**TABLE 3-14 When to Initiate Weight Loss Diets in Children**

| | | |
|---|---|---|
| Children aged 2–7 years | BMI: 85th to 94th percentiles; BMI greater than the 95th percentile with no complications | Maintain weight |
| | BMI is above the 95th percentile with mild complications (mild hypertension, dyslipidemia, insulin resistance) | Gradual weight loss is recommended |
| | Patients with acute complications such as pseudotumor cerebri, sleep apnea, obesity hypoventilation syndrome, or orthopedic problems | Refer to a pediatric obesity center |
| Children 7 years of age and older | BMIs between the 85th and 94th percentiles with no complications | Maintain weight |
| | If the BMI is between the 85th and 94th percentiles with mild complications or the BMI is equal to or above the 95th percentile | Gradual weight loss is recommended |

Source: Marcason W. At what age should an overweight child follow a calorie-restricted diet? *J Am Diet Assoc.* 104:834, 2004.

- Family hx of CHD, diabetes mellitus, hypertension, overweight/obesity
- Breastfed versus formula or other milk (young child)
- BP
- Acanthosis nigricans
- Number of hours of TV watching per day
- Inactive lifestyle?
- Skipping meals?

**Lab Work**

- H&H, Serum Fe
- Chol, Trig (elevated)?
- Serum Hcy
- Gluc
- Alk phos
- Alb
- $Ca^{++}$, $Mg^{++}$
- $Na^{+}$, $K^{+}$
- LFTs Serum insulin

## INTERVENTION

### OBJECTIVES

Health Recommendations (American Academy of Pediatrics, 2009)

- Identify and track patients at risk by virtue of family history, birth weight, or socioeconomic, ethnic, cultural, or environmental factors. Calculate and plot BMI annually in all children and adolescents; use change in BMI to identify rate of excessive weight gain relative to linear growth. Develop a weight maintenance or weight loss plan that is individualized for the child.
- Encourage parents and caregivers to promote healthy eating patterns by offering nutritious snacks, such as vegetables and fruits, low-fat dairy foods, and whole grains; encouraging children's autonomy in self-regulation of food intake and setting appropriate limits on choices; and modeling healthy food choices.
- Routinely promote physical activity, including unstructured play at home, in school, in child care settings, and throughout the community. Limit television, computer, and video time to a maximum of 2 h/d.
- Recognize and monitor for risk factors such as hypertension, dyslipidemia, hyperinsulinemia, impaired glucose tolerance, metabolic syndrome, early puberty, liver disease, eating disorders, skin infections, food allergies, asthma, and sleep apnea.
- Discuss tips that are easily handled by the dietetics professional; refer complex cases to a behavioral specialist. Discourage the use of sweets and foods to reward behavior. Avoid the "clean plate" theory, but be wary about withholding food, which can have the opposite effect.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Overweight**

***Assessment Data:*** Weight and growth charts, physical activity pattern showing sedentary lifestyle and minimal activity (no recess, outside activity swing use set only), BMI >85 percentile for age.

***Nutrition Diagnoses (PES):*** Excessive energy intake (N1–1.5 or N1–2.2) related to low physical activity level and intake of high calorie snacks as evidenced by diet and activity records.

***Interventions:***

Food-nutrient delivery:

ND 1.1. General healthful diet; establishing regular meal patterns (three meals a day with two snacks) that follow healthy plate tool, incorporating fiber into the diet (more whole grains, more fruits and vegetables), increasing to about 26 g/d.

ND 1.3. Specific foods and beverages (healthy snacks and low-calorie beverages).

Education:

E 1. Initial/brief nutrition education on label reading, portion sizes of food, and fiber intake. Educate parents about how to increase activity levels with games, dancing, outside play, and healthier, lower calorie snacks and beverages.

Counseling:

C 2. Nutritional-related cognitive behavioral therapy using motivational interviewing to determine what parents and patient want to change and are willing to work on regarding high fat/sugary foods in the home, frequent fast food meals, and inadequate fiber intake.

Coordination of care

RC 1. Referral to an agency or dietitian who conducts grocery store tours to help choose foods more wisely.

***Monitoring and Evaluation:*** Weight records, growth chart showing improved height for weight (below 85 percentile BMI) for age.

- Help the child "find" the right body for him or her. Encourage self-recognition of hunger cues (e.g., stop eating when feeling "full").

*Advocacy Objectives*

- Help parents, teachers, coaches, and others who influence youth to discuss health habits, not body build, as part of their efforts to control overweight and obesity.
- Enlist policy makers from local, state, and national organizations and schools to support a healthful lifestyle for all children, including a proper diet and adequate opportunity for a regular physical activity.
- Encourage organizations that are responsible for health care and health care financing to provide coverage for effective obesity prevention and treatment strategies.
- Encourage public and private sources to direct funding toward research on effective strategies to prevent overweight and obesity and to maximize limited family and community resources.
- Support social marketing that promotes healthful food choices and increased physical activity.

## FOOD AND NUTRITION

- Support needs for the child according to age and sex of the child: protein at 10–35%, fat at 25–40%, and CHO at 45–65% (Institute of Medicine, 2002). Plan a diet with basal calories plus activity, and likelihood of growth spurts.
- Weight loss is typically recommended for children over age 7 or for younger children who have related health concerns; slow and steady—anywhere from 1 lb/wk to 1 lb/mo (Mayo Clinic, 2009).
- For family teaching, discuss easy behavioral changes. Place less emphasis on a specific calorie level than on portion sizes that are child-appropriate. Use MyPyramid for kids.
- Emphasize low-fat, low-cholesterol foods with elevated cholesterol. Use plant stanols and sterols in the diet.
- Reduce the energy intake by reducing the energy density of foods, increasing fresh fruits and nonstarchy vegetables low-calorie versions of products. Decrease the use of sweets as snack foods or dessert. Decrease the use of fatty or fried foods.
- Include sources of iron, B-complex vitamins, vitamin C, and protein, if needed to correct anemias.
- Limit milk to a reasonable daily amount for the age. Be sure others foods are consumed in addition to milk. Use low-fat or skim milk after 2 years of age.
- Limit juice to 6 oz for young children. Limit sugar-sweetened beverages in general.
- Added sugars in foods/beverages should comprise <25% of total calories consumed.
- Control between-meal snacks; offer fresh fruit or vegetables, plain crackers, pretzels, plain popcorn, cooked egg slices, unsweetened fruit or vegetable juices, and low-fat cheese cubes. Age-appropriate snacks are important; avoid popcorn and other foods that may cause choking in young children.
- Give small helpings at meals; allow more small helpings until "full." Discuss "hunger cues" and "satiety cues."
- Ensure that the family has adequate fluoridated water, as dental caries are common.

### Common Drugs Used and Potential Side Effects

- Discourage the use of drugs for weight loss; no diet medicines are safe for children younger than 16 years. Sibutramine added to a comprehensive behavioral program for teens can induce significant weight loss.
- Antidepressants are sometimes prescribed for childhood depression, which is common. Side effects such as changes in metabolism and appetite can contribute to obesity in some children. Both childhood depression and anxiety are associated with increased BMI percentiles; childhood psychopathology is an important factor that should be carefully monitored (Rofey et al, 2009).

### Herbs, Botanicals, and Supplements

- Herbs and botanicals should not be used for childhood obesity; there are no controlled trials to prove efficacy or safety.
- Physicians may ask dietitians to discuss herbs, botanicals, life cycle, and disease-specific and obesity guidance with their patients.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Responsibilities should be shared. Parents are responsible for a proper emotional setting and for what is offered; the child is responsible for what and how much is eaten (Satter, 2009). Emphasis becomes *supporting each child's normal growth* (Satter, 2009). See Table 3-15 for more tips.
- Intervention should start early and focus on the family, not just the child. Educate parents about the dangers of medical complications.
- Discuss age-appropriate portions and snacks. Many parents innocently overfeed their children; show them child-sized plates and utensils with sample portion sizes.
- Try to alter intake of one "problem food" per visit (regular soda or sugar-sweetened fruit punch). Diluting juice, substituting lower calorie beverages, and calculating number of calories saved can be quite effective. Reading labels is a useful teaching tool.
- Tailor treatment and prevention efforts for each person. Interactive interventions and self-monitoring are keys to success for many individuals (Lombard et al, 2009). Internet-based coaching sessions may be quite helpful as they can be tailored to the individual's needs and concerns (Block et al, 2008).
- Integrate culturally appropriate approaches and strategies. Bilingual professionals might help with developing culturally sensitive programming.
- Encourage regular family meals whenever possible; limit unplanned or habitual snacking. Between meals, ice water can be offered as a special treat instead of sweetened beverages. Good role modeling by parents is essential. Maintain the child's self-image through positive reinforcement.
- Discuss the relationship of food, weight, and energy balance. Metabolic rates are low while watching television. When working with families to prevent and treat childhood

**TABLE 3-15 Components of Successful Weight Loss for Children**

| Component | Comment |
|---|---|
| Reasonable weight loss goal | Initially, a rate of 1 lb per month if <95th or >85th percentile with comorbidity, based on age. |
| Dietary management | Guide family choices rather than dictating; encourage child to eat when hungry and to eat slowly. Encourage family meals. Avoid using food as reward or withholding as punishment. Drink plenty of water and limit sugar-sweetened beverages. Plan healthy snacks. Aim for five servings of fruits and vegetables each day. Promote healthy breakfast each day. Consume milk with dinner instead of soft drinks. |
| Physical activity | Begin according to child's fitness level, with ultimate goal of 60 minutes of moderate activity daily. |
| Behavior modification | Teach self-monitoring, nutritional education, control of cues, modification of eating habits, physical activity, attitude change, reinforcements, and rewards. |
| Family involvement | Review family activity and television viewing patterns; involve parents in nutrition counseling. |

Adapted from: Mullen MC, Shield J. *Childhood and adolescent overweight: the health professional's guide to identification, treatment and prevention*. Chicago, IL: American Dietetic Association, 2004.

weight problems, one should attend to children's time spent with screen media, the frequency of family mealtimes, and parents' perceptions of neighborhood safety for children's outdoor play (Gable et al, 2007). Limit television and nonproductive computer time to <2 hours daily (American Academy of Pediatrics, 2009). Encourage activity, such as jogging, ball games, swimming, bike riding, and school-based physical activities, since many children who enter adolescence overweight will become overweight or obese adults. Dance videos are easy to do at home, especially for children who are self-conscious.

- Discourage potentially dangerous weight-control schemes or practices.
- A system for "traffic light" foods can be used for younger children: green for "go" foods, yellow for "caution" foods, and red for "stop" foods.
- Parents who practice restrained eating with their children tend to be overly indulgent later (fast/feast); the result is chronic anxiety. Eating can become very controlled, inconsistent, and emotional. Highlight nonfood-related achievements; avoid nagging about diet or food.
- In extreme obesity, bariatric surgery may be needed. Improved quality of life generally occurs for teens (Murray, 2008). However, there are profound implications and risks that differ from adults; they may have greater weight regain and may be noncompliant with treatment after surgery (Levitsky et al, 2009). Research on nonsurgical treatments is on-going.

### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing meals.
- Before using tap water as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard beverage or food that has been left at room temperature for 2+ hours.

### *For More Information*

- Blueprint for Physical Activity
  http://movingtothefuture.org/frontpage_files/55/55_frontpage_file1.pdf
- CDP
  http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overwght99.htm
- Center for Health and Health Care in Schools
  http://www.healthinschools.org/Health-in-Schools/Health-Services/Schools-and-Childhood-Overweight.aspx
- CDC Charts for BMI in Children and Teens
  http://apps.nccd.cdc.gov/dnpabmi/
- CDC Contributing Factors in Childhood Obesity
  http://www.cdc.gov/nccdphp/dnpa/obesity/childhood/contributing_factors.htm
- Ellyn Satter Institute
  http://www.ellynsatter.com/
- Healthy Youth
  http://www.cdc.gov/HealthyYouth/nutrition/facts.htm
- International Food Information Council
  http://ific.org/nutrition/obesity/index.cfm
- Maternal and Child Nutrition Center
  http://www.mchlibrary.info/KnowledgePaths/kp_overweight.html
- MyPyramid for Kids
  http://www.cnpp.usda.gov/MyPyramidforKids.htm
- NIDDK Weight Control Network
  http://win.niddk.nih.gov/publications/over_child.htm
- University of Minnesota Nutrition Curriculum
  http://www.epi.umn.edu/let/nutri/chobese/assess.shtm

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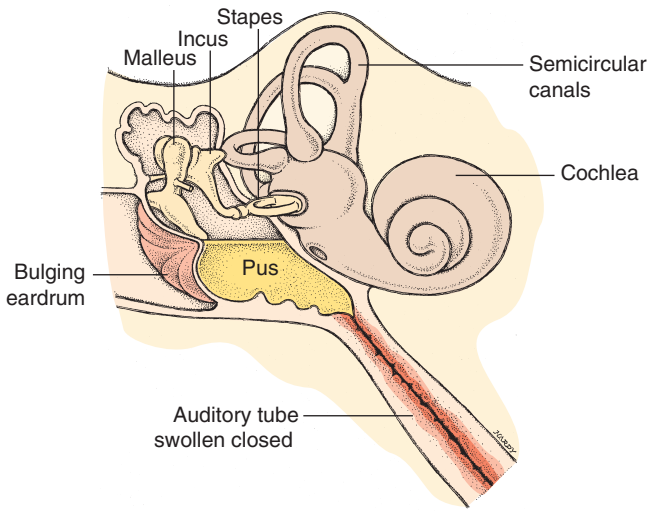
# OTITIS MEDIA

## NUTRITIONAL ACUITY RANKING: LEVEL 1




## DEFINITIONS AND BACKGROUND

Acute OM (acute middle ear infection) occurs when there is a bacterial or viral infection of the fluid of the middle ear that leads to production of pus, excess fluid, or even bleeding in the middle ear. The Eustachian tube becomes clogged. Tubes of the ear of a child are shorter and less slanted than in adults, allowing bacteria and viruses to find their way into the middle ear more easily. Pressure from fluids associated with OM may cause the eardrum to rupture.

Ear infections often occur along with respiratory infections (such as *H. influenza*) or with blocked sinuses and Eustachian tubes caused by allergies. OM with effusions (OME) can lead to significant hearing loss in children if not properly treated. Recent illness of any type and lowered immunity; crowded or unsanitary living conditions; genetic factors; cold climate and high altitude; and bottle feeding of infants (can allow fluid to pool in the throat near the Eustachian tube) may be etiologies. LBW infants may be prone to repeated ear infections.

Breast milk is more protective than formula feeding (Ip et al, 2007). Breast milk contains lactoferrin and a number of other anti-inflammatory factors (Hanson, 2007). The prevalence of early-onset and repeated OM continues to increase among preschool children; an increase in prevalence of allergic conditions is a concern (Auinger et al, 2003).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Susceptibility to infections may run in families.

| Clinical/History | I & O | Lab Work |
|---|---|---|
| Height | Fever? | H & H |
| Birth length | Irritability, fussiness? | Serum Fe |
| Birth weight | Exposure to cigarette smoking? | Chol, Trig |
| Present weight | Audiometry | Gluc |
| Diet/intake history | | Alb |
| Family history of asthma, allergies | | $Na^+$, $K^+$ |
| | | Serum IgE and allergy testing |

## INTERVENTION

### OBJECTIVES

- Promote breastfeeding of newborns, especially for 6 months or longer.
- If formula fed, babies should be positioned in a semi-upright position so that the milk will flow downward into the baby's stomach and not wash up and into the baby's nasal passages and up through the Eustachian tubes. Always position the infant's head higher than the stomach.
- In older children, monitor nutrient density of the diet to maintain a healthy immune system. Inclusion of more fruit and a children's multivitamin–mineral supplement may be needed for poor eaters or during food jags.
- Prevent chronic suppurative OM and hearing loss.

### FOOD AND NUTRITION

- Determine the recommended allowances for the child's age group: kilocal, protein, and other nutrients. Plan a reasonable menu pattern accordingly.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Intake of Unsafe Foods**

***Assessment Data:*** Diet and intake history, recent lab testing indicating allergy to milk and eggs, frequent ear infections, notation that infant was not breastfed after 3 weeks of age.

***Nutrition Diagnoses (PES):*** Intake of unsafe foods related to recent identification of specific food allergies (milk, eggs) as evidenced by six ear infections in past 2 years.

***Intervention:*** Educate parents about foods that are to eat/prepare; food labeling; choices at restaurants, schools. Counsel about adapting recipes and holiday meals that suit the family.

***Monitoring and Evaluation:*** Alleviation of responses to food allergens, improvement or elimination of food allergy reactions at home, at school, and out at restaurants. Improved nutritional quality of life as observed by parent(s).

- Highlight foods that include sufficient levels of iron, vitamins A, $D_3$, and C, and zinc to support a healthy immune system to fight further infections. Studies are under way to determine if antioxidant-rich foods make any difference in healing.
- If child has food allergies, discuss options for maintaining a healthy diet, especially if large food group categories must be eliminated.

### Common Drugs Used and Potential Side Effects

- Antibiotics such as penicillin are often prescribed for bacterial infections; side effects may include rash, vomiting, and diarrhea. Viral infections have to run their course.
- Vaccines may be available for some of the bacterial agents that cause OM.

### Herbs, Botanicals, and Supplements

- Xylitol may have anti-adhesive factors for reducing infections, but no commonly available products have been tested.
- Probiotics in foods such as yogurt can help to replenish the gut and support healthy immunity.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain to parents that overfeeding can aggravate asthma, which is often triggered by bouts of OM. If needed, teach principles for managing food allergies and asthma.
- Toddlers should not be taking a bottle to bed.
- Discuss the role of nutrition and immunity.
- Chronic recurrence should be addressed with an ear-nose-throat specialist to prevent hearing loss and speech delay.
- Smoking around the child should be discontinued (Kuiper et al, 2007).
- Use of pacifiers, cow's milk allergy, environmental smoke or mold, and other issues may be problematic. More clinical trials are needed to determine the best advice.

### *For More Information*

- Family Doctor
  http://familydoctor.org/055.xml
- I-tonsil
  http://www.itonsil.com/index.html
- National Speech and Hearing Association
  http://www.asha.org/public/hearing/disorders/causes.htm
- NIH Health and Hearing
  http://www.nidcd.nih.gov/health/hearing/otitism.asp

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Kuiper S, et al. Interactive effect of family history and environmental factors on respiratory tract-related morbidity in infancy. *J Allergy Clin Immunol.* 120:388, 2007.

# PHENYLKETONURIA

## NUTRITIONAL ACUITY RANKING: LEVEL 4

### DEFINITIONS AND BACKGROUND

Phenylketonuria (PKU) is a rare, inherited condition that occurs in one in 10,000 births. Phenylalanine (Phe) is not metabolized to tyrosine because of a mutation in Phe hydroxylase. Infants are tested for this disorder after birth and after the first feeding and again if levels of Phe are above given cutoff levels (≥6 mg/dL). If very strict diets are followed early and continually, normal development and life span are possible. Children with PKU who follow their special diet for life have fewer intellectual and neurological deficits; do not discontinue at any age. Refer to a metabolic dietitian or special programs at the state level.

Desirable serum Phe levels are below 10 mg/dL; higher levels are associated with declining IQ. There is international consensus that patients with Phe levels <360 μM on a

free diet do not need Phe-lowering dietary treatment, whereas patients with levels >600 μM do. In general, however, "diet for life" is the rule, especially for women with PKU who are considering pregnancy.

Tyrosine is an essential amino acid in patients with PKU because of the limited Phe converted to tyrosine. Treatment with large neutral amino acid supplements may help to correct low or deficient blood concentrations of both tyrosine and tryptophan, which are precursors for dopamine and serotonin (Koch et al, 2003). Individuals who follow diets low in natural proteins should be advised to take selenium and iron supplements (Acosta et al, 2004).

Serum lipids are usually under good control because of the vegetarian-type diet needed for PKU (Schulpis et al, 2003). However, micronutrient status of folic acid and vitamins $B_6$ and $B_{12}$ can be low, and there may be a risk for coronary artery disease.

Tetrahydrobiopterin (BH4) is the natural cofactor that fuels the activity of the phenylalanine hydroxylase (PAH) enzyme. When BH4 is given orally to some people with PKU, the activity of mutated PAH improves; this is "BH4-responsive PKU" in which lowering of Phe by 30% has been seen (Michals-Matalon, 2008; Michals-Matalon et al, 2007). This gives an exciting new treatment for PKU for those who are responsive.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Mutation in the phenylalanine hydroxylase (PAH) gene.

**Clinical/History**

- Birth weight
- Present weight
- Growth (%)
- Diet/intake history
- Developmental delay
- Retardation?
- Musty odor in urine and sweat
- Length/height
- Dry skin or dermatitis
- Blonder hair than siblings
- Electroencephalograms (EEGs)
- Seizures

**Lab Work**

- Response to dose of 10 mg/kg BH4
- Plasma Phe
- Urinary Phe
- Plasma tyrosine
- H & H, serum Fe, ferritin
- Serum pyridoxine, vitamin $B_{12}$, folic acid
- Serum carnitine (may be low)
- Serum zinc and selenium

## INTERVENTION

### OBJECTIVES

- Prevent toxic buildup of abnormal metabolites to prevent mental retardation, and to promote normal intellectual and social development.
- Establish the child's daily requirement for Phe, protein, and energy according to age. The appropriate Phe intake for age is as follows: infants 0–3 months, 60–90 mg/kg; infants 4–6 months, 40 mg/kg; infants 7–9 months, 35 mg/kg; infants 10–12 months, 30 mg/kg; children 1–2 years, 25 mg/kg; and children 2+ years, 20 mg/kg of body weight.
- Provide a diet aiding growth and development with a high energy to protein ratio to spare protein.
- Introduce solids and textures at usual ages; encourage self-feeding when it is possible for the infant.
- Establish a positive attitude toward the modified diet for parents, caretakers, and the child.
- Monitor for deficiency in nutrients, such as DHA, vitamin $B_{12}$, folic acid, selenium, and iron.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Inappropriate Intake of Types of Amino Acids**

***Assessment Data:*** Diet intake records, serum lab values of Phe, previous education on appropriate diet for PKU management; negative response to BH4 administration.

***Nutrition Diagnosis:*** Inappropriate intake of amino acids related to low comprehension of nutrition care plan as evidenced by serum Phe level of 12 mg/dL, where >8 mg/dL indicates loss of dietary control in PKU.

***Intervention:*** Counseling on Phe in diet, use of special formulas and products, referral to State Health Department for resources and financial support, referral to child health clinics.

***Monitoring and Evaluation:*** Serum Phe reports, changes in mental health status and alertness.

### FOOD AND NUTRITION

- Use a diet low in Phe. Use special milk substitutes made from casein hydrolysate, corn oil, corn syrup, tapioca starch, minerals, and vitamins: Phenyl-free, or Maxamaid XP. Phenyl-free does not provide total nutritional needs. Phlexy-10 is available from SHS North America.
- Initially, the infant's tolerance must be assessed individually, and progress in treatment must develop accordingly. A small amount of milk and 85–100% from specialty formula are used to meet the infant's needs. Subtract Phe requirement in formula from total needs (the difference is that which is provided by solid foods).
- Glycomacropeptide (GMP), an intact protein formed from cheese whey, contains minimal Phe and can be supplemented with limiting AAs as a safe and highly acceptable alternative to synthetic AAs as the primary protein source in PKU (van Calcar et al, 2009). It is now recognized that the phenalalanine:tyrosine ratio affects executive function in PKU, so care must be taken to plan the diet properly (Sharman et al, 2009).
- Determine if serum iron, vitamin $B_{12}$, folic acid, selenium, or other nutrient levels are low and enhance diet or use a multivitamin–mineral supplement as needed.
- Introduce solids and textures at the appropriate ages. Omit meat, fish, poultry, bread, milk, cheese, legumes, and peanut butter from the diet of older children. Try

using low-protein bread, pasta, crackers, cookies, and muffin mixes.
- To add calories, try jam, jelly, sugar, honey, molasses, syrups, cornstarch, and oils that are Phe-free. Flavors can be added to the formula to continue its use as a beverage.
- Fish oil supplements may be used to replace the DHA missing from a standard Phe-free diet; this helps improve neurologic development and fine motor coordination (Beblo, 2007). The exact amount of DHA needed is not known (Koletzko et al, 2009).

## Common Drugs Used and Potential Side Effects

- Kuvan (phenoptin) contains sapropterin dihydrochloride, a synthetic dihydrochloride salt of naturally occurring tetrahydrobiopterin (BH4). Individuals with PKU should be tested to determine if they could benefit from taking this medication (Michals-Matalon, 2008; Trefz et al, 2009).

## Herbs, Botanicals, and Supplements

- Herbs and botanical products are not recommended for use in PKU.
- Fish oil supplements may be beneficial (Koletzko et al, 2009; Beblo, 2007).

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Because initial acceptance of formula may be poor due to its strong taste, the mother should be careful not to express her own distaste. Recommend appropriate recipes and cookbooks.
- Monitor the calculation of Phe in the diet. Avoid items sweetened with aspartame (NutraSweet), including diet sodas.
- School challenges vary, but the diet provides good control for most cases (Filiano, 2006). Attention deficit disorder can present as the child grows older.
- Self-management should begin by 7–8 years of age, at least for formula preparation. By 12 years of age, the child should begin calculating his or her own intake of Phe from foods.
- Women who have PKU tend to give birth to children with microcephaly, mental retardation, congenital heart defects, and IUGR. Metabolic control by the end of the first trimester is, therefore, important as a goal. Treatment at any time during pregnancy may reduce the severity of delayed development. Referral to a metabolic dietitian is highly recommended.

### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing formula or meals. Use clean utensils and containers for mixing formula.
- Before using tap water for formula preparation or to give as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard any beverage or food that has been left at room temperature for 2 hours or longer.
- Do not use honey in the diets of infants to decrease potential risk of botulism.

### *For More Information*

- Children's PKU Network
  http://www.kumc.edu/gec/support/pku.html
- Diet Tips for PKU
  http://www.pkunews.org/
- March of Dimes—PKU
  http://www.marchofdimes.com/professionals/14332_1219.asp
- National Coalition for PKU and Allied Disorders
  http://www.pku-allieddisorders.org/
- PKU News
  www.pkunews.org
- Save babies
  http://www.savebabies.org/diseasedescriptions/pku.php

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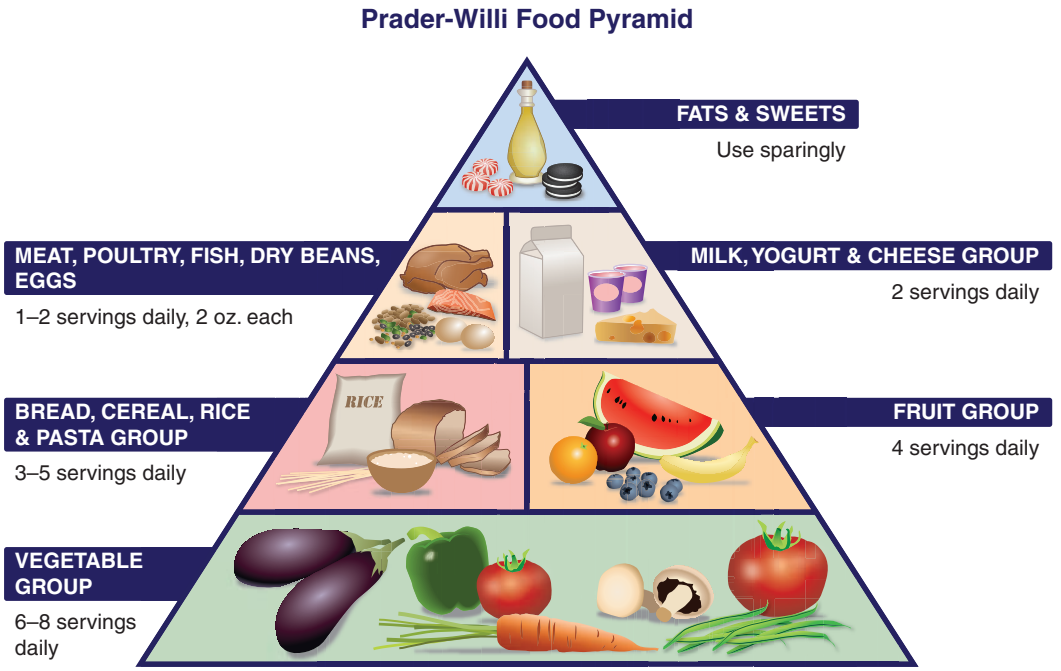
# PRADER-WILLI SYNDROME

## NUTRITIONAL ACUITY RANKING: LEVEL 3–4




Prader-Willi Food Pyramid http://www.pwsausa.org/syndrome/foodpyramid.htm.

### DEFINITIONS AND BACKGROUND

Prader-Willi syndrome (PWS) is a disorder caused by DNA abnormalities of chromosome 15. Major characteristics are infant hypotonia, hypogonadism, mental retardation (average IQ is around 70), small hands and feet, atypical facial features, and obesity because of insatiable appetite in early childhood. Short stature is part of the syndrome and is not nutritional in origin.

The incidence of PWS is 1 in 10,000–16,000 births in the United States. Onset occurs at birth, but symptoms begin by 1–4 years of age. PWS infants often present with absence of crying, poor suck, lethargy, and floppy muscle tone (hypotonia). Motor development is delayed. Hyperphagia results in marked obesity with high risk of metabolic and cardiovascular complications (Schmidt et al, 2008). Lifelong morbidities include osteoporosis, type 2 diabetes, respiratory disorders, and cardiorespiratory failure related to obesity and hypotonia (Allen and Carrel, 2004). Sexual development is incomplete; most PWS individuals are infertile.

Ghrelin levels are high in PWS (DelParigi et al, 2002). It is produced mostly by the stomach but also by the pituitary, hypothalamus, GI tract, lung, heart, pancreas, kidney, and testis. Ghrelin stimulates GH secretion, appetite and food intake, fat mass deposition, and weight gain gastric motility and acid secretion; exerts cardiovascular and anti-inflammatory effects; modulates cell proliferation; and influences endocrine and exocrine pancreatic secretion, as well as glucose and lipid metabolism.

Early dietary treatment starting at the second year of life and continued until the age of 10 years is effective in avoiding excessive weight gain in patients with PWS, but results in shorter stature; GH may be useful (Eiholzer and Whitman, 2004; Schmidt et al, 2008). Individuals with PWS are not able to control their food sneaking, stealing, and gorging behaviors. Because they are difficult to manage, approximately 75% of PWS patients live in group homes. Distinction of behavioral problems from psychiatric illness is important as well (Goldstone et al, 2008).

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** The OCA2 gene is associated with PWS in the long arm of chromosome 15 (region 15q11–q13). In 99% of cases of PWS, the child has two chromosome regions that are both maternally inherited; DNA from the father is missing for chromosome 15 (Isaacs and Zang, 2007).

| | | |
|---|---|---|
| **Clinical/History** | FTT, then rapid weight gain between 1 and 6 years of age | BMI |
| Height | | Slow mental development |
| Birth weight | | Almond shaped eyes |

Irregular areas of skin (bands, stripes, or lines)
Small head, hands, feet
Sleep apnea
Asthma?
Hypotonia, floppy limbs
Down-turned mouth
Hyperphagia
Light hair, eyes

**Lab Work**

DNA-based diagnostic testing of chromosome 15
Hyperghrelinemia
Gluc, Glucose tolerance test (GTT)
Alb
BP
Chol, Trig
LFTs $pCO_2$, $pO_2$
H & H
Serum Fe

## INTERVENTION

### OBJECTIVES

- Reduce excess weight. Monitor weight weekly.
- In preschool children, prevent obesity.
- Maintain recommended dietary intakes for all nutrients, especially protein to promote growth and development.
- Provide feeding assistance if needed.
- Prevent complications including CHD, hypertension, diabetes, sleep apnea, dental problems, and pneumonia. Correct serum lipid levels if elevated.
- Minimize unusual food-seeking behaviors such as eating food from the trash or eating inappropriate or unpalatable food combinations. Correct pica and related nutritional deficits, especially iron.
- Promote an exercise program.

### SAMPLE NUTRITION DIAGNOSES

**PWS**

***Assessment Data***: Diet, weight, and physical activity histories.

***Nutrition Diagnosis:*** Obesity related to excessive nutrient intake as evidenced by BMI of 35 and nutrition history indicating consumption of 2900 kcal/d and sneaking of foods between meals.

***Interventions:***

Food and Nutrient Delivery:

ND 1.3. Use of the Prader-Willi food pyramid to plan meals.

ND 5.7. Adjust availability and locations of foods kept in the house to minimize food sneaking.

Education:

E 2.5. Teach portions based on the PW food pyramid. Give examples of meals and snacks based on total servings from food groups; provide examples of age-appropriate physical activities.

Counseling:

Appropriate nutrient intake, enhancing physical activity, and keeping logs. Use of "Go-Caution-No" foods in color-coded "stoplight" system.

***Monitoring and Evaluation:*** Have patient return in 1 month to assess weight, diet, and activity logs.

### FOOD AND NUTRITION

- Often, these children start with FTT and then become obese by age six; identify where the child is on this continuum. Gavage feeding may be needed for infants.
- Use 10–11 kcal/cm of height to maintain weight; 8.5 kcal/cm for slow weight loss (Lucas, 2004, p. 41). For older teens, reduce the total calorie level to 7–8 kcal/cm for weight loss, or 10–14 kcal/cm to maintain). Patients' needs are about 60% of those without PWS.
- Ensure that the diet provides adequate protein and nutrients with RDAs for age.

### Common Drugs Used and Potential Side Effects

- Weight loss products have not proven to be useful in this population. Drugs may react differently and are, therefore, not recommended.
- GH may be used to correct short stature and hormone replacement therapy may be used to improve signs of osteopenia or osteoporosis (Allen and Carrel, 2004; Schmidt et al, 2008).
- Ghrelin antagonists are being developed. Ghrelin peaks are related to habitual meal patterns and tend to rise in anticipation of eating rather than eliciting feeding (Frecka and Mattes, 2008).

### Herbs, Botanicals, and Supplements

- Persons with PWS may be more sensitive; small doses of herbs and drugs may cause a greater reaction than in other people (see http://www.pwsausa.org/syndrome/herbal.htm). Therefore, herbs and botanicals should not be used.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss feeding practices plus activity factors. Encourage daily activity.
- Behavior modification is an important part of treatment. Help the patient lose weight with specific behavior modification techniques; teach the green/yellow/red (go/caution/stop) method for food choices.
- Record keeping and calorie counting are useful. Control of excess intake is the main goal; locked refrigerators or cupboards may be needed.
- An interdisciplinary approach is useful (Eiholzer and Whitman, 2004). There is a need to reduce guilt and depression; self-monitoring is the eventual goal.

#### *For More Information*

- Heimlich Maneuver—for choking
  http://www.heimlichinstitute.org/howtodo.html#chokingAnchor
- NIH—Prader Willi
  http://ghr.nlm.nih.gov/condition=praderwillisyndrome/show/NIH+Publications
- Prader Willi Pyramid
  http://www.pwsausa.org/syndrome/foodpyramid.htm
- Prader-Willi Syndrome Association
  http://www.pwsausa.org/

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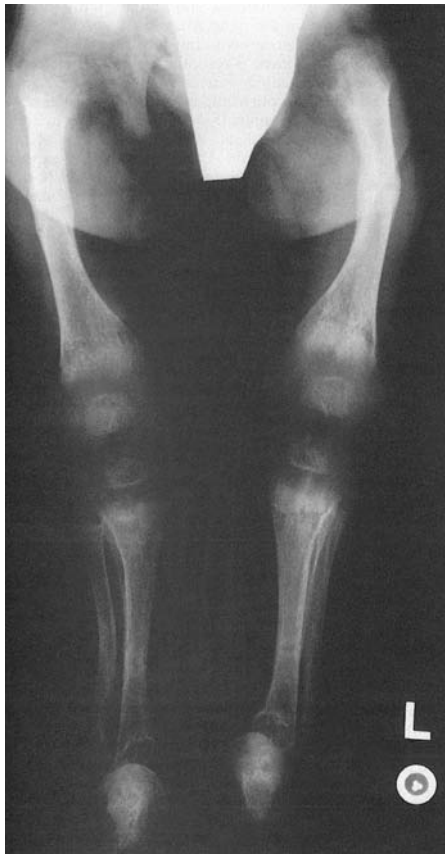
# RICKETS

## NUTRITIONAL ACUITY RANKING: LEVEL 3




Adapted from: Becker KL, Bilezikian JP, Brenner WJ, et al. *Principles and Practice of Endocrinology and Metabolism,* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2001.

## DEFINITIONS AND BACKGROUND

Rickets is generally caused by failure of osteoid to calcify during periods of growth. Vitamin $D_3$ (cholecalciferol) is formed in the skin from cholesterol with stimulation from ultraviolet-B light. Calcitriol is a steroid hormone that has impact on over 1000 genes in the human body (Edlich et al, 2009). Vitamin D insufficiency and deficiency during pregnancy may lead to lower maternal weight gain, disturbed skeletal homeostasis in the infant with reduced bone mineralization, rickets, and fractures (Pawley and Bishop, 2004). Less often, a deficiency of calcium or phosphorus may contribute to rickets. In adults, the condition is known as osteomalacia.

Because sunlight is important to skin production of vitamin D, where exposure is limited, deficiency is likely. Prevention and treatment of vitamin D deficiency is accomplished by regulated sun exposure as well as vitamin D supplementation (Edlich et al, 2009). Natural vitamin D levels, those found in humans living in a sun-rich environment, are between 40 and 70 ng/mL (Cannell and Hollis, 2008).

Rickets can be seen in 30–70% of premature, LBW and VLBW infants; it is also seen in breastfed children from multiple births, and infants with darker skin pigmentation living at higher latitudes. An increased number of children have rickets because more African American women are breastfeeding, fewer infants receive vitamin D supplements, and mothers and children are exposed to less sunlight.

Rickets may also occur in fat malabsorption syndrome, steatorrhea, anticonvulsant use, renal failure, or biliary cirrhosis. One detrimental consequence of untreated vitamin D-rickets is dilated cardiomyopathy (Brown et al, 2009). It is, therefore, important to identify and treat vitamin D deficiency.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** X-linked hypophosphatemic rickets (XLH) is related to the PHEX gene and is the most common cause of vitamin D–resistant rickets.

**Clinical/History**

- Height
- Weight
- Growth (%)
- Diet/intake history
- Decreased linear growth
- Steatorrhea
- Muscle spasm
- Chvostek's sign (facial spasm)
- Metabolic acidosis
- DEXA scan for bone density
- Wrist radiographs

| | Lab Work | |
|---|---|---|
| Radiographs for fractures | 25(OH) D levels: sufficient ≥30 ng/mL; deficient ≤20 ng/mL | Hypophosphatemia? |
| Leg bowing, inability to walk or stand | Urinary $Ca^{++}$ (elevated) | Serum $Ca^{++}$ (often low) |
| Seizures or irritability | Alk phos (increased) | Parathyroid hormone (elevated) |
| | | $Mg^{++}$, $Na^{+}$, $K^{+}$ |

## INTERVENTION

### OBJECTIVES

- Correct body mineral status; prevent further problems and deformity. Vitamin D participates in mineral homeostasis, regulation of gene expression, and cell differentiation. Complement drug therapy with adequate diet.
- Prevent or correct hypocalcemia, dental caries, bone fractures.
- Promote growth; short stature can result if not treated early enough.

### FOOD AND NUTRITION

- Use vitamin D–fortified milk if there are no milk allergies or lactose intolerance. Use calcium-containing foods such as cheeses, yogurt, fortified juice and ice cream, if fluid milk is not tolerated.
- Consuming vitamin D–fortified foods improves 25(OH)D concentrations. Fatty ocean fish are better sources than most other foods; hence, cod liver oil used to be given to children in past generations.
- If diet is inadequate in the specific nutrients, ensure intake of a supplement appropriate for age and sex. Avoid excesses of phytate from high fiber diets.
- Follow guidelines for sensible sun exposure and supplemental vitamin D; 800–1000 IU/d is reasonable (Holick, 2008). The skin forms vitamin D using 5-dihydrotachysterol, then hydroxylating in the liver to vitamin $D_2$ (calcidiol, or 25-hydroxycholecalciferol), which circulates in the plasma. The active form is hydroxylated in the kidney into cholecalciferol (calcitriol or 1,25-dihydroxycholecalciferol). Note that a healthy liver and kidney must be available to make the active form.

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Vitamin Intake**

***Assessment Data:*** Low serum 25(OH) $D_3$, intake history for young child, X-rays.

***Nutrition Diagnoses (PES):*** Inadequate vitamin D intake related to low intake of fortified foods including dairy products as evidenced by low serum levels, X-ray showing bowed legs and rickets.

***Intervention:*** Educate mother about use of vitamin $D_3$ in fortified foods including milk and cereals daily. Discuss good sources of supporting nutrients (calcium, etc.).

***Monitoring and Evaluation:*** X-rays, serum 25(OH) $D_3$, food diary and intake records showing improved intake of vitamins and minerals.

## Common Drugs Used and Potential Side Effects

- A large dose of vitamin D is given upon a rickets diagnosis; 2000–7000 IU vitamin D per day should be sufficient (Cannell and Hollis, 2008).
- With steatorrhea, check serum levels of vitamin D and calcium and supplement appropriately.
- Rickets may occur secondary to prolonged antacid, anticonvulsant, or furosemide (Lasix) use; a vitamin D supplement will be needed.

## Herbs, Botanicals, and Supplements

- Sunlight, artificial ultraviolet B (UVB) radiation, and vitamin $D_3$ supplementation are sources of vitamin D (Cannell and Hollis, 2008).
- Long- term harm from higher doses of vitamin D is not clear (Cranney et al, 2008). No more than the UL should be taken.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss needed alterations of the diet in conjunction with drug therapy.
- Good posture and positioning are important aspects of treatment.
- The recommended intakes for vitamin may not be enough, especially for dark-skinned children and those who live in northern latitudes (Misra et al, 2008; Ward et al, 2007). Discuss the role of sunlight in vitamin D metabolism.
- Infants who are given vegan diets may have low intakes of vitamin D.

#### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing formula or meals. Use clean utensils and containers for mixing formula.
- Before using tap water for formula preparation or to give as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard any beverage or food that has been left at room temperature for 2 hours or longer.
- Do not use honey in the diets of infants to decrease potential risk of botulism.

#### *For More Information*

- American Academy of Family Physicians
  http://familydoctor.org/online/famdocen/home/children/parents/special/bone/902.printerview.html

- NIH—Medline
  http://www.nlm.nih.gov/medlineplus/rickets.html
- Vanderbilt—History of Rickets
  http://www.mc.vanderbilt.edu/biolib/hc/nh8.html

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# SMALL FOR GESTATIONAL AGE INFANT AND INTRAUTERINE GROWTH RETARDATION

## NUTRITIONAL ACUITY RANKING: LEVEL 3

### DEFINITIONS AND BACKGROUND

Infants whose weight is less than 10th percentile for gestational age are "small for gestational age" (SGA). Each year, about 40,000 infants born in the United States are SGA; they are at risk for preterm delivery, perinatal asphyxia, meconium aspiration, and hypoglycemia (Zaw et al, 2003). Another name for SGA infants is intrauterine growth restriction (IUGR). There is usually a wide range in weight gain after birth in SGA infants.

Nongenetic causes may retard intrauterine growth but are not often apparent before 32–34 weeks of gestation. Growth retardation due to nongenetic factors may cause malnutrition while sparing growth of the brain and long bones. Some other genetic disorders and congenital infections result in total growth retardation, in which height, weight, and head circumference are equally affected.

IUGR results from placental insufficiency. This insufficiency can result from maternal diseases (hyperemesis, preeclampsia, primary hypertension, renal disease, or diabetes); from infections such as cytomegalovirus, rubella virus, or *Toxoplasma gondii*; or if the mother is a narcotic or cocaine addict or heavy user of alcohol or tobacco (Dodds et al, 2006) (Table 3-16).

**TABLE 3-16 Risk Factors for Developing IUGR in Pregnancy**

Pregnancies that have any of the following conditions may be at a greater risk for developing IUGR:

- Maternal weight of less than 100 lb
- Poor nutrition during pregnancy
- Birth defects or chromosomal abnormalities
- Use of drugs, cigarettes, and/or alcohol
- Pregnancy induced hypertension (PIH)
- Placental abnormalities
- Umbilical cord abnormalities
- Multiple pregnancy
- Gestational diabetes in the mother
- Low levels of amniotic fluid or oligohydramnios

Source: American Pregnancy Web site at http://www.americanpregnancy.org/pregnancycomplications/iugr.htm, accessed May 10, 2009.

The fetus needs glucose, amino acids, and oxygen to grow normally. If IUGR was caused by chronic placental malnutrition, SGA infants may demonstrate remarkable catch-up growth within the first 2–3 years after delivery, if provided with adequate nutrition. The rates of catch-up growth vary according to many factors including birth weight, gestational age, parental size, adequacy of intrauterine growth, neurological impairment, clinical course, and nutrition (Carver, 2005). Insulin-like growth factor has a critical role in mediating fetal and postnatal growth (Randhawa and Cohen, 2005).

Common complications in SGA infants include hypoglycemia, perinatal asphyxia, meconium aspiration, polycythemia, respiratory distress syndrome, and NEC (Dodds et al, 2006). Prognosis is quite serious for infants who have perinatal asphyxia or congenital conditions.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** SGA is not a genetically oriented condition.

| Clinical/History | | Lab Work |
|---|---|---|
| Prenatal ultrasound | Meconium aspiration? | Glucose |
| Length, Weight | I & O | BUN, creatinine |
| Growth (<10 percentile) | BP | Serum phosphorus |
| Decreased linear growth | Temperature | Serum $Ca^{++}$ |
| | Thin, pale | Polycythemia? |
| | Loose skin? | |

### SAMPLE NUTRITION CARE PROCESS STEPS

**Malnutrition**

***Assessment Data:*** Weight and growth charts, showing <10 percentile for gestational age.

***Nutrition Diagnoses (PES):*** Malnutrition related to intrauterine growth retardation as evidenced by weight/length percentile at 9% and SGA birth.

***Intervention:*** Educate mother about breastfeeding versus formula feeding, the need for gradual catch-up growth without overfeeding, and supporting healthy immunity.

***Monitoring and Evaluation:*** Weight records, catch-up growth rate, health status (fewer infections, illnesses over time).

## INTERVENTION

### OBJECTIVES

- Correct body mineral status; prevent further problems and deformity.
- Complement any necessary drug therapy with adequate diet. Monitor carefully for side effects.
- Identify and treat underlying congenital problems.
- Promote catch-up growth, since short stature can result if not treated early enough. Compensatory catch-up growth may continue into adolescence and adulthood (Carver, 2005). If too rapid in the first 6 months, children may be obese at age 3 (Taveras et al, 2009).
- Prevent or correct hypoglycemia, perinatal morbidity, and other complications.
- Prevent long-term consequences, such as hypertension, insulin resistance and metabolic syndrome, type 2 diabetes mellitus, cardiovascular disease, short stature, and polycystic ovary syndrome (van Weissenbruch et al, 2005).

### FOOD AND NUTRITION

- Promote exclusive breastfeeding whenever possible to promote cognitive development. At least 6 months is needed.
- While nutrient-enriched formulas that provide 22 kcal/oz are often prescribed for VLBW preterm infants after hospital discharge, for promoting greater rates of catch-up growth and increases in head circumference, studies are not as clear in SGA infants (Carver, 2005).
- Use a balanced diet appropriate for older children. Include reasonable snacks with high-quality nutritional value.
- If diet is inadequate in the specific nutrients, ensure intake of a sufficient level of vitamin D, calcium, and phosphorus for age and sex.

### Common Drugs Used and Potential Side Effects

- GH therapy for improving height in these children has been approved by the FDA; it promotes growth acceleration and normalization of height during childhood. High doses can affect carbohydrate metabolism and cause hyperglycemia.

### Herbs, Botanicals, and Supplements

- Herbs and botanicals should not be used in children.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help families adjust to special requirements for their child. The child may have diabetes and other chronic consequences from being born SGA.
- Children born SGA without postnatal catch-up are shorter and have higher weight than children of similar age, height, and sex. In addition, insufficient nutrition during the first 3 years of life is correlated with poor neurodevelopmental outcomes (Belfort et al, 2008). Discuss the importance of good nutrition in infancy.
- Prepare for future pregnancies by discussing the need to avoid alcohol and tobacco.

#### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing formula or meals. Use clean utensils and containers for mixing formula.
- Before using tap water for formula preparation or to give as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard any beverage or food that has been left at room temperature for 2 hours or longer.
- Do not use honey in the diets of infants to decrease potential risk of botulism.

#### *For More Information*

- Intrauterine Growth Retardation
  http://familydoctor.org/online/famdocen/home/women/pregnancy/fetal/313.html

## SMALL FOR GESTATIONAL AGE—CITED REFERENCES

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# TYROSINEMIA

## NUTRITIONAL ACUITY RANKING: LEVEL 4

### DEFINITIONS AND BACKGROUND

Hereditary tyrosinemia type I (HTI), a severe disease affecting primarily the liver, is caused by a deficiency of fumarylacetoacetate hydrolase (FAH). Tyrosine, Phe, and methionine build up. The condition is acute, often causing death within the first year of life. Type I needs to be treated with diet for life and is a much more severe disease than other types. This condition results in liver failure or severe nodular cirrhosis with renal tubular involvement. This form is common in Quebec, Canada; it affects 1 in 100,000 individuals.

Tyrosine accumulation can be aggravated by vitamin C deficiency, a high-protein diet, or liver immaturity. Prenatal diagnosis is possible and can be performed by measuring succinyl acetone in the amniotic fluid or FAH in amniotic fluid cells, allowing for genetic counseling. Liver transplantation may be needed.

Type II tyrosinemia is caused by a deficiency of the enzyme tyrosine aminotransferase (TAT) and affects the eyes with excessive tearing and photophobia; eye pain and redness; painful skin lesions on the palms and soles; and intellectual disability. Type III is caused by a deficiency of the enzyme 4-hydroxyphenylpyruvate dioxygenase (HPD) that presents with seizures, intermittent ataxia, and intellectual disorders.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** Deficiency of FAH, the last enzyme of tyrosine catabolism, leads to accumulation of the toxic substrate fumarylacetoacetate (FAA) in hepatocytes and renal proximal tubular cells (Jacobs, 2006). Mutations in the HPD and TAT genes cause types I and II. The gene is mapped to band 15q23–q25. About 30 distinct mutations have been reported for Type I.

**Clinical/History**

- Birth weight, present weight
- Growth (%)
- Diet/intake history
- Abdominal distention
- Hyperpigmentation
- Dermatitis
- "Cabbage-like" odor
- Rancid butter–like odor (type I)
- FTT
- Irritability
- Jaundice
- Diarrhea, bloody stools
- Annual CT or MRI of liver

**Lab Work**

- Urinary succinyl acetoacetate levels (high)
- FAH (very low?)
- Plasma Phe
- Methionine
- H & H
- Serum Fe
- Plasma tyrosine
- LFTs (elevated)
- Bilirubin (elevated)
- Phosphate
- Gluc (low?)
- Alb (often low)
- Nitisinone level

## INTERVENTION

### OBJECTIVES

- Restrict Phe and tyrosine from the diet to promote normal growth and intellectual development.
- Provide adequate vitamin C for conversion processes.
- Prevent severe liver, kidney and neurological, damage, or rickets (Jacobs, 2006).

### FOOD AND NUTRITION

- Initially, feed a Phe/tyrosine hydrolysate to infants, with small amounts of milk added to provide the minimum requirements of tyrosine and Phe. Mead Johnson product TYROS and 3200-AB; Ross product Maxamaid XPHEN, TYR; or TYROMEX-1 or TYREX from SHS can be used.
- If blood methionine levels are elevated, try PFD1 or PFD2 (Mead Johnson). Use carbohydrate supplements, vitamins, and minerals.
- Low-tyrosine/Phe diet limits foods such as cow's milk and regular formula; avoid meat, eggs and cheese. Regular flour, dried beans, nuts and peanut butter must also be limited. Focus on fruits and vegetables and the special formula.
- Supplement with vitamins C and D appropriate to the patient's age.

### Common Drugs Used and Potential Side Effects

- Nitisinone (Orfadin) reduces the toxic effects of tyrosine in the body when used along with the dietary restrictions (Santra et al, 2008). Fortunately, with this medication,

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Abnormal GI Function**

***Assessment Data:*** Altered LFTs, anorexia, irritability, cabbage-like body odor, bloody diarrhea.

***Nutrition Diagnoses (PES):*** Altered GI function related to missing FAH enzyme as evidenced by bloody diarrhea, poor appetite, altered LFTs.

***Intervention:*** Educate parents about the special formula and diet for managing tyrosinemia. Refer to genetic counselor if they wish to have more children.

***Monitoring and Evaluation:*** Normal weight and growth, LFTs, normal intellectual development after following the diet and taking the medicine. No sign of rickets.

liver transplantation may not be necessary and hepatic carcinoma may be delayed.
- Antibiotics may be needed to correct infections. Use of acidophilus and probiotic products may alleviate loss of intestinal bacteria.
- Vitamin D may be needed if the child has rickets.

