results of the Diabetes Control and Complications Trial (DCCT) and other trials. More than 65% of people with diabetes die from heart disease or stroke (American Diabetes Association, 2009). Diabetes is the chief cause of blindness, renal failure, and amputations and is a leading cause of birth defects.

Intensive therapy to achieve near-normal glucose levels results in lower onset and progression of complications. Caution is needed to prevent hypoglycemia, especially in the very young (<6-year old) and those with vision loss or kidney disease. Fasting blood glucose should be measured three to eight times daily when on a new insulin regimen; this may be tapered down when stable (American Dietetic Association, 2009).

Protein and nutrient metabolism are affected by insulin availability. The long-term effects of diets high in protein and low in carbohydrate (CHO) are unknown at this time. Vitamin and mineral intakes are being studied for their various impacts on blood glucose levels. In addition, it is important to note that antioxidant foods and spices may be helpful in lowering blood glucose levels.

Individuals using insulin should eat at consistent times synchronized with the time-action of the insulin preparation used, monitor their blood glucose levels, and determine their required insulin doses for the amount of food usually eaten. Intensified insulin therapy, including multiple daily injections, continuous subcutaneous insulin infusion with an insulin pump, and rapid-acting insulin, allows for more flexibility in the timing of meals and snacks, as well as in the amount of food eaten.

Medical costs for patients with diabetes account for a significant percentage of all health care costs. National Standards for Diabetes Self-Management have been written to support a team approach to patient care; they include the RD as an essential team member. Ongoing nutrition self-management education includes assessment, care plans, treatment goals, desired outcomes, and monitoring metabolic parameters such as blood glucose, lipids, A1c, and related lab values.

The use of evidence-based MNT can lead to effective lifestyle changes. Use of technology to track quarterly weight and lab values is an effective way to help patients with their self-management goals (Chima et al, 2005). Multidisciplinary team interventions are suggested to help improve patients' A1c levels and to reduce complications, hospital readmissions, and hospital stays. The American Dietetic Association recommends a specific number of MNT visits to promote the desired outcome (see Table 9-6).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The pathogenesis of most DM is multifactorial including both genetic and environmental factors. Interleukin, HLA-DR or HLA-DQ alleles, INS-23, and vitamin D receptor (VDR) gene polymorphisms may be associated with risk of developing T1DM. Much more research is needed to clarify how these present in various haplotypes or ethnic groups.

Hormonal imbalance controls key proteins that regulate the folate-SAM-homocyteine pathways. Poor folate status has been associated with endothelial dysfunction in adolescents with type 1 diabetes (Wiltshire et al, 2008). The methylene-tetrahydrofolate reductase (MTHFR) A>C genotype may confer protection against early nephropathy with lower homocysteine (tHcy) levels, whereas MTHFR 677 TT genotypes may have earlier onset of retinopathy (Wiltshire et al, 2008).

Clinical/History	Lab Work	
Height	HbA1c (goal <7%)	High-density lipoprotein (HDL)
Weight	Fasting blood glucose (FBG) (goal 90–130 mg/dL)	Low-density lipoprotein (LDL)
Weight:height percentiles	OGTT results	Triglycerides (Trig)
Body mass index (BMI)	Urine glucose	Urinary ketones
Diet history	Blood urea nitrogen (BUN)	Na^+, K^+
Waist circumference	Creatinine (Creat)	Ca^{++}, Mg^{++}
Visual acuity	Microalbuminuria	Serum phosphorous (PO_4)
Blood pressure (BP)	C-reactive protein (CRP)	Thyroid-stimulating hormone (TSH)
Intake and output (I & O)	Total Cholesterol (Chol)	Serum D_3 level
Absence of menstruation?		
Increased hunger, thirst, urination		
Nausea, vomiting, abdominal pain		

INTERVENTION

OBJECTIVES

- Individualize MNT as needed to achieve treatment goals, preferably provided by an RD familiar with the components of diabetes MNT (American Diabetes Association, 2009).

TABLE 9-6 Recommended Medical Nutrition Therapy Visits for Type 1 Diabetes

Encounter	Length of Contact	Time between Encounters
1	60–90 minutes	2–4 weeks
2, 3	30–45 minutes	2–4 weeks
4, 5	30–45 minutes	6–12 months
6, 7, 8	30–45 minutes	As indicated by clinical data and/or changes in medication

Adapted from: National Guideline Clearinghouse. Nutrition practice guidelines for type 1 and type 2 diabetes mellitus. Web site accessed September 20, 2009 at http://guidelines.gov/summary/summary.aspx?doc_id=12816&nbr=006618

SAMPLE NUTRITION CARE PROCESS STEPS

Altered Nutrition Labs-Type 1 Diabetes

Assessment Data: Blood glucose log and food history.

Nutrition Diagnosis (PES): Altered nutrition-related lab values related to wrong dose of insulin as evidenced by blood glucose readings consistently ≥250 mg/dL before lunch.

Intervention: Education and counseling to address appropriate timing of food and insulin dose; CHO counting.

Monitoring and Evaluation: Ask patient to fax blood glucose logs for next 3 days.

- Address the metabolic abnormalities of glucose, lipids, and BP (Kulkarni, 2006). Meet the specific needs of the patient; modify drug therapy to enhance outcomes and quality of life.
- Regularly evaluate food/nutrition history, physical exercise, activity patterns, excessive weight gain.
- Develop food/meal planning with client; share plan with medical team so an insulin regimen can be integrated into the client's usual lifestyle. In hospital settings, avoid use of restrictive diets.
- Promote lifestyle changes according to client readiness, educational, and skill level. Early referral for lifestyle changes and advice yields the most benefit (Kulkarni, 2006).
- Plan meal plan, exercise, and medication to achieve blood glucose and lipid goals. Minimize intake of trans fatty acids; saturated fat should be <7% of total calories (American Diabetes Association, 2009).
- Prevent early onset of complications by controlling glucose, lipids, and BP. If there are complications, delay or prevent consequences. The DCCT intensive therapy group had substantially lower complication rates; fewer than 1% became blind, required kidney replacement, or had an amputation because of diabetes (DCCT Research Group, 2009).
- Both the amount (grams) of carbohydrate as well as the type of carbohydrate in a food influence blood glucose level. Monitoring total grams of carbohydrate remains a key strategy in achieving glycemic control (American Diabetes Association, 2009).
- Teach individuals how to use carbohydrate counting and how to adjust insulin doses based on planned carbohydrate intake.
- Promote self-monitoring of blood glucose (SMBG) multiple times per day, and more often during illness.

FOOD AND NUTRITION

- A meal plan based on the individual's usual food intake should be used as the basis for integrating insulin therapy into the usual eating and exercise patterns (American Diabetes Association, 2009).
- Choose a variety of heart-healthy foods, including an average of five servings of fruits and vegetables, six servings of grains (three whole grain), and two servings of low-fat dairy. Foods in the meat and fat groups do not directly affect blood glucose.
- Discourage meal skipping.
- Determine appropriate energy intake for age. Pregnant and growing individuals should receive more: sedentary, 25 kcal/kg; normal, 30 kcal/kg; undernourished or active, 45–50 kcal/kg. Reassess as activity or life stage changes.
- Monounsaturated fatty acids and carbohydrates combined should provide about 60–70% of daily energy intake; limit saturated fats to <10% of energy intake. Use a plan that includes CHO at 45–65% of total energy intake each day to prevent ketosis (American Diabetes Association, 2009).
- Apply carbohydrate counting, dietary guidelines, or MyPyramid food guidance principles. Focus on carbohydrates more than total energy or source of carbohydrate.
- Determine insulin to carbohydrate ratios for each individual. One CHO unit equals 15 g CHO (standard starch, fruit, sweet, or milk servings are based on 15 g CHO).
- Provide consistent carbohydrate with each meal and snack with set doses of insulin. Each of the following portions is one carbohydrate choice (15 g CHO):
  - Grains, breads, cereals: 1 oz bread (1 slice bread, 1/4 large bagel, 6″ tortilla); 1/2 cup cooked dried beans; 1/3 cup pasta or rice; 1 cup soup; 3/4 cup cold cereal; 1/2 cup cooked cereal.
  - Milk and yogurt: 1 cup milk; 2/3 cup unsweetened yogurt (6 oz) or sweetened with noncaloric sweetener.
  - Fruits: 1 small fresh fruit; 1/2 cup fruit; 1 cup melon or berries; 1/2 cup fruit juice; 1/4 cup dried fruit.
  - Sweets and snack foods: 3/4 oz snack food (pretzels, chips, 4–6 crackers); 1 oz sweet snack (two small sandwich cookies, five vanilla wafers); 1 tbsp sugar or honey; 1/2 cup ice cream.
  - Vegetables: 1/2 cup potato, peas, or corn; 3 cups raw vegetables; 1½ cups cooked vegetables; small portions of nonstarchy vegetables.
- Most women need about 3–4 carbohydrate choices (45–60 g CHO) at each meal; men generally need about 4–5 carbohydrate choices (60–75 g CHO) per meal. Active young women may need 2500 kcal, or about 75–90 g CHO/meal; an active young man who needs 3000 kcal/d may need 90–100 g CHO/meal. Use 1–2 carbohydrate choices (15–30 g CHO) for snacks; body size and activity level will determine the number of choices needed.
- Include plenty of fiber from rice, beans, vegetables, barley, oat bran, fruits, and vegetables. Recommendations for fiber intake for people with diabetes are similar to the recommendations for the general public (DRI: 14 g/1000 kcal) and diets containing 44–50 g of fiber daily truly improve glycemia (American Dietetic Association, 2009) Whole grains are good sources of vitamin E, fiber, and magnesium.
- To reduce the risk of nephropathy, protein intake should be limited to the recommended dietary allowance (American Diabetes Association, 2009). If there is microalbuminuria, a more controlled protein intake may be required.
- Cut down or eliminate fried and creamed foods. Include omega-3 fatty acids (as from salmon, mackerel, tuna, walnuts, and canola oil) to control blood lipids and reduce inflammatory processes. A high–monounsaturated fat diet seems to have a favorable effect on lipoproteins in diabetes.
- For minerals, assure adequacy of intake; routine supplementation is not advised. Replenish potassium and magnesium, if needed. Adequate calcium is important: 500 mg in 1–3 year olds, 800 mg in 4–8 year olds, 1300 mg

in 9–18 year olds, and 1000 mg in adults should be attained daily.
- Sodium intake should be limited to 2400 mg daily or less.
- Routine supplementation with antioxidant vitamins E and C and beta-carotene is not advised because of lack of evidence of efficacy (American Diabetes Association, 2009). Folate is important in women of childbearing ages; acquire 400 μg prepregnancy and 600 μg during pregnancy.
- Vitamin D has been found to play an important role in auto-immune disorders. Encourage intake of vitamin-D fortified foods, but also encourage adequate time in the sun.
- If foods containing sucrose are chosen, they should be substituted for other carbohydrate foods. Sucrose intakes of 10–35% of total energy intake do not affect on glycemic or lipid responses negatively when substituted for isocaloric amounts of starch (American Dietetic Association, 2009).
- If adults with diabetes choose to use alcohol, intake should be limited to 1 drink/d or less for adult women and 2 drinks/d or less for adult men. One drink is defined as 12 oz beer, 5 oz wine, or 1.5 oz distilled spirits (American Diabetes Association, 2009). To avoid hypoglycemia, alcohol should be consumed with a carbohydrate-containing food. Abstain in pregnancy, pancreatitis, advanced neuropathy, extremely elevated triglycerides, or a history of alcohol abuse.
- Nonnutritive sweeteners are safe when consumed within the acceptable daily intake levels established by the Food and Drug Administration (FDA) (American Diabetes Association, 2009). The FDA has approved use of several sweeteners; see Table 9-7.
- Adequate carbohydrate replacement during and after exercise is important to prevent hypoglycemia. Decrease rapid-acting insulin doses during the physical activity; 30–50% less is reasonable.
- In critical illness, blood glucose management is a challenge. Enteral feeding is generally preferred but specialty formulas are not usually required. Regular blood glucose monitoring and insulin replacement may be needed.
- If parenteral nutrition is needed, use strict blood glucose control. Hyperglycemia reflects illness severity and results in deleterious consequences. Plan 30% of nutrient intake as fat, 50% as CHO, and 15–20% as protein unless other disease states require alternative plans.

Common Drugs Used and Potential Side Effects

- Anti-CD3 mAbs may prolong beta cell function up to 2 years in patients with new onset Type 1 diabetes (T1DM) but studies are still ongoing (Herold et al, 2009).
- Insulin/diet correlation is essential. Persons with T1DM are usually dependent on insulin for life. The primary potential effect is hypoglycemia. Sources of insulin must be noted because they affect the peak times and duration of effect, and some affect the speed of absorption. Human insulin is produced synthetically from Escherichia coli or yeast with identical amino acid sequence to the human insulin. See Table 9-8 for insulin onset, peak, and duration times.
- Insulin pumps (continuous subcutaneous insulin infusion [CSII]) may be good to maintain glucose levels. If a pump is used, meal schedules are not as strict but should not be abused. With multiple daily insulin injections (MDI), test blood glucose before meals to determine insulin doses. Insulin pens available are convenient for insulin dosing. Insulin glargine (Lantus) is well tolerated in MDI regimens for pediatric patients with T1DM and may be more efficacious than NPH/Lente in those with elevated A1 c (White et al, 2009; Chase et al, 2008).
- Insulin analogs match postprandial blood glucose excursions better than traditional insulin. Bolus analogs are used for more rapid onset to cover meal CHO and to correct hyperglycemia. Basal agents release at a more constant rate to cover between-meal glucose needs or the body's basal insulin needs. Premixed insulins are discouraged except when patients are not able to be compliant with MDI. Adverse effects include the potential for hypoglycemia, especially when HbA1c is less than 7%. Analog insulins are less likely to lead to hypoglycemia, especially overnight hypoglycemia. Weight gain with use of insulin can be counteracted with attention to food intake and physical activity.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician. There is still insufficient evidence to draw definitive conclusions about the efficacy of beans, peanuts, onion, *Coccinia indica*, aloe vera, *Momordica charantia*, and bitter gourd.
- Adequate Vitamin D should be available, especially for bone health (Svoren et al, 2009).
- See Table 9-9 for guidance on specific herbs and supplements in diabetes. The American Diabetes Association also has a book on herbs and supplements for diabetes care, available from the Web site.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The RD should assess food intake (focusing on carbohydrate), medication, metabolic control (glycemia, lipids, and BP), anthropometric measurements, and physical activity to serve as the basis for implementation of the nutrition prescription, goals, and intervention that are tailored for the individual (American Dietetic Association, 2009).
- Diabetes self-management education (DSME) is an essential element of diabetes care, and national standards have been based on evidence. DSME helps patients optimize metabolic control, prevent and manage complications, and maximize quality of life in a cost-effective manner (American Diabetes Association, 2009). Learning can lead to behavioral changes (healthy eating, being active, taking medications, monitoring glucose, problem-solving) which may then lead to desired clinical and health outcomes. Overall, improved health status includes better quality of life, fewer days lost from school or work, fewer complications and health care costs. Weekly problem-based, self-management support interventions can yield health benefits (Tang et al, 2005), as can even a single session with a dietitian (Gaetke et al, 2006).
- Teach patient about the importance of self-care, optimal functioning, the roles of carbohydrate intake and physical activity in maintaining metabolic control. Blood glucose testing is essential. Insulin injections must be given at planned, regular intervals for persons with T1DM.

TABLE 9-7 Sugar and Sweetener Summary

The percentage of calories from carbohydrate (CHO) in the diet for diabetes will vary; it is individualized based on eating habits and glucose and lipid goals.

Glycemic load (GL)	GL = glycemic index × available CHO amount	Although various starches do have different glycemic responses, first priority should be given to the total amount of CHO consumed rather than the source of the CHO. If the use of glycemic index (GI) is proposed as a method of meal planning, the RD should advise on the conflicting evidence of effectiveness of this strategy. Studies comparing high versus low GI diets report mixed effects on A1C.
Sweets	Sugar equivalents (teapoons)	Soft drink, 12 oz (10 tsp) Chocolate bar, 1.5 oz (5.5 tsp) Froot-loop cereal, 1 cup (3.5 tsp) Sweet pickle, 1 oz (2.25 tsp) Catsup, 1 Tbsp (1 tsp) Barbequed chips, 1 oz. (1/2 tsp) Unsweetened cereal, 1 cup (1/3 tsp)
Nutritive Sweeteners		
Sugar (sucrose)	Sucrose = 16 kcal/tsp (4 g CHO)	Sucrose is saccharose ($C_{12}H_{22}O_{11}$). Sucrose is a disaccharide; that is, it is made up of two monosaccharides—glucose and fructose. It is one of the sweetest of sugars. Sucrose-containing foods should be substituted for other carbohydrate foods. Sucrose intakes of 10–35% of total energy intake do not have a negative effect on glycemic or lipid responses when substituted for isocaloric amounts of starch.
Corn syrup, molasses, dextrose, glucose, and maltose	Corn sugar (called glucose, or dextrose), milk sugar (lactose), and malt sugar (maltose).	If sucrose is taken as a standard of 1, the sweetness of glucose is 0.5–0.6, that of lactose is 0.27, and that of maltose is 0.6 There is no evidence that foods sweetened with these sweeteners have any significant advantage or disadvantage over foods sweetened with sucrose in decreasing total calories or CHO content of the diet or in improving overall diabetes control.
Fructose and honey	Fructose: 11 kcal/tsp (3 g CHO); fruit sugar; 1.1–2 times as sweet as sucrose.	Fructose, found in fruits and honey, is the sweetest. Although dietary fructose produces a smaller rise in plasma glucose than equal amounts of sucrose and most starches, it is not to replace more than 20% of calories in adults because of its potential adverse effects on lipids, especially triglycerides. There is no reason to recommend that people with diabetes avoid consumption of fruits and vegetables, in which fructose occurs naturally. In children, fructose works well as a substitute in baked goods because their lipids are not yet a problem and fructose has little impact on blood glucose levels.
Sugar alcohols	Sugar alcohols: 2 kcal/g (50% of kilocalories of other nutritive sweeteners such as sucrose) Sorbitol: 50% as sweet as sucrose Xylitol: 16 kcal/tsp (4 g CHO)	Sugar alcohols produce a lower postprandial glucose response than sucrose or glucose and have lower available energy. With foods containing sugar alcohols, subtraction of one half of sugar alcohol grams from total CHO grams is appropriate, particularly when using the CHO counting method for meal planning. There is no evidence that the amounts of sugar alcohol likely to be consumed will result in significant reduction in energy intake or long-term improvement in glycemia. The use of sugar alcohols appears to be safe. Excessive amounts of polyols may have a laxative effect. The calories and CHO content from all nutritive sweeteners must be accounted for in the meal plan and have the potential to affect blood glucose levels.

(continued)

TABLE 9-7 Sugar and Sweetener Summary *(continued)*

Non-Nutritive Sweeteners

Acesulfame potassium (Sunette®) **Alitame** **Aspartame (Equal®, NutraSweet®)** **Neotame** **Saccharin (Sweet 'N Low®)** **Sucralose (Splenda®)**	Acesulfame: 200 times sweeter than sugar; heat stable Alitame is made from amino acids, 2000 times sweeter than sugar. Still awaiting FDA approval. Aspartame: 180 times sweeter than sugar; 4 kcal/tsp; contains phenylalanine and cannot be used in PKU. Equally contains aspartame, dextrose, and maltodextrin. Neotame: 6000 times sweeter than sugar; heat stable Saccharin: 300–400 times sweeter than sugar; heat stable; leaves a slightly bitter aftertaste Sucralose: 600 times sweeter than sugar; heat stable	The Food and Drug Administration has approved five non-nutritive sweeteners for use in the United States. All have undergone rigorous scrutiny and have been shown to be safe when consumed by the public, including people with diabetes and women who are pregnant. If persons with diabetes choose to consume products containing U.S. Food and Drug Administration (FDA)-approved non-nutritive sweeteners, at levels that do not exceed the acceptable daily intakes (ADIs), the RD should advise that some of these products may contain energy and carbohydrate from other sources that needs to be accounted for. Research on non-nutritive sweeteners reports no effect on changes in glycemic response.

For more information: International Food Information Council, Web site accessed September 22, 2009, at http://www.ific.org/publications/factsheets/lcsfs.cfm; American Diabetes Association, 2009; National Diabetes Evidence-based guidelines, Web site accessed September 20, 2009, at http://guidelines.gov/summary/summary.aspx?doc_id=12816&nbr=006618

- Identify potential or real obstacles; discuss options for resisting temptations, dining away from home, feeling deprived, time pressures, food offers from others, competing priorities, handling social events, family or social support, and food intolerances.
- Encourage regular mealtimes and snacks. Children and teens in particular may need planned snacks.
- A nondiet approach to diabetes management encourages regular eating according to actual hunger and making gradual changes for healthier eating.
- Discuss visual assessment of portions. Practice with measuring cups, spoons, and scales.
- Teach patient how to read labels and how to identify carbohydrates in processed foods.
- Empowerment is important; help patients to gain mastery over their affairs and to effect change (Funnell et al, 2005). Discuss emotional eating, such as from boredom, anger, frustration, loneliness, and depression; alternative choices should be designed from the client perspective. Research reports sustained improvements in A1 c at 12 months and longer with long-term follow-up encounters with an RD (American Dietetic Association, 2009).
- Discuss the weight gain that occurs with intensive insulin therapy and improved glucose control.
- Low-carbohydrate diets are not recommended for the management of diabetes (American Diabetes Association, 2009).

TABLE 9-8 Insulin Onset, Peaks, and Duration[a]

Insulin Type	Begins Working	Peaks at	Ends Working in	Low Occurs at
Rapid, Bolus				
Humalog (Lispro)	15–20 minutes	30–90 minutes	3–4 hours	2–4 hours
Novolog (Aspart)	15–20 minutes	40–50 minutes	3–4 hours	2–4 hours
Short-Acting				
Regular (Novolin R, Humulin R)	30–60 minutes	80–120 minutes	4–6 hours	3–7 hours
Intermediate-Acting				
NPH	2–4 hours	6–10 hours	14–16 hours	6–12 hours
Lente	3–4 hours	6–12 hours	16–18 hours	7–14 hours
Ultralente	4–6 hours	10–16 hours	18–20 hours	12–24 hours
Long-Acting				
Lantus (Glargine)	2–3 hours	Almost no peak; flat action throughout duration	18–26 hours	4–24 hours

[a]Insulin can be used after 1 month if stored at cooler than 86°C and out of direct sunlight or heat. Unopened bottles of insulin should be stored in a refrigerator; do not freeze insulin. Syringes may be prefilled and stored in a refrigerator for up to 3 weeks. Roll the syringes before use to mix the insulin.
Adapted from: North Coast Medical Center, accessed September 22, 2009, at http://www.northcoastmed.com/insulin.htm; and Medscape, accessed September 22, 2009, at http://img.medscape.com/pi/emed/ckb/endocrinology/116364-138562-117853-118003.jpg

TABLE 9-9 Herbs and Supplements in Diabetes Management

Aloe vera	Studies in Japan found that the phytosterols in aloe vera might play a role in lowering blood glucose levels. Further research is needed.
Alpha lipoic acid (thitoic acid)	Alpha lipoic acid (thioctic acid) may have some potential benefits. It is found in the mitochondria and seems to have antioxidant properties that protect vitamin C, vitamin E, and glutathione. Natural sources include red meat, yeast, potatoes, and spinach. Supplementation may provide protection against cataracts, neuropathy in diabetes, cardiovascular disease; research is needed.
Antioxidants	Antioxidants from food should include good sources of beta carotene, vitamins C and E, selenium and zinc.
Bilberry	Bilberry contains anthocyanosides that counteract cellular damage to the retina. Mild drowsiness and skin rashes have been noted.
Biotin	Biotin shows preliminary evidence of being useful for controlling blood glucose.
Bitter melon (*Momordica charantia*)	Bitter melon has traditionally been used as a remedy for lowering blood glucose.
Chromium	Chromium enhances use of insulin. Skin allergies, renal toxicity, and altered iron and zinc absorption can occur. There are no proven benefits if a patient is not deficient (American Diabetes Association, 2009).
Cinnamon	Doses of 1, 3, or 6 g capsules of cinnamon daily lowered blood glucose levels in individuals with diabetes.
Essential oils	Essential oils from cinnamon, cumin, and oregano may enhance insulin sensitivity. Studies are ongoing.
Evening primrose oil	Evening primrose oil may prevent or limit neuropathy due to gamma linoleic acid, an essential fatty acid. It may cause headache and gastrointestinal distress.
Fenugreek	Fenugreek may lower glucose, triglyceride and cholesterol levels due to its psyllium content. Note that it is part of the peanut family and may cause allergic reactions. Do not take with monoamine oxidase (MAO) inhibitors; it has the potential for drug interactions with other medications as well.
Gamma linolenic acid	Evening primrose oil may be useful for preventing neuropathy in diabetes.
Garlic	Garlic may lower blood glucose levels if used in large amounts; more studies are needed.
Gingko biloba	Gingko biloba may help control neuropathy by maintaining integrity of blood vessels and reducing stickiness of blood and clotting. It has some antioxidant properties. Avoid taking with warfarin, aspirin, and other anticoagulant drugs. Headache and interactions with other drugs can occur.
Ginseng, American	Ginseng (American) may lower blood glucose levels. Avoid with warfarin, aspirin, MAO inhibitors, caffeine, antipsychotics, insulin, and oral hypoglycemics because of fluctuations in blood glucose levels, bleeding, platelet functioning, blood pressure and heart rate. Avoid taking with steroids. Headache, insomnia, nausea, or menstrual difficulties can occur. Take with a meal.
Guar gum	Guar gum is a fiber that normalizes the moisture content of the stool, absorbs excess liquid in diarrhea, softens the stool in constipation, and decreases the amount of cholesterol and glucose absorbed from the stomach and intestines.
Gymnema sylvestre	*Gymnema sylvestre* is a hypoglycemic herb. It is highly potent and should be used only under doctor's supervision because it may change insulin requirements.
Magnesium	Magnesium is needed for hundreds of biochemical reactions; it helps regulate blood sugar levels and blood pressure. Some studies suggest that low magnesium levels may worsen blood glucose control in type 2 diabetes. Magnesium supplementation may help with insulin resistance, but avoid high doses.
Turmeric	Turmeric, the rhizome of *Curcuma*, may decrease blood glucose levels.
Vanadum	Vanadium may lower glucose levels. It has been associated with cancer cell growth and can be toxic at therapeutic levels.
Vitamin B_6	Some studies suggest that an adequate intake of vitamin B_6 (pyrodoxine) can helo with both T1DM and T2DM,
Vitamin D	Supplementation of up to 2000 IU daily in infants were less likely to develop type 1 diabetes over the next 30 years in Finland. This vitamin has a strong, protective effect. Experts are now recommending supplementation at the upper end of the current recommendations (i.e., 1000 IU).
Zinc	Zinc is part of the production and storage of insulin in the body. Fresh oysters, ginger root, lamb, pecans, split peas, egg yolk, rye, beef liver, lima beans, almonds, walnuts, sardines, chicken, and buckwheat are sources from the diet. Zinc should not be used with immunosuppressants, tetracycline, ciprofloxacin, or levofloxacin because of potential antagonist effects.

- Discuss the risks for CVD and how to manage them (American Diabetes Association, 2009; American Dietetic Association, 2009).
- Eating disorders often occur in young women with diabetes; refer for psychotherapy if needed.
- A1 c is often unacceptably high in adolescents; there is a need to more fully assess and understand factors such as race/ethnicity, education, and socioeconomic status on treatment recommendations (Paris et al, 2009).
- For sick days: Patients may require more insulin when ill. Liquid diets should provide 200 g CHO in equally divided amounts at mealtime and snacks; liquids should not be sugar free.
- For surgery: Blood glucose should be in good control; perioperative hyperglycemia can be managed with doses of short-acting insulin. Correct abnormalities before surgery when possible.
- Discuss reasonable use of the glycemic index (see Table 9-10).

TABLE 9-10 Glycemic Index and Glycemic Load

Glycemic index (GI) is a measure of serum glucose response to a food relative to a reference food that contains equal amounts of carbohydrate. It does not refer directly to quantified food exchanges. Glycemic load = GI × available carbohydrate amount.

A mixed diet yields varying results on blood glucose levels. Choosing low-GI foods in place of conventional or high-GI foods has a small, clinically useful effect on medium-term glycemic control in patients with diabetes (Brand-Miller et al, 2003).

Carbohydrate Choices

Choose low-GI carbohydrates most often because they digest slower and will be less likely to elevate blood glucose. Each choice = 15 g of carbohydrate. Choose desired number of carbohydrate choices per meal or as needed by the individual. Children and teens, as well as young adults and athletes, will need much more per meal than sedentary adults.

Glycemic Index	Grain or Bean Choices	Fruit Choices	Vegetable Choices
Low (best choice)	Barley, 1/3 cup Beans (kidney, pinto, etc.), 1/2 cup cooked Lima beans, 2/3 cup Muesli, 1/4 cup Oat bran, 1/3 cup	Apple, orange, or pear Cherries, 12 Grapes, 17 Grapefruit, 1/2 Cantaloupe or berries (blueberries, raspberries, strawberries, etc.) = 1 cup	Corn, 1/2 cup Squash, acorn, 1 cup cooked Peas, green or lentils, 1/2 cup cooked 1½ cups cooked or 3 cups raw vegetables (asparagus, beets, broccoli, carrots, cucumbers, mushrooms, onions, peppers, tomatoes, zucchini, and others)
Medium (choose less often)	Baked beans, 1/4 cup Bran flakes, 1/2 cup Bread, 100% whole wheat, 1 slice English muffin, whole wheat, 1/2 Granola, low sugar, 1/4 cup Hamburger or hot dog bun, 1/2 Oatmeal, old fashioned, 1/2 cup Pasta, 1/3 cup cooked Raisin bran, 1/3 cup Rice, converted or wild, 1/3 cup cooked Special K, 3/4 cup Tortilla, 6″	Banana, 1 small Canned fruit, drained, 1/2 cup Dried fruit, 1/4 cup Watermelon, 1¼ cups Raisins, 2 tbsp Orange juice, 1/2 cup Apple juice, 1/2 cup Grape juice, 1/3 cup	Potato, small, “new,” 1/2 cup Sweet potato, 1/2 cup cooked
High (choose rarely)	Bagel, large, 1/4 bagel Bread, white, 1 slice Cream of wheat, 1/2 cup cooked Cheerios, 3/4 cup Corn flakes, 3/4 cup Instant oatmeal, 1/2 cup cooked Pancake, 4″ Rice Krispies, 3/4 cup Rice, white, 1/3 cup cooked Shredded wheat, 1/3 cup Waffle, 4″		Potato, small russet, 3″ diameter Potato, mashed, 1/2 cup

(continued)

TABLE 9-10 Glycemic Index and Glycemic Load *(continued)*

Glycemic Index	Combination Foods	Sweets/Snacks	Milk/Yogurt/Milk Substitutes
Low	Soup, cream, low fat, 1 cup Chili, low fat, 1/2 cup	Ice cream, low fat, 1/2 cup Pudding, sugar free, 1/2 cup Chocolate candy, 15 kisses or 1-oz bar	Milk, skim or low fat (1%), 1 cup Yogurt, low fat, artificially sweetened, 1 cup
Medium	Burrito, bean, flour tortilla, 7″ long = 3 carb choices Pasta dish, 1/2 cup	Popcorn, microwave light, popped, 3 cups Cookie, 3″ across, 1 Muffin, small, 1/2	Soy milk, low fat or nonfat, 1 cup Yogurt, low fat, sweetened, with fruit, 1/3 cup
High	Pizza, thin crust, medium, 1 slice (= 2 carb choices) Pizza, thick crust, medium, 1 slice (= 3 carb choices)	Pretzel twists, mini, 15 pieces Brownie or cake, no frosting, 2″ square Doughnut, 3″, 1/2 doughnut Granola bar, 1 Potato or tortilla chips, 15 chips Syrup, 1 tbsp	

Source: Brand-Miller et al, 2003.

- Aim for 30 min/d of physical activity to burn a minimum of 1500 kcal/wk (see Table 9-11).

