cardiol*. 52:818, 2008.
- Gums JG. Magnesium in cardiovascular and other disorders. *Am J Health Syst Pharm*. 61:1569, 2004.
- Kahn NA, et al. The 2009 Canadian Hypertension Education Program recommendations for the management of hypertension: Part 2—therapy. *Can J Cardiol*. 25:287, 2009.
- Krauser DG, et al. Effect of body mass index on natriuretic peptide levels in patients with acute congestive heart failure: a ProBNP Investigation of Dyspnea in the Emergency Department (PRIDE) substudy. *Am Heart J*. 149:744, 2005.
- Kris-Etherton P, et al. Milk products, dietary patterns and blood pressure management. *J Am Coll Nutr*. 28:103S, 2009.
- Leviton EB, et al. Consistency with the DASH diet and incidence of heart failure. *Arch Intern Med*. 169:851, 2009.
- Molyneux SL, et al. Coenzyme Q10: an independent predictor of mortality in chronic heart failure. *J Am Coll Cardiol*. 52:1435, 2008.
- Nettleton JA, et al. Incident heart failure is associated with lower whole-grain intake and greater high-fat dairy and egg intake in the Atherosclerosis Risk in Communities (ARIC) study. *J Am Diet Assoc*. 108:1881, 2009.
- Paul S, Sneed NV. Strategies for behavior change in patients with heart failure. *Am J Crit Care*. 13:305, 2004.
- Schulman SP, et al. L-arginine therapy in acute myocardial infarction: the Vascular Interaction with Age in Myocardial Infarction (VINTAGE MI) randomized clinical trial. *JAMA*. 295:58, 2006.

Shah KB, et al. The cardioprotective effects of fish oil during pressure overload are blocked by high fat intake. Role of cardiac phospholipid remodeling [published online ahead of print Jul 13, 2009]. *Hypertension*. 54:65, 2009.

Waldo SW, et al. Pro-B-type natriuretic peptide levels in acute decompensated heart failure. *J Am Coll Cardiol*. 51:1874, 2008.

Zhou B, et al. Functional polymorphism of the NFKB1 gene promoter is related to the risk of dilated cardiomyopathy. *BMC Med Genet*. 10:47, 2009.

## HEART TRANSPLANTATION OR HEART-LUNG TRANSPLANTATION

### NUTRITIONAL ACUITY RANKING: LEVEL 4



#### DEFINITIONS AND BACKGROUND

**Heart transplantation (HTx)** is usually performed for terminal HF, often with cardiomyopathy. Usually, the transplantation will be a Jarvik-7 or a live donor heart. Screening includes evaluations for chronic, coexisting illness, psychosocial stability, and normal or reversible cardiac status. The best candidates are younger than 55 years of age with normal hepatic and renal functioning and are free of diabetes mellitus and pulmonary problems, peptic ulcers, and peripheral heart disorders.

**Heart-lung transplantation** is rare, as for complex cases of cystic fibrosis, pulmonary fibrosis, emphysema, Eisenmenger's syndrome, and primary pulmonary hypertension. Graft-host resistance and sepsis are the major concerns.

Survival rates after transplantation are getting better. Pre-HTx diabetes, donor age, and incidences of infection and rejection within 2 years of HTx predict long-term (>10 years) survival (Radovancevic et al, 2005). Main causes of death early after transplantation are rejection, nonspecific graft failure, and right ventricular failure due to pulmonary hypertension (Bauer et al, 2005).

Implantation of circulatory assist devices as a permanent alternative to HTx is an option for patients who are not candidates for HTx (Lietz and Miller, 2005). Ventricular assist devices (VADs) are now quite common. More recently, cell-based cardiac repair using human embryonic stem cells (hESCs) represent an attractive source for obtaining cardiomyocytes (Zhu et al, 2009).

Traditional risk factors, high oxidative stress, and reduced antioxidant defenses play a role in the pathogenesis of atherosclerosis, especially in transplanted hearts. Dietary measures are important. Flavonoid-rich dark chocolate induces coronary vasodilation, improves coronary vascular function, and decreases platelet adhesion (Flammer et al, 2007). Omega-3 fatty acids lower Chol levels, improve endothelial function, and can reduce the risk of sudden death in HTx recipients (Harris et al, 2004; Wenke, 2004). Finally, obesity, dyslipidemia, hypertension, and diabetes mellitus are common after HTx. Therefore, dietary intervention to obtain weight and metabolic control after HTx is needed to decrease TC, TGs, glucose plasma level, and weight loss (Guida et al, 2009).



#### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Cardiac transplantation may be required for idiopathic DCM, which is inherited in approximately one third of cases. In addition, recipient renin-angiotensin-aldosterone system (RAAS) polymorphisms are associated with a higher risk of rejection, graft cytokine expression, graft dysfunction, and a higher mortality after cardiac transplantation (Auerbach et al, 2009).

<b>Clinical/History</b>	Ultrasound of abdomen and blood vessels	BUN, Creat AST, ALT ECG
Height	Cardiac catheterization	pCO <sub>2</sub> , pO <sub>2</sub>
Weight		Alb,
BMI		transferrin
Waist circumference	<b>Lab Work</b>	Chol—HDL, LDL, total
Diet history	Urinary Na <sup>+</sup>	Trig
Edema	Na <sup>+</sup> , K <sup>+</sup>	Gluc
BP	Ca <sup>++</sup> , Mg <sup>++</sup>	tHcy
Stress test	CRP	Serum folate and B <sub>12</sub>
Chest x-ray	H & H	
ECG	Serum Fe	
Coronary angiogram	Transferrin	
Echocardiogram	Complete blood cell count (CBC)	
Cardiopulmonary test		

#### INTERVENTION



#### OBJECTIVES

- Promote adequate wound healing; prevent or correct wound dehiscence.
- Normalize heart functioning; prevent morbidity and death. Control infection and rejection during the first 2 years after HTx to improve survival.

## SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Intake of Bioactive Substances**

**Assessment Data:** Food frequency recall and intake records; computer nutrient analysis showing no intake of omega 3 fatty acid-rich foods or chocolate. Consumes meals only at fast food restaurants. Low HDL and elevated LDL levels. Recent HTx surgery for ASHD and hypertension.

**Nutrition Diagnosis (PES):** Inadequate intake of bioactive substances (flavonols) related to poor diet as evidenced by diet history and daily fast food intake.

**Intervention:** Education about the role of antioxidant-rich foods and omega-3 fatty acids in heart disease, especially after transplantation. Counseling about food choice options besides eating daily at fast food restaurants and how to include chocolate, cocoa, nuts and seeds as easy snack items.

**Monitoring and Evaluation:** Repeat lab values after 3–6 months; dietary recall indicating use of bioactive substances at least once daily and fewer meals at fast food restaurants.

- Control side effects of steroid and immunosuppressive therapy.
- Prevent complications such as hepatic or renal failure and diabetes mellitus.
- Maintain or improve nutritional status and fluid balance.

- Protect against posttransplantation hyperlipidemia, hypertension, and graft coronary vasculopathy (GCV). GCV is an accelerated form of atherosclerosis in transplanted hearts that is one of the most important late complications of HTx and is the single most limiting factor for long-term survival (Wenke, 2004).



## FOOD AND NUTRITION

*Pretransplantation*

- Control calories, protein, sodium, potassium, fat, and Chol as appropriate for specific underlying condition (see appropriate sections). Keep in mind the role of nutrients needed for wound healing, including adequate energy intake.
- Fluid overload must be avoided; limit to 1 L daily, using a nutrient-dense product if needed.
- Avoid alcohol, which can aggravate cardiomyopathies.
- Reduce cardiac stimulants (such as caffeine) until fully recovered.

*Posttransplantation*

- Increase diet as tolerated and as appropriate for status. Alter as needed. Include appropriate levels of calcium, magnesium, potassium, and fiber; the DASH diet is beneficial.
- Increase use of omega-3 fatty acids from fish and fish oils.
- Increase use of cardioprotective agents such as vitamins E, B<sub>6</sub>, and B<sub>12</sub> and folic acid. It is also prudent to use olive, soybean, and canola oils.

TABLE 6-10 Medications Used after 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.
Cardiac medications	Antihypertensives, antilipemics, diuretics, and potassium supplements may be used. Monitor side effects accordingly. Some diuretics spare calcium and protect bone health.
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, hydrocortisone [Solu-Cortef])	Corticosteroids such as prednisone and hydrocortisone 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.
Diuretics	Diuretics such as furosemide 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.
Statins	Statins may be used to manage coronary artery disease or dyslipidemia.
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.

- Nuts contain flavonoids, phenols, sterols, saponins, elagic acid, folic acid, magnesium, copper, potassium, and fiber. Walnuts contain ALA; almonds are a good source of vitamin E. Macadamias, pecans, and pistachios are also beneficial.
- Include chocolate and cocoa frequently.
- For TF, use a product low in sodium and advance gradually.

### Common Drugs Used and Potential Side Effects

- See Table 6-10.

### Herbs, Botanicals, and Supplements

- The patient should not take herbals and botanicals without discussing with the physician.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of nutrition in wound healing, immunocompetence, and cardiovascular health. Specify nutrients that are known to be protective.
- Discuss how exercise affects the use of calories.
- Discuss, as appropriate, fiber intake and sources of fat and Chol. Highlight the importance of maintaining an adequate diet to reduce risks of further heart disease and complications. Transplant CAD proceeds at an accelerated rate; this procedure is not a permanent cure.
- To improve quality of life in transplantation patients, web-based counseling and support may play a vital role in follow-up care and in patient and family adjustments

(Dew et al, 2004). This type of support is helpful, especially in rural areas where access to MNT is limited.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- American Heart Association  
<http://www.americanheart.org/>
- Heart Transplantation  
<http://www.pbs.org/wgbh/nova/cheart/transplant.html>

### HEART TRANSPLANTATION OR HEART-LUNG TRANSPLANTATION—CITED REFERENCES

- Auerbach SR, et al. Recipient genotype is a predictor of allograft cytokine expression and outcomes after pediatric cardiac transplantation. *J Am Coll Cardiol*. 53:1909, 2009.
- Bauer J, et al. Perioperative management in pediatric heart transplantation. *Thorac Cardiovasc Surg*. 53:S155, 2005.
- Dew MA, et al. An internet-based intervention to improve psychosocial outcomes in heart transplant recipients and family caregivers: development and evaluation. *J Heart Lung Transplant*. 23:745, 2004.
- Flammer AJ, et al. Dark chocolate improves coronary vasomotion and reduces platelet reactivity. *Circulation*. 116:2376, 2007.
- Guida B, et al. Role of dietary intervention and nutritional follow-up in heart transplant recipients. *Clin Transplant*. 23:101, 2009.
- Harris WS, et al. Omega-3 fatty acids in cardiac biopsies from heart transplantation patients: correlation with erythrocytes and response to supplementation. *Circulation*. 110:1645, 2004.
- Lietz K, Miller LW. Will left-ventricular assist device therapy replace heart transplantation in the foreseeable future? *Curr Opin Cardiol*. 20:132, 2005.
- Radovancevic B, et al. Factors predicting 10-year survival after heart transplantation. *J Heart Lung Transplant*. 24:156, 2005.
- Wenke K. Management of hyperlipidaemia associated with heart transplantation. *Drugs*. 64:1053, 2004.
- Zhu WZ, et al. Human embryonic stem cells and cardiac repair. *Transplant Rev*. 23:53, 2009.

## HEART VALVE DISEASES

### NUTRITIONAL ACUITY RANKING: LEVEL 2



### DEFINITIONS AND BACKGROUND

The heart has four valves (tricuspid, pulmonary, aortic, and mitral). Inflammation of any or several of these valves can cause stenosis with thickening (which narrows the opening) or incompetence (with distortion and inability to close fully). If the mitral valve is not functioning properly, due to injury or disease, blood leaks back into the left atrium (regurgitates) when the left ventricle contracts and backs up into the lungs. Because some of the blood being pumped by the left ventricle flows back into the left atrium, less blood is pumped into the aorta and throughout the body. The heart compensates for this by increasing the size of the left ventricle to increase the amount of blood it is pumping and to maintain an adequate forward flow of blood throughout the body. Unfortunately, compensation eventually leads to impairment of the left ventricle's ability to contract, which leads to further backup of blood into the lungs.

**Mitral valve prolapse** is the most common cause of severe mitral regurgitation in the United States. Overall prognosis of patients is excellent, but a small subset will develop serious complications, including infective endocarditis or sudden cardiac death (Hayek et al, 2005). Echocardiography is used for diagnosing this condition, and mitral valve repair is the treatment. With advancements in PCIs, some patients benefit from a hybrid approach involving initial planned PCI followed by valve surgery, rather than conventional CABG/valve surgery (Byrne et al, 2005).

**Mitral stenosis** (stiffening) can cause lung congestion, breathlessness after exercise or while lying down, hemoptysis, bronchial infections, chest pains, and right HF. Most people who have had rheumatic heart disease later have primarily mitral stenosis. **Aortic stenosis** can cause symptoms of angina, vertigo, fainting on exertion, and left HF. **Tricuspid stenosis** increases the risk of HF. **Pulmonary stenosis** is rare and occurs in only 2% of all valve disorders.



Patients at high risk for valvular disease should be screened for hyperhomocysteinemia; for prevention, a multivitamin supplement with vitamins B<sub>6</sub> and B<sub>12</sub> and folate should be taken.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Congenital heart defects are a cause of some valve disorders, but most are acquired.

<b>Clinical/History</b>	Cardiac catheterization	H & H Serum ferritin
Height	ECG	Chol—total, HDL, LDL
Weight	Vertigo	Trig
BMI	Fainting	BUN, Creat
Waist-hip ratio	Breathlessness	Na <sup>+</sup> , K <sup>+</sup>
Diet history	after exertion?	Ca <sup>++</sup> , Mg <sup>++</sup>
Weight changes	Heart palpitations	Gluc
Edema in belly, ankles, feet	Shortness of breath	tHcy
Pulse		Serum folate
Cool, moist skin		Serum B <sub>12</sub>
BP	<b>Lab Work</b>	
Urinary output (decreased)	CRP	
I & O	Alb, transthyretin	

## INTERVENTION



### OBJECTIVES

- Prevent HF (right- or left-sided), bacterial endocarditis, emboli or atrial fibrillation, and sudden death. Prevent stroke; mitral annular calcification is an independent predictor of stroke.
- Prepare, if necessary, for valve replacement surgery.
- Prevent or correct cardiogenic shock with tachycardia and other symptoms.
- Correct or manage atherosclerosis.



### FOOD AND NUTRITION

- Avoid excesses of calories, sodium, and fluid (as appropriate for the patient). In some patients with vertigo, fluid and sodium restrictions may actually be detrimental.
- If weight loss has taken place, add extra calories and snacks to return to a more desirable body weight.
- Use adequate vitamins E, B<sub>6</sub>, and B<sub>12</sub> and folic acid.
- The DASH diet is useful. Encourage use of flavonoids such as chocolate, cocoa, pomegranate, or grape juices, apples, onions, tea, or red wine when feasible. Flavonoids may help to reduce blood clot formation.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Excessive Vitamin K Intake

**Assessment Data:** Food intake records; computer nutrient analysis showing intake of vitamin K from multivitamin supplement and fortified nutritional beverages exceeding 150% of recommended levels while on warfarin; INR 1.0–1.5 range.

**Nutrition Diagnosis (PES):** Excessive vitamin K intake related to intake of oral supplements and multivitamin preparation as evidenced by low INR while on anticoagulant therapy.

**Intervention:** Educate patient and family about appropriate levels of vitamin K. Counsel about an appropriate supplement and oral beverage choice with less vitamin K content.

**Monitoring and Evaluation:** Intake from multivitamin and oral supplements in lower range of daily vitamin K requirements; improved INR and bleeding times.

- Ensure adequate intake of omega-3 fatty acids. However, avoid high fat diets as the benefits of omega-3 s are reduced with those diets (Shah et al, 2009).
- Control intake of vitamin K while on warfarin. Changes in dietary vitamin K (phyloquinone) intake may contribute to marked variations in the INR in patients receiving oral warfarin anticoagulant therapy, with potentially serious adverse outcomes (Couris et al, 2006).

## Common Drugs Used and Potential Side Effects

- Anticoagulants are commonly used. Monitor vitamin K-rich foods carefully; use no more than 1 per day (especially green leafy vegetables).
- If aspirin is used, monitor for GI side effects or occult blood loss if used for a long time.
- Diuretics may be used with fluid overload. Monitor potassium and sodium intake carefully. Some diuretics spare calcium and protect bone health.
- Because fenfluramine-phentermine (Fen-Phen) weight reduction drugs promoted valvular heart disease and pulmonary hypertension, they were removed from the market.
- Digoxin may be needed to strengthen the heart's pumping action after surgery. Monitor potassium intake or depletion carefully, especially when combining with diuretics. Avoid excessive intakes of fiber and wheat bran. Avoid use with hawthorn, milkweed, guar gum, and St. John's wort.
- Statins may be used to manage CAD or dyslipidemia. See Table 6-6.

## Herbs, Botanicals, and Supplements

- See Table 6-3 for details.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Careful use of all prescribed medications will be essential, with adequate return visits to the physician at appropriate intervals.
- Alternative food preparation methods may be suggested to reduce sodium or energy intake.

- Persons with a history of heart valve abnormalities may require antibiotic therapy to prevent infections, especially before surgery or dental work.
- After surgery, the patient should receive information about nutrition in wound healing. Some procedures are more invasive than others and will require longer healing time.

#### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

#### For More Information

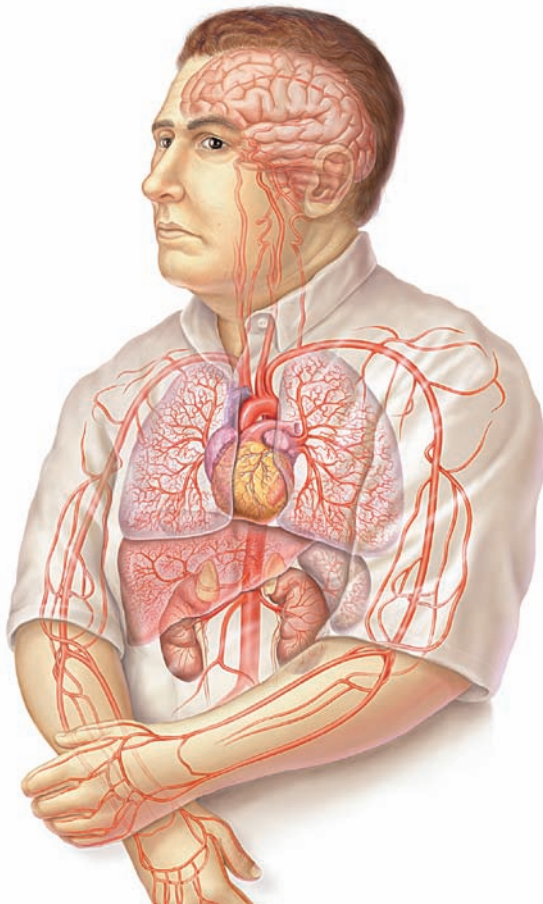
- Heart and Valvular Diseases  
<http://www.nhlbi.nih.gov/health/public/heart/index.htm>
- Heart Murmur  
[http://www.nhlbi.nih.gov/health/dci/Diseases/heartmurmur/hmurmur\\_what.html](http://www.nhlbi.nih.gov/health/dci/Diseases/heartmurmur/hmurmur_what.html)
- Heart Valve Disease  
<http://www.americanheart.org/presenter.jhtml?identifier=4598>
- Medicine Net—Valve Disease  
[http://www.medicinenet.com/heart\\_valve\\_disease/article.htm](http://www.medicinenet.com/heart_valve_disease/article.htm)
- Medline Plus  
<http://www.nlm.nih.gov/medlineplus/heartvalvediseases.html>
- Texas Heart Institute—Heart Anatomy  
<http://www.texasheartinstitute.org/HIC/Anatomy/Anatomy.cfm>

#### HEART VALVE DISEASES—CITED REFERENCES

- Byrne JG, et al. Staged initial percutaneous coronary intervention followed by valve surgery (“hybrid approach”) for patients with complex coronary and valve disease. *J Am Coll Cardiol.* 45:14, 2005.
- Couris R, et al. Dietary vitamin K variability affects International Normalized Ratio (INR) coagulation indices. *Int J Vitam Nutr Res.* 76:65, 2006.
- Hayek E, et al. Mitral valve prolapse. *Lancet.* 365:507, 2005.
- Shah KB, et al. The Cardioprotective effects of fish oil during pressure overload are blocked by high fat intake. Role of cardiac phospholipid remodeling [published online ahead of print Jul 13, 2009]. *Hypertension.* 54:65, 2009.

## HYPERTENSION

### NUTRITIONAL ACUITY RANKING: LEVEL 3



Asset provided by Anatomical Chart Co.



#### DEFINITIONS AND BACKGROUND

Hypertension is defined as having a sustained systolic and diastolic BP greater than 140 and 90 mm Hg, respectively. See Table 6-11. It affects about 600 million people worldwide and about 27% of the U.S. adult population.

Hypertension nearly doubles the risk for heart attack, stroke, and HF, especially for people over age 65. BP often increases with age and is highly prevalent in elderly individuals. Symptoms of hypertension include frequent headaches, impaired vision, shortness of breath, nose bleeds, chest pain, dizziness, failing memory, snoring and sleep apnea, and GI distress.

**TABLE 6-11** Categories for Blood Pressure Levels in Adults (Ages 18 years and Older)

Category	Blood Pressure Level (mm Hg)		
	Systolic		Diastolic
Normal	<120	and	<80
Prehypertension	120–139	or	80–89
High blood pressure:			
Stage 1 hypertension	140–159	or	90–99
Stage 2 hypertension	≥160	or	≥100

From: National Heart, Lung, and Blood Institute. Accessed July 16, 2009, at <http://www.nhlbi.nih.gov/hbp/detect/categ.htm>.

Identifiable causes of high BP include sleep apnea, drug-related causes, CKD, Cushing's syndrome, steroid therapy, pheochromocytoma, primary aldosteronism, thyroid and parathyroid diseases, and reno-vascular disease. Untreated hypertension can result in stroke, HF, renal failure, MI, accelerated bone loss and risk of fractures, and long-term memory problems. The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, the JNC 7 Report (NHLBI, 2009) identified following priority concerns:

- In persons over age 50, systolic BP >140 mm Hg is a more important CVD risk factor than diastolic BP. Those who are normotensive at age 55 have a 90% lifetime risk of developing hypertension. Beginning at 115/75 mm Hg, CVD risk doubles for each increment of 20/10 mm Hg.
- Prehypertensive individuals (systolic BP of 120–139 mm Hg or diastolic BP of 80–89 mm Hg) require health-promoting lifestyle modifications to prevent the progressive rise in BP and CVD. However, patients must be motivated to stay on their treatment plans.

Endothelial activation, oxidative stress, and vascular smooth muscle dysfunction (hypertrophy, hyperplasia, remodeling) can promote inflammation and stiffened blood vessels (Houston, 2005). Elevated CRP is a marker of reduced production of nitric acid in the blood vessels. CRP over 3.5 represents endothelial dysfunction, the earliest event in atherosclerosis (Streppel et al, 2005). With endothelial dysfunction, diet greatly affects vascular reactivity.

Fish oil, antioxidants, folic acid, soy protein, and the Mediterranean diet (high consumption of vegetables, fish, and olive oil and moderate wine consumption) may have a positive effect. Increasing intake of fiber in populations where intake is far below recommended levels may help to prevent hypertension (Streppel et al, 2005). Weight loss is also important; abdominal adiposity promotes high CRP levels.

There is increasing evidence for use of nutrients such as omega-3 PUFA, vitamin C, folic acid, and potassium. The DASH eating plan is rich in fiber, vegetables, fruit, and non-fat dairy products and significantly lowers BP. The decreases are often comparable to those achieved with BP-lowering medication.

For patients with CKD who have hypertension; it is critical to control BP to reduce negative consequences. Current National Kidney Foundation guidelines recommend reducing sodium intake to less than 2.4 g/d; reducing potassium in patients with a GFR of <60 mL/min. Black populations benefit most from reduced salt intake, increased potassium intake, and the DASH diet (Appel et al, 2006).

Because nutrient–gene interactions determine a broad array of consequences such as vascular problems and hypertension, consuming optimal nutrition, nutraceuticals, vitamins, antioxidants, and minerals and moderately restricting alcohol may prevent, delay the onset, reduce the severity, treat, or control hypertension (Houston, 2005). For example, Vitamin D deficiency activates the rennin–angiotensin–aldosterone system and can predispose to hypertension and left ventricular hypertrophy; it can also increase parathyroid hormone that is associated with insulin resistance and hypertension (Lee et al, 2008).

Malignant hypertension, which occurs in only 1% of those with essential hypertension (EH), has medical urgency. In this condition, there is accelerated hypertension (systolic >220 mm Hg or diastolic >120 mm Hg) with no evidence of target organ damage. This differs from a medical emergency, such as a stroke, where it is important to lower elevated BP immediately. African Americans are at higher risk than individuals with European heritage.

Long-term sodium reductions of 400 mg in those with uncontrolled hypertension would eliminate about 1.5 million cases, potentially increasing productivity by \$2.5 billion annually and more aggressive diet changes of 1100 mg of sodium reductions yield potential productivity benefits of \$5.8 billion annually (Dall et al, 2009). Therefore, clinicians are encouraged to work closely with patients to agree on BP goals and develop a treatment plan. Reduced salt intake, weight loss, moderate alcohol consumption among those who drink, increased potassium intake, and use of the DASH diet are among the most effective strategies (Appel et al, 2006). The American Dietetic Association recommends at least three MNT visits for patients with hypertension.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** A genome-wide association study of systolic BP (SBP) and diastolic BP (DBP) and hypertension in the CHARGE Consortium, identified significance in four SNPs for SBP (ATP2B1, CYP17A1, PLEKHA7, SH2 B3), six for DBP (ATP2B1, CACNB2, CSK-ULK3, SH2 B3, TBX3-TBX5, ULK4), and one (ATP2B1) for hypertension (Levy et al, 2009). Severe mutations often result in the development of hypertension at a young age while subtle mutations may take decades to manifest. The MTHFR folate alleles are associated with EH because of their relationship with tHcy metabolism (Ilhan et al, 2008). Identifying genes associated with BP advances understanding of BP regulation and highlights potential drug targets for the prevention or treatment (Levy et al, 2009; Mein et al, 2004; Wang et al, 2009).

Clinical/History	I & O	CRP
Height	Renal arteriography	Alb,
Weight		transthyretin
BMI	Chest x-ray	Urinary Alb to
Waist circumference	Renal ultrasound	Creat ratio
BP pattern	Intravenous	(high levels
Diet history	pyelogram	impair
Headaches,	ECG	arterial dila-
dizziness		tory capacity)
Snoring or sleep	<b>Lab Work</b>	Chol—HDL,
apnea?	H & H	LDL
Polysomnogram	Serum Fe,	Trig
	ferritin	LDH



BUN	Parathormone	Serum folate
Creat, GFR	(PTH)	and B <sub>12</sub>
Uric acid	AST, ALT	Plasma ascorbic
Plasma renin	Alk phos	acid
Na <sup>+</sup> , K <sup>+</sup>	Serum vitamin D	Urinary Ca <sup>++</sup>
Chloride	PT or INR	Urinary cortisol
Serum Ca <sup>++</sup> , Mg <sup>++</sup>	Gluc tHcy	

## INTERVENTION



### OBJECTIVES

- Assess medical risk factors, comorbidities, and identifiable causes of the hypertension. For example, essential hypertension affects 1 billion people worldwide and has a genetic basis (Mein et al, 2004).
- Reduce cardiovascular (HF, stroke) and renal morbidity and mortality (ADA, 2009) by lowering high BP. The DASH dietary pattern reduces SBP by 8–14 mm Hg (ADA, 2009).
- For individuals with hypertension and diabetes or renal disease, maintain BP goal of <130/80 mm Hg (ADA, 2009).
- Increase vascular and lymphocyte beta-adrenergic responsiveness. Dietary sodium intake should be limited to no more than 2300 mg sodium (100 mmol) per day; this level can lower SBP by approximately 2–8 mm Hg (ADA, 2009).
- Achieve and maintain an optimal body weight (BMI 18.5–24.9); weight reduction lowers SBP by 5–20 mm Hg per 22 lb (10 kg) body weight loss (ADA, 2009).
- Increase magnesium, calcium, vitamins D, E, and K where serum levels or dietary intake is low.
- Encourage adequate intake of fluids unless contraindicated. Avoid excesses of alcohol that may increase BP.
- Dietitians should encourage individuals to engage in aerobic physical activity for at least 30 min/d on most days of

the week, as it reduces systolic BP by approximately 4–9 mm Hg (ADA, 2009).



## FOOD AND NUTRITION

- The DASH diet works within 14 days of initiation. This diet is rich in fruits, vegetables, and low-fat dairy foods; it is low in SFA and total fat. Adequate amounts of potassium from skim milk, baked potatoes, grapefruit, oranges, bananas, lima beans, and other fruits and vegetables should be planned daily.
- Tips on eating the DASH way: Start small; make gradual changes in eating habits. Organize meals around carbohydrates such as pasta, rice, beans, or vegetables. Carbohydrates such as beans, whole grains, oat bran, and fruits (apples, blueberries) and vegetables should make up 50% of the diet.
- Treat meat as one part of the whole meal, instead of the main focus. Use fruits or low-fat, low-calorie foods such as sugar-free gelatin for desserts and snacks.
- Increase fruits and vegetables (5–10 servings daily) for their flavonoid, phytochemical, potassium content and properties (ADA, 2009). Besides the DASH diet, Mediterranean and vegetarian diet patterns tend to lower BP and can be beneficial.
- Limit sodium intake. Only 20–50% of patients with hypertension are sodium sensitive. Patients with EH are more sodium sensitive than patients whose conditions are secondary to other disorders (Houben et al, 2005).
- Read labels carefully. If something is sodium free, it has 5 mg or less per serving. Something that is light in sodium has 50% less than the usual recipe. A food that is labeled low sodium must be 149 mg or less per serving. About 77% of salt in the diet comes from processed foods, 12% naturally, and the remainder is either added during cooking or at the table. Table 6-12 lists common salts, salt substitutes, and their content.
- Use an energy-controlled diet if weight loss is needed.
- Olive, soybean, and canola oils can be substituted for some saturated fats in cooking. Pistachios, sunflower kernels, sesame seeds, and wheat germ are good sources of phytosterols; use often.
- Limit alcoholic beverages to one drink for women and to two drinks for men.
- Use sources of omega-3 fatty acids, such as mackerel, haddock, sardines, and salmon, several times weekly. Tuna should be used less often because potential mercury content could elevate BP.
- Higher intake of dairy products seems to reduce serum uric acid levels; high uric acid levels are often correlated with BP and stroke (Choi et al, 2005).
- Increase food sources of folic acid, vitamins B<sub>12</sub>, and B<sub>6</sub> for overall cardiovascular health. Vitamin D is also important. Vitamin D deficiency is associated with increased risk of developing incident hypertension or sudden cardiac death in individuals with preexisting CVD (Judd and Tangpricha, 2009).
- Caffeine from habitual coffee intake is not problematic (Winkelmayer et al, 2005). Hibiscus tea, green tea, cocoa and chocolate should be encouraged.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Hypertension (HTN)

**Assessment:** Diet history and food records, BP records.

**Nutrition Diagnosis (PES):** Excessive intake of sodium related to high intake of commercially prepared and packaged foods as evidenced by BP s of 160/105 mm Hg and lack of knowledge about sources of sodium.