### Herbs, Botanicals, and Supplements

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

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Provide sources of tyrosine and Phe in the diet determined appropriately for age and body size.
- Adjust intake of energy and nutrients according to the patient's age.
- Discuss desirable intake of protein (avoid excesses) and encourage adequate intake of vitamin C to meet recommended levels.
- Genetic counseling is advised for the family members (Scott, 2006).

#### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing formula or meals. Use clean utensils and containers for mixing formula.
- Before using tap water for formula preparation or to give as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard any beverage or food that has been left at room temperature for 2 hours or longer.
- Do not use honey in the diets of infants to decrease potential risk of botulism.

#### *For More Information*

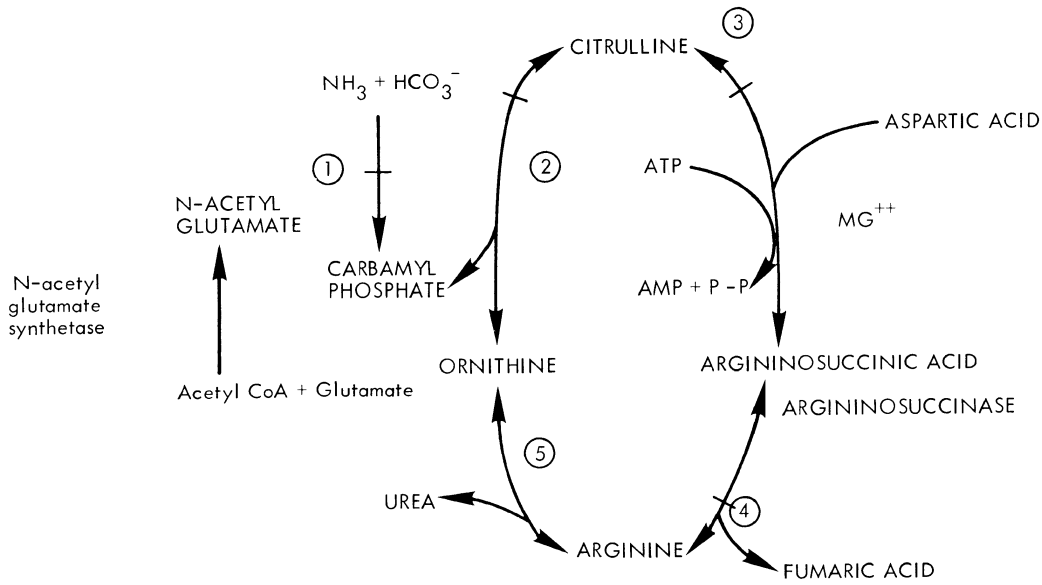
- American Liver Foundation
  http://www.liverfoundation.org/
- Medscape—Tyrosinemia
  http://emedicine.medscape.com/article/949816-overview
- Save Babies
  http://www.savebabies.org/diseasedescriptions/tyrosinemia.php
- University of Washington
  http://depts.washington.edu/tyros/abouttyr.htm

### TYROSINEMIA—CITED REFERENCES

Jacobs SM, et al. Kidneys of mice with hereditary tyrosinemia type I are extremely sensitive to cytotoxicity. *Pediatr Res.* 59:365, 2006.
Santra S, et al. Renal tubular function in children with tyrosinemia type I treated with nitisinone. *J Inherit Metab Dis.* 31:399, 2008.
Scott CR. The genetic tyrosinemias. *Am J Med Genet C Semin Med Genet.* 142:121, 2006.

# UREA CYCLE DISORDERS

## NUTRITIONAL ACUITY RANKING: LEVEL 2–4



## DEFINITIONS AND BACKGROUND

As a group, urea cycle disorders occur in one in 25,000 newborns. Table 3-17 describes the urea cycle disorders and their treatment. Ornithine transcarbamylase (OTC) deficiency is the most common disorder. Statewide newborn screening does not always include these conditions, but screening is important if there is any family history of these disorders.

The urea cycle disorders are manifested most often in the newborn between ages 1 and 5 days, when they are often initially thought to be septic. With later onset, patients have partial enzyme deficiencies and are recognized after a clinical episode months or years later. When they present in childhood, adolescence, and adulthood, there may be FTT, persistent vomiting, developmental delay, behavioral changes, hyperammonemia, irritability, somnolence, seizures, and coma; if not treated rapidly, they may cause irreversible neuronal damage. Diagnosis of urea cycle disorder should be considered in any child or adult with unexplained neurological and psychiatric disorders with anorexia, unexplained coma with cerebral edema, and respiratory alkalosis. Some cases of SIDS may be related to urea cycle disorders.

The hyperammonemia that occurs is a deadly neurotoxin. Chronic hyperammonemia results in increased L-tryptophan metabolites including serotonin. Ammonia levels above 60 μmol/L lead to anorexia, irritability, lethargy, vomiting, somnolence, disorientation, asterixis, cerebral edema, coma, and death (Cohn and Roth, 2004).

Diet is one of the main treatments of these disorders; protein intake should be adjusted according to the metabolic disorder, its severity, the patient's age, growth rate, and preferences (Wilcken, 2004). Poor appetite, nutritional problems, and chronic catabolism are common and difficult to treat (Wilcken, 2004).

Any patient on a low-protein diet should be monitored clinically, with appropriate laboratory tests and an emergency plan; hemodialysis may be needed. Most patients, except those with arginase deficiency, will need supplements of arginine, but the value of other supplements, including citrate and carnitine, is unclear (Wilcken, 2004). Gene therapy and liver transplantation are treatments that show promise.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Deficiencies of CPSI, ASS, ASL, NAGS, and ARG are inherited in an autosomal recessive manner. OTC deficiency is inherited in an X-linked manner. All are genetic diseases associated with lack of a protein or enzyme activity in the urea cycle.

**TABLE 3-17 Urea Cycle Disorders (UCD)**

| UCD | Enzyme Deficiency | Symptoms/Comments | Treatment |
|---|---|---|---|
| Type I hyperammonemia | Carbamoylphosphate synthetase I (CPS I) | Within 24–72 hours after birth, infant becomes lethargic, needs stimulation to feed, vomiting, increasing lethargy, hypothermia and hyperventilation; without measurement of serum ammonia levels and appropriate intervention, infant will die | Arginine that activates *N*-acetylglutamate synthetase |
| *N*-acetylglutamate synthetase deficiency | *N*-acetylglutamate synthetase (NAGS) | Severe hyperammonemia, deep coma, acidosis, recurrent diarrhea, ataxia, hypoglycemia, hyperornithinemia | Carbamoyl glutamate to activate CPS I |
| Type 2 hyperammonemia | Ornithine transcarbamylase (OTC) | Most commonly occurring UCD, only X-linked UCD, ammonia and amino acids elevated in serum, increased serum orotic acid due to mitochondrial carbamoylphosphate entering cytosol and being incorporated into pyrimidine nucleotides, which leads to excess production and consequently excess catabolic products | High-carbohydrate, low-protein diet, ammonia detoxification with sodium phenylacetate or sodium benzoate |
| Classic citrullinemia | Argininosuccinate synthetase (ASS) | Episodic hyperammonemia, vomiting, lethargy, ataxia, seizures, eventual coma | Arginine administration to enhance citrulline excretion; also sodium benzoate for ammonia detoxification |
| Argininosuccinic aciduria | Argininosuccinate lyase (argininosuccinase) (ASL) | Episodic symptoms similar to classic citrullinemia, elevated plasma and cerebral spinal fluid argininosuccinate | Arginine and sodium benzoate |
| Hyperargininemia | Arginase | Rare UCD, progressive spastic quadriplegia and mental retardation, ammonia and arginine high in cerebral spinal fluid and serum arginine, lysine, and ornithine high in urine | Diet of essential amino acids excluding arginine, low-protein diet |

Adapted from: http://themedicalbiochemistrypage.org/nitrogen-metabolism.html#clinical.

**Clinical/History**

Birth weight, present weight
Growth (%)
Diet/intake history
FTT
Vomiting
Irritability
Somnolence
Lethargy, seizures or coma
Developmental delay

**Lab Work**

Plasma amino acid levels (specific to disorder)
Hyperammonemia: 150 µg/dL in neonates; 70 µg/dL in infants to one month of age; 35–50 µg/dL in older children and adults
Phos
Gluc
Alb
H & H
Blood gases
$Na^+$, $K^+$, $Cl^-$
Ketonuria

## INTERVENTION

### OBJECTIVES

- Restrict total protein from the diet to minimize endogenous ammonia production and protein catabolism. Limit one or more essential amino acids while providing adequate energy and nutrients (Trahms, 2008).
- Promote anabolism with normal growth and development for age; use energy from nonprotein sources in amounts to spare protein for other purposes.
- Normalize blood ammonia levels and reduce the effects of hyperammonemia to prevent neuronal damage. Elevated levels of ammonia can come from either muscle breakdown or diet; determine which process is the problem.
- Administer desired substrates of the urea cycles. If necessary, support dialysis if blood ammonia levels are three to four times above normal.

### FOOD AND NUTRITION

- Use a protein-controlled diet (often 1.0–1.5 g/kg daily) with use of special amino acid formulas developed specifically for urea cycle disorders. These formulas provide approximately 50% of the daily dietary protein allowance; some patients may require individual BCAA supplementation.
- Pharmaceutical grade (not over-the-counter) L-citrulline (for OTC and CPS deficiency) or L-arginine free base (ASA and citrullinemia) is also required. These are not to be used in arginase deficiency.
- Add extra energy sources if needed to support growth and development. Weight gain is the best measure of success in infants and children. Metabolic nutritionists routinely prescribe calorie modules such as Prophree, Polycose and ModuCal. If dehydration occurs, intravenous fluids and glucose may be needed.
- Multiple vitamins and calcium supplements are recommended.

**SAMPLE NUTRITION CARE PROCESS STEPS**

**Altered Nutrition-Related Lab Values**

***Assessment Data:*** Weight and growth charts showing FTT, anorexia, elevated serum ammonia.

***Nutrition Diagnoses (PES):*** Altered nutrition-related labs related to hyperammonemia in urea cycle disorder as evidenced by anorexia, lethargy and sleepiness, FTT with weight/height at 3 percentile.

***Intervention:*** Educate parents about low protein formula, enhancing energy intake through high calorie foods and supplements containing calcium.

***Monitoring and Evaluation:*** Weight records, growth, improved appetite and intake, reduced serum ammonia levels, greater alertness.

### Common Drugs Used and Potential Side Effects

- Protein restriction is used in conjunction with medications to remove ammonia from the blood. Medications are given by way of tube feedings, either via gastrostomy tube or NG tube. To provide alternative route for ammonia, what is given depends on where the defect in the urea cycle has occurred.
- Arginine is often supplemented (400–700 mg/d), except for arginine deficiency (Trahms, 2008). For argininosuccinate synthetase and argininosuccinate lyase deficiencies, 0.4–0.7 g arginine/kg/d is given; 0.17 g/kg/d of citrulline is given for carbamyl phosphate synthetase deficiency.
- Sodium phenylbutyrate (Buphenyl) is used to normalize serum ammonia by diverting nitrogen to alternative paths for excretion (Scaglia et al, 2004). It is administered three to four times daily to keep ammonia under control.

### Herbs, Botanicals, and Supplements

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

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Provide sources of all essential amino acids in the diet, determined appropriately for age and body size. There are tables available for these purposes (Trahms, 2008).
- Adjust intake of energy and nutrients according to the patient's age.
- Comprehensive newborn screening is recommended for families who have had the birth of one or more children with these disorders.

#### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing formula or meals. Use clean utensils and containers for mixing formula.

- Before using tap water for formula preparation or to give as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard any beverage or food that has been left at room temperature for 2 hours or longer.
- Do not use honey in the diets of infants to decrease potential risk of botulism.

### *For More Information*

- Children Living with Inherited Metabolic Disorders (Climb)
  http://www.climb.org.uk/
- My Special Diet
  http://www.myspecialdiet.com/
- National Urea Cycle Disorders Foundation
  http://www.nucdf.org
- Organic Acidemia Association
  http://www.oaanews.org/
- UCD Kids Network
  http://www.nucdf.org/

## UREA CYCLE DISORDERS—CITED REFERENCES

Cohn RM, Roth KS. Hyperammonemia, bane of the brain. *Clin Pediatr (Phila).* 43:683, 2004.

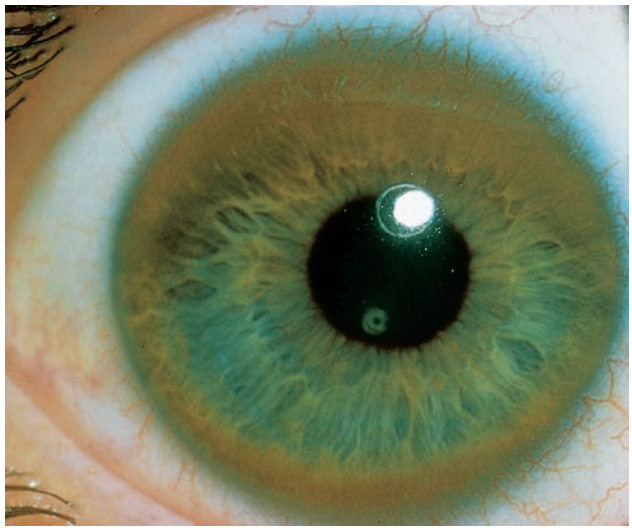
Scaglia F, et al. Effect of alternative pathway therapy on branched chain amino acid metabolism in urea cycle disorder patients. *Mol Genet Metab.* 81:S79, 2004.

Trahms C. Metabolic disorders. In: Mahan K, Escott-Stump S, eds. *Krause's food, nutrition, and diet therapy.* 12th ed. Philadelphia: WB Saunders, 2008.

Wilcken B. Problems in the management of urea cycle disorders. *Mol Genet Metab.* 81:S86, 2004.

# WILSON'S DISEASE (HEPATOLENTICULAR DEGENERATION)

## NUTRITIONAL ACUITY RANKING: LEVEL 3



Adapted from: Gold DH, MD, and Weingeist TA, MD, PhD. *Color Atlas of the Eye in Systemic Disease.* Baltimore: Lippincott Williams & Wilkins, 2001.

## DEFINITIONS AND BACKGROUND

The major physiological role of copper is to serve as a cofactor to a number of key metabolic enzymes. Copper is a trace element essential for normal cell homeostasis, promoting iron absorption for hemoglobin synthesis and for formation of bone and myelin sheath. In hepatic tissues, 90% of the copper in the copper-albumin complex is converted to ceruloplasmin. Wilson's disease is a rare inborn disease related to copper storage (Merle et al, 2007). Tissue deposition occurs instead of formation of ceruloplasmin in Wilson's disease.

An autosomal recessive disorder, Wilson's disease results in hepatolenticular degeneration, cirrhosis, neurologic damage, damage to the kidney, brain, and cornea. Onset occurs at birth, but symptoms may appear from 5 to 40 years of age. The disease may lead to neurodegeneration and behavior abnormalities. Three types of neurological symptoms can occur: dystonic syndrome (dystonic postures and choreoathetosis); ataxic syndrome (postural and intentional tremor and ataxia of the limbs); and parkinsonian syndrome (hypokinesia, rigidity, and resting tremor). Shortened attention span, slurring of speech, and depression are early symptoms. Individuals who present with neuropsychiatric problems are often identified later in life and have poorer outcomes than those with hepatic symptoms (Merle et al, 2007).

A low-copper diet is seldom essential but implemented when other therapies are unsuccessful (e.g., copper-chelating agents). Other dietary treatments under study include the use of increased histidine, specific polyunsaturated fatty acids, low soy, and other plans. If not diagnosed until onset of fulminant liver failure, the patient will die by age 30. Liver transplantation is the best treatment. New ideas regarding the clinical management of this disorder will emerge with elucidation of the cellular basis of the disease (Fink and Schilsky, 2007).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Wilson disease involves two abnormal copies of the *ATP7B* gene, one from each parent. The alteration is on chromosome 13.

| Clinical/History | | |
|---|---|---|
| Height | Kayser–Fleischer ring (gold or gray–brown opacity of peripheral cornea) | Enlarged liver |
| Weight | | Swallowing difficulty |
| BMI | | Drooling? |
| | | Easy bruising |
| | | Jaundice? |

| | Lab Work | |
|---|---|---|
| Fixed pseudo-smile | Ceruloplasmin (often low) | Bilirubin (>300 µmol/L) |
| Postural tremor of arms | Serum Cu (abnormal) | Alk phos, low (<600 IU/L) |
| Rigidity | Liver copper levels | Serum zinc |
| Abrupt personality change | Urinary Cu | Alb |
| Splenomegaly, esophageal varices? | ALT, AST [low transaminases (100–500 IU/L)] | H & H |
| Hepatitis or cirrhosis? | | BUN, Creat |
| | | Serum P |
| | | PT or INR |

## INTERVENTION

### OBJECTIVES

- Keep optimal balance of copper in patient.
- Decrease serum copper levels, generally with drug chelation. Enhance urinary excretion of excesses.
- Prevent or reverse damage to body tissues and liver.
- Watch caloric intake to prevent obesity.
- Monitor changes in gag reflex or dysphagia.
- Provide sufficient zinc to chelate excess copper under doctor's supervision.
- Prepare for transplantation where possible.
- Prevent or correct bone demineralization (Selimoglu et al, 2008).

### FOOD AND NUTRITION

- A normal diet provides 2–5 mg/d of copper. To lower copper in the diet (to 1–2 mg), use limited amounts of liver, kidney, shellfish, nuts, raisins and other dried fruits, dried legumes, brain, oysters, mushrooms, chocolate, poultry, and whole-grain cereals.
- A lacto–ovo–vegetarian diet may be useful to increase content of fiber and phytates; copper is less available in vegetarian diets.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Excessive Mineral Intake**

***Assessment Data:*** Neurological symptoms, altered LFTs, changes in the eye, low ceruloplasmin and elevated serum copper.

***Nutrition Diagnoses (PES):*** Excessive mineral (copper) intake related to Wilson's disease and disordered copper metabolism as evidenced by altered labs for copper and ceruloplasmin.

***Intervention:*** Educate parents about the role of copper in the body, and how zinc interacts; discuss the medication effects and the need for possible transplantation.

***Monitoring and Evaluation:*** Improved neurological symptoms and lab values posttransplantation.

- Control energy intake, food textures, and other nutrients if necessary.
- Increase fluid intake but avoid alcoholic beverages.
- Increase zinc from meat, poultry, fish, eggs, and milk if deemed appropriate for the patient.
- Assure adequate intake of calcium, vitamins D and K for bone health (Selimoglu et al, 2008).

### Common Drugs Used and Potential Side Effects

- Zinc acetate may be used to chelate copper with fewer side effects than D-penicillamine. Doses of 75–150 mg are often prescribed. Oral zinc is a suitable alternative to penicillamine as long-term maintenance therapy.
- D-penicillamine (Cuprimine or Depen), a copper-chelating agent, should be taken orally before meals. A vitamin $B_6$ supplement is needed with this drug; usually a dose of 25 mg.
- Laxatives or stool softeners may be needed. Encourage a diet high in fiber and fluid to wean off medication if possible.
- Corticosteroids and immunosuppressive therapy are used for autoimmune hepatitis. Side effects can be significant, including hyperglycemia, osteopenia, and nutrient depletion.
- Tetrathiomolybdate is being tested for use in Wilson's disease (Brewer et al, 2006).

### Herbs, Botanicals, and Supplements

- A neurological disorder has been noted after taking Chinese herbs; it is best to avoid them in Wilson's disease (Wang and Yang, 2003).

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Teach the patient about the copper and zinc content of foods. Explain that breast milk has higher copper levels than cow's milk to those individuals who need to know.
- Help the patient with feeding at mealtimes, if poor muscular control is demonstrated.
- Discuss effective coping mechanisms, community resources, and genetic counseling.
- Discuss the importance of maintaining prescribed drug therapy, which is essential for life.

#### *Patient Education—Food Safety*

- Hand washing with soap and hot water is recommended before preparing formula or meals. Use clean utensils and containers for mixing formula.
- Before using tap water for formula preparation or to give as a beverage, let cold tap water run for 2 minutes to remove any lead that may be in the pipes.
- Follow the 2-hour rule: discard any beverage or food that has been left at room temperature for 2 hours or longer.

- Do not use honey in the diets of infants to decrease potential risk of botulism.

### *For More Information*

- National Institute of Neurological Disorders and Stroke
  http://www.ninds.nih.gov/disorders/wilsons/wilsons.htm
- NIDDK
  http://digestive.niddk.nih.gov/ddiseases/pubs/wilson/
- Wilson's Disease Association
  http://www.wilsonsdisease.org/
- Wilson's Disease Center
  http://www.wilsonsdiseasecenter.org/

## WILSON'S DISEASE—CITED REFERENCES

Brewer GJ, et al. The use of tetrathiomolybdate in treating fibrotic, inflammatory, and autoimmune diseases, including the non-obese diabetic mouse model. *J Inorgan Biochem.* 100:927, 2006.

Fink S, Schilsky ML. Inherited metabolic disease of the liver. *Curr Opin Gastroenterol.* 23:237, 2007.

Merle U, et al. Clinical presentation, diagnosis and long-term outcome of Wilson's disease: a cohort study. *Gut.* 56:115, 2007.

Selimoglu MA, et al. Bone mineral density of children with Wilson disease: efficacy of penicillamine and zinc therapy. *J Clin Gastroenterol.* 42:194, 2008.

Wang XP, Yang RM. Movement disorders possibly induced by traditional Chinese herbs. *Eur Neurol.* 50:153, 2003.

SECTION 4

# Neuro-psychiatric Conditions

## CHIEF ASSESSMENT FACTORS

- Blunting of Emotions, Apathy, Egocentricity
- Bowel or Bladder Dysfunction
- Confusion, Memory Loss; Disorientation Regarding Place and Time
- Depression, Anxiety
- Disturbed Taste, Smell, Changes in Vision
- Dizziness, Vertigo, Drowsiness
- Dysphagia; Coughing or Choking While Eating/Swallowing
- Easy Aspiration of Food into Lungs
- Extremities: Coldness, Stiffness, Limited Movement, Discoloration, Pain
- Hallucinations, Tremors; Tics, Spasms, Ataxia
- Headaches, Pain
- Impulse Control Disorder
- Loss of Consciousness, Seizures
- Marked Disturbance in Eating Behaviors, Pica
- Mood Swings, Behavioral Changes, Psychotic Delusions
- Nervousness, Irritability
- Numbness, Paralysis, Sensory Pain
- Poor or Weaker Judgment; Difficulty Performing Familiar Tasks
- Problems with Abstract Thinking, Personality Changes
- Status of Food in Oral Cavity
- Stress (may speed up aging process because of protein kinase C)
- Weakness

## OVERVIEW OF NEUROLOGICAL AND PSYCHIATRIC DISORDERS

The central nervous system (CNS) consists of the brain and spinal cord. The brain has three main sections: the cerebrum, the cerebellum, and the brainstem. The normal adult brain weighs 3 lb; it grows steadily until 20 years of age and then loses weight for the rest of life. Gray matter consists of CNS tissue rich in neuronal cell bodies, their dendrites, axons, and glial cells; it includes the cerebral cortex, the central spinal cord, the cerebellar cortex, and the hippocampal cortex. White matter refers to large axon tracts in the brain and spinal cord involved with the cerebral hemispheres, the cerebellum, and the hippocampus. Figure 4-1 shows the brain and the spinal cord.

This chapter provides an overview of neurological and psychiatric disorders that have nutritional implications. A few disorders are found in other relevant sections; autism in Section 3, dysphagia in Section 7, anesthesia in Section 14. The primary neurological disorders are separated from psychiatric disorders. The newest edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) is due in 2012; the American Psychiatric Association (APA) and NIMH worked together to expand the scientific basis for psychiatric diagnosis and classification.

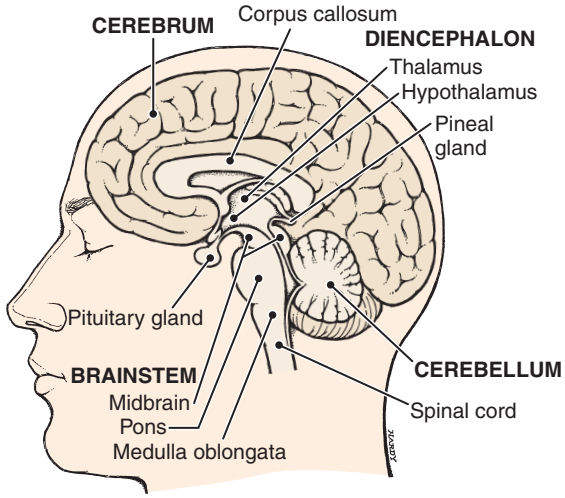



Figure 4-1

The brain and the gut work synergistically with each other and with other organs. Table 4-1 lists functions of the brain. Table 4-2 lists the cranial nerves and highlights those that affect chewing and swallowing. Nutrition influences the genetic onset and consequences of many chronic diseases.

**TABLE 4-1 Brain Parts and Their Functions**

| Parts | Relevant Nerves | Functions |
|---|---|---|
| **FOREBRAIN** | | |
| Cerebrum–temporal lobe | 8th cranial nerve | Controls hearing, expressive language, music and rhythm. Contains the hippocampus. Diseases that affect this area include Alzheimer's disease, depression, and mania |
| Cerebrum–frontal lobe | | Controls personality, mood, behavior, reasoning, emotional control, and cognition. Diseases affecting this area include Alzheimer's disease, depression, mania, and Huntington's disease |
| Cerebrum–parietal lobe | | Comprehension of written language and oral speech; sensory stimulation such as pain, touch, smell, hearing, and heat; body position. Alzheimer's disease affects this area; epilepsy and stroke may also impact this area |
| Cerebrum–occipital lobe | 2nd cranial nerve | Vision |
| Thalamus | | Relays sensory information to the cerebral cortex |
| Hypothalamus | | Lies beneath the thalamus. Secretes corticotropin-releasing hormone, affects metabolism by its influence on pituitary gland. Secretes vasopressin, which regulates sleep and wake cycles |
| Limbic system | System of nerve pathways | **Amygdala** affects depression<br>**Hippocampus** may affect mania, depression, and Alzheimer's. Located inside the temporal lobe (humans have two hippocampi, one in each side of the brain). Part of the limbic system. Plays a part in memory, learning, and navigation |
| **MIDBRAIN** | 3rd cranial nerve | Site between hindbrain and forebrain. Controls oculomotor nerve; eye movement. Affected in Parkinson's disease and some strokes |
| **HINDBRAIN** | | |
| **CEREBELLUM** | 3rd–5th cranial nerves | Found at bottom rear of the head. Posture and balance; voluntary movements such as sitting, standing, and walking. Directing attention and measuring time; other motor and cognitive functions. Stroke often affects this area |
| Pons | 4th–7th cranial nerves | Connects brainstem with cerebellum. Receives information from visual areas to control eye and body movements. Controls patterns of sleep and arousal; coordination of muscular movements; helps maintain equilibrium. Affects sleep disorders |
| Medulla oblongata | 8th–12th cranial nerves | Hearing, balance, some taste, some swallowing. Movement of the tongue. Involuntary functions such as heartbeat, circulation, muscle tone, and breathing. Stroke often affects this area |
| **SPINAL CORD** | | Sends and receives messages to and from brain and body parts |

Developed from: Brainexplorer, http://www.brainexplorer.org, accessed May 13, 2009.

**TABLE 4-2 Cranial Nerves and Those Specifically Affecting Mastication and Swallowing**

| | Nerve | Function | Affected Part of Body |
|---|---|---|---|
| I | Olfactory | Smell | Olfactory bulbs |
| II | Optic | Vision | Retina |
| III | Oculomotor | Eyeball movement<br>Lens accommodation | Four eyeball muscles and one eyelid muscle |
| | Oculomotor | Pupil constriction | |
| IV | Trochlear | Eyeball movement | Superior oblique muscles |
| V | Trigeminal[a] | Sensations<br>General sensory from tongue<br>Proprioception | Face, scalp, teeth, lips, eyeballs, nose and throat lining<br>Anterior two thirds of tongue<br>Jaw muscles for mastication |
| | Trigeminal | Chewing | Muscles of mastication |
| VI | Abducens | Eyeball movement | Lateral rectus muscle |
| VII | Facial[a] | Taste<br>Proprioception | Anterior two thirds of tongue<br>Face and scalp |
| | Facial | Facial expressions | Muscles of the face |
| | Facial | Salivation and lacrimation | Salivary and lacrimal glands via submandibular and pterygopalatine ganglia |
| VIII | Vestibulocochlear | Balance<br>Hearing | Vestibular apparatus of internal ear<br>Cochlear of internal ear |
| IX | Glossopharyngeal[a] | Taste<br>Proprioception for swallowing<br>Blood pressure receptors | Posterior two thirds of tongue<br>Throat muscles<br>Carotid sinuses |
| | Glossopharyngeal | Swallowing and gag reflex<br>Tear production | Throat muscles<br>Lacrimal glands |
| | Glossopharyngeal | Saliva production | Parotid glands |
| X | Vagus | Chemoreceptors<br>Pain receptors<br>Sensations<br>Taste | Blood oxygen concentration, aortic bodies<br>Respiratory and digestive tracts<br>External ear, larynx, and pharynx<br>Tongue |
| | Vagus | Heart rate and stroke volume<br>Peristalsis<br>Air flow<br>Speech and swallowing | Pacemaker and ventricular muscles<br>Smooth muscles of digestive tract<br>Smooth muscles in bronchial tubes<br>Muscles of larynx and pharynx |
| XI | Spinal Accessory | Head rotation | Trapezius and sternocleidomastoid muscles |
| XII | Hypoglossal[a] | Speech and swallowing | Tongue and throat muscles |

Adapted from: http://www.teaching-biomed.man.ac.uk/resources/wwwcal/cranial_nerves/page2.asp, accessed January 16, 2005.
[a]Cranial nerves affecting chewing and swallowing.

One individual may have up to 500,000 single nucleotide polymorphisms (Ferguson, 2007). A steady stream of neurotransmitters is needed for good mental and neurological health, yet they are subject to dietary manipulation. Increases or decreases in dietary precursors of serotonin, dopamine, norepinephrine, and acetylcholine affect nerve functioning. Brain levels of tryptophan, tyrosine, or choline control the rates at which neurons synthesize serotonin, dopamine, or acetylcholine, respectively (Wurtman, 2008). "Brain foods" can be suggested to prevent or treat many stress-related mental disorders (Takeda, 2004).

Heritable abnormalities include major depressive disorder (MDD), attention deficit hyperactivity disorder (ADHD), bulimia nervosa (BN), dysthymic disorder, fibromyalgia, generalized anxiety disorder, irritable bowel syndrome, migraine, obsessive-compulsive disorder, panic disorder, posttraumatic stress disorder, premenstrual dysphoric disorder, and social phobia (Hudson et al, 2003). The biological link between psychiatric and metabolic disorders is now clear (Bazar et al, 2006). Insulin resistance, diabetes, hypertension, metabolic syndrome, obesity, attention-deficit disorders, depression, psychosis, sleep apnea, inflammation, autism, and schizophrenia (SCZ) operate through common pathways; treatments used for one may prove beneficial for others (Bazar et al, 2006). Melatonin (MT), for example, is a powerful antioxidant that protects mitochondrial DNA; it easily crosses blood–brain barrier and has a role in ADHD, Alzheimer's disease (AD), autism, Parkinson's disease, seasonal affective disorder (SAD), and bipolar disorders (BDs).

Lipids are also essential for brain and neuron functioning. Lipid peroxidation is the outcome of free radical-mediated injury to the brain, where it directly damages membranes and generates oxidized products. Brain lipid peroxidation is a therapeutic target early in Alzheimer's and Huntington's diseases (Montine et al, 2004; Wu and Meydani, 2004). Brain P-450 enzymes catalyze the formation

of neurosteroids and eicosanoids and they metabolize substrates such as vitamins A and D, cholesterol, and bile acids (Liu et al, 2004).

Nutrient intake has long-range effects. Deficiencies of vitamins $B_{12}$, folic acid, $B_6$, C, or E, iron, or zinc mimic the effects of radiation on the body by damaging DNA through strand breaks and oxidative lesions; deficient iron or biotin causes mitochondrial decay and oxidant leakage, leading to accelerated aging and neural decay (Ames, 2004).

Micronutrient scarcity during periods of human evolution has altered DNA and promoted some of our modern, late-onset diseases (Ames, 2006). Dietetic professionals should consider the complexities of neuropsychiatric conditions as well as the influence of diet on both reproductive and adult health.

A multidisciplinary approach is most effective. Psychotherapy addresses the will to change, responsibility for self, and search for meaning and identity. Psychiatrists focus on the medical and chemical management of prescribed drugs. Social workers assist with family and relationship issues. Dietitians focus on overall health status, medical issues, prescribed medicines, alternative therapies, and appropriate nutritional treatments. Assessment must include careful review of medical and treatment histories. Interventions must apply a positive approach, prevention of malnutrition, use of the team concept, restoration of feeding abilities, and improved nutritional quality of life. Table 4-3 lists important disorders; Table 4-4 lists neurotransmitters and their nutritional relevance; and Table 4-5 describes nutrients and substances important for brain health.

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**TABLE 4-3 Disorders of Mental Health (*DSM-IV*)**

| Mental Health Disorders | Explanation or Relevance to Nutrition |
|---|---|
| Acute stress disorder | Development of anxiety and dissociative and other symptoms within 1 month following exposure to an extremely traumatic event; other symptoms include re-experiencing the event and avoidance of trauma-related stimuli. |
| Adjustment disorder | Maladaptive reaction to identifiable stressful life events. |
| Amnestic disorder | Mental disorder characterized by acquired impairment in the ability to learn and recall new information, sometimes accompanied by inability to recall previously learned information, and not coupled to dementia or delirium. |
| Anxiety disorders | A group of mental disorders in which anxiety and avoidance behavior predominate, including panic disorders, agoraphobia, specific phobia, social phobia, obsessive-compulsive disorder, posttraumatic stress disorder, acute stress disorder, generalized anxiety disorder, and substance abuse anxiety disorder. |
| Attention-deficit disorder | Mental disorder characterized by inattention (such as distractibility, forgetfulness, not finishing tasks, and not appearing to listen), by hyperactivity and impulsivity (such as fidgeting and squirming, difficulty in remaining seated, excessive running or climbing, feelings of restlessness, difficulty awaiting one's turn, interrupting others, and excessive talking). See Section 3 for details. |
| Autistic disorder | Severe pervasive developmental disorder with onset usually before 3 years of age and a biological basis related to neurological or neurophysiological factors. Characterized by qualitative impairment in reciprocal social interaction (e.g., lack of awareness of the existence of feelings in others, failure to seek comfort at times of distress, lack of imitation), in verbal and nonverbal communication, and in capacity for symbolic play. Restricted and unusual repertoire of activities and interests. |
| Binge eating disorder | An eating disorder characterized by repeated episodes of binge eating, as in bulimia nervosa, but not followed by inappropriate compensatory behavior such as purging, fasting, or excessive exercise. |
| Bipolar disorders | Mood disorders characterized by a history of manic, mixed, or hypomanic episodes, usually with concurrent or previous history of one or more major depressive episodes. |
| Body dysmorphic disorder | A mental disorder in which a normal-appearing person is either preoccupied with some imagined defect in appearance or is overly concerned about some very slight physical anomaly. |
| Catatonic disorder | Catatonia due to the physiological effects of a general medical condition and neither better accounted for by another mental disorder nor occurring exclusively during delirium. |
| Childhood disintegrative disorder | Pervasive developmental disorder characterized by marked regression in a variety of skills, including language skills, social skills or adaptive behavior, play, bowel or bladder control, and motor skills, after at least 2, but less than 10, years of apparently normal development. |

*(continued)*

**TABLE 4-3 Disorders of Mental Health (*DSM-IV*) *(continued)***

| Mental Health Disorders | Explanation or Relevance to Nutrition |
|---|---|
| Circadian rhythm sleep disorder | Lack of synchrony between the schedule of sleeping and waking required by the external environment and that of a person's own circadian rhythm. |
| Conduct disorder | A type of disruptive behavior disorder of childhood and adolescence characterized by a persistent pattern of conduct in which rights of others or age-appropriate societal norms or rules are violated. Misconduct includes aggression toward people or animals, destruction of property, deceitfulness, theft, and serious violations of rules. |
| Conversion disorder | Mental disorder with loss or alteration of voluntary motor or sensory functioning suggesting physical illness (such as seizures, paralysis, dyskinesia, anesthesia, blindness, or aphonia) having no demonstrable physiological basis. |
| Delusional disorder | Mental disorder marked by well-organized, logically consistent delusions but lacking other psychotic symptoms. Most functioning is not markedly impaired, the criteria for schizophrenia have never been satisfied, and symptoms of a major mood disorder have been present only briefly if at all. |
| Depersonalization disorder | Dissociative disorder characterized by one or more severe episodes of depersonalization (feelings of unreality and strangeness in one's perception of the self or one's body image) not due to another mental disorder, such as schizophrenia. The perception of reality remains intact; patients are aware of their incapacitation. Episodes are usually accompanied by dizziness, anxiety, fears of going insane, and derealization. |
| Depressive disorders | Mood disorders in which depression is unaccompanied by manic or hypomanic episodes. |
| Disruptive behavior | Group of mental disorders of children and adolescents consisting of behavior that violates social norms, is disruptive, distressing others more than it does the person with the disorder. |
| Dissociative disorders | Mental disorders characterized by sudden, temporary alterations in identity, memory, or consciousness, segregating normally integrated memories or parts of the personality from the dominant identity of the individual. |
| Dissociative identity disorder | Multiple personality disorder; characterized by the existence in an individual of two or more distinct personalities, each having unique memories, characteristic behavior, and social relationships. |
| Dysthymic disorder | Mood disorder characterized by depressed feeling (sad, blue, low), by loss of interest or pleasure in one's usual activities, and by at least some of the following: altered appetite, disturbed sleep patterns, lack of energy, low self-esteem, poor concentration or decision-making skills, and feelings of hopelessness. Symptoms have persisted for more than 2 years but are not severe enough to meet the criteria for major depressive disorder. |
| Eating disorder | Any of several disorders (anorexia nervosa, bulimia nervosa, pica, and rumination disorder) in which abnormal feeding habits are associated with psychological factors. |
| Factitious disorder | Repeated, intentional simulation of physical or psychological signs and symptoms of illness for no apparent purpose other than obtaining treatment. |
| Generalized anxiety disorder | Excessive, uncontrollable anxiety and worry about two or more life circumstances, for 6 months or longer, accompanied by some combination of restlessness, fatigue, muscle tension, irritability, disturbed concentration or sleep, and somatic symptoms. |
| Impulse control disorders | Repeated failure to resist an impulse to perform some act harmful to oneself or to others. |
| Learning disorders | Academic functioning that is substantially below the level expected on the basis of the patient's age, intelligence, and education, interfering with academic achievement or other functioning. Included are reading disorder, mathematics disorder, and disorder of written expression. |
| Mental disorder | Any clinically significant behavioral or psychological syndrome characterized by the presence of distressing symptoms, impairment of functioning, or significantly increased risk of suffering pain, disability, loss of freedom or death. Mental disorders manifest a behavioral, psychological, or biological dysfunction in the individual. |
| Motor skills disorder | Inadequate development of motor coordination, severe enough to limit locomotion or restrict the ability to perform tasks, schoolwork, or other activities. |
| Obsessive-compulsive disorder | Anxiety disorder characterized by recurrent obsessions or compulsions, which are severe enough to interfere significantly with personal or social functioning. Performing compulsive rituals may release tension temporarily; resisting them causes increased tension. |
| Oppositional defiant disorder | Disruptive behavior characterized by a recurrent pattern of defiant, hostile, disobedient, and negativistic behavior directed toward those in authority, including such actions as defying the requests or rules of adults, deliberately annoying others, arguing, spitefulness, and vindictiveness that occur much more frequently than would be expected on the basis of age and developmental stage. |
| Pain disorder | A somatoform disorder characterized by a chief complaint of severe chronic pain that causes substantial distress or impairment in functioning; the pain is neither feigned nor intentionally produced, and psychological factors appear to play a major role in its onset, severity, exacerbation, and maintenance. |
| Panic disorder and agoraphobia | Recurrent panic attacks, episodes of intense apprehension, fear, or terror associated with somatic symptoms such as dyspnea, palpitations, dizziness, vertigo, faintness, shakiness; and psychological symptoms such as feelings of unreality, depersonalization, fears of dying, going crazy, or losing control. There is usually chronic nervousness and tension between attacks. |

*(continued)*

**TABLE 4-3 Disorders of Mental Health (*DSM-IV*) *(continued)***

| Mental Health Disorders | Explanation or Relevance to Nutrition |
|---|---|
| Personality disorders | Enduring, inflexible, and maladaptive personality traits that deviate markedly from cultural expectations, are self-perpetuating, pervade a broad range of situations, and either generate subjective distress or result in significant impairments in social, occupational, or other functioning. Onset is by adolescence or early adulthood. |
| Pervasive developmental disorders | Impaired development in multiple areas, including the acquisition of reciprocal social interaction, verbal and nonverbal communication skills, and imaginative activity and by stereotyped interests and behaviors; included are autism, Rett's syndrome, childhood disintegrative disorder, and Asperger's syndrome. |
| Premenstrual dysphoric disorder | Premenstrual syndrome with signs of depression, lethargy. |
| Reading disorder | Learning disorder in which the skill affected is reading ability, including accuracy, speed, and comprehension. |
| Rumination disorder | Eating disorder seen in infants under 1 year of age. After a period of normal eating habits, the child begins excessive regurgitation and rechewing of food, which is then ejected from the mouth or reswallowed. If untreated, death from malnutrition may occur. |
| Schizoaffective disorder | A major depressive episode, manic episode, or mixed episode occurs along with prominent psychotic symptoms characteristic of schizophrenia, the symptoms of the mood disorder being present for a substantial portion of the illness, but not for its entirety, and not being due to the effects of a psychoactive substance. |
| Seasonal affective disorder | A cyclically recurring mood disorder characterized by depression, extreme lethargy, increased need for sleep, hyperphagia, and carbohydrate craving; it intensifies in specific seasons, most commonly winter. It is hypothesized to be related to melatonin levels. "Mood disorder with seasonal pattern." |
| Separation anxiety disorder | Excessive, prolonged, developmentally inappropriate anxiety and apprehension in a child concerning removal from parents, home, or familiar surroundings. |
| Shared psychotic disorder | A delusional system that develops in one or more persons as a result of a close relationship with someone who already has a psychotic disorder with prominent delusions. |
| Sleep disorders | Chronic disorders involving sleep. Primary sleep disorders comprise dyssomnias and parasomnias; causes of secondary sleep disorders may include a general medical condition, mental disorder, or psychoactive substance. |
| Speech disorder | Defective ability to speak, either psychogenic or neurogenic. |
| Substance-related disorders | A variety of behavioral or psychological anomalies resulting from ingestion of or exposure to a drug of abuse, medication, or toxin. |

Adapted from: Merck Manual, http://www.mercksource.com/pp/us/cns/cns_home.jsp, accessed May 13, 2009.

**TABLE 4-4 Neurotransmitters and Nutritional Relevance**

| Type | Neurotransmitter | Postsynaptic Effect | Functions and Nutritional Relevance |
|---|---|---|---|
| Amino acids | Gamma aminobutyric acid (GABA) | Inhibitory | Glutamate is a precursor. Pyridoxal phosphate is a cofactor for both synthesis and break-down. |
| | Glycine | Inhibitory | Glycine inhibits neurotransmitter signals in the CNS. Available from dietary proteins, but most contain only small amounts (exception is collagen). Unique role as a type of antioxidant. |
| | Glutamate | Excitatory | The most important neurotransmitter for normal brain function; >50% of the neurons in the brain release glutamate. Glutamate can be used to synthesize glutamine by taking up ammonia; this reduces excessive ammonia levels in the brain and is important in diseases such as hepatic encephalopathy. The "sodium salt" of glutamic acid, monosodium glutamate (MSG), is responsible for one of the five basic tastes of the human sense of taste, umami; MSG is extensively used as a food additive. No specific dietary precursors. Primarily synthesized in the brain from alpha-keto glutarate and glucose. Glutamate is a precursor of GABA. |
| | Aspartate | Excitatory | Acidic analog of asparagine. No specific dietary precursors. Synthesized primarily from glutamate. |
| Biogenic mono-amines | Dopamine | Excitatory | A monoamine neurotransmitter, concentrated in the basal ganglia. It is widely distributed throughout the brain in the nigrostriatal, the mesocorticolimbic, and the tuberohypophyseal pathways. Decreased brain dopamine levels contribute to Parkinson's disease, while an increase in dopamine concentration has a role in psychosis. Synthesized from phenylalanine and tyrosine. |
| Biogenic amine | Acetylcholine | Excitatory | Main neurotransmitter in the parasympathetic nervous system that controls heart rate, digestion, secretion of saliva, and bladder function. Drugs that affect cholinergic activity produce changes in these body functions. Affected by choline from the diet (eggs, soybeans). Some antidepressants act by blocking cholinergic receptors; this anticholinergic activity is an important cause of dry mouth. Botulism suppresses release of acetylcholine, and nicotine increases receptors for acetylcholine. Alzheimer's disease seems to be related to a malfunction in this neurotransmitter. |

*(continued)*

**TABLE 4-4 Neurotransmitters and Nutritional Relevance *(continued)***

| Type | Neurotransmitter | Postsynaptic Effect | Functions and Nutritional Relevance |
|---|---|---|---|
| | Epinephrine | Excitatory | Affects fight or flight reactions; secreted in greater quantity during anger and fear, with resulting increase in heart rate and hydrolysis of glycogen to glucose. Used as a stimulant in cardiac arrest, as a vasoconstrictor in shock, as a bronchodilator in asthma, and to lower intraocular pressure in glaucoma. Secreted by the adrenal medulla. |
| | Noradrenaline | Excitatory | Synthesized from phenylalanine and tyrosine. A monoamine neurotransmitter that affects "fight or flight," attention and arousal, and blood pressure. |
| | Serotonin | Excitatory | Synthesized from tryptophan in the diet. Affects mood control, regulation of sleep, pain perception, body temperature, blood pressure, and hormonal activity. Also affects gastrointestinal and cardiovascular systems. |
| | Histamine | Excitatory | A potent agent believed to be involved in sleep–wake cycles and allergy. |

From: http://www.brainexplorer.org/neurological_control/Neurological_index.shtml, accessed May 13, 2009.

**TABLE 4-5 Nutrients for Brain Health**

**Diets that provide adequate amounts of complex carbohydrates, essential fats, amino acids, vitamins and minerals, and water support a balanced mood. Diet is one part of the jigsaw in the promotion of good mental health (Mental Health Foundation, 2009).**

| Nutrient or Factor | Role in the Brain | Comments |
|---|---|---|
| **PROTEINS** | | |
| Aromatic amino acids (tryptophan, tyrosine, and phenylalanine) | Precursors of serotonin, dopamine, and norepinephrine | Increase in brain tryptophan from eating a carbohydrate-rich/protein-poor meal causes parallel increases in the amounts of serotonin released into synapses. Tryptophan can induce sleep from high-carbohydrate meals; high-protein meals tend to increase alertness. |
| Corticotropin-releasing hormone | Disturbances occur in periods of stress in the hypothalamic–pituitary–adrenal axis | Eating is often suppressed during stress due to anorectic effects of corticotropin-releasing hormone and increased during recovery from stress due to appetite-stimulating cortisol. Night eating syndrome is related to cortisol levels. |
| Cytokines | Influence sleep and eating behaviors; involved in many infectious, inflammatory, neoplastic, metabolic, and degenerative illnesses | Implicated in depressive and anxiety disorders; schizophrenic disorders (chronic and acute); autistic disorder; eating disorders; and obsessive-compulsive disorder. |
| Dietary antioxidants | Improve cognitive functioning | Fruits, vegetables, coffee, and tea. Strong inverse relationship between coffee intake and risk of suicide (Takeda, 2004). Quercetin (in red apples with skins, onions, blueberries, cranberries, strawberries) seems to protect against brain-cell damage (Silva et al, 2004). |
| **LIPIDS** | | |
| Endocannabinoids | A class of lipids including amides, esters, and ethers of long-chain polyunsaturated fatty acids (Battista et al, 2004). They are activated by a CB2 receptor agonist, arachidonoylglycerol, and by elevated endogenous levels of endocannabinoids (Van Sickle et al, 2005) | Anandamide (N-arachidonoylethanolamine; AEA) and 2-arachidonoylglycerol are the main endogenous agonists of cannabinoid receptors, able to mimic several pharmacological effects of delta(9)-tetrahydrocannabinol, the active principal component of *Cannabis sativa* preparations such as hashish and marijuana (Battista et al, 2004). Nonpsychotropic therapeutic interventions using enhanced endocannabinoid levels may be used in localized brain areas (Van Sickle et al, 2005). |
| Essential fatty acids (EFA) | Fluidity of neuronal membrane, synthesis and functions of brain neurotransmitters, immune system integrity (Yehuda et al, 2005) | The blood–brain barrier determines the bioavailability. The myelination process determines the efficiency of brain and retinal functions of EFA. Since they must be supplied from the diet, a decreased bioavailability is induces major disturbances (Yehuda et al, 2005). |
| Omega-3 fatty acids *DHA, EPA | Control inflammatory and autoimmune processes; part of the brain lipid membranes. DHA depletion leads to losses in neuronal function (Lim et al, 2005) | Helpful in depression, bipolar disorder, multiple sclerosis, and other neurological conditions. DHA and arachidonic acid (AA) may not distribute evenly in the brain. There are age-induced regional changes in fatty acid composition of brain phospholipids. DHA, uridine (as uridine monophosphate), and choline are all found in mother's milk, and included in most infant formulas; these substances are part of a regulatory mechanism through which plasma composition influences brain development (Wurtman, 2008). |
| Omega-6 polyunsaturated fatty acids<br>ALA, GLA | Part of the brain lipid membranes | Useful in anorexia nervosa and several neurological conditions. May reduce risk for Parkinson's disease (de Lau et al, 2005). |

*(continued)*

**TABLE 4-5 Nutrients for Brain Health *(continued)***

| Nutrient or Factor | Role in the Brain | Comments |
|---|---|---|
| **MINERALS** | | |
| Iron | Normal amounts are required for normal functioning. 7% of the general population, and 19% of women between ages of 12–50 may be deficient (UC Davis, 2009) | Iron sufficiency prevents anemia. Deficiency causes DNA breaks (UC Davis, 2009). |
| Selenium | Parts of the United States and China have areas where soil is deficient in selenium | Antioxidant properties protect the brain and nerves from damage. Brazil nuts are a rich source. |
| Zinc | 18% of the US population is deficient (UC Davis, 2009) | Zinc functions as part of the insulin molecule and hundreds of enzymes. Deficiency causes DNA damage with chromosome breaks; this leads to brain and immune dysfunction (UC Davis, 2009). |
| **VITAMINS** | | |
| Niacin | 2% of the general population may be deficient (UC Davis, 2009) | Deficiency disables DNA repair (poly ADP ribose); this leads to neurological damage and memory loss (UC Davis, 2009). |
| Vitamins $B_6$, $B_{12}$, and folic acid | Lower elevated amounts of homocysteine. 10% of the population may be deficient in these $B_6$; 4% may be deficient in $B_{12}$ (UC Davis, 2009) | These play a role in many neurological conditions. Methylated forms may be needed in individuals who have the MTHFR genotype. $B_{12}$ deficiency leads to neurological damage (UC Davis, 2009). |
| Vitamins C and E | Vitamins C and E function as antioxidants | Antioxidant properties are protective. |
| Vitamin D | Vitamin D has been found to delay onset of multiple sclerosis and effects of depression (especially seasonal affective disorder). It is being studied in schizophrenia | Vitamin D has nuclear hormone receptors that regulate gene expression and nervous system development. |
| **OTHER** | | |
| [a]Uridine | A pyrimidine nucleoside that is formed when uracil is attached to a ribose ring | Component of RNA. Its nucleotides participate in the biosynthesis of polysaccharide compounds. Foods containing uridine may help to alleviate depression (Carlezon et al, 2005). |
| [a]Choline | A nutrient that improves the environment of the brain cells (Zeisel, 2004) | Choline is a nutrient found in egg yolks, milk, nuts, fish, liver and other meats, and human breast milk. It is the essential building block acetylcholine, and it plays a vital role in the formation of phospholipids in cell membranes. Pregnant women should include a good source in their daily diets. |

[a]NOTE. A preparation containing uridine, docosahexaenoic acid (DHA,) and choline is being tested for improved cognition, and enhanced neurotransmitter release (Wurtman et al, 2009).