Patient Education—Foodborne Illness

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

For More Information

- American Dietetic Association: Type 1 Diabetes Evidence-Based Guidelines for Practice
  http://www.eatright.org/cps/rde/xchg/ada/hs.xsl/home_21231_ENU_HTML.htm
- American Diabetes Association–T1DM
  http://www.diabetes.org/type-1-diabetes.jsp
- Juvenile Diabetes Research Foundation
  http://www.jdrf.org/index.cfm?page_id=101982
- Mayo Clinic – T1DM
  http://www.mayoclinic.com/health/type-1-diabetes/DS00329

TABLE 9-11 American Diabetes Association General Guidelines for Regulating Exercise

Metabolic control before exercise	Avoid exercise if ketosis is present. Use caution if glucose levels are >300 mg/dL without ketosis.
	Ingest added carbohydrate if glucose levels are <100 mg/dL.
Blood glucose monitoring before and after exercise	Identify when changes in insulin or carbohydrate are necessary.
	Learn the glycemic response to different exercise conditions.
Food intake	Consume added carbohydrate, as needed, to avoid hypoglycemia.
	Carbohydrate-based foods should be readily available during and after exercise.

- Medline Plus
  http://www.nlm.nih.gov/medlineplus/ency/article/000305.htm

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ISLET CELL TRANSPLANTATION

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

Some native pancreatic beta cell function persists even years after disease onset in most type 1 diabetic patients (Liu et al, 2009). However, the destruction of beta cells leads to permanent insulin dependence. Islet cell transplantation can lead to insulin independence with excellent metabolic control. Islet transplantation can require more than one donor pancreas to achieve insulin independence (Rickels et al, 2005). Transplants may be performed by a radiologist, who uses x rays and ultrasound to guide placement of a catheter through the upper abdomen and into the portal vein, where islets are slowly infused. If this is not feasible, a surgeon may perform the procedure under anesthesia.

The acute posttransplantation phase lasts up to 2 months; the chronic phase starts after 2 months. Drugs are needed to prevent rejection. Islet cell programs try to avoid using large doses of glucocorticoids. Most protocols are designed to use tacrolimus and sirolimus so that high-dose glucocorticoids would not have to be used.

During the acute period of care, there is the issue that patients are variably managed with insulin therapy to allow for recovery of the islets after they have been placed into their new and not totally adequate environment in the liver. Most centers treat patients for up to a month, and treating hypoglycemia during this time is a concern.

Long-term complications may include hyperlipidemia. In T1DM, the release of many hormones, not only from beta-cells, but also from adipocytes (adipokines) is altered (Stadler et al, 2009). After successful pancreas–kidney transplantation (PKTx), T1DM patients can revert to a nondiabetic metabolism, but altered adipokines are still present after PKTx; adipokines include visfatin, retinol-binding protein-4 (RBP-4), adiponectin, and high-molecular-weight (HMW) adiponectin (Stadler et al, 2009). More research in this area is needed.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Transplantation may occur for T1DM, where there is a genetic component.

Clinical/History

- Height
- Dry weight, present weight
- BMI
- Diet history
- I & O
- BP
- Temperature

Lab Work

- Albumin (Alb), transthyretin
- HbA1c
- Alkaline phosphatase (Alk phos)
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)
- Bilirubin
- Na^+, K^+
- Hemoglobin and hematocrit (H & H)
- Serum Fe
- Serum folacin
- BUN, Creat
- Ca^{++}, Mg^{++}
- White blood cell (WBC) count
- Total lymphocyte (TLC) count
- Glucose (Gluc)
- Chol, Trig
- CRP
- Nitrogen (N) balance
- Glomerular filtration rate (GFR)

INTERVENTION

OBJECTIVES

- Meet the specific needs of the patient; modify drug therapy to enhance outcomes and quality of life.
- Prevent infection and promote healing.
- Monitor for abnormal electrolyte levels.
- Monitor CHO intolerance but make sure that diet provides enough CHO to spare proteins. Treatment goals should reflect those of the American Diabetes Association; adjust as new evidence suggests.
- Alleviate rejection episodes.
- Control infections, especially during the acute phase. Support protein intake to prevent additional infections.
- Force fluids unless contraindicated, as in retention. Match fluid output.
- Help patient adjust to a lifelong medical regimen during chronic phase. Improve survival rate by supporting immune response.
- Correct or manage complications that occur.
- Control weight gain in the first year after transplantation.

SAMPLE NUTRITION CARE PROCESS STEPS

Poor Nutrition Quality of Life

Assessment Data: Weight, labs, nutritional history prior to transplantation, statement of "not liking to eat any more because of the pain."

Nutrition Diagnosis (PES): Poor nutritional quality of life related to abdominal pain after meals as evidenced by weight changes, vacillating lab results and statements about not enjoying meals.

Intervention: Food and Nutrient Delivery—gradual weaning from nutritional support back to oral diet, using small portions of favorite foods. Education—how the transplant would eventually allow life without insulin shots and more normal meal experiences.

Monitoring and Evaluation: Tolerance after weaning from nutrition support to oral diet. Acceptance of several favorite foods, and trying new foods with assistance of family members.

TABLE 9-12 Medications Used after Islet Cell Transplantation

Medication	Description
Cyclosporine	Cyclosporine does not cause retention of sodium as much as corticosteroids do. Intravenous doses are more effective than oral doses. Nausea, vomiting, and diarrhea are common side effects. Hyperlipidemia, hyperglycemia, and hyperkalemia may also occur; decrease fat intake as well as sodium and potassium if necessary. Magnesium may need to be replaced. The drug is also nephrotoxic; a controlled renal diet may be beneficial. Take omega-3 fatty acids to reduce toxic side effects such as high blood pressure and kidney damage. Avoid St. John's wort.
Diuretics	Diuretics such as furosemide (Lasix) may cause hypokalemia. Aldactone actually spares potassium; monitor drug changes closely. In general, avoid use with fenugreek, yohimbe, and ginkgo.
Immunosuppressants	Immunosuppressants such as muromonab (Orthoclone OKT3) and antithymocyte globulin (ATG) are less nephrotoxic than cyclosporine but can cause nausea, anorexia, diarrhea, and vomiting. Monitor carefully. Fever and stomatitis also may occur; alter diet as needed.
Insulin	Insulin may be necessary during periods of hyperglycemia. Monitor for hypoglycemic symptoms during use; teach patient self-management tips.
Pancreatic enzymes	Pancreatic enzymes may be needed if pancreatitis occurs after transplantation.
Tacrolimus (Prograf, FK506)	Tacrolimus suppresses T-cell immunity; it is 100 times more potent than cyclosporine, thus requiring smaller doses. Side effects include gastrointestinal distress, nausea, vomiting, hyperkalemia, and hyperglycemia; adjust diet accordingly by controlling carbohydrate and enhancing potassium intake.
Tetranectin	Tetranectin binds plasminogen and may have a role in regulating pericellular proteolysis and in the survival of islets in the liver after islet transplantation.

FOOD AND NUTRITION

- Progress solids as quickly as possibly postoperatively. Monitor fluid status, and adjust as needed.
- Daily intake of protein should be appropriate for age and sex; 1.5 g/kg while on steroids may be recommended. Energy needs should be calculated as 30–35 kcal/kg.
- Daily intake of sodium should be 2–4 g until the drug regimen is reduced. Adjust potassium levels as needed.
- Daily intake of calcium should be 1–1.5 times the daily requirements to offset poor absorption. Children especially need adequate calcium for growth. Daily intake of phosphorus should be equal to calcium intake.
- Supplement diet with vitamin D, magnesium, and thiamin as needed. Adequate vitamin intake will be essential to maintain immunity and to support wound healing.
- Control CHO intake with hyperglycemia (45–50% total kcal); encourage healthy food sources of carbohydrate. Transplantation patients are at risk of further glucose intolerance from multiple medications.
- Plan fats at 25–35% of total kilocalories (encourage monounsaturated fats and omega-3 fatty acids). Low saturated fats and cholesterol may be needed. A controlled fat intake is recommended for prevention and treatment of hyperlipidemia.
- Reduce gastric irritants as necessary if GI distress or reflux occurs.
- Monitor electrolytes carefully; hyperkalemia is common with cyclosporine or tacrolimus.
- Special diets may be discontinued when drug therapy is reduced to maintenance levels. Encourage exercise and a weight control plan thereafter.

Common Drugs Used and Potential Side Effects

- Thymoglobulin is an antibody preparation to prevent organ transplant rejection. The START trial is underway to determine whether Thymoglobulin treatment can halt the progression of newly diagnosed type 1 diabetes when given within 6 weeks of disease diagnosis.
- See Table 9-12 for other drugs used for the transplant.

Herbs, Botanicals, and Supplements

- Herbals should be discouraged after transplantation. Those who self-medicate with herbals are taking a chance that their use of a herb may interact with their immunosuppressive drugs and either cause higher or lower than desired drug levels of these agents.
- Chaparral is an herbal product that may cause severe hepatitis or liver failure. St. John's wort interferes with the metabolism of immunosuppressants and should not be used after transplantation.
- See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Indicate which foods are sources of key nutrients such as protein in the diet. If patient does not like milk, discuss how other sources of calcium may be used in the diet.
- Alcohol should be avoided unless permitted by the doctor.
- Patients should know when to seek medical attention.
- Discuss problems with long-term obesity and hypercholesterolemia.
- Encourage moderation in diet; promote adequate exercise.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association–Islet Cell Transplantation
  http://www.diabetes.org/type-1-diabetes/islet-transplants.jsp
- Immune Tolerance Network (ITN)
  http://www.immunetolerance.org
- Insulin-Free
  http://www.insulin-free.org/
- NIDDK–Islet Cell Transplantation
  http://diabetes.niddk.nih.gov/dm/pubs/pancreaticislet/

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METABOLIC SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

The metabolic syndrome (MetS; insulin resistance syndrome or syndrome X) has simultaneous clustering of low levels of HDL cholesterol, hyperglycemia, high waist circumference, hypertension, and elevated triglycerides. Any three of the following five criteria constitute diagnosis of MetS (Grundy et al, 2005):

- Elevated waist circumference: 40″ or 102 cm in men; 35″ or 88 cm in women.
- Elevated triglycerides (TG) ≥150 mg/dL or drug treatment for elevated TG.
- Reduced HDL cholesterol: <40 mg/dL in men and 50 mg/dL in women or drug treatment for low HDL cholesterol levels.
- Elevated BP: >130 mm Hg systolic BP or 85 mm Hg diastolic BP or drug treatment for hypertension.
- Elevated fasting glucose: >100 mg/dL or drug treatment for elevated glucose.

It is associated with CVD and often leads to T2DM. This condition affects some young people but usually affects persons aged 55 years and older. More than 64 million Americans have MetS, roughly one in four adults and 40% of adults aged 40 years and older.

Increased birthweight, excessive energy intake, physical inactivity, obesity, smoking, inflammation, and hypertension contribute to MetS. Individuals who are obese and insulin resistant are particularly prone to this syndrome. An "apple" shaped figure (high waist circumference) is riskier because fat cells located in the abdomen release fat into the blood more easily than fat cells found elsewhere.

Serum adiponectin levels are associated with insulin sensitivity; they are decreased in T2D and obesity. Genetic and environmental factors contribute to risk (Gable et al, 2006). The initial insult in adipose inflammation and insulin resistance is perpetuated through chemokine secretion, adipose retention of macrophages, and elaboration of pro-inflammatory adipocytokines (Shah et al, 2008). In women, depressive symptoms are associated with MetS, especially with elevated afternoon and evening cortisol (Muhtz et al, 2009). Clearly, more research is needed.

Management of MetS should focus on lifestyle modifications, especially reduced caloric intake and increased physical activity (Deedwania and Volkova, 2005). Phytochemicals, MUFA, antioxidant foods, spices such as turmeric, cumin, and cinnamon have anti-inflammatory effects. Intake of whole milk, yogurt, calcium, and magnesium protect against MetS whereas intake of cheese, low-fat milk, and phosphorus do not (Beydoun et al, 2008; McKeown et al, 2009). Mild-to-moderate alcohol consumption is acceptable but binges and early onset of drinking are not acceptable.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Candidate genes associated with CVD represent potential risk factors for the MetS (Goulart et al, 2009). Chronic inflammation and IL1beta genetic variants may be a concern; genetic influences are more evident among subjects with low (n-3) PUFA intake (Shen et al, 2007).

Clinical/History		
Height	BMI	Waist circumference (35″ or 88 cm in women)
Weight	Diet history	BP (≥130/85 mm Hg)
Weight pattern history	Waist circumference (40″ or 102 cm in men)	
Central Obesity?		

Sleep apnea?
Depression?

Lab Work

Gluc (>100 mg/dL)
HbA1 c
HDL Chol (<40 mg/dL for men, <50 mg/dL for women)
LDL Chol (high?)
Trig (>150 mg/dL)
Mg^{++}
Na^{+}, K^{+}
Serum insulin
Serum uric acid (elevated?)
CRP (elevated)
High fibrinogen or plasminogen activator inhibitor [−1] in the blood?
Prothrombin time (PT)
International normalized ratio (INR)

INTERVENTION

OBJECTIVES

- Reduce the inflammatory state and insulin resistance caused by excessive adipose tissue. Improve body weight; lessen abdominal obesity in particular. A realistic goal for weight reduction should be 7–10% over 6–12 months (Bestermann et al, 2005).
- Promote physical activity. Recommendations should include practical, regular, and moderated regimens of exercise, with a daily minimum of 30–60 minutes and equal balance between aerobic and strength training (Bestermann et al, 2005).
- Achieve and maintain cholesterol, blood glucose, and BP at levels indicated by the American Heart Association, as follows (Grundy et al, 2005):

For Atherogenic Dyslipidemia

- For elevated LDL cholesterol: Give priority to reduction of LDL cholesterol over other lipid parameters. Achieve LDL cholesterol goals based on patient's risk category. LDL cholesterol goals for different risk categories are:
  - High risk: seek 70–100 mg/dL
  - Moderately high risk: seek 100–130 mg/dL
  - Moderate risk: seek 130 mg/dL
  - Lower risk: 160 mg/dL is acceptable

SAMPLE NUTRITION CARE PROCESS STEPS

Inappropriate Intake of Types of CHO—Metabolic Syndrome

Assessment Data: Food intake records: high refined CHO and soft drink intake; high juice but minimal fruit intake; low dietary fiber intake.

Nutrition Diagnosis (PES): Inappropriate intake of types of CHO related to knowledge deficit about MetS as evidenced by FBS of 140 mg/dL and triglycerides of 300 mg/dL.

Intervention: Education about desirable CHO, whole grains, and fiber; shopping tips; dining out.

Monitoring and Evaluation: Improved labs in 3–6 months; dietary records showing improvement in intake of whole grains, whole fruits, and vegetables.

- If TG is >200 mg/dL, then goal for non-HDL cholesterol for each risk category is 30 mg/dL higher than for LDL cholesterol. If TG is >200 mg/dL after achieving LDL cholesterol goal, consider additional therapies to attain non-HDL cholesterol goal.
- If HDL cholesterol is <40 mg/dL in men or <50 mg/dL in women, raise HDL cholesterol to extent possible with standard therapies for atherogenic dyslipidemia. Either lifestyle therapy can be intensified or drug therapy can be used for raising HDL cholesterol levels, depending on patient's risk category.

For Elevated BP

- Reduce BP to at least achieve BP of >140/90 mm Hg (or <130/80 mm Hg if diabetes is present). Reduce BP further to extent possible through lifestyle changes.
- For BP >120/80 mm Hg: Initiate or maintain lifestyle modification via weight control, increased physical activity, alcohol moderation, sodium reduction, and emphasis on increased consumption of fresh fruits, vegetables, and low-fat dairy products in all patients with MetS.
- For BP >140/90 mm Hg (or >130/80 mm Hg if diabetes is present), add BP medication as needed to achieve goal BP.

For Elevated Glucose

- For IFG, delay progression to T2DM. Encourage weight reduction and increased physical activity.
- In diabetes, for hemoglobin A1 c at or above 7.0%, lifestyle therapy and pharmacotherapy, if necessary, should be used. Modify other risk factors and behaviors (e.g., abdominal obesity, physical inactivity, elevated BP, or lipid abnormalities).

For Prothrombotic State

- Reduce thrombotic and fibrinolytic risk factors. Low-dose aspirin therapy or prophylaxis is recommended.

For Proinflammatory State

- There are no specific therapies beyond lifestyle changes. The antioxidants and omega-3 fatty acids may be helpful.

FOOD AND NUTRITION

- The general recommendations include low intake of saturated fats, trans fats, and cholesterol. Increase use of omega-3 PUFA intake (Shen et al, 2007) and MUFA (especially extra virgin olive oil).
- Plan a Mediterranean-type diet using more fiber and starches, especially whole grains, raw fruits, and vegetables. A plant-based diet may be useful (Barnard et al, 2005).
- The DASH diet contains 3–4 g sodium with good sources of potassium, calcium, and magnesium. Dairy products provide calcium, magnesium, and potassium.
- Monitor blood glucose levels. Carbohydrate restriction (CR) has been shown to improve dyslipidemias associated with MetS more than a low fat diet (Al-Sarraj et al, 2010).

- Encourage soy protein as a meat substitute several times a week; soy protein may help with weight reduction and dyslipidemia (Bestermann et al, 2005).
- Ensure adequate intake of folate, vitamins B_6, B_{12}, C, and E, preferably from food.
- Dark chocolate in small amounts regularly may help lower BP, improve cholesterol, and help with insulin sensitivity.
- Spread out the energy load by eating smaller meals.

Common Drugs Used and Potential Side Effects

- To lower lipids, a statin should be used initially unless contraindicated (Bestermann et al, 2005). Statins decrease biomarkers of inflammation and oxidative stress in a dose-related manner; atorvastatin 80 mg compared with a 10-mg dose is superior for decreasing oxidized LDL, hs-CRP, matrix metalloproteinase-9, and NF-kB activity (Singh et al, 2008).
- Glucose-lowering medications must be carefully prescribed and monitored. Metformin may be indicated (Orchard et al, 2005).
- BP medications may be prescribed; monitor for necessary restrictions of sodium and/or higher need for potassium. An ACE inhibitor or an angiotensin receptor blocker is usually the first medicine (Bestermann et al, 2005).
- Medications that diminish insulin resistance and directly alter lipoproteins are necessary; combination therapy is often required (Bestermann et al, 2005).
- If patients with MetS have elevated fibrinogen and other coagulation factors leading to prothrombotic state, aspirin is used (Deedwania and Volkova, 2005). Low-dose aspirin is not generally a problem; taken with a meal or light snack to prevent potential for GI bleeding.

Herbs, Botanicals, and Supplements

- Antioxidant supplements are not recommended, but intake of antioxidant-rich foods should be suggested (Czernichow et al, 2009). Thus far, trials with alpha tocopherol have been disappointing; further trials with gamma and alpha-tocopherols are warranted. Include phytochemicals, antioxidant foods, and spices such as turmeric, cumin, and cinnamon in the diet. See Tables 8-13 and 8-14.
- Chromium picolinate (CrPic) enhances insulin action by lowering plasma membrane (PM) cholesterol (Horvath et al, 2008). Coenzyme Q10 may have some merit, but further research is needed.
- High serum selenium concentrations have been associated with prevalence of higher FPG, LDL, TG, and glycosylated hemoglobin levels; further research is needed to determine its role in the development or the progression of MetS (Bleys et al, 2008).
- Other herbs and botanical supplements should not be used without discussing with physician. See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Widespread screening is recommended to slow the growth of this syndrome. Prevention should start in childhood with healthy nutrition, daily physical activity, and annual measurement of weight, height, and BP beginning at 3 years of age (Bestermann, 2005).
- Discuss the role of nutrition (DASH or Mediterranean diet principles) in managing this syndrome. Obesity is a major contributor to the problem, so weight loss (even 10 lb) can help improve health status. A diet rich in antioxidants and DHA is beneficial. Finally, the 2005 Dietary Guidelines are consistent with lowering risk for MetS (Fogli-Cawley et al, 2007).
- Regular physical activity can help to lower elevated blood cholesterol levels and BP. Walking, or an exercise that is pleasant for the individual, is the one to select. Aerobic and strength training exercises are beneficial. Reduce sedentary activities, including television and computer time (Ford et al, 2005).
- Smoking cessation measures may be needed. Offer guidance on how not to gain weight after quitting.
- Limit consumption of alcoholic beverages, which can elevate triglycerides in large doses.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association–Metabolic Syndrome
  http://www.diabetes.org/diabetes-research/summaries/ekelund-metabolic.jsp
- American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=534
- Mayo Clinic
  http://www.mayoclinic.com/health/metabolic%20syndrome/DS00522

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PREDIABETES

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

Prediabetes (impaired glucose tolerance, IGT) distinguishes those people who are at increased risk of developing diabetes. People with prediabetes have IFG or IGT, or both. More than 54 million Americans are affected with this combination of genes, obesity, and physical inactivity. People who have prediabetes may have a family history of heart disease and apple-shaped (intra-abdominal) obesity. Elevated triglycerides and low HDL cholesterol are risks for T2DM (Zacharova et al, 2005); adiposity-associated inflammation and insulin resistance are also implicated (Shah et al, 2008). NIH has also reported that smoking increases insulin resistance and prediabetes.

Under recent criteria, a normal blood glucose level is <100 mg/dL. Table 9-13 shows the tests and classification for IGT and IFG.

Most people with prediabetes go on to develop T2DM within 10 years unless they make lifestyle changes. Studies have evaluated various interventions among people with IGT and risks for developing diabetes. The Diabetes Prevention Program (DPP) was a large prevention study of people at high risk for diabetes (American Diabetes Association, 2006). Half of the participants had MetS at the beginning of the study; lifestyle intervention and metformin therapy reduced development of the syndrome in the remaining participants (Orchard et al, 2005). Lifestyle interventions include modest weight loss from a low-calorie, low-fat diet, and increased moderate-intensity physical activity (such as walking for 2½ hours each week).

People treated with an intensive lifestyle intervention reduce their risk of developing diabetes by half over 4 years, whereas people treated with metformin reduce their risk by only one third (American Diabetes Association, 2006). Metformin is most effective among younger, heavier people (those 25–40 years of age, 50–80 lb overweight) and less effective among older people and people with lower BMIs. In the STOP-NIDDM Trial, treatment of people with IGT with the drug acarbose reduced the risk of developing diabetes by 25% over 3 years.

The benefits of weight loss and physical activity strongly suggest that lifestyle modification should be the first choice to prevent or delay diabetes (American Diabetes Association, 2009). The dietitian plays an important role in nutrition education and counseling for these behavioral changes.

TABLE 9-13 Prediabetes Classifications and Tests

Condition/Classification	Test Used and Diagnostic Values
Impaired glucose tolerance (IGT)	• Oral Glucose Tolerance Test (OGTT), 75 g of glucose • 2-hour plasma glucose = 140–199 mg/dL
Impaired fasting glucose (IFG)	• Fasting plasma glucose (FPG) after 8-hour fast • Fasting plasma glucose = 100–125 mg/dL

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Researchers are learning how to predict a person's odds of getting diabetes; Caucasians with type 1 diabetes have genes called HLA-DR3 or HLA-DR4 (American Diabetes Association, 2009). The HLA-DR7 gene may put African Americans at risk, and the HLA-DR9 gene may put Japanese at risk.

The MTHFR C>T polymorphism has a significant association with diabetic neuropathy in Caucasians and in persons with T2DM (Zintzaras et al, 2007). More rigorous studies are needed to define the role of genotypes in diabetes complications and management.

Clinical/History		
Height	Waist circumference	I & O
Weight	BMI	BP (>140/90?)
	Diet history	Smoker?
		PCOS?

Heart disease?
Hx gestational diabetes?
High risk ethnic background?
Sedentary lifestyle?
Family hx T2DM?

Lab Work

Gluc
OGTT with 2 hours post-prandial (140–199 mg/dL?)
Fasting glucose after 8 hours (100–120 mg/dL?)
HbA1 c
Chol, HDL and LDL profiles
Trig
CRP
Alb, transthyretin
Na^+, K^+
H & H
Serum Fe
Serum folacin
BUN, Creat
Ca^{++}, Mg^{++}
GFR

SAMPLE NUTRITION CARE PROCESS STEPS

Not Ready For Lifestyle Change

Assessment Data: Blood glucose log and food history; BMI 29 at age 48; family Hx diabetes (T2DM); previous counseling on lifestyle changes to prevent diabetes. Recent random blood glucose >140 mg/dL.

Nutrition Diagnosis (PES): Not ready for lifestyle change related to potential for diabetes as evidenced by BMI 29, family history of diabetes, statement of "not being interested in many changes at this time."

Intervention: Counseling about the importance of lifestyle changes to reduce likelihood of diabetes.

Monitoring and Evaluation: Follow-up clinic visit indicating that client did make some lifestyle changes after considering the last counseling session; random blood glucose 100 mg/dL; weight loss of 5 lb in two months; increase in physical activity noted.

INTERVENTION

OBJECTIVES

- Increase the probability of reverting from IGT to normal glucose tolerance. Prevent further insulin resistance, hyperglycemia, or progression to diabetes.
- Modest weight loss (5–10% of body weight) and modest physical activity (30 minutes daily) are the recommended goals (American Diabetes Association, 2006). Walking can be encouraged for most people.
- Prevent or delay heart and kidney diseases, stroke, eye disease, and other undesirable conditions.
- Design a program that includes elements from the successful DPP trial:

Clearly defined weight loss and physical activity goals	A flexible maintenance program
Individual case managers or "lifestyle coaches"	Culturally appropriate materials and strategies
Intensive, ongoing intervention	Local and national network of training, feedback and clinical support
A core curriculum	Supervised exercise sessions at least twice weekly

Source: CDC, Web site accessed September 25, 2009, at http://www.cdc.gov/diabetes/faq/prediabetes.htm.

FOOD AND NUTRITION

- Control CHO intake (about 45–50% of total kilocalories) and limit excess added sugars. Encourage less processed carbohydrate and fiber food sources. Individualize according to lab work, BMI, and other risk reduction requirements.
- Maintain protein at about 20% of total energy.
- Plan fats at about 30–35% of total kcal (encourage monounsaturated fats and omega-3 fatty acids). Low saturated fats and cholesterol may be needed to prevent or treat hyperlipidemia.
- A moderate reduction in total energy intake is important. Modest weight loss of 5–7% of body weight may be recommended.
- Sufficient intake of minerals such as magnesium may be protective against diabetes. Include more almonds, whole wheat, cooked spinach, baked potatoes, and other magnesium-rich foods.
- Excesses of iron and selenium should be avoided; studies suggest that high intakes cause deposits in the internal organs, including the pancreas.
- Include adequate intake of fiber. A low fat vegan diet has been found to improve glycemia and elevated lipids (Barnard et al, 2009).
- Essential oils such as fenugreek, cinnamon, cumin, and oregano enhance insulin sensitivity (Talpur et al, 2005). Cinnamon may improve blood sugar and lipid levels (1/2 teaspoon daily).
- When a tube feeding is needed, there is insufficient evidence to support the use of specialty products as compared with standard products at this time.

Common Drugs Used and Potential Side Effects

- Acarbose treatment in IGT subjects decreases the risk of progression to diabetes by 36% (Delorme and Chiasson, 2005).
- Metformin reduces risk of developing diabetes, especially among younger, heavier people such as those 25–40 years of age who are 50–80 lb overweight (Herman et al, 2005). It is less effective among older people and people who were not as overweight.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. See Table 9-9 for tips.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Community Health Workers (CHW) may be trained for peer counseling when administered through a Diabetes Care Center (Katula et al, 2010). Lifestyle weight (LW) loss intervention has a goal of ≥7% weight loss achieved through increases in physical activity (180 min/wk) and decreases in caloric intake (approximately 1500 kcal/d) with CHW-led group-mediated cognitive behavioral meetings that occur weekly for 6 months and monthly thereafter for 18 months (Katula et al, 2010). Monthly small-group education sessions for a year are recommended by the CDC.
- Weight loss is a key goal (Davis et al, 2009; Norris et al, 2005). Discuss foods and meal patterns that will help to reduce risk factors for the individual client.
- Look AHEAD (Action for Health in Diabetes) trial participants (overweight adults diagnosed with diabetes) exceed recommended intake of fat, saturated fats, and sodium, which may contribute to increasing their risk of CVD and other chronic diseases (Vitolins et al, 2009). Therefore, it is important to address total fat intake (type of fat) and to suggest increasing intake of antioxidant-rich, high fiber foods. A vegan or vegetarian diet can be safely recommended (Barnard et al, 2009).
- Promote 8 oz or more of low fat milk, an ounce a day of nuts, filtered coffee, 1/2 teaspoon of cinnamon, and plenty of nonstarchy vegetables.
- Exercise seems to promote more reliable glycemic control compared with specific dietary protocols (Nield et al, 2007). Promote physical activity that matches client interest and ability. Discuss how to handle medication changes accordingly.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association–Prediabetes
  http://www.diabetes.org/diabetes-prevention/pre-diabetes.jsp
- CDC – Prediabetes
  http://www.cdc.gov/diabetes/faq/prediabetes.htm
- Mayo Clinic – Prediabetes
  http://www.mayoclinic.com/health/prediabetes/DS00624
- NIDDK–Prediabetes and Insulin Resistance
  http://diabetes.niddk.nih.gov/dm/pubs/insulinresistance/

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TYPE 2 DIABETES IN ADULTS

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

T2DM arises because of insulin resistance, when there is failure to use insulin properly combined with relative insulin deficiency. Increased and unrestrained hepatic glucose production as well as diminished glucose uptake and utilization results from insulin resistance occurring in the cells of the liver and other peripheral tissue, especially skeletal muscle. Previous names for T2DM include "non–insulin-dependent diabetes (NIDDM)," "adult-onset," "type II," "maturity-onset," and "ketosis-resistant" diabetes. T2DM accounts for 90% of all forms of diabetes. A significant percentage (about one third) of individuals who have T2DM are unaware of their diagnosis. Because T2DM is progressive, most patients will have already lost at least 50% of beta-cell function at the time of diagnosis.

The American Diabetes Association recommends diagnostic screening at 3-year intervals beginning at the age of 45, especially in individuals who have a BMI greater than 25 kg/m^2 (overweight). Risk factors include genetics, obesity, age, history of gestational diabetes, sedentary lifestyle, and smoking. Smoking cessation, decreasing BMI, and decreasing BP are major modifiable risk factors that are also major determinants of acquiring T2DM (Smith et al, 2005).

TABLE 9-14 Number of Medical Nutrition Therapy Visits Recommended for Type 2 Diabetes

Encounter Number	Length of Contact	Time between Encounters
1	60–90 minutes	2–4 weeks
2, 3	30–45 minutes	2–4 weeks
4, 5	30–45 minutes	6–12 months

From: National Guideline Clearinghouse. Nutrition practice guidelines for type 1 and type 2 diabetes mellitus. Accessed August 1, 2009, at http://guidelines.gov/summary/summary.aspx?doc_id=12816&nbr=006618&string=nutrition+AND+visits+AND+diabetes

For reducing BP, numerous clinical trials and experts support reduction of sodium intake; modest weight loss; increased physical activity; a low-fat diet that includes fruits, vegetables, and low-fat dairy products; and moderate alcohol intake. MNT from RDs in the treatment of T2DM improves glycemic outcomes with a decrease in A1 c of approximately 1–2%, depending on the duration of diabetes. Early referral for lifestyle changes and advice will yield the most benefit (Kulkarni, 2006).

The body's first defense against invading pathogens or tissue injury is the innate immune system; the cholinergic anti-inflammatory pathway is a neural mechanism that suppresses the innate inflammatory response such as excessive cytokine release in T2DM (Oke and Tracey, 2009). The use of antioxidant foods, spices and the Mediterranean diet has shown positive results as compared with a low fat diet (Esposito et al, 2009).

Patients should be educated about the progressive nature of diabetes and the importance of glycemic control with appropriate food choices and physical activity in conjunction with antidiabetes medication (Kulkarni, 2006). Maintain focus on lifestyle strategies that will improve blood glucose, BP, and lipids. Diabetes self-management training (DSMT), consisting of a 4-hour class, followed by individual dietitian consults and monthly support meetings can lower A1 c at a very low cost.

Weight management is especially helpful (Davis et al, 2009). Addition of a modest-cost, RD-led lifestyle case-management intervention to usual medical care does not increase health care costs and yields modest cost savings among obese patients with T2DM (Wolf et al, 2007). Weight loss significantly reduces diabetes costs; for every 1% weight lost, there is a 3.6% savings in total health costs and a 5.8% savings in diabetes care costs (Yu et al, 2007). The American Dietetic Association recommends four MNT visits initially, then one visit every 6–12 months Table 9-14). Referral to a dietitian within the first month after diagnosis is important.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: T2DM has a stronger genetic basis than type 1, but depends more on environmental factors such as obesity and a western diet. From whole-genome studies, affected family members are studied; calpain 10 (CAPN10) and hepatocyte nuclear factor 4 alpha (HNF4A) have been identified.