**Intervention:** Initial/brief nutrition education (E-1) to communicate the relationship between nutrition and HTN. Discuss lower sodium food choices, label reading, and options to reduce BP. Discuss choices for healthier meals and snacks. Promote use of BP monitoring records, easy preparation meals made from scratch, recipe alterations, shopping tips, tips for dining away from home.

**Monitoring and Evaluation:** BP reports, food records showing improved intake of sodium in daily diet.



**TABLE 6-12 Sodium and Potassium in Salt, Salt Substitutes, Herbal Seasonings**

Type	Milligrams (mg) of Sodium per $\frac{1}{4}$ tsp	Milligrams (mg) of Potassium per $\frac{1}{4}$ tsp
Morton's table salt	590	0
Morton sea salt	560	0
Morton's salt balance	440	200
Diamond crystal salt sense	390	0
Lawry's seasoned salt	380	0
Morton's lite salt	290	350
Papa dash	240	0
Baking soda	205	0
Monosodium glutamate	123	0
Sterling lo-salt mixture	115	150
Baking powder	85	0
Nu salt	0	795
Nosalt	0	650
Morton salt substitute	0	610
Also salt	0	300
Mrs. dash salt-free seasoning blends	0	5–15
Benson's gourmet salt free seasonings	<5	minimal
Durkee smart seasons	0	0–15
McCormick salt-free seasoning blends	1	20–40

Derived from: Edwards A. Salt, salt substitutes, and seasoning alternatives. *J Renal Nutr.* 18:e23, 2008.

### Common Drugs Used and Potential Side Effects

- Dietitians should assess food/nutrient-medication interactions in patients that are on pharmacologic therapy for hypertension, as many antihypertensive medications interact with food and nutrients (ADA, 2009). See Table 6-13.
- In uncomplicated stage 1 hypertension, dietary changes are the initial treatment, then drug therapy. In hypertensive patients already on drug therapy, lifestyle modification and reduced salt intake, can further lower BP (Appel et al, 2006).
- Use of a diuretic is part of the treatment plan in most patients. For stage 1, thiazide diuretics may suffice; in stage 2, two-drug combinations may be needed. Drug classes that have been shown to be effective in reducing hypertension's cardiovascular complications include ACE inhibitors, angiotensin receptor blockers, beta-blockers, and calcium channel blockers. Most persons will need multiple medications to lower BP to a desired level.
- Note that use of estrogens and oral contraceptives can increase BP.
- Antihypertensive therapy is challenging in the elderly because of metabolic and physiological alterations, comorbidities, polypharmacy, and biological variability. For example, ACE inhibitors and beta-blockers may

provide beneficial therapeutic effects to the EH patients by decreasing tHcy levels, while thiazide diuretics increase tHcy (Poduri et al, 2009).

### Herbs, Botanicals, and Supplements

- Green coffee bean extract, oolong, and moderate-strength green teas may be beneficial in lowering BP (Kozuma et al, 2005). Hibiscus tea may also be beneficial.
- While vitamin D supplementation is simple, safe, and inexpensive, large randomized controlled trials are needed to identify how much is needed to prevent hypertension (Lee et al, 2008).
- Use of 1000 mg of a specific olive leaf extract EFLA943 has been shown to lower BP in twins (Perrinjacquet-Moccetti et al, 2008). Larger population studies are suggested.
- See Table 6-3 for more information.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage patience; it takes 2 weeks to see the results while following the DASH diet.
- Remove the salt shaker from the table. Have the patient taste food before salting. Avoid excesses of processed and

**TABLE 6-13 Medications for Hypertension**

Medication	Description
<b>Angiotensin-converting enzyme (ACE) inhibitors</b> (benazepril/Lotensin, captopril/Capoten, enalapril/Vasotec, fosinopril/Monopril, lisinopril/Prinivil or Zestril, moexipril/Univasc, perindopril/Aceon, quinipril/Accupril, ramipril/Altace, trandolapril/Mavik)	ACE inhibitors prevent angiotensin I from conversion; they are useful in heart failure. Ramipril has been noted to prevent diabetes in hypertensive patients. Nausea, vomiting, and abdominal pain may occur; do not take with potassium supplements. Captopril (Capoten) can alter BUN/creatinine; take 1 hour before meals and reduce calories and sodium. Loss of taste can occur. Patients who take captopril and enalapril may develop zinc deficiency.
<b>Angiotensin II receptor antagonists</b> (candesartan/Atacand, eprosartan/Teveten, irbesartan/Avapro, losartan/Cozaar, olmesartan/Benicar, telmisartan/Micardis, valsartan/Diovan)	Use with low-sodium, low-calorie diet. GI distress can occur. Some are mixed with thiazide diuretics; monitor for potassium depletion.
<b>Direct renin inhibitors (DRIs)</b> (aliskiren/Tektura)	
<b>Beta-blockers</b> (atenolol/Tenormin, pindolol/Visken, propranolol/Inderal, acebutolol/Sectral, bisoprolol/Zebeta, metoprolol/Lopressor, timolol/Blocadren, prazosin/Minipress)	Beta-blockers decrease the force and rate of heart contractions, thereby decreasing blood pressure. Dizziness and nausea are common side effects. Low-calorie, low-sodium diet may be useful. Metoprolol (Lopressor) should be taken with a low-calorie, low-sodium diet. Diarrhea, nausea, vomiting, or abdominal cramps may occur. Prazosin (Minipress) may cause nausea, weight gain, anorexia, diarrhea, or constipation. Use with low-sodium, low-calorie diet. Avoid natural licorice.
<b>Calcium channel blockers</b> (amlodipine/Norvasc; diltiazem/Cardizem; felodipine/Plendil)	
<b>Diuretics</b> Thiazides (furosemide/Lasix, indapamide/Lozol, chlorthalidone/Clorpres, hydrochlorothiazide/Hydrodiuril Microzide)	Thiazides deplete potassium and may require supplementation; diarrhea or GI bleeding can occur. Avoid natural licorice. Chlorthalidone may alter blood glucose or potassium levels; it may cause anorexia, vomiting, constipation, and nausea. In general, avoid use with fenugreek, yohimbe, and ginkgo.
<b>Diuretic-antihypertensives</b> (amiloride/Moduretic)	A low-calorie, low-sodium diet is important. Potassium loss is minimized. Avoid use with alcohol.
<b>Melatonin</b>	The nocturnal decline of blood pressure (BP) coincides with the elevation of melatonin, which may exert vasodilating and hypotensive effects; prolonged administration of melatonin may improve the day-night rhythm of BP.

canned foods. The normal adult needs only 1/2 teaspoon of sodium (200 mg) per day. Greater amounts of salt are required only in hot, humid conditions, during lactation, or with other salt-losing states. In such conditions, 2000 mg of salt is sufficient.

- Low fat dairy products have been linked with lower risk for hypertension; include often.
- Interesting food flavors are often hidden by salt. Discuss use of other seasonings and recipes. Monitor potassium in salt substitutes and medications to prevent hyperkalemia; read all labels carefully.
- Obesity leads to a proinflammatory and prothrombotic state that potentiates hypertension. Work on a weight loss program if needed.
- In children, an assessment of psychological and psychosocial factors that lead to obesity and hypertension should be undertaken (Kiessling et al, 2008).
- Increase physical activity when possible. Encourage use of pedometers for daily feedback.
- Omit or reduce alcohol intake severely, if needed.
- Assure adequate sleep; adverse changes to BP are noted with shorter duration.

#### **Patient Education—Foodborne Illness**

- Careful food handling will be important. Hand washing is key as well.

#### **For More Information**

- DASH Diet  
<http://www.nih.gov/news/pr/apr97/Dash.htm>
- JNC 7  
<http://www.nhlbi.nih.gov/guidelines/hypertension/jnc7full.htm>
- Malignant hypertension  
<http://www.nlm.nih.gov/medlineplus/ency/article/000491.htm>
- National High Blood Pressure Education Program  
<http://www.nhlbi.nih.gov/about/nhbpep/index.htm>  
<http://www.nhlbi.nih.gov/hbp/index.html>
- World Hypertension League  
<http://www.mco.edu/whl/>

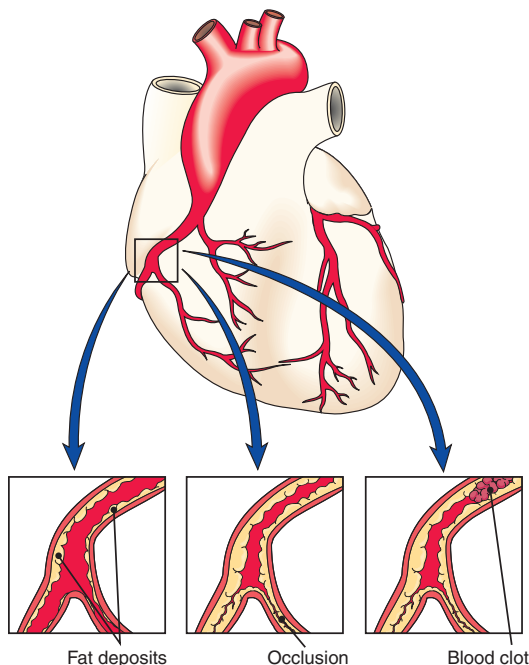
## HYPERTENSION—CITED REFERENCES

- ADA. American Dietetic Association Evidence Analysis Library. Accessed July 15, 2009, at [http://www.adaevidencelibrary.com/template.cfm?key=1947&cms\\_preview=1](http://www.adaevidencelibrary.com/template.cfm?key=1947&cms_preview=1).
- Appel LJ, et al. Dietary approaches to prevent and treat hypertension: a scientific statement from the American Heart Association. *Hypertension*. 47:296, 2006.
- Choi HK, et al. Intake of purine-rich foods, protein, and dairy products and relationship to serum levels of uric acid: the Third National Health and Nutrition Examination Survey. *Arthritis Rheum*. 52:283, 2005.
- Dall TM, et al. Predicted national productivity implications of calorie and sodium reductions in the American diet. *Am J Health Promot*. 23:423, 2009.
- Houben AJ, et al. Microvascular adaptation to changes in dietary sodium is disturbed in patients with essential hypertension. *J Hypertens*. 23:127, 2005.
- Houston MC. Nutraceuticals, vitamins, antioxidants, and minerals in the prevention and treatment of hypertension. *Prog Cardiovasc Dis*. 47:396, 2005.
- Ilhan N, et al. The 677 C/T MTHFR polymorphism is associated with essential hypertension, coronary artery disease, and higher homocysteine levels. *Arch Med Res*. 39:125, 2008.
- Judd SE, Tangpricha V. Vitamin D deficiency and risk for cardiovascular disease. *Am J Med Sci*. 338:40, 2009.
- Kiessling SG, et al. Obesity, hypertension, and mental health evaluation in adolescents: a comprehensive approach. *Int J Adolesc Med Health*. 20:5, 2008.

- Kozuma K, et al. Antihypertensive effect of green coffee bean extract on mildly hypertensive subjects. *Hypertens Res*. 28:711, 2005.
- Lee JH, et al. Vitamin D deficiency an important, common, and easily treatable cardiovascular risk factor? *J Am Coll Cardiol*. 52:1949, 2008.
- Levy D, et al. Genome-wide association study of blood pressure and hypertension [published online ahead of print May 10, 2009]. *Nat Genet*. 41:677, 2009.
- Mein CA, et al. Genetics of essential hypertension. *Hum Mol Genet*. 13:169, 2004.
- NHLBI. Seventh Report of the Joint National Committee on Prevention, Detection and Treatment of High Blood Pressure. Website Accessed July 16, 2009, at <http://www.nhlbi.nih.gov/guidelines/hypertension/jnc7full.htm>.
- Perrinjacquet-Moccetti T, et al. Food supplementation with an olive (*Olea europaea* L.) leaf extract reduces blood pressure in borderline hypertensive monozygotic twins. *Phytother Res*. 22:1239, 2008.
- Poduri A, et al. Effect of ACE inhibitors and beta-blockers on homocysteine levels in essential hypertension. *J Hum Hypertens*. 22:289, 2009.
- Streppel MT, et al. Dietary fiber and blood pressure: a meta-analysis of randomized placebo-controlled trials. *Arch Intern Med*. 165:150, 2005.
- Wang Y, et al. From the cover: whole-genome association study identifies STK39 as a hypertension susceptibility gene. *Proc Natl Acad Sci USA*. 106:226, 2009.
- Winkelmayer WC, et al. Habitual caffeine intake and the risk of hypertension in women. *JAMA*. 294:2330, 2005.

## MYOCARDIAL INFARCTION

## NUTRITIONAL ACUITY RANKING: LEVEL 3



Adapted from: Cohen BJ, Wood DL. *Memmler's The Human Body in Health and Disease*. 9th ed. Philadelphia: Lippincott Williams & Wilkins, 2000.



## DEFINITIONS AND BACKGROUND

**MI** is necrosis in the heart muscle caused by prolonged inadequate blood supply or oxygen deficit. A **coronary occlusion** (heart attack) is the closing of a coronary artery feeding heart muscle by fatty deposits or a blood clot; it manifests with heavy squeezing pain radiating to the jaw or back, nausea, vomiting, diaphoresis, anxiety, and weakness. Abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, inadequate consumption of fruits and vegetables, alcohol consumption, and lack of regular physical activity account for most of the risk factors for MI worldwide; Table 6-14 provides a complete list.

Women have symptoms of an MI that differ from men. Prodromal symptoms include unusual fatigue, shortness of breath, and pain in the shoulder blade/upper back; medical practitioners must develop an awareness of and a more comprehensive approach to treating women.

Stages after an MI include critical (first 48 hours), acute (3–14 days), and convalescent (15–90 days). Treatment with Chol-lowering medications and antioxidants may decrease MI and may reduce adverse coronary events. Table 6-15 lists potential complications after an MI. CVDs cause 12 million deaths worldwide, and MI is a significant problem.

**TABLE 6-14 Risk Factors for Myocardial Infarction**

- Family history of heart disease
- Patient history of heart disease
- Diabetes or elevated blood glucose, even in nondiabetics
- Hypertension
- Advanced age
- High lipoprotein (a) lipids
- African American ethnicity
- Stress, smoking, sedentary lifestyle, compulsive personality
- Poor diet (high sodium, high fat, high intake of alcohol; low intake of B-complex vitamins, calcium, magnesium, and potassium; low intake of fruits and vegetables)
- Obesity

Fruit and vegetable intake should be encouraged. Platelet aggregation is central in acute coronary syndromes, including MI and unstable angina. Effects of flavonoids on endothelial and platelet function might explain their protective benefits on cardiac risk (Vita, 2005). Studies relate wine/resveratrol with reduction in myocardial damage.

An **arrhythmia** is a variation from normal heartbeat rhythm. Among its many forms is a slowing of the heartbeat to less than 60 beats per minute (bradycardia), a speedup to more than 100 beats per minute (tachycardia), and premature or “skipped” beats. Post-MI patients will need to monitor themselves for arrhythmias.

On the basis of the ECG, a distinction is made between ST-elevation (where thrombolysis, PCI, angioplasty or stent insertion are used), versus non-ST elevation where medications suffice. In some cases, coronary artery bypass surgery (CABG) is an option.

**TABLE 6-15 Complications After Myocardial Infarction**

- Arrhythmias with risk of sudden death
- Cardiogenic shock
- Cardiac tamponade
- Cholesterol emboli due to cardiac catheterization or during CABG
- Heart failure with pulmonary edema
- Left ventricular free wall rupture
- Pericarditis
- Re-infarction
- Renal failure
- Splenic infarction with fever, tachycardia, left upper quadrant abdominal pain
- Thrombosis or CVA with ischemic bowel or renal infarct
- Valve insufficiency
- Ventricular septal defect

Sources: O’Keefe JH Jr, et al. Thromboembolic splenic infarction. *Mayo Clin Proc.* 61:967, 1986; Puletti M, et al. Incidence of systemic thromboembolic lesions in acute myocardial infarction. *Clin Cardiol.* 9:331, 1986; Prieto A, et al. Nonarrhythmic complications of acute MI. *Emerg Med Clin North Am.* 19:397, 2001.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Genetic factors and interactions between multiple genes and environmental factors indicate an association with MI or CHD of polymorphisms in MTHFR, LPL, APOE, and those in LTA and at chromosomal region 9p21.3 by genome-wide scans (Yamada et al, 2008).

Clinical/History	BP	LDH
Height	I & O	(increased)
Weight	Radionucleotide	WBC count
BMI	imaging	(increased)
Waist circum-	Echocardiogra-	Na <sup>+</sup> , K <sup>+</sup>
ference	phy	Ca <sup>++</sup> , Mg <sup>++</sup>
Diet history	ECG	pCO <sub>2</sub> , pO <sub>2</sub>
Temperature	Chest X-ray	PT or INR
(low grade		Chol—total,
fever?)	<b>Lab Work</b>	HDL, LDL
Pulse (NL =	Creatine kinase-	Trig (often
60–100	MB (CK-MB)	increased)
beats/min)	Troponin I	BUN, Creat,
Tightness in the	(TnI) or tro-	GFR
chest	ponin T	Cystatin C (ele-
Nausea, anxiety	(TnT) levels	vated with
Syncope	CRP	low GFR)
Wheezing,	Gluc (elevated	tHcy
diaphoresis	levels will	Serum folate
Chest pain, radi-	increase risk)	and B <sub>12</sub>
ating	AST	H & H
Severe fatigue	Serum Cu	Serum Fe,
Migraine	(increased)	ferritin
headaches?		

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Myocardial Infarction (MI)

**Assessment:** Diet history and food records, lipid profile, review of current and previous medications used for heart disease.

**Nutrition Diagnosis (PES):** Inappropriate intake of types of food fats due to nutrition-related knowledge deficit as evidenced by LDL Chol of 155 mg/dL and use of large meat portions at lunch and dinner meals.

**Intervention:** Easy meal preparation and recipe adaptations, shopping and dining away from home tips, one-on-one counseling and goal setting for making changes in choices of food fats.

**Monitoring and Evaluation:** Lipid profile, food diary and records, goal achievement.



## INTERVENTION



## OBJECTIVES

- Promote rest to reduce heart strain. Avoid the distention of heavy meals.
- Prevent arrhythmias by serving food at body temperature.
- Avoid both constipation and flatulence.
- Avoid excessive heart stimulation from caffeine. Energy drinks may contain 50–500 mg of caffeine.
- Reduce elevated levels of lipids: keep Chol below 200 mg/dL, TGs below 200 mg/dL, HDL between 40 and 60 mg/dL, and LDL between 100 and 129 mg/dL.
- Decrease energy required to chew, prepare meals, perform activities of daily living during convalescence.
- Identify modifiable risk factors and complications; reduce risks when possible (see Tables 6-14 and 6-15). For example, lose weight if obese.
- Consume more fish and a diet rich in ALA to reduce the risk of fatal heart attack.



## FOOD AND NUTRITION

- Initially, use liquids to promote rest while reducing the dangers of aspiration or vomiting. Reduce fluid and caffeine intake to that recommended by the physician. As treatment progresses, diet should include soft, easily digested foods that are low in saturated fats, Chol, and gas-forming foods.
- Limit diet to 2 g of sodium, or remove salt from the table. Schedule three to six small meals daily.
- If needed, use an energy-controlled diet to reduce the heart's workload.
- The DASH diet and Mediterranean diet are useful. Increase intake of fish, whole grains, and olive oil. Onions, tea, apples, grape juice, and pomegranate contain flavonoids and should be used often. Red wine is recommended, if approved by the physician.
- Adequate calcium, magnesium and potassium will be needed, but not in excess. Consuming micronutrients at levels exceeding those provided by a dietary pattern consistent with American Heart Association (AHA) Dietary Guidelines will not confer additional CVD risk reduction (Kris-Etherton et al, 2004).
- Decrease intake of whole-milk products, red meats, visible fat on meat/poultry, and commercial baked goods. Limit egg yolks to four to five times weekly if lipids are elevated.
- Increase food sources of vitamins E and K, folic acid, and vitamins B<sub>6</sub> and B<sub>12</sub>.
- Fiber is especially important; choose vegetables, fruits, and cereal grains.
- Include judicious use of nuts, such as walnuts, almonds, macadamias, pecans, and pistachios. Walnuts contain ALA; almonds are a good source of vitamin E. Nuts also contain flavonoids, phenols, sterols, saponins, elegendic acid, folic acid, magnesium, copper, potassium, and fiber.
- Pistachios, sunflower kernels, sesame seeds, and wheat germ are highest in phytosterols; use often.

## Common Drugs Used and Potential Side Effects

- Appropriate drugs are provided according to needs established by the profile (elevated BP or lipids). Review specific drugs given to the patient and treat accordingly. Nitrates, beta-blockers, calcium channel blockers, statins, and related drugs are given according to the cause and risk factors for the patient.
- Anticoagulants such as warfarin (Coumadin) may be given where bleeding tendencies are not present. Limit to one vitamin K-rich food per day (green leafy vegetables such as kale, broccoli, spinach, and turnip greens). Avoid taking with dong quai, fenugreek, feverfew, excessive garlic, ginger, ginkgo, and ginseng.
- Aspirin is often recommended later to prevent recurrent MIs. Watch for GI bleeding or other side effects such as occult blood loss.
- Mexiletine (Mexitil) and propafenone (Rythmol) are used to treat arrhythmias. Nausea, vomiting, or constipation may occur. Procainamide (Procan) may result in a bitter taste, nausea, anorexia, or diarrhea.
- Morphine is used for relief of pain but should be given in minimal amounts to prevent hypotension and other side effects.

## Herbs, Botanicals, and Supplements

- Dietary supplementation with omega-3 fatty acids should be considered in the secondary prevention of cardiovascular events (Marik and Valon, 2009). Study of the associations between EPA and DHA intake and disease requires a valid biomarker of dietary intake; RBC delta(15)N as a biomarker of EPA and DHA intake is rapid and inexpensive (O'Brien et al, 2009).
- At this time, the scientific data do not justify the use of antioxidant vitamin supplements.
- See Table 6-3 for more guidance on herbs and botanical products.



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Position patient and arrange utensils to avoid or lessen fatigue. Encourage relaxation, especially at mealtimes.
- If needed, use a weight-control diet.
- Discuss roles of fats, Chol, sodium, potassium, calcium, magnesium, and fiber in the diet. Encourage use of the DASH or Mediterranean diets (see Table 6-4). A vegetarian diet may also be beneficial (Craig et al, 2009).
- Monitor for changes in renal status. A GFR of <60 mL/min may indicate high risk for cardiac death in women (Kurth et al, 2009).
- Avoid excesses of carbohydrate and alcohol, especially with diabetes or elevated BP.
- Individuals continue to have multiple risk factors for CAD that place them at high risk for future events. Discuss convalescence and prevention of HF.
- The patient should stop smoking, follow the recommended diet, and manage other risk factors. A positive attitude toward the modified diet is essential for changing food behaviors.

- The reduction of exposure to second-hand smoke is also beneficial (Glantz et al, 2008; Pell et al, 2008).
- Management of anxiety is an important feature for recovery (Kuhl et al, 2009).
- Discuss a gradual increase in activity. Cardiac rehabilitation programs are very helpful (Lisspers et al, 2005). Aerobic physical activities, such as exercise or walking at work, seem to reduce the risk of MI, whereas anaerobic activities, such as heavy lifting, may increase risk of MI (Fransson et al, 2004).
- A comprehensive management plan uses the “ABCDE” approach: “A” for antiplatelet therapy, anticoagulation, ACE inhibition, and angiotensin receptor blockade; “B” for beta-blockade and BP control; “C” for Cholesterol treatment and cigarette smoking cessation; “D” for diabetes management and diet; and “E” for exercise (Gluckman et al, 2005).

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- Heart Attack  
<http://www.heartpoint.com/mi.html>
- JAMA Patient Page  
<http://jama.ama-assn.org/cgi/reprint/299/4/476.pdf>
- Medicine Net  
[http://www.medicinenet.com/heart\\_attack/article.htm](http://www.medicinenet.com/heart_attack/article.htm)

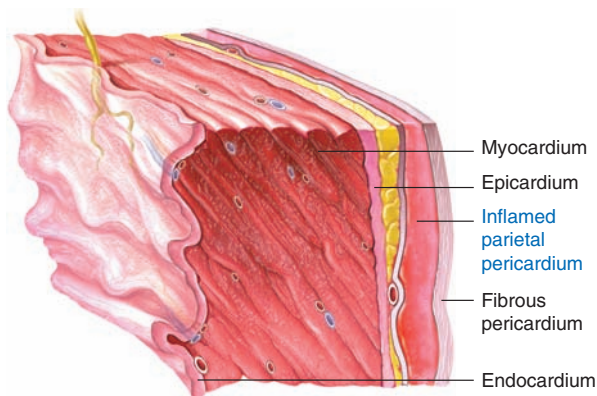
- Myocardial Infarction  
<http://www-medlib.med.utah.edu/WebPath/TUTORIAL/MYOCARD/MYOCARD.html>

### MYOCARDIAL INFARCTION—CITED REFERENCES

- Craig WJ, et al. Position of the American Dietetic Association: vegetarian diets. *J Am Diet Assoc.* 109:1266, 2009.
- Fransson E, et al. The risk of acute myocardial infarction: interactions of types of physical activity. *Epidemiology.* 15:573, 2004.
- Glantz S. Meta-analysis of the effects of smoke free laws on acute myocardial infarction: an update. *Prev Med.* 47:452, 2008.
- Gluckman TJ, et al. A simplified approach to the management of non-ST-segment elevation acute coronary syndromes. *JAMA.* 293:349, 2005.
- Kris-Etherton PM, et al. Antioxidant vitamin supplements and cardiovascular disease. *Circulation.* 110:637, 2004.
- Kuhl EA, et al. Relation of anxiety and adherence to risk-reducing recommendations following myocardial infarction. *Am J Cardiol.* 103:1629, 2009.
- Kurth T, et al. Kidney function and risk of cardiovascular disease and mortality in women: a prospective cohort study. *BMJ.* 338:2392, 2009.
- Lisspers J, et al. Long-term effects of lifestyle behavior change in coronary artery disease: effects on recurrent coronary events after percutaneous coronary intervention. *Health Psychol.* 24:41, 2005.
- Marik PE, Varon J. Omega-3 dietary supplements and the risk of cardiovascular events: a systematic review. *Clin Cardiol.* 32:365, 2009.
- O'Brien DM, et al. Red blood cell delta15 N: a novel biomarker of dietary eicosapentaenoic acid and docosahexaenoic acid intake. *Am J Clin Nutr.* 89:913, 2009.
- Pell JP, et al. Smoke-free legislation and hospitalizations for acute coronary syndrome. *N Engl J Med.* 359:482, 2008.
- Vita JA. Polyphenols and cardiovascular disease: effects on endothelial and platelet function. *J Clin Nutr.* 81:292S, 2005.
- Yamada Y, et al. Molecular genetics of myocardial infarction. *Genomic Med.* 2:7, 2008.

## PERICARDITIS AND CARDIAC TAMPONADE

### NUTRITIONAL ACUITY RANKING: LEVEL 2



Asset provided by Anatomical Chart Co.



### DEFINITIONS AND BACKGROUND

Pericarditis is inflammation of the pericardium due to human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), MI, rheumatic diseases, radiation

treatment, viral infection, trauma, neoplasm, chronic renal failure, or lupus. Bacterial pericarditis occurs by direct infection during trauma, thoracic surgery or catheter drainage, spread from *Staphylococcus*, *Streptococcus*, *Haemophilus*, and *Mycobacterium tuberculosis* (Pankuweit et al, 2005).

Severe substernal chest pain, dyspnea, shortness of breath, fever, chills, diaphoresis, nausea, fatigue, and anxiety are common in the acute stage. The chronic stage of pericarditis often results from tuberculosis and may involve ascites, edema of the extremities, HF, shrinkage of the pericardium, shortness of breath, coughing, fatigue, ascites, and leg edema.

Although pericarditis usually is not a life-threatening condition, other life-threatening conditions may cause chest pain and should be ruled out. These include MI, dissection of the aorta, pulmonary embolus, collapsed lung, and perforation or rupture of parts of the esophagus or stomach. Control of symptoms through diuretics is the usual treatment.

The most serious complication is **cardiac tamponade**, with accumulation of fluid or blood within the pericardial sac. If uncontrolled, this condition may lead to HF, arrest, or

shock (Meltzer and Karia, 2005). Decreased heart sounds, distended neck veins with inspiratory rise in venous pressure (Kussmaul's sign), decreased BP, and abdominal pain may occur. In children, cases of cardiac tamponade have occurred as a complication of malignancies, cardiac surgery, trauma, infections, central venous catheter placement, rheumatologic and autoimmune diseases (Cousineau and Savitsky, 2005). Pericardial effusions causing tamponade are rare in patients with lupus, but high-dose corticosteroids are often needed (Rosenbaum et al, 2009).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** A deficiency in the interleukin-1 receptor activated kinase 4 (IRAK-4) has recently been associated with severe recurrent, Gram-positive bacterial infections and related heart infections (Comeau et al, 2008). In addition, Familial Mediterranean fever is an autosomal recessive disease that presents with recurrent Peritonitis. More studies of the genetics of pericardial diseases are needed.

<b>Clinical/History</b>	Cyanosis?	Alb,
Height	Cough	transferrin
Weight	Dysphagia	BP
Weight changes (loss?)	Pericardial friction rub	BUN, Creat, GFR
BMI	ECG	H & H
Waist circumference	Cardiac catheterization	Serum Fe
Diet history	Magnetic resonance imaging (MRI)	Transferrin
Low-grade intermittent fever?	Computed tomography (CT)	Na <sup>+</sup> , K <sup>+</sup>
High fever?		Ca <sup>++</sup> , Mg <sup>++</sup>
I & O		WBC
Ascites or hepatomegaly?		tHcy
Tachypnea, dyspnea		Serum folate and B <sub>12</sub>
	<b>Lab Work</b>	
	CRP	
	Gluc	

## INTERVENTION



### OBJECTIVES

- It is critical for anyone who experiences chest pain to seek immediate medical attention to determine the cause and receive prompt, appropriate treatment to improve cardiac functioning.
- Maintain bed rest during acute stages.
- Prevent sepsis, HF, and shock, especially if cardiac tamponade occurs.
- Decrease fever and inflammation, which may last 10–14 days in the acute form.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Malnutrition

**Assessment Data:** Diet history and food records indicating intake of about 60% of usual intake; dyspnea, anorexia, nausea, recent fevers up to 103°F. BMI 17; usual BMI 20.