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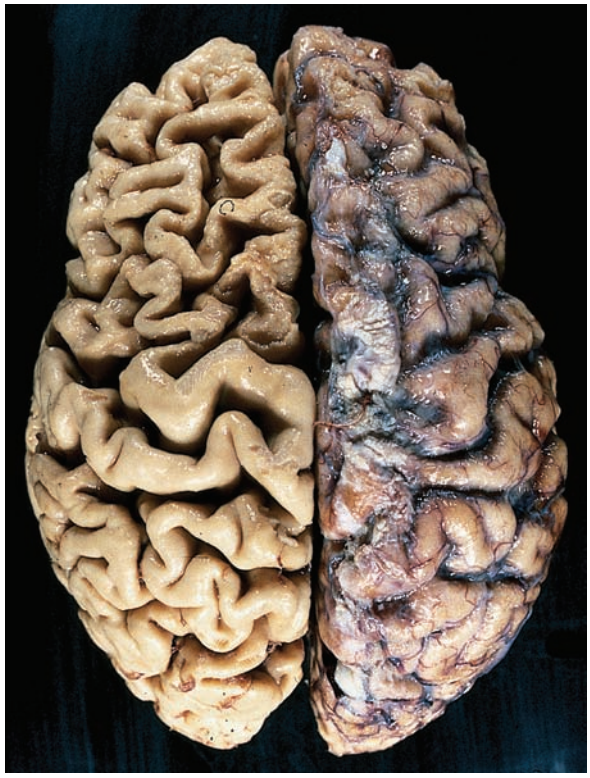
## *For More Information*

- American Academy of Neurology
  http://www.aan.com
- American Association of Neuroscience Nurses
  http://www.aann.org
- American Neurological Association
  http://www.aneuroa.org
- American Psychiatric Association—DSM-V
  www.dsm5.org
- American Society of Neurorehabilitation
  http://www.asnr.com
- Brain Research Foundation
  http://brainresearchfdn.org/
- Society for Neuroscience
  http://www.sfn.org

## Neurological Disorders

# ALZHEIMER'S DISEASE AND DEMENTIAS

## NUTRITIONAL ACUITY RANKING: LEVEL 3



Adapted from: Raphael Rubin, David S. Strayer, *Rubin's Pathology: Clinicopathologic Foundations of Medicine,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

## DEFINITIONS AND BACKGROUND

**Dementias** include multiple cognitive defects with memory loss; they often involve aphasia, apraxia, agnosia, and disturbed daily functioning. Risk factors include diabetes, cardiovascular disease, stroke, hypertension, head injury, aging, depression, and family history. Of over 50 dementias, **AD** is the most common, with progressive deterioration of intellect, memory, personality, and self-care. Other conditions should be ruled out by medical evaluation. Early stages of AD manifest with short-term memory loss; problems finding the appropriate word; asking the same questions over and over; difficulty making decisions and planning ahead; suspiciousness; changes in senses of smell and taste; denial; depression; loss of initiative; personality changes; and problems with abstract thinking.

AD is characterized by deposition of extracellular neuritic, beta-amyloid peptide-containing plaques in cerebral cortical regions and presence of intracellular neurofibrillary tangles in cerebral pyramidal neurons (Schliebs, 2005). Acetylcholine-containing neurons are especially affected. Acetylcholine normally triggers breakdown of the beta-amyloid precursor protein (APP) in brain cells. Impaired cerebral energy metabolism and pyruvate dehydrogenase activity are also found (Martin et al, 2005).

Insulin and associated signaling molecules begin to disappear from the brain during early AD, suggesting a form of "type 3 diabetes." Insulin deficiency and insulin resistance mediate AD-type neurodegeneration; indeed, experimental brain diabetes is treatable with drugs currently used to treat T2DM (de la Monte and Wands, 2008). Altered glycogen synthase kinase-3 (GSK-3) function, decreased serum insulin-like growth factor I (IGF-I) levels, and carotid atherosclerosis are independent risk factors for AD (Lester-Coll et al, 2006; Wantanabe et al, 2005).

Apolipoprotein E (ApoE) is an important determinant of lipoprotein metabolism and risk for oxidative damage (Dietrich et al, 2005). Because ApoE also influence cognitive function and decline, prevention of cardiovascular disease may slow the onset of AD (Kang et al, 2005). Because age-related proinflammattory cytokines are also involved, there is a reduced risk of AD in users of nonsteroidal anti-inflammatory drugs (Staehelin, 2008).

Mood instability and increased distractibility, irritability, agitation, and irregular sleep can be present. Behavioral changes, such as aggressive behavior, psychosis, and overactivity, occur frequently and determine the need for institutionalization or use of mood stabilizers. A lifetime of depression may actually precede AD and should be corrected (Ownby et al, 2006; Rapp et al, 2006).

Declining body mass over time is strongly linked to the risk of developing AD (Buchman et al, 2005). Metabolic acidosis promotes muscle wasting; diets that are rich in net acid-producing protein and cereal grains relative to their content of net alkali-producing fruit and vegetables may therefore contribute to a reduction in lean tissue mass in older adults (Dawson-Hughes et al, 2008).

Circadian patterns of food intake change and disturbed eating patterns occur, with altered macronutrient selection and preference for carbohydrates (Greenwood et al, 2005).

Numerous observational studies demonstrate a positive correlation between a high intake of antioxidants and better cognitive function in the elderly (Staehelin, 2008). Use of nutrients that increase the levels of brain catecholamines and protect against oxidative damage is critical: vitamins C and E, zinc, iron, copper, and selenium should be provided (Squitti et al, 2005). Antioxidants in foods such as blueberries, cranberries, strawberries, kale, and spinach may improve cognitive function; and coffee drinkers tend to have lower levels of AD later in life (Eskelinen et al, 2009). There is substantial epidemiological evidence from a number of recent studies that demonstrate a protective role of omega-3 fatty acids, such as docosahexaenoic acid (DHA), in AD and cognitive decline (Morris, 2009).

Finally, vitamins $B_6$ and $B_{12}$ and folate should be used to lower homocysteine (tHcy) levels. Comprehensive treatment of AD requires thorough caregiver support and thoughtful use of medications for cognition enhancement, neuroprotection, and treatment of agitation. While the prognosis for

AD is improving, death most often occurs from renal, pulmonary, or cardiac complications between 2 and 20 years after onset of symptoms. Pneumonia is a common cause of morbidity and death, related to dysphagia, aspiration, altered mobility, decreased nutritional status, and lowered immune response.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** ApoE epsilon4 allele is a primary gene that has been confirmed in AD. Hypomethylation of the amyloid A4 precursor gene and hyperhomocysteinemia may contribute to the pathophysiology of AD (Abdolmaleky et al, 2004; Aisen et al, 2008). Cerebrospinal-fluid biomarkers beta-amyloid 42 (Aß42), T-tau, and P-tau may identify early-stage AD (Mattsson et al, 2009).

**Clinical/History**

- Height, weight
- Subtle weight loss
- Current BMI,% change
- Dietary/intake history
- Intake and output (I & O)
- Anorexia and poor intake
- Nausea, vomiting
- Diarrhea
- Bowel incontinence
- Electroencephalogram (EEG)
- Loss of sense of smell
- Hx of Down syndrome or depression?
- Computed tomography (CT) scan
- Brain magnetic resonance imaging (MRI)
- Medial temporal lobe thickness (often thinned)
- Mini-Mental State Examination
- Severe Impairment Battery and Global Deterioration Scale
- Mattis Dementia Rating Scale
- Behavioral disturbances: Neuropsychiatric Inventory
- Behavioral function: London Psychogeriatric Rating Scale

**Lab Work**

- Cerebrospinal fluid biomarkers [Aβ42, T-tau, P-tau]
- Choline acetyltransferase activity (ChAT)
- C-reactive protein (CRP)
- Cholesterol (may be high)
- Glucose (gluc)
- Serum tHcy (elevated?)
- Serum folate; methyltetrahydrofolate reductase (MTHFR) levels
- Serum vitamin $B_{12}$
- Serum zinc
- Serum copper
- Alanine aminotransferase (ALT)
- Aspartate aminotransferase (AST)
- Dopamine (DA)
- Cerebral spinal fluid (CSF) pyruvate and lactate levels
- $Na^+$, $K^+$
- Blood urea nitrogen (BUN)
- Creatinine (creat)
- H & H
- Serum Fe
- Albumin or transthyretin

## INTERVENTION

### OBJECTIVES

- To effectively enhance intake, interventions must work with changing needs and intake patterns (Young et al, 2005). Prevent weight loss from altered activity levels, poor eating habits, depression, impaired memory, and self-feeding difficulty.
- Maintain activity to preserve function. Walking 90 minutes per week helps to maintain lean body mass and can possibly help alleviate anorexia.
- Avoid constipation or impaction; support bowel or bladder continence through proper scheduling.
- Encourage self-feeding at mealtimes as long as possible. Begin using finger foods and items easy to consume without utensils.
- Prevent or correct dehydration, pressure ulcers, and other signs of nutritional decline. Preserve muscle mass.
- Monitor dysphagia, pocketing of food, or aspiration. Use gastrostomy feeding, especially in early stages. While the

### SAMPLE NUTRITION CARE PROCESS STEPS

**Self-Feeding Difficulty**

***Assessment Data:*** Food records indicating poor intake; weight loss; forgetting how to feed self. Requires total feeding. Anorexia and complaints of taste changes, lost sense of smell. Lab results showing low albumin and transthyretin. Recent dehydration and urinary tract infection (UTI).

***Nutrition Diagnoses (PES):*** Self-feeding difficulty related to inability to use utensils (fork, spoon) at advanced stage of Alzheimer's as evidenced by weight loss, I & O with recent dehydration, dietary intake records showing intake of about 25–50% of meals served.

***Interventions:***

Food and Nutrient Delivery:
ND 1.3 Specific foods/beverage—finger foods and nutrient-dense diet.
ND 3.1.1.Commercial beverage supplement 1 can twice daily.
ND 3.2.1 Supplement with multivitamin–mineral once daily.
ND 4.5 Feeding assistance—fed by staff.

**Education:** Educate caregivers about introducing finger foods items, reducing distractions at mealtime, scheduling of meals to give structure, use of frequent and portable snacks throughout the day to improve intake of nutrients and energy. Reinforce safe feeding strategies with caregivers to avoid aspiration.

**Counseling:** C-2.2 Goal setting: improve oral intake, prevent aspiration, regain lost weight. Avoid constipation, dehydration, and impaction.

**Coordination of Nutrition Care:** RC-1.1 Team meeting with nursing, social services, speech therapy, recreation therapy.

***Monitoring and Evaluation:*** Improved weight, visceral protein levels; less agitation.

terminal stage of AD is generally indicated by the inability to swallow, the benefit of enteral feeding (EN) is limited at that point.
- Use creative feeding strategies. Offer frequent snacks, day and night if desired.
- Protect patient from injuries and provide emotional support for patient and family.
- To assist with eating-related behavioral problems, there are many tips. For example, for attention deficit, verbally direct the patient through the eating process; make food and fluids visible. Give one food at a time; offer small bites. Serve soft foods to reduce the need for chewing.
- Keep simple routines and a consistent environment. Minimize distractions at mealtime. Provide a quiet environment. If group dining is a concern, provide meals in room or with a single partner.
- Use finger foods and cups with cover/spout if the patient paces.

## FOOD AND NUTRITION

- Ensure a healthy diet, including protein and increased calories for age, sex, and activity, especially for "wanderers" and those who pace. Persons with AD may need 35 kcal/kg of body weight or more.
- Adequate **vitamin E** (such as in nuts, creamy salad dressings). Include nutrient-dense foods that are high in antioxidants. Coffee, cocoa, and red wine contain flavonols that increase blood flow to the brain; one serving of wine daily can be included.
- Provide more oily fish (salmon, halibut, trout, and tuna) for **omega-3 fatty acids**. Fish oil capsules have not been shown to be quite as effective.
- **Omega 6 fatty acids** may also be beneficial, such as from vegetable oils.
- Extra **folic acid** may lessen decline of cognitive function; offer leafy greens, orange juice, broccoli. To lower tHcy levels, folic acid, vitamins B and $B_{12}$ are important (Zhou and Practico, 2009). Avoid excesses of vitamin $B_{12}$ from red meat and high-fat dairy products.
- Use color-rich fruits and vegetables (blueberries, cranberries, strawberries, spinach, kale, broccoli, oranges, and other citrus fruits). Higher intake of foods rich in potassium, such as fruit and vegetables, favors preservation of muscle mass (Dawson-Hughes et al, 2008).
- Foods high in copper include liver, kidney, oysters, nuts, dried beans and legumes, cocoa, eggs, prunes, and potatoes.
- Offer meals at regular and consistent times each day. Allow sufficient time for eating. Offer one course at a time (first salad, then entree, then fruit dessert) to prevent confusion. Avoid distractions; use calming background music. Cueing to eat is also useful.
- Use dishes without a pattern; white is a good choice.
- Use a simple place setting and a single eating utensil. Use a bowl for easier scooping and special spoons or adaptive equipment as needed. Serve soup in mugs.
- Finger foods, such as sandwiches cut into four, cheese cubes, pancakes or waffles cut in smaller pieces, hard cooked egg halves, chicken strips, julienne vegetables, and brownies (vs. pie), are easier to eat and help to maintain weight.
- Provide a high-carbohydrate meal for dinner to increase food intake during later stages of the disease; this reflects the preference for high-carbohydrate foods (Young et al, 2005). Plan menus accordingly; offer nutrient-dense desserts, such as fruit tarts, puddings topped with fruit, and custards.
- Tube feed or use texture-altered foods with thickened liquids as needed to compensate for dysphagia.
- Choline may be beneficial; use soybeans or eggs (Michel et al, 2006).
- Adequate fluid intake is essential. Offer regular drinks of water, juice, milk, and other fluids to avoid dehydration.
- Cut back on saturated fats, which increase brain beta-amyloid levels. Omit high-fat dairy products, fast food items, fried foods, and processed foods.

### Common Drugs Used and Potential Side Effects

- See Table 4-6.

### Herbs, Botanicals, and Supplements

- Allopregnanolone (APalpha, a metabolite of progesterone) is reduced in the serum, plasma, and brain of aged individuals and Alzheimer's patients; researchers are evaluating its possible use (Wang et al, 2008).
- Chinese medicines show promise in combinations; Huperzine A (Chinese club moss,) gingko biloba, and ginseng are under Study (Fu and Li, 2009).
- Coenzyme Q10 and choline supplements are under study.
- Curry, cumin, and turmeric may block beta-amyloid plaque formation in the brain and can be encouraged as seasonings.
- Folic acid, vitamins $B_6$, and $B_{12}$ supplements do not slow cognitive decline in dementia but can lower tHcy levels (Aisen et al, 2008; Malouf et al, 2003). Cerefolin® contains N-acetylcysteine (NAC) and L-methylfolate. Where there are MTHFR polymorphisms, the active form of folate (methyltetrahydrofolate) may be needed (Mischoulon and Raab, 2007).
- Genetic risk factors for cognitive decline may remain latent pending age-related decline in nutrition, suggesting the importance of early intervention with key dietary supplements (including alpha-lipoic acid [ALA], DHA) to delay the progression of age-related cognitive decline (Suchy et al., 2009).
- Gingko biloba interacts with anticoagulants and antiplatelets such as aspirin, warfarin, and dipyridamole. Clinical trials have not shown effectiveness when used alone (Birks et al, 2009).
- Horse balm, rosemary, dandelion, procaine, sage, and lecithin have been suggested but long-term trials have not confirmed their usefulness.

**TABLE 4-6 Medications for Alzheimer's Disease and Possible Side Effects**

| Medication | Side Effects |
|---|---|
| Antidepressants | Minimal improvements have been noted. Mirtazapine may be useful in the treatment of the comorbid symptoms of weight loss, insomnia, and anxiety, a reflection of its enhancement of brain serotonergic and noradrenergic neurotransmission. Large, randomized controlled trials (RCTs) are needed. |
| Atypical antipsychotics | The efficacy of risperidone and olanzapine for the treatment of psychotic symptoms has been demonstrated by large RCTs in Alzheimer's disease. |
| Cholinesterase inhibitors donepezil (Aricept®), galantamine (Reminyl® or Razadyne®), rivastigmine (Exelon®) | Can slow agitation and the progression of cognitive and functional deficits in Alzheimer's disease in early stages by blocking acetylcholine breakdown. They improve cognitive function, behavior, and daily functioning. Nausea, diarrhea, insomnia, fatigue and loss of appetite may occur. |
| Cerefolin® | Contains methylated $B_{12}$ and folic acid, as well as N-acetylcysteine. |
| Coenzyme Q10 | Studies are inconclusive at this time. Indivuduals who take statins may need a supplement to reduce side effects such as rhabdomyolysis. |
| Hydergine | Relieves symptoms of declining mental capacity. Nausea and gastrointestinal (GI) distress are common. |
| Ibuprofen and other nonsteroidal, anti-inflammatory drugs | May reduce the risk of development of Alzheimer's disease by reducing inflammatory processes. |
| Insulin | If Alzheimer's disease is related to diabetes, it may be important to assure that adequate levels of insulin are available to the brain. |
| Laxatives | To control constipation. Offer high-fiber foods and sufficient fluid. |
| Mood stabilizers (lithium) | Low doses may be useful when combined with antipsychotics. Lithium regulates amyloid-beta precursor protein processing (Su et al, 2004). |
| Memantine (Namenda) | This regulates the activity of glutamate, a messenger chemical involved in learning and memory. |
| Selegiline (Eldepryl) | Selegiline should not be used with ginseng, ma huang (ephedra), yohimbe, or St. John's wort. |
| Statins | People taking statin drugs may have lower blood cholesterol and less incidence of Alzheimer's disease. They may have the ability to break down plaque-building amyloid protein. |
| Tacrine (Cognex) | For use in mild-to-moderate AD. May cause nausea, vomiting, or liver damage. Used less often today. |
| Vitamin E | Use of alpha-tocopherol has had mixed results in the literature (Petersen et al, 2005). |

http://www.alz.org/national/documents/topicsheet_treatments.pdf, accessed September 1, 2009.

- Omega-3 fatty acids are known to reduce cytokines and then lower the AD risk (Fotuhi et al, 2009; Morris, 2009; Staehelin, 2008).
- Panax ginseng has demonstrated efficacy in some studies (Lee et al, 2009).
  - SAM as a supplement can facilitate glutathione and acetylcholine utilization (Chan et al, 2008). Longer trials are needed.
  - Repeat testing of vitamin $B_{12}$ every 2 years. Where deficiency occurs, treat and prevent neurological and hematological consequences by providing $B_{12}$ (Prodan et al, 2009).

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- A nutrition education program intended for caregivers of AD patients can yield positive effects on patient weights and cognitive function.
- There is growing evidence for possible dietary risk factors in the development of AD and cognitive decline with age, such as antioxidant nutrients, fish, dietary fats, and B-vitamins (Morris, 2009). Promote use of the Mediterranean or DASH diets. Encourage use of fruits such as blueberries and other berries. Exercise and an antioxidant-rich diet may be protective against further cognitive decline.
- Encourage routines such as regular mealtimes, and good mouth care. Reduce distractions at mealtime.
- Refer family or caretakers to support groups. Long-term care or home care may be needed at some point in time.
- Special feeding methods may be needed. If the patient must be spoon fed, gently holding his or her nose will force the mouth open.
- Liquid supplements can add extra calories and protein without excessive expense. Baking nutritious cookie bars or snacks enhances calorie intake and addresses the need for sensory stimulation.
- Use unbreakable dishes and utensils to avoid injury. Cutting and preparing foods for the patient are useful.
- Offspring of affected family members should be tested and treated for hypertension; pro-inflammatory cytokines and ApoE genes should also be assessed (van Exel et al, 2009).

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing, especially with incontinence. Use of hand wipes before meals is recommended.

### *For More Information*

- Alzheimer's Association
  http://www.alz.org/
- Alzheimer's Disease Education and Referral (ADEAR) Center
  http://www.alzheimers.org
- Alzheimer's Disease International
  http://www.alz.co.uk/
- Alzheimer's Research Forum
  http://www.alzforum.org/
- Web MD
  http://www.webmd.com/alzheimers/default.htm

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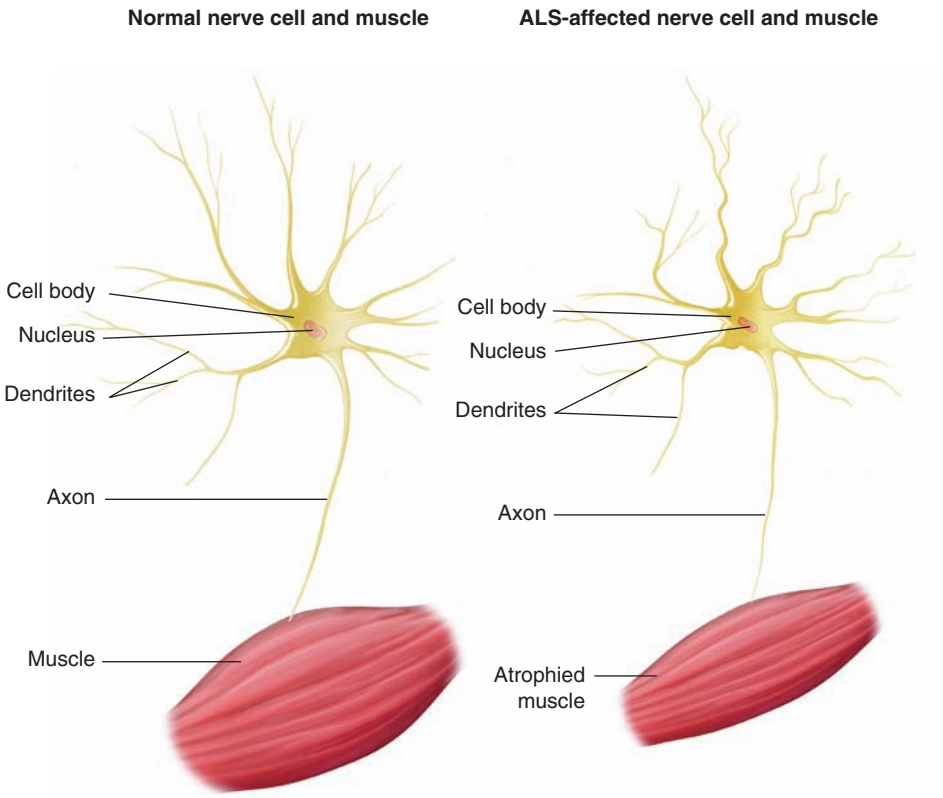
# AMYOTROPHIC LATERAL SCLEROSIS

## NUTRITIONAL ACUITY RANKING: LEVEL 3




Asset provided by Anatomical Chart Co.

## DEFINITIONS AND BACKGROUND

Amyotrophic lateral sclerosis (ALS) is a progressive motor neuron disease of adult life that destroys nerve cells from the spinal cord to muscle cells. The name "no muscle nourishment." Symptoms include muscular wasting and atrophy, drooling, loss of reflexes, respiratory infections or failure, spastic gait, and weakness. Respiratory failure occurs as a result of bulbar, cervical, and thoracic loss of motor neurons; inspiratory muscles are affected.

Men and women are affected equally, in about 20,000 people in the United States. ALS usually occurs after age 40; it is also known as progressive spinal muscular atrophy or Lou Gehrig's disease. Management of respiratory failure includes the use of strategies that limit aspiration pneumonia, the reduction in secretions, positioning of the patient to a maximal mechanical advantage, and use of noninvasive positive pressure ventilation.

Malnutrition is aggravated by elevated metabolic needs and swallowing dysfunction in the lower set of cranial nerves. The malnutrition produces neuromuscular weakness and adversely affects patients' quality of life. In later stages of the disease, percutaneous endoscopic gastrostomy (PEG) feeding may be needed. However, consider patient preferences and advance directives; individuals have the right to accept or refuse treatment (American Dietetic Association, 2008). Quality of life does not necessarily improve (Langmore et al, 2006).

Dietary factors have been suspected of being risk factors for ALS. Lycopene and magnesium are important nutrients (Oyanagi et al, 2006). Environmental exposure to arsenic depletes s-adenosyl-methionine (SAM) especially with folate insufficiency (Dubey and Shea, 2007). Elevated tHcy damages motor neurons and may be linked to faster progression of ALS (Zoccolella et al, 2008). However, there is no known cure at this time and ALS patients usually have respiratory distress, anxiety, pain, choking episodes, or pneumonia at the end of life.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Mutations in the c-1 (SOD-1) gene result in familial amyotrophic ateral sclerosis (FALS). Over 135 mutations have been identified in the various forms of ALS.

**Clinical/History**

Height
Weight
Weight changes
BMI (<18.5 indicates undernutrition)
Dietary/intake history
Swallowing difficulty
Temperature, fever?
I & O

Electromyogram (EMG)
Gag reflex
ALS Functional Rating Scale (ALSFRS-R)
Ventilatory dependency?

**Lab Work**

H & H
Serum Fe
$Na^+$, $K^+$
$Ca^{++}$, $Mg^{++}$
Albumin (alb), transthyretin
Transferrin
BUN, Creat
Nitrogen (N) balance
Gluc
CRP
tHcy
Serum folate, $B_{12}$, $B_6$
$pCO_2$, $pO_2$

## INTERVENTION

### OBJECTIVES

- Maintain good nutrition to prevent further complications. Meet extra energy requirements (Vaisman et al, 2009).
- Reduce difficulties in chewing and swallowing. Monitor gag reflex.
- Reduce the patient's fear of aspiration; test swallowing reflexes with water and feed slowly.
- Minimize the possibility of UTI and constipation.
- Correct negative nitrogen balance and nutritional deficiencies.
- Ease symptoms to maintain independence as long as possible.
- Reduce fatigue from the eating process; provide a slow pace to avoid choking.

### FOOD AND NUTRITION

- In initial stages, use a soft diet. Flaky fish, ground meats, and casseroles may be encouraged, along with foods moistened with gravies and sauces. Provide adequate fiber in the diet, perhaps Benefiber or psyllium when fibrous foods are no longer tolerated.
- The diet should include 2–3 L of water daily. Thicken liquids as needed with commercial thickeners, gelatin powder, or mashed potato flakes. Sips of liquid are best tolerated between bites of food.
- Place food at side of mouth and tilt head forward to facilitate swallowing, when possible.
- Enhance energy intake (Vaisman et al, 2009). Five to six small meals should be scheduled daily.
- Increase protein intake to counteract wasting.
- Diet and feedings should provide antioxidants such as vitamins E and C and selenium; zinc, magnesium, potassium, folate, omega-3 fatty acids, lycopene, and phosphorus.
- Foods should be moistened and not dry or crumbly. Cake and crackers should not be served plain; yogurt, applesauce, and pudding generally are acceptable.
- PEG or percutaneous endoscopic jejunostomy (PEJ) tube placement is well tolerated with dysphagia.

**SAMPLE NUTRITION CARE PROCESS STEPS**

**Swallowing Difficulty**

***Assessment Data:*** Food records indicating poor intake; weight loss; paralysis of throat muscles with difficulty swallowing.

***Nutrition Diagnoses (PES):*** Difficulty swallowing related to paralysis of throat muscles as evidenced by weight loss and dietary intake records showing 20–25% of meals consumed orally.

***Interventions:*** Education of patient and family about structured meals, use of tolerated liquids and pureed foods to improve intake of nutrients and energy, prevent further weight loss.

***Monitoring and Evaluation:*** Improved weight; delay of the need for tube feeding, or initiation early in the illness if patient wishes to have it.

### Common Drugs Used and Potential Side Effects

- Ceftriaxone alters glutamate and has been found to prolong survival in animal models of ALS.
- Riluzole (a glutamate release inhibitor and membrane stabilizer) has been used to block nerve cell destruction. It seems to slow the disease but not curb its progress. Adding vitamin E to this therapy does not seem to cause any improvement (Graf et al, 2005).
- Studies suggest that recombinant erythropoietin, magnesium, lithium and valproate, lycopene, or anti-inflammatory drugs may be beneficial; however, more clinical trials are needed.

### Herbs, Botanicals, and Supplements

- While no specific herbs and botanical products have demonstrated efficacy, supplement use is common in this population.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Dietary counseling is important, but oral intake rapidly becomes insufficient and enteral nutritional support may be needed. Discuss care plan in front of patient; include the patient in the decision.
- In early stages, discuss adding fiber to the diet to prevent constipation and explain which foods have fiber.
- Encourage the planning of small, adequately balanced meals.
- Carefully monitor the patient's weight loss; 10% loss is common.
- Lightweight utensils are beneficial. A referral to an occupational therapist is recommended.
- Minimize chewing, but avoid use of baby food. Puree adult foods, especially preferred foods that are seasoned as usual for the individual.

- In later stages, decide if enteral nutrition will be used. If care will be given at home, teach family members what they can do to provide the feedings.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

### *For More Information*

- ALS Association
  http://www.alsa.org/
- ALS Neurology Channel
  http://www.neurologychannel.com/als/index.shtml
- ALS Therapy Development Foundation
  http://www.als.net/

## AMYOTROPHIC LATERAL SCLEROSIS—CITED REFERENCES

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# BRAIN TRAUMA

## NUTRITIONAL ACUITY RANKING: LEVEL 4

### DEFINITIONS AND BACKGROUND

There are two types of brain injury: nontraumatic or traumatic. Nontraumatic head injury develops slowly, from arthritis, cancer, infections, or degeneration of the vertebrae. Traumatic brain injury (TBI) results from head injury after motor vehicle or industrial accidents, falls, fights, explosions, and gunshot wounds (40% involve alcohol use). The term TBI is not used for persons who are born with a brain injury or for injuries that happen during the birth process.

Any sudden impact or blow to the head (with or without unconsciousness) may cause a TBI, and two thirds of patients with TBI die before reaching a hospital. Low cerebral blood flow and cerebral perfusion pressure (CPP) are associated with poor outcome (White and Venkatesh, 2008).

Immediate signs of concussion (seen within seconds or minutes) include any loss of consciousness, impaired attention, vacant stare, delayed responses, inability to focus, slurred or incoherent speech, lack of coordination, disorientation, unusual emotional reactions, and memory problems. Classification is by location, effect, and severity. **Hypothalamic lesions** can promote hyperphagia. **Lateral lesions** can lead to aphasia and cachexia. **Frontal lobe damage** may result in loss of voluntary motor control and expressive aphasia. **Occipital lobe damage** impairs vision. **Temporal lobe damage** results in receptive aphasia and hearing impairments.

Hours, days, or even weeks after head injury, the patient may have persistent headache, dizziness with vertigo, poor attention or concentration, memory problems, nausea or vomiting, easy fatigue, irritability, intolerance for bright lights or loud noises, anxiety, depression, and disturbed sleep. Long-term TBI patients may exhibit dyspnea, vertigo, altered consciousness, seizures, vomiting, altered blood pressure (BP), weakness or paralysis, aphasia, and problems with physical control of hands, head, or neck with resulting difficulty in self-feeding. A brain injury often causes problems with understanding words, learning and remembering things, paying attention, solving problems, thinking abstractly, talking, behaving, walking, seeing, and hearing.

Brain trauma is accompanied by regional alterations of brain metabolism, overall reduction in metabolic rates, and persistent metabolic crisis (Vespa et al, 2005). Pyruvate dehydrogenase complex (PDHC) enzyme activity is lost with cerebral ischemia (Martin et al, 2005). Severe head injuries are also associated with negative nitrogen balances. With severe head injuries (Glasgow Coma Scale Score of 8), there is an increased tendency for gastric feeding to regurgitate into the upper airway; keeping the patient upright and checking residuals is important in such patients. Jejunal feedings are less apt to be aspirated. If the gastrointestinal (GI) tract cannot be used to reach nutritional goals within 3 days, total parenteral nutrition (TPN) is begun within 24–48 hours.

New neurons arise from progenitor cells that are maintained throughout adult life; they are enhanced by growth factors, drugs, neurotransmitters, and physical exercise and suppressed by aging, stress, glucocorticoids (Elder et al, 2006). Stem cell therapy has been studied for possible use in brain injuries.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** Brain trauma is acquired.

| | | |
|---|---|---|
| **Clinical/History** | BMI | BP |
| Height | Dietary/intake history | Temperature |
| Weight | | |

Visual field examination
Glasgow Coma Scale
Dysphagia?
Weight changes
Intracranial pressure
CT scan
Skull x-rays
Brain scan
Cerebral angiography
EEG
I & O

**Lab Work**

$pCO_2$ and $pO_2$
Alb, transthyretin
Urinary urea nitrogen (UUN) excretion (24-hour specimens)
Gluc (increased with brain ischemia)
CRP
Folic acid
Complete blood count (CBC)
Total lymphocyte count (TLC)
Transferrin
H & H
Serum Fe
$Na^+$, $K^+$
$Ca^{++}$, $Mg^{++}$
AST (increased with brain necrosis)
BUN, Creat
Alkaline phosphatase (alk phos)
Serum ethanol (ETOH)

## INTERVENTION

### OBJECTIVES

- Prevent life-threatening complications, such as aspiration pneumonia, meningitis, sepsis, UTIs, syndrome of inappropriate antidiuretic hormone (SIADH), hypertension, pressure ulcers, Curling's ulcer, and GI bleeding.
- Assess regularly the substrate needs to prevent malnutrition, cachexia, or overfeeding. Indirect calorimetry to determine the respiratory quotient and resting energy expenditure should be determined twice weekly.
- Based on the level of nitrogen wasting and the nitrogen-sparing effect of feeding, full nutritional replacement is desirable by day 7.
- Prevent or correct hyperglycemia by carefully regulating glucose and insulin intake.
- Provide adequate protein for improving nitrogen balance (serum albumin tends to be low, especially if comatose, and urinary losses may be twice the normal).
- Monitor hydration; prevent either dehydration or overhydration.
- Correct self-feeding, breathing, and swallowing problems. Promote return to self-care where possible.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Oral Food and Beverage Intake**

***Assessment Data:*** Food records indicating poor intake; comatose; requiring tube feeding.

***Nutrition Diagnoses (PES):*** Inadequate oral food and beverage intake related to coma following TBI as evidenced by requirement for tube feeding to meet elevated energy requirements.

***Interventions:*** Provision of enteral nutrition to meet energy, protein, glucose, lipid and micronutrients requirements and prevent weight loss.

***Monitoring and Evaluation:*** Tolerance of enteral nutrition; no physical signs of malnutrition; improvement in cognition or resolution of coma through recovery period.

- Prevent or reduce seizure activity, convulsions, intracranial edema, fluid overload (especially with TPN).
- After patient is stabilized, adapt to residual impairments.

### FOOD AND NUTRITION

- EN should begin as soon as the patient is hemodynamically stable, attempting to reach 35–45 kcal/kg and a protein intake of 2.0–2.5 g/kg as soon as possible. Tube feeding is preferable; if malabsorption persists, a short course of supplemental PN may help.
- The need for surgery or ventilation will have an effect on the ability to progress to any oral intake.
- Patients who are immobile for a long period of time may have a 10% decrease in weight from lowered metabolic rate. Energy intake will need to be varied accordingly.
- Increased urinary zinc losses can occur. Monitor potassium, phosphorus, and magnesium requirements as losses are often high. Otherwise, a general multivitamin–mineral supplement should suffice.
- Progress, when possible, to oral intake. A dysphagia may be needed.
- Use of probiotics (such as yogurt or buttermilk) can help to maintain GI integrity and immunity.
- Over time, a patient may gain excessive weight if the brain injury affected the hypothalamus. Some patients forget that they have eaten and state their constant hunger. Monitor energy intake carefully.

### Common Drugs Used and Potential Side Effects

- Analgesics are used for pain. Antacids and Pepcid may then be needed to reduce the onset of stress ulcers.
- Anticonvulsants may be needed to reduce seizure activity; these may deplete folic acid levels and other nutrients. Presence of food reduces effectiveness of the liquid form of phenytoin (Dilantin). Adjust phenytoin dosage rather than holding feedings.
- Insulin is used when hyperglycemia occurs or persists.
- Reglan may be used as a promotility agent in tube-fed patients to assist in transit time and to decrease the risk of aspiration.
- Soluble or mixed fibers (Benefiber or other soluble fiber supplements) or laxatives (Metamucil) are often helpful in alleviating constipation. However, bloating, nausea, diarrhea, or vomiting may result.

### Herbs, Botanicals, and Supplements

- Avoid using phenytoin (Dilantin) with evening primrose oil, gingko biloba, and kava.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage the patient to chew and swallow slowly, if and when the patient is able to eat solids.
- Gradually relearn self-feeding techniques.
- Be wary of extreme food temperatures; patients may have become less sensitized to hot and cold.

- Serve colorful and attractive meals for better acceptance.
- The team approach is beneficial, with occupational therapists, speech therapists, psychologists, and physical therapists helping design treatment plans.
- Plate guards, long-handled utensils, and other adaptive feeding devices may be useful. Discuss with the occupational therapist.
- Discuss a healthy eating pattern and use of foods such as yogurt for probiotics.
- Emotional changes are common after a head injury. Family members should be prepared to address changes that relate to mealtimes, eating patterns, weight management, and the need for consistency and structure.
- Many brain injury patients do not receive counseling about the long-term effects of their injury. Provide written instructions for review later.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important.
- Sanitize work areas before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

### *For More Information*

- Brain and Spinal Cord
  http://www.brainandspinalcord.org/
- Brain Injury Association
  http://www.biausa.org
- Brain Trauma Foundation
  http://www.braintrauma.org/
- National Institute of Neurological Disorders and Stroke
  http://www.ninds.nih.gov/

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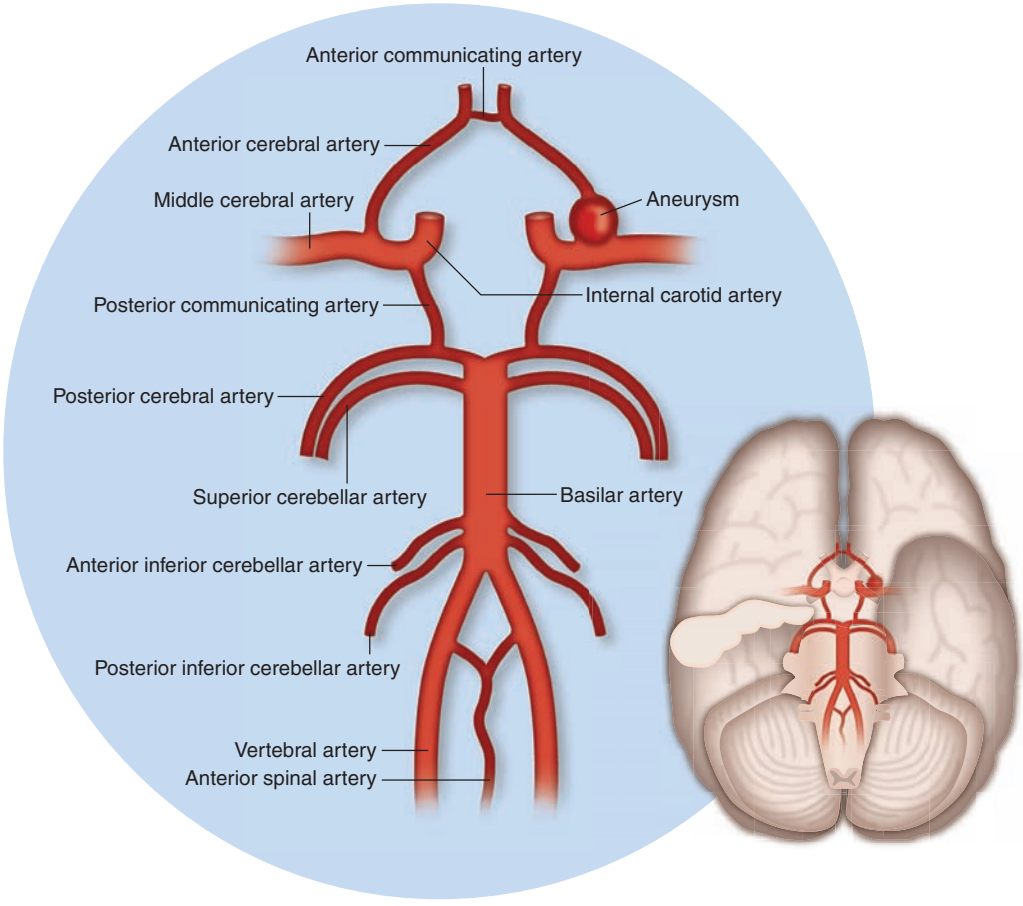
# CEREBRAL ANEURYSM

## NUTRITIONAL ACUITY RANKING: LEVEL 2–3



## DEFINITIONS AND BACKGROUND

A cerebral aneurysm may involve the dilation of a cerebral artery resulting from a weakness of the blood vessel wall. Symptoms include altered consciousness, drowsiness, confusion, stupor or coma, headache, facial pain, eye pain, blurred vision, vertigo, tinnitus, hemiparesis, elevated BP, and dilated pupils. Aneurysms may burst and cause hemorrhage. Epidemiological evidence suggests that most intracranial aneurysms do not rupture. It is important to identify which unruptured intracranial aneurysms (UIAs) are at greatest risk of rupture when considering which to repair (Wiebers et al, 2004).

An **intracranial** hemorrhage is bleeding inside the skull, usually from head injury. Bleeding within the brain is **intracerebral**. Hemorrhages between the brain and the subarachnoid space are **subarachnoid hemorrhages**; those between the meninges are **subdural hemorrhages**; and those between the skull and covering of the brain are **epidural hemorrhages**. Hemorrhagic stroke may occur. After an aneurysmal subarachnoid hemorrhage, nearly half of patients die, and the half who survive suffer from irreversible cerebral damage (Chen et al, 2004).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Alagille syndrome (AGS) is a dominantly inherited multisystem disorder involving the liver, heart, eyes, face, and skeleton, caused by mutations in Jagged1; intracranial bleeding is a recognized complication and cause of mortality. Otherwise, most aneurysms are not genetic.

**Clinical/History**

Height
Weight
BMI
Weight changes
Dietary/intake history
I & O
BP (increased)
Brain MRI
CT scan results
Angiography
Cerebrospinal fluid analysis
Pneumonia?
Fever?

**Lab Work**

Chol (LDL, HDL)
Triglycerides (trig)
Alb, transthyretin
Gluc
$Na^+$, $K^+$
H & H
Serum Fe
$pO_2$, $pCO_2$

## INTERVENTION

### OBJECTIVES

- Limit fluids as necessary to reduce cerebral edema.
- Rest is essential. Avoid constipation and straining at stool.
- Decrease or manage hypertension.
- Prevent further complications and lingering neurological problems.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Oral Food and Beverage Intake**

***Assessment Data:*** Semi-concious state, inability to eat orally.

***Nutrition Diagnoses (PES):*** Inadequate oral food and beverage intake related to recent TBI after a car accident as evidenced by cognitive deficits and high risk for aspiration and pneumonia.

***Interventions:*** EN because oral feeding not safe or feasible. Calculate protein, energy, fluid requirements as well as rate and goal intake.

***Monitoring and Evaluation:*** Tolerance of enteral nutrition; labs stable for glucose, prealbumin, electrolytes. Gradual return to oral diet if possible.

- Prepare for surgery if safe and possible. Reduce fever prior to surgery (Todd et al, 2009).
- Gradually encourage self-feeding.

### FOOD AND NUTRITION

- Nothing by mouth unless ordered; appropriate IVs are used. With cognitive progress, a tube feeding or diet will be prescribed.
- Restrict fluid, sodium, saturated fat and cholesterol if deemed necessary. Enhance potassium if necessary.
- Alter dietary fiber intake, as appropriate.
- Offer sufficient antioxidant foods and omega-3 fatty acids where appropriate.

### Common Drugs Used and Potential Side Effects

- In some cases, aspirin may be given at levels of 150–300 mg daily.
- Cardiovascular drugs are usually ordered according to significant parameters. Adjust dietary intake accordingly.
- Diuretics may be used. Monitor potassium replacement if furosemide is prescribed.
- Nimodipine is used to treat symptoms resulting from hemorrhage by increasing blood flow to injured brain tissue.
- Papaverine is used to improve blood flow in patients with circulation problems by relaxing the blood vessels.

### Herbs, Botanicals, and Supplements

- No specific herbs and botanical products have been used for cerebral aneurysm in any clinical trials.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- If enteral nutrition must continue at home, the caregiver should be taught appropriate and safe techniques.
- Discuss fiber sources from the diet. Foods such as prune juice or bran added to cereal can be helpful in alleviating constipation.

- Counsel regarding self-feeding techniques.
- Discuss role of nutrition in preventing further cardiovascular or neurological problems.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important.
- Sanitize counters or work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

### *For More Information*

- American Association of Neurological Surgeons
  http://www.aans.org/
- Brain Aneurysm Foundation
  http://www.bafound.org/
- National Institute of Neurological Disorders and Stroke
  http://www.ninds.nih.gov/disorders/cerebral_aneurysm/detail_cerebral_aneurysm.htm

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Uhl E, et al. Intraoperative computed tomography with integrated navigation system in a multidisciplinary operating suite. *Neurosurgery.* 64:651, 2009.

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# COMA OR PERSISTENT VEGETATIVE STATE

## NUTRITIONAL ACUITY RANKING: LEVEL 4

## DEFINITIONS AND BACKGROUND

Coma is the unconscious state in which the patient is unresponsive to verbal or painful stimuli. Impaired consciousness or coma can occur from a stroke, head injury, meningitis, encephalitis, sepsis, lack of oxygen, epileptic seizure, toxic effects of alcohol or drugs, liver or kidney failure, high or low blood glucose levels, or altered body temperature. Coma usually only lasts for a few weeks and most people recover fully. Medical staff use the Glasgow Coma Scale to determine prognosis. Often, the 1-month performance on measures such as Disability Rating Scale (DRS) and Glasgow Outcome Scale (GOS) scores help predict status 6 months post injury (Pastorek et al, 2004). Nutritional support is associated with improved survival in coma patients. Most patients are tube fed because it is safer and more practical than hand feeding. Clinical care factors such as time delay for orders and enteral access are impediments to EN provision in the first week of neurocritical illness (Zarbock et al, 2008).

A patient in a **permanently vegetative state (PVS)** does not have the ability to request or refuse treatment. The doctor determines the diagnosis of PVS. According to the American Dietetic Association position (2008), the definition of brain death is central to the dilemma of feeding permanently unconscious patients. Dietitians play an integral role with other members of the team in developing and implementing ethical guidelines for feeding patients (American Dietetic Association, 2008). Table 4-7 lists the consequences of withholding food and fluid from patients whose advance directives indicate no "heroic measures."

**TABLE 4-7 Consequences of Withholding Food and Fluid in Terminally Ill Patients**

Neither nutrition nor hydration improves comfort or quality of life in terminally ill patients. Physiological adaptation allows patients not to suffer from the absence of food, as follows:

1. Two thirds of the patients who are not fed or hydrated at the end of life feel no hunger. They usually have loss of appetite and reduced enjoyment of food.
2. Thirst and dry mouth are common initially. Use ice chips, lubricating the lips, and small amounts of food and water to reduce the thirst sensations from dehydration.
3. Dehydration eventually results in hemoconcentration and hyperosmolality with subsequent azotemia, hypernatremia, and hypercalcemia. These changes produce a sedative effect on the brain just before death.
4. Withholding or minimizing hydration can reduce disturbing oral and bronchial secretions, the need for frequent urination, and coughing from diminished pulmonary congestion.

Adapted from: American Dietetic Association. Position of the American Dietetic Association: ethical and legal issues in nutrition, hydration, and feeding. *J Am Diet Assoc.* 108:879, 2008.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Coma is generally a result of an accident or other head injury. Some genetic disorders may lead to coma if undiagnosed and untreated.

| Clinical/History | Dietary/intake history | motor, and verbal responses |
|---|---|---|
| Height | Unconsciousness | BP |
| Weight | Glasgow Coma Scale score (>13 mild; <8 severe) for eye, | I & O |
| BMI | | CT scan or MRI |
| Weight change (using a bed scale) | | |

**Lab Work**

H & H
Serum Fe
$pCO_2$, $pO_2$
Chol, full lipid profile (HDL, LDL)
Trig
Alb
Gluc
BUN, Creat
Urine tests for chemicals, glucose
Serum alcohol level
tHcy levels
Serum folic acid
Serum vitamin $B_{12}$

## INTERVENTION

### OBJECTIVES

- Maintain standards related to primary condition.
- When possible, elevate head to prevent aspiration during feeding process.
- Assess daily energy and fluid requirements. Adequate caloric intake is associated with improved outcome (Zarbock et al, 2008).
- Prevent or treat pressure ulcers, constipation, and other complications of immobility.
- For terminally ill patients, follow their wishes as directed by advance directives.

### FOOD AND NUTRITION

- Immediately give intravenous glucose until etiology is clearly identified. Parenteral fluids may also be appropriate at this time.
- Tube feed for increased energy and protein requirements every 2–3 hours, or as ordered by the physician. If tube fed, a formula with fiber can be helpful in preventing or easing constipation; be sure sufficient fluid is included as well.
- TPN may be appropriate for some persons, following evaluation of the original disorder, sepsis, and other complicating factors.
- Progress, when or if possible, to oral feedings.
- For patients who are terminally ill, gradual withdrawal of food and fluid is appropriate if directed by the patient's advance directives.

**SAMPLE NUTRITION CARE PROCESS STEPS**

**Inadequate Energy Intake**

***Assessment Data:*** Inability to eat orally, leading to poor intake. Weight changes; % usual body weight.

***Nutrition Diagnoses (PES):*** Inadequate energy intake related to coma and inability to chew and swallow as evidenced by intake meeting less than 10% of estimated requirements.

***Interventions:*** Enteral nutrition to meet estimated needs for energy and nutrients.

***Monitoring and Evaluation:*** Weight status, stabilization of labs, and improved energy intake.

### Common Drugs Used and Potential Side Effects

- Anticonvulsants, such as phenytoin (Dilantin), decrease folic acid over time. Avoid use with evening primrose oil, gingko biloba, and kava.
- Antacids may be needed to prevent stress ulcers.
- Cathartics are often used. Monitor for electrolyte imbalances.
- Steroids may be used. Side effects include sodium retention, increased losses of potassium and calcium and magnesium, and nitrogen depletion.

### Herbs, Botanicals, and Supplements

- No specific herbs and botanical products have been used in comatose patients in any clinical trials.
- With phenytoin (Dilantin), avoid use with evening primrose oil, gingko biloba, and kava.

### INTERVENTION: NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss with caretaker or family any necessary measures that are completed to provide adequate nourishment. Explain importance of prevention of complications such as aspiration.
- Evaluate potential for self-feeding over time.
- A Medic Alert bracelet or other ID is useful for persons with disorders that can lead to unconsciousness.

#### *Patient Education—Foodborne Illness*

- Careful food handling will be important. Caregivers must wash their hands before initiating tube feeding, TPN, or oral feeding process.
- Sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

#### *For More Information*

- Coma
  http://www.neuroskills.com/coma.shtml
- Glasgow Coma Scale
  http://www.neuroskills.com/glasgow.shtml
- Neurology Channel
  http://www.neurologychannel.com/coma/index.shtml

## COMA—CITED REFERENCES

American Dietetic Association. Position of the American Dietetic Association: ethical and legal issues in nutrition, hydration, and feeding. *J Am Diet Assoc.* 108:879, 2008.

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# EPILEPSY AND SEIZURE DISORDERS

## NUTRITIONAL ACUITY RANKING: LEVEL 2

### DEFINITIONS AND BACKGROUND

Epilepsy is a disturbance of the nervous system with recurrent seizures, loss of consciousness, convulsions, motor activity, or behavioral abnormalities. The seizures result from excessive neuronal discharges in the brain. A grand mal seizure involves an aura, a tonic phase, and a clonic phase. A petit mal seizure involves momentary loss of consciousness. A single seizure does not imply epilepsy. There are many forms of epilepsy, each with its own symptoms. In two thirds of cases, no structural abnormality is found. Incidence is two to six in 1000 people. Approximately 45,000 children under the age of 15 develop epilepsy each year, often those with cerebral palsy or spina bifida.

A ketogenic diet should be considered for refractory epilepsy (Papandreou et al, 2006; Vaisleib et al, 2004). Chronic ketosis modifies the tricarboxcylic acid cycle, increases GABA synthesis in brain, limits reactive oxygen species generation, and boosts energy production in brain tissue; these changes stabilize synaptic function and increase the resistance to seizures throughout the brain (Bough and Rho, 2007).

A ketogenic diet contains 70–90% fat with the remainder as protein and carbohydrates (CHO). A medium-chain triglyceride (MCT) diet alleviates some of the obstacles of compliance and acceptance. While overall caloric restriction may improve efficacy of the ketogenic diet (Cheng et al, 2004), the diet may slow growth (Peterson et al, 2005). An alternative diet that is not as strict uses a low–glycemic index treatment, with more liberal total carbohydrate intake (Pfeifer and Thiele, 2005).

Bone health, altered hepatic cytochrome P-450 enzymes, decreased metabolism of vitamin D, resistance to parathyroid hormone, inhibition of calcitonin secretion, and impaired calcium absorption are affected by use of antiepileptic drugs (Fitzpatrick, 2004).

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** In close relatives (parents, brothers, sisters, and children) of people with generalized epilepsy, the risk of epilepsy is about four times as high as in the general population. In the close relatives of people with partial epilepsy, the risk is twice as high as in the general population.

**Clinical/History**
Height
Weight

BMI
Dietary/intake history of fatty acids

BP
I & O
CT scan
Skull x-ray
EEG
DEXA scan

**Lab Work**
Urinary acetone (AM levels)

Serum $Ca^{++}$, $Mg^{++}$
$Na^{+}$, $K^{+}$
Alb, transthyretin
H & H
Alk phos

Chol, Trig
Serum folate
Urinary calcium
Uric acid

### INTERVENTION

#### OBJECTIVES

- Minimize seizures via medications, ketogenic diet, or lesionectomy.
- If drug therapy does not work (as in the case of intractable myoclonic or akinetic seizures of infancy), a ketogenic diet may be used (Neal et al, 2008). Reverse the usual ratio of cholesterol and fat. Provide a diet that avoids excess of CHO. Beware of changing the diet abruptly; a gradual approach is preferred.
- With signs of hyperuricemia or hypercalciuria, increase fluid intake and consider use of diuretics.
- Correct nutritional deficits from long-term anticonvulsant medication use (disorders of vitamin D, calcium, and bone metabolism). Phenytoin therapy (PHT) decreases serum folate by half, thereby increasing risk of deficiency.
- Monitor for possible long-term cardiac problems or a decline in bone health.