Common genetic variation near melatonin receptor MTNR1B contributes to raised plasma glucose and increased risk of type-2 diabetes among Indian Asians and European whites (Chambers et al, 2009). Variants of the FTO (affectionately called the FATSO) gene are identified an obesity risk allele; this gene is associated with increased risk of TDM.

Clinical/History

- Height
- Weight
- BMI
- Obesity?
- Diet history
- Waist circumference
- BP (increased?)
- Blurred vision?
- Erectile dysfunction
- Fatigue
- Frequent, slow-healing infections
- Increased appetite, thirst, urination
- Numbness or burning sensation in feet, ankles, legs
- Signs of dehydration?
- Smoking history
- Electrocardiogram (ECG) before exercise program

Lab Work

- CRP
- FPG
- HbA1c
- Blood or urinary ketones
- Total Chol, LDL, HDL
- Trig (often increased)
- BUN, Creat
- Na^+, K^+
- ALT, AST
- Lactate dehydrogenase (LDH)
- Alk phos
- Ca^{++}, Mg^{++}
- Microalbuminuria
- Plasminogen activator inhibitor type 1
- Fasting insulin
- Fasting C-peptide levels

SAMPLE NUTRITION CARE PROCESS STEPS

Self-Monitoring Deficit In T2DM

Assessment Data: Blood glucose self-monitoring records, food diary worksheets and meal records, blood glucose levels (fasting, 2-hour postprandial and/or HbA1c levels).

Nutrition Diagnosis (PES): Self-monitoring knowledge deficit related to lack of understanding how to record food and beverage intake as evidenced by incomplete food records at last two clinic visits and lab of HbA1c = 8.5 mg/dL.

Intervention: Teaching patient and family member(s) about use of simple blood glucose self-monitoring records (recording of timing, amount, blood glucose levels) and meal records.

Monitoring and Evaluation: HbA1c levels (goal <7 mg/dL); other glucose labs, food diary and records, discussion about complications of using the records.

INTERVENTION

OBJECTIVES

- Provide persons with diabetes initial basic nutrition messages and schedule time for MNT with an RD who has expertise in diabetes management.
- Maintain as near-normal blood glucose levels as possible by balancing food intake with insulin (either endogenous or exogenous) or oral glucose-lowering medications and activity levels. Improvement with MNT, if successful, is usually seen within 6 weeks and up to a maximum of 6 months. Medication may be needed if blood glucose levels are not under control.
- Maintain glycosylated A1c levels <7%, preprandial capillary plasma glucose levels between 90 and 130 mg/dL, and peak postprandial capillary plasma glucose levels <180 mg/dL. The target goal range for the patient may vary by age and underlying disorders.
- Protect beta-cell function by controlling hyperglycemia. A1c tests should be done at least two times a year in patients who are meeting treatment goals and quarterly in patients whose therapy has changed or who are not meeting glycemic goals.
- Achieve optimal serum lipid levels to reduce the risk for macrovascular disease. Dyslipidemia is a central component of insulin resistance in all ethnic groups.
- Emphasis of MNT is on lifestyle strategies to reduce glycemia, dyslipidemia, and BP. Strategies should lead to reduced energy intake and increased energy expenditure through physical activity. The A to Z (Atkins to Zone Diet) weight loss diet analysis found that premenopausal overweight and obese women who followed the Atkins diet (lowest carbohydrate intake), lost more weight at 12 months than women assigned to follow the Zone diet (Gardner et al, 2007). Therefore, for some patients, a low-carbohydrate, high-protein, high-fat diet may be a feasible alternative recommendation for weight loss.
- Maintain or improve overall health. The Dietary Guidelines for Americans and the MyPyramid food guide illustrate healthy nutritional guidelines and can be used by people with diabetes.
- Encourage regular mealtimes. Maintain lifestyle changes through behavior modification, education, and problem-solving strategies.
- Address individual needs according to culture, ethnicity, and lifestyle, while respecting willingness to change. For older adults, meet nutritional and psychosocial needs. For pregnant or lactating women, provide adequate energy and nutrients for optimal outcomes. Encourage therapeutic lifestyle changes (TLC), considering client's readiness, skills, resources, and current needs.
- Achieve BP levels that reduce risk for vascular disease and renal function decline.
- Manage problems related to compulsive or binge eating. Patients often report deliberate omission of insulin or oral hypoglycemic agents (OHA) to lose weight.
- Manage children with T2DM or maturity-onset diabetes of youth (MODY) differently than children who have T1DM; see appropriate entries.
- Prevent and treat the acute and long-term complications of diabetes listed in Table 9-2.

FOOD AND NUTRITION

- The dietitian should calculate CHO and fat requirements individually according to lipid and glucose levels. Assess dietary history, physical exercise, and activity patterns.
- Include foods containing CHO from whole grains, fruits, vegetables, and low-fat milk. A vegan diet is also an effective consideration (Barnard et al, 2009; Turner-McGrievy et al, 2008).
- Moderate weight loss in an obese patient (5–10% of starting weight) may reduce hyperglycemia, dyslipidemia, and hypertension. A moderate caloric restriction (250–500 calories less than average daily intake as calculated from a food history) and a nutritionally adequate meal plan should be recommended. Extremely low-calorie diets for adults should be performed only in a hospital setting.
- In morbidly obese patients, bariatric surgery may be the only effective treatment. There are risks from surgery and anesthesia, but the benefits often outweigh the risks.
- Space meals and spread nutrient intake, particularly carbohydrate, throughout the day.
- Eating breakfast has beneficial effects on fasting lipid and postprandial insulin sensitivity. Avoid meal skipping. Individualize meal plan according to patient preferences.
- Carbohydrate should be calculated between 45% and 65% of energy intake. Consistency is important.
- Protein should be calculated at 15–20% of daily energy intake with normal renal function and control at 0.8–1.0 g/kg with renal disease. Where weight loss is needed, use of a diet slightly higher in protein may help to enhance insulin sensitivity.
- Fat should be 25–35% of energy intake (7–10% of kcal saturated fats, 10% polyunsaturated fats, and 10–15% monounsaturated fats). Limit intake of cholesterol to <200–300 mg/d.
- Foods that benefit both the carbohydrate and lipid abnormalities should be consumed often. Recommendations are the same as for the general population concerning fiber: use more brown rice, beans, green leafy vegetables, oat bran, legumes, barley, produce with skins, apples, oranges, and other whole fruit and produce.
- As part of a healthy lifestyle and to manage hypertension, teach the principles of the DASH diet; <2400 mg/d of sodium is recommended.
- For most vitamins and minerals, ensure adequate dietary intakes. Higher levels of magnesium (Rumawas et al, 2006), chromium, and zinc are recommended. A multivitamin–mineral supplement may be used. Because altered vitamin D and calcium homeostasis may play a role in the development of T2DM, dual supplementation may prevent T2DM in high-risk populations (Tremblay and Gilbert, 2009; Pittas et al, 2007). Vitamin K as phylloquinone may also have a role in glucose management (Yoshida et al, 2008).
- Discuss usefulness of artificial sweeteners and food diaries.
- Discuss use of alcohol. Avoid hypoglycemia by consuming alcohol with food. Limit to 1 drink daily for women and two for men. Light-to-moderate amounts of alcohol do not increase triglyceride or BP levels. Indeed, moderate alcohol consumption has been associated with a decreased incidence of diabetes and heart disease; alcohol has been

reported to increase insulin sensitivity and raise HDL cholesterol levels. Alcoholic beverages should be considered an addition to the regular food/meal plan. If consumed daily, calories from alcohol are calculated into the total energy intake. Abstain from alcohol in pregnancy, pancreatitis, advanced neuropathy, severe hypertriglyceridemia, and a history of alcohol abuse.

Common Drugs Used and Potential Side Effects (see Table 9-15)

- T2DM is a progressive disease. Medications will need to be combined with lifestyle strategies. Insulin may be used in a combination with oral therapy or alone. If oral glucose-lowering medications do not achieve normoglycemia, combined oral therapies (drugs from two or more classes) can be initiated.
- If combination oral therapy does not work, insulin is needed. Insulin may also be needed for refractory hyperglycemia, diabetic ketoacidosis, stress, infection, or pregnancy.
- Multiple drug therapy is generally required to achieve BP targets. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), beta-blockers, diuretics, and calcium channel blockers may all be helpful.
- Aspirin therapy (75–162 mg/d) is recommended to protect against cardiovascular events.
- Overwhelming evidence suggests that the renin-angiotensin system (RAS) plays an important role in the pathogenesis of DM and its associated cardiovascular risks; RAS blockers may delay or prevent the onset (Braga and Leiter, 2009).
- In individuals with diabetes over the age of 40 years and without overt CVD, statin therapy to achieve an LDL <100 mg/dL is recommended. For people with diabetes and overt CVD, an LDL cholesterol goal of <70 mg/dL is recommended. Lifestyle modifications focusing on reducing saturated fat and increasing physical activity are also important.
- Antipsychotic agents contribute to the development of the metabolic syndrome and increase the risk for T2DM and heart disease. Monitor their use carefully.

TABLE 9-15 Medications Used for Type 2 Diabetes

Combination therapy rather than monotherapy is typically used to achieve glucose control in patients who are not at glycemic goals.

Classification	Medication	Route	The Way it Works	Time and Dose	Comments
Sulfonylureas	Glimepiride (Amaryl) Glipizide (Glucotrol) Glipizide ER (Glucotrol XL) Glyburide (Diabeta, Micronase)	Oral	Increases insulin production. Long-acting.	One or two times a day	Never give to a patient who is fasting for any reason. Do not use with alcoholic beverages. May cause weight gain, nausea, diarrhea or heartburn. Check LFTs.
Biguanides	Glucophage (Metformin) Glucophage XR	Oral	Lowers glucose from digestion; inhibits glucose release from the liver. Sensitizer.	Two to three times a day, XR once a day	Take with food. May cause diarrhea, flatulence, abdominal pain. Do not use with inflammatory bowel disease. Enhances weight loss.
Alpha-Glucosidase Inhibitors	Miglitol (Glyset) Acrobose (Precose)	Oral	Slows digestion, slows glucose production. Starch blocker that delays intestinal glucose absorption.	Take before each meal; swallow with first bite of food.	GI intolerance can occur. Exercise enhances effectiveness.
Thiazolidinediones	Rosiglitazone (Avandia) Pioglitazone (Actos)	Oral	Lowers glucose production; increases tissue glucose utilization, mostly in muscle.	Once daily with or without food	Liver damage is possible. Can lead to weight gain or heart failure.
Meglitinides	Repaglinide (Prandin) Senaglinide (Starlix)	Oral	Increases insulin production; short-acting.	5–30 minutes before meals	Offers better control of postprandial hyperglycemia and is associated with a lower risk of delayed hypoglycemic episodes.
DPP-4 Inhibitors	Sitagliptin (Januvia)	Oral	Lowers glucose by blocking an enzyme	100 mg. Once a day	
Incretin Mimetics	Exenatide (Byetta)	Injectable	Helps the pancreas make insulin, slows digestion	10 μg. Inject within an hour of AM and PM meals	Acid or sour stomach, belching, diarrhea, dizziness, nervousness.
Anti-hyperglycemic	Amylin (Symlin)	Injectable	Controls postprandial blood glucose	15 μg. Inject before major meals	

Source: Diabetes Medications, NIDDK. Web site accessed September 22, 2009, at http://diabetes.niddk.nih.gov/dm/pubs/medicines_ez/

- Medications such as steroids, beta-blockers, and diuretics may cause hyperglycemia in some patients. Supplements of Vitamin C may cause false-positive urinary glucose levels when given in large doses.
- Melatonin may have a role. Carriers of the risk genotype exhibit increased expression of MTNR1B in islet cells; these individuals may be more sensitive to the actions of melatonin, leading to impaired insulin secretion. Blocking the inhibition of insulin secretion by melatonin may be a novel therapeutic avenue for T2DM.
- Metformin improves insulin sensitivity in patients with IGT, but it does not reduce inflammatory biomarker levels (Pradhan et al, 2009).
- Xenical (fat blocker) may help cut heart risk in patients with T2DM. The obese diabetic patient who is poorly controlled may benefit from weight-reducing agents, such as sibutramine or orlistat.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. See Table 9-9 for more details.
- Vitamin D and calcium in combined supplementation may be beneficial in optimizing glucose metabolism (Pittas et al, 2007).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Medical nutrition intervention in patients with diabetes should address the metabolic abnormalities of glucose, lipids, and BP (Kulkarni, 2006). To meet the specific needs of the patient, adapt drug therapy to enhance outcomes and quality of life.
- DSME is an essential element of diabetes care, and national standards have been based on evidence for its benefits. DSME helps patients optimize metabolic control, prevent and manage complications, and maximize quality of life in a cost-effective manner. Health care providers should refer newly diagnosed patients to a dietitian who should then regularly reassess patients over time for modification of medications and food intake patterns.
- Emphasize the importance of regular mealtimes, use of medications, self-care and optimal functioning with instructions on how to handle illness, stress, exercise, label reading, use of sucrose or fructose and sugar alcohols.
- Vegan diets increase intakes of carbohydrate, fiber, and several micronutrients even more than the American Diabetes Association recommended diet (Turner-McGrievy et al, 2008). It would be acceptable to promote a vegan lifestyle for diabetes management (Barnard et al, 2009).
- Some restaurants offer foods lower in cholesterol, fat, and sodium, and higher in fiber. All restaurants offer low calorie sweeteners in the blue, yellow, or pink packets, and diet drinks. Many offer reduced-calorie salad dressings, low-fat or fat-free milk, and salt substitutes. Choose salads, fish, vegetables, baked or broiled food, and whole-grain breads.
- Clinical trials using antihyperglycemic medications to improve glycemic control have not demonstrated the anticipated cardiovascular benefits in patients with T2DM (Jenkins et al, 2008).
- To improve glycemic control, assist with weight maintenance, and reduce risk of CVD, at least 150 min/wk of moderate-intensity aerobic physical activity (50–70% of maximum heart rate) is recommended or at least 90 min/wk of vigorous aerobic exercise (>70% of maximum heart rate). Activity should be distributed over at least 3 d/wk, with no more than 2 consecutive days without physical activity.
- While high-intensity exercise is beneficial, even in older adults, it should start gradually and increase to desired intensity and duration. Avoid high-intensity exercise and resistance training in persons with neuropathy, coronary heart disease, or retinopathy. In the absence of contraindications, people with T2DM should perform resistance exercise three times a week, targeting all major muscle groups and progressing to three sets of 8–10 repetitions.
- Decrease sedentary behaviors, especially prolonged TV or computer time.
- Identify potential or real obstacles. Discuss options for handling negative emotions, temptations, dining away from home, feeling deprived, time pressures, competing priorities, social events, family support, food refusal, and lack of support from friends.
- Regular consumption of breakfast, self-weighing, and infrequent intake of fast foods are strategies that work well (Raynor et al, 2008).
- Use culturally appropriate methods for teaching and guidance. For example, use of the Medicine Wheel in Plains Indians has shown more effective results than usual techniques (Kattelmann et al, 2009).
- Encourage group support, behavior modification, and nutritional counseling for overweight.
- Small changes lead to greater self-esteem. Sequential rather than simultaneous dietary changes work well for most people and can improve the sense of self-efficacy.
- A comprehensive lifestyle self-management program using the Mediterranean low–saturated fat diet, stress management training, exercise, group support, and smoking cessation can reduce cardiovascular risk factors.
- Dietitians have a distinct role in MNT versus DSME; but both education and counseling are more medically effective than either one alone (Daly et al, 2009).
- A low-literacy tool can be useful in teaching key changes for those who need it. The goal is to achieve a sense of control over self-management of the diabetes.
- Before any type of surgery, blood glucose should be maintained between 100 and 200 mg/dL. Perioperative hyperglycemia may be managed with doses of rapid-acting insulin. Correct abnormalities before surgery, when possible.
- In noncritically ill hospitalized patients, premeal blood glucose should be kept as close to 90–130 mg/dL as possible, with a postprandial blood glucose <180 mg/dL; use insulin when necessary.
- In critically ill hospitalized patients, blood glucose levels should be kept as close to 110 mg/dL as possible and generally <180 mg/dL. These patients will usually require intravenous insulin.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association
  http://www.diabetes.org/food-nutrition-lifestyle/nutrition.jsp
- American Diabetes Association–Medicare Policy and Benefits
  http://www.diabetes.org/for-health-professionals-and-scientists/recognition/dsmt-mntfaqs.jsp
- American Diabetes Association–Type 2 Diabetes
  http://www.diabetes.org/type-2-diabetes.jsp
- American Dietetic Association: Type 2 Diabetes Practice Guidelines
  http://www.eatright.org/cps/rde/xchg/ada/hs.xsl/index.html
- Annals of Internal Medicine – Mediterranean Diet Implications for Patients
  http://www.annals.org/cgi/summary_pdf/151/5/306.pdf
- Mayo Clinic – T2DM
  http://www.mayoclinic.com/health/type-2-diabetes/DS00585
- Web MD – T2DM
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TYPE 2 DIABETES IN CHILDREN AND TEENS

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

T2DM arises because of insulin resistance, when there is failure to use insulin properly combined with relative insulin deficiency. In children, obesity or puberty often unmasks T2DM in genetically susceptible individuals. For example, the emergence of T2DM presents a new challenge for pediatricians and other health care professionals; preventive efforts, early diagnosis, and collaborative care of the patient and family are essential.

Breastfeeding, reduced television and sedentary lifestyles, healthy eating principles, family-based approaches, and screening are all important in managing this epidemic. Screening should be available for children whose BMI is greater than the 85th percentile at puberty or at age 10 plus any two of the following risk factors: family history, increased risk by ethnicity, or signs of insulin resistance such as polycystic ovary syndrome (PCOS), hypertension, dyslipidemia, or acanthosis. Weight greater than 120% of ideal for height also warrants screening.

In addition to obesity, family history of T2DM, and absence of GAD-65 antibodies, children with new-onset T2DM may be distinguished from those with T1DM by a combination of biochemical parameters of C-peptide, IGFBP-1, CO(2), and urine ketones (Katz et al, 2007). However, one third of the children with T2DM may have at least one detectable beta-cell autoantibody, classified as latent autoimmune diabetes in youth.

Fasting blood glucose levels should be checked at least every 2 years in high-risk children. T2DM in childhood can lead to end-stage renal disease and mortality in middle age; long duration is more detrimental (Pavkov et al, 2006). There is an ongoing effort for diabetes prevention in trials specifically designed to address the adolescent population (Karam and McFarlane, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The presence of multiple risk alleles amounts to a significant difference in children who have diabetes (Kelliny et al, 2009). Genetic variations for glucokinase (GCK), GCK regulatory protein (GCKR), islet-specific glucose 6 phosphatase catalytic subunit-related protein (G6PC2), and melatonin receptor type 1B (MTNR1B) are associated.

Clinical/History

Height
Weight
BMI
Diet history
Waist circumference
BP (often increased)
Sleep-disordered breathing?

Lab Work

FPG
HbA1c
C-peptide, IGFBP-1, CO(2)
Beta-cell autoantibodies (anti-GAD, anti-IA-2, and anti-ICA)?
Urine ketones
CRP
Urinary microalbuminuria
Plasminogen activator inhibitor type 1
Total Chol, LDL (high?)
HDL (usually low)
Trig (often increased)
BUN, Creat
Na^+, K^+
ALT, AST
LDH
Alk phos
Ca^{++}, Mg^{++}
Alb

INTERVENTION

OBJECTIVES

- Maintain as near-normal blood glucose levels as possible by balancing food intake with insulin (either endogenous or exogenous) or oral glucose-lowering medications and activity levels.
- Try to maintain glycosylated A1c levels <7%. Target goal range for the patient may vary by age and underlying disorders: <6-year old, 7.5–8.5%; 6- to 12-year old, <8%; 13- to 9-year old, <7.5%. Preprandial capillary plasma glucose levels should be kept between 90 and 130 mg/dL.
- Protect beta-cell function by controlling hyperglycemia. A1c tests should be done at least twice a year in patients who are meeting treatment goals and quarterly in patients who are not meeting glycemic goals.
- Achieve optimal serum lipid levels to reduce the risk for macrovascular disease.
- Emphasis of MNT is on lifestyle strategies to reduce hyperglycemica, dyslipidemia, and hypertension.
- Prevent and treat acute complications, short-term illnesses, and exercise-related problems, the long-term complications of renal disease, autonomic neuropathy, hypertension, and CVD.
- Maintain BP levels that reduce risk for vascular disease. Elevated BP has a major impact on renal function.
- Improve overall health through optimal nutrition and physical activity. Dietary Guidelines for Americans and the MyPyramid food guidance system illustrate nutritional guidelines and can be used by young people with diabetes.
- Encourage regular mealtimes. Maintain lifestyle changes through behavior modification, education, and problem-solving strategies.
- Working with the whole family is important. Promote family and individualized psychosocial counseling to handle depression and emotions.
- Address individual needs according to culture, ethnicity, and lifestyle, while respecting willingness to change. Encourage TLC, considering the child's needs and family circumstances.
- Manage problems related to compulsive or binge eating. Patients often report deliberate omission of medication in order to lose weight.

SAMPLE NUTRITION CARE PROCESS STEPS

Altered Nutritional Labs – T2DM In Teenager

Assessment Data: Blood glucose self-monitoring records, food diary worksheets and meal records, hour HbA1c levels 9 mg/dL, BMI >85% for age. Family Hx T2DM (both parents). BP normal but LDL cholesterol slightly elevated

Nutrition Diagnosis (PES): Altered nutritional labs related to overweight, knowledge deficit about diabetes and self-monitoring difficulty as evidenced by HbA1c = 8.5 mg/dL and LDL chol >130 mg/dL and statement that "I don't really understand my diabetes very well."

Intervention: Teaching patient and family member(s) about use of simple blood glucose self-monitoring records (timing, blood glucose testing, role of HbA1c in evaluating past 3 months, meal records).

Monitoring and Evaluation: HbA1c levels <7 mg/dL. Other labs WNL; food diary and glucose records showing improvement in outcomes of lab testing.

FOOD AND NUTRITION

- Calculate CHO and fat requirements individually according to age, serum lipid, and glucose levels. Assess dietary history, physical exercise, and activity patterns. Studies support the importance of carbohydrate from whole grains, fruits, vegetables, and low-fat milk.
- Moderate weight loss (5–10% of starting weight) may reduce hyperglycemia, dyslipidemia, and hypertension. A moderate restriction of 250–500 calories less than average daily intake (as calculated from a food history) and a nutritionally adequate meal plan are recommended.
- For teen boys over age 15, it may be useful to calculate caloric needs as 18 kcal/lb for usual activity and 16 kcal/lb if sedentary. Teen girls over age 15 have needs that are estimated the same as those for an adult.
- Extremely low-calorie diets are not recommended for children or teens. In morbidly obese children, gastric bypass surgery should be the last option as malnutrition is a permanent side effect.

- Spacing of meals (spreading carbohydrate throughout the day) and eating breakfast have beneficial effects on fasting lipid and postprandial insulin sensitivity.
- Meal skipping should be discouraged. Individualize meal plan according to patient preferences.
- Carbohydrate should be calculated as 45–65% of energy intake. Daily consistency is important.
- Protein should be calculated at 15–20% of daily energy intake with normal renal function; control at 0.8–1.0 g/kg with presence of renal disease.
- Fat should be 25–35% of energy intake, with 7–10% of kilocalories saturated fats, 10% polyunsaturated fats, and 10–15% monounsaturated fats. Limit intake of cholesterol to 200–300 mg/d.
- Recommendations for fiber include rice, beans, vegetables, oat bran, legumes, barley, produce with skins, apples, oranges, other produce. Include 19–38 g/d, larger amounts for older children and teens.
- As part of a healthy lifestyle and to manage hypertension, teach the principles of the DASH diet; 2400 mg/d of sodium is recommended.
- For vitamins and minerals, ensure that patient has adequate dietary intakes. Higher levels of magnesium, chromium, and potassium are recommended when serum levels are low. There is no need for supplements of any singular nutrient; a pediatric multivitamin–mineral supplement may be used.
- Include phytochemicals and antioxidants in the diet (see Table 8-14 in the pancreatic chapter).
- Discuss use of artificial sweeteners, food diaries, menu options at school, fast food choices that are lower in total fat, benefits and goals for physical activity, and the need for adequate fluid intake.

Common Drugs Used and Potential Side Effects (see Table 9-15 also)

- Oral agents may be used when blood glucose and other treatment goals are not met through diet and exercise alone. Glucophage should be the first oral agent used but not in children with known liver and kidney disease, low oxygen problems, or severe infections. Other oral agents such as a sulfonylurea or meglitinide can be added if control does not improve after 3–6 months. Thiazolidinediones probably should not be used in children.
- Insulin therapy should be started in children with severely elevated blood sugar levels or children with intense thirst and frequent urination. There are a wide variety of insulin regimens that can be used, anywhere from bedtime alone to multiple daily injections. Once blood sugars are under control, glucophage can be added while decreasing insulin dosage.
- Pharmacologic options for weight loss, including metformin, orlistat, and sibutramine have been studied (Miller and Silverstein, 2006). They should be used only under close medical supervision.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used in children and teens. There is insufficient evidence to draw definitive conclusions about the efficacy of individual herbs and supplements for diabetes, especially in children. However, inclusion of spices and seasonings in the diet would be acceptable.
- See Table 9-9 for guidance on more specific herbs and supplements that the client may be using.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Health care providers are encouraged to refer newly diagnosed patients to a dietitian. The dietitian should regularly reassess children and teens for overall growth and health status.
- Stress the importance of parental supervision and support.
- Children often benefit from attending diabetes camps for youth and support groups for family counseling. DSME is helpful for SMBG, medication use, physical activity goals, and meal planning.
- Emphasize the importance of regular mealtimes, proper use of medications, and balanced activity. Home blood glucose monitoring records and food/exercise records are important.
- Emphasize the importance of self-care and optimal functioning for illness, stress, dining out, exercise.
- Identify potential or real obstacles and discuss options for handling negative emotions, temptations, dining away from home, feeling deprived, time pressures, competing priorities, social events, family support, food refusal, and lack of support from friends.
- Explain food and nutrition labeling as well as how to manage sucrose, fructose, and sugar alcohols in the diet. Reduce sugar-sweetened beverage intake as much as possible.
- Encourage group support, behavior modification, and nutritional counseling for overweight. Small changes lead to greater self-esteem than continued failures. Sequential rather than simultaneous dietary changes work best and can improve self-efficacy.
- Reduce stress where possible. BMI and stress are independent determinants of TNF-alpha (an inflammatory cytokine) and adipocytokine among Latino children (Dixon et al, 2009).
- Suggest guidelines for physical activity: three to four times a week, exercise for 30–60 min/d. Aerobic exercise is protective against age-related increases in visceral adiposity (Kim and Lee, 2009).

Patient Education—Foodborne Illness

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

For More Information

- Centers for Disease Control and Prevention (CDC) Diabetes Projects
  http://www.cdc.gov/diabetes/projects/cda2.htm
- CDC Reference Documents
  http://www.cdc.gov/diabetes/projects/ref.htm
- Children with Diabetes
  http://www.childrenwithdiabetes.com/d_0n_d00.htm
- National Diabetes Education Program
  http://www.ndep.nih.gov/publications/PublicationDetail.aspx?PubId=154

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GESTATIONAL DIABETES

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

GDM involves glucose intolerance with first onset or recognition during pregnancy. It affects 14–25% of all pregnancies, increasing over the past decade (March of Dimes, 2009).

Pregnancy itself is a metabolic stress test. During the first half of pregnancy, transfer of maternal glucose to the fetus occurs; during the second half of pregnancy, placental hormones outweigh glucose transfer and insulin requirements typically double.

The placenta functions as a nutrient sensor, altering placental transport according to the maternal supply of nutrients. Placental transporters are regulated by hormones such as insulin. Insulin resistance occurs when gestational hormones such as human placental lactogen (HPL) interfere with insulin action. The accelerated fetal growth in women with diabetes is characterized by increased activity of placental and glucose transporters (Jansson and Powell, 2006). Cell signaling for insulin changes in GDM; women with diabetes or GDM may need one to two times more insulin than other pregnant women.

All pregnant women should be assessed for risk of GDM at the first prenatal visit; most women are screened between 24 and 28 weeks of gestation. One or more of the following factors identifies women at risk for GDM:

- Ethnicity: Hispanic American, African American, Asian, Pacific Islander, American Indian.
- Family history of diabetes in a first-degree relative (parents or siblings).
- Gestational diabetes or presence of a birth defect in a previous pregnancy.
- History of pregnancy-induced hypertension, urinary tract infections, or hydramnios (extra amniotic fluid).
- History of abnormal glucose tolerance.
- Older maternal age (>25-year old).
- Prepregnancy overweight or obesity; BMI >29.
- Previous birth of a large baby (>9 lb).
- Previous stillbirth or spontaneous miscarriage.

Hyperglycemia in pregnancy poses many complications and risks for the infant such as macrosomia, neural tube defects, hypocalcemia, hypomagnesemia, hyperbilirubinemia, birth trauma, prematurity, or neonatal hypoglycemia. Daily SMBG is important; fasting glucose levels should not exceed 105 mg/dL. Excellent blood glucose control with diet and, when necessary, insulin will result in improved perinatal outcome. HbA1c testing is not useful in GDM.

Women with a prior history of GDM and obesity are at significant high risk of developing MetS (Vohr and Boney, 2009). There is a higher risk of hypertension, pre-eclampsia, urinary tract infections, cesarean section, and future diabetes. About half of women who have GDM will proceed to have T2DM later, especially women who are hypertensive.

Nutrition recommendations should be based on a thorough nutrition assessment. Monitoring blood glucose levels, urine ketones, appetite, and weight gain guides the individualized nutrition prescription and meal plan. Adjustments should be made to the meal plan throughout pregnancy to ensure desired outcomes. See Table 9-16.

In women who have had bariatric surgery, close nutritional evaluation will be needed. Deficiencies in iron, vitamin A, vitamin B12, vitamin K, folate, and calcium can result in both maternal anemia and fetal complications, such as congenital abnormalities, IUGR and failure to thrive (Guelinckx et al, 2009).

Offspring of women with GDM are at increased risk for respiratory distress, macrosomia, jaundice, hypoglycemia, hyperinsulinemia, birth trauma, and developmental problems. Development of MetS in late adolescence and young adulthood is related to maternal glycemia in the third trimester, maternal obesity, neonatal macrosomia, and childhood obesity (Vohr and Boney, 2009).

Although treatment of mild GDM does not significantly reduce the frequency of stillbirth, perinatal death, or several neonatal complications, it does reduce the risks of fetal overgrowth, shoulder dystocia, cesarean delivery, and hypertensive disorders (Landon et al, 2009). Family planning, early screening for fetal abnormalities, compliance, glycemic and BP control during pregnancy, and improved neonatal care can make a difference.

After birth, rapid adaptation is necessary for infants to be able to maintain independent glucose homeostasis; this is compromised in infants who are small for gestational age (SGA), premature, or large for gestational age (Beardsall et al, 2008). Obesity, insulin resistance, and abnormal lipid metabolism bear ominous consequences for future generations (Vohr and Boney, 2009). For women who are offered treatment for mild GDM in addition to routine obstetric care,

TABLE 9-16 Glucose Testing for Gestational Diabetes Mellitus (GDM)

GDM often presents with hyperglycemia and glycosuria. Selective screening at 24–28 weeks of pregnancy is generally recommended, with a glucose challenge test and 1-hour assessment using either a 100-g or 75-g oral glucose load. Two or more of the venous plasma glucose concentrations must be met or exceeded for a positive diagnosis. The test should be done in the morning after an overnight fast.

American Diabetes Association criteria for GDM are noted below:

One-Step Approach

Perform a diagnostic oral glucose tolerance test (OGTT) without prior plasma or serum glucose screening. The one-step approach may be cost effective in high-risk patients or populations.