**Nutrition Diagnosis (PES):** Malnutrition related to pericarditis and chronically poor intake as evidenced by BMI of 17 and weight loss over the past year of 15 lb.

**Intervention:** Add protein powder to mixed dishes that are served. Educate about nutrient density and ways to add calories and protein. Review medications and suggest ways to increase appetite; discuss with medical team.

**Monitoring and Evaluation:** Improved intake and appetite; BMI closer to usual. Fewer complaints of fever, nausea, anorexia.

- Reduce nausea and anorexia.
- Prevent fluid overload if CPN is administered.



### FOOD AND NUTRITION

- Maintain an adequate diet as needed for any underlying conditions; increase protein and calories if tolerated and if needed to prevent loss of LBM.
- Alter sodium and fluids intake if necessary.
- Small, frequent feedings to reduce nausea may be indicated.
- Monitor diet and supplements adequately. Thiamin for the heart muscle and potassium may be especially necessary; vitamins B<sub>6</sub> and B<sub>12</sub> and folic acid may be needed if tHcy levels are elevated. It may also be prudent to increase vitamin E levels.
- The DASH diet, TLC diet, and Mediterranean diet are good choices.

### Common Drugs Used and Potential Side Effects

- Analgesics or nonsteroidal anti-inflammatory drugs (NSAIDs) may be used to relieve pain. Monitor for specific side effects.
- Antibiotics are needed for bacterial infections. Intravenous antibacterial therapy, such as vancomycin, ceftriaxone, or ciprofloxacin, is used in purulent pericarditis (Pankuweit et al, 2005). Monitor for side effects.
- Diuretics may be used. If a potassium-depleting diuretic is chosen, monitor serum potassium levels closely and manage dietary changes if needed.
- Treatment of tuberculous pericarditis includes isoniazid, rifampin, pyrazinamide, and ethambutol (Pankuweit et al, 2005). Prednisone is given and then progressively reduced in 6–8 weeks; GI distress, hyperglycemia, and calcium and nitrogen depletion may result.
- Some medications can trigger an immune response that causes pericarditis. These medications include isoniazid (Nydrazid), hydralazine (Apresoline), penicillin, antiarrhythmic agents such as procainamide (Procanbid or Pronestyl), and seizure medications such as phenytoin (Dilantin).

## Herbs, Botanicals, and Supplements

- See Table 6-3 for guidance.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the importance of avoiding fatigue.
- The patient should plan rest periods before and after activities and meals.
- Highlight the importance of nutrition in immunocompetence.
- Discuss any drug–nutrient interactions that are possible according to the treatment plan.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- Cardiac Tamponade  
<http://www.nlm.nih.gov/medlineplus/ency/article/000194.htm>

- Medline—Pericarditis  
<http://www.nlm.nih.gov/medlineplus/ency/article/000182.htm>
- Merck Manual—Pericarditis  
<http://www.merck.com/mmpe/sec07/ch078/ch078a.html>
- Mount Sinai  
<http://www.mssm.edu/cvi/pericarditis.shtml>
- Pericarditis  
<http://cardiologychannel.com/pericarditis/>
- Pericarditis and Cardiac Tamponade  
<http://www.emedicine.com/emerg/topic412.htm>

## PERICARDITIS AND CARDIAC TAMPONADE—CITED REFERENCES

- Comeau JL, et al. Staphylococcal pericarditis, and liver and paratracheal abscesses as presentations in two new cases of interleukin-1 receptor associated kinase 4 deficiency. *Pediatr Infect Dis J*. 27:170, 2008.
- Cousineau A, Savitsky E. Cardiac tamponade presenting as an apparent life-threatening event. *Pediatr Emerg Care*. 21:104, 2005.
- Meltzer H, Karia VG. Cardiac tamponade. *Catheter Cardiovasc Interv*. 64:245, 2005.
- Pankuweit S, et al. Bacterial pericarditis: diagnosis and management. *Am J Cardiovasc Drugs*. 5:103, 2005.
- Rosenbaum E, et al. The spectrum of clinical manifestations, outcome and treatment of pericardial tamponade in patients with systemic lupus erythematosus: a retrospective study and literature review. *Lupus*. 18:608, 2009.

# PERIPHERAL ARTERY DISEASE

## NUTRITIONAL ACUITY RANKING: LEVEL 2



### DEFINITIONS AND BACKGROUND

**Peripheral vascular disease (PVD)** can affect the arteries, the veins, or the lymph vessels. The most common and important type of PVD is **peripheral artery disease (PAD)**. PAD, defined as atherosclerosis in the lower extremities, affects nearly 8.5 million people in the United States (Dobesh et al, 2009). Prevalence increases dramatically with age; by age 70, about 20% of the population has PAD. PAD disproportionately affects Mexican Americans and nonhispanic blacks. People with PAD face a six to seven times higher risk of heart attack or stroke. Occlusion of an artery occurs from a clot or by plaque buildup in the extremities, such as the hands and feet numbness, tingling in lower extremities, pain, difficult ambulation, gangrene with potential amputation. See Table 6-16.

Causes of PAD include heavy smoking, arterial embolism, obesity, diabetes mellitus, renal insufficiency, poor circulation, atherosclerosis, or exposure to heavy metals. Urinary cadmium, tungsten, and possibly antimony have been associated with PAD (Navas-Acien et al, 2005). Modifiable risk factors such as smoking, dyslipidemia, hypertension, and diabetes should be addressed (Aronow, 2005; Nijm et al, 2005).

Elevated levels of fibrinogen and CRP may indicate that there is inflammation. PAD is a red flag that the same

process may be going on elsewhere; PAD is often associated with other life-threatening vascular diseases. Exercise is the first-line treatment for intermittent claudication with symptoms of aching pain, numbness, weakness, or fatigue (Dobesh et al, 2009).

**TABLE 6-16 Sites Where Peripheral Arterial Disease Produces Symptoms**

Arteries supplying blood to the brain	Stroke is a serious complication.
Arteries supplying blood to the kidneys	Renal artery stenosis is one of the causes of high blood pressure and renal failure.
Arteries supplying blood to the legs	Diminished ability to walk can occur; worst-case scenario leads to amputation.
Arteries supplying blood to the intestines	Mesenteric arterial disease is less frequent but can cause severe pain, weight loss, and death from intestinal gangrene.



Endothelial dysfunction may reduce functional capacity via attenuations in peripheral blood flow; dietary docosahexaenoic acid (DHA) may improve this dysfunction (Stebbens et al, 2008). When National Health and Nutrition Examination Survey (NHANES) data were evaluated to determine specific nutrients that are associated with PAD, it was found that higher consumption of antioxidants (vitamin A, C, and E), vitamin B<sub>6</sub>, fiber, folate, and omega-3 fatty acids have a significant protective effect (Lane et al, 2008). Because hypovitaminosis D is more common in blacks than Hispanics or whites, check serum levels of serum 25-hydroxyvitamin D (25[OH]D) and supplement as needed (Reis et al, 2008). Dietary supplementation may protect against PAD (Lane et al, 2008).

Angioplasty may be needed to clear obstruction, relieve symptoms, heal ulcers, and prevent amputation. Indications for lower extremity angioplasty, preferably with stenting, or bypass surgery are (1) incapacitating claudication in persons that interferes with work or lifestyle; (2) limb salvage in persons with limb-threatening ischemia as manifested by rest pain, nonhealing ulcers, and/or infection or gangrene; and (3) vasculogenic impotence (Aronow, 2005). If the limb cannot be salvaged, amputation would be needed.

**Buerger's disease** is the obstruction of small- and medium-sized arteries by inflammation triggered by smoking and is usually found among men aged 20–40 years. Skin ulcers or gangrene may result if smoking is not discontinued. Walking is beneficial, unless the person has gangrene, sores, or pain at rest.

**Raynaud's syndrome** allows small arterioles in the fingers and toes to go into spasm, and the skin turns pale or patchy red to blue. Sometimes the underlying cause is not known. Approximately 60–90% of cases occur in young women. Scleroderma, rheumatoid arthritis, atherosclerosis, nerve disorders, hypothyroidism, injury, and reactions to certain drugs are causes. Some people also have migraine headaches and pulmonary hypertension. These individuals must protect their extremities from the cold or take mild sedatives for pain.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Adipokine angiopoietin-like 4 gene (ANGPTL4[E40K]) variant appears to confer reduced genetic risk for CHD and other conditions, including PAD (Folsom et al, 2008). These common alleles are correlated with low TG and high HDL-C (Nettleton et al, 2009). Dietary fat intake modifies relations between HDL-C and polymorphisms in hepatic lipase (LIPC-514 C→T), cholesteryl ester transfer protein (CETP TaqIB), and lipoprotein lipase (LPL S447X) genes (Nettleton et al, 2007).

<b>Clinical/History</b>	BMI	BP
Height	Waist–hip ratio	Smoking history
Weight	Diet history	

Ankle brachial index (ABI) test <0.9	<b>Lab Work</b>	Gluc
Exercise tolerance, stress testing	CRP	Serum insulin
Ulcerations	Chol—total, HDL, LDL	Na <sup>+</sup> , K <sup>+</sup>
Gangrene	Trig	Ca <sup>++</sup> , Mg <sup>++</sup>
ECG	BUN, Creat	Serum 25-hydroxyvitamin D (25[OH]D)
Cardiac catheterization	Uric acid	
MRI or CT scan	tHcy	
	Serum folate and B <sub>12</sub>	

## INTERVENTION



### OBJECTIVES

- Treatment goals are aimed at decreasing cardiovascular risk, as well as improving quality of life (Dobesh et al, 2009). Reduce complications such as angina, HF, heart attack, stroke, renal failure, ulcerative disease, gangrene of lower extremities or toes.
- Prevent sepsis, pressure ulcers, or the need for amputation.
- Correct high levels of tHcy.
- Attain desired body weight if obese.
- Reduce the inflammatory process, as possible. CRP and other inflammatory markers offer risk prediction (Haugen et al, 2007).
- Modify carbohydrate and fat intakes (Nettleton, 2009).
- Correct hypovitaminosis D if needed.



### FOOD AND NUTRITION

- If patient is obese, use a low-calorie diet with high fiber.
- Diet should provide adequate protein intake for wound healing with ulcers or surgery.
- Vitamin E in almonds, filberts, avocados, sunflower seeds, vegetable oils, margarine, mayonnaise, and wheat germ are the best sources. Pistachios, sunflower kernels, sesame seeds, and wheat germ are high in phytosterols and should be used frequently.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Inadequate Intake of Fatty Acids

**Assessment:** Diet history and food records, BP records. Labs with elevated LDL Chol.

**Nutrition Diagnosis (PES):** Inadequate intake of omega-3 fatty acids related to poor diet and peripheral circulation as evidenced by elevated LDL Chol intake.

**Intervention:** Education about the role of omega-3 fatty acids in cardiovascular health and reduction of inflammation.

**Monitoring and Evaluation:** Improved dietary records showing use of omega-3 fatty acids from diet and supplements.

- Increase folic acid, riboflavin, and vitamins B<sub>6</sub> and B<sub>12</sub> if serum tHcy levels are elevated.
- The DASH, TLC, and Mediterranean diet patterns are useful.
- Olive oil consumption along with a dietary supplement of fish oil may be helpful in the management of PVD. Avoid use of butter and saturated fats.
- Correct vitamin D deficiency as needed.

### Common Drugs Used and Potential Side Effects

- Antiplatelet drugs such as aspirin or clopidogrel may be helpful.
- Antibiotics may be used to control infections.
- ACE inhibitors are an important risk-reduction therapy for patients with PAD. Beta-blockers should be given if CAD is present (Aronow, 2005).
- Anticoagulants such as warfarin may be used. Use no more than one high-vitamin K food source daily. Avoid taking with dong quai, fenugreek, feverfew, excessive garlic, ginger, ginkgo, or ginseng because of their effects.
- Isoxsuprine (Vasodilan) may be used to dilate the vessels. If niacin is used as a vasodilator, do not use large doses without monitoring by a physician.
- Pentoxifylline (Trental) improves blood flow. GI distress, nausea, and anorexia may occur; take with meals.
- Statins reduce the incidence of intermittent claudication and improve exercise duration (Aronow, 2005).

### Herbs, Botanicals, and Supplements

- Coenzyme Q10, ginger, purslane, and ginkgo have been recommended for PAD, but no clinical trials have proven efficacy. Use a vitamin D supplement if serum levels are low.
- See Table 6-3 for more information.



#### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Emphasize the importance of weight control and exercise.
- Reduce excess alcohol consumption if TGs are elevated.
- Fish and meatless meals should be used three to four times weekly.

- Hyperbaric oxygen treatments may be needed to heal lesions. Here, oxygen permeates the flesh, and anaerobic bacteria cannot survive.
- Encourage a smoking cessation program.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- American Heart Association  
<http://www.americanheart.org/presenter.jhtml?identifier=4692>
- Amputee Coalition  
<http://www.amputee-coalition.org/>
- Peripheral Artery Disease  
[http://www.nhlbi.nih.gov/health/dci/Diseases/pad/pad\\_what.html](http://www.nhlbi.nih.gov/health/dci/Diseases/pad/pad_what.html)
- Vascular Web  
[http://www.vascularweb.org/patients/NorthPoint/Leg\\_Artery\\_Disease.html](http://www.vascularweb.org/patients/NorthPoint/Leg_Artery_Disease.html)

### PERIPHERAL ARTERY DISEASE—CITED REFERENCES

- Aronow WS. Management of peripheral arterial disease. *Cardiol Rev.* 13:61, 2005.
- Dobesh PP, et al. Pharmacologic therapy for intermittent claudication. *Pharmacotherapy.* 29:526, 2009.
- Folsom AR, et al. Variation in ANGPTL4 and risk of coronary heart disease: the Atherosclerosis Risk in Communities Study. *Metabolism.* 57:1591, 2008.
- Haugen S, et al. Risk assessment in the patient with established peripheral arterial disease. *Vasc Med.* 12:343, 2007.
- Lane JS, et al. Nutrition impacts the prevalence of peripheral arterial disease in the United States. *J Vasc Surg.* 48:897, 2008.
- Navas-Acien A, et al. Metals in urine and peripheral arterial disease. *Environ Health Perspect.* 113:164, 2005.
- Nettleton JA, et al. Associations between HDL-cholesterol and polymorphisms in hepatic lipase and lipoprotein lipase genes are modified by dietary fat intake in African American and White adults. *Atherosclerosis.* 194:e131, 2007.
- Nettleton JA, et al. Carbohydrate intake modifies associations between ANGPTL4[E40 K] genotype and HDL-cholesterol concentrations in White men from the Atherosclerosis Risk in Communities (ARIC) study. *Atherosclerosis.* 203:214, 2009.
- Nijm J, et al. Circulating levels of proinflammatory cytokines and neutrophil-platelet aggregates in patients with coronary artery disease. *Am J Cardiol.* 95:452, 2005.
- Reis JP, et al. Differences in vitamin D status as a possible contributor to the racial disparity in peripheral arterial disease. *Am J Clin Nutr.* 88:1469, 2008.
- Stebbins CL, et al. Effects of dietary docosahexaenoic acid (DHA) on eNOS in human coronary artery endothelial cells. *J Cardiovasc Pharmacol Ther.* 13:261, 2008.

## THROMBOPHLEBITIS

### NUTRITIONAL ACUITY RANKING: LEVEL 1



#### DEFINITIONS AND BACKGROUND

Phlebitis is inflammation of a vein that usually is caused by infection or injury. Blood flow may be disturbed, with blood clots (thrombi) adhering to the wall of the inflamed vein. This condition usually occurs in leg veins, especially in vari-

cose veins. Blood clots in the thigh veins are usually more serious than those in the lower leg and are usually deep vein thromboses (DVTs).

Fatty acids have been implicated in the etiology of thrombophlebitis, but there is no clearly demonstrated mechanism. Blood tHcy levels are an important, independent,

**TABLE 6-17 Causes of Thrombophlebitis**

- Age: Over age 60 is more common, but it can occur any time
- Cancer and its treatment; especially in recently diagnosed patients, patients with cancer that has spread to distant sites (metastases), and those with certain genetic mutations (Blom et al, 2005)
- Central venous catheters
- Inherited conditions that cause increased risk for clotting; factor V Leiden, the genetic defect underlying resistance to activated protein C, is one factor that causes inherited thrombophilia
- Low blood flow in a deep vein due to injury, surgery, or immobilization
- Obesity or overweight
- Oral contraceptives or hormone replacement therapy; both estrogen and progesterone affect the condition
- Pregnancy and postpartum, especially the first 6 weeks after giving birth
- Sitting for a long period of time (long trips in a car or airplane)
- Varicose veins, which are enlarged, twisted, painful superficial veins resulting from poorly functioning valves, usually in the legs. They affect women more commonly than men.

and frequent risk factor for venous thrombosis. The VITRO (Vitamins and ThROMbosis) trial was the first multicenter, randomized, double-blind and placebo-controlled study to evaluate the effect of tHcy-lowering therapy by means of 5 mg folic acid, 0.4 mg vitamin B<sub>12</sub> and 50 mg vitamin B<sub>6</sub>; it did not prevent recurrences (den Heijer et al, 2007).

Venous thromboembolism (VTE) is a major cause of morbidity and mortality worldwide, and the annual incidence of VTE is 1 per 1000 (Bramlage et al, 2005). Table 6-17 lists common causes of thrombophlebitis.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Venous thrombosis is one of the leading causes of maternal morbidity and mortality; it is highly increased in carriers of factor V Leiden or the prothrombin 20210 A mutation (Pomp et al, 2008). It has also been noted with folate MTHFR alleles.

Clinical/History	BP	Duplex ultrasound
Height	Pain, redness,	Doppler study
Weight	warmth, tenderness, itching	Ankle BP measurements
BMI	Hard or cord-like swelling	
Waist-hip ratio	along the affected vein	
Diet history		
Recent weight changes		
Temperature		
I & O		

**Lab Work**  
CRP  
Factor V Leiden kit

Alb, transferrin Chol—total, HDL, LDL Trig BUN, Creat Uric acid WBC	H & H Gluc TLC Na <sup>+</sup> , K <sup>+</sup> Ca <sup>++</sup> , Mg <sup>++</sup> tHcy Serum folate and B <sub>12</sub>	Sex hormone-binding globulin (SHBG)—for oral contraceptive users
--	--	--

## INTERVENTION



### OBJECTIVES

- Stop the clot from getting bigger.
- Reduce inflammation and swelling.
- Prevent septicemia, pulmonary embolism (with chest pain and shortness of breath), and related complications.
- Lower elevated plasma tHcy, which increases the risk of VTE.



## FOOD AND NUTRITION

- Weight control diet may be needed if the patient is obese. The DASH, TLC, and Mediterranean diets may be beneficial.
- Sodium restriction may be beneficial for persons with a generally high salt or sodium intake. Monitor carefully.
- For general cardiovascular health, adequate vitamins B<sub>6</sub> and B<sub>12</sub> and folic acid intakes should be included. Intake of B vitamins through diet, supplementation, and fortified foods effectively reduces tHcy concentration and thus may reduce the risk.
- Thiamin and vitamin E are also beneficial for heart health at levels meeting but not exceeding daily requirements.
- Encourage use of omega-3 fatty acids from fish and other foods.
- Pistachios, sunflower kernels, sesame seeds, and wheat germ are high in phytosterols; use often.
- Flavonoids (including tannins, quercetin, and phenols) in grapes, strawberries, blueberries, apples, kale, broccoli, onions, garlic tea, beer, and red wine may reduce platelet activity and prevent clots.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Assessment:** Diet history and food records, BP records.

**Nutrition Diagnosis (PES):** Inadequate intake of types of fat

**Intervention:** Nutrition education about food and supplemental sources of EPA and DHA.

**Monitoring and Evaluation:** No clotting problems. BP improved or controlled.

- Foods that contain vitamin K can change how well warfarin (Coumadin) will work. Eat a balanced diet that does not vary in usual content of these vitamin K-rich foods so that the medication can be regulated.

### Common Drugs Used and Potential Side Effects

- The anticoagulant warfarin (Coumadin) may be used, with side effects that alter use of vitamin K. Monitor intake carefully. Avoid supplements that are high in vitamins E, C, or A during use. Avoid taking with dong quai, fenugreek, feverfew, excessive garlic, ginger, ginkgo, and ginseng because of their effects.
- Another anticoagulant drug, ximelagatran (Exanta), may be useful as well (O'Brien and Gage, 2005). It is an oral direct thrombin inhibitor (DTI) that binds noncovalently and reversibly to both fibrin-bound and freely circulating thrombin (Petersen, 2005).
- Antibiotics are used in bacterial infections.
- Aspirin or acetaminophen may be used to reduce fever or pain. Chronic use of aspirin may decrease serum ferritin by increasing occult blood loss.
- Thrombolytics may be given to quickly dissolve blood clots that cause symptoms during life-threatening situations. Thrombin inhibitors may be used to interfere with the clotting process.

### Herbs, Botanicals, and Supplements

- See Table 6-3 for guidance.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Bedrest may be important during acute stages of DVT. Leg and foot elevation may be required. Monitor side effects of immobility if patient will be immobilized for a long period of time.
- Zinc ointment may relieve itching.

- The DASH diet plan should be taught. Discuss flavonoids and other nutrients.
- For overall improvement of venous health, encourage patient to stop smoking, increase exercise, lower elevated lipids, and wear compression stockings if needed.
- Discuss sources of the B vitamins, and suggest a supplement if appropriate, especially for individuals with MTHFR mutations (Varga et al, 2005).

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is key as well.

### For More Information

- Coumadin—Interactive Tutorial  
<http://www.nlm.nih.gov/medlineplus/tutorials/coumadinintroduction/htm/index.htm>
- JAMA Patient Page  
<http://jama.ama-assn.org/cgi/reprint/296/4/468.pdf>
- Mayo Clinic  
<http://www.mayoclinic.com/health/thrombophlebitis/DS00223>
- Medline  
<http://www.nlm.nih.gov/medlineplus/ency/article/001108.htm>
- National Institutes of Health—Thrombophlebitis  
<http://www.nlm.nih.gov/medlineplus/deepveinthrombosis.html>

### THROMBOPHLEBITIS—CITED REFERENCES

- Blom JW, et al. Malignancies, prothrombotic mutations, and the risk of venous thrombosis. *JAMA*. 293:715, 2005.
- Bramlage P, et al. Current concepts for the prevention of venous thromboembolism. *Eur J Clin Invest*. 35:4S, 2005.
- den Heijer M, et al. Homocysteine lowering by B vitamins and the secondary prevention of deep vein thrombosis and pulmonary embolism: a randomized, placebo-controlled, double-blind trial. *Blood*. 109:139, 2007.
- O'Brien CL, Gage BF. Costs and effectiveness of ximelagatran for stroke prophylaxis in chronic atrial fibrillation. *JAMA*. 293:699, 2005.
- Petersen P. Ximelagatran: a promising new drug in thromboembolic disorders. *Curr Pharm Des*. 11:527, 2005.
- Pomp ER, et al. Pregnancy, the postpartum period and prothrombotic defects: risk of venous thrombosis in the MEGA study. *J Thromb Haemost*. 6:632, 2008.
- Varga EA, et al. Cardiology patient pages. Homocysteine and MTHFR mutations: relation to thrombosis and coronary artery disease. *Circulation*. 111:289, 2005.

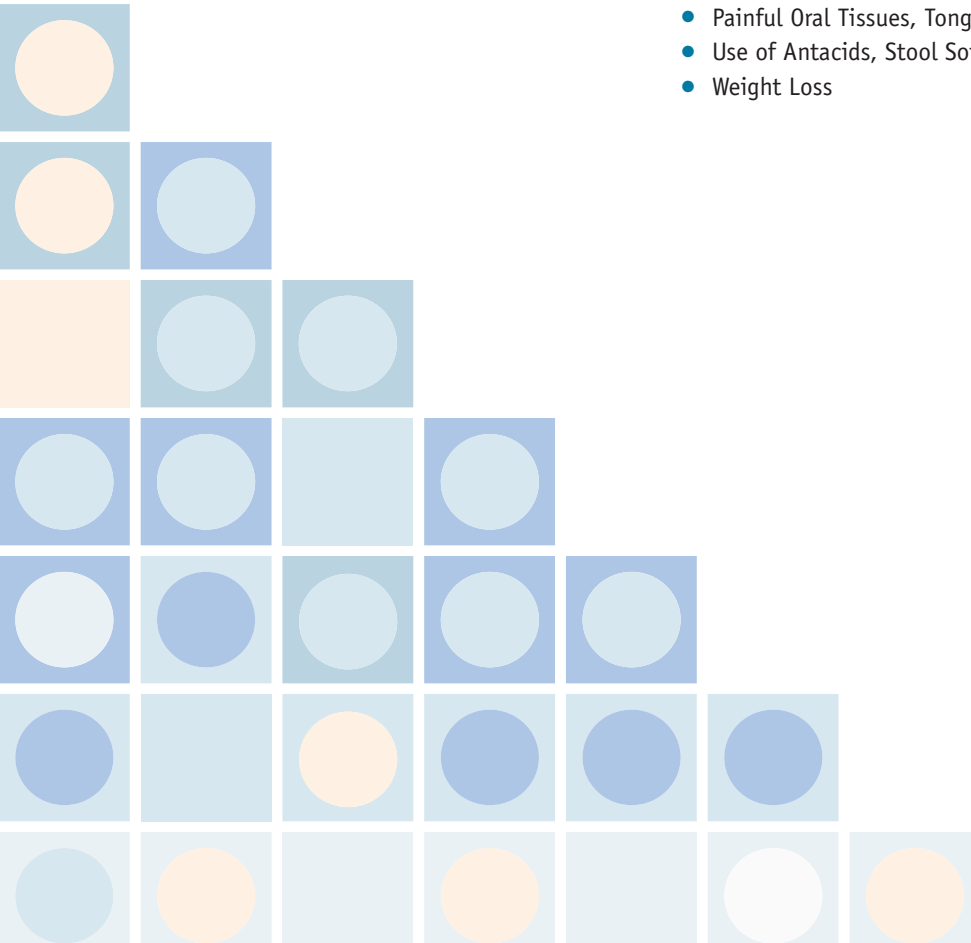




# Gastrointestinal Disorders

## CHIEF ASSESSMENT FACTORS

- Abdominal Pain or Distention
- Appetite, Anorexia
- Ascites, Jaundice
- Bezoars (Undigested Foreign Matter, Usually in Stomach)
- Change in Eating or Bowel Habits
- Change in Stools, Consistency, and Frequency; Fecal Incontinence
- Constipation
- Dentition
- Diarrhea (bloody, explosive)
- Dysphagia
- Easy Fatigue
- Edema of Extremities
- Feeding Method for Digestive or Absorptive Problems
- Hemorrhoids, Rectal Bleeding or Polyps
- Indigestion, Heartburn, Reflux
- Nausea, Vomiting, Regurgitation
- Painful or Cramping Abdomen, Flatulence
- Painful Oral Tissues, Tongue
- Use of Antacids, Stool Softeners, Diuretics, Laxatives, Histamine Blockers
- Weight Loss



## INTRODUCTION AND BACKGROUND FOR GASTROINTESTINAL DISORDERS

### Digestion

Digestion is a process that physically and chemically breaks down food in preparation for absorption. It begins with mastication and mixing of food with salivary fluid and enzymes (oral phase). In the gastric phase, pepsin and gastric acid begin to work. Chyme is then delivered to the small intestine for mixing with pancreatic and biliary juices. The pancreatic phase involves pancreatic amylase and lipase, proteases, and phospholipase. The intestinal phase involves disaccharidases (maltase, lactase, and sucrase), peptidases, and cholecystokinin for bile salts. *Maldigestion* involves the interference at any of these stages, including abnormal emptying of the stomach and pancreatic insufficiency.

### Absorption

Passage of molecular nutrients into the bloodstream from the intestinal cells, starts primarily in the duodenum, with monosaccharides, amino acids and small peptides, mono-glycerides, and free fatty acids. Water is also absorbed to maintain isotonicity of blood and cells. Bile and fat are needed to absorb fat-soluble vitamins A, D, E, and K. Water-soluble vitamins C and B-complex are usually absorbed in the intestinal mucosa with some storage in the liver. *Malabsorption* can result from dysfunction from any of the above steps.

### Small Intestine

It is approximately 3.8 cm in diameter and 4.8-m long, covered with villi projections to increase absorptive surface. Villi cells have a rapid turnover rate of 2–5 days. Fecal fat is a valuable test of lipid digestion/absorption.

### Large Intestine

It is approximately 5 cm in diameter and 1.5-m long, with two sections (colon and rectum) forming a frame around a highly convoluted small intestine. A diet high in whole and unrefined foods (whole grains, dark green and yellow/orange vegetables and fruits, legumes, nuts, and seeds) is high in antioxidant phenolic compounds, fibers, and other phytochemicals, all of which are beneficial.

### Rectum

It is approximately 12-cm long. The area is susceptible to polyps and tumors.

The entire process of digestion/absorption takes about 24 hours, with large variations among individuals.

Digestive problems such as abdominal pain, diarrhea, nausea, and vomiting result in approximately millions of physician visits, billions in direct U.S. medical costs annually, and frequent use of prescription medications. Table 7-1 lists gastrointestinal (GI) conditions that may lead to malnutri-

**TABLE 7-1** Gastrointestinal Conditions That May Lead to Malnutrition

#### *Malabsorption*

Celiac disease	Pancreatic insufficiency
Crohn's disease	Short bowel syndrome
Disaccharidase deficiencies	Ulcerative colitis
Dumping syndrome	
HIV infection or AIDS	

#### *Mechanical Function*

Achalasia or esophageal hypomotility	Esophageal obstruction
Adynamic ileus	Hirschsprung's disease
Bezoar formation after gastric surgery	Pyloric stenosis
Bowel obstruction	Tracheoesophageal fistula
Esophageal stricture	

#### *Conditions that May Cause Fear of Eating*

Aspiration risk	Crohn's disease
Bloating/obstruction/distension/pain	Dental disease
Cholelithiasis and other biliary diseases	Diarrhea
Diverticulitis	Irritable bowel syndrome
Dumping syndrome	Lactose intolerance
Dysphagia	Pancreatitis (acute or chronic)
Esophageal spasm	Peptic ulcer
Flatulence	Proctitis
Food allergies	Rectal fissures
Gastritis	Reflux esophagitis
Ill-fitting dentures	Ulcerative colitis

tion. The enteric nervous system (ENS) consists of many different types of enteric neurons forming complex reflex circuits that underlie or regulate many gut functions (Young, 2008). The identification of the genetic, molecular, and cellular mechanisms responsible for the colonization of the gut by enteric neuron precursors provides an exciting future management for GI disorders.