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Intake of Fatty Acids**

***Assessment Data:*** Frequent, treatment-refractable seizures in child; difficulty with self-feeding.

***Nutrition Diagnoses (PES):*** Inadequate intake of fatty acids related to high CHO intake as evidenced by frequent seizures and use of regular diet.

***Interventions:*** Food-Nutrient Delivery: Alter diet or tube feeding to increase ratio of lipid to CHO for ketogenic effect. Education of patient/caregiver about the role of lipids in brain health and preparation of the ketogenic diet; consequences of long-term use of anticonvulsants on bone health and folate status.

***Monitoring and Evaluation:*** Decreased frequency of seizures, tolerance of ketogenic diet, sufficient growth or weight maintenance, adequate bone density.

## FOOD AND NUTRITION

- Provide a diet reflecting the patient's age and activity. Protein should meet needs; such as 0.8–1 g/kg body weight.
- The high-fat, high-protein, low-carbohydrate Atkins diet is somewhat ketogenic and may be useful in managing medically resistant epilepsy (Kossoff and Rho, 2009).
- The ketogenic diet is a low-carbohydrate, adequate-protein, high-fat diet that biochemically mimics the fasting state and has been used to successfully treat seizures for 85 years (Huffman and Kossoff, 2006). However, the diet may be unpalatable. The diet follows a ratio of 3:1 or 4:1 of fats to carbohydrate and protein.
- MCTs are more ketogenic, having more rapid metabolism and absorption. MCTs provide 60% of kcal (the rest of the diet would consist of 10% other fats, 10% protein, and 20% carbohydrates). Stimulants such as tea, coffee, colas, and alcohol are not usually recommended with the ketogenic diet. If the pure ketogenic diet is not tolerated, modify it with low–glycemic index foods (Pfeifer and Thiele, 2005).
- Supplements may be needed, especially calcium, vitamin D, folic acid, vitamins $B_6$ and $B_{12}$.
- Add sufficient fiber and fluid for relief of constipation.

## Common Drugs Used and Potential Side Effects

- Cough syrups, laxatives, and other medications may contain a high CHO content; monitor for interactions with the diet.
- Common anticonvulsants and potential side effects are listed in Table 4-8. Anticonvulsant therapy interferes with vitamin D metabolism, leading to a calcium imbalance, rickets or osteomalacia. Therapy with D3 is recommended.

## Herbs, Botanicals, and Supplements

- Vitamin $B_6$ is associated with neuronal function. Avoid high doses of pyridoxine with phenobarbital or phenytoin because seizure control might be compromised. If vitamin $B_6$ is added to either drug regimen, keep it at the lowest effective dose and monitor serum drug levels.
- St. John's wort should not be used with monoamine oxidase inhibitors (MAOI), selective serotonin reuptake inhibitors (SSRIs), cyclosporine, digoxin, oral contraceptives, HIV protease inhibitors, theophylline, warfarin, or calcium channel blockers such as amlodipine, diltiazem,

**TABLE 4-8 Medications Used in Epilepsy**

| Generic Name | Trade Name | Possible Side Effects |
|---|---|---|
| Carbamazepine | Carbatrol, Tegretol XR, Tegretol | Dry mouth, vomiting, nausea, anorexia, low red blood cell and white blood cell counts. |
| Clonazepam | Klonopin | Anorexia, weight loss or gain, increased thirst. |
| Diazepam | Diazepam Intesol, Diastat, Valium | Anorexia, weight loss or gain, increased thirst. |
| Ethosuximide | Zarontin | Gastrointestinal upset, anemia, and weight loss. Take with food or milk. |
| Felbamate | Felbatol | Constipation, nausea, vomiting, and anorexia. |
| Fosphenytoin | Cerebyx | Water-soluble phenytoin. May need vitamin D, calcium, thiamin, magnesium. |
| Gabapentin | Neurontin | Weight gain and increased appetite occur. Take magnesium supplement separately by 2 hours. |
| Lamotrigine | Lamictal | Anorexia, weight loss, nausea and vomiting, abdominal pain. |
| Levetiracetam | Keppra | Anorexia, headache. |
| Oxcarbazepine | Trileptal | Restrict fluid with hyponatremia. |
| Phenobarbital | Luminal | Depletes vitamins D, K, $B_{12}$, $B_6$, folate, and calcium. Nausea, vomiting, constipation, sedation, and anorexia can occur. Limit caffeine and alcohol. May elevate serum cholesterol levels. |
| Phenytoin | Dilantin | Gum hyperplasia and carbohydrate intolerance. It binds serum proteins and decreases folate, vitamins $B_{12}$ and C, and magnesium absorption. Be careful with vitamin $B_6$; excesses can reduce drug effectiveness. Stop tube feedings 30 minutes before and after administration of the medication; nutritional intake may need to be calculated over 21 versus 24 hours. |
| Primidone | Mysoline | Gastrointestinal upset, anemia, and weight loss. Take with food or milk. Primidone is similar to a barbiturate; vomiting may occur. |
| Tiagabine | Gabitril | Mouth ulcers, nausea and vomiting may occur. |
| Topiramate | Topamax | Weight loss and anorexia are common. |
| Valproate, valproic acid, divalproex sodium | Depacon, Depakote, Depakene, Depakote ER | Nausea, vomiting, anorexia, weight gain, or hair loss. |
| Vigabatrin | Sabril | Visual field loss can occur. |
| Zonisamide | Zonegran | Anorexia and weight loss are common. |

or verapamil. Avoid use with benzodiazepines such as alprazolam, clonazepam, diazepam, and midazolam.
- Psyllium and ginseng should not be used with divalproex sodium (Depakote) or lithium.
- With phenytoin (Dilantin), avoid use with evening primrose oil, gingko biloba, and kava.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Ketogenic diets cause nausea and vomiting; a small drink of fruit juice can help relieve the symptoms. Regular monitoring of the diet is crucial.
- An ID tag, such as Medic Alert, is recommended.
- To increase long-chain triglycerides (LCTs), add sour cream, whipped cream, butter, margarine, or oils to casseroles, desserts, or other foods. To use MCT, add it to salad dressings, fruit juice, casseroles, and sandwich spreads. Pseudo ice cream may be made with frozen, flavored whipped cream.
- Women who have epilepsy and wish to have children need advice about medications and possible side effects. Pregnancy itself can increase seizure frequency; infants can be born with low birth weight, developmental delay, or epilepsy (Yerby et al, 2004).
- Because of the potential for loss of bone mineral density, discuss use of more calcium and vitamin D-rich foods. A multivitamin–mineral supplement may be recommended.
- Alcohol should be avoided.

#### *Patient Education—Foodborne Illness*

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

#### *For More Information*

- American Epilepsy Society
  http://aesnet.org
- Epilepsy Foundation
  http://www.EpilepsyFoundation.org

### EPILEPSY AND SEIZURE DISORDERS—CITED REFERENCES


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# GUILLAIN–BARRÉ SYNDROME

## NUTRITIONAL ACUITY RANKING: LEVEL 3

### DEFINITIONS AND BACKGROUND

Guillain–Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy with rapidly increasing weakness, numbness, pain, and paralysis of the legs, arms, trunk, face and respiratory muscles. It often occurs after infection with influenza or *Campylobacter jejuni;* bloody diarrhea, fever, cramping, and headache are presenting symptoms. In general, the role of *C. jejuni* has been greatly underestimated (Schmidt-Ott et al, 2006). GBS may progress to respiratory failure; paralysis of lower extremities or quadriplegia; unstable BP; aspiration; dysphagia; difficulty with chewing; impaired speech; muscular pain; low-grade fever; tachycardia; weight loss, anorexia; UTIs. Sometimes ventilatory assistance is needed.

Most people recover fully from GBS, but some may need intensive care support followed by wheelchair assistance. There is no treatment that has been totally effective. Most people recover within a few weeks, but some may still have residual effects for years.

### ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** It is believed that GBS results primarily after infection and not from genetic causes.

| Clinical/History | | Lab Work |
|---|---|---|
| Height | Vomiting | CBC |
| Weight | Bloody diarrhea? | H & H |
| BMI | Fatigue and weakness | Serum Fe |
| Weight changes | Nerve conduction velocity test | Alb |
| Dietary/intake history | Loss of reflexes (knee, etc.) | $pO_2$, $pCO_2$ |
| BP | | Lumbar puncture for CSF protein levels |
| Temperature | | Gluc |
| Dysphagia | | |

### SAMPLE NUTRITION CARE PROCESS STEPS

**Intake of Unsafe Food**

***Assessment Data:*** Diet history revealing intake of undercooked chicken at a sporting event, followed by bloody diarrhea, fever, and vomiting.

***Nutrition Diagnoses (PES):*** Intake of unsafe food related to *C. jejuni and* medical diagnosis of Guillan–Barre as evidenced by GI symptoms and bloody diarrhea, weakness, and fatigue.

***Interventions:*** Education about proper food handling and consumption of foods properly cooked to desired internal temparture.

***Monitoring and Evaluation:*** Improvement in symptoms and gradual improvement in quality of life; better understanding of food handling and issues related to food safety.

## INTERVENTION

### OBJECTIVES

- Meet added energy requirements from fever, weight loss.
- Adjust diet or method of feeding for chewing and swallowing problems.
- Wean, if possible, from ventilator dependency.
- Improve neurological functioning and overall prognosis.

### FOOD AND NUTRITION

- Acute: Intravenous fluids will be required. Tube feeding or TPN may be necessary while patient is acutely ill over a period of time. Increased energy intake and protein may be necessary; increase lipid intake to reduce $CO_2$ production while on the ventilator.
- Progression: a thick, pureed diet may be beneficial with dysphagia. When safe and tolerated, a soft or general diet may be used.
- Supplement oral intake with frequent snacks, such as shakes or eggnog, if unintentional weight loss has occurred. A vitamin–mineral supplement may be beneficial, especially if intake has been poor.

### Common Drugs Used and Potential Side Effects

- Antibiotics may be needed for UTIs.
- Autoimmune globulin may be given.
- Analgesics are used to reduce pain and inflammation.
- Steroids are seldom used except for chronic relapsing polyneuropathy; their effects can be deleterious over time, especially for bone health.
- Vasopressors may be used.

### Herbs, Botanicals, and Supplements

- High doses of pyridoxine ($B_6$) should not be taken with phenobarbital or phenytoin. Keep it at the lowest effective dose and monitor serum drug levels.
- No studies have been conducted for efficacy of herbs or botanical products in GBS.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss adequacy of energy and protein intake to improve weight status and nutritional health.
- Avoid foodborne illnesses, upper respiratory infections, and exposure to other illnesses.
- Avoid constipation by use of fruits, vegetables, crushed bran, prune juice, and adequate fluid intake.
- Encourage self-feeding where possible. Arrange for special feeding utensils if needed.

#### *Patient Education—Foodborne Illness*

- *C. jejuni* is the most frequently diagnosed bacterial cause of human gastroenteritis in the United States. Avoid drinking raw milk; eating raw or undercooked meat, shellfish, and poultry; eating tofu or unwashed raw vegetables.
- Hand washing is important. Wash hands with soap before handling raw foods.
- Prevent cross-contamination in the kitchen. Proper refrigeration and sanitation are also essential.

#### *For More Information*

- CDC: Seasonal Flu and GBS
  http://www.cdc.gov/FLU/about/qa/gbs.htm
- Guillain-Barré Foundation International
  http://gbs-cidp.org/
- NINDS Information Page
  http://www.ninds.nih.gov/disorders/gbs/gbs.htm

## GUILLAIN–BARRÉ SYNDROME—CITED REFERENCE

Schmidt-Ott R, et al. Improved serological diagnosis stresses the major role of *Campylobacter jejuni* in triggering Guillain-Barré syndrome. *Clin Vaccine Immunol.* 13:779, 2006.

# HUNTINGTON'S DISEASE

## NUTRITIONAL ACUITY RANKING: LEVEL 3

### DEFINITIONS AND BACKGROUND

Huntington's disease (HD) is a genetic, autosomal-dominant, neurodegenerative disorder. There is a defective gene that leads to microscopic death of selected neurons (Arrasate et al, 2004; Zhang et al, 2008). Normally, huntingtin's protein is cleared away through the Ubiquitin Proteosome System (UPS) in which proteins that are not needed or that have misfolded are tagged for degradation by ubiquitin (HDSA, 2009). Transglutaminase (TGase) activity is increased in affected regions of the brain.

HD develops in middle to late life, with involuntary, spasmodic, irregular movements (chorea), cerebral degeneration, cognitive decline, and speech difficulties. HD differs from AD in that there is loss of control of voluntary movements. Behavioral changes begin 10 years before the movement disorder, which may begin between age 35 and 40 years.

Nutritional intake plays an important role. Folic acid affects DNA methylation; coenzyme Q10 and unsaturated fatty acids are important for neuroprotection (Bonelli and Wenning, 2006).

Remotivation therapy leads to increased self-awareness, increased self-esteem, and improved quality of life. Duration of HD is generally 13–15 years before death, which often results from pneumonia or a fall. Stem cell transplantation shows promise for treatment; the therapy uses small cell ribonucleic acid (siRNA) to prevent the mutant proteins from being reproduced.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** The defective gene on the short arm of chromosome 4 causes abnormal polyglutamine expansion within the protein huntingtin (HTT).

The DNA segment known as a CAG trinucleotide repeats with three DNA building blocks (cytosine, adenine, and guanine) that appear multiple times in a row, usually 10–35 times; the CAG segment is repeated 36–120 times in HD (NIH, 2009).

**Clinical/History**

- Height
- Weight
- BMI
- Weight changes
- Dietary/intake history
- Ability to self-feed
- Chewing and swallowing difficulties
- I & O
- Brain CT scan
- Depressed mood, irritability
- Obsessive and compulsive
- Dementia
- Grimacing
- Involuntary movements (chorea)
- Unified Huntington's Disease Rating Scale

**Lab Work**

- BUN/Creat
- Serum glucose
- H & H
- CRP
- Acetylcholine and dopamine levels
- Alb, transthyretin
- tHcy levels
- Serum folate
- Vitamin $B_{12}$
- PLP levels or serum vitamin $B_6$

## INTERVENTION

### OBJECTIVES

- Promote normal nutritional status, despite tissue degeneration. Extra energy intake is important (Trejo et al, 2004).
- Encourage the patient to self-feed until this is no longer possible.
- Swallowing problems are significant (see Dysphagia, Section 7). Avoid aspiration of solids and liquids.
- Manage gluten intolerance if celiac disease is present.

### FOOD AND NUTRITION

- Provide a diet that gives sufficient energy and protein to prevent pressure ulcers and other sequelae. Usually 1–1.5 g/kg protein is needed. A patient with HD may need up to 5000 kcals/d. In later stages, if weight gain is a problem, change diet as needed.
- Use a thick, pureed, or chopped diet as appropriate. Feed slowly to prevent choking. Small, frequent meals are suggested.
- Include adequate liquid as dehydration is common.
- Tube feed when necessary; bolus feedings are usually tolerated.
- Provide adequate fiber (e.g., prune juice or tube feedings that contain fiber) for normal elimination.
- Supplement with a multivitamin–mineral supplement; folic acid and other B-complex vitamins may be especially important.

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Swallowing Difficulty**

***Assessment Data:*** Weight changes and eating difficulty, dysphagia, and lack of muscle coordination.

***Nutrition Diagnoses (PES):*** NC 1.1 Swallowing difficulty related to dysphagia and lack of muscle coordination as evidenced by choking at mealtime and with thin liquids and recent loss of 10 lb.

***Interventions:*** Education of caregiver about thickening liquids for safer swallowing and the possible use of percutaneous gastrostomy feeding tube as needed.

***Monitoring and Evaluation:*** No choking episodes; tolerance of thickened liquids or enteral nutrition. Improved weight status.

- If gluten intolerance occurs, omit gluten from the diet from wheat, barley, rye and oat products, and flours. Label reading is essential.

### Common Drugs Used and Potential Side Effects

- Supplement with vitamin E, antioxidants, and omega-3 fatty acids to reduce inflammation; folic acid, vitamins $B_{12}$ and $B_6$ to lower serum tHcy levels if elevated.
- Minocycline may slow the disease process by blocking caspases from entering the brain.
- Riluzole has been used with some success and few side effects as a membrane stabilizer and glutamate-release inhibitor.
- Tetrabenazine (Xenazine) reduces chorea, and was approved in 2008 in the United States. It can cause insomnia or nausea. Neuroleptics and benzodiazepines may also be used.
- Antiparkinsonian medications such as Sinemet help with hypokinesia and rigidity.
- A new drug is being tested; called ACR16, it stabilizes brain levels of dopamine for motor, cognitive, and psychiatric changes.

### Herbs, Botanicals, and Supplements

- Avoid large doses of vitamin $B_6$.
- No clinical trials have proved efficacy for use of herbs and botanicals.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Semisolid foods may be easier to swallow than thin liquids. Teach family or caretakers about the Heimlich maneuver to manage episodes of choking.
- Adding protein and calories through supplements or nutritionally dense foods may be essential.
- If the patient or family wishes to forego tube feeding and hydration, the dietitian should discuss all possible consequences of malnutrition that may occur including dehydration and pressure ulcers.
- Encourage genetic counseling; each child of an affected parent has a 50% chance of inheriting the disease.

#### *Patient Education—Foodborne Illness*

- Careful food handling will be important.
- Sanitize counters or work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of tube feeding formula in the home and institution.

#### *For More Information*

- Huntington's Disease Society of America
  http://www.hdsa.org/
- Huntington's National Research Roster
  http://hdroster.iu.edu/index.asp

### HUNTINGTON'S DISEASE—CITED REFERENCES

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# MIGRAINE HEADACHE

## NUTRITIONAL ACUITY RANKING: LEVEL 1

### DEFINITIONS AND BACKGROUND

Migraine is a neurological process of the trigeminovascular system. Migraine involves paroxysmal attacks of headache, vasospasm, and increased coagulation, often preceded by visual disturbances. Nausea, vomiting, and acute sensitivity to light or sound may occur. These headaches affect 12% of the adult population (28 million people) in the United States and cause a significant economic loss in productivity. Women may be affected as a result of hormones (Martin et al, 2006). Lack of food or sleep, MT disturbances, exposure to light, anxiety, stress, fatigue, or hormonal irregularities can set off a migraine attack. In addition, a drop in serotonin or estrogen, or intake of vasodilators in some foods may cause blood vessels to swell and aggravate migraines. Studies have linked migraine with epilepsy, sleep disorders, ear problems, and vertigo (Eggers, 2007). In addition, elevated tHcy levels may contribute. A B-complex vitamin containing riboflavin, folic acid, vitamins $B_6$ and $B_{12}$ is recommended.

Reactions to food are often within 24 hours after consuming the offending food or beverage. Treatment begins with a headache-food diary and the selective avoidance of foods presumed to trigger attacks; omission of all potential

triggers is not recommended. Immunoglobulin E–mediated food allergy is infrequent.

Celiac disease may present with neurological symptoms, including migraines. Migraine may also arise because of disruption in neurovascular endothelia caused by elevated tHcy (Lea et al, 2009). Lowering tHcy through vitamin supplementation reduces migraine disability in some individuals (Lea et al, 2009).

Vascular-amine toxicity causes a rapid increase in BP when high-tyramine foods such as cheese, wine, beer, fava beans, and sauerkraut are eaten in combination with medications such as MAOIs. Limit tyramine to 25 mg/d; provide instructions for patients on MAOIs.

Exercise, relaxation, massage therapy, biofeedback, and other therapies limit discomfort in migraine treatment. Migraines may be reduced with intake of omega-3 fatty acids from fish oil and from intake of olive oil. Long-term prophylactic drug therapy is appropriate after exclusion of headache-precipitating trigger factors, including dietary factors. Improved sleep hygiene, moderation of caffeine intake, regular exercise, and identification of provocative influences such as stress, foods, and social pressures are essential. People who are prone to migraines are at risk for stroke and should be monitored carefully.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Migraine may be caused by inherited abnormalities. Elevated tHcy promotes migraine disability in a subgroup of patients who have the MTHFRC677 T genotype (Lea et al, 2009).

**Clinical/History**

- Height
- Weight
- BMI
- Dietary/intake history
- Headache symptoms and duration
- Foods eaten in past 24 hours; diary
- History of similar reactions
- Recent illnesses
- Dehydration or edema?
- Migraine Disability Assessment Score
- Migraine with aura?

**Lab Work**

- Serum histamine
- Prothrombin time (PT) or international normalized ratio (INR)
- Serum $Na^+$, $K^+$ $Ca^{++}$, $Mg^{++}$
- Gluc
- Serum tissue transglutaminase IgA (tTGA) antibodies

## INTERVENTION

### OBJECTIVES

- Eliminate stressors and triggers (crowds, bright lights, noises). Reduce or eliminate use of foods that cause migraines for the individual.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Poor Food Choices**

***Assessment Data:*** Food records indicating poor intake of fruits and vegetables, fish, and olive oils while using high amounts of bacon and lard; frequent migraine headaches.

***Nutrition Diagnoses (PES):*** Poor food choices related to nutrient density as evidenced by frequent migraine headaches (>2 weekly) and daily intake of bacon and lard while avoiding use of fish, olive oil, fruits, and vegetables.

***Interventions:*** Education about nutrient density in foods and encouragement to increase fruits, vegetables, fish, and olive oils and reduce use of other fats.

***Monitoring and Evaluation:*** Fewer migraine headaches, better quality of life, reduction in use of migraine medicines.

- Encourage a well-balanced diet, with adequate meal spacing to prevent fasting or skipping of meals.
- Obesity is a factor in some chronic daily headaches; weight loss may be indicated.
- Improve quality of life. Reduce migraine intensity and duration.
- Reduce frequency of migraines and increase responsiveness to therapy.
- Prevent complications, such as ischemic stroke.

### FOOD AND NUTRITION

- Promote regular mealtimes, regular exercise, and adequate relaxation.
- Data surrounding the role of certain foods and substances in triggering headaches are controversial, but certain patients may be sensitive to phenylethylamine, tyramine, aspartame, monosodium glutamate, nitrates, nitrites, alcohol, and caffeine (Sun-Edelstein and Mauskop, 2009). Limit sensitive foods specific to the individual (see Table 4-9).

### Common Drugs Used and Potential Side Effects

- Antiemetics may be prescribed if there is nausea or frequent vomiting.
- Botulinum toxin type A (BoNTA; BOTOX) treatment may be a useful option for headache patients demonstrating poor compliance with oral prophylactic regimens (Cady and Screiber, 2008).
- Medicines can be used to relieve pain and restore function during attacks. Drugs such as almotriptan, eletriptan, naratriptan, rizatriptan, sumatriptan, and zolmitriptan may be used to enhance the effects of serotonin with few side effects.
- Drugs designed to lower BP also may prevent headaches, such as thiazides, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, and angiotensin II receptor agonists.
- If effective medicines are not found to treat headache at its onset, daily preventive medicines are sometimes used.

**TABLE 4-9 Foods Implicated in Various Types of Headaches**

| Food | Description |
|---|---|
| Alcohol | Champagne and red wine contain both phenols and tyramine; sulfites may also be involved as a trigger. Beer may be another problem.<br>Alcoholic beverages: limit to two normal size drinks of choices such as Cutty Sark scotch, Seagram's VO whisky, Riesling wine (National Headache Foundation, 2010). |
| Caffeine-containing products | Coffee, tea, and cola can trigger caffeine-withdrawal headache from methyl xanthines (18 hours after withdrawal); taper withdrawal gradually. Coffee is the major source of caffeine in adults; soft drinks are the major source for children and teens (Frary et al, 2005). |
| Cheese and tyramine | Aged cheese that contains tyramine has been implicated. Ripened cheeses: cheddar, emmentaler, stilton, brie, and camembert. |
| Chocolate | Chocolate contains phenylethylamine (no clear relationship to migraine). |
| Fermented foods | Chicken livers, aged cheese such as cheddar, red wine, pickled herring, chocolate, broad beans, and beer contain tyramine (no clear relationship to migraine). |
| Fruits | Bananas, figs, raisins; some citrus fruits. |
| Gluten | Celiac disease has been associated with migraine (Bushara, 2005). |
| Histamine-containing foods | Scombroid fish (slightly spoiled). |
| Ice cream | Some individuals are sensitive to the cold. |
| Nuts, peanuts | Some contain vasodilators. Avoid nuts and peanut butter if necessary. |
| Processed meats | Hot dogs, bacon, ham, and salami contain nitrites. |
| Sulfites | Some people respond to the sulfites in shrimp, packaged potato products. |
| Vegetables | Onions, pea pods, lima beans. |

This may include anticonvulsants; nonsteroidal anti-inflammatory drugs (NSAIDs) such as Ibuprofen; tricyclic antidepressants (TCAs), and serotonergic agents.

- MT may be useful in migraine headaches (Masruha et al, 2008; Vogler et al, 2006).
- Avoid using aspirin in children younger than 15 because of the potential for Reye's syndrome.

## Herbs, Botanicals, and Supplements

- While capsaicin from hot chili peppers may be used as a source of relief for cluster headache pain, it does not relieve migraines.
- The following supplements may help in the preventative treatment of migraines, in a decreasing order of preference: magnesium, Petasites hybridus, feverfew, coenzyme Q10, riboflavin, and alpha lipoic acid (Sun-Edelstein and Mauskop, 2009).
- Feverfew and riboflavin have not shown strong efficacy (Tepper, 2008). Side effects of feverfew include decreased platelet aggregation if used with warfarin, aspirin, and ticlopidine. NSAIDs (ibuprofen, indomethacin, advil) decrease the herb's anti-inflammatory action; do not use together.
- Where food-plant sensitivities exist (melon/ragweed, carrot/potato, apple/birch, wheat/grasses, ragweed/dandelion greens) bee pollen and echinacea may cause an allergic reaction.
- Evening primrose, red pepper, willow, and ginger have been recommended; no studies prove efficacy. Counsel about avoiding herbal teas, especially if they contain toxic ingredients.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Fasting can increase likelihood of a headache. Regular mealtimes are important.
- Encourage the patient to identify various, individual triggers. Teach the patient how to keep an accurate food diary for food sensitivities. Read food labels and avoid items containing ingredients that are problematic.
- Monitor drugs taken for underlying conditions such as asthma, reactive airway disease, hypertension, glaucoma, and ear problems. Discuss the possibility of "medication overuse headache" or rebound headaches after caffeine from the diet or medicines.
- Psychotherapy may be useful for mental and emotional stress. Regular sleeping patterns are needed; evaluate for insomnia and sleep apnea.
- Evidence-based behavioral medicine treatments include patient education, cognitive behavioral therapy (CBT), biofeedback, relaxation training, and stress management (Andrasik et al, 2009). Some authorities may also recommend acupuncture.

### *Patient Education—Food Safety*

- Food storage is a major issue. Extended holding times, especially in high-protein foods, is a concern.
- Teach safe food handling, handwashing, and other practices.

### *For More Information*

- American Council for Headache Education
  http://www.achenet.org

- American Headache Society
  http://www.ahsnet.org
- Medline: Headache
  http://www.nlm.nih.gov/medlineplus/headache.html
- National Headache Foundation—Food
  http://www.headaches.org/education/Headache_Topic_Sheets/Diet_and_Headache_-_Foods
- National Migraine Association
  http://www.migraines.org/
- Neurology Channel
  http://www.neurologychannel.com/migraine/index.shtml
- Tyramine-Restricted Diet
  http://www.headaches.org/pdf/Diet.pdf
- World Headache Alliance
  http://www.w-h-a.org

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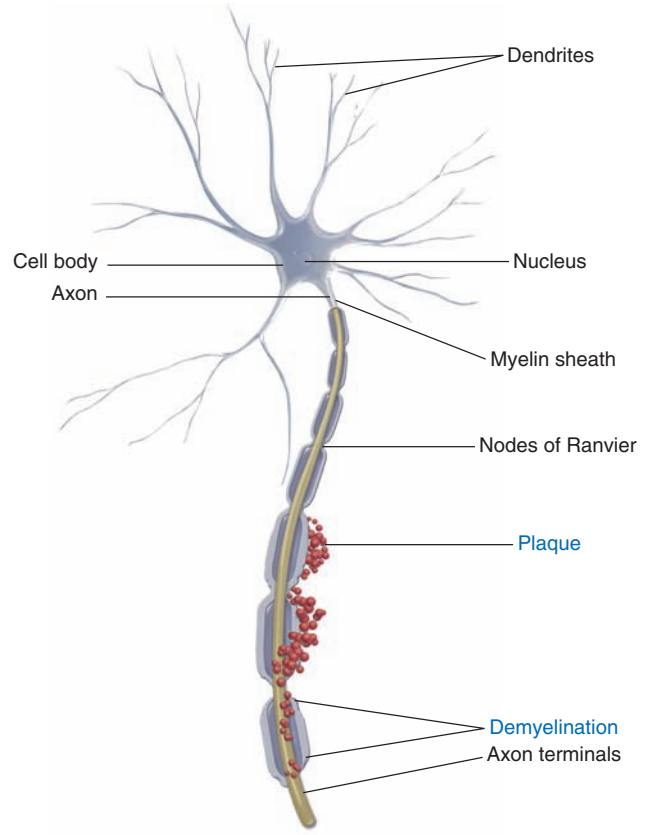
# MULTIPLE SCLEROSIS

## NUTRITIONAL ACUITY RANKING: LEVEL 2




Asset provided by Anatomical Chart Co.

## DEFINITIONS AND BACKGROUND

Multiple sclerosis (MS) involves scarring and the loss of myelin sheath, the insulating material around nerve fibers. The disease causes progressive or episodic nerve degeneration and disability. Insufficient vitamin $D_3$ plays a role; persons living in colder climates and those with less sun exposure are more prone (Kantarci and Wingerchuk, 2006). MS has a much higher incidence among Caucasians than in any other race and affects women two to three times as often as men. MS affects over 400,000 people in the United States and 2.5 million people worldwide.

MS is an autoimmune disease. Proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) instruct the adaptive immune system (T and B cells and other cells and proteins) to launch an attack to suppress the "invader." Th17 T-cells are a type of immune cell known to play a role in the onset of MS. Onset is usually between 20 and 40 years of age (average age, 27 years). Symptoms include tingling; numbness in arms, legs, trunk, or face; double vision; fatigue; weakness; clumsiness; tremor; stiffness; sensory impairment; loss of position sense; and respiratory problems. Dysphagia can occur. Spasticity and bladder dysfunction are also common.

After diagnosis, 70% of persons with MS are as active as previously. Relapsing-remitting MS shows clear relapses with some amount of recovery in between; it affects about 80% of all people with MS. Ten percent of individuals with MS have primary progressive MS, without relapses. A description of the types of MS is in Table 4-10.

**TABLE 4-10 Types of Multiple Sclerosis**

| Name | Characteristics |
|---|---|
| Relapsing-Remitting Multiple Sclerosis (RRMS) | Symptom flare-ups followed by recovery; stable between attacks |
| Secondary-Progressive Multiple Sclerosis (SPMS) | Second phase of RRMS; progressive worsening of symptoms with or without superimposed relapses; treatments may delay this phase |
| Primary-Progressive Multiple Sclerosis (PPMS) | Gradual but steady accumulation of neurological problems from onset |
| Benign | Few attacks and little or no disability after 20 years |
| Progressive-Relapsing Multiple Sclerosis (PRMS) | Progressive course from the onset, sometimes combined with occasional acute symptom flare-ups |
| Malignant of Fulminant Multiple Sclerosis | Rapidly progressive disease course |

Multiple Sclerosis Association of America, http://www.msassociation.org/about_multiple_sclerosis/commontypes, accessed May 25, 2009.

During chronic CNS inflammation, nicotinamide adenine dinucleotide (NAD) concentrations are altered by T-helper Th1-derived cytokines; use of pharmacological doses of nontryptophan NAD precursors have been suggested (Penberthy and Tsunoda, 2009).

Magnesium, vitamin $B_6$, vitamin $B_{12}$, zinc, vitamin $D_3$, vitamin E, selenium, and omega-3 fatty acids have been suggested. Vitamin $B_{12}$ is important for proper myelination of the spinal cord (Montanha-Rojas et al, 2005).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** MS tends to run in families. Altered human leukocyte antigen (HLA) genes in chromosome 6; IL2RA and IL7RA as receptors for interleukins; and the gene that encodes kinesin (KIF1B) may enhance the onset of MS.

**Clinical/History**
- Height
- Weight
- BMI
- Dietary/intake history
- BP
- I & O
- Edema
- Temperature

**Lab Work**
- Alb
- Chol
- Trig (may be low in autoimmune disorders)
- $Na^+$, $K^+$
- Serum $D_3$
- Alk phos
- H & H
- Serum Fe
- Gluc
- MRI scan
- Evoked potentials test
- CSF (WBC, γ-globulin are increased)
- EEG
- L:S ratio

### SAMPLE NUTRITION CARE PROCESS STEPS

**Self-Feeding Difficulty**

***Assessment Data:*** Difficulty with self-feeding, loss of strength in hands, dependent on assistants.

***Nutrition Diagnoses (PES):*** Self-feeding difficulty related to poor hand strength and MS as evidenced by inability to use standard meal utensils.

***Interventions:*** Alteration of food consistency and use of adaptive feeding utensils to adjust and enhance ability to feed self; finger foods where possible, such as julienne vegetables, sandwiches cut into strips, soup in a mug.

***Monitoring and Evaluation:*** Ability to grasp items at mealtime as evidenced by increased ability to do some self-feeding with finger foods and less dependency on staff for assistance.

## INTERVENTION

### OBJECTIVES

- During the chronic phase of the disease, treatment goals are to reduce the incidence of respiratory infections, UTIs, bowel problems, muscle spasms, contractures, pressure ulcers, constipation or fecal impactions.
- Adjust energy intake to avoid excessive weight gain, if this becomes a problem.
- Maintain good nutritional status. Since vitamin $D_3$ seems to play a role in autoimmunity, supplement with 800 IU daily.
- Reduce fatigue associated with mealtimes. Frequent, small meals may be better tolerated than three large ones.
- During the active phase of the disease, corticosteroids may be used to decrease symptoms. Alter diet accordingly.
- Prevent chronic diseases such as coronary heart disease or osteoporosis, which may occur with immobilization.

### FOOD AND NUTRITION

- Normal protein and adequate carbohydrate intakes are recommended. Use olive oil and fish oil (omega-3 fatty acids) more often.
- Provide adequate intake of multivitamins, especially vitamins $D_3$ and $B_{12}$.
- Laxative foods and liquids may ease constipation.
- Control sodium intake during steroid therapy. Otherwise, sodium plays an important role in lipid/protein transport in myelin tissues.
- Small, frequent meals may be better tolerated than large meals. If swallowing difficulties increase and coordination decreases, foods may need to be pureed, liquefied or fed by tube.
- To prevent UTIs, cranberry juice is quite effective (Raz et al, 2004).

## Common Drugs Used and Potential Side Effects

- A man-made retinoid (AM80) prevents early symptoms of the autoimmune disease by blocking the function of Th17 T-cells.
  - Immune-modifying drugs must have FDA approval. Interferon injections are useful. Interferon-β1 a (Avonex or Rebif) may cause nausea, diarrhea, liver damage, flu-like symptoms, headache, infections, or anemia. Interferon-β1b (Betaseron) may cause weight changes, abdominal pain, diarrhea, constipation, fever, headache, hypertension, or tachycardia.
  - Corticosteroids are not FDA-approved for use in MS. If used, they require controlled sodium intake. Glucose intolerance, negative nitrogen balance, and decreased serum zinc, calcium, and potassium may occur.
  - A combination of CT and a subtherapeutic dose of 1,25(OH)(2)$D_3$ suppresses autoimmune encephalitis (EAE) without causing hypercalcemia (Becklund et al, 2009). Further studies are needed.
  - Antispasticity drugs such as baclofen (Lioresal) may cause nausea, diarrhea, and constipation. If Sinemet (L-dopa) is used, avoid large doses of vitamin $B_6$.
  - The immunosuppressant azathioprine (Imuran) reduces new brain lesions in relapsing-remitting MS (Massacesi et al, 2005). Its use is still experimental.
  - Cannabinoids are potent immunosuppressive and anti-inflammatory agents; cannabinoid receptor 1 (CB1) is expressed on the cells of the CNS and cannabinoid receptor 2 (CB2) is expressed on immune cells; they affect apoptosis, and suppress cytokine and chemokine production (Rieder et al, 2009).
  - A complete list of medications is available at: http://www.msassociation.org/about_multiple_sclerosis/medications/types/

## Herbs, Botanicals, and Supplements

- Many patients with MS seek alternative therapies for their pain, fatigue, and stress. Diet, essential fatty acid (EFA) supplements, vitamin–mineral supplements, homeopathy, botanicals (Shinto et al, 2004) as well as exercise, herbal therapy, cannabis, massage, and acupuncture (Olsen, 2009).
- Based on available evidence, the prophylactic use of vitamin D is a viable option as an adjunct to conventional medicine (Kimball et al, 2007; Namaka et al, 2008).
- St. John's wort should not be used with MAO inhibitors, SSRI antidepressants, cyclosporine, digoxin, oral contraceptives, HIV protease inhibitors, theophylline, warfarin, or calcium channel blockers such as amlodipine, diltiazem, or verapamil. No studies have been conducted for efficacy in MS patients.
- Stinging nettle, pineapple, black currant, and purslane have been recommended for MS; no clinical trials have proven efficacy.
- Despite beneficial reports regarding nonherbal supplements such as ALA, luteolin, evening primrose oil and vitamins such as $B_{12}$, the lack of evidence does not support their prophylactic use (Namaka et al, 2008).

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Teach the patient how to control energy intake, especially if inactive.
- Discuss the role of fat and vitamin E in myelin sheath formation and maintenance, and where to find sources of linoleic acid and omega-3 fatty acids from the diet.
- Teach the patient about foods high in fiber.
- Avoid total inactivity. Physical therapy may be beneficial.
- Encourage moderate exposure to sunlight for vitamin D.
- Use tabletop cooking methods and equipment to avoid lifting. Utensils with large handles may be useful in food preparation and self-feeding. Foods may need to be cut before serving.
- Allergen-free, gluten-free, pectin-free, fructose-restricted, raw foods diets, and liquid diets are ineffective.
- Avoid smoking.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

### *For More Information*

- Consortium of Multiple Sclerosis Centers
  http://www.mscare.org
- Multiple Sclerosis Association of America
  http://www.msaa.com/
- National MS Society
  http://www.nmss.org/

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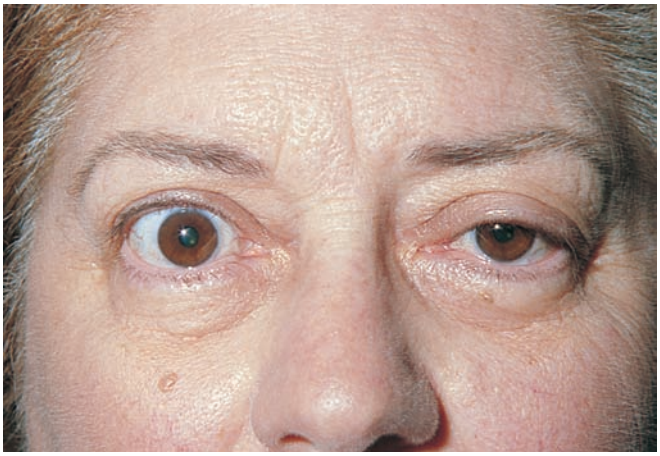
# MYASTHENIA GRAVIS AND NEUROMUSCULAR JUNCTION DISORDERS

## NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Tasman W, Jaeger E. *The Wills Eye Hospital Atlas of Clinical Ophthalmology,* 2nd ed. Lippincott Williams & Wilkins, 2001.

## DEFINITIONS AND BACKGROUND

Myasthenia gravis (MG) is an autoimmune disorder caused by autoantibodies against the nicotinic acetylcholine receptor on the postsynaptic membrane at the neuromuscular junction (Thanvi and Lo, 2004). The neuromuscular junction lies beyond the protection of the blood–brain barrier and is particularly vulnerable to antibody-mediated attack. Individuals with seronegative MG have autoantibodies to acetylcholine receptors and muscle-specific kinase (MuSK protein) for cell signaling (NINDS, 2009).

Few types are congenital, most are acquired. Acquired neuromuscular junction disorders include botulism, autoimmune MG, and drug-induced MG. General immunosuppression is primary. Treatments that reduce complement-mediated damage or inhibit the binding of pathogenic antibodies are under development (NINDS, 2009). The disease frequently is associated with thymic morphologic abnormalities; removal of the thymus gland works for about 70% of cases (Juel and Massey, 2005).

Symptoms and signs of MG include drooping eyelids (ptosis), double vision (diplopia), fatigue, general weakness, dysphagia, weak voice, inability to walk on heels, pneumonia or respiratory arrest. Diabetes, sleep apnea, and thyroid problems are common in persons who have MG and should be closely monitored.

MG occurs in approximately 18,000 affected people in the United States. Incidence begins with the first peak in the third decade and a second peak in the sixth decade (Thanvi and Lo, 2004). Plasmapheresis can be used during a crisis to remove the abnormal antibodies. A myasthenic crisis is defined as the need for mechanical ventilatory support, usually following progressive weakness and oropharyngeal symptoms (Bershad et al, 2008).

When MG is suspected, a tensilon test involves insertion of a small intravenous catheter through which tensilon is given; this very short-acting drug blocks the degradation of acetylcholine. The short-term availability of acetylcholine results in improved muscle function, often in the eye area.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** In MG, the expression of acetylcholine receptors (AChRs) in the thymus is under the control of the autoimmune regulator protein (AIRE). Polymorphisms in the AChR promote early onset of disease. Congenital myasthenia and congenital myasthenic syndrome are caused by defective genes.

**Clinical/History**

- Height
- Weight
- BMI
- Dietary/intake history
- I & O
- Weight changes
- Ptosis
- Diplopia
- Tensilon test
- Chest CT scan for thymoma
- Sleep apnea?

**Lab Work**

- $Na^+$, $K^+$
- BP
- CRP
- Acetylcholine antibodies test
- EMG
- Gluc
- $Mg^{++}$, $Ca^{++}$
- Alb
- H & H
- Serum Fe
- T3, T4, TSH

### SAMPLE NUTRITION CARE PROCESS STEPS

**Dysphagia**

***Assessment Data:*** Swallowing study, weight changes, BMI 17.

***Nutrition Diagnoses (PES):*** Difficulty swallowing related to neuromuscular disorder (MG) as evidenced by aspiration of food and liquids into lung on swallowing evaluation and weight loss (BMI now 17).

***Interventions:*** Thickened liquids and pureed foods as tolerated; PEG tube placement and enteral nutrition at night to regain lost weight.

***Monitoring and Evaluation:*** Weight gain, no further incidents of aspiration, tolerance of thickened liquids and pureed foods and/or enteral nutrition when necessary.

## INTERVENTION

### OBJECTIVES

- Increase the likelihood of obtaining adequate nutrition by altering the consistency of foods. This is necessary when muscles used in chewing and swallowing are weakened. Work with Speech Therapist accordingly.
- Feedings should be small to reduce fatigue. Allow adequate time to complete meals.
- Prevent permanent structural damage to the neuromuscular system during crises.
- Prevent or manage respiratory failure.
  - Encourage participation in rehabilitation programming.

### FOOD AND NUTRITION

- Diet should include frequent, small feedings of easily masticated foods.
- Provide tube feeding when needed.
- If corticosteroids are part of treatment, use a low-sodium diet. Provide adequate potassium supplements.
- Use a high-energy diet if weight loss occurs, which is common.
- Avoid giving medications with thin liquids such as coffee or juice; give with milk and crackers or bread.

### Common Drugs Used and Potential Side Effects

- Prednisone is usually started at a high dose every day, then reduced to every other day. In the event of intolerable side effects or failure of treatment, other immunosuppressants may be used, most commonly azathioprine (Imuran). GI distress, nausea, vomiting, and anorexia may occur.
- Transient symptomatic control can be achieved by the initiation of pyridostigmine (Mestinon) that blocks the degradation of acetylcholine at the neuromuscular junction, increasing the level of acetylcholine with better muscle response to stimulation by the nerve. Mestinon is a temporary symptomatic treatment and does not reverse the course of the illness. Limit sodium intake. Anorexia, abdominal cramps, diarrhea, and weakness may result. Long-acting capsules may be needed if morning weakness persists.
- Long-term use of antacids negatively affects calcium and magnesium metabolism.

### Herbs, Botanicals, and Supplements

- No studies have been conducted for efficacy of herbs or botanicals in MG patients.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Show the patient how to prepare foods and nutrient-dense beverages with the use of a blender, if necessary.
- Indicate how to take medication with food or milk. Discuss potential side effects.
- Avoid alcohol.
- Food and utensils should be arranged within easy reach of the patient; lightweight items are preferable.
- The International Classification of Functioning (ICF), Disability and Health rehabilitation practitioners is a worldwide accepted model providing a universal language for the description and classification of functioning (Rauch et al, 2008).

#### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

#### *For More Information*

- Myasthenia Gravis Foundation of America
  http://www.myasthenia.org/
- National Institute of Neurological Disorders and Stroke
  http://www.ninds.nih.gov/disorders/myasthenia_gravis/detail_myasthenia_gravis.htm
- Neuromuscular Junction Disorders
  http://www.neuro.wustl.edu/neuromuscular/synmg.html

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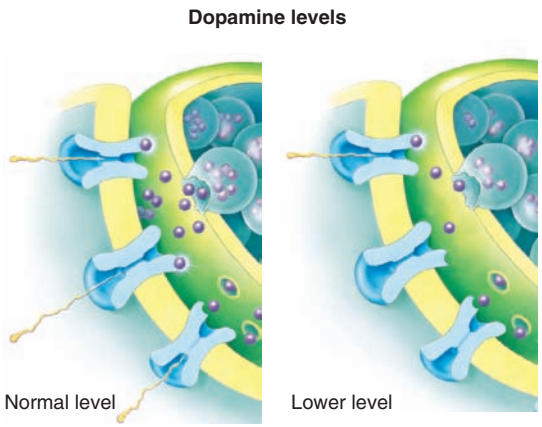
# PARKINSON'S DISEASE

## NUTRITIONAL ACUITY RANKING: LEVEL 2

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## DEFINITIONS AND BACKGROUND

Parkinson disease (PD) is an age-related neurodegenerative disorder that affects 1–2% of persons aged 60 years and older (Olanow et al, 2009). There are diminished levels of dopamine at the basal ganglia of the brain, causing tremor of hands, arms, legs, jaw, and face; rigidity of limbs and trunk; slowness of gait; coordination difficulty; chewing problems; dysphagia; problems with speech. A test for PD progression includes a decline in ability to smell and the speed of wrist movements. Depression and dementia-related symptoms may also occur. Levodopa must be provided. Although levodopa continues as the gold standard for efficacy, its chronic use is associated with potentially disabling motor complications (Poewe, 2009).

Approximately 24 conditions are categorized as PDs. Between 1–1.5 million people are affected, men slightly more often than women. Life expectancy is 12.5 years after diagnosis. Yet, causes and pathophysiology are poorly understood. Oxidative stress contributes to apoptotic death of dopamine neurons (Bournival et al, 2009). Long-term exposure to manganese, herbicides, pesticides, or high intake of iron with high manganese may promote PD symptoms (Ascherio et al, 2006; Fitsanakis et al, 2006). Some medications such as major tranquilizers or metoclopramide can also cause PD-like symptoms.

Esophageal motor abnormalities and constipation are common. Constipation appears about 10–20 years prior to motor symptoms (Ueki and Otsuka, 2004). Unintentional weight loss is frequent, resulting in increased morbidity and mortality. Weight loss occurs from increased energy expenditure due to tremor, dyskinesias, and rigidity; reduced energy intake due to olfactory dysfunction, cognitive impairment, depression, dysphagia, and disability; and medication-related side effects, including dry mouth, nausea/vomiting, appetite loss, anorexia, insomnia, fatigue, and anxiety. PD is progressive. Advancing disease is associated with the emergence of freezing, falling, and dementia which are not adequately controlled with dopaminergic therapies (Olanow et al, 2009).

Increased plasma tHcy accelerates the selective dopaminergic cell death underlying PD (De Lau et al, 2005). Adequate B-complex intake and measurement of serum levels of $B_{12}$ may be important. Higher serum vitamin $B_{12}$ levels are associated with lower dyskinesia risk (Camicioli et al, 2009), whereas folate therapy does not seem to be singularly protective (Chen et al, 2004).

Overall, neuroprotection is desirable. Oxidative stress initiates or promotes degeneration of neurons; antioxidant therapy may be protective. Resveratrol in grape juice and red wine, and quercetin in green tea are two natural polyphenols that have preventive qualities in PD (Bournival et al, 2009). Drinking 4–5 cups of coffee daily and foods rich in vitamin E and vitamin $D_3$ may also be protective (Evatt et al, 2008).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** There is significant variation in the prevalence of PD between different populations. Rates are highest in populations of European origin. Melanocortin 1 receptor (MC1R) genes are associated with red hair and fair skin (Han et al, 2006); risk for PD seems to be highest among those with this Cys/Cys genotype (Gao et al, 2009). There are many other candidate genes for PD: alpha-synuclein, DJ-1, PINK-1 (kinase), and UCHL-1. Two mutated copies of the Parkin gene are needed for the rare, autosomal recessive form of PD, with early onset in the fourth decade.

**Clinical/History**

- Height
- Weight
- BMI
- Dietary/intake history
- BP
- I & O
- Dysphagia
- Tremors
- Bradykinesia
- Difficulty arising from a chair
- Slowed activities of daily living
- Depression, anorexia
- Constipation
- Micrographia
- Reduced arm swing
- Postural instability
- DEXA for bone mineral density
- Insomnia
- Stooped, shuffling gait
- Urinary frequency and urgency
- Skin evaluation; melanoma?
- Unified Parkinson Disease Rating Scale (UPDRS)

**Lab Work**

- Plasma 25-hydroxyvitamin D (25[OH]D)
- $Na^+$, $K^+$
- N balance
- H & H
- Serum Fe
- Alb, transthyretin
- Dopamine
- Norepinephrine
- BUN, Creat
- ALT, AST
- Gluc
- Plasma urate
- $Ca^{++}$, $Mg^{++}$
- Serum manganese
- Serum tHcy
- Serum folate and $B_{12}$

### SAMPLE NUTRITION CARE PROCESS STEPS

**Dehydration**

***Assessment Data:*** Weight loss, poor intake of food and fluids, swallowing difficulty, easy fatigue at mealtimes and early satiety, coughing after swallowing, poor skin turgor.

***Nutrition Diagnoses (PES):*** Inadequate fluid intake related to swallowing difficulty and poor intake as evidenced by I & O records showing consumption of 25% of estimated needs and decreased intake of thin liquids because of coughing and early satiety.

***Interventions:*** Swallowing evaluation (Speech Therapy) with selection of appropriate thickened liquids or pureed foods and ways in which fluids can be added to foods; use of IV fluids as needed.

***Monitoring and Evaluation:*** Improvement in I & O related to fluids from food and beverages; improved skin turgor; return to previous weight.

## INTERVENTION

### OBJECTIVES

- Supply dopamine to the brain; monitor diet therapy accordingly.
- Maintain optimal physical and emotional health. Exercise may be protective, especially for men (Carne et al, 2005).
- Improve the ability to eat. Use semisolid foods rather than fluids when sucking/swallowing reflexes are reduced. Drooling may be a problem. Request a swallow evaluation from a speech therapist to determine proper consistency of foods, and (See Dysphagia entry in Section 7).
- Provide adequate energy to prevent weight loss, and avoid gaining excessive weight as well.
- Provide adequate hydration, especially when thickened liquids are needed. Coffee is a good choice.
- Correct alterations in GI function (i.e., increased transit time, heartburn, and constipation).
- Preserve functioning; delay disability as long as possible.
- When used MAOIs, use a tyramine-restricted diet to prevent severe headaches, blurred vision, difficulty thinking, seizures, chest pain or symptoms of a stroke.
- Lower elevated tHcy levels where needed (Caccamo et al, 2007).

### FOOD AND NUTRITION

- A high intake of protein diminishes the effectiveness of levodopa; use 0.5 g/kg of body weight. If unplanned weight loss occurs, up to 1–1.5 g/kg plus extra energy may be needed. For some, a protein redistribution diet is used (i.e., low-protein breakfast and lunch with high-protein dinner and snack). This diet is not always effective, and often provides insufficient protein. Timing of levodopa should be monitored to avoid conflicting responses to protein at mealtimes.
- Plan diet according to results of swallowing evaluation. Cut, mince, or soften foods as required. Use small, frequent meals if needed.
- To increase fiber, add crushed bran to hot cereal or try prune juice.
- A multivitamin–mineral supplement may be beneficial, especially for vitamins C, E, and the B-complex vitamins. Folate, vitamins $B_6$ and $B_{12}$ will be important to lower elevated tHcy levels (Biselli et al, 2007; Grimble, 2006).
- Highlight foods such as vegetable oils, salad dressings, nuts, green tea, coffee, turmeric, and antioxidant-rich fruits and vegetables.
- If needed, follow the Tyramine-restricted diet: avoid aged and fermented meats, sausages, and salamis; pickled herring; spoiled or improperly stored meat, poultry and fish with changes in coloration, odor, or mold; spoiled or improperly stored animal livers; broad bean pods; sauerkraut; aged cheeses; red wines and all varieties of tap beer and beers that have not been pasteurized; over-the-counter supplements containing tyramine; concentrated yeast extract; soybean products such as soy sauce and tofu.