Two-Step Approach

Perform an initial screening by measuring the plasma or serum glucose concentration 1 hour after a 50-g oral glucose load (glucose challenge test [GCT]), and perform a diagnostic OGTT on the subset of women exceeding the glucose threshold value on the GCT. When the two-step approach is used, a glucose threshold value >140 mg/dL (>7.8 μmol/L) identifies approximately 80% of women with GDM, and the yield is further increased to 90% by using a cutoff of >130 mg/dL (>7.2 μmol/L).

Glucose Load	Glucose (mg/dL)
Glucose load, 100 g	
Fasting	95
1 hour	180
2 hours	155
3 hours	140

Sources: American Diabetes Association. Standards of Medical Care in Diabetes–2009.

additional charges are incurred but fewer babies experience serious perinatal complication or death (Moss et al, 2007). Nutritional intervention for GDM saves thousands of dollars per case. All women with GDM should receive nutritional counseling by an RD. MNT, initiated within 1 week of diagnosis and with a minimum of three nutrition visits, results in decreased hospital admissions and insulin use, improves likelihood of normal fetal and placental growth, and reduces risk of perinatal complications, especially when diagnosed and treated early (American Dietetic Association, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: GDM is associated with increased anti–human leukocyte antigen (HLA) class II antibodies in the maternal circulation (Steinborn, 2006). There may be some link between both autoimmune and nonautoimmune forms of diabetes and GDM.

Clinical/History

- Height
- Pregravid weight
- Pregravid BMI
- Pregravid waist circumference
- Expected date of confinement (EDC)
- No. of risk factors for GDM
- Current weight
- Goal weight
- Weight gain pattern
- Diet history
- BP
- Edema?
- Periodontal disease?
- Frequent urinary tract infections?
- Ultrasonography for fetal growth

Lab Work

- Ketones, fasting
- H & H
- Serum Gluc on awakening (95 mg/dL or lower)
- 1-hour postprandial glucose (not above 140 mg/dL)
- 2-hour postprandial glucose (not above 120 mg/dL)
- Na^+, K^+
- BUN
- Creat (often elevated)
- Alb, transthyretin
- Microalbuminuria
- Serum homocysteine levels
- Ca^{++}, Mg^{++}
- TSH (altered?)
- OGTT
- CRP

INTERVENTION

OBJECTIVES

- Prevent complications, perinatal morbidity, and mortality by normalizing the levels of glycemia and other metabolites (i.e., lipids and amino acids) to the levels of nondiabetic pregnant individuals.

SAMPLE NUTRITION CARE PROCESS STEPS

Inconsistent CHO Intake and Knowledge Deficit

Assessment Data: Blood glucose self-monitoring records, food diary worksheets and meal records, blood glucose levels (fasting, 2-hour postprandial).

Nutrition Diagnoses (PES):

(1) Inconsistent CHO intake (NI 5.8.4) related to lack of knowledge and confusion concerning gestational diabetic meal plan and CHO portion sizes as evidenced by postprandial blood sugars above and below desired ranges.

(2) Food and nutrition-related knowledge deficit (NB 1.1) related to gestational diabetic meal plan as evidenced by patient reported confusion and lack of confidence in meal planning and reported diet recall indicating inconsistent carbohydrate intake.

Interventions: E 2.2—review purpose of meal plan, CHO containing foods, portion sizes, CHO limits at meals/snacks. E 2.3, 2.5—provide advanced training in CHO counting, food label reading, menu planning, shopping tips.

Monitoring and Evaluation: Improvements in blood glucose levels; food diary and meal records showing more consistent intake of CHO throughout the day and before bedtime. Monitor at routine clinic visits for HgbA1c and weight gain. Assess need for further education/counseling; patient verbalizes better understanding of a meal plan and agrees to continue following plan.

- Optimize growth and development of the fetus. Desirable maternal weight gain will vary according to prepregnancy and current weight. Optimal weight gain is generally as follows: first trimester 0.5–1 kg (1–2 lb); second and third trimesters 0.2–0.5 kg/wk (0.5–1 lb/wk). Prevent weight loss.
- Obese women should control weight gain carefully. Excessive weight gain may be a forerunner of the MetS in offspring (Pirkola et al, 2008).
- Prevent infections and unexpected outcomes. Fetal glucose exposure and consequent fetal insulin secretion is normally tightly regulated by glucose delivery from the mother during pregnancy (Beardsall et al, 2008). The risk of spontaneous preterm birth increases with increasing levels of pregnancy glycemia.
- Control BP.
- Avoid incidents of starvation ketosis (where glucose is needed) and diabetic acidosis (where insulin and potassium are needed) by regular preprandial and postprandial SMBG. Ketosis is harmful to the baby.
- Use insulin when necessary, based on measures of maternal glycemia with or without assessment of fetal growth. Insulin therapy is recommended when nutrition intervention fails to maintain fasting whole-blood glucose at desired levels.
- Prevent hypoglycemic episodes, urinary tract infections, and candidiasis.
- Promote healthy lifestyle changes for the mother that will last long after delivery.
- Women with GDM should be encouraged to breastfeed.
- Physical activity, especially after meals, can help to maintain blood glucose control. Contraindications to exercise may include pregnancy-induced hypertension, intrauterine growth retardation, preterm labor or history of preterm labor, incompetent cervix/cervical cerclage, and persistent second or third trimester bleeding (American Dietetic Association, 2009).

FOOD AND NUTRITION

- Use a carbohydrate-controlled meal plan with adequate intake to keep weight gain appropriate while preventing glycemic shifts or ketonuria; manage for age and weight goals. Provide adequate energy and nutrients to meet the needs maternal blood glucose goals that have been established. The typical diet may include 30–35 kcal/kg; 20% protein, 40–45% CHO, and 35–40% fat.
- Select CHO from whole grains, one fruit portion, or one milk portion at a time. Limit juices, sweets, or desserts. Sufficient CHO intake is needed, 175 g at minimum. Many women with GDM undereat out of fear of needing insulin; interval weight loss may indicate presence of fasting ketones. Insulin may be needed.
- While a low glycemic index diet may be beneficial for some outcomes for both mother and child, results are inconclusive at this time (Tieu et al, 2008).
- For obese women (BMI >30), a calorie limit ~25 kcal/kg actual weight per day has been shown to reduce hyperglycemia and plasma triglycerides with no increase in ketonuria. For many women, this translates to an energy intake around 1700–1800 kcal. Artificial sweeteners may be used, but not saccharin.
- Maintain an adequate intake of polyunsaturated fats; keep saturated fats to 10% of total fat or less.
- Most women require three meals and four to five snacks; snacks are eaten at least 2–3 hours between feedings and should contain carbohydrate.
- Smaller, more frequent meals and snacks will be beneficial because of insulin resistance. Work closely with a dietitian to establish the best pattern according to typical blood glucose levels. A snack before bedtime may be needed.
- DASH diet principles may be helpful if BP is elevated. Include regular use of antioxidant foods and spices such as cinnamon.
- Ensure intake of a prenatal vitamin–mineral supplement (especially containing 600 μg folic acid, 30–60 mg iron, vitamin C, and adequate calcium). Include adequate chromium intake from diet (as in yeast breads).
- Tube feeding may be useful in patients who cannot be fed orally or with hyperemesis. CHO-controlled products are not necessarily required; monitor closely.
- After delivery, a review of glucose tolerance and postpartum nutrition is suggested at 6–12 weeks.

Common Drugs Used and Potential Side Effects

- Investigators found no substantial maternal or neonatal outcome differences between use of glyburide or metformin compared with use of insulin in GDM (Nicholson et al, 2009). Insulin may be required to control blood glucose if diet and exercise do not help. Careful physician and self-monitoring is important. The insulin lispro is associated with fewer hypoglycemic events.
- Use prenatal vitamin–mineral supplements as prescribed. Avoid large doses of vitamin C, which may show false-positive urinary glucose levels; more than 200 mg/d is not needed.
- Low-dose estrogen–progesterone oral contraceptives may be used after GDM, if no medical contraindications exist. However, medications that worsen insulin resistance (e.g., glucocorticoids, nicotinic acid) should be avoided if possible.

Herbs, Botanicals, and Supplements

- In general, pregnant women should not take any herbs and botanical products. They should discuss concurrent or previous use with their physician.
- Supplements of 1,25-dihydroxyvitamin D_3 may influence glucose metabolism in GDM by increasing insulin sensitivity. More research is needed on postpartum requirements.
- Only limited evidence exists to support prenatal omega-3 supplementation; use caution for an unbalanced high DHA intake during the first two trimesters of pregnancy, that is, DHA without additional amino acid (AA) supplementation (Hadders-Algra, 2008).

- Conflicting results have been found for specific nutrients. Zinc and selenium seem to be protective, whereas chromium does not; more research is needed for micronutrient recommendations to be made (American Dietetic Association, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The RD should monitor and evaluate blood glucose, weight, food intake, physical activity, and pharmacological therapy (if indicated) in women with GDM at each visit; MNT results in improved maternal and neonatal outcomes (American Dietetic Association, 2009).
- Communicate the importance of meal spacing, timing, adequacy, and consistency (i.e., patient should not skip meals). Instructions on what to eat and what to avoid will be important. Carrying a snack at all times is helpful (e.g., fruit, peanut butter, crackers). Follow current evidence-based guidelines.
- Regular aerobic exercise, such as walking, is beneficial. Exercise after meals may help to control blood sugar, but do not exercise until short of breath. Upper body exercises are also beneficial. Avoid exercises while lying on back as this decreases blood flow when the weight of the fetus presses on the main artery. Avoid exercises that increase the risk of falling.
- Encourage breastfeeding.
- Babies born to women who have GDM may have low levels of lipids such as arachidonic acid and docosahexaenoic acid (DHA); further studies are needed (Bitsanis, 2006).
- Encourage family planning to ensure optimal glucose regulation for all subsequent pregnancies. Women with a history of GDM are more likely to have recurrent diabetes if they are older, heavier and wait longer between pregnancies (e.g., 3 vs. 2 years) in many cases (Holmes et al, 2010). Recurrence of GDM is common, especially among non-Caucasian populations (Kim et al, 2007).
- Counsel regarding risk for T2DM. Any degree of abnormal glucose homeostasis during pregnancy independently predicts an increased risk of glucose intolerance and CVD postpartum (Retnakaran, 2009). Elevated FBG, OGTT 2-hour blood glucose, and OGTT glucose A1c are strong and consistent predictors of subsequent T2DM (Golden et al, 2009). Long-term lifestyle modifications, maintenance of normal body weight, and physical activity should be discussed.
- Discuss the potential impact of GDM on offspring, who are at increased risk of obesity, glucose intolerance, and diabetes in late adolescence or adulthood.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association
  http://www.diabetes.org/gestational-diabetes.jsp
- Baby Center – GDM
  http://www.babycenter.com/0_gestational-diabetes_2058.bc
- Diabetes and Pregnancy
  http://www.otispregnancy.org/pdf/diabetes
- NIDDK
  http://diabetes.niddk.nih.gov/dm/pubs/gestational/

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PRE-ECLAMPSIA AND PREGNANCY-INDUCED HYPERTENSION

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Pre-eclampsia is defined as the association of pregnancy-induced hypertension (PIH) and proteinuria of 300 mg/24 h or more after 20-weeks estation. It is more common in primigravidas and in patients with multiple gestation, T1DM, teenage mothers, family history of PIH, age over 40 years, or underlying vascular or renal disease. It affects 2–8% of pregnancies and is a severe complication, which may lead to fetal morbidity and mortality. Risks for the baby include poor growth and prematurity (Duley, 2009).

Pre-eclampsia can lead to problems in the liver, kidneys, brain, and the clotting system (Duley, 2009). Maternal morbidity includes placental abruption, HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) or eclampsia; treatment consists of ending the pregnancy. Early sonogram is recommended; many women will have to have total bed rest. Severe pre-eclampsia that develops at <34 weeks of gestation is associated with high perinatal mortality and morbidity (Sibai and Barton, 2007). Women who are 37-week pregnant are induced immediately.

Criteria for **mild pre-eclampsia** include hypertension as defined at 140/90 to 159/109 mm Hg; proteinuria >300 mg/24 h; mild edema with weight gain >2 lb/wk or >6 lb/month; and urine output >500 mL/24 h. Signs and symptoms include increased BP, proteinuria, facial edema, pretibial pitting edema, irritability, nausea and vomiting, nervousness, headache, altered states of consciousness, epigastric pain, and oliguria.

Criteria for **severe pre-eclampsia** include: BP >160/110 mm Hg on two occasions with patient on bed rest; systolic BP rise >60 mm Hg over baseline; diastolic BP increase of >30 mm Hg over baseline; proteinuria >5 g/24 h or 3+ or 4+ on a urine dipstick; massive edema; and oliguria <400 mL/24 h. Symptoms include pulmonary edema, severe headaches, visual changes, right upper quadrant pain, elevated liver enzymes, and thrombocytopenia in addition to the symptoms listed for mild pre-eclampsia.

In **eclampsia,** a seizure occurs. Eclamptic seizures and symptoms of the HELLP syndrome occur. In severe cases, there may also be hepatic rupture, pulmonary edema, acute renal failure, placental abruption, elevated creatinine, intrauterine growth restriction, cerebral hemorrhage, cortical blindness, and retinal detachment. Maternal mortality rate is 8–36%.

Because plasma homocysteine is often increased, sufficient intakes of folic acid, vitamins B_{12} and B_6 are recommended (Mignini et al, 2005). Studies have also shown the effectiveness of magnesium in eclampsia and pre-eclampsia (Guerrera et al, 2009).

SAMPLE NUTRITION CARE PROCESS STEPS

Disordered Eating Pattern

Assessment Data: Food records, weight records. Diet hx reveals intake of one large meal daily, mostly canned soup and sandwiches; no fruits and few vegetables. BP 170/90 average; abnormal lung function tests (LFTs).

Nutrition Diagnosis (PES): Disordered eating pattern (NB 1.5) related to lack of knowledge concerning nutrition during pregnancy as evidenced by excessive prenatal weight gain, frequent meal skipping, increased BP, and diet lacking in vitamins and minerals and statement that "I can't eat more than once a day because it isn't good for the baby."

Intervention: ND 3.2.1—daily use of prenatal vitamin. Education and counseling about appropriate dietary intake and meal frequency during pregnancy; dangers of skipping meals; when to contact the doctor. E 1.2—priority modifications of diet include not skipping meals, ensure adequate calcium, folic acid, potassium and magnesium; control sodium intake. C 1.3—Health Belief Model of counseling used to motivate patient in making necessary dietary changes.

Monitoring and Evaluation: Weight and prenatal growth charts; improved intake. Lab reports and BP records showing improvement in all aspects. Check urine protein and presence of edema. Successful outcome for infant and mother, even if hospitalization is required.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Women with pre-eclampsia have higher levels of a peptide that increases BP in the pieces of tissue linking mother and fetus.

Clinical/History

- Height
- Pregravid weight
- Pregravid BMI
- Pregravid waist circumference
- EDC
- Current weight
- Weight gain pattern
- Diet history
- Edema in lower extremities
- BP (mild, ≥140/90 mm Hg; severe, >160/110 mm Hg)
- Decreased urine output
- Confusion, apprehension
- Shortness of breath
- Right upper quadrant abdominal pain
- Dizziness
- Visual changes (blurring, double vision)
- Excessive nausea or vomiting
- Severe headaches
- Fever?
- Blood in urine?

Lab Work

- GFR
- Alb, transthyretin (often low)

Proteinuria (>300 mg/d is mild; >500 mg/d is severe)	BUN	Liver function tests (AST, ALT, LDH)
H & H	Creat (may be elevated)	Microalbuminuria
Serum Fe	Homocysteine	
Chol, Trig (elevated?)	CRP	
Gluc	PT, INR	
Ca^{++}	HELLP syndrome (decreased platelets, abnormal liver function tests)	
Mg^{++}		
Na^{+}, K^{+}		
Uric acid (elevated)		

INTERVENTION

OBJECTIVES

- Reduce maternal and neonatal mortality and morbidity. In pregnant patients with diabetes and chronic hypertension, BP target goals of 110–129/65–79 mm Hg are suggested. Avoid lower BP because fetal growth may be impaired.
- Lessen edema when present. Correct any underlying malnutrition or micronutrient deficiencies.
- Monitor any sudden weight gains (1 kg/wk) that are unexplained by food intake.
- Prevent, if possible, chronic hypertension and MetS after delivery.

FOOD AND NUTRITION

- Maintain diet as ordered for age and pregnancy stage (generally 300 kcal more than prepregnancy diet). Use extra fruits and vegetables and less sucrose.
- Sources of magnesium include green leafy vegetables, nuts, legumes, and whole grains.
- Supplement with prenatal vitamins. Include folic acid, calcium, other B-complex vitamins, protein, selenium, and potassium from diet. The role of vitamins C and E does not benefit women at risk for pre-eclampsia (Villar et al, 2009). Antioxidant supplements are also not recommended (Rumbold et al, 2008).
- Sodium intake may need to be controlled to 2 g/d if edema is severe. Diuretics generally are not used.
- There may be some merit for including a sufficient intake of omega-3 fatty acids from salmon, tuna, walnuts, and flaxseed oil.

Common Drugs Used and Potential Side Effects

- The only interventions shown to prevent pre-eclampsia are antiplatelet agents, primarily low-dose aspirin, calcium supplementation and magnesium sulfate for eclamptic seizures (Duley, 2009). Avoid excessive magnesium from supplements if this medication is being used.
- Corticosteroids for lung maturity may be necessary.
- During pregnancy, typical diuretics and cardiac drugs are not used. ACE inhibitors and ARBs are contraindicated. Calcium pump inhibitors are often a first line choice.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used in pregnancy.
- Evening primrose has been suggested for this condition. Coenzyme Q10 and alpha-tocopherol are potent antioxidants.
- See Table 9-9 for guidance on more specific herbs and supplements that the client may have used.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Rest is essential during this time. Biofeedback, yoga, meditation, and other forms of stress reduction are often beneficial.
- Meal skipping should be avoided at all costs.
- Discuss adequate sources of calcium from the diet, especially if dairy products are not tolerated or preferred.
- Good sources of potassium and magnesium include nuts, fruits and vegetables. The DASH diet is an excellent diet to continue, even after pregnancy.

Patient Education—Foodborne Illness

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

For More Information

- Diabetes and Preeclampsia
  http://diabetes.healthcentersonline.com/womensdiabetes/preeclampsia.cfm
- Mayo Clinic
  http://www.mayoclinic.com/health/preeclampsia/DS00583
- Pregnancy and Preeclampsia
  http://familydoctor.org/064.xml

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DIABETIC GASTROPARESIS

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

Gastroparesis occurs in approximately 50% of all cases of diabetes (Aring et al, 2005), with delayed gastric emptying (DGE) in the absence of mechanical obstruction. Diabetic gastroparesis is the result of ongoing damage to the nerves that are responsible for peristalsis and normal motility. Foods digest abnormally slowly or peristalsis is diminished so that it is difficult to match insulin action to digestion and absorption of the meals. If food stays in the stomach too long, infection is possible. Also, the food can harden into a solid lump; these bezoars may cause pain, nausea, and blockages in the digestive tract.

Hypoglycemia can occur if insulin is working and if food remains in the stomach too long. Later, insulin action is diminished and food finally digests, causing hyperglycemia. Problems occur more often in type 1 than in T2DM. Strict glycemic control is key.

A gastrostomy is rarely indicated, but a jejunostomy may be helpful in maintaining nutrition. Prokinetic agents are the best treatment option; see Section 7 for more details on management of gastroparesis. Gastric electrical stimulation may be used to send out brief, low-energy impulses to the stomach to decrease nausea and vomiting.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: T1DM has a genetic component, but gastroparesis would be individually acquired.

Clinical/History

- Height
- Weight
- BMI
- I & O
- Diet history
- Early satiety
- DGE
- Diarrhea or constipation
- Vomiting undigested food
- Nausea
- Reflux or heartburn
- Abdominal bloating or pain
- Upper endoscopy
- Barium x-ray
- Gastric emptying scintigraphy (GES)
- Gastric manometry

Lab Work

- HbA1c
- H & H
- Serum Fe
- Gastrin
- Gluc (fasting and 30 minutes after meals)
- Na^+, K^+
- Alb, transthyretin
- CRP
- Chol, Trig

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function

Assessment Data: Blood glucose self-monitoring records, nausea, gastroesophageal reflux (GERD) and reflux with meals, abdominal discomfort. Irregular FBG levels from day to day.

Nutrition Diagnosis (PES): Abnormal GI function related to gastroparesis as evidenced by nausea, GERD, and abdominal discomfort.

Intervention: Meal records and food diary listing symptoms and frequency of discomfort. Blood glucose self-monitoring records. Discussion about use of prokinetic agents.

Monitoring and Evaluation: Glucose records, food diary and records, discussion about GI symptoms.

INTERVENTION

OBJECTIVES

- Correct the fluctuating blood glucose levels through careful food and insulin management, frequent blood glucose checks and monitoring records.
- For nausea and vomiting, restore volume and hydration and provide antiemetics generously.
- Relieve symptoms (pain, diarrhea, constipation) and maintain adequate nutritional status.
- Differentiate from ketoacidosis, which has similar symptoms of nausea and vomiting.
- Prevent infection or bezoar formation from accumulation of indigestible solids.

FOOD AND NUTRITION

- Monitor intake carefully; blood glucose fluctuations are common. Include 200–240 g CHO daily, spread throughout the day. Use two to three CHO servings per meal. Be consistent from day to day.
- Soft-to-liquid diet may be useful to prevent delay in gastric emptying. Six small meals may be better tolerated than large meals.
- Use a low-fat diet to improve digestion and to improve stomach emptying. Stay sitting after meals.
- Decrease overall fiber intake from meals or supplements. Oranges, broccoli, green beans, berries, figs, and fresh apples may be difficult to digest.
- For dry mouth, add extra fluids and moisten foods with broth or allowed sauces or gravies.
- If nausea occurs, cold foods and low-odor choices may be better tolerated.

- In severe problems, a percutaneous jejunostomy (PEJ) tube feeding may be indicated. It can be used temporarily if needed to correct malnutrition.

Common Drugs Used and Potential Side Effects

- Prokinetics such as metoclopramide (Reglan) may be given 30 minutes before meals to increase gastric contractions and to relax pyloric sphincter. Dry mouth, sleepiness, diarrhea or nausea can be side effects. Emitasol is a nasal spray form of this medication. Metoclopramide prophylaxis to reduce gastric volumes before elective surgery is unnecessary unless the patient has a prolonged history of poor blood glucose control (Jellish et al, 2005).
- Rapid-acting insulin should be injected with or after meals. Use SMBG to monitor delayed absorption and glucose changes. Insulin lispro (Humalog) is quite effective because it starts working shortly after injection. To control blood glucose, it may be necessary to take insulin more often, to take insulin after eating instead before, and to check blood glucose often after eating.
- If a liquid diet is used, more insulin may be needed. Insulin pumps may be beneficial because insulin delivery rates can be programmed to the patient's individual needs.
- Because there is a reciprocal relationship between gastric emptying and ambient glucose concentrations, newer diabetes therapies that decelerate the rate of gastric emptying may be a beneficial tool (Samsom et al, 2009).
- Antiemetics may be used for vomiting. Monitor for specific side effects. Zofran may cause constipation or headache.
- Erythromycin improves stomach emptying by increasing the contractions that move food through the stomach. Side effects are nausea, vomiting, diarrhea, and abdominal cramps.
- Dramamine (dimenhydrinate) is an antihistamine that helps prevent nausea and vomiting; slight-to-moderate drowsiness and thickening of bronchial secretions may occur.
- Motilium (domperidome) is used to manage the upper GI problems; side effects may include headache.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss delayed digestion and absorption of food.
- Discuss role of diet in maintaining weight and controlling discomfort. Emphasize nutrient-dense foods if intake has been poor. Blenderized foods or smoothies may be well tolerated.
- Bezoar formation may occur after eating oranges, coconuts, green beans, apples, figs, potato skins, Brussels sprouts, broccoli, sauerkraut, corn, sauerkraut, and other high-fiber foods.
- Discuss insulin management, as appropriate. Include information about SMBG and meal spacing. Advise the doctor if blood glucose levels are >200 mg/dL.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association
  http://www.diabetes.org/type-1-diabetes/Gastroparesis.jsp
- Ask the Dietitian
  http://www.dietitian.com/drugnutr.html
- Diabetic Gastroparesis
  http://digestive.niddk.nih.gov/ddiseases/pubs/gastroparesis/
- Gastroparesis and Dysmotilities Association (GPDA)
  http://www.digestivedistress.com/main/page.php?page_id=26

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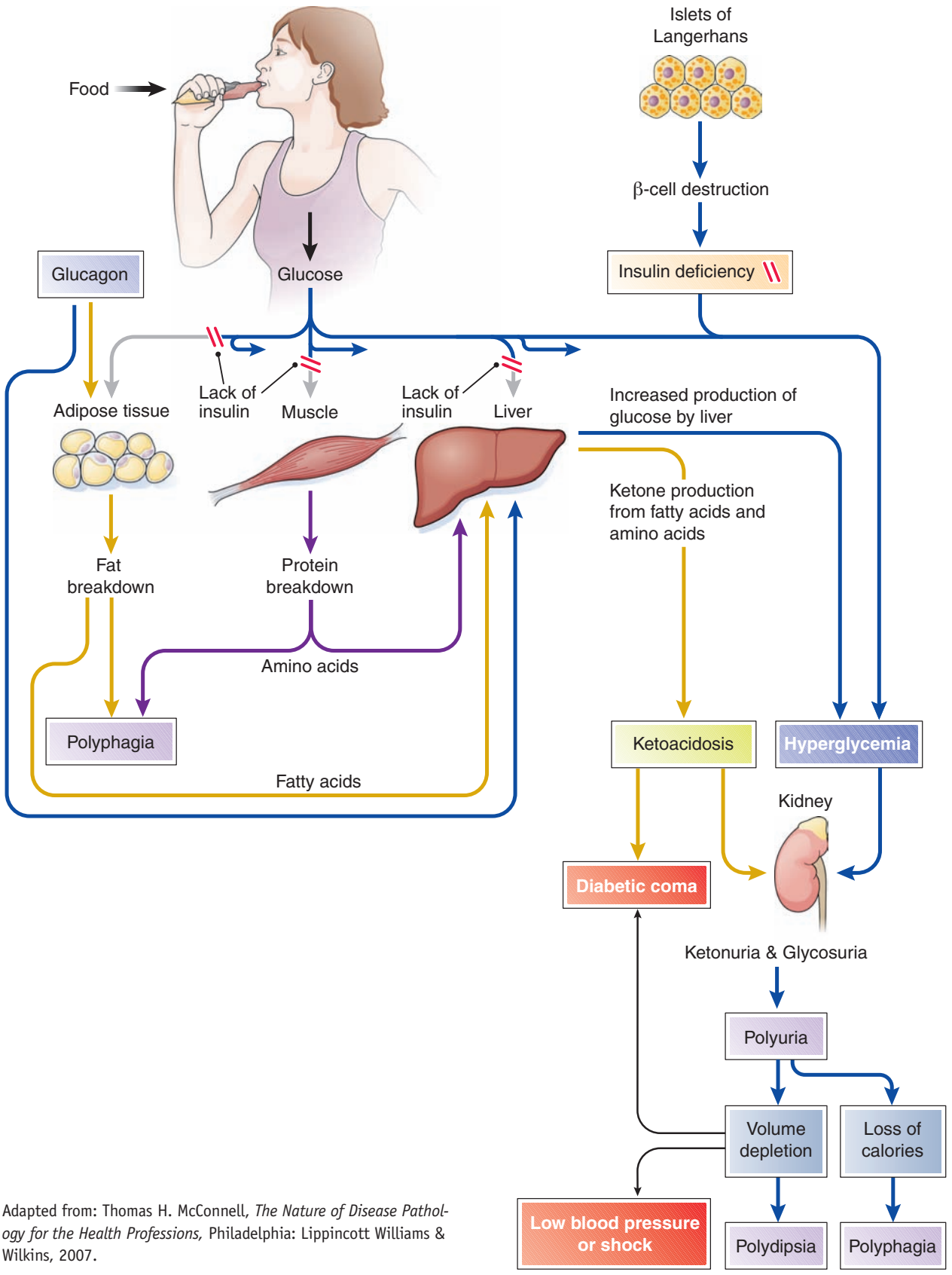
van der Voort IR, et al. Gastric electrical stimulation results in improved metabolic control in diabetic patients suffering from gastroparesis. *Exp Clin Endocrinol Diabetes.* 113:38, 2005.

DIABETIC KETOACIDOSIS

NUTRITIONAL ACUITY RANKING: LEVEL 3–4



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

DEFINITIONS AND BACKGROUND

Ketones in the urine mean that the body is burning fat to get energy because glucose is not available. The lack of insulin leads to gluconeogenesis in the liver, then glucose spills into the urine and causes an osmotic diuresis with dehydration. Free fatty acids are released from the adipose tissue and converted into acidic ketones (acetoacetate and β-Hydroxybutyrate) by the liver.

Large amounts of ketones in the serum and urine can be dangerous. Diabetic ketoacidosis (DKA) is classic metabolic acidosis, a medical emergency that accounts for more than 100,000 hospital admissions yearly in the United States. A patient with a low serum potassium level should be assumed to have a potentially life-threatening crisis.

Causes of DKA include not getting enough insulin from missed injections; alkaline reserves depleted by too little insulin, flu or colds, fever, pregnancy, stress, trauma, or myocardial infarction; infection (leading cause); urinary tract infections and pneumonia. The triad of uncontrolled hyperglycemia, metabolic acidosis, and increased total body ketone concentration characterizes DKA. These metabolic derangements result from the combination of absolute or relative insulin deficiency and increased levels of the counterregulatory hormones of glucagon, catecholamines, cortisol, and growth hormone. Table 9-17 provides defining characteristics for DKA and hyperosmolar hyperglycemic state (HHS).

DKA is seen primarily in patients with T1DM; a few type 1 diabetic patients presenting with DKA on initial diagnosis. In children, DKA is a leading cause of hospitalization and is a cause of cerebral edema, which may lead to death if untreated. DKA can also occur in T2DM, usually from urinary tract infections, trauma, stress, pregnancy, surgery, or myocardial infarction. Inflammatory processes may play a role. Both DKA and its treatment produce varying degrees of immunological stress (Jerath et al, 2005). All individuals with DKA should be tested for hyperthyroidism (Potenza et al, 2009). Use of standardized written guidelines have merit.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: There are no specific genes for DKA, but diabetes itself has a genetic component.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Temperature
- I & O
- BP (often low)
- Nausea and vomiting
- Hot or dry and flushed skin
- Diarrhea
- Fruity breath
- Intense thirst
- Profound dehydration
- Dim vision
- Labored or rapid breathing (Kussmaul)
- Pruritus
- Polyuria
- Cramping
- Seizures or drowsiness

Lab Work

- *Mild:* blood pH 7.25–7.30; bicarbonate 15–18 µmol/L; alert
- *Moderate:* pH 7.00–7.25, bicarbonate 10–15, mild drowsiness
- Severe: pH < 7.00, bicarbonate <10, stupor or coma
- Gluc (>250 mg/dL)
- HgbA1c
- Beta-hydroxybutyrate levels
- K^+ (low?)
- Urinary glucose, ketones, osmolality
- T3, T4, TSH levels
- CRP
- BUN (increased)
- Creat
- Na^+ (decreased)
- Cl^- (decreased)
- HbA1c
- Amylase (increased)
- WBC (elevated with infections)
- pCO_2 (decreased)
- pO_2
- Uric acid (increased)
- AST (decreased)
- ALT, LDH, creatine kinase (CK) (increased)
- Phosphate (often decreased)
- Mg^{++} (often decreased)
- Chol (increased), Trig
- Ca^{++}

INTERVENTION

OBJECTIVES

- Correct the high blood glucose level by giving more insulin. Frequent blood glucose and ketone monitoring is necessary. Note that urine ketones lag behind serum ketones because it takes time to the empty the bladder, where they have accumulated and been stored.
- Replace fluids and electrolytes lost through excessive urination and vomiting.
- Promote return to wellness after poor health for several days.
- Evaluate precipitating factors such as surgery, trauma, urinary tract infection, pneumonia, or influenza.
- Monitor frequently to prevent recurrence of DKA.