The GI tract does not respond well to inflammation where digestion, absorption, and gut barrier protection is impaired (Tappenden, 2008). Arachidonic acid (AA) is the precursor of inflammatory eicosanoids-like prostaglandin E(2) and leukotriene B(4); the n-3 PUFAs eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are anti-inflammatory. Fish oil supplementation in patients with inflammatory bowel diseases (IBD) results in n-3 PUFA incorporation into gut mucosal tissue with the potential for modified inflammation, improved gut histology, decreased disease activity, reduced use of corticosteroids (Calder, 2008), and decreased risk of colorectal cancer (Habermann et al, 2009). Glutamine (an amino acid needed in stress or

**TABLE 7-2 Enteral Nutrition, Prebiotics, Probiotics, and Synbiotics in Gastrointestinal Tract Function**

When an oral diet is not feasible, **enteral nutrition (EN)** is needed to avoid prolonged starvation, to prevent deterioration of intestinal integrity, and to avoid translocation of gut bacteria. One or two basic formulas can meet most patients' needs.

**Prebiotics:** Low-digestible carbohydrates that are not digested in the upper GI tract become fermented in the large intestine. They have physiological benefits similar to those of dietary fiber. Short-chain fatty acids (SCFAs) are produced from fermentation of fiber; the SCFAs are fuel for mucosal cells, so they benefit the gut tissue. Fermentation leads to the selective stimulation and growth of beneficial gut bacteria such as *bifidobacteria* (prebiotics). Carbohydrates that offer desirable physiological properties are resistant starch (RS), oligofructose, and polydextrose.

**Probiotics** are live microbial food supplements; they support balance in the intestinal tract. Probiotics have the ability to modify gut pH, antagonize pathogens, produce lactase, and stimulate immunomodulatory cells. Functional foods such as yogurt with live cultures may decrease the incidence of cancer, allergic reactions, and lactose intolerance. Probiotics also have immunomodulating properties and enhance the mucosal barrier in inflammatory bowel disease, pancreatitis, liver transplantation, and diarrhea (Jenkins et al, 2005).

**Synbiotics** are combinations of prebiotics and probiotics. By combining specific prebiotics and plant fibers into multifiber synbiotics, immunosupportive effects are possible (Bengmark and Martindale, 2005). The use of these products may become common practice in intensive care and GI units. Crohn's disease and ulcerative colitis are caused by overly aggressive immune responses to a nonpathogenic enteric bacteria in genetically predisposed individuals; administration of probiotics, prebiotics, or synbiotics can restore a predominance of beneficial *Lactobacillus* and *Bifidobacterium*. They may be protective against colon cancer as well (Pool-Zobel, 2005).

#### REFERENCES

- Bengmark S, Martindale R. Prebiotics and synbiotics in clinical medicine. *Nutr Clin Pract.* 20:244, 2005.  
 Jenkins B, et al. Probiotics: a practical review of their role in specific clinical scenarios. *Nutr Clin Pract.* 20:262, 2005.  
 Pool-Zobel BL. Inulin-type fructans and reduction in colon cancer risk: review of experimental and human data. *Br J Nutr.* 93:S73, 2005.

**TABLE 7-3 Conditions That May Benefit from Use of Intestinal Fuels<sup>a</sup>**

Bowel resection  
 Constipation  
 Diarrhea  
 Diverticulosis  
 Dumping syndrome  
 Inflammatory bowel disease (IBD)  
 Irritable bowel syndrome (IBS)  
 Parenteral nutrition-induced bowel atrophy  
 Radiation/chemotherapy damage to the GI tract  
 Tube feeding

<sup>a</sup>Glutamine, short-chain fatty acids, soy, fermentable fiber, prebiotics, probiotics

sepsis) requires GI processing to become effective; this is likely true for other nutrients. Threonine is an essential amino acid that is abundantly present in intestinally produced glycoproteins; studies in infants suggest a high obligatory visceral need for it in protein synthesis (van der Schoor et al, 2007). Lysine, arginine, and other amino acids are also used in intestinal health, providing support for early enteral feeding whenever possible. Table 7-2 gives details about the role of enteral nutrition, prebiotics, probiotics, and synbiotics in GI health. The right product and the right ingredients can make a big difference. Therefore, it is beneficial to employ a nutrition support specialist or team in facilities where many patients need enteral or parenteral support. Table 7-3 lists GI conditions that may benefit from nutritional enhancements and Table 7-4 describes the role of dietitians in the GI specialty area.

**TABLE 7-4 Knowledge and Skills of Dietitians in Gastrointestinal Specialty**

#### The GI dietetics practitioner:

- Knows sites of digestion and absorption of macronutrients and micronutrients
- Understands normal digestion, nutrient secretion, and absorption
- Identifies key nutritional screening factors for persons with GI disease
- Explains the association between diet and other therapies in treating GI disorders
- Knows the value and limitations of enteral and parenteral nutrition formulas and common functional food ingredients
- Knows how to handle complex GI problems in conditions as diverse as cystic fibrosis (Mascarenhas, 2003) and celiac disease (Niewinski, 2008)
- Recognizes extremes of dietary intake and the effects of diet on GI function and symptoms
- Understands how GI dysfunction, surgical resections, and diseases affect nutritional status
- Understands how other diseases and conditions can affect GI function, such as diabetes and gastroparesis, postanesthetic ileus, neurological injury, and hormonal changes
- Understands the consequences of eating patterns in both healthy persons and in those who have GI disease

#### REFERENCES

- Beyer PL. Gastrointestinal disorders: roles of nutrition and the dietetics practitioner. *J Am Diet Assoc.* 98:272, 1998.  
 Mascarenhas MR. Treatment of Gastrointestinal Problems in Cystic Fibrosis. *Current Treat Options Gastroenterol.* 6:427, 2003.  
 Niewinski MM. Advances in celiac disease and gluten-free diet. *J Am Diet.* 108:661, 2008.



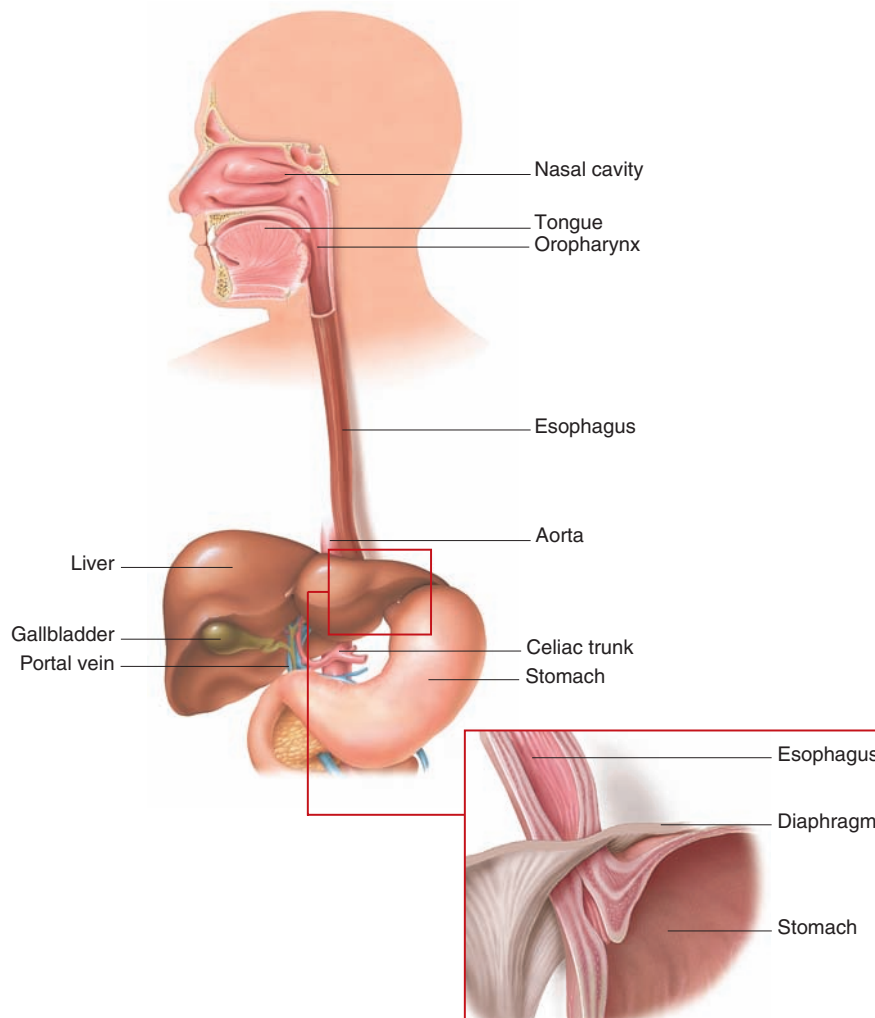
**For More Information**

- American College of Gastroenterology  
<http://www.acg.gi.org/>
- American Digestive Health Foundation  
<http://www.fdhf.org/>
- American Gastroenterological Association  
<http://www.gastro.org>
- American Society of Gastrointestinal Endoscopy  
<http://www.asge.org>
- Cleveland Clinic Foundation  
<http://www.clevelandclinic.org/gastro/>
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)  
<http://www.niddk.nih.gov/>
- Society of American Gastrointestinal Endoscopic Surgeons  
<http://www.sages.org>

- Society of Gastroenterology Nurses and Associates  
<http://www.sgna.org>
- World Organization for Digestive Endoscopy  
<http://www.omed.org>

**CITED REFERENCES**

- Calder PC. Polyunsaturated fatty acids, inflammatory processes and inflammatory bowel diseases. *Mol Nutr Food Res.* 52:885, 2008.
- Habermann N, et al. Modulation of gene expression in eicosapentaenoic acid and docosahexaenoic acid treated human colon adenoma cells. *Genes Nutr.* 4:73, 2009.
- Tappenden KA. Inflammation and intestinal function: where does it start and what does it mean? *JPEN J Parenter Enter Nutr.* 32:648, 2008.
- van Der Schoor SR, et al. The gut takes nearly all: threonine kinetics in infants. *Am J Clin Nutr.* 86:1132, 2007.
- Young HM. Functional development of the enteric nervous system—from migration to motility. *Neurogastroenterol Motil.* 20:20S, 2008.

**UPPER GI: ESOPHAGUS****DYSPHAGIA****NUTRITIONAL ACUITY RANKING: LEVEL 3–4**

**TABLE 7-5 Standard Questions in the Evaluation of Dysphagia**

Describe any difficulty swallowing that you have.  
 Is the swallowing difficulty greater for solids or liquids?  
 How long have you had this swallowing difficulty?  
 Do you have heartburn along with this difficulty swallowing?  
 Is swallowing painful?  
 Do you get chest pain?  
 Does food get stuck when you swallow?  
 Do you choke or cough with swallowing?  
 Do you have temperature sensitivity (especially cold foods and beverages)?  
 Has there been any weight loss?

Adapted from: <http://www.ncbi.nlm.nih.gov/bookshelf/br.fcgi?book=cm&part=A2747>, accessed July 25, 2009.



## DEFINITIONS AND BACKGROUND

Anatomic or physiological swallowing problems of dysphagia create a disturbance in the normal transfer of food from the oral cavity to the stomach. Swallowing requires 5–10 seconds and three phases for completion—oral phase, pharyngeal phase, and esophageal phase. All must be adequate to prevent choking and/or aspiration into the lung. Signs of possible dysphagia include coughing with meals, choking, drooling, or pocketing of foods. Consult speech therapist for a full evaluation; a barium swallow may reveal silent aspiration. Videofluoroscopy (VF) is the gold standard. Questions to ask about swallowing difficulty are listed in Table 7-5.

Progress diet when possible, under guidance of the therapist. Inadequate dietary intake, weight loss, nutrient deficiencies, protein-energy malnutrition, and dehydration may result from prolonged dysphagia. In children with developmental disabilities, diagnosis-specific treatment of feeding disorders results in significantly improved energy consumption and nutritional status.

Dysphagia is classified into oropharyngeal dysphagia due to malfunction of the pharynx and upper esophageal sphincter, or esophageal dysphagia due to malfunction of the esophagus. Common causes of dysphagia are listed in Table 7-6.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Mutations in the PABPN1 gene can cause an oculopharyngeal muscular dystrophy.

#### Clinical/History

Height  
Weight  
Body mass index (BMI)

Weight changes  
Diet history and swallowing problems—current, history, duration

Fiberoptic endoscopic evaluation of swallowing (FEES)

Videofluoroscopic swallowing study	Wet, gurgly voice after drinking or eating	Upper GI bleeding Blood pressure (BP)
Esophagoscopy	Slurred speech	<b>Lab Work</b>
Barium swallow	Poor tongue control or excessive movement	Hemoglobin and hematocrit (H & H)
Cookie swallow test	Hoarseness, breathy voice	Blood urea nitrogen (BUN)
Pocketing of food under tongue or in cheeks	Recurrent pneumonia	Albumin (Alb)
Spitting food out of mouth	Mealtime resistance—clenching teeth or throat	C-reactive protein (CRP)
Facial weakness	Regurgitation of food through nose or mouth	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup> Ca <sup>++</sup> , Mg <sup>++</sup>
Slow oral transit time		Other labs specific for the disorder
Choking		
Coughing before, during, or after swallowing		
Excessive eating time		

**TABLE 7-6 Common Causes of Dysphagia**

Achalasia	Head trauma
Aging	Hiatal hernia
Alzheimer's disease	Huntington's disease
Amyotrophic lateral sclerosis	Lung cancer
Cerebral palsy	Meningitis
Cerebrovascular accident (stroke)	Multiple sclerosis
Chronic obstructive lung disease	Muscular dystrophy
Cleft lip or palate	Myasthenia gravis
Closed head injury	Myotonic dystonia
Dehydration from medications	Parkinson's disease
Diabetes, type 1 (long term)	Pneumonia with aspiration history
Encephalopathy	Poliomyelitis
Esophageal inflammation	Prematurity
Esophageal fistula	Pulmonary disorders
Esophageal obstruction or stricture	Radiation treatment to head/neck
Esophageal trauma	Sjögren's disease
Gastroparesis	Spinal cord injury
Gastroesophageal reflux (GERD)	Throat cancer or injury
Goiter	Tongue cancer
Guillain-Barré syndrome	Tracheoesophageal fistula
Head or neck cancer	Zenker's diverticulum

## SAMPLE NUTRITION CARE PROCESS STEPS

**Dysphagia**

**Assessment Data:** Weight loss, dysphagia and jaw pain, throbbing headache

**Nutrition Diagnoses (PES):** Dysphagia related to progression of Parkinson's disease as evidenced by weight loss, difficulty swallowing solids, and choking on thin liquids.

**Interventions:** Education about blending foods and simplifying meal planning to increase intake. Swallowing evaluation for determination of appropriate textures and thickening of liquids. Counseling on positioning at mealtime to reduce likelihood of aspiration.

**Monitoring and Evaluation:** Improved intake for meals and snacks; fewer complaints of difficulty swallowing. No further weight loss.

## INTERVENTION



## OBJECTIVES

- Prevent choking and aspiration of foods and beverages.
- Promote weight maintenance or gain if losses have occurred. In head and neck cancer patients, anorexia and dysphagia are factors that adversely affect outcome of treatments (Kubrak et al, 2010).
- Individualize diet based on patient needs and preferences. Refer to speech therapist, who will help to determine the level of consistency that is required (e.g., nectar or syrup, honey, pudding). Monitor for pocketing of food.
- For some patients, thin liquids may be needed. Modify levels of dysphagia diets as impairment level changes; upgrade when and if safe for the patient.
- Support independence in eating whenever possible. Provide foods that stimulate the swallowing reflex.
- For persons who have viscous oral secretions or dry mouth, liquefy foods before serving by adding broth, juice, or water. Provide moistened foods or thickened beverages for adequate hydration.
- Correct any nutrient deficits. Prevent pressure ulcers, if relevant.



## FOOD AND NUTRITION

- The patient may be fed enterally if needed. Results from the FOOD (Feed or Ordinary Food) Collaboration Trials showed that nasogastric (NG) tube feeding (TF) was favored over percutaneous endoscopic gastrostomy (PEG) as the early route of feeding in dysphagic stroke patients (Prosser-Loose and Paterson, 2006).
- If needed, jejunostomy feedings may be more appropriate for the patient's condition. Home TF may be needed,

depending on the medical condition and cause of dysphagia.

- If central parenteral nutrition (CPN) is needed, monitor closely for ability to progress back to TF or oral diet.
- Following a protocol, clinical guideline, or algorithms improves dysphagia management.
- Calculate needs at 30–35 kcal/kg. Use a 1- to 1.5-g protein intake per kilogram to assure adequacy and to prevent loss of lean body mass. Monitor cardiac, hepatic, and renal status accordingly.
- Prevent aspiration by careful selection of foods and beverages, such as thick, soft, pureed foods instead of thin liquids. Thickening may be at honey, nectar, or pudding consistency; the label on the thickener will indicate the amount required for the differing levels. When a thickened liquid diet is ordered, foods such as gelatin should not be used because they liquefy at body temperature. Thicken foods and beverages with special products such as Thick-It, Thicken-Up, and Thick 'n Easy; these products use thickeners to make semisolids out of coffee, soup, beverages, juices, and shakes.
- It is possible to use mashed potato flakes to thicken some meat and casserole dishes. Baby rice cereal is an inexpensive thickener as well. Fruit purees are also helpful in thickening juices and some desserts.
- Progress over time to a soft diet.
- With a decreased saliva production, moisten foods with small amounts of liquid and use extra fats, mild sauces, and gravies.
- Monitor for deficiencies in fiber, vitamins A and C if whole grains, fruits, and vegetables are not consumed.
- Avoid alcoholic and extremely hot or cold liquids and beverages.
- Avoid foods that cause choking or are hard to manage: tart juices and foods, dry or crisp foods such as crackers and chips, bony fish, chewy meats such as steak, sticky foods such as peanut butter or bananas, thinly pureed foods that are easily aspirated, foods with varying consistency, excessively sweet drinks that aggravate drooling, carbonated beverages, dry bread in sandwiches.
- Avoid foods that are easily aspirated: popcorn, bran cereals, nuts, dry mashed potatoes, cottage cheese, corn, celery, pineapple, other fruits or vegetables with skins or fibrous pulp.
- Where there is reduced oral sensation, position food in the most sensitive area and use cold foods.
- To form a more cohesive bolus of food in the mouth, serve semisolid consistencies.
- For a severely sore mouth, avoid acidic foods and use soft foods at moderate temperatures.
- For delayed or absent swallowing reflex, temperature extremes and spicy foods may help excite the nerves necessary to function better. Some thickened liquids may actually be beneficial. Use cohesive foods that do not fall apart.
- Finely crushed bran on cereal, powdered fiber, or high-fiber tube-feeding products can help alleviate constipation.
- An interdisciplinary care plan is available for use.

# INTERDISCIPLINARY NUTRITION CARE PLAN

## Dysphagia

Client Name: \_\_\_\_\_ #: \_\_\_\_\_ Initiated by: \_\_\_\_\_ Date: \_\_\_\_\_

### SCREEN

Nutrition Screen diagnosis: Dysphagia

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### GOALS (Check any/all):

- ☐ Safely eat and drink without risk in \_\_\_\_ (goal time).
  - ☐ Swallow efficiently to maintain nutrition and hydration in \_\_\_\_ (goal time).
  - ☐ Advance to normal texture of food in \_\_\_\_ (goal time).
- Weight ☐ maintained, or ☐ loss/ ☐ gain of \_\_\_\_ lb in \_\_\_\_ (goal time).

### ASSESS (Check any/all)

#### Food/liquid texture modification

#### Weight/BMI

- ☐ Weight loss >3 lb/wk or >5%/mo or >10%/6 mo
- ☐ BMI <20 (High risk)
- ☐ BMI <27

#### Medications

#### Infection (e.g., pneumonia)

#### Pressure ulcers/wounds

#### Poor oral intake symptoms

- ☐ Complex diet order
- ☐ Nausea/vomiting
- ☐ Poor appetite/early satiety
- ☐ Problems chewing/swallowing
- ☐ Depression/anxiety
- ☐ GI distress

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### MODERATE RISK INTERVENTIONS (Check any/all)

- ☐ Foods that are easy to swallow provided and explained
- ☐ Food record provided and explained
- Obtain Dr. orders as needed:**
  - ☐ RD chart consult
  - ☐ SLP chart consult
  - ☐ OT chart consult
  - ☐ Monitor weight q: \_\_\_\_\_
  - ☐ Monitor I & O q: \_\_\_\_\_
  - ☐ BID/TID supplements
- ☐ Other: \_\_\_\_\_  
(See notes for documentation.)

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### HIGH-RISK INTERVENTIONS (Check any/all)

- ☐ Foods that are easy to swallow provided and explained
- ☐ Food record provided and explained
- Obtain Dr. orders as needed:**
  - ☐ RD referral for home visit(s)
  - ☐ SLP referral for home visit(s)
  - ☐ OT referral for home visit(s)
  - ☐ Monitor weight q: \_\_\_\_\_
  - ☐ Monitor I & O q: \_\_\_\_\_
  - ☐ BID/TID supplements
- ☐ Medication adjustment
- ☐ Other: \_\_\_\_\_  
(See notes for documentation.)

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### ASSESS RESPONSE (Check any/all)

- ☐ Further food/liquid texture modification required
- ☐ Weight change not appropriate per goal
- ☐ Dehydration
- ☐ Onset of pulmonary infection
- ☐ Exhibiting Poor Oral Intake Symptoms
- ☐ Other: \_\_\_\_\_  
(See notes for documentation.)

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### OUTCOMES ACHIEVED

- ☐ Food/liquid texture advanced toward normal
- ☐ Weight maintained or improved
- ☐ Hydration status maintained or improved
- ☐ Other: \_\_\_\_\_  
(See notes for documentation.)
- ☐ Repeat Nutrition Risk Screen in \_\_\_\_ days

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### ASSESS RESPONSE (Check any/all)

- ☐ Further food/liquid texture modification required
- ☐ Continued poor oral intake symptoms
- ☐ Weight change not appropriate per goal
- ☐ Dehydration
- ☐ Onset of pulmonary infection
- ☐ Other: \_\_\_\_\_  
(See notes for documentation.)

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### OUTCOMES ACHIEVED

- ☐ Food/liquid texture advanced toward normal
- ☐ Weight maintained or improved
- ☐ Hydration status maintained or improved
- ☐ Other: \_\_\_\_\_  
(See notes for documentation.)
- ☐ Repeat Nutrition Risk Screen in \_\_\_\_ days

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

### OUTCOMES NOT ACHIEVED

Reassess/evaluate need for EN/PN (refer to Tube Feeding Nutrition Care Plan). Document on Nutrition Variance Tracking form.



## Common Drugs Used and Potential Side Effects

- For thick saliva and gagging, artificial saliva such as lemon glycerin may be useful.
- Papain or citrus juices may be useful for thinning secretions.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- It is important to stress that no supplement or diet can cure dysphagia.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Follow meals by brushing teeth to reduce dental caries; encourage optimal mouth care.
- Offer suggestions for specific changes in food preparation (e.g., adding moistening sauces, gravies, etc.) and cutting or mincing foods to increase control of the swallowing process.

- Encourage regular review of changes in swallowing abilities to identify early decline or to lessen restrictions when possible.
- Monitor for quality of life factors and adjust where possible.

### Patient Education—Foodborne Illness

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

### For More Information

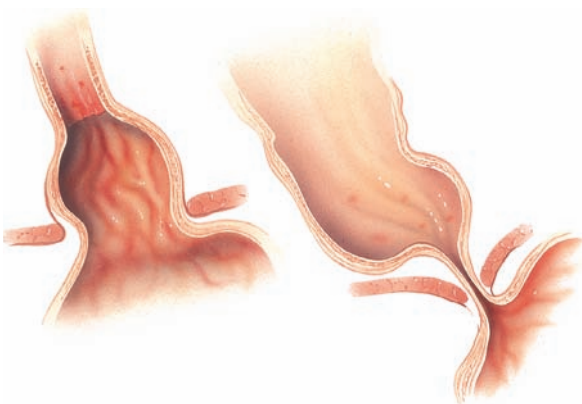
- Dysphagia On-Line (Nestle)  
[http://www.dysphagiaonline.com/en/pages/01\\_what\\_is\\_dysphagia.aspx](http://www.dysphagiaonline.com/en/pages/01_what_is_dysphagia.aspx)
- E-medicine  
<http://emedicine.medscape.com/article/324096-overview>
- NIH—Dysphagia  
<http://www.nidcd.nih.gov/health/voice/dysph.asp>

## DYSPHAGIA—CITED REFERENCES

- Kubrak C, et al. Nutrition impact symptoms: key determinants of reduced dietary intake, weight loss, and reduced functional capacity of patients with head and neck cancer before treatment [published online ahead of print July 22, 2009]. *Head Neck*. 32:290, 2010.
- Prosser-Loose EJ, Paterson PG. The FOOD Trial Collaboration: nutritional supplementation strategies and acute stroke outcome. *Nutr Rev*. 64:289, 2006.

# ESOPHAGEAL STRICTURE OR SPASM, ACHALASIA, OR ZENKER'S DIVERTICULUM

## NUTRITIONAL ACUITY RANKING: LEVEL 3, STRICTURE; LEVEL 2 ACHALASIA



Asset provided by Anatomical Chart Co.



### DEFINITIONS AND BACKGROUND

**Esophageal stricture** is caused by injury or chemical ingestion (such as lye), sliding hiatal hernia, esophageal cancer, reflux esophagitis (RE), peptic ulcer disease, or prolonged use of an NG tube. Another cause is eosinophilic esophagitis, with solid food dysphagia on presentation. Scar tissue builds up and prevents normal swallowing.

In **esophageal spasm**, segmented, concentric contractions occur simultaneously in the lower two thirds of the esophagus. Barium and manometric studies are useful in the evaluation. Achalasia and diffuse esophageal spasm are associated with hypertrophy of circular and longitudinal muscle layers (Mittal et al, 2005). Chemical denervation with *Clostridium botulinum* toxin (Botox) is effective in relieving spasm.

**Achalasia** is failure of the cardiac sphincter to relax, with obstruction of food passage into the stomach. In addition, the esophagus does not demonstrate normal waves of contraction after swallowing. The mechanisms remain poorly understood (Kraichely and Farrugia, 2006; Sonnenberg, 2009). Esophageal dilatation or surgical myotomy is common. Laparoscopic procedures are preferred (Pastor et al, 2009).

**Zenker's diverticulum** (ZD, or pharyngeal pouch) generally presents after 60 years of age, but patients may have years of dysphagia symptoms. Regurgitation of undigested food when patient bends over or lies down may occur. Serious complications include aspiration and malnutrition (Ferreira et al, 2008). Diagnosis is by barium swallow, and treatment is open surgical diverticulectomy. Minimally invasive (endoscopic stapling) devices are often used. Oral feeding is usually possible the day after surgery as general anesthesia is not needed.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Achalasia is usually of idiopathic origin but may be secondary to cancer or mast cell disorders.

Esophageal stricture secondary to radiation treatment may show signs of altered interleukin or tumor necrosis factor (TNF).

Clinical/History		Lab Work
Height	Regurgitation, halitosis	Glucose (Gluc)
Weight	Heartburn or	Alb
Weight loss?	other specific	CRP
BMI	symptoms	Gastrin
Diet history	Endoscopy	H & H
Intake and output (I & O)	Esophageal manometry	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>
Dysphagia	Esophagoscopy	Ca <sup>++</sup> , Mg <sup>++</sup>
Substernal pain after meals	Cineesophagography	
	BP	

## INTERVENTION



### OBJECTIVES

- **Esophageal stricture:** Avoid large boluses of food. Provide adequate nutrition. Prevent weight loss. Remove cause or dilate, if necessary.
- **Esophageal spasm:** Avoid either very cold or very hot foods or beverages. Monitor dysphagia.
- **Achalasia:** Individualize diet according to patient tolerances and preferences. Monitor chronic dysphagia. Avoid aspiration.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Dysphagia and Esophageal Stricture

**Assessment Data:** Weight loss, choking after swallowing, regurgitation, heartburn and chest pain after eating.

**Nutrition Diagnoses (PES):** Dysphagia related to altered GI function and esophageal stricture as evidenced by difficulty swallowing, abnormal barium swallow, and weight loss of 10 lb in last month.

**Interventions:** Education about blending foods and simplifying meal planning to increase intake. Swallowing evaluation for determination of appropriate textures and thickening of liquids. Counseling on positioning at mealtime; preparation for surgery

**Monitoring and Evaluation:** Improved intake of meals and snacks. Resolution of esophageal structure after surgery with no further weight loss.



## FOOD AND NUTRITION

- **Esophageal stricture:** Begin with liquid diet and progress to soft diet as tolerance increases. Adequate calories are needed. Gastrostomy may be needed. Antireflux regimen (no alcohol, weight loss) may be helpful. Avoid sticky and dry foods. Use thin liquids and pureed or soft foods.
- **Esophageal spasm:** Use diet as tolerated with modified temperatures for foods and beverages.
- **Achalasia:** Patients should take smaller bites of food, chew well, and eat slowly. Provide large volumes of fluids with each meal, unless dysphagia prevents appropriate swallowing of liquids. Tube feed if needed; may need to use gastrostomy TF.

### Common Drugs Used and Potential Side Effects

- Antacids: Check the label for aluminum, calcium, magnesium, or sodium if other medical problems exist. Beware of long-term side effects.
- Isosorbide dinitrate and calcium channel blockers such as nifedipine may be needed 30 minutes before meals.
- Nitroglycerin often helps spasm. Headache is one possible side effect.

### Herbs, Botanicals, and Supplements

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



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Emphasize the importance of spacing meals and achieving relaxation. Recommend intake of food at moderate temperature only.
- Elevate head of bed for 30–45 minutes after meals and at bedtime.
- Encourage fluids at mealtimes.
- Avoid foods that aggravate dysphagia.
- Bland foods are not clearly beneficial and not required.