### Common Drugs Used and Potential Side Effects

- Elevated plasma tHcy levels have been observed in PD patients treated with levodopa. New approaches may be needed for management of PD in persons who have MTHFR alleles (Caccamo et al, 2007; Camicioli et al, 2009; Todorovic et al, 2006).
- See Table 4-11 for specific medications used in PD.

### Herbs, Botanicals, and Supplements

- Because creatine kinase/phosphocreatine system plays a significant role in the CNS with the high and fluctuating energy demand, exogenous creatine supplementation tends to reduce neuronal cell loss (Andres et al, 2008).
- Coenzyme Q10 may be beneficial, but more research is needed.
- Other forms of CAM therapy are common, but patients do not always tell their health providers. Because of potential interactions with medications and excess costs, patients should be encouraged to discuss their use with their doctor before taking any forms of alternative medicine. Use of evening primrose, St. John's wort, passionflower, velvet bean, or gingko have not been proven effective.
- Kava should not be taken by patients with PD; it decreases effectiveness of medications.
- Ginseng, ma huang (ephedra), yohimbe, and St. John's wort should not be used with MAOIs, including selegiline (Eldepryl).

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Education for the patient and family, access to support groups, regular exercise, and good nutrition are essential. Provide tips on antioxidant foods such as blueberries, green tea, coffee.
- Explain how to blenderize food or how to make food and beverages more nutrient dense, as needed.

**TABLE 4-11 Medications for Parkinson's Disease and Possible Side Effects**

| Medication | Side Effects |
|---|---|
| **Antidepressants** | Weight gain, dry mouth, or nausea can result. |
| **Anticholinergics**<br>Cogentin (benztropine), Artane (trihexyphenidyl), Ethopropazine | Confusion, agitation, dizziness, sedation, euphoria, tachycardia, hypotension, dry mouth, constipation, nausea, urinary retention and blurred vision. |
| **Antiviral agent**<br>Symmetrel (amantadine) | Possible adverse effects include anorexia, dry mouth, nausea, constipation, dizziness, insomnia, blurred vision, depression, ataxia, confusion, fatigue, leg/ankle edema, hallucinations, anxiety, and livedo reticularis (skin discoloration). |
| **Catechol-O-methyltransferase (COMT) inhibitors**<br>Tasmar (tolcapone), Comtan (entacapone) | These drugs slow the breakdown of dopamine. Diarrhea, orthostatic hypotension, hallucinations, sleep disturbances, dyskinesias, muscle cramping, and vivid dreams may occur with use. Liver function testing should be scheduled regularly while on these medications. |
| **Dopamine agonists**<br>Parlodel (bromocriptine), Mirapex (pramipexole), Requip (ropinirole) | These stimulate dopamine receptors. Edema, psychosis, nausea, headache, fatigue, confusion, somnolence and "sleep attacks." With ropinirole, fewer side effects, such as dyskinesia, have been identified. Some studies suggest that dopamine agonists, rather than levodopa, should be the initial symptomatic therapy in Parkinson's disease. |
| Apokyn (apomorphine hydrochloride) | Approved for the treatment of acute, intermittent hypomobility episodes associated with advanced Parkinson's disease. |
| **Ibuprofen** | Users of ibuprofen are less likely to develop Parkinson's disease than nonusers. |
| **Levodopa/carbidopa**<br>Sinemet, Sinemet CR, Atamet, Madopar | Nausea, vomiting, weakness, hallucinations, mental confusion, orthostatic hypotension, fatigue, sudden daytime sleep onset, insomnia, elevated serum glucose and homocysteine, and anemia. Large neutral amino acids block levodopa absorption, both from the gut and at the blood–brain barrier. Levodopa preparations should be taken 30–60 minutes prior to meals; intake of vitamin $B_6$ should be limited to DRI levels. Up to 15 mg of vitamin $B_6$ can be taken daily in either food or supplement form. Today's preparations combine levodopa with carbidopa. Increase intake of foods rich in vitamin $B_{12}$, folate, and vitamin C. |
| **MAO type B inhibitors**<br>Eldepryl (selegiline), Azilect (rasagiline) | Insomnia, dry mouth, confusion, hypertension, abdominal pain, and weight loss. Selegiline should not be used with ginseng, ma huang (ephedra), yohimbe, or St. John's wort. A low tyramine diet should be used with rasagiline. |

- Help patient to control weight, which may fluctuate from either reduced mobility or the inability to ingest sufficient quantities.
- Place all foods within easy reach of the patient. Braces may help the patient control severe tremors at mealtime.
- Music therapy, Tai Chi, and yoga help to relieve depression and improve balance.
- Discuss how to resolve issues such as weight loss, constipation, osteopenia, gastroesophageal reflux disease (GERD), side effects of medications, xerostomia, and dehydration.
- Deep brain stimulation of the subthalamic nucleus shows promise. Medtronic has two new devices that can be used with PD patients to help control tremors: Activa RC and Activa PC.
- Maintain bone mineral density, as hip fractures are common. BMD is related to leg muscle strength (Pang and Mak, 2009).

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

### *For More Information*

- American Parkinson's Disease Association
  http://www.apdaparkinson.org/
- Michael J. Fox Foundation for Parkinson's Research
  http://www.MichaelJFox.org/
- National Parkinson's Foundation
  http://www.parkinson.org/
- Nutrition for Parkinson's
  http://www.nutritionucanlivewith.com/index.html
- Parkinson's Disease Foundation, Inc.
  http://www.pdf.org/
  http://www.pdf.org/pdf/FactSheet_Nutrition.pdf
- Parkinson's Genetic Research Group
  http://depts.washington.edu/pgrgroup/
- Society for Neuroscience
  http://web.sfn.org/
- We Move
  http://www.wemove.org/par/
- Young Parkinson's
  http://www.youngparkinsons.org/pages/index/siteindex.htm

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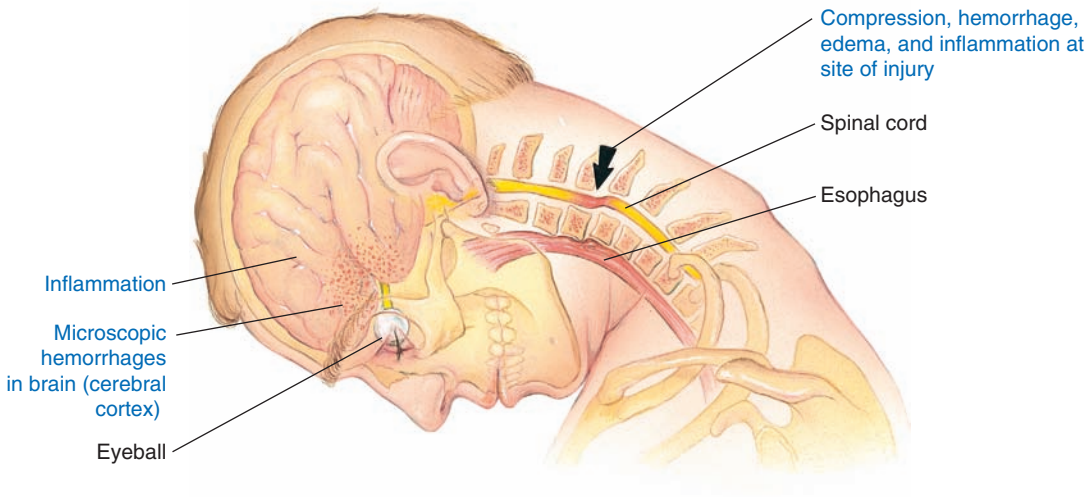
# SPINAL CORD INJURY

## NUTRITIONAL ACUITY RANKING: LEVEL 3

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## DEFINITIONS AND BACKGROUND

Spinal cord injury (SCI) is often caused by traffic accidents, falls, diving accidents, sports injury, or gunshot wounds. Partial versus total self-care deficits depend on resulting paralysis or loss of sensation below the site of the injury. Classification usually includes the cause, direction of injury, level of injury, stability of vertebral column, and degree of cord involvement. See Table 4-12.

Most injury causes permanent disability or loss of movement (paralysis) and sensation below the site of the injury. Paralysis that involves the majority of the body, including the arms and legs, is called quadriplegia or tetraplegia because of injuries to one of the eight cervical segments of the spinal cord. Those with paraplegia have lesions in the thoracic, lumbar, or sacral regions of the spinal cord.

The nervous system of a patient with neurological trauma is vulnerable to variations in oxygen, glucose, and other nutrients. Indirect calorimetry is the best method for identifying energy requirements (Shepherd, 2009). Pressure ulcers are very common in this population; see Section 2.

SCI patients with a BMI of >22 should be considered at risk for obesity (Laughton et al, 2009). After long-term immobility, SCI patients may require weight loss, using varied psychosocial, behavioral, and dietary interventions (Chen et al, 2006).

Treatment using fetal stem cells has profound implications for recovery in the SCI population. More research is forthcoming.

**TABLE 4-12 Expected Functional Level of Spinal Cord Disruption**

| Quadriplegia | Paraplegia |
|---|---|
| **C1-C3**<br>Vagus domination of heart, respiration, blood vessels, all organs below injury.<br>Movement in neck and above, loss of innervation to diaphragm, absence of independent respiratory function.<br>Ability to drive power wheelchair equipped with portable respirator by using chin control or sip and puff, lack of bowel and bladder control. | **T1–T6**<br>Sympathetic innervation to heart, vagus domination of the rest.<br>Full innervation of upper extremities, back, essential intrinsic muscles of hands; full strength and dexterity of grasp; decreased trunk stability; decreased respiratory reserve.<br>Full independence in self-care and in a wheelchair, ability to drive a car with hand controls (in most clients), ability to use full body brace for exercise but not for functional ambulation, lack of bowel and bladder control. |
| **C4**<br>Vagus domination of heart, respiration, and all vessels and organs below injury.<br>Sensation and movement above neck.<br>Ability to drive power chair using chin control or sip and puff, lack of bowel and bladder control. | **T6–T12**<br>Vagus domination only of leg vessels, gastrointestinal, and genitourinary organs.<br>Full, stable thoracic muscles and upper back; functional intercostals, resulting in increased respiratory reserve.<br>Full independent use of wheelchair; ability to stand erect with full body brace, ambulate on crutches with swing (though gait is difficult); inability to climb stairs; lack of bowel and bladder control. |
| **C5**<br>Vagus domination of heart, respiration, and all vessels and organs below injury.<br>Full neck, partial shoulder, back, biceps; gross elbow, inability to roll over or use hands; decreased respiratory reserve.<br>Ability to drive power chair with mobile hand supports, ability to use hand splints (in some clients), lack of bowel and bladder control. | **L1–L2**<br>Vagus domination of leg muscles.<br>Varying control of legs and pelvis, instability of lower back.<br>Good sitting balance, full use of wheelchair. |
| **C6**<br>Vagus domination of heart, respiration, and all vessels and organs below injury.<br>Shoulder and upper back abduction and rotation at shoulder, full biceps to elbow flexion, wrist extension, weak grasp of thumb, decreased respiratory reserve.<br>Ability to assist with transfer and perform some self-care, feed self with hand devices, push a wheelchair on smooth, flat surface; lack of bowel and bladder control. | **L3–L4**<br>Partial domination of leg vessels, gastrointestinal, and genitourinary organs.<br>Quadriceps and hip flexors, absence of hamstring function, flail ankles.<br>Completely independent ambulation with short leg braces and canes, inability to stand for long periods, bladder and bowel continence. |
| **C7**<br>Vagus domination of heart, respiration, and all vessels and organs below injury.<br>All triceps to elbow extension, finger extensors and flexors, good grasp with some decreased strength, decreased respiratory reserve.<br>Ability to transfer self to a wheelchair, roll over and sit up in bed, push self on most surfaces, perform most self-care; independent use of wheelchair; ability to drive a car with hand controls (in some clients); lack of bowel and bladder control. | |

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** A SCI is not usually genetic.

**Clinical/History**

- Height
- Weight
- BMI
- Indirect calorimetry (rest 30 minutes prior)
- Dietary/intake history
- I & O
- BP (tends to be elevated)
- Cervical x-rays
- Somatosensory-evoked potentials
- Myelogram
- MRI or CT scan
- DEXA
- Triceps skinfold
- Mid-arm circumference
- Pulmonary edema
- Pneumonia

**Lab Work**

- H & H (decreased)
- Serum Fe
- Alb
- Serial transthyretin levels
- $Na^+$, $K^+$
- Creat (eventually decreased)
- BUN
- Gluc
- $pCO_2$, $pO_2$
- RQ (if over 1.0, evaluate for overfeeding)
- PT or INR
- $Ca^{++}$, $Mg^{++}$
- Hypercalciuria
- Parathormone (may be low)
- 25-hydroxyvitamin $D_3$
- N balance
- Erythrocyte sedimentation rate (ESR)
- CRP (elevated)
- Serum $B_{12}$

### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Protein Intake**

***Assessment Data:*** SCI 6 months ago; poor intake of protein foods.

***Nutrition Diagnoses (PES):*** Inadequate protein related to poor intake of protein-rich foods as evidenced by dietary recall and early skin breakdown.

***Interventions:*** Change diet order to provide protein-enhanced foods that are nutrient dense. Educate patient about rationale for the protein to protect the skin integrity.

***Monitoring and Evaluation:*** Improved protein intake; better quality of life by increased ability to participate in physical and occupational therapies.

## INTERVENTION

### OBJECTIVES

#### *Immediate*

- Monitor for acid–base and electrolyte imbalances. Assess needs on admission and then daily thereafter.
- Maintain blood glucose at or below 140 mg/dL in critical phase (ADA, 2009). If on a ventilator, keep blood glucose levels between 80 and 110 mg/dL.
- Reduce the danger of aspiration by avoiding oral feedings in supine patients. Initiate tube feeding within 24–48 hours after admission to intensive care (Shepherd, 2009). Early EN is desirable because of the demands of the brain and nerves.
- If needed, use parenteral nutrition if there is intolerance of EN, such as high gastric residuals or aspiration.
- Ensure adequate fluid and calcium intake to prevent renal stones.
- Slow down weight loss and any progressive muscle wasting by maintaining protein and energy sufficiency.
- Increase opportunities for rehabilitation by monitoring weight changes; loss of 10–30% in the first month is common.
- Prevent UTIs, paralytic ileus, pneumonia, malnutrition, pressure ulcer, constipation, stress ulcer, and fecal impaction. Note that elevated CRP is associated with UTIs or pressure ulcers within a year (Gibson et al, 2008; Morse et al, 2008).

#### *Long Term*

- Mobilize, prevent complications, and regain as much independence as possible. Participate in muscle vibration stimulation and other therapies whenever possible.
- Monitor weight gain since excessive weight gain can lead to pressure ulcers and make patient transfers more difficult. Maintain an ideal body weight with a BMI of 18–22 (Laughton et al, 2009).
- Promote neuronal growth and survival, encourage the formation of synapses, enhance the production of myelin, and restore conduction capabilities and thus restore the compromised circuitry in the injured spinal cord.
- Prevent osteoporosis and risk of fractures.
- Manage long-term problems with bowel motility with fiber, fluid and laxatives (Shepherd, 2009).
- Prevent heart disease. While women with paraplegia tend to maintain healthier diets (i.e., lower calorie and fat intakes, more nutrient density, less overweight,) individuals with tetraplegia tend to be overweight or obese (Groah et al, 2009).

### FOOD AND NUTRITION

- Provide patient with intravenous solutions as soon as possible after injury. Check blood gas measurements and chemistries. Once peristalsis returns, patient may be tube fed. Elevate head of bed 45°, if possible, to prevent aspiration (ADA, 2009).
- Determine energy needs by indirect calorimetry. Patients with paraplegia need 28 kcal/kg/d and those with quadriplegia need 23 kcal/kg/d (Shepherd, 2009).
- Paraplegics initially need 1.5–1.7 g protein/kg. Progress to more normal intake, such as 0.8 g/kg, when nitrogen balance returns after several weeks. Patients with large pressure ulcers will need an increase in protein back to 1.5 g/kg.
- Ensure adequate CHO and fat intake, including at least 1–2% EFAs.
- Encourage adequate fluid (1 mL/kcal/d); use more with fever or pressure ulcers.
- Include adequate fiber (15 g/d). Be careful with gas-forming foods; monitor tolerance.
- Ensure adequate intake of thiamin, niacin, vitamins $B_6$, $B_{12}$, and C, and amino acids. Monitor iron stores and adjust diet as needed.
- Provide adequate vitamin $D_3$ and calcium intake.
- With hypertension, the DASH diet may be useful (increases in calcium, potassium, and magnesium are beneficial).
- Increase intake of antioxidant-rich foods and omega-3 fatty acids (DHA) for neuroprotection (King et al, 2006).
- Tube feeding should be used over parenteral nutrition where possible (ADA, 2009). An immune-enhancing formula may be recommended but is not always essential. Do not recommend use of blue dye to detect aspiration (ADA, 2009).

### Common Drugs Used and Potential Side Effects

- Corticosteroids such as prednisone are used to prevent swelling. Long-term use can cause hyperglycemia and nitrogen, calcium, and potassium losses. Sodium retention occurs.
- Analgesics for pain relief (e.g., aspirin/salicylates) can prolong bleeding time. GI bleeding may eventually result.
- Laxatives may be used; encourage fiber and fluid instead.
- For a bowel regimen, use of erythromycin, metoclopramide, a stool softener and a stimulant laxative may be prescribed (Shepherd, 2009). There are no contraindications to the use of promotility agents (ADA, 2009).
- Anabolic steroids (oxandrolone) help alleviate anorexia and may be helpful in managing pressure ulcers.
- To protect against fractures, bisphosphonates may help prevent acute bone loss; use IV rather than oral route to reduce reflux.

### Herbs, Botanicals, and Supplements

- Studies have found usefulness in pretreatment of patients with creatine for neuroprotection before spinal surgery.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help promote a structured feeding routine. Feed slowly (over 30–45 minutes) using small bites of food.
- The majority of people with SCI would benefit from nutritional counseling to prevent emerging secondary conditions (Groah et al, 2009; Tomey et al, 2005).
- Provide weight control measures for successful rehabilitation (Chen et al, 2006).
- Teach patient about good sources of iron and other minerals, vitamins, and protein.
- Discuss long-term risks of heart disease.
- Encourage participation in weight-bearing exercise to reduce calcium loss and risk of fracture or osteoporosis.

#### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

#### *For More Information*

- American Spinal Injury Association (ASIA)
  http://www.asia-spinalinjury.org/
- Children and Spinal Cord Injury
  http://www.childrenshospital.org/az/Site1150/mainpageS1150P0.html
- Christopher and Dana Reeve Paralysis Foundation
  http://www.christopherreeve.org/
- Cure Paralysis Now
  http://www.cureparalysisnow.org/
- Dermatome Chart
  http://www.asia-spinalinjury.org/publications/2006_Classif_worksheet.pdf
- Foundation for Spinal Cord Injury Prevention, Care, & Cure
  http://www.fscip.org/
- International Spinal Cord Society
  http://www.iscos.org.uk/
- Model Spinal Cord Injury System Dissemination Center
  http://www.mscisdisseminationcenter.org/
- National Spinal Cord Injury Association
  http://www.spinalcord.org/
- NIH—Medline
  http://www.nlm.nih.gov/medlineplus/spinalcordinjuries.html
- NINDS Spinal Cord Injury
  http://www.ninds.nih.gov/disorders/sci/sci.htm
- Paralyzed Veterans of America
  http://www.pva.org
- Spinal Cord Centers
  http://www.sci-recovery.org/sci-centers.htm
- Spinal Cord Injury Information Network
  http://www.spinalcord.uab.edu/
- Spinal Cord Recovery
  http://www.sci-recovery.org/

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Groah SL, et al. Nutrient intake and body habitus after spinal cord injury: an analysis by sex and level of injury. *J Spinal Cord Med.* 32:25, 2009.

King VR, et al. Omega-3 fatty acids improve recovery, whereas omega-6 fatty acids worsen outcome, after spinal cord injury in the adult rat. *J Neurosci.* 26:4672, 2006.

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Shepherd E. Nutrition care for the spinal cord injured patient. *Support Line.* 30(6):25, 2009.

Tomey KM, et al. Dietary intake and nutritional status of urban community-dwelling men with paraplegia. *Arch Phys Med Rehabil.* 86:664, 2005.

# STROKE (CEREBROVASCULAR ACCIDENT)

## NUTRITIONAL ACUITY RANKING: LEVEL 3

### DEFINITIONS AND BACKGROUND

A cerebrovascular accident (CVA) (stroke) is caused by damage to a portion of the brain resulting from loss of blood supply due to a blood vessel spasm, clot, or rupture. Sporadic strokes can occur, but most have a genetic, polygenic component. Eighty percent of strokes are ischemic. Transient ischemic attacks (TIAs) are brief episodes of blood loss to the brain from a clot or an embolism; 10% of victims will have a major CVA within a year. Stroke patients need to be seen medically within 60 minutes to begin appropriate treatment. Some people recover completely; others may be seriously disabled or die. The urgent presenting symptoms are listed in Table 4-13.

Hypertension, smoking, diabetes mellitus, atrial fibrillation, and oral contraceptive use are key risk factors for strokes. Unconsciousness, paralysis, and other problems may occur depending on the site and extent of the brain damage. Left CVA affects sight and hearing most commonly, including the ability to see where foods are placed on a plate or tray. Patients with a right-hemisphere, bilateral, or brainstem CVA have significant problems with feeding and swallowing of food; speech problems also occur.

**TABLE 4-13 Most Common Stroke Symptoms**

- Sudden numbness or weakness of face, arm, or leg, especially on one side of the body
- Sudden confusion, trouble speaking or understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking, dizziness, loss of balance or coordination
- Sudden severe headache with no known cause

Neurogenic deficits may include motor deficits with muscle weakness of the tongue and lips; nerve damage with resulting lack of coordination; apraxia; sensory deficits with an inability to feel food in the mouth. Cognitive deficits include difficulty sustaining attention, poor short-term memory, visual field problems, impulsiveness, aphasia, and judgment problems such as not knowing how much food to take or what to do with the food once it reaches the mouth. Strokes cause 10% of all fatalities in the United States.

A dietary pattern with high intakes of red and processed meats, refined grains, sweets and desserts may increase stroke risk, whereas a diet higher in fruits and vegetables, fish, and whole grains may protect against stroke. Intake of cruciferous and green leafy vegetables, citrus fruits, and carotenoids seem to be protective. Among elderly individuals, consumption of tuna or other broiled or baked fish is associated with lower risk of ischemic stroke, while intake of fried fish is associated with higher risk (Mozaffarian et al, 2005). Both fish and omega-3 fatty acids seem to prevent thrombotic strokes.

Individualized dietary advice to those with coronary heart disease can reduce stroke mortality and morbidity, yet this is often overlooked by physicians (Spence, 2006). Risk reduction from controlled trials with supplemental vitamins C or E has not been consistent. Vitamin E influences the activity of enzymes PKC, PP2 A, COX-2, 5-lipooxygenase, nitric oxide synthase, NADPH-oxidase, superoxide dismutase, and phospholipase A2 and modulates gene expression. Vitamin C levels tend to be lower among stroke patients, probably due to the relationship to inflammation and oxidative stress.

Lowering elevated serum tHcy is also important. For example, homocystinuria is a metabolic disorder that is a known life-threatening risk factor for ischemic stroke. Folate deficiency and hyperhomocysteinemia increase oxidative DNA damage and ischemic lesion size in stroke patients (Endres et al, 2005). Intake of folate, vitamins $B_6$ and $B_{12}$ should be maintained in high-risk groups.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Pro-inflammatory gene polymorphisms are related to both coronary heart disease and stroke. Alterations in the 9p21 chromosome relate to atherosclerotic stroke (Gschwendtner et al, 2009), whereas MTHFR C677 T, beta-Fg—455 A/G, beta-Fg—48 T/C, PAI-1 4G/5G, and ApoE epsilon2–4 are associated with ischemic stroke (Xu et al, 2008).

**Clinical/History**

Height
Weight
BMI
Waist to hip ratio
Dietary/intake history
Positron emission tomography (PET) scan
Sleep apnea
Chewing ability
Hand to mouth coordination
Gag reflex absent?
BP
Temperature
Visual field scan
EEG
Carotid ultrasound
CT scan or MRI
I & O

**Lab Work**

CRP
PT
INR: 2.0–4.0 desirable
$Na^+$, $K^+$
$Ca^{++}$, $Mg^{++}$
Chol (total, HDL, LDL)
Trig
tHcy
Serum folate
Ferritin
H & H
Gluc (often increased)
Creatine phosphokinase (CPK)
Serum uric acid

### SAMPLE NUTRITION CARE PROCESS STEPS

**Imbalance of Nutrients**

***Assessment Data:*** Food records indicating high sodium intake and limited use of fruits and vegetables for calcium, magnesium, and potassium.

***Nutrition Diagnoses (PES):*** Imbalance of nutrients related to dietary intake of mostly processed, high sodium foods and few fruits, vegetables, or dairy as evidenced by diet history.

***Interventions:*** Education about use of the DASH diet (enhanced fruits, vegetables, low fat dairy) and whole grains.

***Monitoring and Evaluation:*** Improved BP; no more TIAs or strokes; improved balance between sodium and other minerals (calcium, potassium, magnesium).

**Difficulty Swallowing**

***Assessment Data:*** Results of swallow study and x-rays, food diary, and problems noted with specific types of liquids/foods; swallow studies and conferences with speech therapist; patient is also aphasic.

***Nutrition Diagnoses (PES):*** Swallowing difficulty related to consumption of general diet after CVA as evidenced by coughing after intake of thin liquids.

***Intervention:*** Alter diet to thicken liquids with all meals, snacks, medication passes, special events, dining out. Provide recipes for use of thickened liquids in daily meals.

***Monitoring and Evaluation:*** Reduced incidence of coughing after drinking beverages; no hospital admissions for pneumonia. Able to communicate in spite of aphasia using head nod and eyes to communicate preferences and choices.

## INTERVENTION

### OBJECTIVES

- **Immediate treatment:** Maintain fluid-electrolyte balance for lifesaving measures.
- **Ongoing treatment:** Improve residual effects such as dysphagia, hemiplegia, and aphasia. Correct side effects such as, constipation, UTIs, pneumonia, renal calculi, and pressure ulcers.
- If the patient is excessively overweight, weight reduction is necessary to lower elevated BP or lipids and to lessen the workload of the cardiovascular system.
- Chewing should be minimized with dysphagia; prevent choking. Avoid use of straws if there is dysphagia.
- Lower elevated serum lipids; try to improve HDL cholesterol levels.
- Promote self-help, self-esteem, and independence.
- Prevent additional strokes, which are common. See Table 4-14. Since inflammation may be caused by a response to oxidized low-density lipoproteins, chronic infection, or other factors, monitor CRP.

**TABLE 4-14 Strategies Used to Prevent Strokes**

| | |
|---|---|
| GOAL: Lower or Less | Blood pressure at 120/80 or below. |
| | Smoking; quitting is best. |
| | Limit sodium from salt shaker, processed meats, pickles, and olives. |
| | Maintain serum cholesterol levels of 200 or lower; LDL of 100 or lower; triglycerides of 150 or less |
| | Energy intake: avoid obesity. |
| GOAL: Higher or More | Exercise moderately each day for at least 30–60 minutes. Brisk walking (about 3 mph) is most protective; but any walking is good. |
| | High-dose statin, low to standard doses of antihypertensive therapy, aspirin, cardiac rehabilitation (Robinson and Maheshwari, 2005). |
| | Eat a balanced diet including fruits, vegetables, whole grains, low fat dairy products for more potassium, vitamin C, calcium, magnesium. |
| | Include omega-3 fatty acids in foods regularly (fatty fish, flaxseed, and walnuts). May use omega-3 fish oil. |
| | Include natural sources of fatty acids and vitamin E, such as mayonnaise, creamy salad dressings, margarine, and nuts. |
| | Vitamin K may curtail vascular calcification; 500 μg may be needed. |
| | Take a multivitamin–mineral supplement daily, especially for folic acid, vitamins $B_6$, and $B_{12}$, to reduce homocysteine levels and to help lower blood pressure. |
| | Achieve and maintain body weight within BMI range for height. |
| | Maintain or elevate HDL to >60. |
| | Drink alcoholic beverages in moderation only (one drink for women, two for men per day). Alcohol boosts HDL and may reduce clot formation. |

## FOOD AND NUTRITION

- **Initial treatment:** Nothing by mouth (NPO) with intravenous fluids for 24–48 hours. Avoid overhydration. Tube feeding may be needed, especially gastrostomy or jejunostomy. If the patient is comatose, tube feeding definitely is required, and the head of the bed should be elevated at least 30°–45° during feeding to prevent aspiration.
- **Recovery:** Treatment should progress from NPO to liquids. Sip feeding may improve nutrient intake and nutritional status of stroke patients who do not have swallowing difficulties with liquids.
- Provide adequate energy intake (patient's weight should be checked frequently). Monitor the patient's activity levels. From 25–45 kcal/kg and 1.2–1.5 g protein/kg may be needed, depending on weight status and loss of lean body mass.
- Texture modification to compensate for dysphagia should be made to reduce risk of choking and/or aspiration. Thick pureed liquids or a mechanical soft diet may be needed. Liquids can be thickened with gels.
- Always start with small amounts of food. Use easy-to-chew foods and spoon rather than fork foods. Progress slowly.
- With dysphagia, avoid foods that cause choking or that are hard to manage (e.g., tart juices and foods, dry or crisp foods, fibrous meats, unboned fish, chewy or stringy meats, sticky peanut butter or bananas, thinly pureed foods that are easily aspirated, mixed foods with varying consistency, excessively sweet drinks or tart fruits that aggravate drooling, raw vegetables). Mashed potatoes or soft breads for some patients may be hard to swallow.
- With decreased salivation, moisten foods with small amounts of liquid. Use thickener products to make semisolids out of soup, beverages, juices, and shakes. Test swallowing periodically. When ready, use of a syringe or training cup is beneficial.
- The amount of saturated and trans fatty acids in the planned diet should be <10% of total calories, and the dietary cholesterol intake should be <300 mg/d. A useful recommendation is to reduce the quantity of fat by 20–25%, reduce animal fats, and decrease the amount of salt added to foods in cooking and at the table.
- Replace saturated fat with monounsaturated sources; use more olive, soybean or canola oils and nuts such as walnuts, almonds, macadamias, pecans, and pistachios. Walnuts contain alpha linolenic acid; almonds are a good source of vitamin E. Nuts also contain flavonoids, phenols, sterols, saponins, elegiac acid, folic acid, magnesium, copper, potassium, and fiber. The Mediterranean diet is a useful diet to follow; in this diet, unsaturated fats replace most of the saturated fat, and fruits and vegetables are highlighted (Spence, 2006).
- Use plant sterols and stanols, as from margarines and related products.
- Increase omega-3 fatty acids from fish.
- Use skim milk products whenever possible. Milk fat is negatively correlated with certain cardiovascular disease risk factors.
- Increase potassium to reduce risk of additional strokes. Avoid use of potassium-sparing diuretics or with end-stage

renal disease. Fruits and vegetables are the best sources (oranges, bananas, prunes, baked potatoes); milk is another good source. Magnesium, vitamin E, folic acid, and vitamins $B_{12}$ and $B_6$ should be included in sufficient quantities to meet at least minimum daily requirements. Use the DASH diet plan (see Section 6).
- Fluid should be given in sufficient quantity if tolerated; estimate needs at 30 mL/kg and increase to 35 mL/kg if dehydration occurs. Give oral beverages at the end of the meal to increase solid food intake in patients who have early satiety or fatigue with meals.
- The diet should provide adequate fiber from prune juice, bran, whole grain breads and cereals, oatmeal, bran, wheat germ, popcorn, brown rice.
- Use caution with supplemental vitamin C; excesses may act as a pro-oxidant.
- Flavonoids such as grape juice, green tea, and red wine are useful if the patient can tolerate thin liquids.

## Common Drugs Used and Potential Side Effects

- Angiotensin-converting enzyme inhibitors/angiotensin receptor blockers may cause diarrhea or GI distress. Atacand, Teveten, Avapro, Cozaar, Benicar, Micardis, or Diovan should be used with a low sodium, low calorie diet. They may cause anemia or hyperkalemia. Be careful with salt substitutes; read contents carefully.
- Anticoagulants used to prevent thromboembolism, such as warfarin (Coumadin), require a controlled amount of vitamin K. Monitor tube feeding products and supplements. Many patients who are taking warfarin can safely monitor their INR levels at home and adjust their medications accordingly. Monitor supplements containing vitamins A, C, and E with these drugs because of potential side effects. Avoid taking with dong quai, fenugreek, feverfew, excessive garlic, ginger, gingko, and ginseng.
- Aspirin is often used to prevent future strokes as a blood thinner (generally 1 tablet per day). Monitor for GI bleeding or occult blood loss. Aspirin is safer than warfarin and just as effective for treating blocked arteries in the brain (Koroshetz, 2005).
- Grapefruit juice decreases drug metabolism in the gut (via P450-CYP3A4 inhibition) and can affect medications up to 24 hours later. Avoid taking with alprazolam, buspirone, cisapride, cyclosporine, statins, tacrolimus, and many others.
- Products containing phenylpropanolamine (PPA) are a risk for stroke. PPA has been pulled from the shelves but may still be in some cough medicines in the home.
- Statins (Lipitor, Lescol, Mevacor, Pravachol, Crestor, and Zocor) are commonly prescribed. Nausea, abdominal pain, and other GI effects are common. Do not take with grapefruit juice or St. John's wort. Monitor liver enzymes.
- Stool softeners may be used. Tube feeding containing a mix of soluble and insoluble fiber can be used. If a low-residue formula is used, a fiber supplement such as Benefiber can be mixed with water and administered via Y-port.
- Thiazide diuretics, such as Lasix, may be used and can deplete potassium.

## Herbs, Botanicals, and Supplements

- The patient should not take herbals and botanicals without discussing with the physician.
- Coenzyme Q10 should not be used with gemfibrozil, TCAs, or warfarin. Coenzyme Q10 may act similarly to vitamin K.
- Niacin (nicotinic acid) should not be taken with statins, antidiabetic medications, and carbamazepine because of potentially serious risks of myopathy and altered glucose control.
- Large doses of vitamin E should not be taken with warfarin because of possible increased bleeding. Avoid doses greater than 400 IU/d.
- Garlic, willow, pigweed, gingko, or evening primrose have been recommended; no clinical trials have proven efficacy.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help the patient simplify meal preparation. Arrange food and utensils within reach. Discuss the use of appropriate assistive devices.
- Explain which sources of adequate nutrition do not aggravate the patient's condition. Discuss fat, cholesterol; sodium, potassium, calcium, magnesium, specific vitamins, and other nutrients in the DASH diet. Correlate with drug therapy.
- Help the patient make mealtime safe and pleasant. Encourage small bites of food and slow, adequate chewing.
- Discuss ways to prevent future strokes; linolenic acid from walnut, canola, and soybean oils may be protective. Increased fruit and vegetable intake is also protective.
- Physical therapy is very important in early stages after a stroke, especially to regain use of limbs such as hands and arms.
- Manage depression, which is common after a stroke. Treatments may include patient and family counseling and education, reestablishment of sleep pattern, improving diet, regular physical activity, or medication.
- Future prevention strategies should be taught to stroke patients and their families. Modifiable risk factors include hypertension, exposure to cigarette smoke, diabetes, atrial fibrillation and certain other cardiac conditions, dyslipidemia, carotid artery stenosis, sickle cell disease, postmenopausal hormone therapy, poor diet, physical inactivity, and obesity and body fat distribution (Goldstein et al, 2006). Potentially modifiable risk factors include the metabolic syndrome, alcohol use, oral contraceptive use, migraine headache, hyperhomocysteinemia, inflammation (Goldstein et al, 2006).
- The American Heart Association, American Cancer Society, and American Diabetes Association agree that lifestyle changes are essential for prevention of stroke and associated disability.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. If tube feeding is needed, discuss proper sanitation of work counters during preparation.

- Hand washing is important, especially for caregivers if the patient is unable to feed himself or herself.

### *For More Information*

- American Academy of Physical Medicine and Rehabilitation
  http://www.aapmr.org/
- American Stroke Association
  http://www.strokeassociation.org/
- National Aphasia Association
  http://www.aphasia.org
- National Institute of Neurological Disorders and Stroke
  http://www.ninds.nih.gov/
- National Rehabilitation Awareness Foundation
  http://www.nraf-rehabnet.org/
- National Rehabilitation Hospital
  http://www.nrhrehab.org/
- National Stroke Association
  http://www.stroke.org/
- North Carolina Stroke Association
  http://www.ncstroke.org/
- UCLA Stroke Center
  http://www.stroke.ucla.edu/

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# TARDIVE DYSKINESIA

## NUTRITIONAL ACUITY RANKING: LEVEL 2

### DEFINITIONS AND BACKGROUND

Tardive dyskinesia (TD) is a movement disorder characterized by involuntary oro-facial, limb, and truncal movements (Tsai et al, 2009). It is usually caused by the use of drugs that block dopamine receptors (dopamine receptor antagonists [DRAs]). When used in the classic sense, TD is produced by the long-term use of drugs to treat SCZ that act by blocking dopamine receptors. TD occurs in 20–40% of all patients receiving long-term antipsychotic drugs. Patients are often elderly and chronically institutionalized.

Phenylalanine sensitivity has been speculated as the cause of TD. Amine-depleting agents such as reserpine (Serpalan, Serpasil) and tetrabenazine (Nitoman) deplete dopamine, norepinephrine, and serotonin. Tarvil, a medical food with high branched-chain amino acids, targets excess phenylalanine and has few side effects.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** There have been a sizeable number of candidate gene studies. A total of 128 candidate genes were studied in 710 subjects—2580 SNPs in 118 candidate genes selected from the literature (e.g., dopamine, serotonin, glutamate, and GABA pathways) and composite genotypes for 10 drug-metabolizing enzymes. No single marker or haplotype association reached statistical significance after adjustment for multiple comparisons (Tsai et al, 2009).

**Clinical/History**

- Height
- Weight
- BMI
- Dietary/intake history
- BP
- Chorea, athetosis, dystonia
- Tics or facial grimacing
- Abnormal Involuntary Movement Scale
- Tremors

**Lab Work**

- CRP
- BUN, Creat
- H & H
- Serum Fe
- Serum tHcy
- Serum folate
- Serum prolactin (often increased)
- Acetylcholine levels
- Gluc
- Alb, transthyretin
- Ceruloplasmin
- Serum Cu
- Serum phenylalanine

## INTERVENTION

### OBJECTIVES

- Prevent or correct malnutrition, weight loss, and other problems.
- Identify and assist with problems, such as puckering of the lips, difficulty sucking and eating.
- Restore capacity for eating orally as far as possible.
- Alter textures as necessary (eating problems are rare or occur late in the condition).

## SAMPLE NUTRITION CARE PROCESS STEPS

**Self-Feeding Difficulty**

***Assessment Data:*** Food intake sporadic because of difficulty feeding self; weight loss.

***Nutrition Diagnoses (PES):*** Self-feeding difficulty related to dyskinesia as evidenced by food intake <75% desirable amount and weight loss of 10 lb in past 3 months and BMI 18.

***Interventions:*** Enhance nutrient intake and offer easy to handle finger foods at frequent intervals. Educate about nutrient density. Counsel about ways to increase energy intake to improve weight.

***Monitoring and Evaluation:*** Improved weight status. Increased ability to eat sufficient amounts of kilocals and nutrient-dense choices using finger foods and frequent meals or snacks.

## FOOD AND NUTRITION

- Offer the usual diet with soft textures to reduce chewing as needed.
- Decrease energy intake if obese; increase intake if underweight.
- Carbohydrate craving is common. Watch overall intake of sweets or offer nutrient-dense varieties to reduce hyperglycemia.
- Increase dietary choline from foods such as eggs, soybeans, peanuts, and liver.
- Moisten foods with gravy, sauces, and liquids if dry mouth is a problem.
- Alter fiber intake if needed to prevent or correct constipation.
- Ensure adequate intake of antioxidants and omega-3 fatty acids (colorful fruits and vegetables, nuts, fish and seafood).

### Common Drugs Used and Potential Side Effects

- Psychiatric conditions are often treated with phenothiazines, butyrophenones, dibenzodiazepines, indolones, diphenylbutylpiperidines, and thioxanthenes. These are more likely to cause TD than the newer antipsychotic agents.
  - Incidence of TD with the use of newer atypical antipsychotic agents such as clozapine (Clozaril), olanzapine (Zyprexa), risperidone (Risperdal), and quetiapine (Seroquel) is minimal (Casey, 2006). Risperidone (Risperdal) appears to bring out the symptoms of TD more frequently, as compared to the other newer atypical antipsychotic agents.
  - Drugs other than those used to treat psychiatric illnesses can also block the dopamine receptors. These include anticholinergics and SSRIs, which are used to treat depression. Whether MAOIs and tricyclics cause TD is not known.
  - Reduction in the use of the drugs that caused TD is desirable. Changing to a different medication, such as an atypical antipsychotic, is recommended. Patients on antipsychotics should be checked regularly.
- TD has also been known to develop in patients who have been treated for digestive and GI disorders with medications such as metoclopramide (Reglan).
- Tetrabenzine, a medication that reduces levels of dopamine, has been of some use in treating TD symptoms.
- "Anti-Parkinsonian" drugs such as Aricept and Miraplex appear to offer some benefit.

### Herbs, Botanicals, and Supplements

- No studies have been conducted for efficacy of herbs or botanicals in TD.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Diet instructions should be offered directly to the patient unless this is not possible.
- Discuss major issues related to nutrition, self-feeding practices, moistening of foods, use of adaptive equipment as needed.
- Discuss sources of foods that contain branched-chain amino acids and how to obtain medical foods that may be used.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.

### *For More Information*

- NIMH
  http://www.nimh.nih.gov/index.shtml
- NINDS—Tardive Dyskinesia
  http://www.ninds.nih.gov/disorders/tardive/tardive.htm
- Tardive Dyskinesia
  http://www.tardivedyskinesia.com/
- We Move—Tardive Dyskinesia
  http://www.wemove.org/td/

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# TRIGEMINAL NEURALGIA

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2

### DEFINITIONS AND BACKGROUND

Trigeminal neuralgia (TN) or tic douloureux manifests as a disorder of the fifth cranial nerve and is characterized by paroxysms of excruciating pain of a burning nature. The painful periods alternate with pain-free periods. The frequency of the paroxysms ranges from a few to hundreds of attacks a day; remission can last for months to years, but tend to shorten over time (Zakrewska and Linskey, 2009).

The disorder is rare before 40 years of age and is more common in elderly women. The right side of the face is affected more often; the pain can be incapacitating. Dentists often play a role in identifying this condition (Bagheri et al, 2004). Loss of taste after surgery can occur. Recent use of radiosurgery or a gamma knife procedure has shown promise.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** Whether TN is related to celiac disease has yet to be determined.

**Clinical/History**

Height
Weight
BMI
Dietary/intake history
PET scan
Chewing ability
BP
Temperature
EEG
Carotid ultrasound
CT scan or MRI
I & O

**Lab Work**

CRP
$Na^+$, $K^+$
Folate
$Ca^{++}$, $Mg^{++}$
Chol (total, HDL, LDL)
Trig
Ferritin
H & H
Gluc

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Difficulty Chewing**

***Assessment Data:*** Problems with weight loss, pain with eating meals, difficulty chewing solids.

***Nutrition Diagnoses (PES):*** Inadquate oral food and beverage intake related to difficulty chewing as evidenced by diagnosis of TN with excruciating pain, especially with solids.

***Interventions:*** Education of patient about pureed foods and liquefying meal items to reduce chewing; nutrient-dese food and beverage choices.

***Monitoring and Evaluation:*** Regain of lost weight, no further complaints of pain with chewing, improved nutrient intake.

### INTERVENTION

#### OBJECTIVES

- Control pain with medications, especially before meals.
- Provide appropriate counseling and assistance with consistency of meals (foods and beverages).
- Individualize for preferences and tolerances.
- Maintain body weight within a desirable range.
- Manage celiac disease if present.

#### FOOD AND NUTRITION

- Use a normal diet as tolerated, perhaps altering to soft or pureed foods as needed. Omit gluten if celiac disease is diagnosed.
- Small, frequent feedings may be better tolerated than large meals.
- Liquids may be preferred if given by straw. Individualize.
- Avoid extremes in temperature.
- Use nutrient-dense foods if weight loss occurs.

#### Common Drugs Used and Potential Side Effects

- Anticonvulsants such as topiramate, phenytoin (Dilantin) or carbamazepine (Tegretol) are used. Diarrhea, nausea, and vomiting are common (He et al, 2006). Ensure adequate intake of folate.
- Nonsteroidal anti-inflammatory medications or narcotics may be used to reduce pain.
- Opiate-based analgesics offer the best relief. In some cases, Botox has been used.
- Baclofen may help patients eat when jaw movement tends to aggravate the symptoms.

#### Herbs, Botanicals, and Supplements

- No studies have been conducted for efficacy of herbs or botanicals in TN.
- With anticonvulsants such as phenytoin (Dilantin), avoid use with evening primrose oil, gingko biloba, and kava.

#### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The importance of oral and dental hygiene should be stressed, even with pain. Use pain medications as directed.
- The patient should be encouraged to avoid eating when tense or nervous.
- Relaxation therapy, yoga, tai chi, and biofedback may be beneficial.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.

### *For More Information*

- Mayo Clinic—Trigeminal Neuralgia
  http://www.mayoclinic.com/health/trigeminal-neuralgia/DS00446
- NIH—Trigeminal Neuralgia
  http://www.ninds.nih.gov/disorders/trigeminal_neuralgia/detail_trigeminal_neuralgia.htm
- Neuropathic Facial Pain
  http://www.endthepain.org/

## TRIGEMINAL NEURALGIA—CITED REFERENCES

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PSYCHIATRIC DISORDERS—EATING DISORDERS

# ANOREXIA NERVOSA

## NUTRITIONAL ACUITY RANKING: LEVEL 3

### DEFINITIONS AND BACKGROUND

Anorexia nervosa (AN) is an eating disorder (ED) in which the patient severely rejects food, causing extreme weight loss, low basal metabolic rate, and exhaustion. About 6–15% of the population is affected. AN is more common in girls, especially just after, peaking at 12–13 and 19–20 years of age. But AN can occur at any age.

Signs include relentless pursuit of thinness, misperception of body image, and restrained eating, binge eating, or purging (see Bulimia and Binge Eating Disorder entries). Generally, cases are separated into "restricting" or "binge-purging" types: anorectic restrictor (AN-R) and anorectic bulimics (AN-B). Fear of fatness and a codependent focus outside of one's self are common in AN. The intense fear of becoming fat (not diminishing as weight loss progresses) has no known physical cause. Patients with EDs may have dermatologic manifestations secondary to starvation; recognition of these signs can lead to early diagnosis and treatment.

Weight is 85% or less of former weight; there is usually amenorrhea. Length of amenorrhea, estrogen exposure (age minus age at menarche minus years of amenorrhea), and body weight have independent effects on bone densities; therefore, osteopenia is common. Long-term sequelae may include Cushing's disease and osteoporosis. Without treatment, death may occur, usually from cardiac arrhythmias.

Problems include perfectionism, denial, impulse control, manipulative behavior, trust issues, power and misinformation within the family, low tolerance for change and new situations, fear of growing up and assuming adult responsibilities. Individuals with AN are overly dependent on parents or family, obsessive-compulsive, meticulous, introverted, emotionally reserved, socially insecure, overly rigid in thinking, self-denying, and overly compliant. Individuals with AN have high constraint, constriction of affect and emotional expressiveness, ahendonia and asceticism; restricting food intake becomes powerfully reinforcing because it provides a temporary respite from dysphoria (Kaye, 2009).

Because patients deny the severity of their illness, they delay seeking psychiatric treatment. Teens with ED often use subterfuge to give the impression that they are cooperating with treatment plans, when they in fact are not. These behaviors prolong treatment and can lead to malnutrition. Group parenting education may be quite helpful (Zucker et al, 2006).

Some careers promote a thin body for success (e.g., fashion, air travel, entertainment, and athletics). Many female athletes struggle with an ED. The main concern is inadequate energy intake (Gabel, 2006). Studies suggest that individuals with AN have lower than optimal levels of polyunsaturated fatty acids (including ALA and GLA). Two or more consecutive spontaneous menses implies resumption of menses; this depends on body weight but not on body fat. Weight regain in subjects with AN is associated with an increase in serum leptin concentrations (Mauler et al, 2009). Insulin-like growth factor I (IGF-I) is a biochemical marker of malnutrition and a sensitive index of nutritional repletion in patients with EDs. Fortunately, the majority of patients with EDs make a full recovery.

Dietitians must be able to identify and refer patients with EDs. Death by suicide occurs in a disproportionate percentage of individuals with AN (Zucker et al, 2007). Yet, evidence for successful treatment is weak (Bulik et al, 2007). Tube feeding, when accepted, helps to increase weight and improve cognitive and physical functioning. Medical nutrition therapy for EDs is a specialization that requires training beyond entry level. The American Dietetic Association has recommended eight medical nutrition therapy visits by a trained professional for persons who have EDs.

### ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** A genetic component may play a role in determining a person's susceptibility to anorexia. Researchers are currently attempting to identify the particular gene or genes that might affect a person's

tendency to develop this disorder. The agouti gene may play a role in AN.

### Clinical/History

Height
Present weight
Usual weight
Recent weight
Percentage of weight changes
BMI (often <17.5)
Weight goal/timing
Dietary/intake history
Bulimia or vomiting?
Tooth erosion or decay
Laxative or diuretic abuse?
BP; hypotension?
Amenorrhea
Blotchy or yellow skin
Lanugo hair
Muscle wasting
Edema
Blood in stool?
MRI or CT scan for ventricular enlargement from malnutrition
DEXA (after 6 months underweight)
EKG Arrthymias?
Parental Authority Questionnaire (PAQ)
Eating Disorders Inventory-2 (EDI-2)
Eating Attitudes Test (EAT-26)
Perfectionism, compulsiveness

### Lab Work

Luteinizing hormone (LH) response to gonadotropin-releasing hormone (GnRH)
Follicle-stimulating hormone (FSH)
IGF-I
Serum estradiol (low)
Serum cortisol (high)
Urinary cortisol (high)
Sex hormone–binding globulin (SHBG)
Alb, transthyretin, N balance
Chol (low?)
Trig
Gluc
BUN (low?)
H & H
Serum Fe
Thyroid-stimulating hormone (TSH)
TLC; Leukopenia?
$Na^+$, $Cl^-$
$K^+$ (hypokalemia?)
Serum amylase (with vomiting)
Serum $Ca^{++}$, $Mg^{++}$
Serum phosphorus
Liver function tests
Leptin levels
*Ghrelin levels
* Plasma levels of ghrelin increase before meals and decrease strongly after meals.

## SAMPLE NUTRITION CARE PROCESS STEPS

**Harmful Beliefs About Food And Nutrition**

***Assessment Data:*** BMI charts and current versus desirable weight for height; body fat measurements.

***Nutrition Diagnosis (PES):*** Disordered eating pattern related to complaints of being "too fat" as evidenced by current weight of 82 lb and self-limited dietary intake of 450 kcals daily on nutrient analysis.

***Intervention:*** Provide meals appropriate for refeeding protocol; education and counseling about appropriate body size, weight, BMI; goal setting for self-management.

***Monitoring and Evaluation:*** Improved weight and height for age; intake records; discussion about perceptions of mealtimes and eating, body image.

## INTERVENTION

### OBJECTIVES

- Restore normal physiological function by correcting starvation and its associated changes, including electrolyte imbalance, bradycardia, and hypotension.
- Check weight or growth charts to determine difference and to set goals. Promote weight gain of 1–3 lb weekly (in-patient) and 0.5–1 lb weekly (outpatient) to reach a weight closer to a healthy BMI.
- Promote adequate psychotherapy and use of medications to protect the heart, fluid, and electrolytes, which are the most important.
- Obtain diet history to assess bulimia, vomiting, and use of diuretics or laxatives.
- Do not force feedings; rejection of food is part of the illness. Promote normal eating behavior instead.
- Gradually increase intake to a normal or high-energy intake to lessen likelihood of edema and other consequences of malnutrition.
- For young women, promote normal menstrual cycles. Estrogen seems to be correlated with cognitive function (Chui et al, 2008).
- Reduce preoccupation with weight and food. Erroneous perceptions of "normal" should be alleviated. Promote adequate self-esteem.
- Refer to appropriate care for psychiatric maladies and comorbid conditions, especially insulin-dependent diabetes mellitus.
- Coordinate nutrition education and counseling with the overall team plan. Table 4-14 shows how average women compare with "fashion women"; counseling will need to be adjusted according to the individual's self-perception.