TABLE 9-17 Diagnostic Criteria for Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycemic State (HHS)

	DKA			
Criteria	**Mild**	**Moderate**	**Severe**	**HHS**
Plasma glucose (mg/dL)	>250	>250	>250	>600
Arterial pH	7.25–7.30	7.00–7.24	<7.00	>7.30
Serum bicarbonate (mEq/L)	15–18	10–<15	<10	>15
Urine ketones	Positive	Positive	Positive	Small
Serum ketones	Positive	Positive	Positive	Small
Effective serum osmolality (mOsm/kg)	Variable	Variable	Variable	>320
Altered mental status	Alert	Alert/ drowsy	Stupor/ coma	Stupor/ coma

Adapted with permission from Kitabchi AE, et al. Hyperglycemic crises in diabetes. *Diabetes Care.* 27:S95, 2004.

SAMPLE NUTRITION CARE PROCESS STEPS

Self Monitoring Deficit

Assessment Data: Admission to unit with DKA, random blood glucose levels >600, semi-comatose state, unable to eat orally.

Nutrition Diagnosis (PES): Self monitoring deficit (NB-1.4) related to blood glucose monitoring and diabetes management as evidenced by DKA, BG > 600, lethargy, nausea and vomiting past 2 days, and report by family that "he doesn't test his sugars as he should."

Intervention: Food and nutrient delivery with IVs and tube feeding until semicomatose state resolves and glucose levels are below 200 consistently. Educate patient when possible about the dangers of not monitoring BG levels. Teach methods for simplifying records, tracking lab results, and self-monitoring of glucose and activity levels to match insulin therapy.

Monitoring and Evaluation: Improved cognition and ability to resume an oral diet. Able to describe how to track glucose, lab results, physical activity, and insulin; defined own goals for reporting signs and symptoms to doctor when hyperglycemia persists. Able to state the signs of impending DKA and measures to take immediately.

- Prevent complications such as shock, arterial thrombosis, renal failure, or cerebral edema (CEDKA). CEDKA remains a significant problem with a high mortality rate (Lawrence et al, 2005). Young children are especially vulnerable and may slip into a coma.
- For chronically high fasting glucose levels, adjust evening intermediate- or long-acting insulin doses or timing.

FOOD AND NUTRITION

- If patient is comatose, intravenous insulin, electrolytes, and fluids are used. A nasogastric tube may be placed to prevent aspiration during feeding. If patient is alert and oriented, offer plenty of fluids orally.
- As treatment progresses, a 5% glucose solution is usually given as glucosuria and hyperglycemia subside. If glucosuria and hyperglycemia do not decrease, try tea and salty broth. Later, fruit juices and liquids high in potassium may be given.
- Resolution of DKA leads to the ability to tolerate oral nutrition and fluids, normalization of blood acidity (pH>7.3), and absence of ketones in blood (<1 μmol/l). Once this has been achieved, insulin may be switched to the usual regimen.

Common Drugs Used and Potential Side Effects

- Insulin and fluid are usually given intravenously to decrease blood glucose by 50–75 mg/dL/h. Guidelines may recommend a bolus (initial large dose) of insulin of 0.1 unit of insulin/kg of body weight if potassium level is higher than 3.3. Regular and glulisine insulin are equally effective during the acute treatment of DKA; a basal bolus regimen with glargine and glulisine is safer than NPH and regular insulin after the resolution of DKA (Umpierrez et al, 2009).
- Dextrose in saline is given once plasma glucose decreases to avoid accidental hypoglycemia.
- Potassium, phosphate and magnesium levels may be low. Bicarbonate therapy is needed only rarely (Trachtenbarg, 2005).
- Intravenous antibiotic therapy will be needed where there is sepsis.
- Atypical antipsychotics, thiazide diuretics, and corticosteroids can lead to DKA if the patient is not carefully monitored.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain demand for more insulin during illness and infection. Use of a diabetes record may be useful if episodes of high blood glucose are frequent.
- Never exercise when there are ketones present.
- Because every episode of DKA implies breakdown in clinical communication, appropriate diabetes education should be reinforced. Education of patients and their social environment to promote frequent testing—especially during sick days—and to lower their glucose levels, as well as to recognize the early symptoms of hyperglycemia and DKA is of paramount importance in preventing the development of severe DKA (Weber et al, 2009).
- When strict control through insulin is administered, weight gain is common. Insulin omission and reduction, which are eating disorder symptoms unique to diabetes mellitus, are associated with an increased risk of DKA. Attention to this disorder is important in overall treatment planning and management.
- The biggest risk of insulin pump therapy is DKA since no long-acting insulin is used. Any interruption to insulin delivery or pump malfunctioning can cause DKA. To prevent this reaction, monitor blood glucose regularly. Insulin pump users also need to check often to see that insulin is still flowing through the tubing.

Patient Education—Foodborne Illness

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

For More Information

- American Diabetes Association
  http://www.diabetes.org/type-1-diabetes/ketoacidosis.jsp
- Merck manual – DKA
  http://www.merck.com/mmpe/sec12/ch158/ch158c.html
- NIDDK–Diabetic Ketoacidosis
  http://diabetes.niddk.nih.gov/DM/pubs/dictionary/K-O.htm

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Trachtenbarg DE. Diabetic ketoacidosis. *Am Fam Physician.* 71:1705, 2005.
Umpierrez GE, et al. Insulin analogs versus human insulin in the treatment of patients with diabetic ketoacidosis: a randomized controlled trial. *Diabetes Care.* 32:1164, 2009.
Weber C, et al. Prevention of diabetic ketoacidosis and self-monitoring of ketone bodies: an overview. *Curr Med Res Opin.* 25:1197, 2009.

HYPEROSMOLAR HYPERGLYCEMIC STATE

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

Diabetic HHS is a life-threatening emergency with marked elevation of blood glucose, hyperosmolarity, and minimal or no ketosis (Stoner, 2005). HHS is preventable if blood glucose is monitored regularly in order to correct elevating levels before problems occur. The condition occurs in patients older than age 70 years with T2DM and profound dehydration as precipitating factors. If recognized early, HHS can frequently be treated in the outpatient setting if the patient can take fluids. Table 9-18 provides diagnostic criteria. Sometimes DKA is the first time that diabetes is diagnosed.

Identification and treatment of the underlying and precipitating causes of HHS are absolutely essential. Predisposing factors for this syndrome include long-term uncontrolled hyperglycemia, pancreatic disease, infections or sepsis, stroke, surgery, extensive burns, renal or CVD, corticosteroid use, diuretics, excessive total parenteral nutrition (TPN), dialysis.

Underlying poor compliance with medications, infections, alcohol, or cocaine abuse are the most common causes. In children, corticosteroid use and gastroenteritis are common causes. The mortality rate is high at 10–20%. Areas of future research include prospective randomized studies to determine the pathophysiological mechanisms for the absence of ketosis in HHS and to investigate the reasons for elevated proinflammatory cytokines and cardiovascular risk factors (Kitabchi et al, 2008).

TABLE 9-18 Quick Sources of Glucose

4 oz grape, cranberry, or prune juice	19 g CHO
1 tbsp honey	17 g CHO
1/2 cup sweetened gelatin	17 g CHO
1 tbsp corn syrup, jam, jelly or glucose	15 g CHO
5 Lifesavers candies	15 g CHO
4 oz orange, apple, pineapple, grapefruit juice	12–15 g CHO
4 oz regular soft drink	13 g CHO
1 tbsp sugar, dissolved in water	13 g CHO
2 tablespoons raisins	10–15 g CHO
5–6 Lifesaver candies	10–15 g CHO
1 cup 2% or skim milk	12 g CHO
3–4 glucose tablets	4–5 g CHO each

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: HHS is acquired and not genetic, although T2DM has a genetic component. African Americans, Hispanics, and Native Americans have the highest rates.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP (hypotension?)
- I & O
- Profound dehydration (8–12 L)
- Fever or hypothermia?
- Excessive thirst and urination
- Dry, parched mouth
- Sunken eyes
- Dry skin with no sweating
- Weakness, lethargy
- Leg cramps
- Sleepiness or confusion
- Rapid pulse and abnormal respirations
- Convulsions, grand mal seizures, or coma

Lab Work

- Gluc (>600 mg/dL)
- Na^+, K^+
- Bicarbonate >15 mEq/L
- Serum osmolality (usually >320 mOsm)
- pH levels greater than 7.30
- Mild or absent ketonemia
- Elevated serum urea nitrogen (BUN)-to-creatinine ratio
- Alb
- H & H
- PO_4
- Chol, Trig
- CRP

INTERVENTION

OBJECTIVES

- Correct dehydration, shock, and cardiac arrhythmias.
- Reduce elevated blood glucose levels with isotonic saline, then hypotonic saline along with insulin.
- Monitor fluid status and replace deficits, which may be 10–20% of total body weight. This may require up to 9 L in 48 hours (Stoner, 2005).

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Alcohol Intake

Assessment Data: Blood glucose self-monitoring records with FBG >650; recent heavy intake of alcohol; fever; profound dehydration and signs of HHS; admitted to Emergency Room from a wedding reception; lives with son and daughter-in-law.

Nutrition Diagnosis (PES): Excessive alcohol intake related to lack of understanding about the impact of alcohol on diabetes as evidenced by confusion, profound dehydration, FBG >650, and fever after intake of four alcoholic beverages within 3 hours at a wedding reception.

Intervention: Teaching patient and family members about dangers of large doses of alcohol mixed with diabetes medication, especially with aging. Counseling about how to include an occasional alcoholic beverage and ways to participate in social events without excessive intake.

Monitoring and Evaluation: Improved glucose labs, resolution of HHS, fever and dehydration.

- Prevent future crises by appropriate DSME and regular monitoring of blood glucose.
- Prevent acute renal failure, which could result from prolonged hypovolemia.

FOOD AND NUTRITION

- Offer fluid replacement, often 1 L/h until volume is restored; 9–12 L may be needed.
- Patient is likely to nothing by mouth (NPO) during a crisis or perhaps tube fed during a comatose state. As appropriate, intake may be progressed gradually to a balanced diet, controlling calories as needed.
- Correct electrolyte deficits. Potassium or magnesium may be needed.
- The reported sodium level should be corrected when the patient's glucose level is markedly elevated. In this circumstance, extracellular fluid (ECF) osmolality rises and exceeds that of intracellular fluid (ICF), since glucose penetrates cell membranes slowly in the absence of insulin, resulting in movement of water out of cells into the ECF (Merck Manual, 2009). Types of fluids administered will depend on the corrected serum sodium level, calculated using the following formula: measured sodium + [(serum glucose − 100)/100) × 1.6].
- A renal diet plan may be needed if renal failure is identified.

Common Drugs Used and Potential Side Effects

- Insulin is needed to normalize blood glucose levels. Infusions will be needed until full rehydration is complete. DKA and HHS are associated with elevation of proinflammatory cytokines; insulin therapy provides a strong anti-inflammatory effect (Stentz et al, 2004).
- Potassium replacements may be needed. Monitor carefully.
- Antibiotic therapy may be needed in cases of underlying infections.
- Drugs that may precipitate HHS include diuretics, beta blockers, clozapine, olanzapine, H2 blockers, cocaine, alcohol.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss, where possible, predisposing factors, how to avoid future incidents, and blood glucose monitoring.
- CHO-controlled diets may be beneficial if patient can comprehend. Family intervention may be required.
- Discuss possible neglect or abuse, if suspected.
- Continuous management of the fluid, electrolyte, and glucose disturbances is necessary until resolved.
- Provide diabetes teaching to prevent recurrence. Adjust insulin or oral hypoglycemic therapy on the basis of the insulin requirement once serum glucose level are stable.
- Coordinate home visits from nursing or dietitian if needed to evaluate the inadequate access to water.

Patient Education—Foodborne Illness

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

For More Information

- Diabetic Hyperosmolar Hyperglycemic State
  http://www.diabetes.org/type-2-diabetes/treatment-conditions/hhns.jsp
- E-medicine
  http://www.emedicine.com/emerg/topic264.htm

HYPEROSMOLAR HYPERGLYCEMIC STATE—CITED REFERENCES

Kitabchi AE, et al. Thirty years of personal experience in hyperglycemic crises: diabetic ketoacidosis and hyperglycemic hyperosmolar state. *J Clin Endocrinol Metab.* 93:1541, 2008.

Merck Manual. Hyperosmolar Hyperglycemic State. Web site accessed October 13, 2009, at http://www.merck.com/mmpe/sec12/ch158/ch158d.html.

Stentz FB, et al. Proinflammatory cytokines, markers of cardiovascular risks, oxidative stress, and lipid peroxidation in patients with hyperglycemic crises. *Diabetes.* 53:2079, 2004.

Stoner GD. Hyperosmolar hyperglycemic state. *Am Fam Physician.* 71:1723, 2005.

HYPOGLYCEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 3

DEFINITIONS AND BACKGROUND

Hypoglycemia occurs primarily in patients with T1DM. Except in diabetic patients receiving insulin or sulfonylureas, hypoglycemia is a rare disorder. True low blood sugar (70 mg/100 dL or lower) releases hormones such as catecholamines, which produce hunger, trembling, headache, dizziness, weakness, and palpitations.

Sources of bodily glucose are from dietary intake, glycogenolysis, and gluconeogenesis. The metabolism of glucose involves oxidation and storage as glycogen or fat. The body makes great effort to supply glucose for the CNS and red blood cells. Glycogenolysis is stimulated by secretion of glucagon from the alpha-cells of the pancreas; this is impaired in patients with T1DM.

As different causes can stimulate hypoglycemia, treatments will differ accordingly. Skipped or insufficient meals, errors in insulin dosing, taking too much insulin, alcohol consumption, or extra physical exertion can lead to hypoglycemic episodes. Oxidative stress and zinc release contribute to neuronal death after hypoglycemia (Suh et al, 2008). Recurrent episodes of iatrogenic hypoglycemia induce a state of hypoglycemia unawareness and defective counterregulation, which defines hypoglycemia-associated autonomic failure (HAAF).

It is essential to manage these episodes carefully. A prooxidant state is promoted in certain brain regions during hypoglycemia and after glucose reperfusion, which results from the activation of several oxidative stress pathways; subsequent cell death occurs in particular brain regions including the cerebral cortex, the striatum, and the hippocampus (Haces et al, 2010).

Approaches to the prevention of hypoglycemia include glucose monitoring, patient education, meal planning, and medication adjustment. Adequate carbohydrate replacement during and after exercise seems to be an important measure to prevent hypoglycemia. Insulin dosage adjustment with a decrease from 20% to 30% is needed only for exercise duration over an hour (Grimm et al, 2004).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: A genetic deficiency in the p47(phox) subunit of NADPH oxidase may be relevant (Suh et al, 2008).

Clinical/History

- Height
- Weight
- BMI
- Diet history
- I & O
- Temperature
- BP
- Trembling
- Headache
- Dizziness, weakness
- Palpitations
- Seizures or coma
- Blood glucose and insulin regimen records
- Exercise history and habits
- Hypoglycemia unawareness?
- Type and duration of diabetes

Lab Work

- Serum Gluc (≤70 mg/dL)
- HbA1c
- Serum insulin
- Na^+, K^+
- Alb
- CRP
- Chol, Trig
- Ca^{++}
- Mg^{++}
- OGTT results using 1 g glucose/kg

INTERVENTION

OBJECTIVES

- Normalize blood glucose levels. If the problem is recurrent, stabilize blood glucose levels through consistent mealtimes, CHO consistency, insulin dose adjustments, and blood glucose self-monitoring. For those individuals on insulin, appropriate insulin timing with food or use of CHO to insulin ratio are important factors to consider.
- Minimize length of time between meals.
- Prevent seizures and coma, with precipitating symptoms such as neuroglycopenia with confusion, light headaches, and aberrant behavior.
- Determine frequency, symptoms of hypoglycemia, activity levels for the individual.
- Delayed hypoglycemia after strenuous or prolonged exercise may occur up to 24 hours after exercise and is related to increased insulin sensitivity from exercise as well as

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Physical Activity

Assessment Data: Blood glucose self-monitoring records, food diary worksheets and meal records, blood glucose levels (fasting, 2-hour postprandial and/or HbA1c levels). Recent exercise program including aerobic dancing for >1 hour thrice weekly. Low blood glucose levels and signs/symptoms of hypoglycemia twice weekly.

Nutrition Diagnosis (PES): Excessive physical activity related to a new exercise regimen as evidenced by two bouts of blood glucose levels below 70 in past week.

Intervention: Education of patient, exercise partner, and family member(s) about blood glucose self-monitoring records (timing, blood glucose levels before and after exercise), insulin dosing, and activity records. Counseling about timing of FBG testing, insulin reductions, and close monitoring before and after dance sessions.

Monitoring and Evaluation: Glucose lab results within desirable range; no symptoms of hypoglycemia. Food diary and activity records showing effective self-management.

repletion of glycogen stores. Plan appropriately by increasing carbohydrate, decreasing insulin for periods of activity, and increasing frequency of blood glucose monitoring.
- Always recheck blood glucose 15–20 minutes after treatment to make sure that problem is resolved. Very low blood glucose levels (<50 g/dL) may take 30 g CHO or more to resolve, especially after exercise.

FOOD AND NUTRITION

- For insulin-induced hypoglycemia, use a normal diet with adequate CHO content. Consider a possible reduction of medication if the hypoglycemia is recurrent.
- Have patient ingest fruit juice when needed or use candy for an immediate corrective measure. If there are symptoms of hypoglycemia (blood glucose <70 g/dL), carry quick sources of glucose (see Table 9-18). Fat is not as effective as CHO in normalizing blood glucose.
- In most cases, mild hypoglycemia can be handled with use of readily available CHOs, including milk, fruit, and crackers. Balanced, regular mealtimes or frequent small snacks are also useful.
- For hypoglycemia at night that is caused by excessive insulin or insufficient dinner meal or evening snack, adjust evening and bedtime doses of insulin. A slightly larger dinner or snack containing CHO may be needed.

Common Drugs Used and Potential Side Effects

- Insulin and other glucose-lowering medications must be carefully prescribed and monitored. Careful use of sliding scales of insulin is the goal (Smith et al, 2005). Glycemic control to a lower glucose target range can be achieved using a computerized insulin dosing protocol with particular attention to timely measurement and adjustment of doses (Juneja et al, 2009).
- Glucose tablets have 4–5 g CHO per tablet. Patients should receive instructions regarding quantity, when to use, and when not to use. Most people will need three to four tablets to treat low blood glucose levels.
- If the individual passes out from hypoglycemia and uses insulin on a daily basis, it may be necessary to give a glucagon shot and call for emergency assistance. Glucagon can cause vomiting. Intravenous dextrose is administered by medical professionals.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Clinical trials are lacking for this condition.
- See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Teach benefits of frequent blood glucose monitoring. Explain signs, symptoms, and treatment of hypoglycemia.
- Review appropriate timing of meals, snacks, and medications. Promote regular mealtimes, meal spacing, and planned exercise.
- Teach routine blood glucose before and after exercise. Discuss carrying a quick source of glucose.
- Encourage patients to obtain and carry diabetes identification.
- Discuss observations that require medical attention. Teach when to contact physician for medication adjustment.
- Discuss use of alcoholic beverages and potential effects. Excess alcohol increases the risk of hypoglycemia. Alcohol is processed in the liver to acetaldehyde at a time when the liver cannot do gluconeogenesis because nicotinamide adenine dinucleotide (NAD) is depleted. The biggest risk occurs when a patient drinks alcohol without carbohydrates being available, such as 4 hours or longer after the last meal. One drink takes 1–1.5 hours to process in the liver. Women should stick to one alcoholic beverage a day; men should limit their intake to two per day; both should consume a CHO with their drinks.
- In the elderly who are monitoring blood glucose levels closely, the benefits of intensive therapy in an effort to lower A1c must be weighed against the greater risk of unpredictable hypoglycemia (Alam et al, 2005).

Patient Education—Foodborne Illness

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

For More Information

- Mayo Clinic
  http://www.mayoclinic.com/health/hypoglycemia/DS00198
- NIDDK–Hypoglycemia in Diabetes
  http://diabetes.niddk.nih.gov/dm/pubs/hypoglycemia/

HYPOGLYCEMIA—CITED REFERENCES

Alam T, et al. What is the proper use of hemoglobin A1c monitoring in the elderly? *J Am Med Dir Assoc.* 6:200, 2005.

Grimm JJ, et al. A new table for prevention of hypoglycaemia during physical activity in type 1 diabetic patients. *Diabetes Metab.* 30:465, 2004.

Haces ML, et al. Selective vulnerability of brain regions to oxidative stress in a non-coma model of insulin-induced hypoglycemia. [published online ahead of print October 7, 2009] *Neuroscience.* 165:28, 2010.

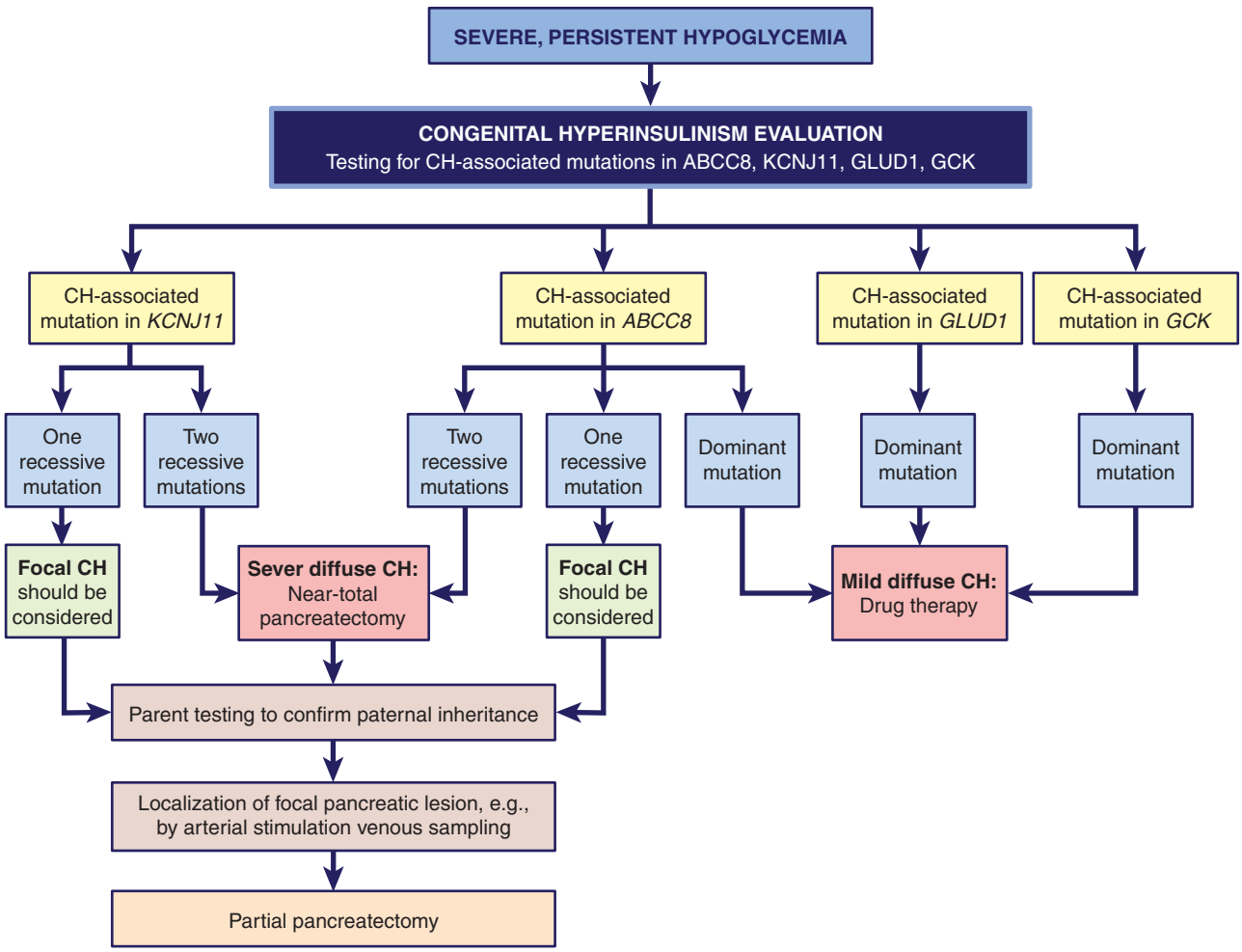
Juneja R, et al. Computerized intensive insulin dosing can mitigate hypoglycemia and achieve tight glycemic control when glucose measurement is performed frequently and on time. *Crit Care.* 13:165, 2009.

Smith WD, et al. Causes of hyperglycemia and hypoglycemia in adult inpatients. *Am J Health Syst Pharm.* 62:714, 2005.

Suh SW, et al. Sequential release of nitric oxide, zinc, and superoxide in hypoglycemic neuronal death. *J Cereb Blood Flow Metab.* 28:1697, 2008.

HYPERINSULINISM AND SPONTANEOUS HYPOGLYCEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 3–4



DEFINITIONS AND BACKGROUND

Hyperinsulinism consists of insulin levels above 3 μU/mL when glucose levels are below 50 mg/dL. Treatment depends on the cause and the severity of the hyperinsulinism. **Congenital hyperinsulinism (CH)** is familial hyperinsulism (FHI) with profound hypoglycemia because of excessive insulin secretion. CH can lead to developmental delay, mental retardation or death if untreated. CH occurs at an approximate frequency of 1/25,000 to 1/50,000 live births. New procedures spare healthy cells in the pancreas while allowing surgeons to remove abnormal tissue. **Spontaneous hypoglycemia** is an underlying symptom of some other diseases besides diabetes. Fasting and reactive hypoglycemias fall into the category of spontaneous forms. In **fasting hypoglycemia**, the body is not able to maintain adequate levels of sugar in the blood after a period without food in heavy drinkers who do not eat; people with islet cell tumors, viral hepatitis, cirrhosis, or liver cancer; and children with carbohydrate metabolic disorders. Hereditary fructose intolerance, galactosemia, leucine sensitivity can cause this type of hypoglycemia. **Reactive hypoglycemia** describes recurrent episodes of hypoglycemia occurring 2–4 hours after a high CHO or glucose load. Etiologies include alimentary hypoglycemia (dumping syndrome following gastric surgery); hypothyroidism or other hormonal disorders; *h. pylori*-induced gastritis; or occult diabetes with an exaggerated hyperglycemia during a glucose tolerance test. Symptoms include weakness and agitation 2–4 hours after meals, perspiration, nervousness, and mental confusion. Diet remains the main treatment, although alpha-glucosidase inhibitors and some other drugs may be helpful. Brain glycogen supports energy metabolism when glucose supply from the blood is inadequate and its levels rebound to levels higher than normal after a single episode of moderate hypoglycemia (Oz et al, 2009). Glutamate serves important intracellular signaling functions; glutamate dehydrogenase (GDH) catalyzes the oxidative deamination of glutamate to alpha-ketoglutarate in brain, liver, kidney, and the pancreatic islets (Stanley, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Individuals with congenital hyperinsulinism (CH) should be tested for gene mutations. Mutations in *GLUD1, GCK,* or *HADHSC* are response to drug therapy, but CHI associated with mutations in *ABCC8* or *KCNJ11* often requires pancreatectomy.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Recent weight losses
- Craving for sweets
- BP
- Seizures, confusion
- Heart palpitation or fibrillation
- Dizziness
- Lightheadness, tremors
- Headache
- Flushing
- Irritability
- Numb or cold extremities

Lab Work

- Gluc
- Serum insulin
- Serum glucagon
- Growth hormone levels
- Chol
- Trig
- Na^+, K^+
- Trig
- HgA1c
- Ca^{++}, Mg^{++}
- Acetone
- PO_4
- CRP

INTERVENTION

OBJECTIVES

- Reduce intake of concentrated CHO to a level that does not overstimulate the pancreas to secrete inappropriately large amounts of insulin, which may cause blood sugar to drop. Limit total intake of carbohydrate to 130 g/d to reduce the severity of symptoms.
- If required, ensure that patient loses weight gradually.
- Reduce counterregulatory hormone responses to excessive insulin.
- Exercise regularly and maintain consistency in meal times and daily routines.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive CHO Intake

Assessment Data: Recent bouts of low blood glucose, (<50 mg/gL) without Hx of diabetes or insulinoma. Dumping syndrome 2–3 hours after meals with weakness, perspiration, heart palpitations, headache, and craving for sweets. Diet history reveals CHO intake averaging 250 g/d, especially between meals and before bedtime.

Nutrition Diagnosis (PES): Excessive CHO intake related to high intake of sweets as evidenced by diet history.

Intervention: Food and nutrient delivery—tracking of CHO intake at meals and snacks. Education—sources of CHO from each food group. Counseling—ways to decrease hunger; food diary for episodes of hypoglycemia; signs and symptoms and when to call the doctor; glycemic index and glycemic load of foods.

Monitoring and Evaluation: Improvement through decreased episodes of hypoglycemia following meals and snacks with limitation of intake to 130–150 g daily.

FOOD AND NUTRITION

- Diet should include frequent small meals, about every 3 hours. Eat a variety of foods, including meat, poultry, fish, or nonmeat sources of protein; whole grains; fruits and vegetables; dairy products.
- Choose high-fiber foods and food with a moderate-to-low glycemic load. Avoid white rice, potatoes, corn.
- Avoid or limit foods high in sugar, especially on an empty stomach; this includes sweetened beverages and dried fruits.
- Avoid alcohol, caffeine.
- Maintain protein intake at DRI levels; fat furnishes the remainder of calories. The traditional high protein, low CHO diet is not evidenced-based.
- For the GLUD1 genetic form of HI, a low leucine diet and use of diazoside may be needed. Most of the other genetic forms will require surgery or just drug use.

Common Drugs Used and Potential Side Effects

- Diazoside is needed for the GLUT1 form of HI. Diazoxide is believed to inhibit insulin secretion through opening KATP channels. Octreotide (a somatostatin analogue) or continuous dextrose is often used; but in KATP HI, drug therapy fails, and pancreatectomy is required.
- Chemotherapy (e.g., streptozocin, fluorouracil) may be used for insulinomas. Monitor GI side effects and nephrotoxicity.
- Alpha-glucosidase inhibitors may be where diabetes is identified.
- Monitor all medications for their potential hypoglycemic effects.

Herbs, Botanicals, and Supplements

- Products such as hemicelluose and pectin may help in dumping syndrome.
- Herbs and botanical supplements should not be used without discussing with physician.
- See Table 9-9 for guidance on more specific herbs and supplements.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain that alcohol blocks gluconeogenesis and should be avoided.
- Ensure that patient keeps snacks available such as cheese and crackers. More frequent feedings are needed.

- Avoid skipping meals; eat meals on time. Avoid any one meal that is unbalanced or especially high in carbohydrates.
- Control caffeine intake and use of other stimulants, which may aggravate condition.

Patient Education—Foodborne Illness

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

For More Information

- Congenital Hyperinsulinism
  http://www.chop.edu/service/congenital-hyperinsulinism-center/home.html?id=47690
- Congenital Hyperinsulinism International
  http://www.congenitalhi.org/
- NIDDK–Hypoglycemia (Nondiabetes)
  http://diabetes.niddk.nih.gov/dm/pubs/hypoglycemia/#nodiabetes

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Oz G, et al. Human brain glycogen metabolism during and after hypoglycemia. *Diabetes* 58:1978, 2009.

Stanley GA. Regulation of glutamate metabolism and insulin secretion by GDH in hypoglycemic children. *J Am Clin Nutr.* 90:862S, 2009.

Other Endocrine Disorders

Hormones can be separated into three categories: **amines**, these are simple molecules, **proteins and peptides**, which are made from chains of amino acids, and **steroids**, which are derived from cholesterol. **Glands** discharge hormones directly into the bloodstream, and they have feedback mechanisms that maintain a proper balance of hormones and prevent excess secretion. Endocrine disorders require varying levels of nutritional intervention. If medications are taken appropriately, many conditions can be readily managed. See Table 9-19.

TABLE 9-19 Other Endocrine Conditions

There are many other disorders of endocrine function besides those affecting the pancreas that require some level of nutritional intervention. Listed below are essential hormones and functions of the endocrine glands.