### Patient Education—Foodborne Illness

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

### For More Information

- Achalasia  
<http://www.medicinenet.com/achalasia/article.htm>
- Baylor College of Medicine—Zenker's diverticulum  
[http://www.bcm.edu/oto/grand/05\\_06\\_04.htm](http://www.bcm.edu/oto/grand/05_06_04.htm)
- Esophagitis  
<http://chorus.rad.mcw.edu/doc/00858.html>

## ESOPHAGEAL STRICTURE OR SPASM, ACHALASIA, AND ZENKER'S DIVERTICULUM—CITED REFERENCES

Ferreira LE, et al. Zenker's diverticula: pathophysiology, clinical presentation, and flexible endoscopic management. *Dis Esophagus*. 21:1, 2008.

Kraichely RE, Farrugia G. Achalasia: physiology and etiopathogenesis. *Dis Esophagus*. 19:213, 2006.

Mittal RK, et al. Sensory and motor function of the esophagus: lessons from ultrasound imaging. *Gastroenterology*. 128:487, 2005.

Pastor AC, et al. A single center 26-year experience with treatment of esophageal achalasia: is there an optimal method? *J Pediatr Surg*. 44:1349, 2009.

Sonnenberg A. Hospitalization for achalasia in the United States 1997–2006. *Dig Dis Sci*. 54:1680, 2009.

# ESOPHAGEAL TRAUMA

## NUTRITIONAL ACUITY RANKING: LEVEL 3–4



### DEFINITIONS AND BACKGROUND

Esophageal trauma is a major traumatic condition that affects the esophagus; it is often caused by chemical burns, ingestion of foreign bodies, or injury. Diagnosis of penetrating pharyngeal and esophageal injuries is difficult when the patient has severe facial injuries, is obese, intubated, or hemodynamically unstable (Ahmed et al, 2009).

**Boerhaave syndrome** involves complete laceration of the esophagus, sometimes spontaneously from alcohol abuse with retching or secondary to endoscopy or vagotomy. **Mallory–Weiss syndrome** involves a mucosal gastric tear that can occur at the gastroesophageal (GE) junction or proximal stomach; it is also associated with retching or alcohol abuse.



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Esophageal trauma is acquired, not genetic.

<b>Clinical/History</b>	Respiratory distress	Na <sup>+</sup> , K <sup>+</sup>
Height	Esophageal perforation?	TLC
Weight	Dysphagia	H & H
BMI	Temperature	Serum Fe, ferritin, ferritin
Weight changes		Alb or transthyretin
Videoendoscopy		Transferrin
I & O	<b>Lab Work</b>	Ca <sup>++</sup> , Mg <sup>++</sup>
Nausea, vomiting	Gluc	
Loss of consciousness	BUN, creatinine (Creat)	

## INTERVENTION



### OBJECTIVES

- Emergency care, such as adequate ventilation, is given as needed.

- Allow the esophagus to rest and heal. Prepare for esophageal surgery if necessary.
- Keep the patient adequately hydrated.
- Improve swallowing capacity as rapidly as possible; prevent aspiration.
- Prevent malnutrition, weight loss, sepsis, constipation, fluid loss from exudates, and other complications.
- For serious injuries with permanent damage, it may be necessary for a gastrostomy TF to be used.



### FOOD AND NUTRITION

- Nothing by mouth (NPO) as needed. Provide CPN, gastrostomy, or jejunostomy feedings as appropriate for the patient's condition.
- Calculate needs with extra protein, if applicable. Monitor cardiac, hepatic, and renal status.
- Progress over time to a soft diet. Avoid alcoholic beverages, extremely hot liquids and beverages, caffeine, and spicy foods if not tolerated.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Unintentional Weight Loss

**Assessment:** Prior gunshot wound to the neck that required esophageal repair. Loss of 10 lb this year even though eating "a lot."

**Nutrition Diagnosis (PES):** Inability to digest/absorb nutrients (NC-1.4) related to surgical alteration in GI anatomical structure as evidenced by weight loss, albumin indicative of malnutrition.

**Interventions:** Food and Nutrient Delivery: ND 1.2—Alter diet as tolerated. ND 32.3 and 32.4—Initiate vitamin and mineral supplementation.

Education: E-1.2—Discuss ways to increase energy and nutrient density in food choices.

Counseling: C-2.3—Keep a food diary for 1 month. C-2.2—Set goal of only nutrient and energy-dense meals for 1 month if tolerated.

**Monitoring and Evaluation:** Review food diary after 1 month. Monitor weight for resolution of unintentional weight loss; goal is gain of 1–2 lb weekly.

- Provide adequate fluids but avoid overhydration.
- When able to eat orally, monitor for signs of dysphagia. Work with a speech therapist for proper consistency evaluations. Use appropriately thickened or thinned liquids and pureed foods until swallowing ability returns.
- Home TF may be needed, for which either a gastrostomy or jejunostomy will be used, depending on location and extent of injury and surgical repair.

### Common Drugs Used and Potential Side Effects

- Liquid topical anesthetizing agents (such as lidocaine) may be used before meals to reduce pain.
- Antibiotics may be used in bacterial infections.

### Herbs, Botanicals, and Supplements

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



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- When the patient can swallow, discuss the need to chew well and swallow carefully. The patient should also learn to eat slowly to prevent aspiration.
- Discuss the appropriate food textures for different stages of progress. This plan will be developed in accordance with the speech therapist and the physician.

### Patient Education—Foodborne Illness

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

### For More Information

- Esophageal trauma  
<http://emedicine.medscape.com/article/775165-overview>

### ESOPHAGEAL TRAUMA—CITED REFERENCES

Ahmed N, et al. Diagnosis of penetrating injuries of the pharynx and esophagus in the severely injured patient. *J Trauma*. 67:152, 2009.

## ESOPHAGEAL VARICES

### NUTRITIONAL ACUITY RANKING: LEVEL 2–3



### DEFINITIONS AND BACKGROUND

Acute bleeding from esophageal varices due to portal hypertension is a frequent and severe complication of liver cirrhosis (Lata et al, 2006). Esophageal varices occur in about half of all people with alcoholic cirrhosis and one third of these will experience variceal hemorrhage, a life-threatening event (Smith, 2010). Portal hypertension is defined as a portal pressure gradient exceeding 5 mm Hg where porto-systemic collaterals decompress the portal circulation and give rise to varices (Toubia and Sanyal, 2008). Small esophageal veins become distended and may rupture due to increased pressure in the portal system. Thrombocytopenia and splenomegaly are independent predictors of large esophageal varices.

All cirrhotic patients should undergo endoscopic screening to detect varices. Severe fibrosis and esophageal varices may be diagnosed through a prothrombin index of less than 60%, alkaline phosphatase activity greater than 110 IU/L, and hyaluronate greater than 100 g/L in alcoholic patients (Vanbiervliet et al, 2005). Maintenance of a good renal function is essential in these patients (Lata et al, 2006). Fortunately, great strides have been made because of noninvasive imaging and pressure measurement. Mortality has been substantially reduced. Band ligation is the first-line endoscopic treatment. If this fails, the more invasive surgical shunt may be needed.



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Cirrhotic patients have an increased risk for thrombosis because of portal blood flow stasis; known risk factors include G20210 A mutation of prothrombin or factor V Leiden (Amitrano et al, 2007).

Clinical/History	Lab Work	
Height	Confusion	Transferrin
Weight	Abdominal distention,	Bilirubin
BMI	Jaundice or	(>20 mg/dL)
Diet history	hepatic coma	Platelet count
Weight changes		(<200,000/mm <sup>3</sup> )
Esophagoscopy		Prothrombin index
Edema	H & H	(<60%)
Melena	Serum Fe, ferritin	Hyaluronate
Upper GI bleeding	Ammonia (NH <sub>3</sub> )	(>100 g/L)
BP	BUN	Alkaline phosphatase
Respiratory distress	Alb (<4.0 g/dL)	(Alk phos)
Aspiration of emesis	Ascites	(>110 IU/L)
	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>	
	Ca <sup>++</sup> , Mg <sup>++</sup>	



## SAMPLE NUTRITION CARE PROCESS STEPS

**Excessive Alcohol Intake**

**Assessment:** Intake history revealing poor intake of nutrient-dense foods and frequent meal skipping with use of one fifth of rum daily for 12+ years. Lab results showing elevated ammonia level, blood in stool, low H & H with documented anemia.

**Nutrition Diagnoses (PES):** Excessive alcohol intake related to intake of rum instead of meals as evidenced by bleeding and varices, diet history, altered hepatic labs.

**Interventions:** Enhance oral intake, or offer PEG TF as needed. Remove access to alcohol and coordinate care for referral to rehabilitation center when feasible.

**Monitoring and Evaluation:** No rebleeding. Adequate intake from TF and slow progression back to oral diet. Improved intake of all macro- and micronutrients, and improvement in labs. Corrected anemia.

## INTERVENTION



## OBJECTIVES

- Promote healing and recovery.
- Prevent variceal rebleeding, which is a very frequent and severe complication in cirrhotic patients (Kravetz, 2007).
- Avoid constipation or straining with stool.
- Prevent or correct hepatic encephalopathy or coma; see Section 8.



## FOOD AND NUTRITION

- Generally, unless comatose, the patient can tolerate five to six small meals of soft foods. Avoid foods such as tacos, tortilla chips, or large pieces of raw fruits and vegetables.
- Alter carbohydrate, protein, and fat intake according to hepatic function and state of consciousness. Monitor micronutrient needs, such as iron if patient is anemic.
- Provide adequate fluid as allowed or controlled.
- To prevent constipation and straining, use foods such as prune juice or formulas with added fiber.
- Consider use of branched-chain amino acid formula for cirrhosis.

## Common Drugs Used and Potential Side Effects

- Antacids may be beneficial to buffer gastric acidity. Extended use can cause problems with pH, altered mineral and nutrient use, and other imbalances.

- Beta-blockers; propranolol reduces the risk of bleeding, especially in cirrhosis (Suzuki et al, 2005). However, the combination of beta-blockers with mononitrate of isosorbide is superior to beta-blockade alone (Kravetz, 2007).
- Vasoactive agents, such as somatostatin analog and terlipressin, are useful.
- Vitamin K may be needed to help with clotting.

## Herbs, Botanicals, and Supplements

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



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The importance of good nutrition with proper consistency should be addressed. Avoid rough or crunchy foods that are fibrous or sharp; chew all foods well before swallowing.
- If gastrostomy is needed, discuss how to manage the process.
- If relevant, discuss the role of alcohol in contributing to the disease process with the patient and family. Advise the patient to avoid mouthwashes, cough syrups, and other products that contain alcohol.

## Patient Education—Foodborne Illness

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

## For More Information

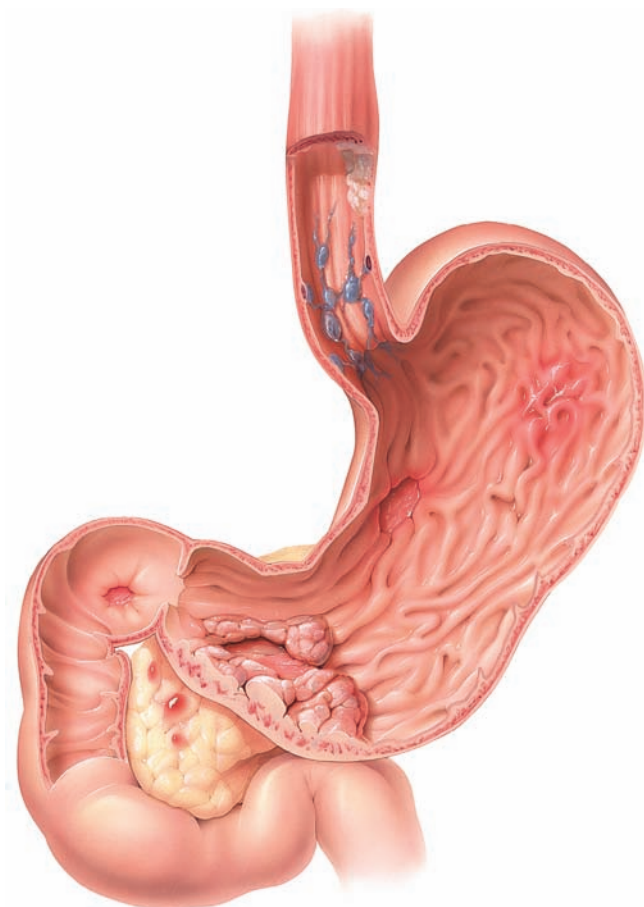
- Esophageal Varices  
<http://www.nlm.nih.gov/medlineplus/ency/article/000268.htm>

## ESOPHAGEAL VARICES—CITED REFERENCES

- Amitrano L, et al. Coagulation abnormalities in cirrhotic patients with portal vein thrombosis. *Clin Lab*. 53:583, 2007.
- Kravetz D. Prevention of recurrent esophageal variceal hemorrhage: review and current recommendations. *J Clin Gastroenterol*. 41:318S, 2007.
- Lata J, et al. Factors participating in the development and mortality of variceal bleeding in portal hypertension—possible effects of the kidney damage and malnutrition. *Hepatogastroenterology*. 53:420, 2006.
- Smith MM. Emergency: Variceal hemorrhage from esophageal varices associated with alcoholic liver disease. *Am J Nurs*. 110:32, 2010.
- Suzuki A, et al. Diagnostic model of esophageal varices in alcoholic liver disease. *Eur J Gastroenterol Hepatol*. 17:307, 2005.
- Toubia N, Sanyal AJ. Portal hypertension and variceal hemorrhage. *Med Clin North Am*. 92:551, 2008.
- Vanbiervliet G, et al. Serum fibrosis markers can detect large oesophageal varices with a high accuracy. *Eur J Gastroenterol Hepatol*. 17:333, 2005.

# ESOPHAGITIS, GASTROESOPHAGEAL REFLUX DISEASE AND HIATAL HERNIA

NUTRITIONAL ACUITY RANKING: LEVEL 2–3



Asset provided by Anatomical Chart Co.



## DEFINITIONS AND BACKGROUND

**Esophagitis** results from gastric juice being forced into the esophagus from the stomach. Pill-induced esophageal injury may occur from use of aspirin, tetracycline, vitamin C, ferrous sulfate, potassium chloride, or nonsteroidal anti-inflammatory drugs (NSAIDs). Take these with plenty of liquid.

**Eosinophilic esophagitis (EoE or EE)** is a disorder characterized by a severe, isolated eosinophilic infiltration of the esophagus that is unresponsive to aggressive acid blockade but responsive to the removal of dietary antigens (Liacouras et al, 2005). Adult patients usually present with dysphagia, food impaction and reflux-like symptoms (Gupte and Draganov, 2009). Known causes of tissue eosinophilia include GE reflux disease (GERD), infections, malignancy, collagen vascular diseases, hypersensitivity, and IBD (Gupte and Draganov, 2009). It is highly associated with atopic disease.

**Barrett's esophagus** is a condition that affects men more than women, and length of impact is greater in men than in women (Falk et al, 2005). It is also more common in Caucasians and in persons older than age 50. Symptoms are similar to GERD, but Barrett's esophagus is more likely to precede esophageal adenocarcinoma. Upper endoscopy and surveillance biopsies may be needed (Liu and Saltzman, 2006). Antioxidants (vitamins C, E, and beta-carotene) are important protective factors (Kubo et al, 2008), whereas obesity and the western diet may be promoters of cancer.

**GERD** and peptic ulcer disease are common in elderly individuals. GERD affects approximately 19 million Americans; prevalence is as high as 80% among asthma patients. There is a significantly higher prevalence of RE in an *Helicobacter pylori*-infected individuals of any age or sex (Moon et al, 2009). Distal esophageal cancer is associated with symptomatic GI reflux disease and Barrett's esophagus; surveillance programs are identifying patients early for curable esophageal adenocarcinoma (Demeester, 2006).

GERD may occur in infants but usually resolves by 6–12 months of age. Management involves thickened feedings and positioning. The recommended approach for infants with uncomplicated regurgitation is the reassurance of the parents about the physiological nature of excessive regurgitation and dietary recommendations for formula feeding. Symptoms of pediatric GERD include colic, inconsolable crying, frequent spitting up or vomiting, food refusal, failure to thrive, heartburn, stomach pains, chronic sore throat, chronic respiratory problems, asthma, and apnea. GERD diagnosis in older children warrants review for upper GI tract disorders, cow's milk allergy, or metabolic, infectious, renal, or central nervous system diseases.

Treatment guidelines address lifestyle changes, patient-directed (over the counter) therapy, acid suppression, pro-motility therapy, maintenance therapy, and antireflux surgery (DeVault and Castell, 2005). Intractable GERD may require minor surgery to strengthen a weak sphincter. Laparoscopic antireflux surgery is highly effective as a long-term treatment for severe GERD.

**Hiatal hernia** is caused by protrusion of part of the stomach through the diaphragm muscle, which separates the chest from the abdomen. This causes an enlarged diaphragm opening (hiatus) through which the esophagus passes to join the stomach. An increase in BMI is associated with the increased prevalence of hiatal hernia, esophageal mucosal injury, and complications because of increased intragastric pressure and increased GE pressure gradient (Fass, 2008). Hiatal hernia may show no symptoms or may contribute to heartburn, swallowing difficulty, reflux, or vomiting. Hiatal hernia surgery has evolved from anatomic repair to physiological restoration (Stylopoulos and Rattner, 2005).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Proton pump inhibitors (PPIs), such as omeprazole, lansoprazole, and rabeprazole are metabolized by CYP2C19 in the liver; there are genetic differences in the activity of this enzyme (Furuta et al, 2010). These genotypic differences influence the healing and eradication rates for GERD and *H. pylori* infection (Furuta et al, 2010). The pathogenesis of EE involves multiple tissues, cell types, and genes, and derives from complex genetic and environmental factors (Blanchard and Rothenberg, 2008).

Clinical/History	Reactive airway disease or nocturnal asthma	<i>H. pylori</i> infection?
Height		Cholesterol (Chol)
Weight		Triglycerides (Trig)
BMI	Choking attacks	Transferrin
Diet history	Dental erosion or caries	Total iron-binding capacity (TIBC)
Weight changes		Bernstein test:
Upper GI endoscopy	<b>Lab Work</b>	HCl solution is dripped into the distal esophagus; positive test mimics patient symptoms
Esophagoscopy	H & H	
PillCAM (noninvasive visualization)	Mean cell volume (MCV)	
Manometry	Na <sup>+</sup> , K <sup>+</sup>	
Feeding difficulties in children	Ca <sup>++</sup> , Mg <sup>++</sup>	
Recurrent vomiting	Gluc	
	Gastrin	
	Alb, transthyretin	
	CRP	

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Gastroesophageal Reflux Disease (GERD)

**Assessment:** Knowledge about the role of diet in GERD; food diary; timing of meals and related symptoms. Use of antacids with every meal and at bedtime.

**Nutrition Diagnosis (PES):** Undesirable food choices related to lack of knowledge regarding role of diet in GERD symptoms and complications as evidenced by frequent consumption of large, high-fat meals and alcoholic beverages.

**Intervention:** Teach about dietary changes that will alleviate GERD symptoms and possibly prevent complications. Counsel about lifestyle changes, such as weight loss, loose clothing, and upright posture during and after eating.

**Monitoring and Evaluation:** Report of relief from symptoms. No complications from GERD. Gradual reduction in need for antacids and medication.

## INTERVENTION



### OBJECTIVES

- In stage 1, simple lifestyle modifications may be successful: elevate head of the bed, decrease fat intake, stop smoking, lose excess weight, avoid eating large meals, and do not eat 3 hours before lying down. Large meals increase gastric pressure and alter pressure on the lower esophageal sphincter (LES), thereby allowing reflux or aspiration to occur. Avoid tightly fitted garments around the abdomen.
- In stage 2, add pharmacological treatments, such as histamine-receptor blockers or antacids.
- In stage 3 with erosive esophagitis, it may be necessary to add a PPI as first-line therapy.
- In stage 4, maintenance therapy, use the lowest possible dose of medications to manage symptoms.
- In severe stage 5, surgery may be needed. This may include laparoscopic Nissan fundoplication.



### FOOD AND NUTRITION

- Provide an individual diet reflecting patient needs. Assess intake of fat, alcohol, spices, and caffeine.
- If needed, a reduced-energy diet should be used to promote weight loss.
- During acute episodes, provide small, frequent feedings of soft foods.
- Diet should be high in protein to stimulate gastrin secretion and to increase LES pressure. Avoid foods that decrease LES pressure, including chocolate, peppermint, onions, garlic, and spearmint.
- Use fewer fried foods, cream sauces, gravies, fatty meats, pastries, nuts, potato chips, butter, and margarine.
- Dietary fiber and physical exercise may be protective. Increased fiber intake benefits a number of GI disorders including GERD (Anderson et al, 2009). Dietary fiber intakes for children and adults should be calculated as 14 g/1000 kcal.
- Avoid foods that may irritate the esophagus, such as citrus juices, tomatoes, and tomato sauce. Other spicy foods are to be eliminated according to individual experience.
- If there is EoE, try a dietary elimination diet and add back foods one at a time to identify potential allergens (Liacouras et al, 2005).
- Fluids can be taken between meals to reduce abdominal distention and discomfort.
- Preterm infants may benefit from transpyloric feedings if they show signs suggestive of reflux and apnea (Malcolm et al, 2009).

### Common Drugs Used and Potential Side Effects

- Antacids neutralize gastric contents. They destroy thiamin and may provide excess sodium for the body; check labels carefully. If the antacid contains calcium (e.g., Tums, which contains calcium carbonate), excess calcium may decrease levels of magnesium and phosphorus. Aluminum hydroxide (Maalox) depletes phosphorus, which is acceptable for patients with certain types of renal diseases, but otherwise this is not desirable for the long term. When

used as an antacid, sodium bicarbonate can decrease iron absorption and causes sodium retention; use with caution.

- Calcium glycerophosphate (Prelief) is somewhat useful for relief of heartburn by neutralizing the acid in foods; it is available over the counter.
- PPIs, such as lansoprazole (Prevacid), omeprazole (Prilosec), and esomeprazole (Nexium) are popular treatments. When clarithromycin-resistant *H. pylori* (CRHP) occur, PPIs tend to inhibit the growth and motility of CRHP. Omeprazole is useful for refractory RE. Because CYP2C19 genotypes affect the recurrence rate of GERD symptoms during PPI maintenance therapy, genotype-based tailored therapy is needed (Furuta et al, 2010; Saitoh et al, 2009).

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Chamomile, fennel, cardamom, cinnamon, dill, and licorice have been recommended for this condition, but no clinical trials have proven efficacy.



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage the patient to avoid late evening meals and snacks and to avoid lying down or sleeping or swimming soon after a meal to guard against reflux.
- Teach the proper measures for controlling weight, including small, frequent feedings.
- Instruct the patient about lifestyle modifications listed in the Objectives. Patients with heartburn probably should not sleep in a waterbed.
- Chewing sugarless gum after meals may reduce reflux somewhat because of the saliva production.
- More effective communication and consumer education is required to enhance fiber consumption from foods or supplements (Anderson et al, 2009).

## Patient Education—Foodborne Illness

- Careful food handling and washing hands before eating are useful recommendations.

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

## For More Information

- International Foundation for Gastrointestinal Disorders <http://www.aboutgerd.org/>
- Heartburn and Regurgitation Algorithm [http://www.uwgi.org/guidelines/ch\\_03/ch03.htm](http://www.uwgi.org/guidelines/ch_03/ch03.htm)
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) <http://digestive.niddk.nih.gov/ddiseases/pubs/gerd/>
- Prelief <http://www.akpharma.com/prelief/preliefindex.html>

## HIATAL HERNIA, ESOPHAGITIS, AND GERD—CITED REFERENCES

- Anderson JW, et al. Health benefits of dietary fiber. *Nutr Rev*. 67:188, 2009.
- Blanchard C, Rothenberg ME. Basic pathogenesis of eosinophilic esophagitis. *Gastrointest Endosc Clin N Am*. 18:133, 2008.
- Demeester SR. Adenocarcinoma of the esophagus and cardia: a review of the disease and its treatment. *Ann Surg Oncol*. 13:12, 2006.
- DeVault K, Castell D. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease. *Am J Gastroenterol*. 100:190, 2005.
- Falk GW, et al. Barrett's esophagus in women: demographic features and progression to high-grade dysplasia and cancer. *Clin Gastroenterol Hepatol*. 3:1089, 2005.
- Fass R. The pathophysiological mechanisms of GERD in the obese patient. *Dig Dis Sci*. 53:2300, 2008.
- Furuta T, et al. Influences of different proton pump inhibitors on the antiplatelet function of clopidogrel in relation to CYP2C19 genotypes. *Br J Clin Pharmacol*. 70:383, 2010.
- Gupte AR, Draganov PV. Eosinophilic esophagitis. *World J Gastroenterol*. 15:17, 2009.
- Kubo A, et al. Dietary antioxidants, fruits, and vegetables and the risk of Barrett's esophagus. *Am J Gastroenterol*. 103:1614, 2008.
- Liacouras CA, et al. Eosinophilic esophagitis: a 10-year experience in 381 children. *Clin Gastroenterol Hepatol*. 3:1198, 2005.
- Liu JJ, Saltzman JR. Management of gastroesophageal reflux disease. *South Med J*. 99:735, 2006.
- Malcolm WF, et al. Transpyloric tube feeding in very low birthweight infants with suspected gastroesophageal reflux: impact on apnea and bradycardia. *J Perinatol*. 29:372, 2009.
- Moon A, et al. Positive association between helicobacter pylori and gastroesophageal reflux disease in children [published online ahead of print 9 June 2009]. *J Pediatr Gastroenterol Nutr*. 49:283, 2009.
- Saitoh T, et al. Influences of CYP2C19 polymorphism on recurrence of reflux esophagitis during proton pump inhibitor maintenance therapy. *Hepatogastroenterology*. 56:703, 2009.
- Stylopoulos N, Rattner DW. The history of hiatal hernia surgery: from Bowditch to laparoscopy. *Ann Surg*. 241:185, 2005.

## STOMACH

# DYSPEPSIA/INDIGESTION OR BEZOAR FORMATION

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2



## DEFINITIONS AND BACKGROUND

Indigestion (dyspepsia) may be secondary to other systemic disorders such as atherosclerotic heart disease, hypertension, liver disease, or renal disease. It may have psychogenic causes as well, such as during periods of anxiety. Symptoms may be graded from mild to severe for the individual patient. Gastric hypersensitivity is an important factor.

In rare cases, a patient may exhibit signs of **bezoar formation**, such as after gastric surgery or chronic use of medications that do not dissolve easily for that individual. A bezoar (foreign matter accumulation) may be made of plant material, medications, or swallowed foreign objects. The patient complains of vague abdominal pain or indigestion and may experience nausea or vomiting. An x-ray of the GI tract may be useful for a clarifying diagnosis.





## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Genetic factors in indigestion would be related to the primary disorder.

Clinical/History		Lab Work
Height	Anorexia	Gluc
Weight	Nausea or vomiting	<i>H. pylori</i>
Weight changes	Epigastric pain or burning	infection
BMI	Postprandial fullness, early satiation	H & H
Diet history	Gastric burning sensation	Serum Fe, ferritin
Bloating, heartburn, nausea	Gastric barostat tests	MCV
Burping, vomiting	Endoscopy	Alb, transthyretin
Early satiety, postprandial fullness	I & O	CRP
	Bezoar formation?	BUN, Creat
		Na <sup>+</sup> , K <sup>+</sup>
		Ca <sup>++</sup> , Mg <sup>++</sup>

## INTERVENTION



### OBJECTIVES

- Determine whether the problem is psychogenic or organic in etiology. Do not oversimplify the patient's discomfort.
- If the patient has a bezoar, alter food and beverage consistencies.
- If the patient has irritable bowel or other GI condition, work closely with the medical team to manage dietary changes and reduce excessive use of medications.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Dyspepsia

**Assessment Data:** Weight, BMI; recent GI surgery for extensive peptic ulcer disease; positive hx of *H. pylori* infection; heartburn with every meal and complaints of indigestion.

**Nutrition Diagnoses (PES):** Inadequate oral food and beverage intake related to frequent bouts of indigestion after GI surgery as evidenced by unplanned weight loss, reports of heartburn and indigestion with meals.

**Interventions:** Alter foods served to decrease use of acidic foods or stimulants such as caffeine and alcoholic beverages. Educate about simplifying meals and small snacks to increase intake. Counseling on food choices and lifestyle changes to improve intake and decrease discomfort.

**Monitoring and Evaluation:** Improved intake for meals and snacks. Fewer complaints of dyspepsia. No further weight loss. Reduced need for use of antacids for heartburn.



## FOOD AND NUTRITION

- Diet should make use of well-cooked foods that are adequate in amount but not overly seasoned.
- Evaluate for any undetected food allergies and manage accordingly.
- A relaxed atmosphere is helpful, and small meals may be better tolerated.
- If the dyspepsia is organic in etiology, a soft, low-fat diet may be helpful.
- If there is irritable bowel as well, discuss fiber from fruits and vegetables (American Dietetic Association, 2008). Bran is not always well tolerated.
- If the patient has an obstruction or bezoar, a liquid diet may be useful until resolved.

### Common Drugs Used and Potential Side Effects

- Antacids: Beware of nutritional side effects resulting from chronic use or dependency.
- Antisecretory drugs are useful (Suzuki et al, 2006). PPIs may be used, especially if reflux also exists. Lansoprazole (Prevacid), omeprazole (Prilosec), and esomeprazole (Nexium) are commonly used.
- NSAIDs are nonselective cyclooxygenase-1 (COX-1) and COX-2 inhibitors and may be associated with dyspepsia. Other anti-inflammatory medicines such as ibuprofen, aspirin, and naproxen (Aleve) can irritate the stomach. Acetaminophen (Tylenol) is a better choice for pain.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Ginger is often used as an antiemetic. Do not use large doses with warfarin, aspirin, other antiplatelet drugs, antihypertensive drugs, and hypoglycemic drugs. Additive effects can cause unpredictable changes in BP and decreases in blood glucose levels and may decrease platelet aggregation and thus increase bleeding. Ginger ale has few side effects.
- Chamomile, peppermint, red pepper, angelica, and coriander have been recommended, but no clinical trials have proven efficacy.



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage the patient to eat in a relaxed atmosphere.
- Yoga and other stress-relieving lifestyle changes may be beneficial.
- Discuss the role of fiber in maintaining bowel regularity.
- Discuss tips for preparing meals that are lower in acid, stimulants, or other irritants.

**Patient Education—Food Safety**

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the diet of the individual. Preparation and storage techniques are also essential.
- If home TF is needed, teach appropriate sanitation and food handling procedures.