### FOOD AND NUTRITION

- Serve attractive, palatable meals in small amounts, observing food preferences. Small, frequent meals are useful. Encourage variety.
- Limit bulky foods during the early stages of treatment; GI intolerance may persist for a long time. Assure the patient that constipation will be alleviated.
- Diet should be called a "low-calorie diet for AN" to convince the patient of the counselors' good intentions.
- According to the standards set by the APA (2005): start at 30–40 kcal/kg (about 1000–1600 kcals/d) and increase as possible. Promote weekly weight gain by gradually attaining intake of up to 70–100 kcal/kg for some patients. Weight maintenance may need to be 40–60 kcal/kg.
- Protein refeeding takes a long time. Repletion may not be complete until weight has returned to normal. Monitor for improved biochemical results (BUN, albumin).
- Monitor serum cholesterol levels; low levels have been correlated with suicidality (Favaro et al, 2004).
- While not a preferred method, use tube feeding if necessary (i.e., only if the patient weighs 40% of lower end of BMI range for normal). Nocturnal tube feeding may be especially helpful.

- Help the patient resume normal eating habits. Have the patient measure and record food intake at first; then, gradually lessen the emphasis on food.
- A "no added salt" diet may reduce fluid retention.
- It may be useful to avoid caffeine because of stimulant/diuretic effect.
- A vitamin–mineral supplement may be needed for zinc and other nutrients.

## Common Drugs Used and Potential Side Effects

- Pharmacotherapy is not always successful in AN. Olanzapine may have some benefit with reductions in depression and anxiety. Dry mouth and constipation are the most common side effects.
- Antidepressants may be prescribed; nutritional side effects should be monitored carefully. SSRIs are considered to be more effective. However, fluoxetine has failed to demonstrate protection against relapse in AN (Walsh et al, 2006).
- Antiepileptic drugs (AEDs) may be used. Carbamazepine and valproate may be effective in treating patients with AN when used to treat an associated mood or seizure disorder (McElroy et al, 2009).

## Herbs, Botanicals, and Supplements

- No specific herbs and botanical products have been used for AN in clinical trials.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Most treatments are based on consensus rather than evidence (Gowers, 2008). At this time, the evidence base is strongest for family therapy for AN (Keel and Haedt, 2008).
- The RD is uniquely qualified to provide medical nutrition therapy for the normalization of eating patterns and nutritional status (American Dietetic Association, 2006).
- See Table 4-15.

**TABLE 4-15 Average Woman Versus "Fashion Woman"**

| | Average Woman | Barbie Doll | Store Mannequin |
|---|---|---|---|
| **Height** | 5′4″ | 6′0″ | 6′0″ |
| **Weight** | 145 lbs | 101 lbs | Not available |
| **Dress size** | 11–14 | 4 | 6 |
| **Bust** | 36–37″ | 39″ | 34″ |
| **Waist** | 29–31″ | 19″ | 23″ |
| **Hips** | 40–42″ | 33″ | 34″ |

Anorexia Nervosa and Related Eating Disorders. "Statistics: How many people have eating disorders?"http://www.anred.com/stats.html *ANRED*. 2005, accessed July 1, 2008.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for sanitizing work area before and after preparing tube feedings to prevent contamination. Formula companies have good information on safe handling of formula in the home and institution.

### *For More Information*

- Academy for Eating Disorders
  http://www.aedweb.org
- Anorexia Nervosa and Related Eating Disorders (ANRED)
  http://www.anred.com/
- Eating Disorders Anonymous
  http://www.eatingdisordersanonymous.org/
- Eating Disorder Recovery
  http://www.addictions.net/
- International Association of Eating Disorders Professionals (IAEDP)
  http://www.iaedp.com/
- National Association of Anorexia Nervosa and Associated Disorders (ANAD)
  http://www.anad.org/
- Practice Guideline for Eating Disorders
  http://www.psych.org/psych_pract/treatg/pg/prac_guide.cfm

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# BINGE EATING DISORDER

## NUTRITIONAL ACUITY RANKING: LEVEL 3–4

### DEFINITIONS AND BACKGROUND

Food intake and energy balance are regulated by two complementary drives: the homeostatic pathway increases motivation to eat following depletion of energy stores, and the hedonic (reward-based) pathway increases the desire to consume foods that are highly palatable during periods of abundance (Lutter and Nestler, 2009). Binge eating disorder (BED) involves recurrent episodes of eating in a discrete period of time an amount of food larger than most people would eat in the same time, a sense of lack of control over the eating episodes, rapid or secretive eating, guilt, and shame. BED is far more prevalent than AN and BN. The DSM-V will make BED an official diagnosis with frequency and duration at once per week for 3 months (Wilfrey et al, 2007).

Episodes may involve three or more of the following behaviors: eating more rapidly than normal, eating until uncomfortable, eating when not physically hungry, eating these foods alone, and feeling disgusted, guilty, or depressed. Adolescent and young adult vegetarians may be at increased risk for binge eating with loss of control (Robinson-O'Brien, 2009).

Binge eating is a serious problem among a subset of the obese. Weight cycling involves weight loss followed by weight regain along with psychological distress. Chronic dieting may predispose vulnerable individuals to binge eating, alcoholism, or drug abuse.

Many individuals with this problem have a personal or parental history of substance abuse. Persons with BED tend to be depressed and overweight. A single traumatic event, several years of unusual stress or pain, an extended period of emotional pain, or a mood disorder may be involved. Regardless of actual weight, there are high degrees of psychological distress in this group of individuals (Didie and Fitzgibbon, 2005). "Clinical" perfectionism involves both the determined pursuit of self-imposed standards and extremely vulnerable self-evaluation.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** People who have this disorder may be genetically predisposed to weigh more than the cultural ideal, so they may eat little, get hungry, and then binge in response to that hunger.

**Clinical/History**

- Height
- Weight
- BMI
- % Weight changes
- Dietary/intake history
- Binge pattern and frequency
- Socially Prescribed Perfectionism Scale
- Parental Authority Questionnaire (PAQ)
- Eating Disorders Inventory-2 (EDI-2)
- Eating Attitudes Test (EAT-26)

**Lab Work**

- Serum cortisol (high?)
- $Na^+$, $K^+$, $Cl^-$
- BUN, Creat
- Gluc
- Urinary acetone
- Chol, Trig

### INTERVENTION

#### OBJECTIVES

- Support the individual's counseling and therapy to identify the causes of binges. Help them follow a step-wise plan using self-monitoring records; regular patterns of eating to displace binge eating; alternative behaviors to help resist urges.
- Educate about food, eating, body shape, and weight patterns. Eliminate all aspects of restrained eating.
- Develop skills for dealing with difficulties that triggered past binges. Identify and challenge problematic ways of thinking.
- Consider the origins of the binge eating problem and then evaluate family and social factors that can be changed.
- Plan for the future. Have realistic expectations and strategies ready for when problems occur.
- Encourage a return to eating that is under the control of the individual.
- Correct any imbalances that have occurred as a result of the binges (e.g., weight, electrolyte imbalances).
- Support therapy, especially if there is a dual diagnosis, such as substance abuse.

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Excessive Oral Food and Beverage Intake**

***Assessment Data:*** Food records indicating periods of binge eating twice a week for the past 8 months.

***Nutrition Diagnoses (PES):*** Excessive oral food and beverage intake related to binge episodes as evidenced by food diary and intake records showing emotional binges when under stress.

***Interventions:*** Education about the hazards of purging and the benefits of eating healthy portions and nutrient-dense foods. Coordination of care with psychotherapist to manage stress through yoga, CBT, and appropriate levels of physical activity.

***Monitoring and Evaluation:*** Reports of fewer episodes of binge-purge cycles; improved quality of life; weight within desirable BMI range.

## FOOD AND NUTRITION

- A balanced diet, using principles of the dietary guidelines and the Food Guide Pyramid, should be planned according to age, sex, and goals for BMI.
- A slightly higher protein intake than usual helps to reduce binge eating, provide more satiety, and lower overall food intake.
- Alter diet according to medications, therapies, medical recommendations, and interdisciplinary care plan. This may include restriction of CHO, protein, fat, sodium, or other nutrients accordingly.

### Common Drugs Used and Potential Side Effects

- Pharmacotherapy is often beneficial in addition to psychotherapy. Antidepressants may be useful; monitor their specific effects.
- Topiramate (Topamax) may be an effective BED treatment; it has mild side effects, such as weight loss, that may be desirable.
- Sibutramine significantly reduces binge eating behavior and body weight in BED.

### Herbs, Botanicals, and Supplements

- No specific herbs and botanical products have been used for binge eating in any clinical trials.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage use of a food diary to record time, place, foods eaten, cues, binge feelings, and other comments.
- Discuss exercise and its effect on sense of well-being; shopping, holidays, and stressors.
- Discuss not skipping breakfast and lunch. This may lead to bingeing late into the evening or night.
- Focus on self-efficacy and proper assertiveness for coping with stressors.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.
- Note any unusual behaviors, such as pica, and discuss food safety issues if relevant.

### *For More Information*

- Academy for Eating Disorders
  http://www.aedweb.org
- Mayo Clinic
  http://www.mayoclinic.com/health/binge-eating-disorder/DS00608
- National Eating Disorders Association
  http://www.edap.org/

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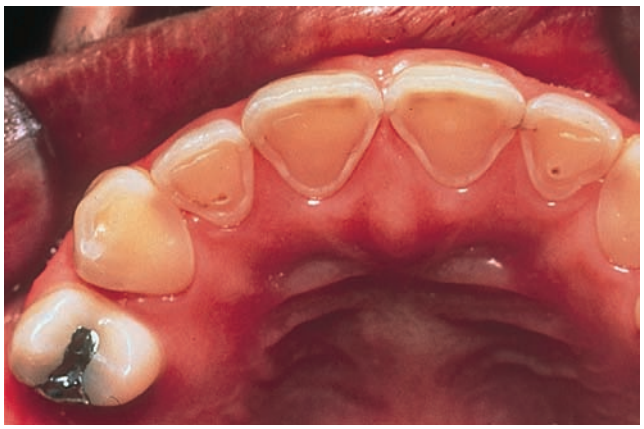
# BULIMIA NERVOSA

## NUTRITIONAL ACUITY RANKING: LEVEL 3–4



Adapted from: Langlais RP, Miller CS. *Color Atlas of Common Oral Diseases.* Philadelphia: Lea & Febiger, 1992.

## DEFINITIONS AND BACKGROUND

BN is an ED with food addiction as the primary coping mechanism. Criteria for diagnosis include recurrent episodes of binge eating, sense of lack of control, self-evaluation unduly influenced by weight or body shape, recurrent and inappropriate compensating behavior two times weekly for 3 months or longer (vomiting, use of laxatives or diuretics, fasting, excessive exercise). In BN, repeated binge episodes increase gastric capacity, which delays emptying, blunts cholecystokinin (CCK) release, and impairs satiety response.

Of the 5–30% of the population with bulimia, 85% are college-educated women. Weight may be normal or near-normal. When not bingeing, individuals with BN tend to be dieting; when hungry, they may binge and purge again. Their self-worth tends to be associated with thinness. The

most restrained patients with BN have the greatest desire to lose weight (Lowe et al, 2007).

Pathogenesis of BN suggests functional abnormalities within a neural system for self-regulatory control, which may contribute to binge eating and other impulsive behaviors (Marsh et al, 2009). Individuals with BN may experience loneliness, irritability, passivity, sadness, addictive behavior patterns, or suicidal behavior. Individuals with BN may shoplift, be promiscuous, and abuse alcohol, drugs, or credit cards. Disordered eating may occur for some time before drug or alcohol problems. Because disturbances in neuronal systems modulate feeding, mood, and impulse control, altered serotonin levels contribute to the disordered eating.

Significantly, the drive for thinness and body dissatisfaction relate to the patient's perception of father as "authoritarian" (Enten and Golan, 2009). Bulimics use food as a coping mechanism. Codependency is a dysfunctional pattern of relating to feelings. Individuals with BN focus on others or on things outside of themselves and deny their feelings. Fear, shame, despair, anger, rigidity, denial, and confusion are integral. Because of the low self-esteem, cognitive dysfunction, use of food or substance to relieve anxiety or depression, secretiveness, social isolation, and denial, psychotherapy is of primary importance. The team approach is best coordinated with the physician, a nutritionist, and a mental health professional.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Alterations in the serotonin (5-HT) 2A receptor are associated with behavioral impulsiveness and BN. Disturbances of 5-HT function occur when people are ill, and persist after recovery (Kaye, 2009).

**Clinical/History**

- Height
- Weight, current
- Usual weight
- BMI
- Percentage of weight changes
- Hx of laxative and diuretic abuse
- BP
- Dietary/intake history
- Oral and dental concerns
- Tooth enamel erosion (perimolysis)
- Knuckles with rough skin
- Broken blood vessels in the eye
- Salivary gland swelling
- Excessive bathroom use (to vomit)
- Perfectionism, obsessive-compulsiveness, dysphoria
- Parental Authority Questionnaire (PAQ)
- Eating Disorders Inventory-2 (EDI-2)
- Eating Attitudes Test (EAT-26)

**Lab Work**

- Serum amylase (high)
- Chol, Trig
- Gastrin
- LH, FSH (may be low)
- Gluc
- Alb
- $Na^+$, $K^+$, $Cl^-$
- Serum cortisol
- Serum folate
- BP
- H & H

### SAMPLE NUTRITION CARE PROCESS STEPS

**Disordered Eating Pattern**

***Assessment Data:*** Food records; binge-purging behaviors as indicated in food diaries.

***Nutrition Diagnosis (PES):*** Disordered eating pattern related to harmful belief about food and nutrition (i.e., that kcals are not available after using vomiting or use of laxatives) as evidenced by purging behaviors after a binge.

***Intervention:*** Educate and counsel about food and absorption after meals, dangers of vomiting and laxatives for weight control. Counsel about stress management and coping mechanisms using biofeedback, yoga or other techniques besides use of food.

***Monitoring and Evaluation:*** Improved food records; decreased use of vomiting and laxatives as a weight-control measure; improved self-esteem and quality of life.

## INTERVENTION

### OBJECTIVES

- Stabilize fluid and electrolyte imbalances.
- Individualize care plan to address weight history, dieting and binge eating episodes, purging behaviors, meal and exercise patterns.
- Promote effective weight control along with stress management. Establish a target weight in accordance with present weight, desirable BMI, reasonable time frame for recovery, and related factors. Modest energy restriction does not promote disordered eating (Wadden et al, 2004).
- Correct or prevent edema.
- Counteract lowered metabolic rate with balanced diet and exercise.
- Prevent oral health problems from vomiting and poor eating habits. About one third of persons with this condition will have erosion. Table 4-16 elaborates the oral manifestations and issues of concern in BN.

### FOOD AND NUTRITION

- Use controlled portions of a regular diet, usually with three meals and two snacks.
- Provide basal energy needs plus 300–400 calories as a beginning stage.
- Decrease sugar and alcohol intake, stressing the importance of other key nutrients. Highlight nutrient density and impact on health, appearance, and stamina.
- Encourage exercise along with diet and psychotherapy. Exercise decreases negative mood, improves EDs, and leads to more overall weight loss (Fossati et al, 2004).

### Common Drugs Used and Potential Side Effects

- The anticonvulsant Topiramate has shown good results in both binge and purge symptoms; it causes anorexic symptoms and weight loss.

**TABLE 4-16 Tips for Helping Patients with Eating Disorders (EDs)**

| Goal | Suggested Action |
|---|---|
| Full participation, especially in-patient | Manage anxiety disorders, which may precede onset of the ED. Patients with the binge eating/purging type of anorexia nervosa are significantly less likely to complete in-patient treatment. |
| Manage avoidant personality style | Persons with anorexia nervosa and avoidant personality style may discontinue therapy early. |
| Become an effective, independently functioning person | Convey principles rather than rigid "rules" to avoid reinforcing the patient's compulsive rituals, preoccupation with food, and perfectionism. |
| Positive, regular habits | Behavioral contracting is useful. |
| Balanced diet | Discuss how a balanced diet affects weight goals. Encourage healthy snacks. Medical nutrition therapy and education are cornerstones of therapy. |
| Identify hunger cues | Discuss signs of hunger and satiety. |
| Positive family relationships and healthy conflict management | Family dynamics play a role. Include family members in education and counseling sessions. Conflict management, support for individuality and personal opinions, and discussion of emotions will be part of therapy. |
| Healthy assertiveness and self-efficacy | Codependent behavior generally is a problem. Help the individual develop healthy reconnections and assertiveness. Computer-based psychosocial counseling may be helpful (Low et al, 2006). |
| Address social pressure for thinness | Preventive actions during middle school years may be helpful. For older individuals, open discussion of these issues may be useful as well. |
| Prevent relapse, which is common | Starvation and self-imposed dieting may lead to binges once food is available. Preoccupation with food and eating, emotional lability, dysphoria, and distractibility are common. |
| Monitor patients who have type 1 diabetes | Monitor for poor control, bulimia, skipping meals, hypoglycemia, hyperglycemia, and complications. Eating disorders are common in type 1 DM. |
| Support healthy pregnancies | Successful treatment includes appropriate pattern of weight gain, decreases in bingeing and purging behaviors, and normal infant birth weight. Special guidance is needed to achieve positive fetal outcomes. Use a team approach. |
| Promote healthy levels of physical activity | Discuss goals for the individual. Sometimes exercise is a goal in itself; therapy may be needed to address this issue along with eating patterns. Excessive exercise and hyperactivity are common. |

- Antidepressants such as sertraline may be used (O'Reardon et al, 2004). Monitor side effects such as glucose changes, dry mouth, constipation, increased BP, abdominal cramps, and weight changes. Avoid use with ma huang (ephedra), St. John's wort, and gingko biloba because they may enhance the effects and cause restlessness.
- Fluoxetine can help reduce binge eating and purging behaviors (Shapiro et al, 2007).
- Laxative and diuretic abuse can cause cardiac arrest and other problems. Discourage this practice.

## Herbs, Botanicals, and Supplements

- Alternative medicines are frequently used in this population; many products are available with potentially significant toxicities, especially diet pills and diuretics.
- Ma huang (ephedra), St. John's wort, and gingko biloba may enhance the effects of antidepressants and cause restlessness.
- With anticonvulsants, avoid use with evening primrose oil, gingko biloba, and kava.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Use of a biopsychosocial approach offers a means of working toward healing the whole person (Kreipe, 2006). The four elements of successful treatment in adolescents are (Kreipe and Yussman, 2003):
  - Recognizing the disorder and restoring physiological stability early in its course.
  - Establishing a trusting, therapeutic partnership with the adolescent.
  - Involving the family in treatment.
  - Using an interdisciplinary team approach.
- The combination of cognitive-behavioral therapy (CBT) with a nutritional education and a physical activity program helps to decrease depression and anxiety (Fossati et al, 2004).
- Help the patient rediscover the ability to be alone without giving in to the urge to binge. Assertiveness training may be of great benefit.
- Information, as from basic nutrition texts, can also encourage improved habits.
- Discuss the outcomes of electrolyte imbalance, such as muscle spasms, kidney problems, or cardiac arrest.
- Assert that there is "no such thing as a forbidden food." Discuss how to handle the cycle of bulimia: hopelessness or anxiety leading to gorging, leading to fear of fatness, leading to vomiting or drug abuse, leading to release from fear, leading to guilt, etc.
- Stringent oral hygiene after vomiting may reduce dental erosion.
- Self-help groups are often beneficial.
- Table 4-17 describes other disordered eating patterns and some tips.

**TABLE 4-17 Assessment of Oral Manifestations in Bulimia Nervosa**

| Condition | Issues of Concern |
|---|---|
| Enamel erosion (perimolysis) | Thermal sensitivity and pain |
| Salivary gland swelling (sialadenosis) | Hypertrophy from regurgitation of acidic contents; malnutrition |
| Dry mouth (xerostomia) | From vomiting, laxative, or diuretic abuse |
| Increased serum amylase | Two to four times increased levels occur after binging and vomiting; a marker for bulimia |
| Mucosal trauma | Abrasions and bleeding from rapid, forceful regurgitation |
| Gingival recession | From frequent and rigorous tooth brushing |
| Dental caries | From increased intake of junk foods, candy, sweets |

### *Patient Education—Foodborne Illness*

- Careful food handling and hand washing are important. If constant hand washing is a concern, referral to a mental health provider may be useful.

### *For More Information*

- Academy for Eating Disorders
  http://www.aedweb.org
- Eating Disorders Anonymous
  http://www.eatingdisordersanonymous.org/
- National Association of Anorexia Nervosa and Associated Disorders (ANAD)
  http://www.anad.org/
- National Eating Disorders Association
  http://www.edap.org/
- Women's Health—Bulimia Nervosa
  http://www.womenshealth.gov/FAQ/bulimia-nervosa.cfm

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MENTAL DISORDERS—OTHER

# BIPOLAR DISORDER

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2

## DEFINITIONS AND BACKGROUND

Abnormalities in brain biochemistry and circuits are responsible for the extreme shifts in mood, energy, and functioning that characterize BD. Bipolar affective disorders are characterized by mood swings from mania (exaggerated feeling of well-being, stimulation, and grandiosity in which a person can lose touch with reality) to depression (overwhelming feelings of sadness, anxiety, and low self-worth, which can include suicidal thoughts and suicide attempts). The disorders affect men and women equally. Children are rarely diagnosed. See Table 4-18.

The old name for BD is manic-depressive illness. The spectrum involves depression with varying degrees of excitatory signs and symptoms. Genetics seem to be involved. Relatives of people with bipolar affective disorder and depression are more likely to be affected. In general, the less severe the case, the later the onset of clinically observable mood disorder.

According to the DSM-IV, BD is a severe, recurrent, lifelong illness that affects up to about 7% of Americans. Lifetime prevalence rates for bipolar I and II disorder range up to 2%; for cyclothymia, a milder form of BD, prevalence ranges from 3% to 5%. More recent prevalence estimates are even higher. The World Health Organization reports that BD is the sixth leading cause of years lived with disability, worldwide.

For doctors working in a primary care setting, it is important to recognize the signs and symptoms of BD; it is commonly misdiagnosed as unipolar depression. Patients with bipolar depression are significantly more likely to report hallucinations, current suicidal ideation, and low self-esteem than patients with unipolar depression but less likely to report disturbed appetite (Das et al, 2005; Olfson et al, 2005).

The cyclical nature of the disorder poses challenges and barriers. Mood swings significantly impair the ability to function in social situations and to hold down a job. Patients often need to take days off from work either due to worsening clinical symptoms or hospitalization. When at work, problems may result from mood episodes such as poor concentration or low motivation during depression and inappropriate behavior during mania.

In mania, a person's behavior is often reckless and self-damaging. During mania, patients may spend excessive amounts of money or may have excessive urges to drive fast.

**TABLE 4-18 Other Disordered Eating Patterns**

| Disorder | Description |
|---|---|
| Anorexia athletica (compulsive exercising) | The person repeatedly exercises beyond the requirements for good health and is a fanatic about weight and diet. Not a formal diagnosis; behaviors are usually a part of anorexia nervosa, bulimia, or obsessive-compulsive disorder. Focuses on challenge and does not savor victory; proud of being an "elite athlete." Rarely satisfied with athletic achievements or performance. Needs a team approach for therapeutic intervention. |
| Body dysmorphic disorder (BDD) | BDD is thought to be a subtype of obsessive-compulsive disorder. It is not a variant of anorexia nervosa or bulimia nervosa. The person feels "ugly" and suffers from shyness and acts withdrawn in new situations or with unfamiliar people. Often strikes before age 18; affecting 2% of people in the United States. Sufferers are excessively concerned about appearance, in particular perceived flaws of face, hair, and skin. They are convinced these flaws exist despite reassurances from friends and family members who usually can see nothing to justify such intense worry and anxiety. High risk for despair and suicide; may undergo unnecessary expensive plastic surgery. BDD is treatable and begins with an evaluation by a physician and mental health care provider. Treatments include medication that adjusts serotonin levels in the brain and cognitive-behavioral therapy. A clinician makes the diagnosis and recommends treatment based on the needs and circumstances of each person. |
| Cyclic vomiting syndrome | Cycles of frequent vomiting, usually (but not always) found in children, often related to migraine headaches. Careful medical assessment is needed. Not a true eating disorder. |
| Eating disorders not otherwise specified (ED-NOS) | Official diagnosis describing atypical eating disorders where a person meets some but not all criteria for one specific eating disorder. Food behaviors are not normal and healthy; for example, behavior that resembles bulimia nervosa because of purging but without binge eating. |
| Gourmand syndrome | Person is preoccupied with fine food, including its purchase, preparation, presentation, and consumption. Exceedingly rare; thought to be caused by injury to the right side of the brain (as from tumor, concussion, or stroke). Relationship to addictions or obsessive-compulsive disorder possible. Had normal relationship with food prior to injury. Start with a neurologist evaluation. |
| Muscle dysmorphic disorder (bigorexia) | Sometimes called bigorexia, muscle dysmorphia is the opposite of anorexia nervosa. People with this disorder obsess about being small and undeveloped. They worry that they are too little and too frail. Even if they have good muscle mass, they believe their muscles are not big enough or are inadequate. Depression is the underlying concern. May understand the risks of steroid use but continue anyway. This condition results in disordered eating with very high protein and very low fat and often very low carbohydrate, often combined with excessive supplements. |
| Night eating syndrome | Affects 1–2% of general population. Likely that over one quarter of all morbidly obese persons may have this condition. This disorder is being considered for next psychiatric diagnostic classification manual. The person has little or no appetite for breakfast, delays first meal for several hours after waking up, and is often upset about how much was eaten the night before. Most calories are eaten late in the day or during the night. Sertraline, a selective serotonin reuptake inhibitor, may be beneficial in the treatment of night eating syndrome (O'Reardon et al, 2004). Psychotherapy is recommended. Self-help groups such as Overeaters Anonymous or group therapy can help. |
| Nocturnal sleep-related eating disorder | More of a sleep disorder than a true eating disorder. Individual eats while asleep and may sleep walk. No conscious memory of eating when they awaken again. Much guilt and confusion ensues. |
| Orthorexia nervosa | Eating the "right" food becomes an important, or even the primary, focus of life. One's worth or goodness is seen in terms of what one does or does not eat. Personal values, relationships, career goals, and friendships become less important than the quality and timing of what is consumed. May be a type of obsessive-compulsive disorder. |
| Pica | A craving for nonfood items such as dirt, clay, plaster, chalk, or paint chips. Pica may occur in pregnancy, in people whose diets are deficient in minerals found in the substances, in people with psychiatric conditions or developmental disabilities, or in people with family history of similar customs. Sometimes people who diet become hungry and ease their hunger with nonfood substances. May cause a medical emergency if obstruction or severe constipation occurs or if electrolyte imbalances occur. |
| Rumination syndrome | Person eats, swallows, and then regurgitates food back into the mouth where it is chewed and swallowed again. Process may be repeated several times or for several hours per episode. Process may be voluntary or involuntary. Ruminators report that regurgitated material does not taste bitter and that it is returned to the mouth with a gentle burp, not violent gagging or retching, not even nausea. Consequences range from minor inconveniences to life-threatening crises and include bad breath, indigestion, chapped lips and chin, damage to dental enamel and tissues in the mouth, aspiration pneumonia, failure to grow (children), weight loss, electrolyte imbalance, and dehydration. |

From: O'Reardon RP, Stunkard AJ, 2004. Anorexia Nervosa and Related Eating Disorders, Inc., http://www.anred.com/defslesser.html, accessed January 18, 2005.

During the depressive phase of the illness, patients may try to self-medicate themselves with alcohol or other substances, leading to problems with abuse or dependence. A series of four or more manic or depressive episodes in 12 months is known as "rapid cycling," a condition that can be more difficult to treat. Patients with bipolar I disorder are ill nearly half the time and have a high probability of relapse; bipolar II is more chronic, more depressive, and associated with more neuroticism and emotional instability between episodes than bipolar I (Keller, 2004).

Magnetic resonance spectroscopy imaging (MRSI) of the brains of patients (before starting medication) shows

different patterns in the chemical fingerprint in more severe cases than patients with mild-to-moderate disease. Severe BD requires more aggressive treatment. Cholesterol levels are lower during manic and depressive phases. Individuals treated with omega-3 fatty acids (in combination with their usual mood stabilizing medications) for 4 months experience fewer mood swings (Keck et al, 2006).

For treatment of resistant BD, high-dose thyroid hormones, calcium channel blockers, electroconvulsant therapy, omega-3 fatty acids, vitamin $D_3$, and psychosocial strategies have been investigated. Comorbid conditions are almost always involved. Anxiety, substance use, and conduct disorders are the most common; overeating, hypersexuality, attention-deficit hyperactivity, impulse control, autism spectrum disorders, and Tourette's syndrome are less common (McElroy, 2004). For these "dual diagnoses," both psychotherapy and appropriate medications are important (Levin and Hennessey, 2004).

The most common medical comorbidities include migraine, thyroid illness, obesity, type 2 diabetes, cardiovascular disease, chronic fatigue syndrome, asthma, chronic bronchitis, multiple chemical sensitivities, hypertension, and gastric ulcer tend to be significantly higher in bipolar patients (McElroy, 2004; McIntyre et al, 2006). Painful physical symptoms are common. Treatment of both physical and emotional symptoms associated with mood disorders may increase a patient's chance of achieving remission; abnormalities of serotonin and noradrenaline are strongly associated (Wise et al, 2005).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** BD and severe major depression are highly heritable, but differ from single-gene (Mendelian) diseases in that they are the end products of multiple causes (Cannon and Keller, 2006).

**Clinical/History**

- Height
- Weight
- BMI
- Dietary/intake history
- BP
- I & O
- Constipation
- Food pica?
- Disordered eating?
- Bipolar symptoms
- Mood Disorder Questionnaire
- Sheehan Disability Scale
- Clinical Global Impression Severity and Improvement Scores for Bipolar Disorder (CGI-BP)
- DEXA scan

**Lab Work**

- H & H
- Serum Fe
- Alb
- Chol with full profile
- Trig
- Serum tHcy
- CRP
- Serum $Ca^{++}$, $Mg^{++}$
- Gluc
- Serotonin
- Thyroid tests (T3, T4, TSH)
- $Na^{+}$, $K^{+}$

### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Intake of Certain Types of Fats**

***Assessment Data:*** Low serum cholesterol (LDL and HDL); cycling BD with little or no intake during manic and depressive phases; low weight for height.

***Nutrition Diagnoses (PES):*** Inadequate intake of omega-3 fatty acids related to cycling BD with poor appetite and low fat intake as evidenced by total cholesterol <140 (low LDL and HDL) and BMI of 17.5.

***Interventions:*** Food and nutrient delivery related to change in diet to include more omega 3 fatty acids and total kilocals. Education about the role of lipid in proper brain functioning. Counseling about healthy "brain foods" such as salmon, tuna, antioxidant foods.

***Monitoring and Evaluation:*** Dietary intake records showing improved intake of omega-3 fatty acids, energy, and regular meals; BMI improving and closer to low normal (18.5).

## INTERVENTION

### OBJECTIVES

- Support efforts at maintaining a balance between nutritional intake, physical activity, medications, and well-being.
- Monitor energy intake; counsel appropriately and offer tips for reducing kilocals from meals, snacks, and beverages. Metabolic syndrome can result.
- Monitor for medical problems related to weight gain, such as low HDL and high LDL cholesterol levels, elevated triglyceride levels, hyperglycemia or diabetes, and cardiovascular problems.
- Seek stable periods that are relatively normal ("euthymia"). Reduce stress, which elevates protein kinase C levels in the brain.
- Manage comorbid conditions that occur, such as diabetes, obesity, cardiovascular disease, and thyroid disorders.
- Maintain bone density since losses are common with use of various medications.

### FOOD AND NUTRITION

- Persons with BD may need an energy-controlled diet if their medications cause weight gain or obesity. Those who eat poorly will need a change in habits to gain weight.
- Snacks that are low in energy or fat may be useful between meals. Offer suggestions on what to keep on hand.
- During episodes of depression, keep prepared meals such as frozen dinners or packaged meals on hand.
- Discuss fluid and fiber if constipation is a problem.
- Include sufficient to higher levels of calcium and vitamin D intake from diet; a multivitamin–mineral supplement may be beneficial.
- Include omega-3 fatty acids. Fish oil–enriched diets increase omega-3 fatty acids in tissue phospholipids; flax oil increases circulating 18:3ω-3, thereby presenting tissue with this EFA for further elongation and desaturation (Barcelo-Coblijn et al, 2005).

## Common Drugs Used and Potential Side Effects

- Because of the risks of treating BD with antidepressant monotherapy, physicians should assess their depressed patients for mania before prescribing antidepressants (Olfson et al, 2005).
- The APA guideline for the treatment of BD recommends optimizing individual medications before switching to combination therapy, especially preventing discontinuation of therapy because of side effects (Bowden, 2004). Depressive symptoms of BD have a more negative impact on a patient's life than manic symptoms (Gao and Calabrese, 2005).
- **New antipsychotics** are effective for acute mania, and some may ultimately prove effective in acute depression (e.g., olanzapine combined with fluoxetine, quetiapine) and maintenance (Gao and Calabrese, 2005). Some antipsychotics, particularly olanzapine, clozapine, chlorpromazine, and thioridazine, result in serious weight gain. Energy expenditure is lower in people taking atypical antipsychotics; weight management programs may need to offer 280 kcals less per day (Sharpe et al, 2005). Aripiprazole (Abilify) is effective and has fewer side effects than some of the other atypical antipsychotics (Perlis et al, 2006).
- **Mood stabilizers**, such as lithium carbonate (Lithane, Lithobid, Lithotabs) and valproate, stabilize mood by significantly decreasing the manic, hypomanic, and depressive symptoms. Lithium causes weight gain; up to 25% of lithium users become clinically obese. Lithium requires constancy in sodium intake and limits on caffeine. Metallic taste, nausea, vomiting, and diarrhea may also occur. Valproate increases testosterone levels in teenage girls and may lead to polycystic ovary syndrome; careful monitoring is needed. These medications are contraindicated in pregnancy and lactation; lithium is associated with cardiac malformations, and valproate has been associated with neural tube defects.
- **New anticonvulsants** may be useful for aspects of BDs. Lamotrigine is used for maintenance or for acute bipolar depression. Compared with lithium and divalproex, lamotrigine is more effective in preventing bipolar depression (Gao and Calabrese, 2005). Topiramate may be used for problems related to obesity, bulimia, alcohol dependence, and migraine.
- **Multinutrient combinations** of vitamins, minerals, herbals, and the omega-3 fatty acids eicosapentaenoic acid (EPA) and DHA have been found to be somewhat effective (Kidd, 2004). Pramipexole, a dopamine D2/D3 receptor agonist, and omega-3 fatty acidsare used to augment mood stabilizers and are excellent in reducing depressive symptoms (Gao and Calabrese, 2005).
- Treatment of comorbid conditions may include use of gabapentin for anxiety or pain and zonisamide for obesity.
- **Medicines that can cause mania** include: amphetamines, Antabuse, anticholinergics, baclofen, benztropine, bromocriptine, bupropion, captopril, cimetidine, corticosteroids, cyclosporine, hydralazine, isoniazid, levodopa, MAOIs such as Nardil or Parnate, Ritalin, Synthroid, opioids, procarbazine, and yohimbe.
- **Medicines that can cause depression** include: acyclovir, alcohol, anticonvulsants, asparaginase, baclofen, barbiturates, benzodiazepines, beta-blockers, bromocriptine, calcium channel blockers, corticosteroids, cycloserine, dapsone, estrogens, fluoroquinolone, histamine $H_2$–receptor antagonists, interferon, isotretinoin, mefloquine, methyldopa, metoclopramide, narcotics, progestins, statins, sulfonamides.

## Herbs, Botanicals, and Supplements

- Ginger, licorice, purslane, rosemary, and ginseng have been suggested; no clinical trials have proven efficacy in BDs.
- Ginseng and yohimbe should not be used with MAO inhibitors.
- Gingko biloba interacts with anticoagulants and antiplatelets such as aspirin, warfarin, and dipyridamole.
- L-tryptophan may be tried for insomnia or depression. Do not use with MAO inhibitors, SSRI antidepressants, or serotonin receptor antagonists.
- Ma huang (ephedra) and kava should not be taken by patients with depression.
- Omega-3 fatty acids have a benefit in managing depression. Supplementation may improve symptoms of depression in children and BD in adults; 360 mg/d EPA and 1560 mg/d DHA for 6 weeks show good results (Clayton et al, 2009).
- Psyllium and ginseng should not be used with divalproex or lithium.
- St. John's wort is used as a natural antidepressant. Do not use with MAO inhibitors, SSRI antidepressants, cyclosporine, digoxin, oral contraceptives, HIV protease inhibitors, theophylline, warfarin, or calcium channel blockers such as amlodipine, diltiazem, or verapamil.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Teach creative menu planning and food preparation methods that address the side effects and symptoms the patient is experiencing.
- Teach the patient how to moisten foods for dry mouth syndrome resulting from certain medications. Sugar-free candy may help.
- Limit caffeine-containing foods and beverages in the late evening to improve sleep.
- Individuals who are prone to bouts of depression or mania may find it difficult to eat properly. Simple meals and snacks should be readily available.
- Since there is a higher risk for suicide, individuals with BD should be carefully monitored for signs of severe depression and should seek help from a mental health professional immediately.
- Functional outcomes are reliable measure of response to treatment. Changes in circadian rhythm and sleep patterns may predict onset of relapse. Patients and families may benefit from education and therapy.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.

### *For More Information*

- Bipolar Disorder
  http://www.cmellc.com/topics/bdfaq.html
- Depression and Bipolar Support Alliance
  http://www.dbsalliance.org/
- Food and Mood
  http://www.dbsalliance.org/pdfs/foodmoode2.pdf
- JAMA Patient Page
  http://jama.ama-assn.org/cgi/reprint/301/5/564.pdf
- Medline—Bipolar Disorder
  http://www.nlm.nih.gov/medlineplus/bipolardisorder.html
- Mental Health Links
  http://mentalhealth.samhsa.gov/links/default2.asp
- NIMH—Bipolar Disorder
  http://www.nimh.nih.gov/health/publications/bipolar-disorder/complete-index.shtml
- National Mental Health Information Center
  http://www.mentalhealth.samhsa.gov/

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# DEPRESSION

## NUTRITIONAL ACUITY RANKING: LEVEL 1 (MILD); LEVEL 2 (NUMEROUS MEDICATIONS)

### DEFINITIONS AND BACKGROUND

Depression involves changes in body chemistry (neurotransmitters) because of genetics or after a traumatic event, hormonal changes, altered health habits, the presence of another illness, or substance abuse. Persons with major depressive disorder (MDD) have had at least one major depressive episode over a 14-day period or longer. It may be recurrent throughout their lives. MDD is debilitating and has a high morbidity rate (Farah, 2009).

The lifetime risk for depression is 10–25% in women, 5–12% in men (Fava, 2007). Depression frequently develops between the ages of 25 and 44. Approximately 20 million adult Americans experience depression every day. Diagnosis of depression is indicated by four of eight of the following symptoms: SIGECAPS—Sleep changes, loss of Interest, inappropriate Guilt (hopelessness), Energy decline, Concentration changes, Appetite changes, Psychomotor changes, and Suicidal tendencies. In addition, prolonged sadness and unexplained crying spells, chronic irritability and agitation or anxiety, chronic pessimism or indifference, indecisiveness, social withdrawal, and unexplained aches and pains may also be present.

Folate seems to have a causal relationship in depression (Lewis et al, 2006). When there are lower red blood cell folate levels, episodes of depression are longer and more severe (Fava, 2007). Deplin® contains 7.5 mg L-methylfolate and may help in managing depressive episodes. It unmasks vitamin $B_{12}$ anemia whereas folic acid masks vitamin $B_{12}$ anemia. After 4-week trials of Deplin®, if patients are not feeling better, psychiatrists often recommend doubling the dose to 15 mg/d (Zajecka, 2007).

Depression is a leading cause of disability in the United States. Careful assessment is needed to determine the specific type of depression and its most effective treatments. **Dysthymia** is a chronic, moderate type of depression expressed as poor appetite or overeating, insomnia or oversleeping, low energy, fatigue, irritability, and high stress. Children and teens who experience depression may experience frequent headaches and absences from school. Men often mask signs of depression by working long hours or drinking too much. Women may have depression during times of hormonal change (menstruation, pregnancy, miscarriage, the postpartum period, and menopause). **Postpartum depression** negatively affects both mother and child; early detection and treatment are needed.

Older individuals may experience depression along with a chronic disease such as heart disease, diabetes, or hypertension. CRP tends to be elevated in depression and may contribute to heart disease (Ford and Erlinger, 2004). Elevated tHcy levels should be treated. In nursing homes, it is expected that about 50% of individuals will have some form of depression for which medication should be prescribed.

Many persons with depression have a deficiency of brain serotonin. A mixed diet of protein/CHO should provide tryptophan, a precursor of serotonin. Intake of dietary protein

high in tryptophan increases the ratio of tryptophan to large neutral amino acids (LNAA). Antidepressants that are serotonin reuptake modulators actually promote growth of serotonin innervation in the forebrain (Zhou et al, 2006). Painful physical symptoms commonly exist comorbid with depressive disorders; abnormalities of serotonin and noradrenaline are strongly associated and play a role in pain perception (Wise et al, 2005).

Psychotherapy can change brain chemistry after cognitive restructuring. For some persons for whom medications and therapy are not effective, electroconvulsive therapy (ECT) may also be helpful. A pacemaker-like device that sends electrical stimulations into the vagus nerve of the neck may help to alleviate depressive symptoms. And, recent studies show that vitamin D is an important component for alleviating depression. SAD increases with latitude, so vitamin D and sunlight or light therapy may be beneficial.

Monitoring physical health, including nutrition, is an important adjunct to medication or psychotherapy. Adequate intake of the omega-3 fatty acids may reduce depression (Casper, 2004). The omega-3 fatty acids are important components of nerve cell membranes and help nerve cells communicate with each other. In medicated patients, added treatment with omega-3 fatty acids, particularly EPA, may ameliorate symptoms of MDD (Casper, 2004). Finally, use of fresh versus highly processed foods should be considered. A highly processed food dietary pattern is a risk factor for depression years later, whereas a whole food pattern is protective (Akbaraly et al, 2009).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** About 40% of risk for major depression is inherited; 60% is from environmental factors such as substance abuse. Multiple genes play a role in depression (Psychiatric GWAS Consortium, 2009). Individuals with the C>T type of MTHFR allele are at high risk for depression (Almeida et al, 2008). Testing for MTHFR levels and augmenting therapy with methylfolate and omega-3 fatty acids is suggested (Fava, 2007; Shelton, 2007). Research has identified some genetic mutations found only in women, related to female hormone regulation; these biological differences are being clarified.

**Clinical/History**

- Height
- Weight
- BMI
- Weight changes
- Dietary/intake history
- BP
- Food pica
- I & O
- Constipation
- DEXA scan

**Lab Work**

- H & H
- Serum Fe
- Alb
- $Ca^{++}$, $Mg^{++}$
- Gluc
- Serotonin
- Thyroid tests (T3, T4, TSH)
- $Na^{+}$, $K^{+}$
- N balance
- MTHFR
- Serum tHcy
- Serum folate and $B_{12}$
- CRP

### SAMPLE NUTRITION CARE PROCESS STEPS

**Altered Nutrient Utilization**

***Assessment Data:*** Altered methyltetrahydrofolate reductase (MTHFR) with C>T allele; signs of severe depression with insomnia and poor dietary intake; elevated tHcy levels.

***Nutrition Diagnoses (PES):*** Altered nutrient utilization of folate related to MTHFR allele as evidenced by lab test, diagnosis of MDD, and poor intake from oral diet.

***Interventions:*** Education about the role of folate in depression; discussion about genetics and how the C>T allele can aggravate depression. Counseling about use of L-methylfolate and dietary sources of folate, vitamins $B_6$ and $B_{12}$.

***Monitoring and Evaluation:*** Improved lab results for tHcy; fewer episodes of prolonged depression; improved quality of life.

## INTERVENTION

### OBJECTIVES

- Provide adequate nutritional intake (e.g., excessive weight loss or shock therapy requires increased energy intake). Monitor weight at least twice monthly to evaluate status and changes.
- Assess usual eating habits and related problems, which may include loneliness, difficulty in activities of daily living, boredom, lack of hobbies and interests, and poor sleep habits. Adequate drug therapy usually helps appetite improve.
- Monitor for consequences from certain antidepressants, such as weight gain.
- Assure adequate intake of amino acids, omega-3 fatty acids, folic acid, vitamins $D_3$ and $B_{12}$.
- Reduce stress, which elevates protein kinase C levels in the brain.

### FOOD AND NUTRITION

- Use a diet providing high-quality protein. Inadequate protein intake may reflect decreased intake of iron, thiamine, riboflavin, niacin, and vitamins $B_6$ and $B_{12}$ as well.
- Increase intake of omega-3 fatty acids and uridine from foods such as fish, walnuts, molasses, and sugar beets (Carlezon et al, 2005). Supplements may also be used, but dietary change is beneficial.
- Low serum folate is common in many depressed adults, especially women (Ramos et al, 2004; Tolmunen et al, 2004). Intake of 400 μg is needed daily. Use L-methylfolate where MTHFR alleles have been identified.
- Vitamin $D_3$ and calcium should be supplemented (Mussolino et al, 2004; Payne et al, 2008).
- If serum tHcy levels are high, include vitamins $B_6$ and $B_{12}$ in addition to folate.
- Use a tyramine-restricted diet for patients given MAOI drugs. Such a diet excludes aged cheese, beer, red wine, ale, chicken livers, broad bean pods, sausage, salami,

pepperoni, commercial gravies, ripe avocado, fermented soy sauce, and pickled or smoked herring.
- If overeating, limit access to food and provide low-calorie diet information.
- Encourage increased physical activity, which often helps to lift depressed moods.
- Sometimes a craving for carbohydrates occurs; monitor if weight gain is a problem or if overall nutrient density decreases.
- Liquid supplements may be useful when preparing meals seems overwhelming.
- TPN is not advised for patients who are suicidal.

## Common Drugs Used and Potential Side Effects

- SSRIs and TCAs do not always provide symptom relief. Yet, the SSRIs increase the density of nerve-impulse carrying axons in the brain, thus rewiring the neocortex and limbic system (Zhou et al, 2006). Their use may be more beneficial than previously anticipated.
- Some serotonergic antidepressants (e.g., fluoxetine, Prozac) can reduce hyperglycemia, normalize glucose homeostasis, and increase insulin sensitivity, whereas some noradrenergic antidepressants (e.g., desipramine) exert opposite effects (McIntyre et al, 2006).
- L-methylfolate may be needed to help alleviate depression (Mischoulon and Raab, 2007; Stahl, 2007).
- See Table 4-19 for more details.

## Herbs, Botanicals, and Supplements

- Ginseng and yohimbe should not be used with MAO inhibitors.
- Licorice, ginger, purslane, rosemary, and ginseng have been suggested for depression; no clinical trials prove efficacy.
- L-tryptophan may be tried for insomnia or depression. Do not use with MAO inhibitors, SSRI antidepressants, or serotonin receptor antagonists.
- Ma huang (ephedra) and kava should not be used with antidepressants.
- Omega-3 fatty acids (EPA and DHA) may be taken in capsule form; usually 1000 mg several times a day will be needed.
- St. John's wort is used as a natural antidepressant. Do not use with MAO inhibitors, SSRI antidepressants, cyclosporine, digoxin, oral contraceptives, HIV protease inhibitors, theophylline, warfarin, or calcium channel blockers such as amlodipine, diltiazem, or verapamil.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Encourage full involvement with psychotherapy, CBT, or interpersonal therapy (IPT). These are helpful adjuncts for medication. Hopefulness and resilience are also important.

**TABLE 4-19 The Bipolar Spectrum and Symptoms**

| | | |
|---|---|---|
| Bipolar type I | Depression and varying degrees of excitatory signs and symptoms up to full mania | Onset during teens and early adulthood; 60% will have problems with substance abuse |
| Bipolar type II | Discrete hypomanic episodes; may appear to have just depression, but mood stabilizers seem to help more than antidepressants | 50% will have problems with substance abuse |
| Bipolar type III | Hypomania associated with antidepressants and/or psychostimulants | |
| Bipolar type IV | Hyperthymic temperament | Onset during 4th or 5th decade of life |
| Bipolar type V | Recurrent depressions without discrete irritability, hypomania, but mixed hypomanic episodes with agitation, and racing thoughts during depression | |
| Bipolar type VI | Alzheimer's type | Onset in the 6th or 7th decade |
| Manic symptoms | Severe changes in mood, either extremely irritable or overly silly and elated<br>Overly inflated self-esteem, grandiosity<br>Increased energy<br>Decreased need for sleep, ability to go with very little or no sleep for days without tiring<br>Increased talking, talks too much, too fast; changes topics too quickly; cannot be interrupted<br>Distractibility, attention moves constantly from one thing to the next<br>Hypersexuality, increased sexual thoughts, feelings, or behaviors; use of explicit sexual language<br>Increased goal-directed activity or physical agitation<br>Disregard of risk, excessive involvement in risky behaviors or activities | |
| Depressive symptoms | Persistent sad or irritable mood<br>Loss of interest in activities once enjoyed<br>Significant change in appetite or body weight<br>Difficulty sleeping or oversleeping<br>Physical agitation or slowing<br>Loss of energy<br>Feelings of worthlessness or inappropriate guilt<br>Difficulty concentrating<br>Recurrent thoughts of death or suicide | |

- Teach creative menu planning and food preparation methods that address the side effects and symptoms the patient is experiencing.
- Promote the use of whole foods (fruits, vegetables, fish) and discourage processed foods (fried foods, sweetened desserts, processed meats; Akbarahly et al, 2009).
- Teach the patient how to moisten foods for dry mouth syndrome resulting from certain medications. Sugar-free candy may help.
- Promote exercise, which seems to help reduce symptoms of depression. Teens may be especially vulnerable to depression, and exercise may help improve other health-related behaviors, including eating better (Fulkerson et al, 2004).
- Limit caffeine-containing foods and beverages in the late evening to promote better sleep.
- Nicotine dependence and major depression are often related; treat mutually (Manley et al, 2009).
- After giving birth, postpartum "blues," postpartum depression, or postpartum psychosis may occur. Maternal depression in the perinatal period may lead to poor growth for infants (Rahman et al, 2004). Education is needed for new mothers.
- In the elderly, "failure to thrive" usually includes impaired physical function, malnutrition, depression, and cognitive impairment (Robertson and Montagnini, 2004). Treatment of depression may help to improve appetite. Adding breakfast to homebound meals is a good start (Gollub and Weddle, 2004).

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.

### *For More Information*

- Academy of Cognitive Therapy
  http://www.academyofct.org
- American Mental Health Counselors Association
  http://www.amhca.org/
- American Psychiatric Association
  http://www.psych.org/
- American Society of Geriatric Psychiatry
  http://www.aagpgpa.org/
- Anxiety Disorders of America
  http://www.adaa.org/
- Depression
  http://www.depression.com/
- National Depression Screening
  http://www.mentalhealthscreening.org/infofaq/depression.aspx
- NIMH—Depression
  http://www.nimh.nih.gov/health/publications/depression/complete-index.shtml

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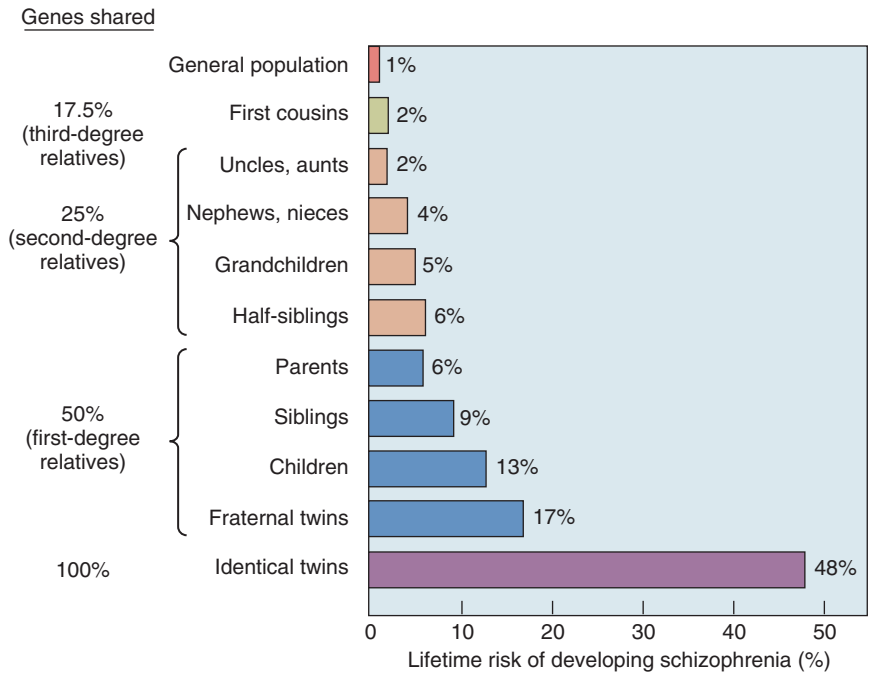
# SCHIZOPHRENIA

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2




Adapted from: Gottesman, 1991, p. 96.

## DEFINITIONS AND BACKGROUND

**SCZ** is a group of disorders manifested by disordered thinking, hallucinations, delusions, apathy, social withdrawal, and mood or behavioral disturbances (delusional, catatonic, or paranoid). In the resulting psychosis, the individual loses contact with reality. SCZ has a heritability estimated at 73–90% (International Schizophrenia Consortium, 2008).