Gland	Hormones	Functions
Pancreas	Glucagon (from alpha-cells)	Alpha-cells in the pancreatic islets secrete the hormone glucagon in response to a low concentration of glucose in the blood.
	Insulin (from beta-cells)	Beta-cells in the pancreatic islets secrete insulin in response to high blood glucose concentrations. See diabetes section.
Gastric mucosa	Gastrin	Stimulates production of hydrochloric acid and the enzyme pepsin, used in digestive processes.
Small intestinal mucosa	Secretin	Stimulates the pancreas to produce bicarbonate-rich fluid to neutralize stomach acid.
	Cholecystokinin	Stimulates contraction of the gallbladder to release bile after a meal containing fat. Also stimulates the pancreas to secrete digestive enzymes.
Placenta	Human chorionic gonadotropin	Signals the mother's ovaries to secrete hormones to maintain the uterine lining during pregnancy.
Hypothalamus	Gonadotropin-releasing hormone (GnRH) and other releasing hormones	Hunger and thirst; emotional and sexual responses of the limbic system; heart rate and blood pressure; circadian cycles; body temperature; bladder function; moods. Hypothalamus links the nervous system by synthesizing and secreting neurohormones that stimulate release of hormones from the anterior pituitary gland.
Pituitary (anterior)	Adrenocorticotropic hormone (ACTH)	Reacts with receptor sites in the cortex of the adrenal gland to stimulate the secretion of cortical hormones, particularly cortisol.
	Human growth hormone (hGH or somatotropin hormone)	Growth of bones, muscles, and other organs by promoting protein synthesis; influences height.
	Thyroid-stimulating hormone (TSH or thyrotropin)	Causes thyroid to secrete thyroid hormone.
	Gonadotropins (follicle-stimulating hormone [FSH]; luteinizing hormone [LH])	React with gonads to regulate growth and function of these organs. In females, FSH stimulates egg production; in males, it stimulates sperm production. In females, LH causes ovulation; in males, it causes the testes to secrete testosterone.
	Prolactin (PRL)	Promotes development of glandular tissue in the female breast during pregnancy and stimulates milk production after the birth of the infant.
Pituitary (posterior)	Oxytocin (OT)	Causes contraction of the smooth muscle in the wall of the uterus. It also stimulates the ejection of milk from the lactating breast.
	Vasopressin (antidiuretic hormone [ADH])	ADH promotes the reabsorption of water by the kidney tubules; less water is lost as urine to conserve water for the body. Insufficient amounts of ADH cause excessive water loss in the urine.

(continued)

TABLE 9-19 Other Endocrine Conditions *(continued)*

Gland	Hormones	Functions
Pineal gland	Melatonin (5-methoxy-*N*-acetyl-tryptamine)	Reproductive development and daily sleep–wake cycles; skin blanching. Derived from tryptophan. Production is stimulated by darkness and inhibited by light. Beta-blockers decrease release of melatonin.
Adrenal cortex		Adrenal cortex has control of 28 hormones, all of which are steroids.
	Mineralocorticoids (such as aldosterone)	Aldosterone conserves sodium ions and water.
	Glucocorticoids (such as cortisol)	Cortisol increases blood glucose levels.
	Gonadocorticoids (sex hormones: androgens and estrogens)	Normal reproductive functioning.
Adrenal medulla	Epinephrine (adrenalin) and norepinephrine (noradrenalin)	These two hormones are secreted in response to stimulation by the sympathetic nerve, particularly during stressful situations. They cause faster heartbeat and increased blood glucose levels.
		A lack of hormones from the adrenal medulla produces no significant effects. Hypersecretion, usually from a tumor, causes prolonged or continual sympathetic responses.
Thyroid	Thyroid hormones: thyroxine (95%) and triiodothyronine (5%)	Growth and development, metabolism. Thyroid hormones need iodine.
	Calcitonin	Calcitonin is secreted by the parafollicular cells of the thyroid gland. This hormone opposes the action of the parathyroid glands by reducing the calcium level in the blood. If blood calcium becomes too high, calcitonin is secreted until calcium ion levels decrease to normal.
Thymus	Thymosin	Immunity; T cells; lymphocytes. Currently being studied for its possible role in AIDS and hepatitis.
Parathyroid glands	Parathyroid hormone (PTH)	Regulation of calcium and phosphorus; secreted in response to low blood calcium levels in order to increase those levels. PTH mobilizes calcium by increasing calcium resorption from bone and by raising calcium reabsorption in the proximal kidney tubule.
Gonads (testes and ovaries)	Androgens (testosterone)	Growth and development of the male reproductive structures; increased skeletal and muscular growth; enlargement of the larynx accompanied by voice changes; growth and distribution of body hair; increased male sexual drive.
	Estrogen and progesterone	Development of the breasts; distribution of fat evidenced in the hips, legs, and breasts; maturation of reproductive organs such as the uterus and vagina. Progesterone causes the uterine lining to thicken in preparation for pregnancy. Together, progesterone and estrogens are responsible for the changes that occur in the uterus during the female menstrual cycle.

Sources: Surveillance, Epidemiology, and End Results. Endocrine glands and their functions. Accessed July 1, 2005, at http://training.seer.cancer.gov/module_anatomy/unit6_3_endo_glnds.html and Cotterill S. The endocrine system (hormones). Accessed July 1, 2005, at http://www.cancerindex.org/medterm/medtm12.htm#function

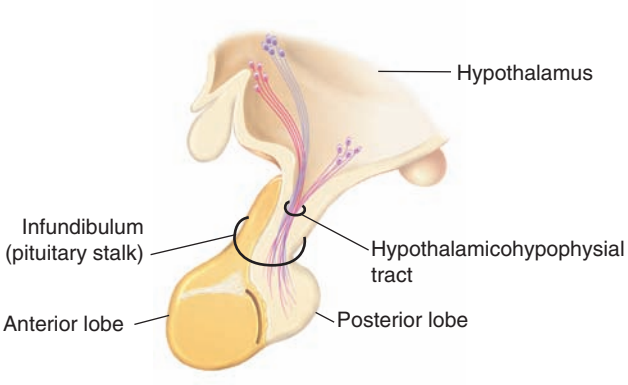
For More Information on Other Endocrine Conditions

- American Association of Clinical Endocrinologists
  http://www.aace.com/
- American Medical Association
  http://www.ama-assn.org/ama/pub/category/7157.html
- Endocrine Society
  http://www.aace.com/
- The Hormone Foundation
  http://www.hormone.org/
- National Institutes of Health–Endocrine Disorders
  http://www.niddk.nih.gov/health/endo/endo.htm

PITUITARY GLAND

HYPOPITUITARISM

NUTRITIONAL ACUITY RANKING: LEVEL 1



Asset provided by Anatomical Chart Co.

DEFINITIONS AND BACKGROUND

Hypopituitarism is an underactive pituitary gland. A deficiency in production of pituitary hormones may be caused by tumor, trauma, radiation to the brain, stroke or aneurysm, or surgery.

In hypopituitarism, there is a lack of one or more of these hormones and loss of function in the affected gland or organ. Table 9-20 lists symptoms of pituitary disorders. It may take years for an accurate diagnosis to be made. If all pituitary hormones are missing, this is panhypopituitarism; it is relatively rare.

Adrenocorticotropic hormone (ACTH) stimulates the adrenal gland to release cortisol to maintain BP and blood glucose levels. If ACTH is missing, depression, fatigue, low BP, nausea and diarrhea, dizziness, pale skin, weakness, and weight loss are signs and symptoms. A shortage of cortisol can be life threatening.

Arginine vasopression (AVP) was formerly known as antidiuretic hormone (ADH). AVP controls water loss by the kidneys. If AVP is deficient, severe thirst and excessive urination occur; diabetes insipidus may result. In rare instances, deficiency may occur after an event such as brain surgery.

TABLE 9-20 Symptoms of a Pituitary Disorder

Headaches	Short stature if during growth period
Depression	Cold intolerance
Mood/emotion swings	Weight loss
Abdominal pain, anorexia	Infertility in women
Loss of memory	Impotence in men
Loss of sleep	Cessation of or irregular menses
Sexual dysfunction	Lethargy, fatigue
Failure in lactation	Weakness
Low blood pressure	Loss of armpit, body, pubic or facial hair

Source: Pituitary Network Association. Available at http://www.pituitary.org/

Growth hormone (GH) regulates somatic growth, carbohydrate and lipid metabolism, and adipocyte functions. There is a complex interplay between GH and insulin signaling (Perrini et al, 2008). If GH is deficient in children, short stature (below 5 feet) can result. This condition causes 10% of all dwarfism. In adults, there is abnormal body composition, osteopenia, impaired quality of life, cardiac dysfunction, and an adverse lipid profile. Follicle-stimulating hormone (FSH) and Luteinizing hormone (LH) control sexual function and fertility in males and females. When gonadotropin (FSH, LH) deficiency occurs, men and women will lose interest in sex and can experience fatigue, weakness, loss of body hair, impotence in men, and loss of menstruation in women. Oxytocin stimulates the uterus to contract during labor and the breasts to release milk. Oxytocin mechanisms are necessary for successful pregnancies (Kubler et al, 2009). Pregnancy is uncommon when oxytocin levels are low.

Prolactin stimulates female breast development and milk production. Prolactin deficiency is rare but can stop milk production in women.

TSH stimulates the thyroid gland to release hormones that affect the body's metabolism. When TSH is deficient, this can lead to an underactive thyroid (hypothyroidism). Cold intolerance, constipation, weight gain, and pale and waxy or dry skin can occur.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Transcription factors have an impact on target genes; different phenotypes result when the gene encoding the relevant transcription factor is mutated. Many genes are involved.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- Brain scan
- Pituitary MRI
- Dual-energy x-ray absorptiometry (DEXA) scan

Lab Work

- ACTH level
- Cortisol
- Serum estradiol
- Serum testosterone
- Serum IGF-1
- Thyroxine (T4) (decreased?)
- Triiodothyronine (T3)
- FSH, LH, TSH (decreased?)
- Protein-bound iodine (PBI) uptake (decreased?)
- Glucose
- Glucose tolerance test (GTT)
- Chol, Trig
- Homocysteine
- CRP
- Alb
- Complete blood count (CBC)

Osmolarity	Serum Gluc	H & H, serum
Na^+, K^+	Uric acid	ferritin
Ca^{++}, Mg^{++}		Transferrin

INTERVENTION

OBJECTIVES

- Replenish missing hormones.
- Prevent dehydration, hypoglycemia, and related problems.
- Improve lean muscle mass stores.
- Monitor serum levels of cholesterol and triglycerides; prevent vascular complications.

FOOD AND NUTRITION

- Dietary alterations may be needed, such as higher or lower energy intake, until hormone levels are normalized. A modified fat, cholesterol, and carbohydrate intake may be needed. Ensure sufficient intake of protein.
- Six small feedings may be better tolerated than larger meals.
- Increase fluids unless contraindicated.
- Ensure adequate intake of all vitamins and minerals. Calcium and vitamin D should be taken in sufficient amounts to prevent osteoporosis.

Common Drugs Used and Potential Side Effects

Hormone replacement therapy may include any of all of the following:

1. Corticosteroids (hydrocortisone [Cortef], cortisol) are often used and can alter glucose, calcium, and phosphate tolerance. Potassium and folacin must be increased; sodium must be decreased. Monitor for signs of hyperglycemia.
2. Thyroid preparations (levothyroxine) may be needed.
3. GH (somatotropin) requires no specific dietary interventions. It may help alleviate elevated triglycerides. Long-term GH replacement therapy in adults is safe for lifelong therapy in order to maintain the benefits.
4. Estrogen, progesterone, or testosterone replacement should be monitored for side effects related to heart disease and elevated lipids.
5. Cortisone may be needed during periods of stress or illness if ACTH is deficient.

SAMPLE NUTRITION CARE PROCESS STEPS

Unsafe Food Handling

Assessment Data: On home tube feeding. Unsanitary habits noted during home visit.

Nutrition Diagnosis (PES): Unsafe food handling related to preparation and administration of tube feeding as evidenced by observations during home visit.

Intervention: Education on how to sanitize counters and maintain safe procedures while handling tubing and enteral product.

Monitoring and Evaluation: No signs of diarrhea or GI distress from contaminated TF.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Have patient avoid fasting and stress.
- Discuss the need to use small, frequent meals instead of large meals.
- Discuss the possibility of hyperglycemia and how to manage.
- Hormone replacement is usually permanent, so doctor visits will be needed to check for diabetes and signs of osteoporosis.

Patient Education—Foodborne Illness

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

For More Information

- Hormone Foundation–Pituitary Hormone
  http://www.hormone.org/public/pituitary.cfm
- Mayo Clinic
  http://www.mayoclinic.com/health/hypopituitarism/DS00479
- Medline Plus
  http://www.nlm.nih.gov/medlineplus/pituitarydisorders.html
- Pituitary Disorders Education and Support
  http://www.pituitarydisorder.net/
- The Pituitary Society
  http://www.pituitarysociety.org/

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Perrini S, et al. Metabolic implications of growth hormone therapy. *J Endocrinol Invest.* 31:79, 2008.

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PITUITARY GLAND (ANTERIOR)

ACROMEGALY

NUTRITIONAL ACUITY RANKING: LEVEL 1

Adapted from: Weber J RN, EdD and Kelley J RN, PhD. *Health Assessment in Nursing*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.

DEFINITIONS AND BACKGROUND

Acromegaly is a hormonal disorder caused by overproduction of human GH by the pituitary gland. Incidence is rare, with 50–70 cases per million in the U.S. population. Diagnosis is made common about a decade after oversecretion of GH begins. If GH-producing tumors occur in puberty, the condition is called gigantism. Genetics and nutrition impact the height of most children; unusual, continuing growth in stature beyond the twenties should be evaluated.

GH (somatotropin) affects the growth of almost all cells and tissues and has direct and indirect effects. Direct effects of excessive GH include hyperinsulinism, lipolysis, insulin resistance in peripheral tissues, ketogenesis, hyperglycemia, and sodium and water retention. In over 98% of cases, overproduction of GH is related to a benign pituitary tumor. In a few patients, acromegaly is caused by tumors of the pancreas, lungs, or adrenal glands. Symptoms and signs of acromegaly include enlarged extremities with disproportionate growth of nose, lips, brow, lower jaw, tongue, hands, and feet. Serious side effects include heart failure, colon polyps that become cancerous, and diabetes.

Elevated insulin-like growth factor I (IGF-I) also occurs in acromegaly, resulting in greater protein synthesis, amino acid transportation, muscle and bone growth, DNA and RNA synthesis, and cell proliferation. High levels of GH and increased IGF-1 levels (three to ten times above normal) may be used to diagnose acromegaly. Hormone-secreting pituitary tumors account for about 30% of all pituitary tumors (Patil et al, 2009). Surgical removal of the pituitary gland provides a 50–70% chance of cure (Vance and Laws, 2005). Acromegaly is complicated by an increased incidence of diabetes mellitus caused by impaired insulin sensitivity and reduced beta-cell function (Higham et al, 2009). Bone and cartilage growth may lead to arthritis. Acromegalic patients present with hypoadiponectinemia; studies suggest a link between adiponectin, visfatin, fat mass, and bone changes (Sucunza et al, 2009). Premature death may result if left untreated. Treatment may include surgical removal of the tumor, radiation therapy, or injection of a GH blocking drug. Somatostatin analogs have been key in medical therapy. Pegvisomant, a GH-receptor antagonist, competitively binds to the GH receptor, blocking IGF-I production and allowing for a better control of cardiac disorders and glucose metabolism (Vance and Lawes, 2005). Improved insulin sensitivity occurs after use from a reduction in overnight endogenous glucose production (Higham et al, 2009). Somatuline Depot (lanreotide) can be used for a long-term treatment in patients who have had an inadequate response to surgery and/or radiation.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Most pituitary tumors are from a genetic mutation that is acquired and not present from birth.

Clinical/History

- Height
- Weight
- BMI
- Weight changes
- Diet history
- BP (increased)
- I & O
- Increased coarse body hair
- Coarse, leathery skin
- Excessive diaphoresis and oily skin
- Skin tags
- Impaired vision
- Osteoarthritis, carpal tunnel syndrome
- Deepening voice
- Headaches
- Fluid retention

Lab Work

- IGF-I levels (best marker)
- GH (>5× higher than normal)
- N balance
- BUN
- Serum Creat (increased)
- Urine sugar
- Gluc, HbA1c
- GTT
- Serum insulin
- Ca^{++} (decreased)
- Phosphorus (increased?)
- Alb
- Na^{+}, K^{+}
- H & H

INTERVENTION

OBJECTIVES

- Control weight.
- Prevent or control diabetes, hypertension, or heart disease when present.
- Prevent osteoporosis with calcium balance, which is often negative.
- Monitor for complications such as colon polyps, which may lead to cancer.

SAMPLE NUTRITION CARE PROCESS STEPS

Involuntary Weight Gain

Assessment Data: Diagnosis of acromegaly with recent weight gain of 10 lb over 6 weeks; FBG 130 mg/dL.

Nutrition Diagnosis (PES): Involuntary weight gain (NC-3.4) related to excessive GH and IGF-1 levels as evidenced by 10 lb weight gain in 6 weeks.

Intervention: Education—importance of controlling CHO and energy intake to manage weight and prevent diabetes. Counseling—tips for identifying hidden CHO sources and controlling energy intake.

Monitoring and Evaluation: Weight stabilized; HgbA1c, FBS and other available lab results. No additional signs of hyperglycemia and no further weight gain.

- Achieve the goal for GH levels as 1–2 μg/L (Sheppard, 2005).

FOOD AND NUTRITION

- An CHO-controlled diet may be needed if diabetes is present.
- Extra fluid intake may be needed.
- Control sodium and fluid intake if there is heart failure.
- Offer sufficient intake of calcium and vitamin D; a multivitamin–mineral supplement may be useful.

Common Drugs Used and Potential Side Effects

- Bromocriptine (Parlodel) can be taken orally to reduce GH secretion. Side effects include gastrointestinal upset, nausea, vomiting, light-headedness when standing, and nasal congestion. Take with food.
- Octreotide (Sandostatin) injection is a synthetic form of somatostatin. Side effects include diarrhea, nausea, gallstones, and loose stools.
- Insulin may be needed if diabetes is also present. Be wary of excess doses; hypoglycemia is a dangerous side effect.
- Cardiac medications may be needed; monitor for specific side effects accordingly. Atorvastatin treatment is safe, well-tolerated, and effective (Mishra et al, 2005).
- Pegvisomant is used to normalize circulating levels of IGF-I, the principal mediator of GH action.
- Somatuline Depot delivers an injection subcutaneously instead of into the muscle; side effects include diarrhea, cholelithiasis, abdominal pain, nausea, injection site reactions, flatulence, arthralgia, and loose stools.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss body changes and altered self-image. Exercise can help to improve physical functioning and quality of life (Woodhouse et al, 2006).
- Teach patient about control of diabetes or heart failure, where present.
- After surgery, potential complications include cerebrospinal fluid leaks, meningitis, and damage to the surrounding normal pituitary tissue, requiring lifelong pituitary hormone replacement.

Patient Education—Foodborne Illness

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

For More Information

- Acromegaly
  http://www.acromegaly.org/
- NIDDK–Acromegaly
  http://www.niddk.nih.gov/health/endo/pubs/acro/acro.htm
- Pituitary Network Association
  http://www.pituitary.com/
- Skull Base Institute
  http://www.skullbaseinstitute.com/acromegaly_gigantism.htm
- Treatment Guidelines
  http://www.aace.com/pub/pdf/guidelines/AcromegalyGuidelines2004.pdf

ACROMEGALY—CITED REFERENCES

Higham CE, et al. Pegvisomant improves insulin sensitivity and reduces overnight free fatty acid concentrations in patients with acromegaly. *J Clin Endocrinol Metab.* 94:2459, 2009.

Mishra M, et al. The effect of atorvastatin on serum lipoproteins in acromegaly. *Clin Endocrinol.* 62:650, 2005.

Patil CG, et al. Non-surgical management of hormone-secreting pituitary tumors. *J Clin Neurosci.* 16:985, 2009.

Sheppard MC. GH and mortality in acromegaly. *J Endocrinol Invest.* 28:75S, 2005.

Sucunza N, et al. A link between bone mineral density and serum adiponectin and visfatin levels in acromegaly. *J Clin Endocrinol.* 94:3889, 2009.

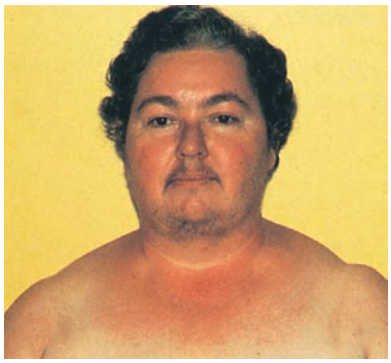
Vance ML, Laws ER Jr. Role of medical therapy in the management of acromegaly. *Neurosurgery.* 56:877, 2005.

Woodhouse LJ, et al. The influence of growth hormone status on physical impairments, functional limitations, and health-related quality of life in adults. *Endocr Rev.* 27:287, 2006.

Pituitary Gland (Anterior)

CUSHING'S SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: Rubin E. *Essential Pathology,* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2000.

DEFINITIONS AND BACKGROUND

Cushing's syndrome (CS) is a disease caused by an excess of cortisol. It can be caused by extrinsic and excessive hormonal stimulation of the adrenal cortex by tumor of the anterior pituitary gland, adrenal hyperplasia, or exogenous cortisol use. Differential diagnosis is not simple. No existing test is accurate when used alone; focused imaging, including computed tomography (CT), magnetic resonance imaging (MRI), and nuclear imaging modalities can provide the diagnosis (Lindsay and Nieman, 2005).

Pituitary CS occurs after puberty with equal frequency in boys and girls. In adults, it has a greater frequency in women than men, with most diagnosed between ages 20 and 50 years. The total incidence is about 10–15 million people per year. It is a disorder characterized by virilism, upper body obesity with thin arms and legs, hyperglycemia, glucosuria, hypertension, red moon face, vertigo, emotional liability, buffalo hump, purple striae over obese areas, acne, female balding or hirsutism, blurry vision, and pitting ankle edema. In some cases, osteoporosis and severe depression are present.

Chronic cortisol hypersecretion causes central obesity, hypertension, insulin resistance, dyslipidemia, and prothrombotic state, manifestations of a MetS. There is a complex interaction between CS and inflammation; raised levels of IL-8 and OPG in CS patients, despite glucocorticoid excess, may represent an inflammatory and pro-atherogenic phenotype (Kristo et al, 2008). If left untreated, CS can be fatal. Diagnosis is not simple. Late-night salivary cortisol may be a useful test (Carroll et al, 2009). Treatments differ according to the cause: ACTH dependent (pituitary or ectopic) or independent (an adrenal tumor), or iatrogenic (from excessive steroid hormone use). If iatrogenic, depletion of steroid hormones will be needed. If pituitary, the gland may need to be removed. CS caused by ACTH production from solid tumors can result in life-threatening hypercortisolemia (Uecker and Janzow, 2005). Radiation, chemotherapy, or surgery may be needed. After surgical removal of the adrenal glands, most symptoms of CS disappear. Psychological impairment can persist despite successful treatment (Iacabone et al, 2005).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Rarely, CS results from an inherited tendency to develop tumors of one or more endocrine glands. With multiple endocrine neoplasia type 1 (MEN1), hormone-secreting tumors of the parathyroid glands, pancreas, and pituitary develop and may lead to CS.

Clinical/History

Height
Weight
BMI
Diet history
BP (increased)
Edema
Buffalo hump
Moon face
Easy bruising, slow healing
Red or purple striae on skin
Weakened bones
Sore, aching joints in hip, back, shoulders
Excess facial hair in women
Decreased fertility in men

CT
MRI for tumors
DEXA scan

Lab Work

24-hour urinary free cortisol level (>50–100 μg/d)
Dexamethasone suppression test
Late-night salivary cortisol
Corticotropin-releasing hormone (CRH) stimulation test for ACTH levels (altered)
Urinary Gluc (increased)
Gluc (increased)
HbA1c
Ca^{++}, Mg^{++}
Urinary Ca^{+}
Lipid profile, Chol, Trig
K^{+} (decreased)
Na^{+} (increased)
Alb, N balance
CRP
pCO_2 (increased), pO_2
WBC, TLC (decreased)

INTERVENTION

OBJECTIVES

- Control elevated blood glucose or lipids; manage diabetes and CVD.
- Promote weight loss if needed; decrease fat stores while increasing lean body mass.
- Control or lower BP.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Sodium Intake

Assessment Data: Long-term corticosteroid use for lupus; dietary history reveals high intake of sodium from snack foods, canned soups and vegetables, luncheon meats; buffalo hump, slightly elevated glucose and lipids. BMI 27. BP averaging 190/80. Feet and hands edematous.

Nutrition Diagnosis (PES): Excessive sodium intake related to high intake of salted and processed foods as evidenced by diet history and requirement for corticosteroids.

Intervention: Education about a desirable lowering of sodium from dietary sources. Counseling about sources of well-flavored snack foods, use of more fruits and raw vegetables, use of varied spices and seasonings; choices in the supermarket, at restaurants, while traveling.

Monitoring and Evaluation: Evaluation of impact of lower sodium from diet on BP and edema. Improved intake of fruits, raw vegetables and nonprocessed meats. Improved quality of life.

- Prevent or control side effects of corticosteroid therapy: vertebral collapse, heart failure, bone demineralization, osteoporosis, and hypokalemia.

FOOD AND NUTRITION

- Restrict sodium if steroids are used.
- Use an energy-controlled diet, if needed. Calculate diet according to patient's desirable body weight.
- Control glucose levels when elevated; carbohydrate counting can be useful.
- Ensure adequate intake of calcium and potassium.
- Ensure adequate intake of protein if losses are excessive (e.g., 1 g protein/kg or more).
- Use an anti-inflammatory diet rich in omega-3 fatty acids, herbs, spices and antioxidant foods; see Tables 8-13 and 8-14.

Common Drugs Used and Potential Side Effects

- With glucocorticoid therapy, osteoporosis and hypercalciuria are common side effects. Withdrawal of these medications after autoimmune or cancer management may cause iatrogenic CS. Adrenal insufficiency or steroid withdrawal symptoms may occur (Hopkins and Leinung, 2005).
- Large doses of vitamin D may be necessary; do not use for extended periods without monitoring for toxicity.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help patient control weight as needed.
- Explain which foods are good sources of calcium in the diet.
- Explain how to control elevated blood sugars and lipids through balanced dietary intake.
- Manage symptoms of MetS through dietary changes and exercise.

Patient Education—Foodborne Illness

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

For More Information

- Cushing's Support and Research Foundation
  http://csrf.net/
- Hormone Foundation
  http://www.hormone.org/Other/upload/cushings-syndrome-billingual-032309.pdf
- NIDDK–Cushing's Syndrome
  http://www.niddk.nih.gov/health/endo/pubs/cushings/cushings.htm

CUSHING'S SYNDROME—CITED REFERENCES

Carroll T, et al. Late-night salivary cortisol for the diagnosis of Cushing syndrome: a meta-analysis. *Endocr Pract.* 15:335, 2009.

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Iacabone M, et al. Results and long-term follow-up after unilateral adrenalectomy for ACTH-independent hypercortisolism in a series of fifty patients. *J Endocrinol Invest.* 28:327, 2005.

Kristo C, et al. Biochemical markers for cardiovascular risk following treatment in endogenous Cushing's syndrome. *J Endocrinol Invest.* 31:400, 2008.

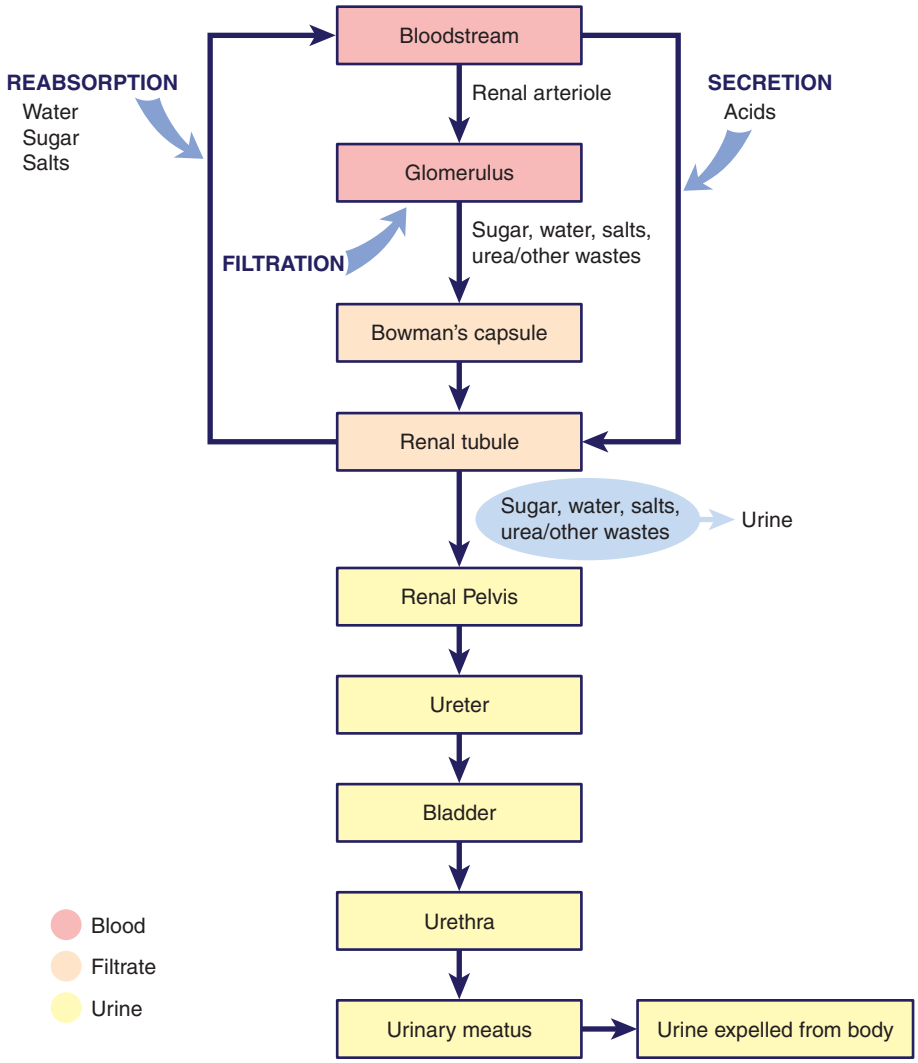
Lindsay JR, Nieman LK. Differential diagnosis and imaging in Cushing's syndrome. *Endocrinol Metab Clin North Am.* 34:403, 2005.

Uecker JM, Janzow MT. A case of Cushing syndrome secondary to ectopic adrenocorticotropic hormone producing carcinoid of the duodenum. *Am Surg.* 71:445, 2005.

PITUITARY GLAND (POSTERIOR)

DIABETES INSIPIDUS

NUTRITIONAL ACUITY RANKING: LEVEL 3



DEFINITIONS AND BACKGROUND

Diabetes insipidus (DI) can be caused by defects in the posterior pituitary gland or from an insufficient renal response to AVP, formerly ADH. Distinction is essential for effective treatment (Makaryus and MacFarlane, 2006). DI may be primary (congenital) or secondary (acquired after trauma, surgery, tumor, or infection). Both forms of DI are marked by excessive thirst, copious urination, and dry skin. There is potential for dehydration and weakness. Urine output may be 5–10 L/24 h.

Nephrogenic DI is characterized by the kidney's inability to respond to AVP. The multiple and complex functions of the renal tubule in regulating water, electrolyte, and mineral homeostasis make it prone to numerous genetic abnormalities (Chadra and Alon, 2009). Nephrogenic DI requires careful monitoring of body chemistry and adequate hydration.

Neurogenic (primary) DI is more common in males. Children with DI may be irritable or listless and may have problems with bedwetting, fever, vomiting, or diarrhea (Linshaw, 2007). Neurogenic DI responds to nasal administration of 1-deamino-8-D-arginine vasopressin (desmopressin acetate) (DDAVP), a vasopressin analogue.

Dipsogenic DI is a very rare forms of DI from a defect in the thirst mechanism in the hypothalmus.

Gestational DI (GDI) is also very rare. DI can complicate up to 1 in 30,000 pregnancies. If DI occurs with pre-eclampsia, the baby may have to be delivered early. DDAVP may be used (Ananthakrishnan, 2009).

Sometimes, the exact cause of DI is unknown. Patients undergoing surgery for pituitary tumors present with DI (Dumont et al, 2005). In acquired forms, the kidneys' ability to respond to AVP can be impaired by drugs such as lithium,

or by chronic disorders including polycystic kidney disease, sickle cell disease, kidney failure, partial blockage of the ureters, and inherited genetic disorders.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Abnormal posterior pituitary development is found in genetic forms of central DI. The exact genes are being studied.