**For More Information**

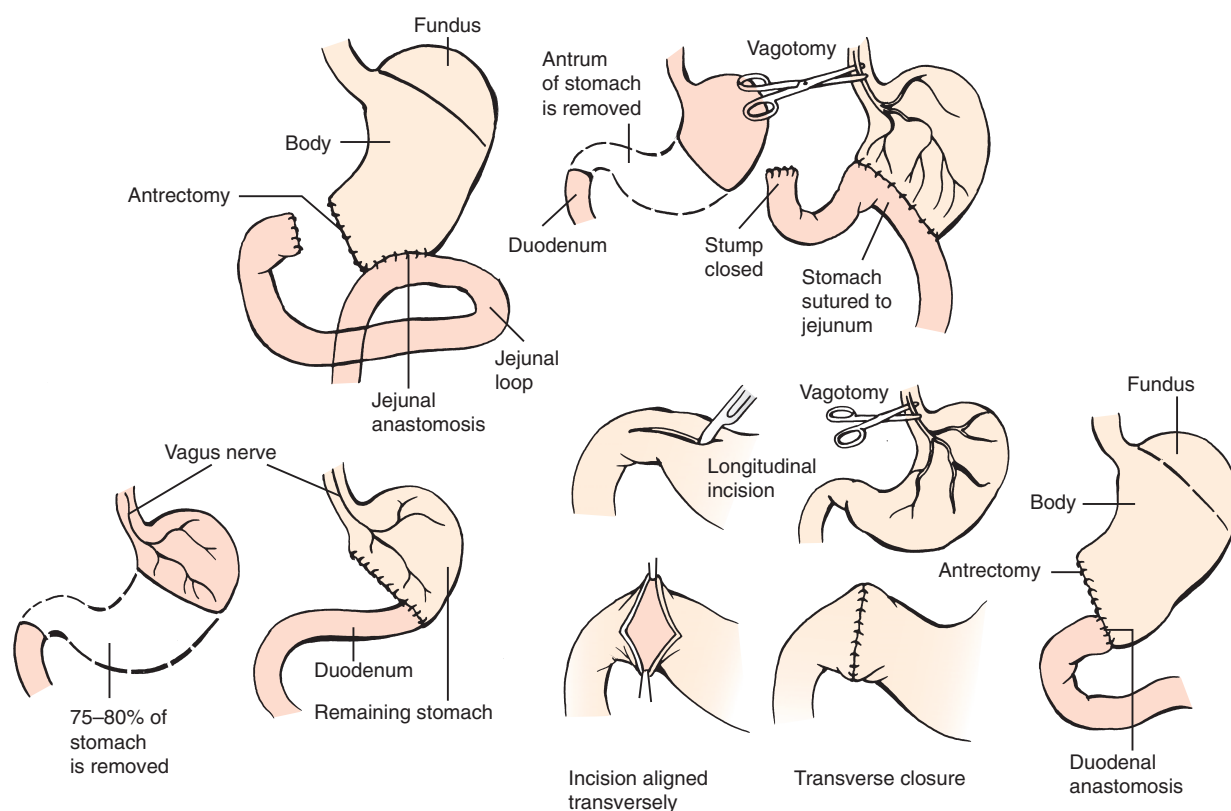
- Dyspepsia  
<http://familydoctor.org/474.xml>

- Dyspepsia Algorithm  
[http://www.uwgi.org/guidelines/ch\\_02/ch02.htm](http://www.uwgi.org/guidelines/ch_02/ch02.htm)

**DYSPEPSIA/INDIGESTION—CITED REFERENCES**

- American Dietetic Association. Position of the American Dietetic Association: health implications of dietary fiber. *J Am Diet Assoc.* 108:1716, 2008.
- Suzuki H, et al. Therapeutic strategies for functional dyspepsia and the introduction of the Rome III classification. *J Gastroenterol.* 41:513, 2006.

# GASTRECTOMY AND VAGOTOMY

**NUTRITIONAL ACUITY RANKING: LEVEL 3–4**

Adapted from: Nettina, Sandra M., MSN, RN, CS, ANP, *The Lippincott Manual of Nursing Practice*, 7th ed. Lippincott, Williams & Wilkins, 2001.

**DEFINITIONS AND BACKGROUND**

**Gastrectomy** and **vagotomy** are surgical procedures that are used for gastric cancer, when medical management for peptic ulcer has failed, or for perforation. The frequency with which elective gastric surgeries are performed has decreased in the past 20 years as drugs have become increasingly effective in treating ulcers. Laparoscopic-assisted gastrectomy is an increasingly common procedure for gastric cancer, with fewer side effects (Mochiki et al, 2005).

Billroth I (gastroduodenostomy) is an anastomosis between the stomach and duodenum after removal of the distal portion of the stomach. Billroth II (gastrojejunostomy) is an anastomosis between the stomach and jejunum after removal of two thirds to three fourths of the stomach; iron loss can occur. While the Billroth I and Billroth II operations have been used for reconstruction after a distal gastrectomy for gastric cancer, a Roux-en-Y reconstruction is increasingly performed to prevent duodenogastric reflux (Hoya et al, 2009).

Vagotomy is a procedure in which the vagus nerve is cut to reduce pain; much less nutritional intervention is required. Gastrectomy or vagotomy may lead to reactive hypoglycemia, which may drop plasma glucose levels to as low as 30–40 mg/dL due to rapid digestion and absorption of food, especially carbohydrates. Gastric emptying rate for solids may increase in some patients. In most of them, however, there is a normal to decreased emptying rate.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Gastrectomy and vagotomy are surgical procedures. Any genetic relationships would be from the original problem.

Clinical/History	Lab Work	
Height	H & H	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>
Weight	Serum Fe, ferritin	Blood guaiac
BMI	Gluc	Urine acetone
Diet history	Glucose	White blood cell (WBC) count
BP	tolerance test (GTT)	BUN
Temperature, fever	Chol, Trig	Alb, transthyretin
Electrogastrogram (EGG)	Pro-time (PT) or International Normalized Ratio (INR)	Serum amylase
Dual-energy x-ray absorptiometry (DEXA) scan		Serum B <sub>12</sub>
		Ca <sup>++</sup> , Mg <sup>++</sup>
		Serum folate
		TIBC

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Inconsistent Carbohydrate Intake

**Assessment Data:** Weight loss, frequent emesis after meals, irregular blood glucose levels with reactive hypoglycemia several times weekly.

**Nutrition Diagnoses (PES):** Inconsistent carbohydrate intake related to lack of knowledge about nutrition after gastrectomy as evidenced by weight loss, complaints of profuse sweating, diarrhea, and vomiting after meals high in carbohydrate, and blood glucose levels that vary with bouts of reactive hypoglycemia.

**Interventions:** (ND-1) Order diet that is low fat, high protein, no concentrated sweets (NCS). Educate about foods that are high in simple carbohydrate and those that are complex. Counsel on eating slowly and drinking beverages between meals.

**Monitoring and Evaluation:** Improved intake for meals and snacks; fewer complaints of dumping syndrome. No further weight loss.

## INTERVENTION



### OBJECTIVES

#### Preoperative

- Empty the stomach and upper intestines.
- Ensure high-calorie intake for glycogen stores and weight maintenance or weight gain if needed. Ensure adequate nutrient storage to promote postoperative wound healing.
- Maintain normal fluid and electrolyte balance.

#### Postoperative

- Prevent distention and pain. Reduce the likelihood of the dumping syndrome: nausea, vomiting, abdominal distention, diarrhea, malaise, profuse sweating, hypoglycemia, hypotension, increased bowel sounds, and vertigo. Additional use of soy and fermentable fiber may be useful; liquid pectin may prolong gastric emptying time and reduce the onset of dumping.
- Compensate for loss of storage/holding space and lessen dumping of large amounts of chyme into the duodenum/jejunum at one time. Overcome effects of decreased hormonal output (secretin, pancreaticozymin, and cholecystokinin).
- Overcome negative nitrogen balance after surgery; restore healthy nutritional status.
- Prevent or correct iron malabsorption; steatorrhea, calcium malabsorption, and vitamin B<sub>12</sub> or folacin anemias.
- Prevent or treat metabolic bone disease, which can occur over time.
- Prevent or treat problems such as bezoars, gastric stasis, or gastroparesis.



## FOOD AND NUTRITION

#### Preoperative

- Use a soft diet that is high in calories with adequate protein and vitamins C and K.
- Regress to soft diet with full liquids and then NPO about 8 hours before surgery.

#### Postoperative

- Within a total quantity limit, intake of complex carbohydrates such as bread, rice, and vegetables should be liberal (50–60%). To lessen the hyperosmolar load, use only 0–15% of diet from foods made with sucrose, fructose, and glucose. Initial diet may need to be 20 mL of liquid nutritional supplement every few hours, progressing as tolerated. Gastrectomy patients will be limited by the size of the remaining stomach as well.
- A protein-rich food should be consumed with each meal. Include eggs, cheese, dried beans or peas, tender meat or poultry, boneless fish, yogurt, peanut butter, nuts, tofu, and cottage cheese.
- Lactose intolerance is common in patients with these conditions; use less milk or omit if needed. Monitor calcium intake carefully.
- Use a moderate fat intake (about one third of energy intake). If needed, medium-chain triglycerides (MCTs) may be beneficial with fat maldigestion, and pancreatic enzymes may also be needed in some cases.

- The diet should also provide adequate chromium, vitamin B<sub>12</sub>, riboflavin, iron, folacin, calcium, and vitamin D. A liquid multivitamin–mineral supplement may be needed.
- If weight loss becomes a problem, a liquid supplemental beverage may be useful between meals and can be sipped throughout the day and evening.
- Diet should provide a moderate sodium intake; excess salt draws fluid into the duodenum. If there is diarrhea, losses of sodium in the stool may occur.
- Fluids should be taken 1 hour before or after meals, rather than with meals; assure adequate fluid intake overall.
- Sit upright while eating. Encourage slow eating and adequate chewing for all meals and snacks.
- Diet should provide frequent, small meals. Avoid extremes in food temperature.

### Common Drugs Used and Potential Side Effects

- Antibiotics may be used to control bacterial overgrowth.
- Antidiarrheals such as Kaopectate and loperamide may be useful. Dry mouth, nausea, vomiting, and bloating may occur. Use plenty of fluids.
- For reactive hypoglycemia, use of an alpha-glucosidase inhibitor, acarbose, may be beneficial. GI side effects are common.
- Pancreatic enzymes may be useful; the usual dose provides two to three capsules with meals.
- If vitamin B<sub>12</sub> deficiency occurs, shots may be needed.
- If bone density loss occurs, the use of calcium, vitamin D, or bisphosphonates may be prescribed, and these should be taken as directed.

### Herbs, Botanicals, and Supplements

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



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Stress the importance of self-care and optimal functioning—what to do for illness, episodes of vomiting, eating away from home, and how to read labels for carbohydrate (CHO) content.
- Discuss the use of artificial sweeteners.
- Instruct the patient to eat slowly in an upright position and to remain upright for awhile after meals.
- Help the patient to overcome reluctance and the fear of eating. Discuss the dumping syndrome and its effects on nutrient absorption if untreated.

### Patient Education—Foodborne Illness

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

### For More Information

- Anti-Dumping Syndrome Diet  
<http://www.gicare.com/Diets/Dumping.aspx>
- Gastrectomy—Surgical Channel  
<http://www.surgerychannel.com/gastrectomy/index.shtml>
- Medline  
<http://www.nlm.nih.gov/medlineplus/ency/article/002945.htm>

### GASTRECTOMY AND VAGOTOMY—CITED REFERENCES

- Hoya Y, et al. The advantages and disadvantages of a Roux-en-Y reconstruction after a distal gastrectomy for gastric cancer. *Surg Today*. 39:647, 2009.
- Mochiki E, et al. Laparoscopic assisted distal gastrectomy for early gastric cancer: Five years' experience. *Surgery*. 137:317, 2005.

## GASTRITIS AND GASTROENTERITIS

### NUTRITIONAL ACUITY RANKING: LEVEL 3



### DEFINITIONS AND BACKGROUND

**Gastritis** involves inflammation of the stomach. Types of gastritis include bacterial (from *H. pylori*), autoimmune gastritis with pernicious anemia, erosive gastritis (from aspirin or NSAID use), alcohol-induced gastritis, bile reflux gastritis, or atrophic gastritis. The treatment of gastritis will depend on its cause; reduction of stomach acid by medication is often most helpful.

*Helicobacter pylori* infects half the world's population, causing **chronic gastritis** (Shanks and El-Omar, 2009). Bacterial, environmental and host genetic factors combine to define the degree of gastric damage in gastritis (Shanks and El-Omar, 2009). **Hemorrhagic gastritis** may result from chronic intake of alcohol or medications, Crohn's disease or HIV

infection. **Atrophic gastritis** is chronic inflammation of the gastric mucosa without erosion but with hypochlorhydria or achlorhydria; it is important to monitor vitamin B<sub>12</sub>, calcium, and ferric iron intake.

**Gastroenteritis (GE)** is an inflammation of the stomach and intestinal lining that may occur from eating chemical toxins in food (such as seafood, mushrooms, arsenic, or lead), drinking excessive alcohol, foodborne illness and viruses, cathartics or other drugs. GE produces malaise, nausea, vomiting, intestinal rumbles, diarrhea with or without blood and mucus, fever and prostration.

Viral gastroenteritis is contagious. Many different viruses can cause gastroenteritis, including rotaviruses or adenoviruses. Norovirus infection is associated with 90% of nonbacterial acute gastroenteritis (Sala et al, 2005).



Contaminated shellfish and raw oysters are major contributors. People who get viral gastroenteritis almost always recover completely if they consume adequate fluids to replace what they lose through vomiting or diarrhea.

Because the intestinal epithelium constitutes the largest and most important barrier against intraluminal toxins, antigens, and enteric flora, its dysfunction is a major factor contributing to the predisposition to inflammatory diseases (Groschwitz and Hogan, 2009). Post-infectious irritable bowel syndrome (PI-IBS) is a disorder where symptoms begin after an episode of acute GE with persistent subclinical inflammation, changes in intestinal permeability and alteration of gut flora (Thabane and Marshall, 2009). Some children acquire functional GI disorders after an episode of acute bacterial gastroenteritis (AGE) from *Salmonella* (54%), *Campylobacter*, or *Shigella* (Saps et al, 2008). Other issues related to intestinal dysfunction include food allergy, IBD, and celiac disease (Groschwitz and Hogan, 2009).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Gastroenteritis is acquired and not genetic. Host genetic factors define the severity and extent of *Helicobacter*-induced gastritis; interleukin-1 and TNF- $\alpha$  gene clusters are involved (Shanks and El-Omar, 2009).

Clinical/History	Barium swallow study	MCV
Height	Upper GI	Serum folate
Weight	endoscopy	Serum B <sub>12</sub>
BMI	Gastric biopsy	Gluc
Diet history	Diarrhea	Alb,
Anorexia, nausea	Stool culture	transferrin
Upset stomach	Blood in stool?	Ca <sup>++</sup> , Mg <sup>++</sup>
Hiccups		Hydrogen
I & O		breath test
Signs of	<b>Lab Work</b>	BUN, creatinine
dehydration?	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>	
Fever?	H & H	
Esophageal	Serum Fe,	
manometry	ferritin	

## INTERVENTION



### OBJECTIVES

- Prevent or correct dehydration, shock, hypokalemia, and hyponatremia.
- If hemorrhage occurs, consider a medical emergency.
- Allow the stomach and GI tract to rest. Empty stomach to permit mucous lining to heal.
- Omit lactose if not tolerated during bouts of gastroenteritis.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Abnormal GI Function—Diarrhea

**Assessment Data:** Diarrhea, GI pain, slight fever; recent intake of undercooked beef and sushi while traveling.

**Nutrition Diagnoses (PES):** Altered GI function (NC-1.4) with foodborne illness as evidenced by diarrhea, slight fever (100°F) for 7 days and stool culture positive for *Escherichia coli* O157:H7.

**Interventions:** Alter diet as tolerated; soft diet or liquids may be accepted. Provide oral rehydration products. Educate about foodborne illness related to *E. coli* O157:H7 including food sources. Counseling about safe food-handling procedures and better choices at restaurants while traveling.

**Monitoring and Evaluation:** Resolution of diarrhea. Stool cultures free from *E. coli* O157:H7. Improved knowledge about food safety as documented by correct responses to questions.



## FOOD AND NUTRITION

- **Gastritis:** Omit foods that are poorly tolerated. Provide adequate hydration. If chronic, mucosal atrophy can lead to nutritional deficits (e.g., pernicious anemia, achlorhydria). Alter diet accordingly.
- **Acute gastroenteritis:** Patient will be NPO or on partial parenteral nutrition (PPN) for the first 24–48 hours to rest stomach. Use crushed ice to relieve thirst. Oral rehydration therapy may be useful. Progress to a soft diet, if desired. Alcohol is prohibited. Omit lactose if needed. Gradually add fiber-containing foods as tolerance improves. Rehydration solutions may be effective.
- **Chronic gastritis:** Use small, frequent feedings of easily tolerated foods. Progress with larger amounts and greater variety of foods, as tolerated. Restrict fat intake, which depresses food motility, and alcohol intake. Monitor lactose intolerance. Add fiber-containing foods as tolerated.

## Common Drugs Used and Potential Side Effects

- Antacids: Watch for constipation caused by aluminum and calcium agents. Watch for diarrhea caused by magnesium agents.
- Antibiotics are used for infection. If used in excess over a long period of time, they may cause or aggravate gastroenteritis. Monitor carefully and suggest use of probiotics such as yogurt with live and active cultures.
- Sucralfate may be useful. Take separately from calcium or magnesium supplements by 30 minutes. Constipation may occur.
- Early postoperative medication with a PPI is effective in preventing gastritis after open-heart surgery (Hata et al, 2005). Lansoprazole (Prevacid), omeprazole (Prilosec), and esomeprazole (Nexium) are commonly used.
- Eliminate use of aspirin and other agents that may aggravate gastritis.
- If the gastritis is caused by pernicious anemia, B<sub>12</sub> vitamin shots will be given.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician. Products such as ginger and ginger ale may alleviate some nausea.
- The Polynesian traditional food, poi, is a starchy paste made from taro plants (Brown et al, 2005). It may be useful as a probiotic.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Omit offenders in chronic conditions: alcohol, caffeine, and aspirin.
- Patients with chronic gastritis should be assessed for folate and vitamin B<sub>12</sub> status. Atrophy of the stomach and intestinal lining interferes with folate and vitamin B<sub>12</sub> absorption.
- Discuss calcium and riboflavin food sources if dairy products must be omitted.
- Discuss the role of fiber in achieving or maintaining bowel integrity.
- Discuss foodborne illness and its prevention (e.g., avoiding raw shellfish).
- Oral rehydration therapy (ORT) is recommended as first-line therapy for both mildly and moderately dehydrated children with gastroenteritis (Spandorfer et al, 2005).

### Patient Education—Foodborne Illness

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

- Careful food handling will be important. To prevent gastroenteritis, cook all foods to proper temperatures; wash all produce before cutting or eating; use careful hand washing.

### For More Information

- Gastritis  
<http://digestive.niddk.nih.gov/ddiseases/pubs/gastritis/>
- Gastroenteritis  
<http://www.cdc.gov/ncidod/dvrd/revb/gastro/faq.htm>
- Merck manual  
<http://www.merck.com/mmpe/sec02/ch013/ch013c.html>

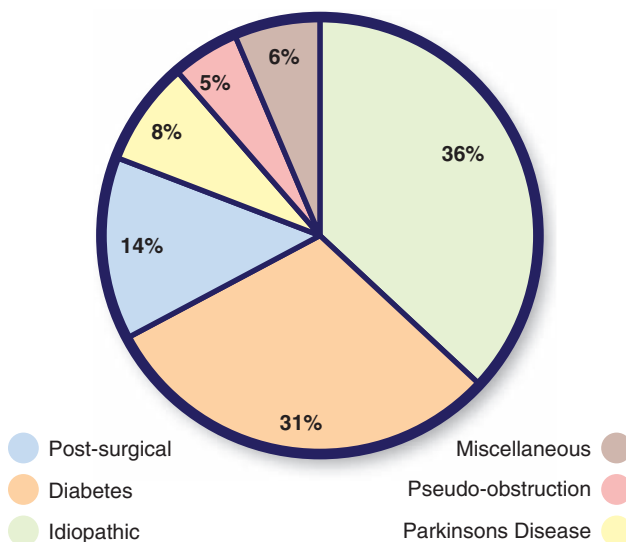
## GASTRITIS AND GASTROENTERITIS—CITED REFERENCES

- Brown AC, et al. A non-dairy probiotic's (poi) influence on changing the gastrointestinal tract's microflora environment. *Altern Ther Health Med*. 11:58, 2005.
- Groschwitz KR, Hogan SP. Intestinal barrier function: molecular regulation and disease pathogenesis. *J Allergy Clin Immunol*. 124:3, 2009.
- Hata M, et al. Prospective randomized trial for optimal prophylactic treatment of the upper gastrointestinal complications after open heart surgery. *Circ J*. 69:331, 2005.
- Sala MR, et al. An outbreak of food poisoning due to a genogroup I norovirus. *Epidemiol Infect*. 133:187, 2005.
- Saps M, et al. Post-infectious functional gastrointestinal disorders in children. *J Pediatr*. 152:812, 2008.
- Shanks AM, El-Omar EM. *Helicobacter pylori* infection, host genetics and gastric cancer. *J Dig Dis*. 10:157, 2009.
- Spandorfer PR, et al. Oral versus intravenous rehydration of moderately dehydrated children: a randomized, controlled trial. *Pediatrics*. 115:295, 2005.
- Thabane M, Marshall JK. Post-infectious irritable bowel syndrome. *World J Gastroenterol*. 15:3591, 2009.

# GASTROPARESIS AND GASTRIC RETENTION

## NUTRITIONAL ACUITY RANKING: LEVEL 2–3

Etiologies of Gastroparesis



## DEFINITIONS AND BACKGROUND

Phases of normal digestion include: phase I (45–60 minutes of inactivity), phase II (30–45 minutes of intermittent peristaltic contractions), phase III (10 minutes of intense, regular contractions), and phase IV (brief transition between cycles). **Gastric retention** is caused by a partial obstruction at the outlet of the stomach into the small bowel. Gastric retention may result from diabetes, prolonged hyperglycemia, vagal autonomic neuropathy, scleroderma, Parkinson's disease, hypothyroidism, postviral syndromes, gastric surgery, or vascular insufficiencies.

**Gastroparesis** is a chronic disorder of gastric motility that is characterized by delayed emptying of either solids or liquids from the stomach in the absence of any mechanical obstruction; diabetes mellitus and postsurgical states cause the majority of these problems. Complications include ketoacidosis, infection, and bezoar formation (Feigenbaum, 2006). Bezoars further obstruct the flow from the stomach to the small intestine. Gastroparesis may occur as a

complication of end-stage liver disease and portal hypertension. Idiopathic gastroparesis is characterized by severely delayed gastric emptying of solids without obvious underlying organic cause (Karamanolis et al, 2007).

Because ghrelin is produced by enteroendocrine cells in the gastric mucosa, vagal function and regulation of ghrelin are impaired in gastroparesis (Gaddipati et al, 2006; Levin et al, 2006). Severe gastroparesis can result in recurrent hospitalizations, malnutrition, and even death (McKenna et al, 2008).

Gastroparesis can be difficult to treat. Prokinetic drugs are commonly used. Treatment may involve use of an implantable gastric electrical stimulation (GES) device or endoscopic botulinum toxin injection (Gumaste and Baum, 2008; Monnikes and van der Voort, 2006; Vittal and Pasricha, 2006). The surgical procedure has been found to be effective in reducing symptoms within 6 weeks (McKenna et al, 2008).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** No specific genetic disorder predicts the onset of gastric retention or gastroparesis.

<b>Clinical/History</b>	Flatulence	H & H
Height	Early satiety	BUN, Creat
Weight	Gastric x-rays–	Alb,
Weight changes	EGG	transferrin
BMI	Gastric emptying	CRP
Diet history	test (slow	Gastrin
I & O	emptying of	Ghrelin levels
Nausea,	liquids	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>
vomiting	and/or solids)	Ca <sup>++</sup> , Mg <sup>++</sup>
Abdominal pain	<b>Lab Work</b>	Serum folate
after eating		and B <sub>12</sub>
Heartburn	Gluc (poorly con-	
Belching,	trolled, over	
bloating	200 mg/dL?)	

## INTERVENTION



### OBJECTIVES

- Decrease volume of meals served. Use liquids or foods that liquefy at body temperature so they are able to pass by a partial obstruction before or during digestion. Bypass or correct obstruction or other causes of retention.
- If diabetes is present, manage control of blood glucose. Differentiate from ketoacidosis, which has similar symptoms of nausea and vomiting. Pernicious vomiting may occur; distinguish from bulimia.
- Correct dehydration and electrolyte abnormalities.
- Reduce or control pain, diarrhea, or bouts of constipation.
- Ensure adequate intake of diet as prescribed to prevent weight loss and control malnutrition.
- Prevent or correct bezoar formation of indigestible solids.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Phytobezoar

**Assessment Data:** Weight loss. Abdominal pain. Swallowing evaluation showing gastroparesis and GI obstruction from phytobezoar.

**Nutrition Diagnoses (PES):** Altered GI function (NC-1.4) related to phytobezoar as evidenced by delayed gastric emptying, abdominal pain and vomiting, and labs showing glucose levels of 250–300 mg/dL.

**Interventions:** Food and Nutrient Delivery: Use a liquid, low residue diet until bezoar is resolved. Educate: Blending foods to improve intake. Coordinate care: insulin as needed to improve serum glucose control; try doses of coca cola to dissolve phytobezoar.

**Monitoring and Evaluation:** Improved intake for meals and snacks. Improved glucose management. No further abdominal pain or vomiting. Resolution of phytobezoar.

- Monitor use of, or avoid where possible, medications that cause gastric stasis.



## FOOD AND NUTRITION

- A soft-to-liquid diet lower in fat may be useful to prevent delay in gastric emptying. Isotonic liquids empty more quickly than hypertonic liquids.
- Six small meals may be better tolerated than large meals.
- Calculate protein and energy requirements according to underlying medical condition(s).
- Alter fiber intake according to needs (more to alleviate diarrhea, constipation; less with a history of bezoar formation).
- If patient complains of dry mouth, add extra fluids and moisten foods with broth or allowed sauces or gravies.
- For patients with a lesser obstruction of the stomach, progress to a mechanical soft diet.
- For patients with greater obstruction of the stomach, use a low-fiber diet or tube feed, checking residuals frequently.
- A jejunostomy TF may be indicated for persistent problems, even temporarily if needed to correct malnutrition. Consider if there is a history of significant weight loss, cyclical nausea and vomiting, or repeated hospitalizations for gastroparesis.
- Ensure that the patient sits upright during meals.

## Common Drugs Used and Potential Side Effects

- Medications that may cause or aggravate gastric emptying include: alcohol, antacids containing aluminum, anticholinergics, calcitonin, calcium channel blockers, glucagon, interleukin-1, levodopa, lithium, octreotide, narcotics, nicotine, potassium salts, progesterone, sucralfate, tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs) including Celexa, Paxil, Prozac, and Zoloft.
- Dopamine antagonists, such as metoclopramide and domperidone, and the motilin receptor agonist erythromycin have been the cornerstones in drug treatment of severe gastroparesis for more than a decade (Abrahamsson, 2007).

These drugs are less than ideal (Gumaste and Baum, 2008). Give metoclopramide (Reglan) 30 minutes before meals to increase gastric contractions and to relax the pyloric sphincter. Dry mouth, sleepiness, anxiety, and nausea can be side effects.

- If there is hyperglycemia, oral agents or insulin may be needed.
- Ghrelin receptor agonists may have a role as prokinetic agents (Levin et al, 2006).

### Herbs, Botanicals, and Supplements

- Products such as ginger and ginger ale may alleviate some nausea. Use of herbal remedies is common in the Hispanic population. Herbs and botanical supplements should not be used without discussing with the physician.
- *Cuminum cyminum* is widely used in Ayurvedic medicine for the treatment of dyspepsia.
- Adolph's Meat Tenderizer (1 teaspoonful in 8 oz of water before each meal for 7 days) provides papain, a proteolytic enzyme that is a safe treatment for bezoars (Baker et al, 2007). Other studies suggest the use of coca cola.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help the patient determine a specific dietary regimen. If there is diabetes, optimize glycemic control.
- Discuss methods for liquefying foods as needed.
- Bezoar formation may occur after eating oranges, coconuts, green beans, apples, figs, potato skins, Brussels sprouts, broccoli, and sauerkraut in a vulnerable individual.

### Patient Education—Foodborne Illness

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

### For More Information

- Association of Gastrointestinal Motility Disorders, Inc. <http://www.agmd-gimotility.org>
- Cyclic Vomiting Association <http://www.cvsaonline.org>
- Gastroparesis and Dysmotilities Association <http://gpda.net>
- International Foundation for Functional Gastrointestinal Disorders <http://www.iffgd.org>

### GASTROPARESIS AND GASTRIC RETENTION—CITED REFERENCES

- Abrahamsson H. Severe gastroparesis: new treatment alternatives. *Best Pract Res Clin Gastroenterol*. 21:645, 2007.
- Baker EL, et al. Resolution of a phytobezoar with Adolph's Meat Tenderizer. *Pharmacotherapy*. 27:299, 2007.
- Feigenbaum K. Update on gastroparesis. *Gastroenterol Nurs*. 29:239, 2006.
- Gaddipati KV, et al. Abnormal ghrelin and pancreatic polypeptide responses in gastroparesis. *Dig Dis Sci*. 51:1339, 2006.
- Gumaste V, Baum J. Treatment of gastroparesis: an update. *Digestion*. 78:173, 2008.
- Karamanolis G, et al. Determinants of symptom pattern in idiopathic severely delayed gastric emptying: gastric emptying rate of proximal stomach dysfunction. *Gut*. 56:29, 2007.
- Levin F, et al. Ghrelin stimulates gastric emptying and hunger in normal weight humans. *J Clin Endocrinol Metab*. 91:3296, 2006.
- McKenna D, et al. Gastric electrical stimulation is an effective and safe treatment for medically refractory gastroparesis. *Surgery*. 144:566, 2008.
- Monnikes H, Van Der Voort IR. Gastric electrical stimulation in gastroparesis: where do we stand? *Dig Dis*. 24:260, 2006.
- Vittal H, Pasricha PF. Botulinum toxin for gastrointestinal disorders: therapy and mechanisms. *Neurotox Res*. 9:149, 2006.

## GIANT HYPERTROPHIC GASTRITIS AND MÉNÉTRIER'S DISEASE

### NUTRITIONAL ACUITY RANKING: LEVEL 3



Adapted from: Ronald L. Eisenberg, *An Atlas of Differential Diagnosis*. 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2003.