SCZ can be either episodic or chronic. When patients are at risk of self-harm or harm to others, hospital treatment is appropriate. Delusions may involve control, persecution, grandiosity, or abnormal fears. Hallucinations are perceptions of an external stimulus without a source in the external world.

Nearly 1% of the population develops SCZ, with onset generally between ages 15 and 25 years (earlier in males). SCZ has been thought to have a genetic component and is associated with reduced dopamine signaling and executive function impairment (Roffman et al, 2007). However, there are many structural DNA copy number variants (CNVs) in the brains of individuals with SCZ; these rare deleterious variants may be more important in SCZ predisposition than common polymorphisms (Need et al, 2009).

Molecular mechanisms critical for adolescent brain development are disturbed in SCZ patients from genes associated with energy metabolism and protein and lipid synthesis (Harris et al, 2009). Oxidative stress, oxidative injury, and abnormal membrane phospholipid metabolism suggest that PUFA fatty acid depletion occurs (duBois et al, 2005). Similar to diabetes, conversion of ALA to EPA or DHA is inefficient in SCZ. Supplementation with antioxidants and omega-3 fatty acids is recommended. Otherwise, effective serum lipid control is difficult to attain (Weiss et al, 2006).

Low vitamin D availability during brain development interacts with susceptibility genes to alter brain development. Vitamin D supplementation during the first year of life is important, especially in families with a history of SCZ.

Folate, cobalamin ($B_{12}$), tHcy, and MTHFR polymorphisms are also found in SCZ. Patients with SCZ who have the MTHFR A>C genotype have a slight correlation with positive symptoms such as hallucinations and hearing voices, whereas the C>T allele promotes negative symptoms (depression, dysthymia) but actually protects against positive symptoms (Roffman et al, 2007; Zintzaras, 2006). Both folate and cobalamin deficiencies should be identified (Miller, 2008). Supplementation with S-adenosylmethionine (SAM) may be useful (Frankenburg, 2007; Gilbody et al, 2007).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** No single gene has been identified. Reelin (a secretory protease that manages neuronal migration, dopamine signaling, and synaptogenesis) is heavily methylated in SCZ (Guidotti et al, 2009; Roffman et al, 2007; Suzuki et al, 2008). Viral infections

reduce reelin levels and may trigger SCZ. If the reelin-VLDLR/ApoER2 signaling pathway is significant, peripheral VLDLR mRNA levels may be biological markers of SCZ (Suzuki et al, 2008). Other susceptibility genes are neuregulin 1 (NRG1); DISC-1 genes (Lipska et al, 2006); catechol-O-methyltransferase (COMT) for dopamine transmission (Roffman et al, 2008; Woodward et al, 2007); and the potassium channel KCNH2 gene, for neuronal firing and higher mental functions (Huffaker et al, 2009).

MTHFR alleles promote some symptoms seen in SCZ (Roffman et al, 2007) and the tryptophan-serotonin pathway has been implicated from 5-HTT gene polymorphic variants. SCZ patients may have large deletions on chromosome 15q13.3 and 1q21.1 (International Schizophrenia Consortium, 2008). However, very few SCZ patients share identical genetic alterations; this complicates efforts to personalize treatment regimens (International Schizophrenia Consortium, 2008; Need et al, 2009).

### Clinical/History

- Height
- Weight
- Weight changes
- BMI
- Dietary/intake history
- I & O
- BP
- Global Assessment of Functioning Scale (GAF)
- Positive and Negative Syndrome Scale (PANSS)
- Delusions, auditory hallucinations
- Blunted speech
- Flat affect
- Lack of motivation
- Asociality
- Anhedonia
- Heightened emotionality to stress
- Disorganized speech and thought disorder
- EEG and brainwave patterns
- MRI with hippocampus reduction (Velakoulis et al, 2006)
- PET scans

### Lab Work

- Peripheral VLDLR mRNA levels
- Gluc
- Creatine phosphokinase (CPK) (elevated in acute episodes)
- Chol (total profile)
- Trig
- Alb, transthyretin
- $Na^+$, $K^+$
- H & H
- Serum Fe
- tHcy
- Methylmalonic acid
- MTHFR polymorphisms
- Serum folate and $B_{12}$
- CRP
- Serum insulin
- Serum $D_3$
- $Ca^{++}$, $Mg^{++}$

## INTERVENTION

### OBJECTIVES

- Develop a trusting relationship; make expectations clear to the patient.
- Provide adequate nourishment to prevent significant weight changes; gain is common with many antipsychotic medications.
- Correct any nutritional deficits in folate, vitamins $B_6$ and $B_{12}$, and vitamin D. Use L-methylfolate with MTHFR alleles.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Poor Food Choices**

***Assessment Data:*** Food records indicating poor intake; weight gain; elevated serum LDL and triglycerides; hypertension; high waist circumference.

***Nutrition Diagnoses (PES):*** Poor food choices related to regular intake of high fat, fast foods and few fruits and vegetables as evidenced by weight gain while on antipsychotics, BP of 170/95, LDL 190, triglycerides 300, waist circumference 42 and BMI 32.

***Interventions:*** Education about better food choices to lower serum lipids and BP; importance of managing weight and health status to prevent cardiovascular disease. Counseling about regular mealtimes, ways to increase fiber, antioxidant-rich fruit and vegetable intake. Use motivational interviewing.

***Monitoring and Evaluation:*** Improved weight status (lower BMI, waist circumference); lipids closer to normal for age and sex; no further weight gain with medicines; better food choices as indicated on food records.

- Promote a normal pattern of dietary intake and exercise routines.
- Prevent or correct constipation or impaction.
- Manage diabetes and coronary heart disease, which tend to be common and often under-diagnosed (Henderson, 2005). Metabolic syndrome is—two to four times higher in SCZ than in the general population (Ellingrod et al, 2008; McEvoy et al, 2005). Both decreased MTHFR activity (in C>T allele carriers) and elevated tHcy can increase risk of cardiovascular disease (Ellingrod et al, 2008).
- Reduce stress, which increases protein kinase C in the brain, increases forgetfulness, and speeds up the aging process.

### FOOD AND NUTRITION

- A balanced diet for age and sex should be used. Reduce sugars and saturated fats if diabetes or metabolic syndrome are present.
- Adjust calories according to goal weight for patient and medications.
- Reduce potential accidents by avoiding glass containers and serving dishes.
- Vitamins C, D, $B_6$, and $B_{12}$, folate, and selenium levels may be low in persons with SCZ; encourage improved intake accordingly.
- Highlight the use of antioxidant-rich foods (berries, nuts, green tea) and omega-3 fatty acids (salmon, tuna, sardines).
- Suggest use of the DASH diet if BP is high.

### Common Drugs Used and Potential Side Effects

- Some drugs can cause psychiatric symptoms. Anxiety, mania, hallucinations, suicidal thoughts, and bizarre

behavior may result from various medications, and a doctor should be contacted if these occur:

Confusion: acyclovir, propoxyphene (Darvon), and cimetidine (Tagamet)
Depression: oral contraceptives, ibuprofen, metronidazole (Flagyl), barbiturates, cimetidine, and diazepam (Valium)
Excitement or agitation: alprazolam, amphetamines, barbiturates, metronidazole, and diazepam Insomnia: acyclovir and alprazolam
Paranoia: amphetamines, cannabis, ibuprofen, cimetidine, and TCAs

- Try offering a beverage or snack to reduce anxiety before adding new medications (Ativan, Xanax, Klonopin, Paxil).
- To reduce hypermethylation of GABAergic promoters, COX-2 inhibitors or valproate may be used along with atypical antipsychotics (Table 4-20).
- What works for one person may not work for another. See Table 4-21 for more guidance on medications.

## Herbs, Botanicals, and Supplements

- Ginseng should not be used with CNS stimulants, caffeine, hormones, steroids, antipsychotics, MAO inhibitors, lithium.
- Gingko biloba interacts with anticoagulants and antiplatelets such as aspirin, warfarin, and dipyridamole.
- Indian snakeroot is used for mental illness in some cultures. Do not use with digoxin, phenobarbital, levodopa, albuterol, furosemide, thiazide diuretics, MAO inhibitors, beta-blockers such as atenolol or propranolol, or tranquilizers. Problems include potential sedation, increased BP, arrhythmias, and CNS excitation.
- Kava and valerian should not be taken with anxiety-reducing drugs (e.g., alprazolam, diazepam, lorazepam).
- Tryptophan metabolism may be altered by proinflammatory cytokines; this affects serotonin production and functioning. If L-tryptophan is taken, avoid use with MAO inhibitors, SSRI antidepressants, or serotonin receptor antagonists. Deaths have been related to the use of L-tryptophan in the past.
- Ma huang (ephedra) and kava should not be taken in patients with depression.
- Psyllium should not be used with divalproex or lithium.
- St. John's wort should not be used with MAO inhibitors, SSRI antidepressants, cyclosporine, digoxin, oral contraceptives, HIV protease inhibitors, theophylline, warfarin, or calcium channel blockers such as amlodipine, diltiazem, or verapamil.
- Yohimbe should not be used with MAO inhibitors.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Teach nutrition principles to the patient or the caregiver.
- Encourage self-care. Successfully terminate client relationship when independence is possible.
- Provide follow-up, especially with regression. If daily medications are a problem, monthly injectable medications may be useful.
- Trauma, pain, endocrine disorders, infection, and metabolic disorders may cause agitation (Marco and Vaughan, 2005). A quiet environment is needed, especially for meals.
- Weight gain is common (Patel et al, 2009). Weight loss programs, cholesterol-lowering (TLC) plans, or the DASH diet may be needed.

**TABLE 4-20 Antipsychotic Medications and Possible Side Effects**

| Medication | Side Effects |
|---|---|
| Tranquilizers | Triavil combines an antidepressant with a tranquilizer. Nausea, diarrhea, and vomiting may result. |
| Typical antipsychotics: clozapine (Clozaril),butyrophenone (Haldol), thiothixene (Navane) | May cause dizziness, drowsiness, dry mouth, weight gain, edema, nausea, constipation, or vomiting. They help to quiet symptoms and help when the patient is resistant to other drugs and alternatives. |
| Phenothiazines: perphenazine (Trilafon), fluphenazine (Prolixin), prochlorperazine (Compazine), chlorpromazine (Thorazine) | Chlorpromazine (Thorazine) contains sulfites. It may cause dry mouth, constipation, and weight gain. |
| Atypical antipsychotics (aAPs): aripiprazole (Abilify), quetiapine (Seroquel), risperidone (Risperdal), olanzapine (Zyprexa), ziprasidone (Geodon) | The phospholipids in the neuronal membranes of the brain are rich in highly unsaturated essential fatty acids (EFAs). With a beneficial effect on dyskinesia as well, EPA is an effective adjunct to antipsychotics.<br>Aripiprazole exhibits high affinity for dopamine, serotonin, and histamine receptors. Ziprasidone is not as likely to cause weight gain as some other antipsychotics. Olanzapine performs modestly better than most other medications, but weight gain can be significant. Long-acting risperidone injection may increase adherence and lead to improved clinical and economic outcomes for individuals with schizophrenia (Edwards et al, 2005).<br>Typical doses may be: olanzapine (schizophrenia: 15 mg; mania: 20 mg); quetiapine (schizophrenia: 750 mg; mania: 800 mg); ziprasidone (schizophrenia and mania: 160 mg); aripiprazole (schizophrenia and mania: 30 mg). |
| SSRIs and obsessive-compulsive disorder medications | Prozac, Anafranil, Luvox, and Zoloft have been used with some success. Avoid use with ma huang (ephedra) and St. John's wort. Fluvoxamine (Luvox) is an SSRI that may cause anorexia, dry mouth, nausea, diarrhea, and constipation. |

**TABLE 4-21 Medications for Depression and Mood Disorders and Possible Side Effects**

| Medication | Side Effects |
|---|---|
| Dual-mechanism antidepressants: Cymbalta (duloxetine), Effexor (venlafaxine) | Approved for the treatment of major depressive disorder in 2004, these are serotonin and norepinephrine reuptake inhibitors. Dual-mechanism antidepressants do not appear to disrupt glucose homeostatic dynamics.<br>Brain-derived neurotrophic factor, which is increased with antidepressant treatment, appears to influence regulation of mood and perception of pain; evidence indicates that dual-acting agents may have an advantage in modulating pain over those agents that increase either serotonin or noradrenaline alone (Wise et al, 2005). |
| Other antidepressants | Clomipramine (Anafranil) is used in obsessive-compulsive disorders. Dry mouth is common; hard sugar-less candy or chewing gum may be useful. Anorexia and abdominal pain are also common.<br>Norpramin (desipramine) may cause abdominal cramps, altered blood glucose levels, and vomiting. Avoid use with ma huang (ephedra), St. John's wort, and ginkgo biloba.<br>Nortriptyline (Aventyl, Pamelor) may cause increased appetite for sweets, GI distress, vomiting, and diarrhea.<br>Wellbutrin (bupropion) tends to have a stimulating effect but may also cause weight loss, dry mouth, nausea, and vomiting. It may be used to help with smoking cessation. |
| Monoamine oxidase inhibitors: Parnate (tranylcypromine), Nardil (phenelzine), Marplan (isocarboxazid) | Nonselective hydrazine monoamine oxidase inhibitors (e.g., phenelzine) are associated with hypoglycemia and an increased glucose disposal rate (McIntyre et al, 2006). Tyramine is a pressor amine.<br>Tyramine-restricted diet to prevent hypertensive crisis: spoiled, overripe, and aged products are the most problematic. Beware of Chianti wines, beer, fava beans, and sauerkraut. Constipation, weight gain, and GI distress are common side effects. Avoid ginseng, L-tryptophan, yohimbe, St. John's wort, kava, and ma huang (ephedra). |
| SAMe (S-5-adenosyl-methionine) | Useful for mild depression but may trigger coronary problems. A positive side effect is that it may actually help with degenerative joint disease symptoms. |
| Selective serotonin reuptake inhibitors (SSRIs): Paxil (paroxetine), Prozac (fluoxetine), Zoloft (sertraline) | May cause abdominal pain, anorexia, diarrhea, and weight changes; SSRIs are used to treat despair and helplessness.<br>Prozac may also cause nausea, vomiting, glucose changes, and decreased sodium.<br>Do not use in pregnancy; neurobehavioral effects have been noted in otherwise healthy infants. Fetal exposure to a mother's antidepressants during pregnancy may leave her newborn in withdrawal, known as neonatal abstinence syndrome (Levinson-Castiel et al, 2006).<br>Zoloft can cause dry mouth and diarrhea; avoid use with St. John's wort and ma huang (ephedra). |
| Tranquilizers, benzodiazepines: Halcion (triazolam), Versed (midazolam), Serax (oxazepam), Librium (chlordiazepoxide), Xanax (alprazolam), Restoril (temazepam), Ativan (lorazepam), Klonopin (clonazepam), Tranxene (clorazepate) Valium (diazepam), Dalmane (flurazepam) | The main use of the short-acting benzodiazepines is in insomnia, while anxiety responds better to medium- to long-acting substances that will be required all day. Benzodiazepines may cause either weight loss or gain and GI distress. Avoid use with sedatives or chamomile. Increased thirst is common. |
| Tricyclic antidepressants: Tofranil (imipramine), Elavil (amitriptyline), Asendin (amoxapine), Doxepin (sinequan) | May cause dry mouth, increase in appetite, weight gain, nausea, vomiting, syndrome of inappropriate antidiuretic hormone (SIADH), constipation, anorexia, or stomatitis. |

- Osteoporosis may be a problem with long-term medication use; monitor carefully (Hummer et al, 2005).
- Breastfeeding mothers should avoid use of medications as much as possible. Should psychiatric medication be necessary, available information regarding the effects of these medications on the infant should be provided.
- Nicotine addiction is much higher in the SCZ population. Cigarette smoke contains many pro-oxidants that contribute directly to oxidative stress. Support efforts to quit, but make sure the patient is aware that psychotic symptoms may be heightened during that time.
- Substance abuse reduces the effectiveness of treatment; amphetamines, cocaine, PCP, and marijuana may make the symptoms of SCZ worse.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.

### *For More Information*

- CATIE study
  http://www.nimh.nih.gov/health/trials/practical/catie/index.shtml
- Mayo Clinic—Schizophrenia
  http://www.mayoclinic.com/health/schizophrenia/DS00196
- Medline—Schizophrenia
  http://www.nlm.nih.gov/medlineplus/schizophrenia.html
- NIMH—Schizophrenia
  http://www.nimh.nih.gov/health/publications/schizophrenia/complete-index.shtml
- Schizophrenia
  http://www.schizophrenia.com/

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# SLEEP AND CIRCADIAN RHYTHM DISORDERS

**NUTRITIONAL ACUITY RANKING: LEVEL 1**

## DEFINITIONS AND BACKGROUND

Primary insomnia is described as difficulty getting to sleep or staying asleep over a time period of at least one month; it occurs in 10% of the population. This equals about 70 million people. The prevalence of insomnia is high among older adults; 44% of older persons experience nighttime symptoms of insomnia at least a few nights per week (National Sleep Foundation, 2009).

Circadian rhythm sleep disorders have wake–sleep cycles differ from the typical pattern. Delayed sleep phase syndrome involves a longer time to get to sleep with periods of alertness during the night. The irregular sleep–wake pattern causes the individual to sleep at irregular times, with wakefulness at night and naps during the day. Other sleep disorders include sleep apnea, where breathing is interrupted during sleep, daytime sleepiness (narcolepsy), and restless legs syndrome.

Sleep affects energy balance. Short sleep duration may be related to high BP, depression and obesity in children and teens (Javaheri et al, 2008; Landhuis et al, 2008; Liem et al, 2008; Shaikh et al, 2008).

MT is an indole formed from L-trytophan that plays a major role in sleep and circadian rhythm (Konturek et al, 2007).

MT is produced primarily in the GI tract, but it is also found in the pineal gland, retina, lens, bone marrow, and skin (Pandi-Perumal et al, 2008). Its production increases with darkness and drops again with light, including artificial lighting. Production also decreases with age.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Sleep disorders with an established genetic basis include fatal familial insomnia, familial advanced sleep-phase syndrome, chronic primary insomnia, and narcolepsy with cataplexy. Recent gene association studies have identified multiple gene mutations in several other sleep disorders.

**Clinical/History**

Height
Weight
BMI
Obesity?

EEG
EMG
Wake (W),
nonrapid eye movement (NREM) sleep
Rapid eye movement (REM) sleep

| | | |
|---|---|---|
| BP | **Lab Work** | Gluc |
| Loud snoring? | $Ca^{++}$, $Mg^{++}$ | Chol (total profile) |
| Restless leg syndrome | $Na^{+}$, $K^{+}$, $Cl^{-}$ | |
| Depression or anxiety | H & H | |
| | Serum Fe | |

## INTERVENTION

### OBJECTIVES

- Improve nutritional status and outcome.
- Calculate energy requirements and promote a weight loss program if obesity is a problem.
- Manage intake of bioactive substances that affect sleep (i.e., caffeine, alcohol).
- Teach principles related to good health: balanced diet without excessive energy intake, adequate physical activity, routine sleeping habits.

### FOOD AND NUTRITION

- Adequate intake of thiamin and other B-complex vitamins, zinc, protein, vitamin A, and any other depleted nutrients will be important.
- An energy controlled plan may be needed. The DASH diet may also be beneficial with hypertension.
- Limit caffeine and alcohol, especially later in the evening.

### Common Drugs Used and Potential Side Effects

- Benzodiazepines are commonly used. They may cause dry mouth or dehydration.
- MT analogues have a rapid onset of action, improve sleep quality, and enhance mood. Agomelatine has 5-HT(2c) antagonist properties that may be used in treating patients with major depression, insomnia, and some other sleep disorders (Pandi-Perumal et al, 2008). Ramelteon is another MT agonist recently approved for long-term treatment of insomnia.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Excessive Bioactive Substance Intake**

***Assessment Data:*** Sleep problems; no hx of medical conditions that cause insomnia; diet hx with high caffeine intake.

***Nutrition Diagnoses (PES):*** Excessive bioactive substance intake (caffeine) related to consumption of 10–12 cups of coffee throughout the day as evidenced by insomnia, chronic fatigue, and heartburn.

***Interventions:*** Education about the need to reduce caffeine intake to promote healthier sleep.

***Monitoring and Evaluation:*** Fewer complaints about heartburn; better sleeping habits and less chronic fatigue; intake of two to three cups of coffee per day and increased intake of other beverages.

- MT, when taken with calcium, acts as an immunostimulator. Because this could aggravate conditions such as rheumatoid arthritis, use these supplements with caution.
- As appropriate, antidepressants or anti-anxiety medications may be prescribed. Side effects vary.
- Pain may cause insomnia. Cancer, arthritis, and other conditions may be present. Adequate pain medication will be needed.

### Herbs, Botanicals, and Supplements

- Valerian is a popular botanical used for insomnia. Side effects may include pruritis, headache, or GI distress.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- CBT and family, group, and self-help therapies are all recommended.
- Many people benefit from relaxation training.
- If the individual is obese, a weight management program, sleep enhancement, and exercise plan will be needed.
- Avoid alcohol, nicotine, and caffeine for 3–4 hours before bedtime.
- Exercise in the afternoon rather than too close to bedtime.

#### *Patient Education—Foodborne Illness*

- Basic food handling and handwashing techniques are important.

#### *For More Information*

- American Academy of Sleep Medicine
  http://www.aasmnet.org/
- Medline—Sleep Disorders
  http://www.nlm.nih.gov/medlineplus/sleepdisorders.html
- National Center on Sleep Disorders Research
  http://www.nhlbi.nih.gov/about/ncsdr/
- National Sleep Foundation
  http://www.sleepfoundation.org
- National Institutes of Health—Sleep Tutorial
  http://www.nlm.nih.gov/medlineplus/tutorials/sleepdisorders/htm/index.htm
- Valerian
  http://ods.od.nih.gov/factsheets/valerian.asp
- Your Guide to Healthy Sleep
  http://www.nhlbi.nih.gov/health/public/sleep/healthy_sleep.pdf

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# SUBSTANCE USE DISORDERS AND ADDICTION

## NUTRITIONAL ACUITY RANKING: LEVEL 1 (WITHDRAWAL/REHABILITATION)

### DEFINITIONS AND BACKGROUND

Substance use often leads to addiction. Addiction is a brain disorder, a chronic disorder with compulsive and relapsing behavior. Three predisposing factors exist: constitutional liability (biochemical), personality factor (psychological vulnerability), and social factors (environmental conditioning). Sensitivity to reward (STR) seems to play a role (Davis et al, 2004). The master "pleasure" molecule of addiction is dopamine (D2 receptor gene). Heroin, amphetamines, marijuana, alcohol, nicotine, and caffeine all trigger the release of dopamine. Abnormalities in the metabolism of dopamine, serotonin, and norepinephrine contribute to substance dependency. In some cases, use of antidepressant medications alleviate the dependency.

Abuse of chemical substances may be chronic or acute and may involve abuse of alcohol, prescription or over-the-counter drugs, or illicit drugs. Initial use of drugs of abuse converge on the mesolimbic dopamine pathway from the ventral tegmental area (VTA); these drugs modulate glutamatergic transmission activating the dopamine neurons, a critical early stage in the development of addiction (Heikkinen et al, 2009; Xiao et al, 2009). Physiological problems that result are definite, specific to the abused substance. Social, emotional, vocational, and legal problems may arise.

Persons with substance dependency tend to have type A personalities and are prone to perfectionism and depression. Substance abusers are codependent, neglecting their own feelings and emotions. EDs and substance disorders may represent different expressions of the same underlying problem, with cognitive dysfunction, use of food or substance to relieve negative affect (anxiety or depression), secretiveness about the problem, and social isolation. Addiction to food may also be a reality; brain circuits can also be deranged with natural rewards such as food (Davis et al, 2004). Overweight individuals may use food as a reward, just as substance abusers use pharmacological substances for a reward for the dopamine-specific part of the brain.

Alcoholism is a chronic relapsing disease that is frequently unrecognized and untreated; approximately one third of patients remain abstinent, and one third are fully relapsed 1 year after withdrawal from alcohol (Oroszi and Goldman, 2004). Many alcoholics are malnourished; nutritional interventions are needed to prevent liver disease. Antioxidants as precursors of endogenous glutathione show promise (Lieber, 2003). Protein calorie malnutrition (PCM) is predictive of mortality in alcoholic liver disease; deficiencies of folate, thiamine, pyridoxine, and vitamin A promote anemia, altered cognitive states, and night blindness (Halsted, 2004).

Addictions share some of the same biological pathways. Because polydrug use may alter food intake, taste preferences, and nutrient metabolism and because denial is common, psychotherapy along with substance withdrawal is recommended. Dysfunctional eating patterns and excessive weight gains have been observed during recovery from drug and alcohol addictions; food deprivation during active addiction may contribute (Cowan and Devine, 2008). Assertive outreach is effective in engaging and linking persons to substance abuse treatment services, even if they are homeless (Fisk et al, 2006). Giving up should not be an option (Table 4-22).

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** The collaborative study on genetics of alcoholism (COGA) supports the premise that alcohol dependence is inherited in 50–60% of cases (Dick et al, 2008). Alcohol dehydrogenases, catechol-O-methyltransferase (COMT), opioid receptors, and HTTLPR (which alters serotonin transport) all affect the process of addiction and relapse (Oroszi and Goldman, 2004). In addition, the gamma-amino-butyric acid (GABA) neurotransmitter mediates actions of alcohol.

| Clinical/History | Lab Work | |
|---|---|---|
| Height | Prolactin levels | Serum folate |
| Weight | Serotonin levels | Serum $B_{12}$ |
| BMI | $Ca^{++}$, $Mg^{++}$ | Serum tHcy |
| Weight changes | $Na^{+}$, $K^{+}$, $Cl^{-}$ | CRP |
| Dietary/intake history | H & H | Liver function tests |
| I & O | Serum Fe | |
| BP | Alb or transthyretin | |
| Tremors, delirium? | Gluc | |
| Multidimensional Addictions and Personality Profile (MAPP) | Chol (total profile) | |
| | Trig (often very high in alcoholics) | |
| | Serum thiamin | |

### INTERVENTION

#### OBJECTIVES

- Normalize brain levels of neurotransmitters.
- Correct fluid and electrolyte imbalances or dehydration.
- Protect during withdrawal (e.g., alcohol detoxification may cause tremors, hallucinations, seizures, and delirium tremens). Of persons with delirium tremens, 20% may die, even with therapy; monitor closely.
- Modify diet for medical conditions. Alcoholics experience problems such as liver failure, cirrhosis, pancreatitis, GI

**TABLE 4-22 Common Addictions and Issues**

| Substance | Issues |
|---|---|
| Alcohol | The most consistent predictor of alcohol dependency is alcoholism in a biological parent. Alcoholics are more likely to diet from stroke or cirrhosis. An estimated 3 million children between 14 years and 17 years of age are problem drinkers; the earlier the exposure, the more likely dependency will occur. To assess for problems, C-A-G-E questions include: Have you tried to cut back? Has anybody ever annoyed you regarding this behavior? Have you ever felt guilty about it? Have you ever needed an early morning eye opener? With two or more yes answers, a problem should be addressed. |
| Caffeine | Caffeine and nicotine are the most common psychostimulant drugs used worldwide. Caffeine affects the brain in ways that are similar to cocaine and other stimulants, but it is not as addictive overall. |
| Chocolate | Chocolate may be used as self-medication for low magnesium levels and to balance low neurotransmitters for mood (serotonin and dopamine). Chocolate contains methylxanthines, biogenic amines, and cannabinoid-like fatty acids. Chocolate cravings may occur (Smit et al, 2004), especially related to menses in women. |
| Club drugs: LSD (acid), MDMA (Ecstasy), GHB, GBL, ketamine (special K), Fentanyl, Rohypnol, amphetamines, methamphetamine | "Club drug" is a vague term that refers to a wide variety of drugs including MDMA (ecstasy), GHB, Rohypnol, ketamine, methamphetamine, and LSD. Uncertainties about the drug sources, pharmacological agents, chemicals used to manufacture them, and possible contaminants make it difficult to determine toxicity, consequences, and symptoms. Serious health problems may result from their use. |
| Cocaine | The pure chemical, cocaine hydrochloride, has been an abused substance for more than 100 years, and coca leaves, the source of cocaine, have been ingested for thousands of years. Use has increased, and now over 1.5 million Americans are users. Young adults aged 18–25 are most likely to initiate use. Years later, cocaine use may be linked to Parkinson's disease.<br>A herbal supplement can reduce the cravings associated with chronic cocaine use. N-acetylcysteine (NAC) is a potential agent to modulate the effects of cocaine addiction, heroin addiction, and possibly alcoholism. |
| Heroin | Heroin is processed from morphine, a naturally occurring substance extracted from the seedpod of the Asian poppy plant. Heroin usually appears as a white or brown powder. Street names for heroin include smack, H, skag, and junk. Other names may refer to types of heroin produced in a specific geographical area, such as Mexican black tar. Use of heroin may be fatal. Use during pregnancy may cause spontaneous abortion. |
| Marijuana | Marijuana is the most commonly used illicit drug in the United States. The main active chemical in marijuana is THC (delta-9-tetrahydrocannabinol). The membranes of certain nerve cells in the brain contain protein receptors that bind to THC. Once in place, THC kicks off a series of cellular reactions that ultimately lead to the high that users experience. |
| Nicotine | Along with directly stimulating the brain's reward system, nicotine stimulates it indirectly by altering the balance of inputs from two types of neurons that help regulate its activity level. |
| Prescription medications | Pain relievers, tranquilizers, stimulants, and sedatives are very useful treatment tools. When people do not take them as directed, they may become addicted. Inappropriate or nonmedical use of prescription medications is a serious public health concern. Nonmedical use of prescription medications like opioids, central nervous system (CNS) depressants, and stimulants can lead to abuse and addiction, which are characterized by compulsive drug seeking and use. |
| Steroids, anabolic | Anabolic-androgenic steroids are man-made substances related to male sex hormones. They are available legally only by prescription to treat conditions that occur when the body produces abnormally low amounts of testosterone, such as in delayed puberty and impotence. They are also prescribed to treat body wasting in patients with AIDS and other diseases that result in loss of lean muscle mass. Athletes may abuse anabolic steroids to enhance performance and also to improve physical appearance. Anabolic steroids are taken orally or injected, typically in cycles of weeks or months (referred to as "cycling"), rather than continuously. |

Sources: http://www.nida.nih.gov/Drugpages/, accessed June 15, 2009; Smit HJ, et al. Methylxanthines are the psycho-pharmacologically active constituents of chocolate. *Psychopharmacology*. 176:412, 2004.

bleeding, esophageal varices, renal impairment, ascites, and edema. Intravenous drug users are at risk for contracting hepatitis C or HIV infection. See appropriate entries.

- Reorient to reality; develop trusting relationships between patient and care providers. Promote abstinence and long-term treatment.
- Improve nutritional status and outcome. Dietitians can provide nutrition education and can help in drug treatment and rehabilitation programs (Grant et al, 2004).
- Prevent or correct EDs, present in approximately 50% of this population. Avoid major changes in food choices and intake during recovery to prevent drastic weight fluctuations.
- Use motivational interviewing to work through problems, such as resistance and ambivalence for making life changes (Westra, 2004).

## FOOD AND NUTRITION

- According to I & O values, adjust fluid intake. Offer beverages that are nonalcoholic favorites. Reductions in the use of caffeine are often suggested.
- Encourage nutrient-dense foods. Fruits, vegetables, whole grains, and fish are important inclusions.
- Adequate intake of protein will be essential.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Excessive Alcohol Intake**

***Assessment Data:*** Food recall/diet history; alcohol intake; assessment of access to food and resources (money, family support, social service agencies, alcohol counseling or therapy).

***PES:*** Excessive alcohol intake related to social anxiety as evidenced by patient report, admission with alcohol intoxication, and history of rehabilitation for alcohol dependence.

***Intervention:*** Provide nourishing foods and beverages. Counsel about the role of nutrient-dense foods and how alcohol provides only heat and kilocals. Coordinate with social services for additional rehab if appropriate.

***Monitoring and Evaluation:*** Report of improved food and beverage intake; alcohol-free lifestyle changes; improved self esteem and quality of life; reduced social anxiety after rehab program.

- Include adequate calories, especially because patients often become hypoglycemic. Feed several times daily to help regulate blood glucose.
- Adequate intake of thiamin, other B-complex vitamins, zinc, protein, vitamin A, or other depleted nutrients is important during recovery.
- Adjust diet, as appropriate, to reduce excess sweets; many chemical abusers tend to substitute CHO for their dependency drug.
- Adequate fiber intake may be useful to correct or prevent constipation.

## Common Drugs Used and Potential Side Effects

- Antabuse, when mixed with alcohol, can cause severe nausea, vomiting, low BP, and flushing.
- Bromocriptine (Parlodel) may also be used for some drug-recovery patients. Nausea, vomiting, or constipation may occur.
- Methadone maintenance therapy continues to be one of the major effective forms of addiction pharmacotherapy and underscores the importance of biological factors in the physiology and treatment of the addictive diseases (Kreek et al, 2004).
- Naltrexone decreases the pleasurable sensation of alcohol; it is used for narcotic dependency after detoxification. Anorexia, weight loss, nausea, vomiting, and abdominal cramping or pain may occur.
- Subutex/Suboxone (buprenorphine/naloxone) are oral tablets used for the treatment of opiate dependence.
- Stool softeners may be beneficial if constipation results after withdrawal, as with cocaine abuse.
- TCAs (imipramine, desipramine) are often beneficial with some side effects such as dry mouth.

## Herbs, Botanicals, and Supplements

- No studies have been conducted for efficacy of herbs or botanicals in substance abuse patients.
- St John's wort should not be taken with antidepressants.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help the patient accept responsibility for his or her own actions. CBT and family, group, and self-help therapies are all recommended.
- Treatment should focus on sufficient duration and intensity, family support, after-care and follow-up, self-help groups, collaboration with social services, and a drug-free lifestyle. One of five individuals will be drug free or sober after 5 years. New studies will include new, effective medications and evaluations of the changes that occur in the brain.
- Help to maintain abstinence. Avoid discussion of unanswerable questions such as "why" substances have been abused.
- In recovery, simple guidelines are useful: eat breakfast and regular meals daily; eat a variety of foods; make mealtimes pleasant and unhurried; choose healthy snacks; drink decaffeinated coffee.
- Discuss issues regarding personal "control." Coping skills will be needed to reduce helplessness. Include patient in decision making to increase self-esteem and confidence.
- Discuss the dangers of diet pills and starvation to control appetite and weight.
- Long-term alcohol abuse can specifically target beta cells of the pancreas, increasing risk of diabetes. Heroin use can cause glucose intolerance, but unlike in alcohol abuse, this usually resolves with abstinence.
- Heavy drinkers tend to have higher total and HDL cholesterol levels than controls. Moderate alcohol intake does not seem to be protective against coronary heart disease through lipid reduction alone; resveratrol may be one protective factor.
- Sports teams can provide peer-led education about healthy lifestyles and reduction in disordered eating patterns or substance abuse (Elliott et al, 2004).
- If a person smokes cigarettes, having smoking cessation interventions seems to help long-term sobriety for other addictions (Prochaska et al, 2004).
- Help plan adequate discharge planning, follow-up, and family therapy or other support group interactions.

### *Patient Education—Foodborne Illness*

- Careful food handling will be important. The same is true for hand washing.

### *For More Information*

- Addictions
  http://www.addiction.com/
- Alcoholics Anonymous
  http://www.alcoholics-anonymous.org
- American Society of Addiction Medicine
  http://www.asam.org/
- National Clearinghouse for Alcohol and Drug Information
  http://ncadi.samhsa.gov/
- National Council on Alcoholism and Drug Dependence
  http://www.ncadd.org
- National Institute on Drug Abuse (NIDA)
  http://www.nida.nih.gov/
- NIDA Statistics
  http://www.nida.nih.gov/drugpages/stats.html

- Recovery Month
  http://www.recoverymonth.gov/
- Substance Abuse and Mental Health Services
  http://www.icpsr.umich.edu/SAMHDA/

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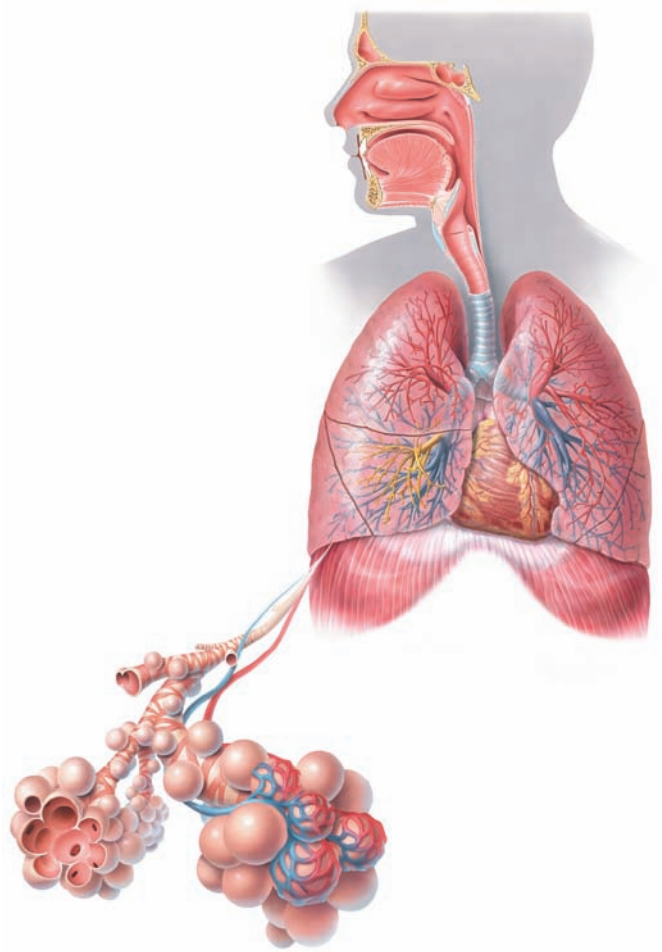
SECTION 5

# Pulmonary Disorders

## CHIEF ASSESSMENT FACTORS

- Altered Respirations
- Anorexia
- Blood Gases: Partial Pressure of Oxygen ($pO_2$); Partial Pressure of Carbon Dioxide ($pCO_2$)
- Clubbing of Nail Beds
- Confusion, Somnolence
- Cough, Especially with Chest Pain
- Cyanosis of Lips, Nail Beds
- Dizziness
- Elevated Blood Pressure (BP)
- Engorged Eye Veins
- Fever or Chills
- Flaring Nostrils; Red, Swollen Nose
- Hemoptysis (Coughing Up Blood)
- Hoarseness
- Orthopnea, Tachypnea
- Pain (Chest, Abdominal)
- Pallor; Ashen or Gray Coloring
- Poor Exercise or Activity Tolerance
- Rapid Breathing, Excessive Perspiration
- Restlessness, Irritability
- Shortness of Breath (Dyspnea)
- Stridor (Crowing Sound on Inhalation)
- Wheezing (Whistling, Musical Sound from Obstructed Airways)

## PULMONARY NUTRITION NOTES



Asset provided by Anatomical Chart Co.

**TABLE 5-1 Causes of Malnutrition in Patients with Pulmonary Disease**

| |
|---|
| Aerophagia and rapid breathing |
| Anemia (low oxygen-carrying capacity) |
| Anorexia of chronic illness |
| Cellular hypoxia |
| Chronic debility |
| Decreased lung immunity |
| Decreased lung surfactant and elasticity |
| Depression, anxiety with anorexia |
| Difficulty in eating with continuous dyspnea |
| Fever |
| Gastric hypomotility |
| Hypermetabolism, as in chronic obstructive pulmonary disease (COPD) |
| Increased mechanical work of breathing |
| Increased workload of the heart |
| Inflammation |
| Lung cancer |
| Malabsorption, as in cystic fibrosis |
| Medications causing nausea and anorexia |
| Pneumonia |
| Polypharmacy |
| Poor respiratory muscle strength and endurance |
| Restricted diet |
| Right-sided heart failure |
| Vitamin deficiency, leading to poor epithelial integrity and weak lung muscles |

NOTE. Death in patients with COPD is typically due to acute respiratory failure, pneumonia, lung cancer, cardiac disease, or pulmonary embolism.
Adapted from: Merck Manual: Chronic obstructive pulmonary disease; Accessed January 28, 2006, at http://www.merck.com/mrkshared/mmg/sec10/ch78/ch78a.jsp.

Pulmonary surfactant is a complex and highly active material composed of lipids and proteins that is found in the fluid lining the alveolar surface of the lungs. It protects the lungs from injuries and infections caused by inhaled particles and micro-organisms (Wright, 2005a). The role for surfactant was first studied in premature infants with respiratory distress syndrome (RDS), which is now routinely treated with an exogenous replacement (Stevens et al, 2004). Biochemical surfactant abnormalities have been described in asthma, bronchiolitis, chronic obstructive pulmonary disease, lung transplantation; infectious and suppurative lung diseases (cystic fibrosis [CF], pneumonia;) adult RDS, pulmonary edema, chronic lung disease of prematurity, interstitial lung diseases. Surfactant replacement therapy has been tested with positive outcomes. In acute respiratory syndrome, exogenous surfactant does not improve survival, but patients who received surfactant had a greater improvement in gas exchange during 24-hour treatment (Spragg et al, 2004).

The evidence for the role of diet in pulmonary disease is clear. Intake of fruit, fish, antioxidant vitamins, fatty acids, sodium or magnesium, helps to alleviate symptoms of asthma and Chronic obstructive pulmonary disease. Because antioxidant nutrients are positively corrected with lung function, vitamin C, vitamin E, beta-carotene, and selenium are important. Flavonoids, such as quercetin and resveratrol, in apples, onions, oranges, berries, and red wine support lung health (Arts and Hollman, 2005; Donnelly et al, 2004; Neuhouser, 2004). Vitamin D helps to maintain healthy lung function (Wright, 2005b). Vitamin E helps to stave off upper respiratory infections; 200 IU daily gives better response to vaccines for diseases such as flu, ear infections, pneumonia, bronchitis, sinusitis, and other pathological conditions (Meydani et al, 2004). Almonds, mango, sunflower seeds, vegetable oils, and whole grains are good sources. Table 5-1 lists factors that contribute to malnutrition with pulmonary disease.

Omega-3 fatty acids can calm inflamed airways; include salmon, respiratory quotients (RQs) tuna, mackerel, walnuts, and flaxseed oil more often. Table 5-2 lists the RQs for fats, protein, and carbohydrates (CHO). In general, it is assumed that fats decrease $CO_2$ output more than CHO.

**TABLE 5-2 Respiratory Quotient (RQ) and Nutrients**

| |
|---|
| RQ = $VO_2/VCO_2$ |
| RQ from fat = 0.7 |
| RQ from protein = 0.8 |
| RQ from carbohydrates (CHO) = 1.0 |

### *For More Information*

- American Lung Association
  http://www.lungusa.org/
- Canadian Lung Association
  http://www.lung.ca/
- National Heart, Lung, and Blood Institute
  http://www.nhlbi.nih.gov/health/public/lung/index.htm
- National Jewish Health
  http://www.nationaljewish.org/healthinfo/index.aspx

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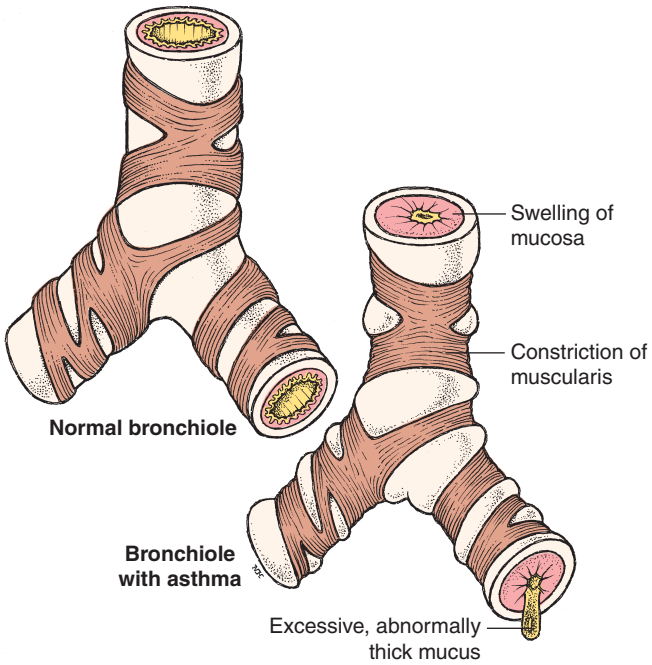
# ASTHMA

## NUTRITIONAL ACUITY RANKING: LEVEL 1




Adapted from: Neil O. Hardy. Wesport, CT. *Stedman's Medical Dictionary,* 27th ed. Baltimore: Lippincott Williams & Wilkins, 2000, p. 158.

## DEFINITIONS AND BACKGROUND

Bronchial asthma involves paroxysmal dyspnea accompanied by wheezing and is caused by spasm of the bronchial tubes or swelling of their mucous membranes. Bronchial asthma differs from wheezing caused by cardiac failure (cardiac asthma), in which an x-ray shows fluid in the lung. Asthma involves inflammation of the lining of the airways, obstruction, and increased sensitivity of the airways. Table 5-3 provides a checklist for signs and symptoms of asthma.

Between 10 and 15 million Americans are affected by asthma, including about 5% of children. Asthma seems to be inherited in two thirds of cases. Two main types of bronchial asthma are recognized: allergic (extrinsic) and nonallergic (intrinsic or infectious). Exercise-induced bronchospasm is much less common.

Children who are exposed to second-hand smoke may have chronic cough or symptoms of asthma. Chronic poor control can lead to a serious condition, status asthmaticus, which generally requires hospitalization and can be life threatening. Brittle asthma is a rare form of asthma with repeated attacks; food intolerance is common. Many infants with wheezing have transient conditions that resolve. The common cold virus and rhinovirus (RV) are major triggers; this pattern continues for adults with allergic asthma (Tan, 2005).

Breastfeeding provides immunological protection when the infant's immune system is immature and a modest protective effect against wheeze in early childhood (Kim et al, 2009; Oddy et al, 2004). Longer duration of breastfeeding seems to be more protective. Supplementation of maternal diet with fish oil is associated with altered neonatal immune responses to allergens (Devereux, 2009). Reduced maternal

**TABLE 5-3 Early Warning Signs of Asthma**

| | |
|---|---|
| Head/eyes | Glassy eyes; dark circles; watery eyes; headache; feverish; pale |
| Nose | Stuffy nose; runny nose; sneezing |
| Mouth/throat | Chin or throat itches; change in sputum; dry mouth; funny feeling in chest |
| Chest/lungs | Fast heartbeat; coughing; changes in breathing; downward trend in peak flow numbers |
| Behavior/mood | Easily upset or irritable; weak; slowing down; feeling sad; more quiet, excited or restless than usual; desire to be alone; insomnia |
| Exercise tolerance | Poor tolerance for exercise; sweaty; easy fatigue |

intake of vitamins D and E, zinc during pregnancy seems to be associated with increased asthma and wheezing outcomes in children up to the age of 5 years (Devereux, 2009).

Nutritional status is important for healthy lungs. Intravenous treatment with multiple nutrients may be of considerable benefit; pulmonary function improves progressively with longer treatment (Schrader, 2004). Diet affects the pathophysiology of asthma by altered immune or antioxidant activity with consequent effects on airway inflammation. Maternal intake of vitamins D and E and zinc can modify fetal lung development (Devereux, 2009). Low serum vitamin D has been shown to be a marker for severity of childhood asthma (Brehm et al, 2009).

Overall, dietary modification may help patients manage their asthma and their overall health. A multidisciplinary approach is required to move forward and understand the complexity of the interaction of dietary factors and asthma (Kim et al, 2009). Obesity and overweight may lead to less-effective therapy from inhaled corticosteroid treatments (Sutherland et al, 2009). While there is currently no conclusive evidence about the role of specific nutrients, food types, or dietary patterns past early childhood on asthma prevalence (Kim et al, 2009); Table 5-4 lists various nutrients and their potential effects on asthma.

**TABLE 5-4 Nutrients and Their Potential Mechanisms in Asthma**

| Nutrient(s) | Activity and Potential Mechanisms of Effect |
|---|---|
| Vitamins A (carotenoids), C, and E | Antioxidants for protection against endogenous and exogenous oxidant inflammation |
| Vitamin C | Prostaglandin inhibition (Harik-Kahn et al, 2004) |
| Vitamin D | Vitamin D modulates T-cell responses (Devereux, 2009) |
| Vitamin E | Membrane stabilization, inhibition of immunoglobulin E (IgE) production. Modulation of T-cell responses (Devereux, 2009) |
| Flavones and flavonoids | Antioxidants; mast cell stabilization |
| Magnesium | Smooth muscle relaxation, mast cell stabilization (Schrader, 2004) |
| Selenium | Antioxidant cofactor in glutathione peroxidase |
| Copper, zinc | Antioxidant cofactors in superoxide dismutase. Zinc modulates T-cell responses (Devereux, 2009) |
| Omega-3 fatty acids | Leukotriene substitution, stabilization of inflammatory cell membranes (Wong, 2005). PUFA modulate T-cell responses (Devereux, 2009) |
| Omega-6 polyunsaturated/trans fatty acids | Increased eicosanoid production (Nagel and Linseisen, 2005) |
| Sodium | Increased smooth muscle contraction; reductions can increase airway responsiveness (Mickleborough and Gotshall, 2004) |

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Tobacco smoke and genetic susceptibility are risk factors for wheezing and asthma (Sadeghnejad et al, 2008). Interleukin 6 (IL6) IL6 receptor (IL6R), and IL13 are candidate genes. Maternal diet plays an epigenetic role by sensitizing fetal airways to respond abnormally to environmental insults (Devereux, 2009; Kim et al, 2009). In addition, beta2-adrenergic receptor (beta2AR) gene polymorphisms are associated with asthma in different racial or ethnic populations.

**Clinical/History**

- Height
- Weight
- Body mass index (BMI)
- BP
- Hypotension?
- Temperature
- Intake and output (I & O)
- Spirometry test
- GERD?
- Respiratory distress
- Audible wheezing
- Decreased breath sounds
- Tachycardia
- Cyanosis
- Anxiety
- Pulmonary edema
- Dehydration
- Hard and dry cough
- Distended neck veins
- Food or sulfite allergies?
- Skin testing

**Lab Work**

- $pCO_2$, $pO_2$
- Glucose (Gluc)
- Albumin (Alb)
- Hemoglobin and hematocrit (H & H)
- Serum Fe, ferritin
- Transferrin
- Serum vitamin $D_3$
- Serum lipids
- Uric acid
- Bilirubin
- $Ca^{++}$, $Mg^{++}$
- Cholesterol (Chol)
- Triglycerides (Trig)
- C-reactive protein (CRP)

## INTERVENTION

### OBJECTIVES

- Prevent distention of stomach from large meals, resulting in distress, GERD, or aggravation of asthma.
- Prevent lung infection and inflammation. Promote improved resistance against infections.
- For allergic asthma, identify and control allergens in the environment.
- Promote adequate hydration to liquefy secretions.
- Optimize nutritional status. Sufficient vitamins C, $B_6$, D, and E, selenium and magnesium are important. Reduce intake of oleic acid, but increase omega-3 fatty acids if tolerated.
- Encourage a health maintenance program, including physical activity where possible.
- Caffeine relaxes muscles and opens the airways; 2–3 cups of coffee daily may be useful in adults.

## SAMPLE NUTRITION CARE PROCESS STEPS

**Overweight**

***Assessment Data:*** BMI >90%tile for age; complaints of heartburn and GERD after meals; asthma triggered by allergies; diet hx showing frequent intake of high sugary snacks between meals.

***Nutrition Diagnoses (PES):*** Overweight related to excessive intake of energy as CHO with asthma as evidenced by BMI >90%tile for age, GERD, and diet hx revealing intake of high CHO snacks throughout the day.

***Interventions:*** Education about the role of weight management and asthma; review of any known food allergies; discussion about appropriate energy intake for age and activity. Counseling about alternative snacks with a mix of protein-CHO-fats.

***Monitoring and Evaluation:*** BMI closer to desirable range; fewer complaints of GERD; better tolerance of drug therapy for asthmatic episodes; improved quality of intake for meals and snacks.

## FOOD AND NUTRITION

- Infants should be exclusively breastfed to reduce the risk of asthma in susceptible families.
- Provide balanced, small meals that are nutrient dense (high-quality protein, vitamins, and minerals), to reduce risk of infections.
- Lose weight by following a lower energy intake if needed (Oddy et al, 2004; Sutherland et al, 2009).
- Encourage extra fluids unless contraindicated. Theobromine in cocoa tends to increase blood flow to the brain and to reduce coughing; use often.
- Use less sodium (Mickleborough and Gotshall, 2004).
- Highlight foods rich in vitamins A and C, magnesium, and zinc. Use more broccoli, grapefruit, oranges, sweet peppers, kiwi, tomato juice, and cauliflower for vitamin C.
- Quercetin in apples, pears, onions, oranges, and berries should be encouraged (5 or more servings per week). Other nutrients that support immunocompetence should be included.
- Omit specific food allergens for children if identified: as milk, eggs, seafood, tree nuts, peanuts, fish, wheat or soy. For adults, tree nuts, peanuts, fish and shellfish allergies tend to persist.
- Sulfites salicylates may aggravate asthma in 5% of this population, especially adults with severe disease. Sulfite-containing foods or beverages should be avoided.
- Salicylate sensitivity is common in 5–20% of asthmatics who are hypersensitive to aspirin. Many fruits and some vegetables contain salicylates.
- If fish is tolerated, consumption of fish two to three times weekly may help reduce leukotriene synthesis (Wong, 2005). If nuts are tolerated, include selenium from Brazil nuts and vitamin E from most nuts.
- Omega-3 fatty acids from fish oils, walnuts, and flaxseed have been suggested. Some studies suggest that EPA is more useful than DHA (Mickleborough et al, 2009) whereas others suggest the opposite (Weldon et al, 2007). The evidence is not yet clear.
- Saturated fatty acids (SFAs) and oleic acid (from margarine) may contribute to clinical onset of asthma; limit their use (Nagel and Linseisen, 2005).