Clinical/History	Lab Work	
Height	Arginine vasopressin (AVP) in serum, urine	Glucose
Weight	Urinary osmolality (may be <300 mOsm/kg)	Bicarbonate
BMI	Fluid deprivation test (neurogenic vs. nephrogenic forms)	Urinary specific gravity (decreased)
Diet history		Uric acid (increased in adults)
BP		BUN
Excessive thirst and urination		Creat
I & O		Na^+ (increased)
Fever?		K^+ (altered?)
Vomiting, diarrhea in children		Alb
Brain MRI		

INTERVENTION

OBJECTIVES

- Reduce urine osmolality and increase electrolyte-free water excretion.
- Raise serum sodium concentration.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Fluid Intake

Assessment Data: Polyuria, polydipsia; preference for cold beverages or ice. Signs of poor skin turgor and dehydration; pale urine. Diagnosis of nephrogenic DI.

Nutrition Diagnosis (PES): Inadequate fluid intake related to losses from frequent urination and diagnosis of DI as evidenced by pale urine, urination 10 times daily and during the night.

Intervention: Education about the importance of taking prescribed medication and increasing fluid intake because of Dx of DI. Counseling about sources of fluid from beverages and foods; discussion of signs of dehydration and when to contact the physician.

Monitoring and Evaluation: Reports of improved hydration status. Consistent use of medication. No emergency visits to the doctor or emergency room. Improved quality of life.

- Avoid fluid overload and rapid fluctuations in sodium concentration, especially in persons who cannot control their fluid intake themselves (Toumba and Stanhope, 2006).
- Check patient's weight three times weekly to determine fluid retention and effectiveness of drug therapy. At home, have weights recorded daily.
- Reduce excess workload for the kidney and prevent stone formation.

FOOD AND NUTRITION

- Adjust fluid, sodium, and potassium intakes according to the cause.
- A low-sodium diet and diuretics may also be needed to minimize workload of the kidney.
- Sometimes a controlled-protein diet is needed to protect renal function.

Common Drugs Used and Potential Side Effects

- DDAVP may cause abdominal pain, headaches, gastrointestinal distress, and weakness. It is administered parenterally, by pill, or by nasal spray. Patients should drink fluids or water only when thirsty, but be aware that a low urine volume is a risk factor for kidney stone formation (Mehandru and Goldfarb, 2005). Hyponatremic hypervolemia leading to seizures is a rare but potentially life-threatening side effect.
- If diuretics such as thiazides or amiloride are used, monitor for side effects. Potassium may be needed if a supplement is not used.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Fluid adjustments will be made according to the type of DI. Caution patients not to limit fluid intake in an effort to lessen urine output.
- Cold or ice water may be preferred.
- Select low-calorie beverages to prevent excessive weight gain.
- Avoid stimulant/diuretic-type beverages (e.g., coffee, tea, alcohol).

Patient Education—Foodborne Illness

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

For More Information

- Diabetes Insipidus Foundation, Inc. http://diabetesinsipidus.com
- Nephrogenic Diabetes Insipidus Foundation http://www.ndif.org/

DIABETES INSIPIDUS—CITED REFERENCES

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Chadra V, Alon US. Hereditary renal tubular disorders. *Semin Nephrol.* 29:399, 2009.

Dumont AS, et al. Postoperative care following pituitary surgery. *J Intensive Care Med.* 20:127, 2005.

Linshaw M. Back to basics: congenital nephrogenic diabetes insipidus. *Pediatr Rev.* 28:372, 2007.

Makaryus AN, MacFarlane SI. Diabetes insipidus: diagnosis and treatment of a complex disease. *Cleve Clin J Med.* 73:65, 2006.

Mehandru S, Goldfarb DS. Nephrolithiasis complicating treatment of diabetes insipidus. *Urol Res.* 33:244, 2005.

Toumba M, Stanhope R. Morbidity and mortality associated with vasopressin analogue treatment. *J Pediatr Endocrinol Metab.* 19:197, 2006.

PITUITARY GLAND

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE (SIADH)

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Syndrome of inappropriate antidiuretic hormone (SIADH) involves hyponatremia and hyperosmolarity of urine. Normal renal and adrenal functioning with abnormal elevation of plasma vasopressin occurs (inappropriate for serum osmolality). Hyponatremia and SIADH can occur among elderly long-term care patients with febrile illness (Arinzon et al, 2005). Other causes of SIADH are listed in Table 9-21.

Hyponatremia is usually the first symptom of SIADH. In severe hyponatremia, convulsions or coma can occur. Other signs and symptoms include irritability, lethargy, seizures, and confusion. SIADH or cerebral salt-wasting syndrome (CSWS) can occur after pituitary surgery; differential diagnosis can be difficult. SIADH syndrome often requires sodium replacement or use of loop diuretics.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: SIADH is usually acquired and not genetic.

Clinical/History

- Height
- Current weight
- Edema-free weight
- BMI
- Diet history
- I & O
- Temperature
- Edema
- Irritability
- Lethargy, confusion
- Low urine volume

Lab Work

- Serum Na^+ <135 µmol/L
- Serum osmolality <280 mOsm/kg
- Serum AVP (elevated)
- Urine osmolality >500 µmol/kg
- Urinary Na^+ (elevated)
- BUN (low, <10 mg/dL)
- Creat
- K^+
- Ca^{++}, Mg^{++}
- Bicarbonate (normal)
- Uric acid (may be low)
- GFR (increased)

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Sodium Intake

Assessment Data: Serum Na+ levels low at 110 mol/L; elevated urinary osmolality.

Nutrition Diagnosis (PES): Inadequate sodium intake related to salt wasting syndrome after pituitary surgery as evidenced by low serum Na+ (110) and high urinary mOsm.

Intervention: Food-nutrient delivery – Add extra salt to meals; send extra salt packet with delivered meals.

Monitoring and Evaluation: Normal serum Na+ and urinary osmolality at next lab visit.

TABLE 9-21 Causes of Syndrome of Inappropriate Antidiuretic Hormone

Acute leukemia
Brain abscess, stroke or meningitis
Drugs: chlorpropamide, cyclophosphamide, carbamazepine
Gillain-Barre syndrome
Head injury
Lung cancer (especially small-cell lung cancer)
Lymphoma
Olfactory neuroblastoma
Pancreatic cancer
Pituitary surgery
Pneumonia
Prostate cancer
Rocky Mountain spotted fever
Sarcoidosis
Temporal arteritis, polyarteritis nodosa

INTERVENTION

OBJECTIVES

- Treat the cause.
- Restrict water intake.
- Replace electrolytes as appropriate; usually intravenous saline is provided.
- Normalize hormone secretion through drug therapy.

FOOD AND NUTRITION

- Restrict fluid intake, usually 1000–1200 mL/d.
- Alter dietary sodium and potassium, as deemed appropriate for the condition. This will vary by patient condition and medications used.
- When enteral feeding is needed, select a formula that is fluid restricted, such as those that are 2 kcal/mL. Monitor carefully for signs of dehydration. Check content of formula for sodium and potassium; select according to patient status and needs.

Common Drugs Used and Potential Side Effects

- Demeclocycline (Declomycin) may be used with side effects like those of tetracycline. Avoid taking with calcium or dairy products.
- Conivaptan is a new agent available for use to antagonize the effects of vasopressin, especially in heart failure patients (Schwarz and Sanghi, 2006).
- Hyponatremia as a result of SIADH is a relatively common serious side effect of the use of selective serotonin reuptake inhibitors (SSRIs) in (mostly elderly) adults (Vanhaesebrouk et al, 2005).

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Provide counseling regarding water and fluid restrictions as ordered.
- Discuss any underlying conditions that may have caused the syndrome; highlight needed dietary alterations.

Patient Education—Foodborne Illness

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

For More Information

- E-medicine
  http://www.emedicine.com/ped/topic2190.htm
- National Library of Medicine–Dilutional Hyponatremia
  http://www.nlm.nih.gov/medlineplus/ency/article/000394.htm

SIADH—CITED REFERENCES

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OVARY

POLYCYSTIC OVARIAN DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Polycystic ovarian disease (PCOD; or polycystic ovarian syndrome [PCOS]) is an endocrine disorder characterized by hyperandrogenism, bilaterally enlarged polycystic ovaries, and insulin resistance. This syndrome affects about 6–10% of women of childbearing age (Barbieri, 2000). There is a lack of consensus between endocrinologists and gynecologists in the definition, diagnosis, and treatment of PCOD (Cussons et al, 2005).

PCOD is currently considered as possibly the most frequent cause of female infertility; it is also closely associated with the MetS (Gleicher and Barad, 2006). Hyperandrogenism, insulin resistance, and acanthosis nigricans (HAIR-AN syndrome) cause presentation of the insulin-resistant syndrome PCOD (Barbieri, 2000). Insulin resistance and hyperandrogenism are caused by both genetic and environmental factors. Acanthosis nigricans is a dark, velvety patch of skin that indicates insulin resistance (Scalzo and McKittrick, 2000). Women of Caribbean-Hispanic or African American descent seem to be more prone to this condition.

Women with PCOD may have had a history of GDM. Many adolescents present with hirsutism and irregular menses. In PCOD, elevated LH to FSH ratio, hirsutism, acne, oily skin, male pattern baldness, menstrual irregularity, oligomenorrhea, and obesity can occur. Abnormally elevated levels of testosterone and LH disrupt the normal maturation process for ovulation. Immature cysts remain on the ovaries, giving the appearance of a "string of pearls."

Girls tested for anorexia nervosa (AN) may also have PCOD, with menstrual irregularities before weight loss and elevated LH and estrogen compared with individuals who have AN alone (Pinhas-Hamiel et al, 2006). In PCOD, biochemical abnormalities include hyperandrogenism, acyclic estrogen production, LH hypersecretion, decreased levels of steroid hormone–binding globulin (SHBG), and hyperinsulinemia (Mascitelli and Pezzetta, 2005). Infertility, hypertension, uterine cancer, diabetes, coronary heart disease, and endometrial carcinoma often follow (Legro, 2001).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Polymorphisms in the PPARGC1A, PPAR-delta and PPAR-gamma2 loci have been associated with PCOS.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Weight gain pattern
- History (Hx) of GDM
- Irregular menses
- Amenorrhea
- Hirsutism
- Infertility
- Acne
- Male pattern baldness
- Acanthosis nigricans (dark, velvety patches on skin)
- Vaginal ultrasound with enlarged ovaries
- Recurrent pregnancy loss?

Lab Work

- Glucose
- CRP
- Chol, Trig (elevated?)
- Homocysteine
- Fasting serum insulin (elevated)
- C-peptide levels for insulin secretion
- Serum estrogen
- Serum testosterone
- LH
- LH:FSH ratio (elevated)
- Plasminogen activator inhibitor-1 (PAI-1) (shows abnormal clotting)
- H & H
- Alb
- BUN, Creat
- ALT
- BP (elevated)

SAMPLE NUTRITION CARE PROCESS STEPS

Food-Drug Interaction

Assessment Data: Recent Dx of PCOD; wt at 110% of normal for height. Currently taking metformin but complaining of GI distress.

Nutrition Diagnosis (PES): Food-drug interaction related to metformin use as evidenced by GI distress and use of the medication on an empty stomach.

Intervention: Education about the need to eat before taking the prescription to evaliate if GI distress resolves.

Monitoring and Evaluation: Follow-up about GI distress or other side effects while taking metformin. Evaluation of timing for food and medication use.

INTERVENTION

OBJECTIVES

- Lose weight or maintain a normal weight for height; obesity occurs in 50% of this population.
- Prevent heart problems, stroke, and heart attack. Improve lipid profile.
- Reduce serum androgens and improve menstrual regularity.
- Decrease risk for endometrial cancer.
- Alleviate glucose intolerance and insulin resistance.
- Improve anxiety, moods, and quality of life.

FOOD AND NUTRITION

- Offer a weight-control and exercise plan to meet weight goals. Loss of 5–10 lb may reduce symptoms.
- Lower elevated blood glucose and lipids. Eat five to six small meals per day.
- The DASH diet may be helpful to lower BP. Include low-fat dairy products and more fruits and vegetables.
- Avoid low-fat, high-CHO diets, which promote extra insulin secretion (McKittrick, 2002). A diet of 30–40% fat, 45–50% complex CHOs, and 15–20% protein may be useful.
- Include sufficient fiber (20–35 g/d).
- Include sources of omega-3 fatty acids (fish, walnuts, and flaxseed).
- Dietary or supplemental chromium should be included.

Common Drugs Used and Potential Side Effects

- Symptoms may be managed by antiandrogen medication (e.g., birth control pills, spironolactone, flutamide, or finasteride).
- Insulin-sensitizing drugs improve ovulation and hirsutism in PCOD (Azziz et al, 2001). Metformin (glucophage) allows for improved insulin sensitivity and reduced LH and testosterone. Metformin induces ovulation, has some marginal benefit in improving aspects of the MetS, improves objective measures of hirsutism, and seems to be effective in both obese and lean individuals (Lord and Wilkin, 2004). Do not use with heart failure, chronic obstructive pulmonary disease, or chronic kidney disease. Take with food; monitor for minor gastrointestinal side effects such as nausea, diarrhea, and flatulence. Hypoglycemia does not usually occur.
- Rosiglitazone (Avandia) and pioglitazone (Actos) pose minimal risk of hepatotoxicity compared with older medications.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Chromium picolinate (1000 μg) may be useful as an insulin sensitizer in the treatment of PCOD (Lydic et al, 2006).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Counsel about weight loss and nutrition. Regular mealtimes and snacks may help control cravings and overeating.
- Encourage regular exercise and reduced sedentary lifestyle.
- Explain relationship of insulin resistance and increased risk for T2DM.
- Medical treatments may be needed to support reproduction. Some women will need in vitro fertilization (IVF) treatments.

Patient Education—Foodborne Illness

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

For More Information

- E-medicine: PCOS
  http://www.emedicine.com/med/topic2173.htm
- Polycystic Ovarian Syndrome Association, Inc.
  http://www.pcosupport.org

POLYCYSTIC OVARIAN DISEASE—CITED REFERENCES

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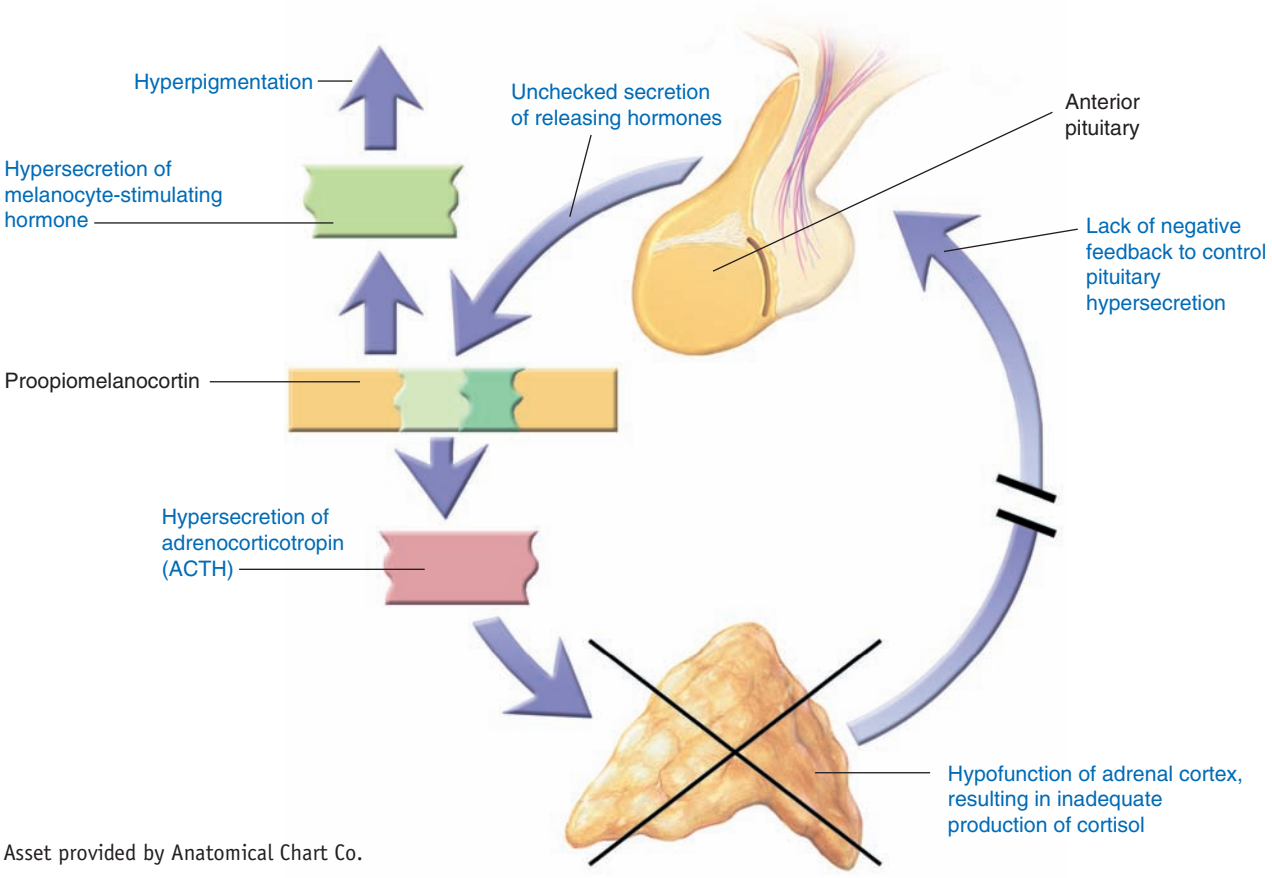
ADRENAL GLAND (CORTEX)

ADRENOCORTICAL INSUFFICIENCY AND ADDISON'S DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Asset provided by Anatomical Chart Co.

DEFINITIONS AND BACKGROUND

In adrenocortical insufficiency, the adrenal cortex atrophies with loss of hormones (aldosterone, cortisol, and androgens). Primary adrenal insufficiency in the pediatric population (0–18 years) is most commonly attributed to congenital adrenal hyperplasia, which occurs in about 1 in 15,000 births, followed by Addison's disease, with a likely autoimmune etiology (Perry et al, 2005). Secondary forms often result from tuberculosis, cancer, or surgery in which the adrenal glands are destroyed or damaged. Of patients with adrenocortical insufficiency, 33% also have diabetes. With T1DM, the expression of organ-specific autoantibodies is very high (Barker et al, 2005), and this suggests a need for careful screening in both of these conditions.

Cortisol, a glucocorticoid, affects almost every organ and tissue in the body. Cortisol helps the body respond to stress. Among other tasks, cortisol helps to maintain BP and cardiac functioning, the immune system's inflammatory response, the effects of insulin in breaking down sugar for energy through metabolism of macronutrients, and proper arousal and well-being.

Aldosterone functions to conserve sodium and excrete potassium. When aldosterone is no longer secreted, the following events occur: excretion of sodium takes place, and the body's store of water decreases, which leads to dehydration, hypotension, and decreased cardiac output. The heart becomes slower due to reduced workload. Increased serum potassium can lead to arrhythmias, arrest, and even death.

Primary adrenal insufficiency causes abdominal pain, vomiting, weakness, fatigue, weight loss, dehydration, nausea, diarrhea, hyperpigmented (tan or bronze) skin, hypotension, low serum sodium, high serum potassium, and low corticosteroid levels. It may be temporary or may become a chronic insufficiency. Salt cravings can occur.

Type I primary adrenal insufficiency (polyendocrine deficiency syndrome) occurs in children and exhibits underactive parathyroid glands, pernicious anemia, chronic *Candida* infections, chronic active hepatitis, and slow sexual development. Autoimmune thyroid diseases are often associated with T1DM and Addison's disease, characterizing the autoimmune polyendocrine syndrome. **Type II primary adrenal insufficiency (Schmidt's syndrome)** affects young adults and presents with underactive thyroid gland, slow sexual development, diabetes, vitiligo and changing skin pigmentation.

In **secondary adrenal insufficiency**, which is much more common than the primary form, there is a lack of ACTH. Production of cortisol drops but not production of aldosterone. A temporary form of secondary adrenal insufficiency may occur when a person who has been receiving a glucocorticoid hormone such as prednisone for a long time abruptly stops or interrupts taking the medication. Glucocorticoid hormones, which are often used to treat inflammatory illnesses such as rheumatoid arthritis, asthma, or ulcerative colitis, block the release of both CRH and ACTH. Normally, CRH instructs the pituitary gland to release ACTH. If CRH levels drop, the pituitary is not stimulated to release ACTH, and the adrenals then fail to secrete sufficient levels of cortisol. In secondary adrenal insufficiency, darkening of the skin does not occur, and GI symptoms are less common. Hypoglycemia, anxiety, nausea, and palpitations can occur.

Addison's disease is a strict insufficiency state of adrenal hormones, including cortisol and aldosterone. It affects about one in 100,000 people. Autoimmune Addison's disease is caused by autoreactivity toward the adrenal cortex; T cells seem to be involved. An Addisonian crisis can be precipitated by acute infection, trauma, surgery, or excessive body salt loss. Patients with adrenal insufficiency, although on treatment, have a poor quality of life and an increased mortality (Debono et al, 2009). A normal lifespan is possible if daily medications are taken as prescribed. Delayed and sustained release oral formulations of hydrocortisone will match the more natural circadian rhythms of the body. Adrenalectomy may require steroid replacement therapy, a 2-g sodium diet, and control of carbohydrate to prevent hyperglycemia. Possibly, the addition of dehydroepiandrosterone (DHEA) to the treatment plan may lead to improved well-being and sexual function (Hahner and Allolio, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Cytotoxic T lymphocyte antigen 4 (CTLA-4), protein tyrosine phosphatase nonreceptor type 22 (PTPN22), major histocompatibility complex class II transactivator (CIITA), and C-lectin type genes (CLEC16A) play a role in the primary form (Michels and Eisenbarth, 2009).

Clinical/History	Lab Work	
Height	Gluc	ACTH stimulation test
Weight	K^+ (increased)	BUN (increased)
BMI	N balance	Cortisol (decreased)
Diet history	Na^+ (decreased)	CRH stimulation test
BP (decreased)	Ca^{++} (increased)	Alb
I & O	Cl_2 (decreased)	Mg^{++} (increased)
Abdominal x-rays	WBC (decreased)	HbA1c
Skin changes	ACTH (increased)	

INTERVENTION

OBJECTIVES

- Relieve symptoms of hormone deficiency by taking synthetic hormones (but not to excess).
- Prevent hypoglycemia; avoid fasting.
- Prevent weight loss. Improve appetite and strength.
- Modify sodium according to drug therapy. Prevent hyponatremia, especially in warm weather when sodium losses from perspiration are higher than usual.
- Prevent dehydration and shock.
- Correct diarrhea, hyperkalemia, nausea, and improper medication administration.
- If hyperglycemia or diabetes occurs, insulin may be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Sodium Intake

Assessment Data: Adrenal insufficiency with inadequate intake of medication replacement; recent labs showing elevated potassium and low serum sodium levels.

Nutrition Diagnosis (PES): Inadequate sodium intake related to insufficient medication for Addison's disease as evidenced by high serum potassium and low serum sodium and diet history revealing low sodium intake for the past week.

Intervention: Educate about the importance of taking sufficient medication to maintain normal electrolyte levels and when to add salt to the diet.

Monitoring and Evaluation: Review of serum electrolytes, quality of life, fewer episodes of Addisonian crisis, diet hx revealing controlled sodium intake as per medical advice.

FOOD AND NUTRITION

- During an Addisonian crisis, low BP, low blood glucose, and high levels of potassium can be life threatening. Intravenous injections of hydrocortisone, saline, and dextrose are given by the medical team.
- Use a high-protein, moderate-carbohydrate diet. Snacks may be needed. Control carbohydrate amount and frequency according to blood glucose and HgbA1c levels.
- Ensure intake of sodium is adequate according to medications given. Monitor potassium levels and adjust diet if needed.
- Force fluids (2–3 L) when allowed.

Common Drugs Used and Potential Side Effects

- If aldosterone is missing, fludrocortisone (Florinef) is used as an oral, synthetic, sodium-retaining hormone. Be wary about overdosing, with potential side effects of hypertension and ankle edema. Postural hypotension can occur with a dose that is too low. Extra may be needed when exercising or in hot weather.
- Long-acting synthetic glucocorticoids, such as oral dexamethasone or prednisone, are given for replacement. Side effects can include loss of calcium, decreased bone density, or risk of osteoporosis. To decrease gastric irritation, take hormones with milk or an antacid.
- Signs of insufficient amounts of cortisol replacement include feeling weak and tired all the time, getting sick or vomiting, and anorexia. Weight loss can occur.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help patient individualize diet according to symptoms. During illness or injury, the body normally makes up to 10 times more cortisol than usual. Be prepared to avoid a crisis with a prompt treatment.
- Ensure patient does not skip meals. Instruct patient to carry cheese or cracker snack to prevent hypoglycemia.
- Discuss simple meal preparation to lessen fatigue.
- Discuss food sources of sodium and potassium according to the medical plan.
- Use of Medic-Alert identification is recommended. Patients with this condition may need to carry a syringe prefilled with dexamethasone.
- Pregnancy is possible, with carefully managed replacement medication. Evidence has accrued that fracture risk might be programmed during intrauterine life, mediated through pituitary-dependent endocrine systems such as insulin, growth hormone, and the HPA (hypothalamic-pituitary-adrenal) system (Cooper et al, 2009).

Patient Education—Foodborne Illness

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

For More Information

- Adrenal Gland Disorders
  http://www.nlm.nih.gov/medlineplus/adrenalglanddisorders.html
- JAMA – Patient Page for Adrenal Insufficiency
  http://jama.ama-assn.org/cgi/reprint/294/19/2528.pdf
- National Adrenal Diseases Foundation
  http://www.medhelp.org/nadf/
- National Institutes of Health
  http://www.cc.nih.gov/ccc/patient_education/pepubs/mngadrins.pdf

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Adrenal Gland (Cortex)

HYPERALDOSTERONISM

NUTRITIONAL ACUITY RANKING: LEVEL 1–2

DEFINITIONS AND BACKGROUND

Hyperaldosteronism is an increased production of aldosterone by the adrenal cortex. Aldosterone controls water and electrolyte balance by acting on mineralocorticoid receptors in the kidney and in the vascular system (Oberliethner, 2005). Hyperaldosteronism causes endothelial dysfunction regardless of high BP. Patients may be quite vulnerable to cardiac events, including stroke (Milliez et al, 2005).

Primary aldosteronism (PA) is usually from an adenoma and involves hypertension, hypokalemia, and low plasma renin. Familial hyperaldosteronism type I (FH-I) represents about 1% of cases of primary hyperaldosteronism. It may be detected in asymptomatic individuals when screening the offspring of affected individuals, or patients may present in infancy with hypertension, weakness, and failure to thrive due to hypokalemia. It is inherited in an autosomal dominant manner.

Conn's syndrome is a benign tumor in one adrenal gland that can cause this condition. Urine 18-hydroxycortisol (18-OHF) measurements are used to detect Conn's syndrome with adrenal adenoma or glucocorticoid-suppressible hyperaldosteronism (Reynolds et al, 2005).

Secondary forms of aldosteronism may occur after cancer, heart failure, hyperplasia, malignant hypertension, pregnancy, estrogen use, or cirrhosis. Aldosteronism is a diagnosis that should be considered in refractory hypertension.

CT, MRI, and adrenal vein sampling (AVS) distinguish unilateral PA from the bilateral form; the unilateral form can be treated surgically, whereas bilateral PA is treated medically (Kempers et al, 2009).

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Sodium Intake

Assessment Data: Hypertension (190/95); diet hx indicating daily use of high sodium foods and salty snacks.

Nutrition Diagnosis (PES): Excessive sodium intake related to intake of high sodium foods and frequent salty snacks throughout the day as evidenced by dietary recall and food records estimated at about 8–9 g Na^+ daily.

Intervention: Food-nutrient delivery—limit dietary sources of sodium to 2–4 g sodium; monitor effects of medication. Educate about sources of sodium, potassium, and use of the DASH diet plan as appropriate. Counsel about ways to reduce sodium from foods and snacks; how to dine away from home; how to monitor for signs and symptoms requiring medical attention.

Monitoring and Evaluation: Lower BP; normal serum labs, especially potassium. Diet history indicating great improvement in sodium regulation. Able to verbalize use of the DASH diet principles.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Primary hyperaldosteronism may be the cause of essential hypertension in some people. Specific DNA mutations have emphasized the role of molecular genetics in this disorder (New et al, 2005).

Clinical/History

- Height
- Weight
- BMI
- Diet history
- I & O
- BP (high)
- Fatigue
- Headache
- Weakness, intermittent paralysis
- Numbness
- Abdominal CT scan for adrenal mass
- ECG with abnormalities from low K^+ levels

Lab Work

- Sodium load test (6 g common)
- Plasma renin (low)
- Plasma aldosterone (elevated)
- Urinary aldosterone (elevated)
- Aldosterone: renin ratio
- Urine sodium (altered)
- Urine 18-hydroxycortisol (18-OHF)
- Na^+ (altered)
- K^+ (low)
- Urine potassium (altered)
- Ca^{++}
- Mg^{++} (altered)
- pCO_2 (altered)
- H & H
- Serum Fe

INTERVENTION

OBJECTIVES

- Hydrate adequately.
- Alter diet as needed (sodium, potassium).
- Correct hypokalemia and hypertension.
- Prepare for surgery if a tumor is involved. Laparoscopic adrenalectomy (LA) is quite successful, after which BP returns to normal. If a tumor is not involved, medical treatment will be for life.

FOOD AND NUTRITION

- Provide adequate fluid intake (unless contraindicated for other reasons).
- A sodium-restricted diet may be needed. A high potassium intake and the DASH diet may be required, depending on the medical or surgical treatment used.
- Small, frequent feedings may be needed.

Common Drugs Used and Potential Side Effects

- Antihypertensives may be used; monitor side effects specifically for the medications prescribed. Aldosterone antagonists have been available for many decades; spironolactone may be used alone or with ACE inhibitors or ARBs.
- Eplerenone, a selective aldosterone antagonist, avoids the androgen and progesterone receptor–related adverse events that sometimes occur with spironolactone, such as breast tenderness, gynecomastia, sexual dysfunction, and menstrual irregularities (Pratt-Ubunama et al, 2005).
- Digitalis may be used. Avoid herbal teas, high-fiber intakes, and excessive amounts of vitamin D. Include adequate amounts of potassium. Take the drug 30 minutes before meals.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Products containing natural licorice should be avoided in this condition.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain altered sodium and potassium requirements. Teach principles of the DASH diet.
- Have patient avoid fasting, skipping meals, and fad dieting.
- Provide recipe suggestions.
- Avoid consuming large amounts of real licorice, which can aggravate the condition.

Patient Education—Foodborne Illness

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

For More Information

- Medline
  http://www.nlm.nih.gov/medlineplus/ency/article/000330.htm
- Merck manual – Hyperaldosteronism
  http://www.merck.com/mmhe/sec13/ch164/ch164e.html
- National Adrenal Foundation – Hyperaldosteronism
  http://www.nadf.us/diseases/hyperaldosteronism.htm

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Adrenal Gland (Medulla)

PHEOCHROMOCYTOMA

NUTRITIONAL ACUITY RANKING: LEVEL 1

DEFINITIONS AND BACKGROUND

Pheochromocytoma (PHEO) is a rare tumor of the chromaffin cells most commonly arising from the adrenal medulla, resulting in increased secretion of epinephrine and norepinephrine. It is slightly more common in males. Hereditary PHEOs are typically intra-adrenal and bilateral, and patients typically present at a young age.

PHEO may occur as a single tumor or as multiple growths. Symptoms and signs include very high BP, headache, excessive diaphoresis, and palpitations. Less-common symptoms are anxiety, chest pain, fatigue, weight loss, abdominal pain, and nervousness. About 10% of these tumors are malignant and can spread. Persons who have difficult-to-treat hypertension, have onset before age 35 or after age 60, or are taking four or more BP medicines may need to be tested for PHEO.

BP often fluctuates up and down, daily testing is recommended. Diagnosis includes measurement of urinary catecholamines or their metabolites, vanillylmandelic acid and total metanephrines. The urinary metanephrines provide a highly sensitive clue to the presence of PHEO; see Table 9-22.