### DEFINITIONS AND BACKGROUND

**Giant hypertrophic gastritis (GHG)** is a pathological condition with increased loss of plasma proteins, resulting in hydrolysis by the proteolytic enzymes of the gut. The hydrolyzed proteins are then reabsorbed as amino acids. Ascites or edema occur if the liver cannot produce sufficient albumin rapidly enough. The condition may precede stomach cancer. The disease is rare and diagnosed in patients with giant gastric folds, dyspeptic symptoms, and hypoalbuminemia due to GI protein loss.

**Ménétrier's disease** is a form of hyperplastic gastropathy and not a form of gastritis because inflammation is minimal. Overexpression of transforming growth factor (TGF)-alpha results in selective expansion of surface mucous cells in the body and fundus of the stomach. Ménétrier's disease is often associated with *H. pylori* infection. Most patients with



Ménétrier's disease are treated nonoperatively with nutritional support, antacids, and pain medications (Sanchez et al, 2007). Gastric resection provides permanent relief if needed.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** The *H. pylori* strain with Ménétrier's disease has high hepatocyte growth factor (HGF) and TNF-alpha mRNA expressions from gastric fibroblasts (Ishikawa et al, 2008).

<b>Clinical/History</b>	Fecal occult blood test	CRP
Height	Stearrhea	Gluc
Weight	Generalized edema	Na <sup>+</sup> , K <sup>+</sup>
BMI	Stomach ulcers?	Ca <sup>++</sup> , Mg <sup>++</sup>
Diet history		Nitrogen (N) balance
Weight changes		Transferrin
Abdominal pain	<b>Lab Work</b>	H & H
Blood in vomit	Pepsin levels	Serum Fe, ferritin
Gastroscopy	<i>H. pylori</i> bacteria	Serum folate and B <sub>12</sub>
Gastric biopsy	Alb, transthyretin	BUN, Creat
Abdominal ultrasound	Globulin	
	A:G ratio	

## INTERVENTION



### OBJECTIVES

- Replace protein; maintain adequate nitrogen balance.
- Reduce edema.
- Spare protein for tissue synthesis and repair.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Inadequate Protein Intake

**Assessment Data:** Diagnosis of GHG with giant gastric folds, dyspepsia, hypoalbuminemia (serum levels <2.0).

**Nutrition Diagnoses (PES):** Inadequate protein intake (NI-52.1) related to GI protein loss as evidenced by serum albumin <2.0 g/dL.

**Interventions:** Alter diet to increase protein intake orally, or tube feed if necessary. Educate patient and family about the role of protein in alleviating edema and correcting low albumin levels.

**Monitoring and Evaluation:** Improvements in serum albumin from diet or transfusion (if needed). Transthyretin levels showing improvement over several weeks. Resolution of edema and improved quality of life.

- Promote normal dietary intake with a return to wellness.
- Delay or prevent onset of stomach cancer if possible.



## FOOD AND NUTRITION

- Use a high-protein/high-calorie diet. The protein level should be approximately 20% of total kilocal unless contraindicated for renal or hepatic problems.
- Omit any food intolerances.
- Include adequate sources of micronutrients in the diet; a basic supplement may be warranted.

### Common Drugs Used and Potential Side Effects

- Ménétrier disease patients have been effectively treated with a specific blocking monoclonal antibody (Coffey et al, 2007). This reduces the frequency of nausea and vomiting, improves serum albumin concentration, and improves abnormalities of the stomach.
- For eradication of *H. pylori*, 2 weeks of treatment with an acid-suppressing drug (one time daily), Pepto-Bismol (four times daily), and antibiotics (three to four times daily) are prescribed. This therapy often must be used more than once. Other combinations may include the antibiotics omeprazole, clarithromycin, and ranitidine bismuth (Tritec).
- Lansoprazole (Prevacid), omeprazole (Prilosec), and esomeprazole (Nexium) may be prescribed.
- If prednisone is used for a long period of time, monitor for changes in glucose levels.

### Herbs, Botanicals, and Supplements

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



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Eliminate aggravating foods specific to the patient.
- Teach the patient about use of high biological value (HBV) proteins to replenish protein levels from GI losses.
- During recovery, the use of probiotics such as yogurt with live and active cultures may be helpful.

### Patient Education—Foodborne Illness

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

### For More Information

- Giant hypertrophic gastritis  
[http://www.cancer.gov/templates/db\\_alpha.aspx?CdrID=589414](http://www.cancer.gov/templates/db_alpha.aspx?CdrID=589414)
- Medical terms on line  
<http://www.medicaltermsonline.org/index.php?section=pages&item=Giant-hypertrophic-gastritis>

## GIANT HYPERTROPHIC GASTRITIS AND MÉNÉTRIER'S DISEASE—CITED REFERENCES

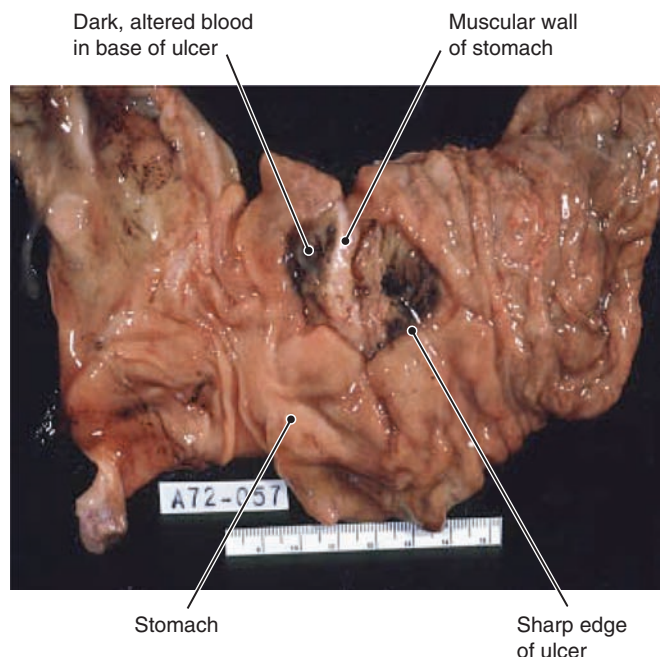
Coffey RJ, et al. Ménétrier disease and gastrointestinal stromal tumors: hyperproliferative disorders of the stomach. *J Clin Invest*. 117:70, 2007.

Ishikawa T, et al. *Helicobacter pylori* isolated from a patient with Ménétrier's disease increases hepatocyte growth factor mRNA expression in gastric fibroblasts: comparison with *Helicobacter pylori* isolated from other gastric diseases. *Dig Dis Sci*. 53:1785, 2008.

Sanchez C, et al. Laparoscopic total gastrectomy for Ménétrier's disease. *J Laparoendosc Adv Surg Tech A*. 17:32, 2007.

# PEPTIC ULCER DISEASE

## NUTRITIONAL ACUITY RANKING: LEVEL 2



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



## DEFINITIONS AND BACKGROUND

A peptic ulcer suggests an imbalance between digestive fluids in the stomach and duodenum, with erosion by gastric acid and pepsin and exposed nerves. Most ulcers are duodenal, within the first 25–30 cm.

One of 10 Americans suffers from peptic ulcer disease. *H. pylori* bacteria play a role in the etiology of 75% or more of peptic ulcers. *Helicobacter pylori* can be transmitted from person to person through close contact, exposure to vomit, or fecal–oral contamination. It can also be found in well water. Because the bacterium is one of the most genetically diverse bacterial species, more than half of the world population in both developed and developing countries are infected (Dube et al, 2009). It has been implicated in stomach cancer.

Hand washing is an important preventive measure. In addition, sulforaphane (SF) from broccoli is a powerful bactericidal agent against *H. pylori* (Yanaka et al, 2009) and intake of soy products may help reduce the effects of inflammation-related IL-10 genetic polymorphisms (Ko et al, 2009).

Individuals who have cirrhosis, chronic obstructive pulmonary disease, renal failure, and organ transplantation

tend to have a higher risk for peptic ulcer disease (PUD). Bland diets neither heal ulcers nor cause a decrease in gastric acid secretion. Drug therapy is most effective in preventing ulcer recurrence and primarily consists of antibiotics and antacids. A vaccine to prevent *H. pylori* infection is being developed. In the meantime, vitamin B<sub>12</sub> tends to be lower in patients who have peptic ulcers; anemia should be monitored.

The decline in duodenal ulcer disease and the established relation of peptic ulcer to *H. pylori* have eliminated the need for elective ulcer surgery. Options for refractory and complicated PUD include vagotomy and pyloroplasty, vagotomy and antrectomy with gastroduodenal reconstruction (Billroth I) or gastrojejunal reconstruction (Billroth II).



## ASSESSMENT, MONITORING, AND EVALUATION



## CLINICAL INDICATORS

**Genetic Markers:** *H. pylori* infection tends to run in families. This pathogen has been shown to follow the routes of human migration; the global *H. pylori* population has been divided into six ancestral populations, three from Africa, two from Asia, and one from Europe (Tay et al, 2009). In addition, the interleukin 1B gene has been identified as a factor, especially inflammation-related IL-10 genetic polymorphisms.

### Clinical/History

Height  
Weight  
BMI  
Diet history  
Weight and appetite changes  
Sharp and sudden abdominal pain  
Burning or gnawing pain (better with meals but returns)

### GI bleeding or

black, tarry stools  
Nausea, vomiting  
Frequent bloating  
Chronic idiopathic urticaria or atopic dermatitis  
Stool test for *H. pylori*  
Endoscopy

### Lab Work

Red blood cell (RBC) count  
Anti-*H. pylori* immunoglobulin G antibody titer  
C-Urea breath test for *H. pylori*  
Chol, Trig  
BUN, Creat  
Alb, transthyretin

Alanine amino-transferase (ALT)	perforated ulcers)	Serum folate PT or INR
Aspartate aminotransferase (AST)	Serum gastrin (increased)	Transferrin
Blood guaiac	Alk phos (increased)	Ca <sup>++</sup> , Mg <sup>++</sup>
Amylase (increased in	H & H	Serum B <sub>12</sub>
	Serum Fe, ferritin	TIBC
		Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>
		Ca <sup>++</sup> , Mg <sup>++</sup>

## INTERVENTION



### OBJECTIVES

- Eradicate any *H. pylori* infection where present. Take medications as directed. Rest during healing stages.
- Reduce pain. Avoid distention from large meals.
- Dilute stomach contents and provide buffering action.
- Correct anemia, if present. Vitamin B<sub>12</sub> deficiency may be corrected after effective *H. pylori* treatment.
- Monitor and prevent steatorrhea, bone disease, dumping syndrome, and complications such as perforation and obstruction.



### FOOD AND NUTRITION

- Use small feedings, frequently if preferred. Include high-protein foods and vitamin C to speed healing.
- Avoid personal intolerances. Citrus and acidic juices may cause pain during exacerbations. If a particular food bothers an individual, it should be avoided.
- Use broccoli and cruciferous vegetables often to enhance chemoprotection of the gastric mucosa against *H. pylori*-induced oxidative stress (Yanaka et al, 2009).
- Encourage soybean intake (Ko et al, 2009).
- Limit gastric stimulants if not tolerated, such as caffeine, alcohol, peppermint, black pepper, garlic, cloves, and chili powder. This is a “liberal bland” diet. See Table 7-7 regarding caffeine in beverages and medications.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Undesirable Food Choices

**Assessment:** Food and symptoms diary. Positive stool guaiac test. Altered GI lab results. No *H. pylori* present.

**Nutrition Diagnosis (PES):** Undesirable food choices related to chronic alcohol intake (beer and wine, 10 drinks/wk) as evidenced by nausea, sharp stomach pains, abdominal discomfort, black tarry stools and altered GI labs.

**Intervention:** Teach about role of alcohol in GI mucosal damage. Counsel about alternative lifestyle changes that will help alleviate GI pain. Encourage intake of broccoli for its chemoprotective effects.

**Monitoring and Evaluation:** Report of decreased alcohol consumption and less GI discomfort and pain. Resolution of peptic ulcer symptoms; improved lab results. No further tarry stools.

**TABLE 7-7 Typical Caffeine Content of Beverages and Medications**

Beverages/Medications	Measure	Caffeine (mg)
Coffee, brewed	5 oz	65–120 (average, 85)
Coffee, instant	5 oz	60–85 (average, 75)
Coffee, Starbucks Frappuccino	8 oz	83
Espresso coffee	1 oz	30–50 (average, 40)
Decaffeinated coffee	5 oz	2–4
Black tea, brewed (most U.S. brands)	5 oz	20–50
Black tea, brewed (imported)	5 oz	25–60
Tea, instant	6 oz	28–30
Mountain Dew	12 oz	54
Cola drinks	12 oz	36–47
Coffee ice cream	4 oz	28
Baker's chocolate	1 oz	26
Dark chocolate	1 oz	5–35 (average, 20)
Milk chocolate	1 oz	1–15 (average, 6)
Cocoa beverage	8 oz	3–32 (average, 6)
Chocolate milk	8 oz	2–7 (average, 5)
Analgesic	1 tablet	30–66
Cold preparation	1 tablet	30
Chocolate syrup	1 oz	4
7-Up or Sprite	12 oz	0
Ovaltine	8 oz	0

Data from Leonard T, et al. The effects of caffeine on various body systems: a review. *J Am Diet Assoc.* 87:1048, 1987.

- Use fewer saturated fats and more polyunsaturated fats if increased lipid levels are found. AA metabolites may play a role in peptic ulcer disease.
- Monitor water supply as a potential source of *H. pylori*.

## Common Drugs Used and Potential Side Effects (see Table 7-8)

- Most patients with PUD should avoid NSAIDs. Analgesics and corticosteroids, when taken over a long time, may cause GI bleeding and ulceration and should be taken with food. High doses of Advil or Motrin (ibuprofen), even for a few days, can significantly increase the risk of GI bleeding.
- For eradication of *H. pylori*, 2 weeks of treatment with an acid-suppressing drug (once daily), Pepto-Bismol (four times daily), and antibiotics (three to four times daily) are prescribed. A 1- to 2-week course of *H. pylori* eradication therapy is an effective treatment (Ford et al, 2006); triple therapy often must be used more than once. Quadruple therapy is also being tested (Feng et al, 2005). Some FDA-approved combinations include the antibiotics omeprazole, clarithromycin, and ranitidine bismuth (Tritec). Other antibiotics include amoxicillin, metronidazole, and tetracycline. Suggest use of probiotics, such as yogurt with live and active cultures.

**TABLE 7-8 Medications Used in Peptic Ulcer Disease**

Medication Type	Description	Specific Drugs
Antacids	<p>Aluminum-containing and magnesium-containing antacids can be helpful in relieving symptoms of gastritis by neutralizing gastric acids. These agents are inexpensive and safe.</p> <p>Aluminum ions inhibit smooth muscle contraction, thus inhibiting gastric emptying. Use aluminum-containing antacids cautiously with upper GI hemorrhage.</p> <p>Magnesium and aluminum antacid mixtures are used to avoid bowel function changes.</p>	<p>Gaviscon contains magnesium as well as aluminum and may decrease absorption of thiamine, phosphate, and vitamin A.</p> <p>Gelusil contains magnesium, aluminum, and simethicone; it may have side effects similar to those of Gaviscon.</p> <p>Mylanta and Amphogel (aluminum hydroxide) may cause nausea, vomiting, and lowered vitamin A, calcium, and phosphate absorption. Take between meals, followed by water.</p> <p>Milk of magnesia (magnesium hydroxide) is a laxative-antacid and can deplete phosphorus and calcium over time.</p> <p>Magaldrate (Riopan) decreases serum vitamin A but can be used on a low-sodium diet.</p>
H <sub>2</sub> -receptor antagonists	<p>These drugs inhibit the action of histamine on the parietal cell, which inhibits acid secretion. The drugs in this class are all equally effective and are available over the counter in half prescription strength for heartburn treatment.</p> <p>Histamine H<sub>2</sub> blockers should be taken with food.</p> <p>Since acid secretion and ulcer pain are most prevalent at night, taking Zantac or Tagamet before bed may be helpful.</p> <p>These drugs can elevate AST/ALT and creatinine, cause confusion in elderly individuals, and cause diarrhea, constipation, or urticaria.</p>	<p>Cimetidine (Tagamet) inhibits histamine at H<sub>2</sub> receptors of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and hydrogen ion concentrations.</p> <p>Famotidine (Pepcid) competitively inhibits histamine at the H<sub>2</sub> receptor of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations.</p> <p>Nizatidine (Axid) competitively inhibits histamine at H<sub>2</sub> receptors of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations.</p> <p>Ranitidine (Zantac) competitively inhibits histamine at the H<sub>2</sub> receptors of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and hydrogen concentrations. Ranitidine can cause nausea, constipation, and vitamin B<sub>12</sub> malabsorption; may alter serum levels of serum iron.</p>
Proton pump inhibitors (PPIs)	<p>PPIs bind to the proton pump of parietal cell, inhibiting secretion of hydrogen ions into gastric lumen.</p> <p>PPIs relieve pain and heal peptic ulcers more rapidly than H<sub>2</sub> antagonists do. Drugs in this class are equally effective.</p> <p>All PPIs decrease serum concentrations of drugs that require gastric acidity for absorption, such as ketoconazole or itraconazole.</p> <p>PPIs are used for up to 4 weeks to treat and relieve symptoms of active duodenal ulcers. Physicians may prescribe for up to 8 weeks to treat all grades of erosive esophagitis.</p>	<p>Lansoprazole (Prevacid) decreases gastric acid secretion by inhibiting the parietal cell H<sup>+</sup>/K<sup>+</sup> ATP pump.</p> <p>Omeprazole (Prilosec) decreases gastric acid secretion by inhibiting the parietal cell H<sup>+</sup>/K<sup>+</sup> ATP pump. Omeprazole is now available over the counter.</p> <p>Esomeprazole (Nexium) is the S-isomer of omeprazole. It decreases gastric acid secretion by inhibiting the parietal cell H<sup>+</sup>/K<sup>+</sup> ATP pump. May increase absorption of digoxin; may decrease absorption of iron.</p> <p>Rabeprazole (Aciphex, Alfence, Pariet) decreases gastric acid secretion by inhibiting the parietal cell H<sup>+</sup>/K<sup>+</sup> ATP pump. It is used for short-term (4–8 weeks) treatment and symptomatic relief of gastritis.</p> <p>Pantoprazole (Protonix) decreases gastric acid secretion by inhibiting the parietal cell H<sup>+</sup>/K<sup>+</sup> ATP pump. It is used for short-term (4–8 weeks) treatment and symptomatic relief of gastritis.</p>
Gastrointestinal agents	<p>These agents are effective in the treatment of peptic ulcers and in preventing relapse. Their mechanism of action is not clear. Multiple doses are required, and they are not as effective as the other options.</p>	<p>Sucralfate (Carafate) binds with positively charged proteins in exudates and forms a viscous adhesive substance that protects the GI lining against pepsin, peptic acid, and bile salts. Used for short-term management of ulcers. Sucralfate may cause constipation as one side effect.</p>
Stomach acid protector	<p>Bismuth subsalicylate</p>	<p>Bismuth is a component of Pepto-Bismol and is used to protect the stomach lining from acid; it kills <i>Helicobacter pylori</i>.</p>

Adapted from: Shayne P. Gastritis and peptic ulcer disease, <http://www.emedicine.com/emerg/topic820.htm>, accessed February 28, 2005.



## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Ginger may be used as an antiemetic. Do not use large doses with warfarin, aspirin, or other antiplatelet drugs, antihypertensive drugs, and hypoglycemic drugs. Additive effects can cause unpredictable changes in BP and decreases in blood glucose levels and may decrease platelet aggregation and thus increase bleeding. Ginger ale is commonly used with few side effects.
- Licorice root may be recommended for gastric and duodenal ulcers. Do not take with digoxin, because it may cause potassium loss and digoxin toxicity. Licorice root may potentiate the effects of steroids, especially hydrocortisone, progesterone, and estrogens. Also avoid taking with thiazide diuretics and antihypertensive medications because of increased sodium and water retention, along with potential hypokalemia; spironolactone is especially antagonized by licorice root.
- Banana, garlic, cabbage, and yellow root have no clinical trials proving efficacy. Broccoli and soy products may be beneficial.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- As needed by the individual, offer guidance about dietary alterations that may be useful.
- Discuss the need to complete treatments for eradication of *H. pylori* bacteria, where present. One treatment is usually not sufficient. Suggest increasing intake of broccoli and soy products.
- Reduce intake of alcoholic beverages; stop smoking; and monitor any family history of ulcer disease to address it as quickly as possible.

- As a preventive measure, recommend endoscopy early in patients older than 45–50 years who have dysphagia, recurrent vomiting, weight loss, or bleeding.

### Patient Education—Foodborne Illness

- Careful food handling will be important. Hand washing is important to reduce the spread of *H. pylori*. Always wash hands after using the bathroom and before eating.
- If home TF is needed, teach appropriate sanitation and food handling procedures.

### For More Information

- Centers for Disease Control and Prevention—Peptic Ulcer <http://www.cdc.gov/ulcer/md.htm>
- Foundation for Digestive Health <http://www.fdh.org/html/education/ulcer/facts.html>
- Helicobacter Foundation <http://www.helico.com/>
- Medline—Peptic Ulcer <http://www.nlm.nih.gov/medlineplus/pepticulcer.html>
- Web MD—Peptic Ulcer <http://www.webmd.com/digestive-disorders/digestive-diseases-peptic-ulcer-disease>

## PEPTIC ULCER DISEASE—CITED REFERENCES

- Dube C, et al. *Helicobacter pylori* in water sources: a global environmental health concern. *Rev Environ Health*. 24:1, 2009.
- Feng LY, et al. Effects of killing *Helicobacter pylori* quadruple therapy on peptic ulcer: a randomized double-blind clinical trial. *World J Gastroenterol*. 11:1083, 2005.
- Ford AC, et al. Eradication therapy for peptic ulcer disease in *Helicobacter pylori* positive patients. *Cochrane Database Syst Rev*. 2:CD003840, 2006.
- Ko KP, et al. Soybean product intake modifies the association between interleukin-10 genetic polymorphisms and gastric cancer risk. *J Nutr*. 139: 1008, 2009.
- Tay CY, et al. Population structure of *Helicobacter pylori* among ethnic groups in Malaysia: recent acquisition of the bacterium by the Malay population. *BMC Microbiol*. 9:126, 2009.
- Yanaka A, et al. Dietary sulforaphane-rich broccoli sprouts reduce colonization and attenuate gastritis in *Helicobacter pylori*-infected mice and humans. *Cancer Prev Res*. 2:353, 2009.

# VOMITING, PERNICIOUS

## NUTRITIONAL ACUITY RANKING: LEVEL 3 (LONGER THAN 7 DAYS)



### DEFINITIONS AND BACKGROUND

Pernicious, uncontrolled vomiting may occur in any of several disorders, including concussion or brain trauma, meningitis or encephalitis, intestinal blockage, migraine headaches, brain tumor or other forms of cancer, foodborne illness, gastroparesis, and pregnancy (hyperemesis gravidarum, see Pregnancy in Section 1). Hyperemesis gravidarum involves pregnancy-induced hormonal changes, often associated with concurrent *H. pylori* infection. The presence of weight loss, GI bleeding, persistent fever,

chronic severe diarrhea, and significant vomiting is associated with a higher prevalence of organic disease in children and should be carefully assessed (American Academy of Pediatrics, 2005).

The biggest immediate risk with pernicious vomiting is dehydration, where losses of water, potassium, and sodium can affect the brain, kidneys, and heart. Watch for signs such as dry mouth membranes, dry lips, sunken eyes, rapid but weak pulse, rapid breathing, cold hands and feet, confusion, and difficulty with arousal. Nutritional deficits are possible when the vomiting is prolonged.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Pernicious vomiting in itself is a symptom of an underlying condition and is not genetic.

<b>Clinical/History</b>	Orthostatic hypotension?	N balance
Height	GI bleeding?	Gluc
Weight		H & H
Weight changes		Serum Fe,
BMI	<b>Lab Work</b>	ferritin
Diet history	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>	Serum folate or
Temperature	Ca <sup>++</sup> , Mg <sup>++</sup>	B <sub>12</sub>
(fever?)	Alb, transthyretin	Gastric emptying
Dehydration?	BUN, Creat	tests

## INTERVENTION



### OBJECTIVES

- Correct electrolyte and fluid imbalances and unintentional weight loss
- Modify oral intake until vomiting resolves.
- Distinguish symptoms that could be related to bulimia nervosa.
- If there is hyperglycemia or diabetes, return to normal blood glucose levels as quickly as possible; insulin may be needed.
- For cancer patients, depending on the type of treatment (either curative or palliative) and on the patient's nutritional status, provide patient-tailored nutritional intervention such as oral supplementation, enteral or total PN (Marin Caro et al, 2007).

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Abnormal GI Function—Vomiting

**Assessment:** Food records, I & O reports, chemotherapy schedule and frequency. Uncontrollable vomiting three to four times daily for past week. Signs of dehydration.

**Nutrition Diagnosis (PES):** Inadequate food and beverage intake related to intolerance to oral diet and chemotherapy treatment as evidenced by dehydration and vomiting after meals, with weight loss of 5 lb over past week.

**Intervention:** Coordinate care: Alter meal pattern according to chemotherapy medications and timing, review timing of fluid intake related to meals. Offer sips of fluid every 1–2 hours while awake.

**Monitoring and Evaluation:** Food records, I & O reports, weight recovery. Resolution of vomiting episodes.



## FOOD AND NUTRITION

- For patients with an acute condition, NPO for 24 hours with intravenous (IV) glucose is common. Oral rehydration solution may be needed. (See Diarrhea entry.)
- When tolerated, gastrostomy TF or jejunostomy may be warranted. An isotonic formula is desirable to reduce imbalances between solute and solvent. CPN may also be a consideration if the condition is prolonged.
- As the patient progresses to an oral diet, clear liquids such as cranberry juice or bouillon may be helpful. Gradually add toast, crackers, jelly, and simple carbohydrates in small, frequent meals.
- Give fluids between meals (a “dry diet”). Avoid acidic fruit and vegetable juices if not tolerated.
- Consider avoiding foods that delay gastric emptying (high-fat, hypertonic, or highly fibrous foods).
- Gradually have the patient resume a normal diet. Decrease fatty foods if not tolerated.

### Common Drugs Used and Potential Side Effects

- Anti-emetic agents may be indicated for some conditions. Meclizine or cyclizine may be prescribed; they are antihistamines and powerful antinausea agents.
- Selective serotonin 5-hydroxytryptamine-3 (5-HT<sub>3</sub>) receptor antagonists have proven to be safe and effective for post-operative nausea and vomiting; these include dolasetron, granisetron, ondansetron, and tropisetron, which bind to 5-HT<sub>3</sub> receptors, blocking serotonin binding at vagal afferents in the gut and in the regions of the CNS involved in emesis (Gan, 2005).
- Insulin or oral agents may be needed if diabetes is also present.
- Peristaltic agents may be used in cases of gastroparesis.
- Chronic use of cannabinoids can lead to hyperemesis; this includes oral use of marijuana for cancer, multiple sclerosis, or social purposes.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Ginger is often used as an antiemetic. Do not use large doses with warfarin, aspirin, other antiplatelet drugs, antihypertensive drugs, and hypoglycemic drugs. Additive effects can cause unpredictable changes in BP, blood glucose levels, platelet aggregation, and bleeding.
- Ginger ale has few side effects and may help.



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain why fluids should be taken between meals. Identify adequate amounts of fluids to be consumed every day. Sips of 1–2 oz every few hours can be tolerated; this may include water, sports drinks, gelatin, and clear broth.
- There are many suitable oral rehydration products available from a pharmacy; discuss appropriate products with

the pharmacist. If needed, share the ingredient recipe if it is to be made at home.

- Do not force eating. Eat and drink slowly; stop when full. Make up lost calories at another time.
- Eat meals in a well-ventilated area free from odors.
- Do not lie down for 2 hours after eating. Do not overeat.
- Discuss the role of carbohydrates and fiber in maintaining blood glucose levels.
- Discuss reasons to seek immediate medical attention, including severe abdominal pain, severe headache, stiff neck, fever over 101°F, vomiting of blood, rapid breathing or pulse.
- In palliative care, nutritional support aims at improving patient's quality of life by controlling symptoms such as nausea, vomiting, and pain related to food intake (Marin Caro et al, 2007).

#### Patient Education—Food Safety

- Careful food handling and hand washing are important to prevent introduction of foodborne pathogens to the diet of the individual.

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

#### For More Information

- Medicine Net  
[http://www.medicinenet.com/nausea\\_and\\_vomiting/article.htm](http://www.medicinenet.com/nausea_and_vomiting/article.htm)
- Nausea and Vomiting Algorithm  
[http://www.uwgi.org/guidelines/ch\\_01/ch01txt.htm](http://www.uwgi.org/guidelines/ch_01/ch01txt.htm)

#### VOMITING, PERNICIOUS—CITED REFERENCES

- American Academy of Pediatrics. Subcommittee on Chronic Abdominal Pain; North American Society for Pediatric Gastroenterology Hepatology, and Nutrition. Chronic abdominal pain in children. *Pediatrics*. 115:370, 2005.
- Gan TJ. Selective serotonin 5-HT(3) receptor antagonists for postoperative nausea and vomiting: are they all the same? *CNS Drugs*. 19:225, 2005.
- Marin Caro MM, et al. Nutritional intervention and quality of life in adult oncology patients. *Clin Nutr*. 26:289, 2007.

## LOWER GI: INTESTINAL DISORDERS

# CARCINOID SYNDROME

### NUTRITIONAL ACUITY RANKING: LEVEL 3



#### DEFINITIONS AND BACKGROUND

Carcinoid tumors are part of a group of GI and pancreatic endocrine tumors that secrete hormones, 5-HT, tachykinins, and other mediators (Druce et al, 2009). The rare neuroendocrine growth develops in the wall of the intestine and is usually discovered in x-rays or during surgery performed for other reasons. GI carcinoid tumors are difficult to diagnose (Gore et al, 2005). Carcinoid cancer patients often have elevated levels of serotonin or its precursor 5-hydroxytryptophan (Shah et al, 2005). Octreotide scanning has a sensitivity of primary tumor detection of 90% (Northrup and Lee, 2007). GI carcinoids comprise 90% of all carcinoid tumors and all carcinoids have malignant potential (Northrup and Lee, 2007). Sometimes, the growth occurs in the appendix area. The growths can be large enough to cause intestinal obstruction. Some growths metastasize to the liver, creating hormone-producing tumors with flushing of the head and neck (usually triggered by alcohol or exercise). Flushing symptoms can last for several hours from the release of vasoactive serotonin, histamine, and prostaglandins.

Other symptoms include swollen or watery eyes, explosive diarrhea, abdominal cramps, wheezing, breathlessness, and symptoms similar to heart failure. Cardiac lesions are also seen; up to one third of patients develop cardiac valvulopathy. Patients may benefit from a valve replacement. If liver involvement occurs, liver resection or transplantation may be needed.