### Common Drugs Used and Potential Side Effects

- An airway renin–angiotensin system is triggered by release of mast-cell renin; ANG II is a critical factor for new therapeutic targets in the management of airway disease (Kano et al, 2008; Reid et al, 2007).
- See Table 5-5.

### Herbs, Botanicals, and Supplements

- Many patients with asthma use alternative therapies. Antioxidant and natural anti-inflammatory and immunomodulatory remedies may prove beneficial.
- In China, a combination of three herbal extracts (ASHMI) may be used in anti-asthma intervention (Wen et al, 2005). Seaweed may be used to treat asthma in Vietnamese and oriental cultures (Dang and Hoang, 2004).
- Dietary fatty acids such as gamma linolenic acid (GLA; borage oil) modulate endogenous inflammatory mediators without side effects (Ziboh et al, 2004).
- Ephedra (ma huang) is an effective bronchodilator, but it increases BP significantly. Problems with blood glucose, arrhythmias, increased heart rate, and central nervous system (CNS) stimulation can also occur. The Food and Drug Administration (FDA) has removed it from the market, but some forms are still available.
- Stinging nettle, licorice, gingko, and anise have not shown efficacy; side effects must be evaluated.
- St. John's wort can inhibit theophylline's effectiveness.

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Mild, chronic asthma can be a warning; if untreated, it can lead to an acute exacerbation.
- Waiting to introduce solids to an infant does not necessarily protect against onset of asthma and allergy (Zutavern et al, 2004).
- Early multivitamin–mineral supplementation may trigger asthma in susceptible children; the exact reasons are not clear (Milner et al, 2004). A healthy, nutrient-dense diet should be consumed instead.
- All medications should be taken as directed by the physician. An emergency pack should be carried at all times containing a rescue inhaler and, if needed, epinephrine injection device and a chewable antihistamine tablet.
- Work with the patient/family to avoid precipitating triggers. Reduce exposure to triggers such as pet dander, food allergens, second-hand smoke. Discuss exercise, rest, and nutrition.
- Massage therapy enhances relaxation, decreases anxiety, and promotes better lung function.

**TABLE 5-5 Medications Used in Asthma**

| Medication | Description |
|---|---|
| Antibiotics | Long-term use can cause diarrhea and other problems. Penicillin should not be taken with fruit juices. |
| Anticholinergics (Atrovent, Combivent) | Quick-relief asthma medications. Dry mouth is common side effect. |
| Beta-agonists (metaproterenol albuterol; levalbuterol; salbumol) | Relaxes smooth muscle around airways. Side effects include shakiness, rapid heart rate, nervousness and elevated blood glucose. Metaproterenol (Metaprel, Alupent) may alter taste and cause nausea or vomiting. Albuterol (Ventolin, Proventil) may have cardiac side effects or may cause nausea or diarrhea. |
| Bronchodilators: theophylline (Theo-Dur, Slo-BID, Slo-Phyllin, Theolair, Uniphyl) | No longer first choice of asthma medication. Nausea, vomiting, and sleeplessness can be a problem Theophylline metabolism is affected by protein and CHO availability; avoid extreme changes in protein and CHO intake. Because it is a methylxanthine, avoid extreme changes in usual intakes of caffeine-containing foods. Theophylline depresses levels of vitamin $B_6$. In addition, lipid levels (cholesterol, HDL, and LDL) are higher in children who take theophylline. |
| Corticosteroids (methylprednisolone [Medrol], Deltasone, Orapred, Prelone) | Many side effects such as fluid retention, low serum potassium, GI distress, retaining excess sodium, causing hyperglycemia, and other problems. Monitor carefully, especially if needed over a long period of time. AeroBid contains an anti-inflammatory steroid and is inhaled; it may cause nausea, vomiting, or diarrhea. Bone mineral density is often decreased after long-term use of inhaled corticosteroids. |
| Epinephrine | May be required for emergencies. Intravenous (IV) administration of epinephrine results in a prolonged increase in resting energy expenditure (REE) as measured by respiratory quotient (RQ); fuel for this is increased CHO oxidation. |
| Expectorants | Potassium iodide may affect existing thyroid problems. |
| Long-term control medications | Anti-immunoglobulin E: Reduces histamine release; may be useful with allergic form of asthma.<br>Combination therapy (Advair): Combining an inhaled corticosteroid and a long-lasting beta$_2$-agonist seems to provide consistent relief for people with asthma.<br>Intal (cromolyn) and Tilade (nedocromil) are inhaled medications useful for asthma triggered by cold weather, exercise, and allergies.<br>Inhaled nasal steroids: AeroBid (flunisolide), Azmacort (triamcinolone), Flovent (fluticasone), Pulmicort (budesonide), and Qvar (beclomethasone HFA). These prevent inflammation and reduce swelling inside airways; they also reduce mucus production.<br>Leukotriene modifiers: Accolate, Singulair, Zyflo. These relax the smooth muscle around the airways and reduce inflammation.<br>Serevent (salmeterol xinafoate), Advair (fluticasone propionate and salmeterol inhalation powder), and Foradil or Tubuhaler Aerolizer (formoterol fumarate); these medications can worsen asthma or cause death. Formoterol may deplete potassium levels and cause heart palpitations. |
| Omega-3 fatty acid supplements | Omega-3 fatty acid supplements may decrease inflammation and improve lung function in adults with asthma, but there is no conclusive evidence. Omega-6 fatty acids tend to increase inflammation and worsen respiratory function. |

### *Patient Education—Foodborne Illness*

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

### *For More Information*

- Allergy and Asthma Advocate
  http://www.aaaai.org/
- Allergy and Asthma Network—Mothers of Asthmatics
  http://www.aanma.org/
- Asthma and Allergy
  http://allergy.healthcentersonline.com/allergyasthmabasics/
- National Asthma Center
  http://www.nationaljewish.org/healthinfo/conditions/asthma/index.aspx
- National Asthma Education and Prevention Program (NAEPP)
  http://aspe.hhs.gov/sp/asthma/
- Salicilate allergy
  http://www.webmd.com/allergies/guide/salicylate-allergy

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# BRONCHIECTASIS

## NUTRITIONAL ACUITY RANKING: LEVEL 1

### DEFINITIONS AND BACKGROUND

Bronchiectasis (BX) is an irreversible widening of portions of the bronchi resulting from damage to the bronchial wall with chronic dilation. It may be present with recurrent bronchitis or pneumonia. The most common acquired cause is acute respiratory illness in patients with COPD. Other causes include measles, whooping cough, tuberculosis (TB), fungal infection, inhaled object, lung tumor, CF, ciliary dyskinesia, immunoglobulin deficiency syndromes, rheumatoid arthritis, ulcerative colitis, human immunodeficiency virus (HIV) infection, and heroin abuse.

BX secondary to primary immunodeficiency in childhood is not always progressive; it is possible to slow or prevent disease progression with appropriate treatment (Haidopoulou et al, 2009). In non-CF BX, airway obstruction deteriorates over time; precaution must be taken to prevent significant morbidity and mortality (Twiss et al, 2006).

Relapse can be controlled with antibiotics, chest physiotherapy, inhaled bronchodilators, proper hydration, and good nutrition. Surgical resection or bilateral lung transplantation may be an option for improving quality of life that has few complications.

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** Congenital BX usually affects infants and children related to problems with lung development in a fetus but is not genetic in origin.

**Clinical/History**

- Height
- Weight
- BMI
- Weight loss?
- Diet history
- BP
- I & O
- Pneumonia?
- Fever?
- Shortness of breath (SOB)
- Fatigue
- Bluish skin or paleness
- Coughing up blood
- Chest x-ray
- Chest high-resolution computed tomography (HRCT)
- Altered respiratory rate
- Chronic cough
- Sputum culture: profuse, foul, or purulent
- Early morning paroxysmal cough
- Decreased breath sounds
- Weight loss, anorexia

**Lab Work**

- Transthyretin
- Retinol-binding protein (RBP)
- Gluc
- $Na^+$, $K^+$
- $Ca^{++}$, $Mg^{++}$
- Chol
- Trig
- H & H
- Serum Fe
- Transferrin
- Blood urea nitrogen (BUN)
- $pO_2$, $pCO_2$

## INTERVENTION

### OBJECTIVES

- Promote recovery and prevent relapse of symptoms. Prevent lung collapse or atelectasis.
- Avoid fatigue associated with mealtimes.
- Prevent or correct dehydration.
- Improve weight status, when necessary.
- Reduce fever and inflammation.
- Support lung function with higher antioxidant intake.
- Prepare patient for surgery if needed.

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Unintentional Weight Loss**

***Assessment Data:*** Fever, anorexia, fatigue, chronic cough with purulent sputum, weight loss of 15 lb in past 2 months.

***Nutrition Diagnoses (PES):*** Unintentional weight loss related to fever, fatigue and poor appetite as evidenced by loss of 15 lb in 2 months.

***Interventions:*** Food and nutrient enhancement through nutrient-dense, energy-rich foods and beverages. Education about recipes and beverages to replace weight that are easy to prepare and consume.

***Monitoring and Evaluation:*** Regain of lost weight; improved appetite; more destable BMI.

## FOOD AND NUTRITION

- Use a diet with 1.0–1.25 g protein/kg and sufficient calories to meet elevated metabolic requirements appropriate for age and sex.
- Small, frequent feedings may be better tolerated.
- Fluid intake of 2–3 L daily may be offered, unless contraindicated.
- Intravenous fat emulsions may be indicated (eicosanoids are inflammatory modulators, and thromboxanes and leukotrienes tend to be potent mediators of inflammation). Omega-3 fatty acids should be enhanced in the oral diet by including salmon, tuna, sardines, walnuts, and flaxseed. Supplements may also be useful.
- Adequate antioxidant use with vitamins C and E and selenium may be beneficial. Ensure adequate potassium intake, depending on medications used.

### Common Drugs Used and Potential Side Effects

- Antibiotics are used if the condition is bacterial. Aerosol administration of high-dose tobramycin in non-CF bronchiectatic patients is safe (Drobnic et al, 2005).
- Expectorants help bring up the mucus. Mucus thinners help make it easier to cough.
- Bronchodilators help open up the airways and corticosteroids help reduce airway swelling and inflammation. Monitor side effects according to the specific drugs used.

### Herbs, Botanicals, and Supplements

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

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the role of nutrition in health and recovery; emphasize quality proteins and nutrient-dense foods, especially if the patient is anorexic.
- A flu shot or pneumonia shot may be needed annually.
- Emphasize fluid intake, perhaps juices or calorie-containing beverages instead of water.
- Discuss desirable sources of fatty acids, such as omega-3 foods.

### *Patient Education—Foodborne Illness*

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

### *For More Information*

- Bronchiectasis
  http://www.lung.ca/diseases/bronchiectasis.html
- Merck Manual
  http://www.merck.com/mmhe/sec04/ch047/ch047a.html

### BRONCHIECTASIS—CITED REFERENCES

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# BRONCHITIS (ACUTE)

## NUTRITIONAL ACUITY RANKING: LEVEL 1

## DEFINITIONS AND BACKGROUND

Bronchitis is caused by inflammation of the air passages. Acute bronchitis is an acute respiratory infection that is manifested by cough and sputum production that lasts for no more than 3 weeks (Braman, 2006). The acute form may follow a cold or other upper respiratory infection, producing hemoptysis, sore throat, nasal discharge, slight fever, cough, and back and muscle pain.

Causes include *Mycoplasma pneumoniae, Chlamydia, or* exposure to strong acids, ammonia, or chlorine fumes, air pollution ozone, or nitrogen dioxide. The chronic form from cigarette smoking and air pollution can produce breathing difficulty, wheezing, blueness, fits of coughing, and sputum production. (See Chronic Obstructive Pulmonary Disease entry.)

Risks for acute bronchitis are much higher in smokers. Mental patients and homeless persons tend to smoke more than other individuals and are at higher risk for acute bronchitis (Himelhoch et al, 2004; Snyder and Eisner, 2004). In addition, smoking has negative consequences for maternal health as well as fetal health during pregnancy; the risk of bronchitis is 15 times higher for smokers than for nonsmokers (Roelands et al, 2009).

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Tachykinins NK receptors, substance P, T lymphocytes, and neurokinin A appear to influence human airway health and susceptibility to bronchitis.

**Clinical/History**

Height
Weight
BMI
I & O
Hydration status
Edema
Productive cough longer than 3 weeks
Green or yellow sputum
Breathing difficulty
Chest x-ray

**Lab Work**

Gluc
$Na^+$, $K^+$
$Ca^{++}$, $Mg^{++}$
Serum lipids (decreased?)
H & H, serum Fe
Alb, transthyretin
Oxygen saturation
CRP

## INTERVENTION

### OBJECTIVES

- Normalize body temperature when there is fever.
- Replenish nutrients used in respiratory distress.
- Prevent complications such as dehydration and otitis media; avoid further infections.
- Allow ample rest before and after feedings.
- Prevent dehydration. Extra fluids are needed.
- Relieve discomfort.
- Support lung function through high antioxidant foods.

### FOOD AND NUTRITION

- Provide a regular or high-calorie diet, specific to the patient's needs.
- If milk gives a sensation of thickening mucus secretions, skim milk may be better tolerated and is important for adequate calcium consumption.

### SAMPLE NUTRITION CARE PROCESS STEPS

**Inadequate Fluid Intake**

***Assessment Data:*** Poor skin turgor, low I & O, frequent cough making drinking difficult, weight loss.

***Nutrition Diagnoses (PES):*** Inadequate fluid intake related to frequent coughing spells and difficulty drinking beverages as evidenced by poor skin turgor, loss of 2 kg fluid, and low I & O.

***Interventions:*** Food and nutrient delivery enhancement with nutrient and calorie-dense beverages such as shakes or nutritional supplements. Education about recipes for calorie-rich beverages.

***Monitoring and Evaluation:*** Improved I & O and skin turgor; improved fluid intake, and recovery of lost weight.

- Provide adequate amounts of vitamins C and E, selenium, and potassium.
- Increase the intake of fluids (2–3 L), unless contraindicated.
- Appropriate fatty acid intake may be beneficial to reduce inflammation.
- A low energy intake may be needed after the acute phase to promote weight loss, improve BMI, and promote a healthier level of respiratory functioning (Canoy et al, 2004).

### Common Drugs Used and Potential Side Effects

- Bronchodilators can cause gastric irritation. They should be taken with milk, food, or an antacid.
- Theophylline can be toxic if a diet high in CHO and low in protein is used. Avoid large amounts of stimulant beverages, namely, coffee, tea, cocoa, and cola, unless the physician permits.
- Use of antibiotics for the treatment of acute bronchitis is not justified (Braman, 2006).

### Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for eucalyptus, mullein, horehound, stinging nettle, or marshmallow.
- Belladonna leaf and root are respiratory antispasmodic agents. They should not be used with tricyclic antidepressants, some antihistamines, phenothiazines, or quinidine. Sedation, dry mouth, and difficult urination may occur.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain to patient that adequate hydration is one of the best ways to liquefy secretions.
- Maintain body weight within a healthy range.
- Promote healthy diet that includes a balance of nutrients, with anti-oxidant-rich foods.

#### *Patient Education—Foodborne Illness*

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

#### *For More Information*

- Medline—Bronchitis
  http://www.nlm.nih.gov/medlineplus/bronchitis.html
- Web-MD—Bronchitis
  http://www.webmd.com/a-to-z-guides/acute-bronchitis-topic-overview

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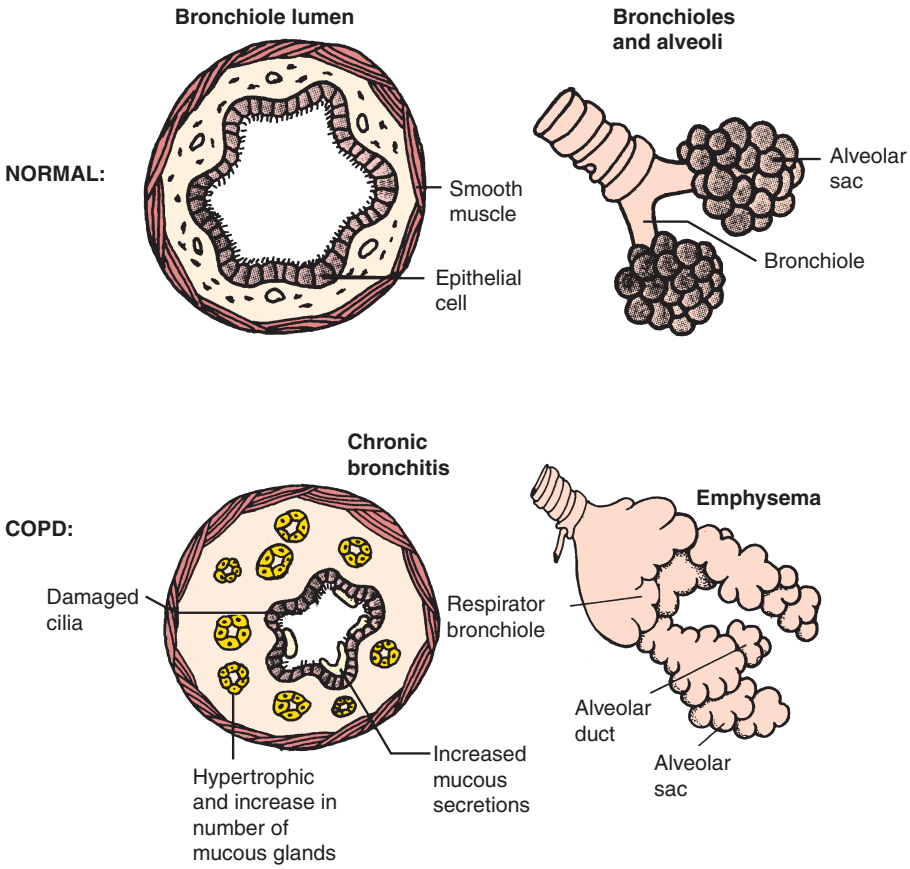
# CHRONIC OBSTRUCTIVE PULMONARY DISEASE

## NUTRITIONAL ACUITY RANKING: LEVEL 3




Adapted from: Nettina, Sandra M., MSN, RN, CS, ANP, *The Lippincott Manual of Nursing Practice,* 7th ed. Lippincott, Williams & Wilkins, 2001.

### DEFINITIONS AND BACKGROUND

Chronic obstructive pulmonary disease (COPD) may result from a history of emphysema, asthma, or chronic bronchitis with persistent lower airway obstruction. COPD is the fourth leading cause of death in the United States. Smoking is the most common cause. According to the Centers for Disease Control and Prevention (CDC), approximately 440,000 persons die each year of a cigarette smoking—attributable illness in the United States. Nonsmoking causes of COPD include alpha-1 antitrypsin deficiency, connective tissue diseases, HIV infection, and some metabolic disorders.

COPD is associated with muscular impairment, nutritional depletion, and systemic inflammation. Symptoms and signs of COPD include dyspnea on exertion, frequent hypoxemia, decreased forced expiratory volume in 1 second ($FEV_1$), and destruction of the alveolar capillary bed. In COPD, total air quantity is blown out much sooner. COPD is a leading cause of death in the United States.

Chronic bronchitis ("blue bloater") patients have inflamed bronchial tubes, excess mucus production, chronic cough (for 3 months each year), SOB, and no weight loss. Cardiac enlargement with failure is common.

Emphysema ("pink puffer") patients have weight loss and thinness without heart failure. It is characterized by tissue destruction, distention, and destruction of pulmonary air spaces by smoking and air pollution. Wheezing, SOB, and chronic mild cough result. Nutritional depletion is significantly greater in patients who have emphysema than in those who have chronic bronchitis. Serious weight loss occurs from anorexia, secondary to significant SOB and gastrointestinal (GI) distress. Malnutrition, tissue wasting, and oxidative stress play a role.

Approximately 75% of patients with COPD suffer from weight loss, where chronic mouth breathing, dyspnea, aerophagia, certain medications, and depression often act in concert. Low body weight or recent weight loss and, in particular, depleted lean body mass (LBM) in patients with COPD are predictors of mortality, outcomes after acute exacerbations, hospital admission rates, and need for mechanical ventilation (Mallampalli, 2004). Risk of respiratory mortality is high.

Elevated resting and activity-related energy expenditure, reduced dietary intake relative to resting energy expenditure, accelerated negative nitrogen balance, medication effects, and an elevated systemic inflammatory response contribute to weight loss (Mallampalli, 2004).

Nutritional supplementation may have a role in the management of COPD when provided as part of an integrated rehabilitation program incorporating a structured exercise component.

The pathological mechanisms of COPD involve neutrophil granulocytes, cytotoxic T cells, macrophages, and mast cells (Ekberg-Jansson et al, 2005). Interventions aimed at controlling cytokine production may be required to reverse cachexia. Starvation, as in anorexia nervosa, can cause emphysema, even without smoking (Coxson et al, 2004).

Recommendations for fats, CHO, proteins, and water must be individualized. For patients with acute exacerbations of COPD in the intensive care unit (ICU), serum total protein is associated with hospital mortality; therefore, protein intake must be carefully monitored (Yang et al, 2004).

Nutritional support is a mainstay of the comprehensive therapeutic approach to patients with COPD because of progressive malnutrition, due to reduced energy intake, increased energy expenditure, and impaired anabolism (Anker et al, 2009).

Fruit and vegetable intake is important and protective. Foods such as meats, vegetables, and coffee may be more bland to the patient than he or she remembers; recognition of this may be important in planning meals.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** Copeptin is a prognostic marker for short-term and long-term prognoses in patients with COPD requiring hospitalization (Stolz et al, 2007). In addition, glutathione-S transferase omega (GSTO) is a candidate gene for COPD (Wilk et al, 2007) and the vitamin D endocrine system is being studied (Janssen et al, 2009).

**Clinical/History**

- Height
- Weight
- BMI
- Diet history
- Temperature
- I & O
- Pulmonary function tests
- Spirometry
- Expiratory airflow limitation
- Excessive mucus production
- Wheezing
- SOB
- Morning headache
- Anorexia, depression
- BP
- Chest x-ray
- Electrocardiogram (ECG)
- Respirations
- Ascites
- Edema

**Lab Work**

- Gluc
- $Na^+$, $K^+$
- $Ca^{++}$, $Mg^{++}$
- Serum Fe
- Hemoglobin
- Hct >48% may reflect chronic hypoxemia
- Serum vitamin $D_3$
- TLC
- pH
- $pO_2$ <50 mm Hg
- $PaCO_2$ >50 mm Hg
- Alb, transthyretin
- BUN
- CRP
- Chol
- Trig

### SAMPLE NUTRITION CARE PROCESS STEPS

**Early Satiety and COPD**

***Assessment:*** Food intake records, weight, preferred foods. Early satiety and problems with coordination of breathing and swallowing.

***Nutrition Diagnosis (PES):*** Involuntary weight loss related to early satiety, problems with breathing and swallowing, and inadequate intake of calorie-dense foods as evidenced by 20-lb weight loss and fatigue at mealtimes.

***Intervention:*** Frequent small meals of easily digested foods with added fats and calorie-dense oral supplements.

***Monitoring and Evaluation:*** Weight changes, improvement in calorie intake, less fatigue while eating.

## INTERVENTION

### OBJECTIVES

- Screen early and correct any malnutrition. Because there is less oxygen available for energy production, the patient is less active, and there is less blood flow to the GI tract and muscles. Malnutrition increases likelihood of infections. Provide medical nutrition therapy (MNT) focusing on prevention and treatment of weight loss and related conditions, especially in underweight patients (ADA, 2009).
- Promote intake of a nutrient-dense diet rich in antioxidant foods.
- Overcome anorexia resulting from slowed peristalsis and digestion. Patient lethargy, poor appetite, and gastric ulceration resulting from inadequate oxygen to the gut.
- Improve ventilation before meals with intermittent positive-pressure breathing and overall physical conditioning to strengthen respiratory muscles. Lessen work efforts by losing excess weight, if needed.
- Prevent respiratory infections or respiratory acidosis from decreased elimination of $CO_2$.
- Alleviate difficulty in chewing or swallowing related to SOB. Patients with COPD have disrupted coordination of breathing and swallowing and may be at risk for aspiration (Gross et al, 2009). Breathe carefully, eat slowly, and rest when the meal is finished.
- Prevent or correct dehydration, which thickens mucus.
- Avoid constipation and prevent straining with defection at stool.
- Avoid distention from large meals or gaseous foods. Eat while sitting up to lessen discomfort.
- Ensure adequate flavor of foods because taste is often minimized (Wardwell et al, 2009).
- Consider nutritional support to prevent progressive weight loss, since restoration of lean and fat body mass may not be achievable (Anker et al, 2009). Medical food supplements should be influenced more by the patient's preference than nutritional factors such as percentage of fat or CHO, as there is limited evidence to support consumption of a particular macronutrient composition (ADA, 2009).
- Assess and help improve quality of life of people with COPD, especially as it relates to their ability to obtain,

prepare, and consume food to meet nutritional needs; impairment with activities of daily living is common (ADA, 2009).

## FOOD AND NUTRITION

- A high-protein/high-calorie diet is necessary to correct malnutrition. Use 1.2–1.7 g protein/kg and sufficient kilocals for anabolism (start with 30–35 kcal/kg, depending on current weight). Use BMI and weight change to assess weight status, body composition, and calorie needs (ADA, 2009).
- A diet without tough or stringy foods and an antireflux regimen are useful. Gas-forming vegetables may cause discomfort for some patients.
- Increased use of omega-3 fatty acids in foods such as salmon, haddock, mackerel, tuna, and other fish sources may be beneficial (ADA, 2009; Romieu et al, 2005).
- Encourage a diet that meets Recommended Dietary Allowances for antioxidant vitamins A, C, and E (ADA, 2009). To enrich the diet with antioxidants, use more citrus fruits, whole grains, and nuts. There is a protective effect of fruit and possibly vitamin E.
- Fluid intake should be high, especially if the patient is febrile. Use 1 mL/kcal as a general rule. This may translate to eight or more cups of fluid daily. For discomfort, consume liquids between meals to increase ability to consume nutrient-dense foods at mealtimes.
- Limit salt intake. Too much sodium can cause fluid retention or peripheral edema, which may interfere with breathing.
- Fiber should be increased gradually, perhaps through use of psyllium, crushed bran, prune juice, or extra fruits and vegetables.
- Use small, concentrated feedings at frequent intervals to lessen fatigue. For example, eggnogs and shakes may be helpful between meals.
- Morning may be the best meal of the day for many patients with COPD. See Tables 5-6 and 5-7 for ways to add extra protein or calories to the diet.
- Parenteral nutrition (PN) is reserved for patients in whom malabsorption has been documented where enteral nutrition has failed (Anker et al, 2009).

### Common Drugs Used and Potential Side Effects

- Bronchodilators (Atrovent, Theo-Dur, etc). are used to liquefy secretions, treat infections, and dilate the bronchi. They can cause gastric irritation and ulceration.
- Antibiotics, steroids, expectorants, antihistamines, diuretics, anticholinergics, and other drugs may be used. Monitor side effects accordingly.
- Oral or parenteral corticosteroids significantly reduce treatment failure and the need for additional medical treatment; adverse drug reactions may occur (Wood-Baker et al, 2005).

### Herbs, Botanicals, and Supplements

- No clinical trials have proven efficacy for use of mullein, camu-camu, licorice, red pepper, peppermint, or eucalyptus.
- Ephedra (ma huang) is an effective bronchodilator, but it increases BP significantly. Avoid taking with digoxin, hypoglycemic agents for diabetes, monoamine oxidase inhibitor (MAOI) antidepressants, antihypertensive medications, oxytocin, theophylline, caffeine, and dexamethasone steroids. Problems with BP, blood glucose, arrhythmias, increased heart rate, and CNS stimulation can occur.
- Vitamin D supplementation may be beneficial to prevent upper respiratory infections (Ginde et al, 2009).

## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Early detection, prevention, and early treatment of involuntary weight loss means putting more emphasis on dietary change (Brug et al, 2004; Weekes et al, 2009). Explain how to concentrate protein and calories in five to six small meals a day rather than three large ones.
- To conserve energy while preparing meals at home, choose foods that are easy to prepare. Try having the main meal early in the day to have more energy later.

**TABLE 5-6 Tips for Adding Calories to a Diet**

| Food | Tip |
|---|---|
| Fats | Butter or margarine, cream, sour cream, gravies, salad dressings, and shortening. Mix butter into hot foods such as soups and vegetables, mashed potatoes, cooked cereals, and rice. Serve hot bread with lots of melted butter. Mayonnaise can be added to salads or sandwiches. Sour cream or yogurt can be used on vegetables such as potatoes, beans, carrots, and squash. Try sour cream or yogurt in gravy or salad dressings for fruit. Whipping cream has 60 kcal per tablespoon; add it to pies, fruits, pudding, hot chocolate, gelatin, eggnog, and other desserts. Fry the entree (e.g., chicken, meat, fish) and sauté vegetables in butter or oil. |
| Sweets | Spread jelly or honey on toast or cereal; mix honey in tea. Add marshmallows to hot chocolate. |
| Snacks | Have snacks ready to eat, such as nuts, dried fruits, candy, buttered popcorn, crackers and cheese, granola, ice cream, and popsicles. |
| Beverages | Drink milk shakes with lots of ice cream added; these will be high in calories and protein. Use sugar-sweetened beverages such as carbonated beverages, coffees with whipped cream and sugar, and sugar-sweetened ades. |

**TABLE 5-7 Tips for Adding Protein to a Diet[a]**

| Food | Tip |
|---|---|
| Meats and meat substitutes | Add diced or ground meat to soups and casseroles. Serve a chef salad with cheese, ham, turkey, and sliced egg. Peanut butter can be spread on crackers, apples, celery, pears, and bananas. Nuts are a good snack with both fat and protein. |
| Dairy products | Add milk powder to hot or cold cereals, scrambled eggs, mashed potatoes, soups, gravies, ground meats (e.g., meat patties, meatballs, meatloaf), casserole dishes, and baked goods. Use milk or half and half instead of water when making soups, cereals, instant puddings, cocoa, and canned soups. Add grated cheese or cheese chunks to sauces, vegetables, soups, casseroles, hot crab dip, and mashed potatoes. Add extra cheese to pizza. Use yogurt as a fruit dip, or add yogurt to sauces and gravies. |
| Milk powder | Add skim milk powder to the regular amount of milk used in recipes or for a beverage. For double-strength milk, add 1 cup of dry powder to 1 quart of fluid milk, let it sit overnight for 286 kcals and 15 g of protein. |
| Beverages | Add protein powder to casseroles, soups, sauces, gravies, milkshakes, and eggnogs. One scoop may have 4 or 5 g of protein, depending on the brand. Some do not stir in as well as others; some dissolve better in hot foods. Buy instant breakfast mixes and use them instead of milk with meals or as snacks; one 8-oz glass provides 280 kcal. Formula products that are high in protein may be useful as supplements with or between meals or with medication pass in an institution. |
| Desserts | Choose dessert recipes that contain egg such as sponge or angel food cake, egg custard, bread pudding, and rice pudding. |

[a]Protein can be added to many foods without having to increase the number of foods eaten.

Encourage slow eating and rest periods before and after meals.
- Encourage the patient to make small, attractive meals.
- Explain that excessively hot or cold foods may cause coughing spells for some individuals.
- Limit fluid intake with meals to decrease early satiety and subsequent decreased food intake.
- Schedule treatments to mobilize mucus (postural drainage, aerosol treatment) 1 hour before and after meals to prevent nausea.
- Improve physical conditioning with planned exercises, especially strengthening exercises and dancing. Consumption of an oral supplement may be beneficial to support exercise.
- If using oxygen, be sure the cannula is worn during and after meals. Eating and digestion require energy and oxygen.
- Maintain a relaxed atmosphere to make meals enjoyable.
- Promote good oral hygiene; periodontal disease is common.
- MNT should be coordinated with the team of clinical professionals to integrate rehabilitative elements into a system of patient self-management and regular exercise (ADA, 2009).

### *Patient Education—Foodborne Illness*

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

### *For More Information*

- AARC—COPD
  http://www.aarc.org/patient_education/tips/copd.html
- American Thoracic Society
  http://www.thoracic.org/
- National Emphysema Treatment Trial (Nett)
  http://www.nhlbi.nih.gov/health/prof/lung/nett/lvrsweb.htm
- Stages of COPD
  http://www.yourlunghealth.org/lung_disease/copd/stages/Stages_of_COPD.pdf
- Your Lung Health
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# CHYLOTHORAX

## NUTRITIONAL ACUITY RANKING: LEVEL 2–4

### DEFINITIONS AND BACKGROUND

Chylothorax involves accumulation of clear lymph (chyle) in the pleural or thoracic space. It may be spontaneous or caused by amyloidosis, congenital chylothorax, coronary artery bypass grafting (CABG), violent vomiting, lymphoma, thoracic cage compression after cardiopulmonary resuscitation (CPR), thoracic duct trauma or surgery, sarcoidosis, or TB. Chylothorax is caused by surgical procedures in about half of all cases (Maldonado et al, 2009).

Chylous effusions look like milk. Since chyle represents direct absorption of fat from the small intestine lacteals, it is rich in triglycerides. Management of chylothorax may include use of total parenteral nutrition (TPN), low-fat enteral nutrition, thoracentesis to remove the chylous fluid, or surgical ligation of the thoracic duct (Suddaby and Schiller, 2004).

In the congenital form, breast milk and/or regular infant feeding formula should be used before proceeding to medium-chain triglyceride (MCT)–rich formula. Surgery may be considered if conservative management fails. Surgery is needed in the care of small babies with massive chylothorax, such as daily output exceeding 50 mL/kg per day (Cleveland et al, 2009).

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** Congenital chylothorax is the leading cause of pleural effusion in newborns but is not genetic in origin.

**Clinical/History**

- Height
- Weight
- BMI
- Weight changes
- Temperature
- I & O
- Lung x-ray
- Pleural fluid analysis for triglyceride >110 mg/dL
- Dyspnea
- Tachypnea
- Decreased breath sounds

**Lab Work**

- Alb, transthyretin
- Chol
- Trig
- TLC (decreased)
- Gluc
- $Ca^{++}$, $Mg^{++}$
- $Na^{+}$, $K^{+}$
- CRP
- BUN, creatinine (Creat)
- $pCO_2$, $pO_2$

### INTERVENTION

#### OBJECTIVES

- Offer continuous chest-tube drainage to decrease pleural chyle.
- Drainage of chyle from the chest or abdomen results in rapid weight loss and profound cachexia. Lessen consequences of a nutritional or immunological nature from drainage (e.g., sepsis, protein–calorie malnutrition, decreased lymphocytes).
- Replace fat, protein, and micronutrient losses from exudates.
- Achieve a positive nitrogen balance.
- Support involvement of a surgical nutrition support team, which is associated with better patient management and a reduction in inappropriate TPN orders (Saalwachter et al, 2004).

#### SAMPLE NUTRITION CARE PROCESS STEPS

**Inappropriate Intake of Types of Fats**

***Assessment Data:*** Chylothorax with high pleural triglyceride levels in infant.

***Nutrition Diagnoses (PES):*** Inappropriate intake of types of fatty acids related to chylothorax as evidenced by pleural triglyceride levels of 120 mg/dL.

***Interventions:*** Use of breast milk or MCT oil formula in the infant.

***Monitoring and Evaluation:*** Reduced pleural triglycerides and signs of chylohorax.

#### FOOD AND NUTRITION

- Decrease enteral fat intake for patients who are tube fed. For patients who are fed orally, reduce total fat intake until condition is resolved; also for these patients, a low-fat diet may be used alone or with an elemental product.
- Some patients may be able to tolerate a low long-chain fatty acid formula given as a tube feeding (TF) (Cormack et al, 2004).
- For patients without sepsis, TPN may be indicated; care is needed to avoid aggravating the condition.
- Replace exudate losses of nutrients such as vitamin A and zinc. Check serum levels and replace with higher levels of the recommended intakes if necessary.

#### Common Drugs Used and Potential Side Effects

- Octreotide (Sandostatin) may be given as conservative medical management (Paget-Brown et al, 2006; Suver et al, 2004). Nausea, vomiting, abdominal pain, diarrhea, and flatulence can occur. Use with the low-fat diet to decrease GI side effects.

- Medications are given, as appropriate, for the etiology. Monitor side effects accordingly, especially in conditions such as TB or cancer in which numerous side effects are created from drug therapies.
- Bronchodilators may be used. Some nausea and vomiting may occur.

### Herbs, Botanicals, and Supplements

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

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss the importance of adequate nutrition in recovery.
- Discuss interventions that are appropriate for the conditions and diagnoses involved.

#### *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/med/topic381.htm
- Medscape
  http://emedicine.medscape.com/article/172527-overview

### CHYLOTHORAX—CITED REFERENCES


Cleveland K, et al. Massive chylothorax in small babies. *J Pediatr Surg.* 44:546, 2009.

Cormack BE, et al. Use of Monogen for pediatric postoperative chylothorax. *Ann Thorac Surg.* 77:301, 2004.

Maldonado F, et al. Pleural fluid characteristics of chylothorax. *Mayo Clin Proc.* 84:129, 2009.

Paget-Brown A, et al. The use of octreotide to treat congenital chylothorax. *J Pediatr Surg.* 41:845, 2006.

Saalwachter AR, et al. A nutrition support team led by general surgeons decreases inappropriate use of total parenteral nutrition on a surgical service. *Am Surg.* 70:1107, 2004.

Suddaby EC, Schiller S. Management of chylothorax in children. *Pediatr Nurs.* 30:290, 2004.

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# COR PULMONALE

## NUTRITIONAL ACUITY RANKING: LEVEL 2–4

### DEFINITIONS AND BACKGROUND

Acute cor pulmonale (right ventricular failure) occurs when relevant increases in pulmonary vascular resistance overwhelm compensatory mechanisms. Cor pulmonale may be acute, subacute, or chronic. The acute form is generally caused by acute respiratory failure (RF) or pulmonary embolism. A heart disease that follows disease of the lung (such as end-stage emphysema, silicosis), chronic cor pulmonale creates hypertrophy and eventual failure.

The body secretes B-type natriuretic peptide (BNP) from the cardiac ventricles in response to ventricular stretch and pressure overload; this counteracts vasoconstriction that occurs as a compensatory mechanism (Prahash and Lynch, 2004).

Long-term exposure to combustion-related fine particulate air pollution is a risk factor. Pulmonary hypertension (PH) and cor pulmonale may affect patients with COPD or CF. Children who have Prader-Willi syndrome may also experience obesity-related cor pulmonale (Stevenson et al, 2004).

### ASSESSMENT, MONITORING, AND EVALUATION

#### CLINICAL INDICATORS

**Genetic Markers:** Genes important in early lung development are also important in determining adult risk for COPD and its consequences (Bush, 2008). Glucocorticoid resistance may be related to cytokines, excessive activation of the transcription factor activator protein 1, reduced histone deacetylase-2 (HDAC2) expression, and increased P-glycoprotein-mediated drug efflux (Barnes and Adcock, 2009).

| Clinical/History | | |
|---|---|---|
| Height | Diet history | Echocardiography |
| Weight | I & O | BP (hypertension?) |
| BMI | Edema of feet, ankles | |
| Obesity? | Chest x-ray | |

| | | |
|---|---|---|
| Right upper quadrant (RUQ) pain | Clubbing of fingers and toes | $Na^+$ |
| SOB | Sleep apnea? | $K^+$ |
| Distended neck veins | Cyanosis | $Ca^{++}$, $Mg^{++}$ |
| Hypoxia | Hepatomegaly | H & H |
| Wheezing, cough | **Lab Work** | Serum Fe |
| Fatigue, weakness | BNP | BUN, Creat |
| | Alb | $pCO_2$ (increased) |
| | | $pO_2$ (decreased) |
| | | CRP |

## INTERVENTION

### OBJECTIVES

- Improve the patient's capacity to eat meals without straining the diaphragm.
- Correct malnourished status but avoid weight gain that stresses the heart.
- Reduce or prevent fluid retention and edema to lessen cardiac workload.
- Prevent additional damage to cardiac and respiratory tissues.
- Improve energy levels and stamina. Oxygen may be needed, even at mealtimes.
- Support adequate lung function with higher antioxidant intake.

### FOOD AND NUTRITION

- Recommend small, frequent meals or oral supplements rather than three large meals (Anker et al, 2006).
- Use a nutrient-dense diet with concentrated protein sources. Double-strength milk, foods with milk powder added to them, high-calorie supplements, and addition of extra gravies or sauces to meals are useful when quantity of food must be kept minimal because of dyspnea.
- Restrict sodium or adjust fluid restriction as needed.
- Use foods that reduce likelihood of gastric irritation and reflux. For example, use low-acidic fruits, vegetables, and juices.
- Provide adequate potassium and magnesium intake with the Dietary Approaches to Stop Hypertension (DASH) diet.
- Include adequate levels of vitamins C, D, and E and selenium for antioxidant properties (Barnes and Adcock, 2009).
- Control CHO if needed. Insulin resistance is also common (Zamanian et al, 2009).
- Oral nutritional supplements or TF enables nutritional intake to be maintained or increased when usual intake is inadequate (Anker et al, 2006).

### SAMPLE NUTRITION CARE PROCESS STEPS

**Excessive Sodium Intake**

***Assessment Data:*** Diet history revealing intake of 8–10 g sodium daily, low intake of potassium, calcium and magnesium; ankle and foot edema; SOB.

***Nutrition Diagnoses (PES):*** Excessive sodium intake related to dietary habits and long-term hypertension as evidenced by BP 212/100, elevated BNP, and dietary intake low in potassium, calcium and magnesium.

***Interventions:*** Offer DASH diet education and alter diet to enhance fruits and vegetables and low-fat dairy products. Monitor sodium intake and offer alternatives for recipes and menu planning. Counsel about dining out and traveling. Evaluate medications for nutritional side effects.

***Monitoring and Evaluation:*** Improved BP and fewer incidents of SOB; alleviation of edema. Improved BNP levels and fewer signs of heart failure.

### Common Drugs Used and Potential Side Effects

- Bosentan or sildenafil may be given by mouth
- Calcium channel blockers and anticoagulants may be used. Monitor specific medicines for side effects.
- Thiazide diuretics can cause potassium depletion.
- Anabolic pharmacotherapy has the potential to improve nutritional status and function (Anker et al, 2006).
- To reverse glucocorticoid resistance, vitamin D may restore interleukin-10 response and use of antioxidants may be recommended (Barnes and Adcock, 2009).

### Herbs, Botanicals, and Supplements

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

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Plan small, attractive meals that are nutrient dense. If fluid and sodium must be limited, provide tips.
- Recommend snacks that are nutrient dense and protein-rich but do not provide excessive sodium.
- Emphasize the importance of eating slowly to reduce SOB.
- Weight loss may be needed (Olson and Zwillich, 2005).
- Monitor heart murmurs in children to identify potential risks or need for surgery. If untreated, cor pulmonale can lead to right-sided heart failure and death.

#### *Patient Education—Foodborne Illness*

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

#### *For More Information*

- Medline
  http://www.nlm.nih.gov/MEDLINEPLUS/ency/article/000129.htm
- Merck Manual
  http://www.merck.com/mmpe/sec07/ch074/ch074c.html

## COR PULMONALE—CITED REFERENCES

Anker SD, et al. ESPEN Guidelines on Enteral Nutrition: cardiology and pulmonology. *Clin Nutr.* 25:311, 2006.
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Prahash A, Lynch T. B-type natriuretic peptide: a diagnostic, prognostic, and therapeutic tool in heart failure. *Am J Crit Care.* 13:46, 2004.
Stevenson DA, et al. Unexpected death and critical illness in Prader-Willi syndrome: report of ten individuals. *Am J Med Genet A.* 124:158, 2004.
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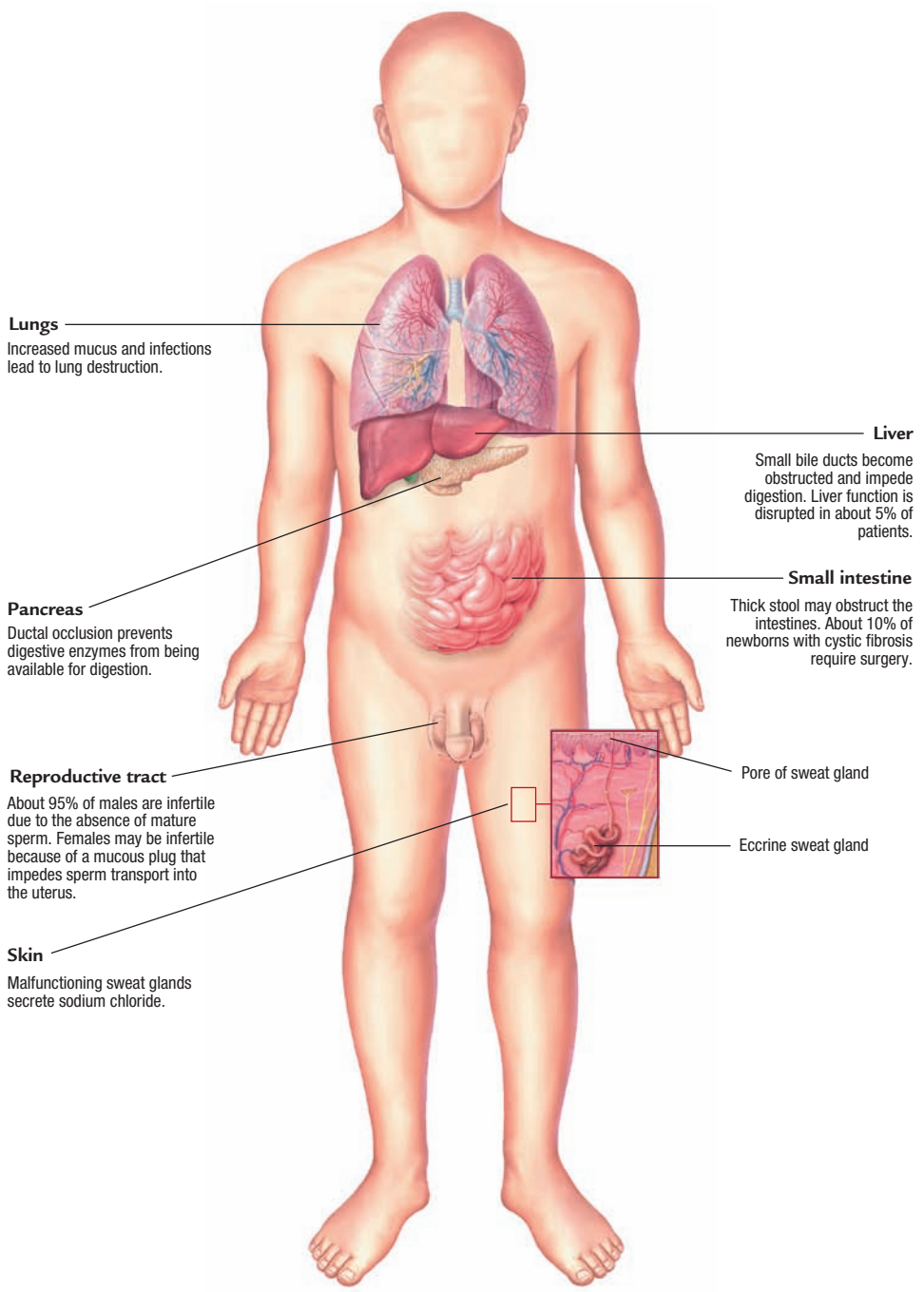
# CYSTIC FIBROSIS

## NUTRITIONAL ACUITY RANKING: LEVEL 3

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## DEFINITIONS AND BACKGROUND

CF is a life-limiting, autosomal recessive inherited disease characterized by thick mucus and frequent pulmonary infections. There is general dysfunction of mucus-producing exocrine glands; high levels of sodium and chloride in the saliva, tears, and sweat; and highly viscous secretions in the pancreas, bronchi, bile ducts, and small intestine. Meconium ileus is a classic sign in newborn infants with CF; it is thicker than usual and passes more slowly.

CF affects approximately 30,000 children and adults in the United States. About one in 3200 Caucasians is affected; 2–5% of Caucasians carry the CF gene. The majority of CF patients have been diagnosed by age 3, but about 10% are not diagnosed until age 18 or older. The median life expectancy for CF patients is 33 years.

The CFTR system controls the efflux of physiologically important anions, such as glutathione (GSH) and bicarbonate, as well as chloride (Hudson, 2004). Interleukin-8 and cytokines also play a role in CF (Augarten et al, 2004). Anti-inflammatory and antioxidant treatments are recommended, including use of omega-3 fatty acids and selenium (Innes et al, 2007). Foods rich in lecithin, choline, betaine, and DHA can safely be recommended to reduce the effects of oxidative stress in CF.

The percentage of CF children who are malnourished varies; weight-based indicators greatly underestimate the extent. A link has been established between the degree of malnutrition and the severity of the disease. Inadequate intake, malabsorption, and increased energy requirements are common. Careful follow-up, better knowledge of energy requirements, dietary counseling, and nutritional intervention help optimize the growth of these patients.

A major goal is to maintain a good nutritional status because it improves long-term survival. Early diagnosis of CF and aggressive nutritional therapy are important to prevent growth failure and malnutrition (Farrell et al, 2005). When appropriate, lung transplantation may be considered.

Pancreatic insufficiency occurs in 80–90% of CF patients; 85% show growth retardation. Intestinal malabsorption is severe in virtually all people who have CF. Deficiency of pancreatic enzymes, bicarbonate deficiency, abnormalities of bile salts and mucosal transport, and anatomical structural changes are relevant. Appropriate pancreatic replacement therapy, combined with pharmacotherapy to address increased acidity of the intestines, achieves near-normal absorption in many patients.

Decreased bone density and increased risk of fractures are seen in patients with CF. Nutrition problems, hypogonadism, inactivity, corticosteroid use, and cytokines may contribute to the low bone mass. Treatment may include calcium, vitamin $D_3$, vitamin K, bisphosphonates, and exercise.

Diabetes may also occur in persons with CF (more commonly in older individuals), reflecting impairment of beta-cell function, which is probably genetically determined. Onset of CF-related diabetes (CFRD) is often associated with a decline in health and nutritional status. Energy requirements may be higher than usual for patients with CF. Microvascular complications are common in CFRD; microalbuminuria is a sensitive indicator of progression to diabetic nephropathy in non-CF diabetes, but it is less sensitive for CF patients (Dobson et al, 2005).

Some patients are diagnosed in adulthood; patients diagnosed as adults differ distinctly from long-term CF survivors diagnosed as children (Nick and Rodman, 2005). While respiratory symptoms are not as severe and prognosis is more favorable, pancreatitis is more common (Nick and Rodman, 2005).

Progressive pulmonary disease associated with chronic bacterial infection and inflammation is the major cause of morbidity and mortality; CRP and IgG levels are indicators of severity (Levy et al, 2007). With anemia from chronic inflammation, treat the underlying inflammation rather than using supplemental iron (Fischer et al, 2007).

Overall, patients with CF who receive optimal nutrition have better growth, maintain better nutritional reserves, and have better pulmonary function than patients with CF who have poor nutrition (Hart et al, 2004). Metabolic and immunological response to infection and the increased work of breathing escalate calorie requirements.

Research supports the potential benefits of gene therapy; compacted DNA is used to get healthy genes into CF cells. Lung transplantation may be needed. Other treatments include use of antibiotics for infections and inhaled medicines to open the airways. Because no single strategy works for every patient, close monitoring of growth, symptoms, and changes in respiratory status must occur. The American Dietetic Association recommends a minimum of 4 MNT visits for patients who have CF.

## ASSESSMENT, MONITORING, AND EVALUATION

### CLINICAL INDICATORS

**Genetic Markers:** CF is genetic, inherited when both parents are carriers of the CF gene. There are hundreds of gene mutations, so each person's symptoms will be unique. The CF transmembrane conductance regulator (CFTR) is an important molecule for chloride that affects sodium transport, fluid, and ion management. CFTR is also expressed in the neurons of the human spinal cord.

**Clinical/History**

- Height, weight
- Growth chart for height and weight
- BMI
- Diet history
- Foul smelling stools
- pH
- Chest x-ray or CT scan
- Pulmonary function test
- DEXA scan
- Upper GI or small bowel series

**Lab Work**

- Pilocarpine iontophoresis sweat test (>60 mEq/L)
- $pCO_2$, $pO_2$
- Chol, Trig (elevated?)
- $Na^+$, $K^+$, $CL^-$
- Alb
- H & H
- Serum Fe, ferritin
- Pancreatic enzymes (amylase, lipase)
- White blood cell count (WBC)
- Prothrombin time (PT)
- International normalized ratio (INR)
- Serum vitamin K