Diversion of tryptophan to 5-HT synthesis occurs, resulting in less tryptophan for protein and nicotinamide synthesis. Pellagra and psychiatric symptoms result from depletion of tryptophan, which is consumed by the tumor for serotonin

synthesis (van der Horst-Schrivers et al, 2004). Psychiatric symptoms should be evaluated carefully.

The malignant carcinoid syndrome is caused by circulating neuroendocrine mediators produced by the tumor and occurs in less than 10% of patients (Bell et al, 2005). Surgery is combined with continuous biotherapy with long-acting somatostatin analogs and interferon, which may alleviate symptoms and slow the disease progression (Akerstrom et al, 2005). Survival ranges from 3 to 20 years after diagnosis.



#### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Carcinoid tumor fibrosis is a CTGF/TGFβ-mediated stellate cell-driven fibrotic response (Kidd et al, 2007).

Clinical/History	(carcinoid syndrome)	Octreotide scanning
Height	Breathlessness	X-rays of GI tract
Weight	Number of stools, consistency	Skin changes (scleroderma, pellagra)?
BMI	Biopsy	Psychiatric symptoms?
Diet history	Endoscopy	
Diarrhea, flushing, wheezing		

Magnetic resonance imaging (MRI)	Serum histamine Serum serotonin (elevated) Alb, transthyretin Transferrin	TLC H & H Serum Fe, ferritin Na <sup>+</sup> , K <sup>+</sup> Ca <sup>++</sup> , Mg <sup>++</sup>
----------------------------------	--	---

**Lab Work**

5-HIAA test  
(urine  
S-HIAA)

**INTERVENTION****OBJECTIVES**

- Ease symptoms of secretory diarrhea and reduce any pain.
- Slow progression of the disease, which is not cured by surgery.
- Control side effects of medications.
- Replenish electrolyte and fluid losses.
- Correct niacin deficiency. Biochemical niacin deficiency is prevalent among newly diagnosed carcinoid syndrome patients (Shah et al, 2005).

**FOOD AND NUTRITION**

- Decrease fiber intake during acute stages of diarrhea. Add pectin and ensure adequate fluid intake during those periods.
- Avoid alcoholic beverages. Limit caffeine intake to a controlled amount.
- During testing, omit foods that contain 5-HIAA: avocados, pineapple, bananas, kiwi fruit, plums, eggplant, walnuts, hickory nuts, and pecans.
- Omega-3 fatty acids may be used for their role in reduction of inflammation; include fish such as salmon, sardines, tuna, and herring often.
- Since tryptophan is not adequately converted to niacin, a daily supplement with DRI levels may be suggested. Avoid excesses.

**SAMPLE NUTRITION CARE PROCESS STEPS****Altered GI Function**

**Assessment Data:** Explosive diarrhea, flushing, fear of eating.

**Nutrition Diagnoses (PES):** Altered GI function related to release of vasoactive substances as evidenced by explosive diarrhea and altered labs with diagnosis of carcinoid syndrome.

**Interventions:** Education about foods or alcohol that may aggravate symptoms. Counseling about postsurgical wound healing, if required. Discuss use of probiotics, adequate fluid, omega-3 fatty acid sources. Avoid alcohol if symptoms are aggravated by intake.

**Monitoring and Evaluation:** Fewer complaints of diarrhea, flushing, GI discomfort, and fear of eating.

**Common Drugs Used and Potential Side Effects**

- Subcutaneous injections of the somatostatin analog octreotide are used. This is expensive, and treatment may be for many years. Newer somatostatin analogs (such as lanreotide) are being tested. Vasoconstricting interleukin or cytotoxic drugs may control side effects.
- If kaolin (Kaopectate) and other medications are used to control diarrhea, constipation is a possible side effect.
- Bronchodilators may be used to control wheezing. Evaluate for potential side effects.

**Herbs, Botanicals, and Supplements**

- Herbs and botanical supplements should not be used without discussing with the physician.
- Monitor side effects if large doses of niacin are taken.

**NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT**

- Discuss measures specifically designed for the patient's status and tolerance levels.
- Suggest ways to make meals more appetizing if appetite is poor.
- Describe techniques for management of diarrhea, abdominal pain, or cramping by reducing fiber and fat intake during those times of flare.
- Probiotics such as acidophilus milk and yogurt with live and active cultures may be useful in the diet.

**Patient Education—Foodborne Illness**

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

**For More Information**

- Carcinoid Syndrome  
<http://www.emedicine.com/med/topic2649.htm>

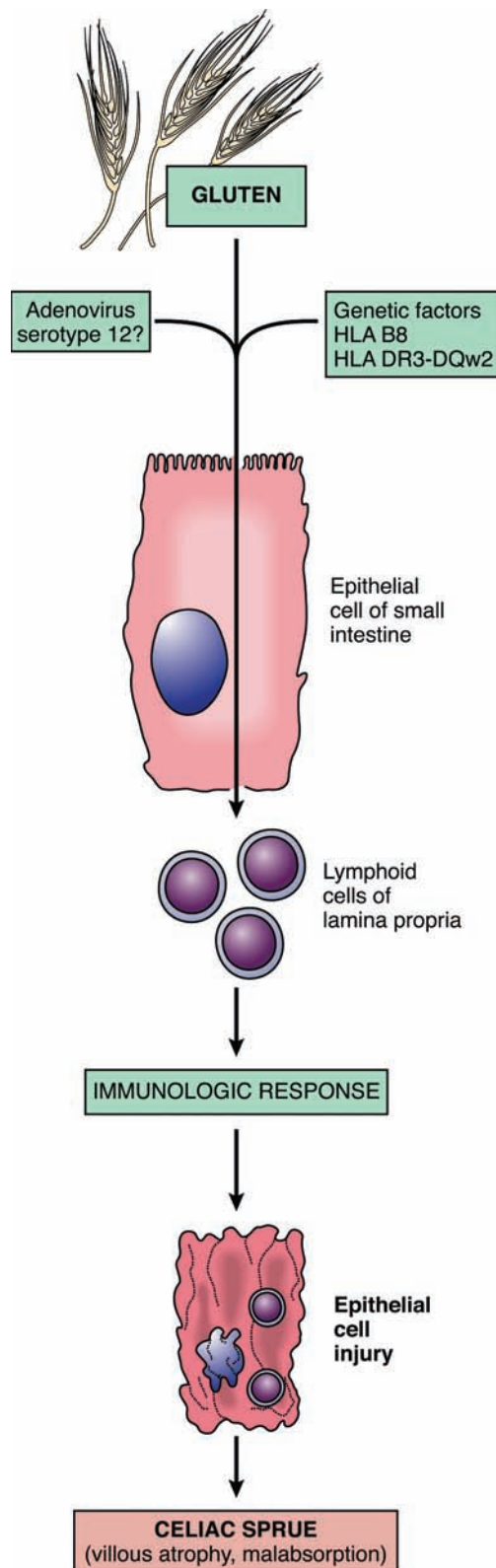
**CARCINOID SYNDROME—CITED REFERENCES**

- Akerstrom G, et al. Management of midgut carcinoids. *J Surg Oncol.* 89:161, 2005.
- Bell HK, et al. Cutaneous manifestations of the malignant carcinoid syndrome. *Br J Dermatol.* 152:71, 2005.
- Druce M, et al. Fibrosis and carcinoid syndrome: from causation to future therapy. *Nat Rev Endocrinol.* 5:276, 2009.
- Gore RM, et al. GI carcinoid tumours: appearance of the primary and detecting metastases. *Best Pract Res Clin Endocrinol Metab.* 19:245, 2005.
- Kidd M, et al. CTGF, intestinal stellate cells and carcinoid fibrogenesis. *World J Gastroenterol.* 13:5208, 2007.
- Northrup JA, Lee JH. Large bowel carcinoid tumors. *Curr Opin Gastroenterol.* 23:74, 2007.
- Shah GM, et al. Biochemical assessment of niacin deficiency among carcinoid cancer patients. *Am J Gastroenterol.* 100:2307, 2005.
- Van Der Horst-Schrivers AN, et al. Complications of midgut carcinoid tumors and carcinoid syndrome. *Neuroendocrinology.* 80:28S, 2004.



# CELIAC DISEASE

## NUTRITIONAL ACUITY RANKING: LEVEL 4



## DEFINITIONS AND BACKGROUND

Celiac disease (CD) is a common, lifelong, genetically based autoimmune disorder that causes inflammation of the proximal small intestine (See and Murray, 2006). CD is characterized by inappropriate T cell-mediated immune response to ingested gluten from wheat, rye, and barley that leads to inflammation, villous atrophy, and crypt hyperplasia in the small intestine. Intestinal villi decrease in number, with less absorptive surface and fewer enzymes. Crypts are markedly elongated, causing mucosal malabsorption.

Other names for this disorder include celiac sprue, gluten enteropathy, or nontropical sprue. A major consensus panel determined that 1% of Caucasians (upwards of 3 million people) may have CD (See and Murray, 2006). While screening suggests that one in 133 people have it, the frequency may be closer to one in 100; undiagnosed CD seems to have increased dramatically in the United States during the past 50 years (Rubio-Tapia et al, 2009).

Diagnosis can occur at any age (infancy through old age) and often occurs after stress, pregnancy, or viral infections. Infants may present with impaired growth, diarrhea, pica, abdominal distention, pallor, edema, or vomiting. Children may have frequent, strong-smelling stools that are pale and foamy, diarrhea, irritability, a distended abdomen, easy fatigue, pallor, weight loss, vomiting, and anemia.

Increased intestinal permeability plays a pathogenic role in various autoimmune diseases including CD and type 1 diabetes (T1D) (Visser et al, 2009). Gliadin may also be involved in the pathogenesis of T1D (Visser et al, 2009). All persons with GI symptoms should be tested for CD, as should individuals with type 1 diabetes (T1D), unexplained iron deficiency anemia, elevated levels of transaminases, thyroid disorders, short stature, delayed puberty, fetal loss, and in relatives of patients who have CD (Thompson, 2005). Screening studies have revealed that CD may be asymptomatic in adults (Niewinski, 2008), or they may have episodic or nocturnal diarrhea, flatulence, intestinal bloating that mimics IBS, steatorrhea, weight loss, recurrent stomatitis, anemias or peripheral neuropathy.

Tissue transglutaminase is the main antigen for the antiendomysial antibodies used to diagnose CD. Deamidated gliadin antibodies (DGP) have shown promising results as serological markers for CD (Setty et al, 2008). False-negative test results can occur in children under age 2 years or in patients who have followed a gluten-free (GF) diet for a month or longer. The diagnostic criteria for CD need re-evaluation; endomysial antibody positivity without atrophy belongs to the spectrum of genetic gluten intolerance, and warrants dietary treatment (Kurppa J, et al. 2010).

Early diagnosis and treatment, together with regular follow-up visits with a dietitian, are necessary to ensure nutritional adequacy and to prevent malnutrition while adhering to the GF diet for life (Niewinski, 2008). Longer duration of exposure to gluten can increase the risk of other autoimmune diseases, including non-Hodgkin's or intestinal lymphoma,

squamous cell cancer of the esophagus, type 1 diabetes, autoimmune thyroid disease, Addison's disease, lupus, primary biliary cirrhosis, osteoporosis, psoriasis, Sjogren's syndrome, and rheumatoid arthritis (National Institutes of Health, 2004).

Dermatitis herpetiformis (DH) is the skin manifestation, with intensely pruritic lesions that occur over the surface of the elbows, knees, legs, buttocks, trunk, neck, or scalp. Abnormal biopsy is evident, but GI symptoms are not always present. There may be immunoglobulin (IgA) deposits around the lesions, and large intake of iodine may trigger flares of DH.

When wheat, rye, and barley grains are consumed, they damage the mucosa of the small intestine, eventually leading to nutrient malabsorption. Moderate amounts of pure oats can be consumed. However, since much of the commercially available oat flour may be contaminated with wheat gluten, caution is advised for new patients.

Response to GF diets in children with autism may be related to amelioration of nutritional deficiency resulting from undiagnosed gluten sensitivity. Therefore, it is recommended that all children with neurodevelopmental problems be assessed for CD (Genius and Bouchard, 2009). Children and adolescents with symptoms of or an increased risk for CD should have the blood test for antibody to tissue transglutaminase (TTG). Those with an elevated TTG should be referred to a pediatric gastroenterologist for an intestinal biopsy. If tests prove positive, a GF diet is needed. Then, a gastroenterologist should follow all patients with CD.

Because oxidative stress plays an important role in the inflammatory process of CD, increased use of lycopene, quercetin, and tyrosol can be recommended (DeStefano et al, 2007). New forms of treatment propose the use of gluten-degrading enzymes to be ingested with meals, the development of GF grains by genetic modification, the use of substrates regulating intestinal permeability to prevent gluten entry across the epithelium, and different forms of immunotherapy (Setty et al, 2008).

Since the defect is permanent, the GF diet is curative and must be a permanent change. Surface cells of the mucosa are replaced within 5 days; swelling is reduced within 14 days; and villi improve within 6 months or up to 5 years. Consultation with a dietetics professional knowledgeable in CD is essential. An individualized, team approach is best because symptoms change over time.

Single-nucleotide polymorphisms (SNPs) in the chromosome 4q27 region containing IL2 and IL21 are associated with CD; this is similar to other autoimmune diseases such as rheumatoid arthritis, type 1 diabetes, Graves' disease, psoriatic arthritis, and ulcerative colitis (UC, Glas et al, 2009).

Clinical/History	Multiple small bowel biopsies showing flat villi	Macrocytic anemia (low vitamin B <sub>12</sub> or folate?)
Height		
Weight		
BMI		
Diet history	Abdominal computed tomography (CT) or MRI scan	Serum homocysteine (tHcy)
Failure to thrive or weight loss		Serum carotene for vitamin A
Aphthous stomatitis	Capsule endoscopy	Serum vitamins D, E, and K
Fatigue, lassitude, depression	Small bowel biopsy	Serum copper (decreased)
Recurrent abdominal pain, bloating		Serum phosphorus (decreased)
Strong-smelling stools that are pale and foamy	<b>Lab Work</b>	Serum zinc (decreased)
Distended abdomen	Anti-TTG antibodies (tTG)—IgA and IgG (95–100% sensitive and specific)	Ca <sup>++</sup> (often low)
Irritability, pallor		Na <sup>+</sup> , K <sup>+</sup>
Steatorrhea, chronic diarrhea	Anti-endomysium antibodies—IgA (EMA)—100% specificity, >90% sensitivity	Mg <sup>++</sup>
DH		Alb, transthyretin
Enamel defects of permanent teeth	HLA-DQ2 and HLA-DQ8 haplotype	CRP
Headaches?	H & H	Transferrin, TIBC
Osteopenia, bone pain?	Serum Fe, ferritin (anemia is common)	Lactic acid dehydrogenase (increased)
Infertility, frequent miscarriages?		Xylose absorption
Short stature?		DEXA scan
		Fecal chymotrypsin level
		Fecal fat study
		Liver function tests (mildly abnormal)



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Like an IBD, gliadin peptides are presented on the Human Leukocyte Antigen DQ2 or DQ-8 molecules of antigen-presenting cells to T helper cells, provoking a T helper 1 response that leads to damage by pro-inflammatory cytokines and interleukins (Festen et al, 2009; Visser et al, 2009). CD is more common among people with other genetic disorders including Down syndrome.

## INTERVENTION



### OBJECTIVES

There are six elements required for the management of CD (Thompson, 2005):

1. Consult a skilled dietetics professional. Medical nutrition therapy (MNT) to promote provided by a registered dietitian is strongly recommended improved self-management (American Dietetic Association, 2009).
2. Educate about the disease.
3. Adhere to a GF diet. Remove the offending protein (gliadin fraction) from the diet; glutenin is harmless. Improvement is noted within 4–5 days.

## SAMPLE NUTRITION CARE PROCESS STEPS

**Altered Nutrient Utilization—Celiac Disease**

**Assessment Data:** Weight loss over the past year. I & O records; stool patterns and frequency; abdominal distention and bloating; DH. GI biopsies reveal CD; positive tTG test.

**Nutrition Diagnosis (PES):** Altered nutrient utilization related to flattened intestinal villi from CD as evidenced by positive biopsies and tTG test, presence of DH, frequent bouts of abdominal distention, bloating, diarrhea and weight loss.

**Intervention:** Education about the sources of gluten from diet and food and nonfood products. Provision of recipes for managing the GF diet. Supplier sources of GF foods and flours or grain products. Counseling about fluid sources and ways to incorporate fluids into meals and nourishments. Meal planning tips for the family.

**Monitoring and Evaluation:** Improved I & O records and weight gain. Normalized stool patterns and frequency. Fewer complaints of GI distress, bloating and diarrhea associated with meals. No signs of protein-energy malnutrition.

## 4. Identify and treat nutritional deficiencies:

- Deficiencies of iron, folate, calcium, and vitamin D may be found.
- Replace nutrients lost from diarrhea and steatorrhea.
- Reverse bone demineralization, hypoalbuminemia, and hypoprote thrombinemia where present.
- Whole-body protein breakdown is common in CD, contributing to a high level of protein-calorie malnutrition.
- Glutamine is an important fuel for the health of intestinal epithelial cells. Replenish as needed.

## 5. Provide access to an advocacy group. A local celiac support chapter is most helpful.

## 6. Provide continuous long-term follow-up care by a multidisciplinary team.



## FOOD AND NUTRITION

- A GF diet excludes wheat, rye, and barley. Avoid products such as breading, stuffing, croutons, graham, bulgur, matzo, broth, breading or coating mixes, communion wafers, pastas, cracked wheat, semolina, farina, malt, malt flavoring, brown rice syrup, commercial soups, imitation bacon, imitation seafood, marinades, processed meats, roux, sauces, seasonings, self-basting poultry, soups and soup bases, thickeners, vegetarian meat substitutes, and commercial potato and rice mixes. Soy sauce, white or nonmalt vinegars, and wheat starch must be pure; read labels.
- Plan a diet that includes acceptable grains and starches; see Table 7-9.
- A Greek-Mediterranean dietary pattern with olive oil, nuts, fruits, and vegetable intake can be recommended. Lycopene, quercetin, and tyrosol may control the pro-inflammatory genes involved in CD (DeStefano et al, 2007). Tyrosol is found in wine and extra virgin olive oil;

**TABLE 7-9 Grains and Starches to Use Freely in Celiac Disease**

Amaranth	Nut flours
Arrowroot	Quinoa
Beans (black, garbanzo, kidney, northern, pinto)	Poha flakes
Black-eyed peas, lentils, split peas	Potato
Buckwheat	Potato starch, potato flour
Cassava	Rice
Corn, corn bran, hasa marina	Sorghum
Gluten-free bread	Soy
Hominy, grits	Tapioca
Indian rice grass	Tef
Montina	Wild rice

lycopene in tomato products, watermelon, pink grapefruit; quercetin in apples, citrus fruit, tea, capers, buckwheat, red grapes, broccoli, and red onions. Buckwheat contains rutin (quercetin-3-O-rutinoside) which produces a catabolite (3,4-dihydroxyphenylacetic acid) with significant reducing power, free-radical scavenging activity and enhanced antioxidant capacity of the colonic lumen (Jaganath et al, 2009).

- Because oats are sometimes contaminated with wheat during processing, avoid in initial stages of treatment. Include small-to-moderate amounts if tolerated after the first few months.
- Diet for adults should provide 1–2 g of protein/kg body weight from fresh meat, fresh fish, milk, cheese, and eggs. Examine processed items carefully since there is often gluten added.
- Diet should provide 35–40 kcal/kg body weight for adults.
- For infants with diarrhea, provide fluids, electrolytes, and a formula that is not high in fat content. Infants may tolerate banana powder; adults and children can eat starchy-type carbohydrates, bananas, lean meats, and fish. Rehydrate with oral rehydration solution or other fluids.
- Initially, the diet should include low amounts of fiber because of flattened mucosal villi; increase as tolerated. Fruits and vegetables are naturally low in gluten and should be included regularly.
- If TF is used, a glutamine-enriched product may be useful. Monitor ingredients carefully to avoid gluten.
- Lactose intolerance may be either temporary or permanent. Initially, dairy products (primarily milk and products made with milk) should be avoided. After 3–6 months of treatment, dairy products may be gradually reintroduced.
- GF products are often low in B vitamins, calcium, vitamin D, iron, zinc, magnesium, and fiber. Few GF products are enriched or fortified, adding to the risk of nutrient deficiencies. Correction of vitamin and mineral deficiencies is important. Supplements to the diet should include water-miscible vitamins A, D, E, and K, iron, calcium,

folic acid, vitamin B<sub>12</sub>, thiamine, and other B-complex vitamins.

- Products containing MCTs are often used when fat malabsorption is present, especially in adults.
- Foods that often are not allowed include commercial cream soups, creamed vegetables, ice cream (labels should be checked for thickening agents), cakes, cookies, and breads unless made with rice, corn, or potato flours. For toddlers, mixed infant dinners and junior dinners that contain flour thickeners, spaghetti, macaroni, and other pastas should not be used.

### Common Drugs Used and Potential Side Effects

- No drug therapy has been proven to suppress the disease.
- Check all labels *each time* for gluten-containing ingredients. GF laxatives include psyllium seed laxatives (Metamucil, Naturacil), docusate sodium (Surfak), and bisacodyl (Dulcolax). Gliadins are often impurities in medications, including acetaminophen; check carefully.
- Corticosteroids may be used with numerous side effects. Take with food. Monitor for negative nitrogen or calcium balances, and for weight gain.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Avoid aloe, cascara, senna, and yellow dock because of enhancing effects when using bisacodyl.
- A B-complex supplement may be warranted if serum tHcy levels are elevated (Hadihi et al, 2009; Hallert et al, 2009). A general multivitamin–mineral supplement may be beneficial.
- With antibiotics, quercetin may interfere with fluoroquinolones. Advise the physician if used in large amounts.
- Studies are ongoing about the need for carnitine supplementation in CD.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- **Strict lifelong elimination of gluten** and dietary adherence are essential (Chand and Mihas, 2006). If strictly followed, improvement will occur in most patients within 2 weeks. If results are not seen after 6–9 months of diet therapy, a new diagnosis should be sought.
- Endomysial antibodies are specific in predicting villous atrophy; patients with endomysial antibodies benefit from a GFD regardless of the degree of enteropathy (Kurppa et al, 2010).
- Intense expert dietary counseling is needed for all patients with CD because the diet is complex (American Dietetic Association, 2009; Niewinski, 2008). An effective plan requires extensive, repeated counseling and instruction of the patient by a skilled dietitian.
- Instruct the patient or family to read food labels *each time* for cereal, starch, flour, thickening agents, emulsifiers,

gluten, stabilizers, hydrolyzed vegetable proteins, semolina, durum, triticale, bulgur, farina, couscous, broth, caramel coloring and monosodium glutamate (MSG).

- The Food Allergen Labeling and Consumer Protection Act (FALCPA) requires food labels to clearly identify wheat and other common food allergens in the list of ingredients. A GF symbol is now widely used by food manufacturers. Contact the manufacturer if there are questionable ingredients.
- “Wheat free” is not the same as gluten free; products may contain rye or barley.
- Because of possible contamination with wheat, oats should be avoided in new patients.
- “Wheat starch” is acceptable because the gliadin/gluten has been removed.
- Toothpaste, mouthwash, lipstick or chapstick, glue on envelopes or teabags, boxed candy, chewing gum wrappers, utensils in buffet lines, toasters, bulk food bins, jars used for various purposes, and other related items should be checked carefully and avoided if gluten is present.
- A Greek–Mediterranean dietary pattern high in olive oil, nuts, fruits and vegetable may be recommended.
- For children at school, it may be necessary to educate the staff, nurses and teachers about the GF diet to enhance compliance.
- Quality of life improves for most individuals with CD who follow the GF diet for at least a year (American Dietetic Association, 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 GI discomfort.
- If home TF is needed, teach appropriate sanitation and food handling procedures.

### For More Information

- Celiac Disease and Gluten-Free Diet Support Group  
<http://www.celiac.com/>
- Celiac Disease Foundation  
<http://www.celiac.org>
- Celiac Sprue Association  
<http://www.csaceliacs.org/>
- Children’s Digestive Health and Nutrition Foundation  
<http://www.cdhnf.org/wmspage.cfm?parm1=40>
- Freeda Vitamins  
<http://www.freedavitamins.com>
- Gluten-Free Diet by Shelley Case  
<http://www.glutenfreediet.ca>
- Gluten Free Guide  
<http://www.cdhnf.org/user-assets/documents/pdf/GlutenFreeDietGuideWeb.pdf>
- Gluten-Free Pantry  
<http://www.glutenfree.com/>
- Gluten Intolerance Group of North America  
<http://www.gluten.net/>
- Guidelines for a Gluten-Free Lifestyle  
<http://www.celiac.org/newsEvents.php>
- National Celiac-Sprue Society  
<http://www.csaceliacs.org>
- NIDDK  
<http://digestive.niddk.nih.gov/ddiseases/pubs/celiac/>



## CELIAC DISEASE—CITED REFERENCES

- American Dietetic Association. Evidence analysis library: Celiac disease. Web site accessed November 8, 2009. at, <http://www.adaevidencelibrary.com/topic.cfm?cat=3677&library=EBG>.
- Chand N, Mihas AA. Celiac disease: current concepts in diagnosis and treatment. *J Clin Gastroenterol*. 40:3, 2006.
- DeStefano D, et al. Lycopene, quercetin and tyrosol prevent macrophage activation induced by gliadin and IFN-gamma. *Eur J Pharmacol*. 566:192, 2007.
- Festen EA, et al. Inflammatory bowel disease and celiac disease: overlaps in the pathology and genetics, and their potential drug targets. *Endocr Metab Immune Disord Drug Targets*. 9:199, 2009.
- Genius SJ, Bouchard TP. Celiac Disease Presenting as Autism [published online ahead of print 29 June 2009]. *J Child Neurol*. 25:114, 2009.
- Glas J, et al. Novel genetic risk markers for ulcerative colitis in the IL2/IL21 region are in epistasis with IL23R and suggest a common genetic background for ulcerative colitis and celiac disease. *Am J Gastroenterol*. 104:1737, 2009.
- Hadihi M, et al. Effect of B vitamin supplementation on plasma homocysteine levels in celiac disease. *World J Gastroenterol*. 15:955, 2009.
- Hallert C, et al. Clinical trial: B vitamins improve health in patients with coeliac disease living on a gluten-free diet. *Aliment Pharmacol Ther*. 29:811, 2009.
- Jaganath IB, et al. In vitro catabolism of rutin by human faecal bacteria and the antioxidant capacity of its catabolites [published online ahead of print 30 July 2009]. *Free Radic Biol Med*. 47:1180, 2009.
- Kurppa J, et al. Celiac disease without villous atrophy in children: a prospective study. *J Pediatr*. 157:373, 2010.
- National Institutes of Health. National Institutes of Health Consensus Development Conference Statement: Celiac disease, 2004. Accessed July 22, 2009 at <http://consensus.nih.gov/2004/2004CeliacDisease118html.htm>.
- Niewinski MM. Advances in celiac disease and gluten-free diet. *J Am Diet Assoc*. 108:661, 2008.
- Rubio-Tapia A, et al. Increased prevalence and mortality in undiagnosed celiac disease. *Gastroenterology*. 137:88, 2009.
- See J, Murray JA. Gluten-free diet: the medical and nutrition management of celiac disease. *Nutr Clin Pract*. 21:1, 2006.
- Setty M, et al. Celiac disease: risk assessment, diagnosis, and monitoring. *Mol Diagn Ther*. 12:289, 2008.
- Thompson T. National Institutes of Health consensus statement on celiac disease. *J Am Diet Assoc*. 105:194, 2005.
- Visser J, et al. Tight junctions, intestinal permeability, and autoimmunity: celiac disease and type 1 diabetes paradigms. *Ann N Y Acad Sci*. 1165:195, 2009.

## CONSTIPATION

### NUTRITIONAL ACUITY RANKING: LEVEL 1–2



#### DEFINITIONS AND BACKGROUND

Constipation occurs when the fecal mass remains in the colon longer than the normal 24–72 hours after meal ingestion or when the patient strains to defecate. Stool type and frequency could be used to determine another problem, such as IBS. Constipation and fecal incontinence are common symptoms in patients with cerebral palsy, traumatic spinal cord injuries, spina bifida, multiple sclerosis, diabetic polyneuropathy, Parkinson's disease, and stroke. Intestinal obstruction, tumors and diverticulosis may narrow the intestine, which can also lead to constipation.

**Atonic constipation** ("lazy bowel") occurs when musculature of the bowel no longer functions properly, sometimes from laxative overuse or poor bowel habits. **Spastic constipation** entails increased narrowing of the colon with small, ribbon-like stools caused by inactivity, immobility, or obstruction; increasing physical activity may be useful. **TF constipation** occurs with use of low-fiber products, medications, or other products.

In infants and children, chronic constipation is a concern, with **encopresis** from poor bowel habits and poor fiber intake. There may be food allergies, such as to milk or wheat, which should also be addressed.

Treatment modalities for constipation include prokinetic agents, enemas administered through the enema continence catheter, and biofeedback. For chronic problems, bowel retraining may be necessary.

There is limited evidence that constipation can successfully be treated by increasing fluid intake unless there is dehydration (Muller-Lissner et al, 2005). Increasing physical activity may be helpful (Muller-Lissner et al, 2005). Some patients may be helped by a fiber-rich diet (American Dietetic Association, 2008). Water-soluble fibers (e.g., pectins, gums, mucilages, and some hemicelluloses) slow down intestinal transit while insoluble fibers from lignin, cellulose, and hemicellulose accelerate intestinal transit. Patients with more severe consti-

pation may get increased bloating or distention when increasing dietary fiber intake; proceed slowly when making a change in fiber intake. Fiber supplements may also help.

Conservative therapies focus on a holistic approach in tandem with evolving drug therapies that target intestinal secretion and transit (Chatoor and Emmanuel, 2009). Chronic constipation decreases quality of life. A Constipation-Related Quality of Life measure is available but should be validated with different treatment methods used for chronic constipation (Wang et al, 2009). Surgical correction of rectocele and intussusception benefit those with anatomical symptoms; for those with predominantly functional features, surgery is best avoided (Chatoor and Emmanuel, 2009).



#### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Chronic constipation may have a genetic basis; research is underway.

Clinical/History	Bowel habits	Gas pain, flatulence
Height	Stool color and number	Heartburn, indigestion
Weight	Hard, lumpy stools	Straining at stool
Recent weight changes	I & O	Feeling of obstruction or incomplete evacuation
BMI	BP	
Diet history	Headaches	
Defecation longer than every 3 days	Abdominal distention, pain, spasms	