Treatment usually involves surgical removal of the tumor. Before surgery, alpha-adrenergic blockers may be used. After surgery, about one quarter of patients will still suffer from hypertension and require lifelong management.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: PHEO may be transmitted as an autosomal dominant trait. At least five different gene mutations can cause PHEO. One type causes multiple endocrine neoplasia. Type IIB (MEN IIB) also leads to thyroid cancer and tumors of nerves in the lips, mouth, eyes and digestive tract. The NF1 neurofibromas may present with café-au-lait spots, an optic glioma, and PHEO.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Increased appetite
- BP (very elevated)
- Orthostatic hypotension
- Headache
- Excessive diaphoresis, flushing
- Intolerance of heat
- Heart palpitations
- Sleep disturbances
- CT scan, MRI
- Adrenal biopsy
- Metaiodobenzylguanidine (MIBG) scanning

Lab Work (see Table 9-22)

- Urinary epinephrine and norepinephrine (increased)
- Urinary metabolites (vanillylmandelic acid and metanephrines)*
- Gluc
- Na^+, K^+
- Alb
- CRP
- H & H
- Glucagon test (positive)
- T3, T4

*Testing may require dietary changes up to 3 days in advance.

INTERVENTION

OBJECTIVES

- Prepare for surgery to remove tumor. Patients with preoperative endocrinopathies present a challenge because the "endocrine axis" is complex (Kohl and Schwartz, 2009). If the tumor cannot be removed, lifelong medication will be needed.

TABLE 9-22 Catecholamines

Catecholamine	Comments	Normal Urinary Value/24 h
Dopamine	A neurotransmitter (a chemical used to transmit impulses between nerve cells) found mainly in the brain. Metabolized by target tissues or by the liver to become inactive substances that appear in the urine—dopamine becomes homovanillic acid.	Dopamine 65–400 µg
Epinephrine	A brain neurotransmitter, but is also a major hormone secreted from the adrenal medulla in response to low blood glucose, exercise, and various forms of stress where the brain stimulates release of the hormone. Epinephrine causes a breakdown of glycogen to glucose in liver and muscle, the release of fatty acids from adipose tissue, vasodilation of small arteries within muscle tissue, and increases in the rate and strength of the heartbeat. In the urine: epinephrine becomes metanephrine and VMA.	Epinephrine 0.5–20 µg Metanephrine: 24–96 µg VMA: 2–7 mg*
Norepinephrine	The primary neurotransmitter in the sympathetic nervous system (controls the "fight or flight" reaction) and is also found in the brain. In the urine: norepinephrine becomes normetanephrine and vanillylmandelic acid (VMA).	Norepinephrine 15–80 µg Normetanephrine: 75–375 µg
Total		Total urine catecholamines: 14–110 µg

*For testing, a VMA-restricted diet may be required for lab results; omit chocolate, vanilla extract, and citrus.
Source: Medline Plus. Catecholamines–urine. Accessed September 22, 2009, at http://www.nlm.nih.gov/medlineplus/ency/article/003613.htm

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Intake of Bioactive Substances

Assessment Data: Hypertensive crisis following intake of caffeine and Chianti wine. Dx of PHEO; high levels of urinary epinephrine and norepinephrine, as well as vanillylmandelic acid and metanephrines. Diet history reveals recent intake of coffee, chocolate and Chianti wine at dinner meal, causing extreme heart palpitations and soaring BP.

Nutrition Diagnosis (PES): Excessive intake of bioactive substances (caffeine, tyramine) related to coffee, chocolate and Chianti wine as evidenced by very high BP and diet history.

Intervention: Education about foods to avoid, such as caffeine from food and beverages, tyramine-rich foods including beer, Chianti wine, processed meats, soy sauce, bananas, avocado.

Monitoring and Evaluation: No further hypertensive crises related to food and beverage intake.

- Stabilize BP before surgery. Avoid overstimulation from even slight exercise, cold stress, or emotional upsets.
- Correct nausea, vomiting, and anorexia.
- Manage long-term hypertension. A severe BP level is any reading above 180/110 mm Hg. Prevent hypertensive crises, which could cause sudden blindness, kidney failure, seizures, acute respiratory distress, arrthymias or stroke.
- Manage any complications such as heart attack or cardiomyopathy (Kassim et al, 2008).

FOOD AND NUTRITION

- For urine testing, some doctors order a VMA diet where patient avoids bananas, caffeine from coffee or Pepsi/Coke, chocolate, tea, vanilla in foods, pineapple, alcoholic beverages, eggplant, plums, walnuts. While this was a common practice in the past, there is no strong evidence to support its use.
- Increase fluids but avoid caffeinated beverages. Avoid tyramine-rich foods, especially if taking MAO inhibitors.
- Six small feedings may be better tolerated than large meals.
- Increase protein and calories if patient is having surgery. Postoperatively, provide adequate vitamins and minerals for wound healing.
- Re-expansion of plasma volume may be accomplished by liberal salt or fluid intake with use of alpha-1 adrenergic receptor antagonists.
- In recurrent cases, long-term drug therapy will be needed. Monitor for specific dietary changes and side effects.

Common Drugs Used and Potential Side Effects

- Pharmacological treatment of catecholamine excess is mandatory.
- Phenoxybenzamine (or an alpha-1 adrenergic receptor antagonist such as prazosin) is needed to block alpha-adrenergic activity. Diuretics should not be used.
- Low doses of a beta-blocker such as propranolol are used to control BP and cardiac tachyarrhythmias but only after alpha blockade. Labetalol, an alpha- and beta-adrenergic blocker, has also been shown to be effective in the control of BP and symptoms of PHEO.
- Avoid decongestants, amphetamines, MAO inhibitors (Nardil, Parnate).
- High-dose MIBG may be used in the treatment of malignant PHEO but can have toxic side effects (Gonias et al, 2009).
- If diabetes occurs, insulin may be needed.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss avoidance of caffeinated foods and beverages (e.g., coffee, tea, and chocolate).
- Maintain a calm atmosphere for patient; prevent undue stress.
- Exercise should be limited until condition is under control.

Patient Education—Foodborne Illness

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

For More Information

- Endocrine Web
  http://www.endocrineweb.com/pheo.html
- Mayo Clinic
  http://www.mayoclinic.com/health/pheochromocytoma/DS00569
- Medline
  http://www.nlm.nih.gov/medlineplus/ency/article/000340.htm
- Merck Manual
  http://www.merck.com/mrkshared/mmanual/section2/chapter9/9d.jsp
- National Cancer Institute
  http://www.cancer.gov/cancerinfo/pdq/treatment/pheochromocytoma/patient
- National Library of Medicine
  http://www.nlm.nih.gov/medlineplus/pheochromocytoma.html
- Urology Health
  http://www.urologyhealth.org/adult/index.cfm?cat=04&topic=114

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Kassim TA, et al. Catecholamine-induced cardiomyopathy. *Endocr Pract.* 14:1137, 2008.

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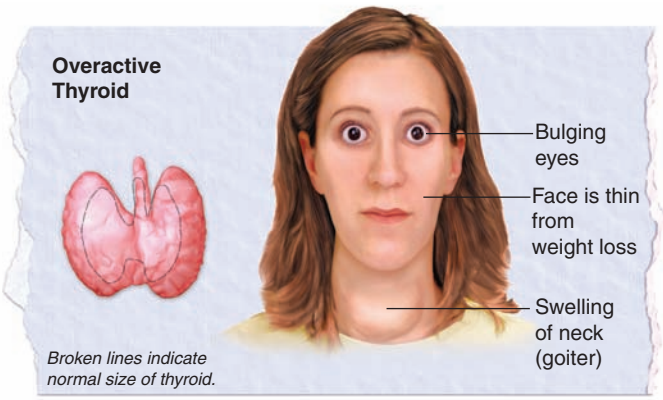
THYROID GLAND

HYPERTHYROIDISM

NUTRITIONAL ACUITY RANKING: LEVEL 1



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DEFINITIONS AND BACKGROUND

Hyperthyroidism results from oversecretion of the thyroid hormones, triiodothyronine (T3) or thyroxone (T4). These hormones affect every cell through controlling body temperature, heart rate, metabolism, and production of calcitonin. Thyroid hormone affects glucose homeostasis through increased hepatic glucose output, increased futile cycling of glucose degradation products between the skeletal muscle and the liver, decreased glycogen stores in the liver and skeletal muscle, altered oxidative and nonoxidative glucose metabolism, decreased active insulin output from the pancreas, and increased renal insulin clearance (Potenza et al, 2009). When the hypothalmus signals the pituitary gland to produce TSH, normally this is regulated to be just enough. See Table 9-23 for interpretation of lab results for thyroid disorders.

The autoimmune thyroid diseases (AITD), Graves' disease, and chronic lymphocytic thyroiditis (CLT) are among the most common endocrine diseases in childhood and adolescence (Brown, 2009). Usually in hyperthyroidism, the entire gland is overproducing thyroid hormone. Rarely, a single nodule is responsible for the excess hormone secretion. An elevated metabolic rate, tissue wasting, diaphoresis, tremor, tachycardia, goiter, heat intolerance, cold insensitivity, nervousness, increased appetite, exophthalmos, and loss of glycogen stores can occur. If left untreated, atrial fibrillation and osteoporosis can result.

Autoimmune thyroiditis (AITD) may start out temporary, then lead to hyperthyroidism. The most common causes include Hashimoto's thyroiditis, subacute granulomatous thyroiditis, or silent lymphocytic thyroiditis. The major environmental triggers of AITD include iodine, medications, infection, smoking, and possibly stress (Tomer and Huber, 2009). Specific digestive diseases (celiac disease or primary biliary cirrhosis) may be associated with autoimmune thyroid processes (Daher et al, 2009). Persons who have T1DM, mood disorders, psychosis, or Addison's disease also seem to be at higher risk.

Graves' disease (diffuse toxic goiter) is the most common form; **thyrotoxicosis** is more severe. Clinical thyrotoxicosis in Graves' disease patients is caused by thyrotropin receptor (TSHR)-stimulating autoantibodies (Schott et al, 2005). Most people who develop Graves' ophthalmopathy have one or more of the following symptoms: dry and itchy eyes, a staring or bug-eyed look (exophthalmos), sensitivity to light, excessive tearing, a feeling of pressure around the eyes, difficulty closing the eyes completely, and peripheral double vision. Thyrotoxicosis can alter carbohydrate metabolism in a type 2 diabetic patient to such an extent that DKA develops if untreated (Potenza et al, 2009).

Thyroid storm (thyroid crisis) is a potentially life-threatening condition that develops in a person with hyperthyroidism. The gland suddenly releases large amounts of thyroid hormone in a short period of time. Signs of thyroid storm include extreme irritability, high systolic BP, low diastolic BP, tachycardia, nausea, vomiting, diarrhea, high fever, confusion, and sleepiness. Shock, delirium, shortness of breath, fatigue, coma, heart failure, and death can result if not treated immediately. Emergency medical treatment is always needed.

If thyroidectomy is needed, antithyroid agents and iodine are often given 4–6 weeks before surgery to minimize the risk of thyroid crisis. With thyroidectomy, the patient may require a high-calorie, high-protein diet preoperatively. Evaluate needs postoperatively with the doctor's care plan.

TABLE 9-23 Thyroid Test Results

TSH	T4	T3	Interpretation
High	Normal	Normal	Mild (subclinical) hypothyroidism
High	Low	Low or normal	Hypothyroidism
Low	Normal	Normal	Mild (subclinical) hyperthyroidism
Low	High or normal	High or normal	Hyperthyroidism
Low	Low or normal	Low or normal	Nonthyroidal illness; rare pituitary (secondary) hypothyroidism

Source: Lab Tests On Line, web site accessed October 25, 2009, at http://www.labtestsonline.org/understanding/analytes/t3/test.html

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: In Graves' disease a specific combination of polymorphisms for thyroglobulin and HLA-DR markedly increases the odds ratio for developing disease

(Brown, 2009). Among the major AITD susceptibility genes that have been identified is the HLA-DR gene locus, as well as CTLA-4, CD40, PTPN22, thyroglobulin, and TSH receptor genes (Tomer and Huber, 2009).

Clinical/History

Height
Weight
BMI
Rapid weight loss?
Diet history
Temperature
BP
I & O
Nervousness, anxiety
Tachycardia or palpitations
Increased sensitivity to heat
Difficulty sleeping
Fine tremor of hands
More frequent bowel movements
Fatigue, muscle weakness
Changes in menstrual habits
Protruding eyeballs?
Excessive tearing from one or both eyes?
Thyroid scan
Goiter (enlarged thyroid gland)

Lab Work

T3 (increased)
T4 (increased)
TSH (normal or low)
Protein-bound iodine (PBI)
Gluc (increased)
Alb, transthyretin
Ca^{++}
Mg^{++} (decreased)
Na^{+}, K^{+}
H & H
Serum ferritin
Chol, Trig (decreased)
BUN, Creat
N balance
Alk phos (increased)

SAMPLE NUTRITION CARE PROCESS STEPS

Altered Nutrient Utilization

Assessment Data: Elevated T3 and T4, low TSH and serum calcium; BMI 18 with weight loss of 15 lb in 6 months. Dx hyperthyroidism.

Nutrition Diagnosis (PES): Altered nutrient utilization (NC-2.1) related to hyperthyroidism as evidenced by elevated T4 of 112 μg/100 mL; T3 of μg/100 mL; low TSH level at 0.10 μU/L; low serum calcium levels; weight loss of 15 lb in less than 6 months.

Interventions: Food and Nutrition Delivery: ND-1.3 provide information on high calorie diet to promote weight gain, ensure high carbohydrate intake, importance of 1 quart of milk daily or equivalent, caffeine to be avoided, be cautious of raw goitrogens ND-3.2.1 continue use of daily multivitamin. ND3.2.4 continue use of daily calcium supplement.

Nutrition Education: E-1.1 Discuss importance of high calorie diet (40 kcal/kg/d) to promote weight gain consisting of following healthy eating habits and to discuss interaction of raw natural goitrogens with antithyroid drugs.

Counseling: C-2.2 RD to work with patient to set goals of weight gain and taking antithyroid drug to be prescribed by MD.

Coordination of Care: RC-1.3 Collaboration with MD and referral to endocrinologist, to speak with MD about weight gain of 1–2 lb per week and drug–food interactions.

Monitoring and Evaluation: Prevent the thyroid from producing excess hormones with use of antithyroid drug, Tapazole. Follow a weight gain of 1–2 lb/wk till patient reaches normal body weight range of 125–130 lb. Daily use of multivitamin and calcium supplementation. Successful food recording in food diary. Patient can recite raw natural goitrogens to be avoid them in her diet. Patient eating frequent snacks. Patient successfully gained 1 lb in 2 weeks bringing her weigh to 116 lb, BMI now 19.3. Patient to visit endocrinologist in next week.

INTERVENTION

OBJECTIVES

- Achieve a euthyroid state to avoid the effects of triiodothyronine (T3) on the heart and cardiovascular system: decreased systemic vascular resistance and increased resting heart rate, left ventricular contractility, blood volume, and cardiac output (Dahl et al, 2008).
- Prevent or treat complications accompanying high metabolic rate, including bone demineralization. This seems to be a greater problem in older women than in adolescents (Poomthavorn et al, 2005).
- Replenish glycogen stores. Replace lost weight (usually 10–20 lb).
- Correct negative nitrogen balance.
- Replace fluid losses from diarrhea, diaphoresis, and increased respirations. However, exophthalmos, caused by increased accumulation of extracellular fluid in the eyes, may require fluid and salt restriction.
- Monitor or treat fat intolerance and steatorrhea.

FOOD AND NUTRITION

- Use a high-calorie diet (start with 40 kcal/kg). The patient's caloric needs may be increased by 50–60% in this condition (or 10–30% in mild cases). Ensure adequate intake of carbohydrates.
- Provide protein in the range of 1–1.75 g/kg body weight.
- Fluid intake should be 3–4 L/d, unless contraindicated by renal or cardiac problems.
- Include 1 quart of milk or equivalent daily to supply adequate calcium, phosphorus, and vitamin D.
- Exclude caffeine and stimulants from diet because they aggravate excitability and nervousness.
- Supplement diet with vitamins A, C, and B-complex vitamins, especially thiamin, riboflavin, B_6, and B_{12}. A general multivitamin–mineral supplement may be beneficial; monitor for iodine content.
- Be aware of iodine in any supplements used. Chronic iodine intakes greater than 500 μg/L are associated with adverse effects (Zimmermann et al, 2005).
- Raw natural goitrogens (cabbage, Brussels sprouts, kale, cauliflower, soybeans, peanuts) should not be used with antithyroid medications because these substances increase side effects of the drugs. Cooking reduces this effect.

Common Drugs Used and Potential Side Effects

- The goal of drug therapy is to prevent the thyroid from producing excess hormones. Antithyroid drugs, also called thionamides, such as methimazole (Tapazole) and propylthiouracil (PTU), can cause nausea, vomiting,

altered taste sensation, and gastrointestinal distress. They should be taken with food. Avoid use of natural goitrogens in raw form; cooked forms are not a problem.
- Thionamides represent the treatment of choice in pregnant women, during lactation, in children and adolescents, and in preparation for radioiodine therapy or thyroidectomy (Bartelena et al, 2005).
- Radioactive iodine may be used to damage or destroy some of the thyroid cells. It can cause a temporary burning sensation in the throat. Hydrochlorothiazide (HCTZ) may be used to improve radioiodine uptake in hyperthyroid patients; replace potassium.
- Some drugs can affect the thyroid glands. Amiodarone is a potent antiarrhythmic drug that also possesses beta-blocking properties; a 100-mg tablet contains an amount of iodine that is 250 times the recommended daily iodine requirement. Amiodarone-induced thyrotoxicosis is a difficult condition to diagnose and treat; monitor carefully (Basaria and Cooper, 2005).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Bugleweed, verbena, lemon balm and kelp have been recommended; no clinical trials prove efficacy.
- Limit use of large quantities of iodine-rich seaweed such as kombu (*Laminaria japonica*).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Thyroid hormone affects adipokines and adipose tissue, predisposing the patient to ketosis (Potenza et al, 2009). Beware of hyperglycemia after carbohydrate-rich meals.
- Encourage quiet, pleasant mealtimes.
- Exclude use of alcohol, which may cause a hypoglycemic state.
- Frequent snacks may be needed. To avoid obesity, adjust patient's diet as condition corrects itself.

Patient Education—Foodborne Illness

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

For More Information

- American Thyroid Association
  http://www.thyroid.org/
- Endocrine Web–Hyperthyroidism
  http://www.endocrineweb.com/hyper1.html
- European Thyroid Society
  http://www.eurothyroid.com/
- Latin American Thyroid Society
  http://www.lats.org/
- National Graves Disease Foundation
  http://www.ngdf.org/
- University of Maryland Medical Center
  http://www.umm.edu/endocrin/hypert.htm

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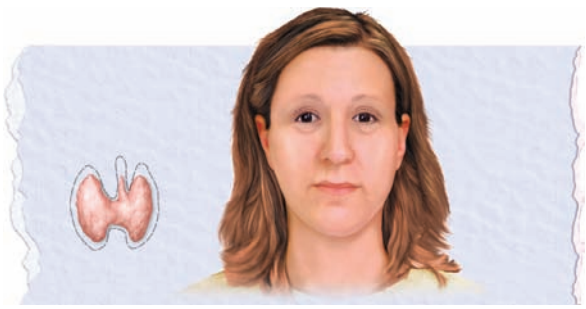
Tomer Y, Huber A. The etiology of autoimmune thyroid disease: a story of genes and environment. *J Autoimmun.* 32:231, 2009.

Zimmermann MB, et al. High thyroid volume in children with excess dietary iodine intakes. *Am J Clin Nutr.* 81:840, 2005.

THYROID GLAND

HYPOTHYROIDISM

NUTRITIONAL ACUITY RANKING: LEVEL 1



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DEFINITIONS AND BACKGROUND

Hypothyroidism is caused by the underfunctioning of the thyroid gland. It can be classified as primary (thyroid failure), secondary (from pituitary TSH deficit), or tertiary (from hypothalamic deficiency of thyrotropin-releasing hormone) or result from peripheral resistance to the action of thyroid hormones. Primary hypothyroidism causes about 95% of all cases.

The most common cause of thyroid gland failure is **Hashimoto's thyroiditis**, which is inflammation caused by the patient's own antibodies (as in pernicious anemia, lupus, rheumatoid arthritis, diabetes, or chronic hepatitis). The

second major cause of thyroid gland failure is from various medical treatments that affect the thyroid gland (e.g., surgery, chronic medication use).

Hypothyroidism affects up to 10% of adult women, usually middle-aged and older women. Women may experience menstrual irregularities or difficulty conceiving. Triiodothyronine (T3) and thyroxine (T4) are elevated during pregnancy and with oral contraceptive and estrogen use. Physicians must treat hypothyroidism to avoid long-term complications, such as depression, infertility, and CVD. Risk decreases if thyroid hormones are taken as directed.

Congenital hypothyroidism (CH) or cretinism is rare; incidence is one in 4000 births. Screening for this type of hypothyroidism is now common (Lanting et al, 2005). Treatment involves replacement of thyroid hormones and iodine. Cretinism can occur in areas where soil content of iodine is low.

Myxedema is a nonpitting edema that can occur in adults with hypothyroidism; hydrophilic mucopolysaccharide accumulates in the skin and muscles. In end-stage myxedema coma, untreated hypothyroidism leads to progressive weakness, stupor, hypothermia, hypoventilation, hypoglycemia, hyponatremia, water intoxication, shock, and even death. It occurs most often in older patients with underlying pulmonary and vascular disease. Signs and symptoms of hypothyroidism are listed in Table 9-24.

Thyroid autoimmunity is common and may contribute to miscarriages as well as to hypothyroidism. Infants are totally dependent on T4 from the mother during the first trimester for normal neurological development (Smallridge et al, 2005). Hypothyroidism from autoimmune disease or suboptimal iodine intake occurs in 2.5% of pregnant women; postpartum thyroid dysfunction (PPTD) occurs in 5–9% of women (Lazarus and Premawardhana, 2005). Because of the potential problems for mother and baby, screening, diagnosis, and treatment of thyroid problems among pregnant women is important.

Endemic goiter is an enlargement of the thyroid gland with swelling in front of the neck, resulting from iodine deficiency due to inadequate dietary intake or drug effects. Over 3 billion people in the world are on iodine supplementation programs. Optimal level of iodine intake to prevent thyroid disease is in a relatively narrow range around the recommended daily iodine intake of 150 μg.

Subclinical hypothyroidism (SCH) is mild thyroid failure and is diagnosed when peripheral thyroid hormone levels are within normal reference laboratory range but serum TSH levels are mildly elevated (Fatourechi, 2009). Iodine, iron, selenium, and zinc deficiencies can impair thyroid function. Iron deficiency impairs thyroid hormone synthesis by reducing activity of heme-dependent thyroid Peroxidase; iron supplementation may improve the efficacy of iodine supplementation. Anemia is a major manifestation in hypothyroidism not only because of impaired hemoglobin synthesis, but also because of iron deficiency from increased iron loss with menorrhagia, impaired intestinal absorption of iron, folate deficiency due to impaired intestinal absorption of folic acid, and pernicious anemia with vitamin B_{12} deficiency. Selenium deficiency and disturbed thyroid hormone status may develop in phenylketonuria, cystic fibrosis, as well as from poor nutrition in children, elderly people, or sick patients. In parts of the world where iodine and selenium are both deficient, dual supplementation may be advisable.

TABLE 9-24 Symptoms of Hypothyroidism By Life Stage

Infants
Constipation
Cyanosis
Developmental delay
Hoarse cry
Marked retardation of bone maturation
Persistent jaundice
Poor feeding
Respiratory difficulty
Somnolence
Umbilical hernia
Children
Evidence of mental retardation
Poor performance at school
Retarded growth
Stunting, short stature
Adults
Abnormal menstrual cycles
Brittle nails
Broad flat nose
Coarse features with protruding tongue
Coarse, dry hair
Cold intolerance
Dry, rough pale skin
Decreased libido
Delayed dentition
Difficulty losing weight
Easy fatigue
Hair loss
Hoarse, husky voice
Impaired mental development
Muscle cramps, frequent muscle aches
Puffy hands and face
Reduced conversion of carotene to vitamin A
Short stature
Weakness
Weight gain
Widely set eyes

Vitamin A deficiency (VAD) and the iodine deficiency disorders (IDD) affect >30% of the global population; these deficiencies often coexist in vulnerable groups (Zimmerman, 2007). Given alone, without iodine repletion, high-dose vitamin A supplementation in combined VAD and ID may reduce thyroid hyperstimulation and reduce risk for goiter (Zimmerman, 2007). More research on vitamin and mineral therapies will be beneficial.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Mutations in the DUOX2, PAX8, SLC5A5, TG, TPO, TSHB, and TSHR genes cause congenital hypothyroidism.

Clinical/History (see Table 9-24)

Height
Weight
BMI
Diet history
Temperature
BP
Goiter?
Thyroid scan

Lab Work

T4 (decreased)
T3 (decreased)
TSH (elevated)
Urinary iodine concentration (UIC)
Ca^{++}
Mg^{++} (increased)
H & H (decreased)
Serum ferritin
Chol, Trig (increased)
Gluc
Alk phos (decreased)
Serum copper (decreased)
Na^{+} and K^{+} (decreased)
Serum folic acid and vitamin B_{12} (low)
Creatine phosphokinase (CPK)
GFR (decreased?)
Thyrotropin
Somatomedin C (increased)
Uric acid (increased)
Carotenoids (increased)
Thyroglobulin (useful measure after introducing iodized salt)
Serum vitamin D_3

SAMPLE NUTRITION CARE PROCESS STEPS

Overweight (NC-3.3)

Assessment Data: Low serum levels of T4 and T3 with increased TSH. Unplanned weight gain of 30 lb in past 2 years. Recent Dx of hypothyroidism and initiation of synthroid. BMI 30.

Nutrition Diagnosis (PES): Overweight (NC-3.3) as related to excess food and nutrition related knowledge deficit and hypothyroidism as evidenced by reports of overconsumption of high fat/calorie dense food and beverages, BMI 30, waist circumference >40 inches, and a fat mass of 51.7%.

Interventions: Nutrition prescription (ND-1) of a weight control/exercise plan with promotion of a diet including 30–40% fat, 45–50% complex carbohydrates, and 15–20% protein with a goal of cutting out 250–500 calories a day resulting in a 0.5–1 lb weight loss each week. Recommend adequate intake of fluid and foods high in fiber. Support use of Synthroid 25 μg once daily. Initial/Brief nutrition education (E-1) to begin instruction on following a weight control/exercise plan with a diet including 30–40% fat, 45–50% complex carbohydrates, and 15–20% protein with a goal of cutting out 250–500 calories a day and provide basic nutrition related educational information on natural goitrogens, fiber, fluids, use of iodized salt, and physical activity.

Monitoring and Evaluation: Follow up in 1 month to repeat a TSH and free T4 to reassess thyroid function status; repeat TSH and free T4 and adjust dose of Synthroid accordingly. Evaluate progress in weight loss and healthy eating habits. Schedule a counseling session for in-depth nutrition related knowledge regarding weight loss as well as hypothyroidism.

INTERVENTION

OBJECTIVES

- Control weight gain that results from a 15–40% slower metabolic rate, especially in the untreated patient. Measure weight frequently to detect fluid losses or retention.
- Correct underlying causes, such as inadequate intake of iodine or congenital deficiency. Hormone replacement will be given.
- Correct vitamin B_{12}, folic acid, or iron deficiency anemias when present.
- Because vitamin D_3 has implications in autoimmune disorders, assure an adequate diet and supplementary source as needed.
- Improve energy levels; reduce fatigue.
- Improve cardiac, neurological, and renal functioning.
- Screen for thyroid problems in pregnant or postpartum women; assure hormone replacement as needed.

FOOD AND NUTRITION

- Use an energy-controlled diet adjusted for age, sex, and height.
- A multivitamin–mineral supplement formula may be beneficial, especially to replace nutrients that have been poorly absorbed. Since T3 is an important hormone needed for vitamin A metabolism, serum levels may be low in this population. Include good sources of carotenoids such as lycopene.
- Ensure an adequate supply of antioxidant and fiber-rich foods as well as fluids.
- For pregnant women or children, make sure that adequate amounts of iodine are consumed.
- Natural goitrogens in cabbage, turnips, rapeseeds, peanuts, cassava, cauliflower, broccoli, and soybeans may block uptake of iodine by body cells; they are inactivated by heating and cooking.
- Achieve optimal iodine intakes from iodized salt (in the range of 150–250 μg/d for adults) to minimize thyroid dysfunction (Zimmerman, 2009). Zinc, copper, and tyrosine are needed.
- Ensure adequate iodine status during parenteral nutrition, particularly in preterm infants (Zimmerman, 2009).

Common Drugs Used and Potential Side Effects

- Thyroid hormones (liotrix, sodium levothyroxine, or Synthroid) are used. Use caution with use of soy protein products since they can decrease effectiveness of the hormones. Thyroid hormones elevate glucose and decrease cholesterol; monitor persons who have diabetes carefully. Monitor

for weight changes and fluid shifts. If rapid weight loss, sweating, or other symptoms of hyperthyroidism occur, the doctor should be contacted for immediate follow-up.
- Lithium treatment for bipolar disorder has been associated with the development of goiter. Monitor patients closely.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Avoid kelp tablets and "thyroid support" supplements. Gentian, walnut, mustard, radish, and St. John's wort have been recommended, but no clinical trials have proven efficacy.
- Curcumin and vitamins A, B_{12}, D_3, and E may be beneficial; longer human studies are needed.
- If large quantities of iodine-rich seaweed such as kombu (*Laminaria japonica*) are consumed, use should be monitored in patients who take thyroid replacement hormones.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss goitrogens in cabbage and *Brassica* vegetables, turnips, rapeseeds, peanuts, cassava, and soybeans; they are inactivated by heating and cooking.
- Encourage use of iodized salt, as permitted. Avoid self-medication with iodine supplements.
- Encourage adequate fluid intake.
- Women who are considering pregnancy may want to be screened to rule out thyroid problems. If their iodine intake is low, or if they live in an area without use of iodized salt, they may need an iodine supplement prescribed from their physician. A normal urinary iodine level would be 150–249 μg/L.
- In many developing countries, children are at high risk of iodine deficiency, VAD, and iron deficiency anemia. Select the proper supplements.
- Because subclinical hypothyroidism is common in individuals who have chronic kidney disease, close monitoring of GFR is recommended (Chonchol et al, 2008).
- Exposure to large doses of metals such as lead, molybdenum, or arsenic may reduce thyroid function, with adverse effects on development, behavior, metabolism and reproduction (Meeker et al, 2009).

Patient Education—Foodborne Illness

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

For More Information

- American Thyroid Association
  http://www.thyroid.org/
- Endocrine Web
  http://www.endocrineweb.com/hypo1.html
- Mayo Clinic
  http://www.mayoclinic.com/health/hypothyroidism/DS00353/DSECTION=symptoms
- Synthroid Information
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PARATHYROID GLANDS

The parathyroid glands have an overall regulatory role with action as a thermostat in the systemic calcium homeostasis to ensure tight regulation of serum calcium concentrations and appropriate skeletal mineralization. Parathyroid hormone (PTH) affects calcium, phosphorus, and vitamin D metabolism by removing calcium from bone to raise serum levels; it promotes hydroxylation of vitamin D to its active form. Calcitonin, in contrast to PTH, decreases serum calcium levels; it is secreted by the thyroid gland.

The body secretes PTH in response to hypocalcemia or hypomagnesemia; the hormone then stimulates osteoclasts to increase bone resorption. PTH also stimulates adenyl cyclase to increase renal tubular calcium resorption and phosphate excretion. PTH works with vitamin D to regulate total body calcium by activating conversion of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D, the active form that stimulates calcium and phosphate absorption from the GI tract. Calcitonin, in contrast to PTH, decreases serum calcium levels and is secreted by the thyroid gland.

Fibroblast growth factor-23 (FGF23) is a hormone that regulates mineral and vitamin D metabolism (Juppner, 2009). Research on the effects of various genes and hormones in bone homeostasis is on-going.

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HYPOPARATHYROIDISM AND HYPOCALCEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Hypoparathyroidism results from a deficiency of PTH from biologically ineffective hormones, damage or accidental removal of the glands, or impaired skeletal or renal response. In the hereditary form, parathyroid glands are either absent or not functioning properly; symptoms appear before age 10. Other causes include magnesium deficiency or neonatal immaturity. If untreated, hypoparathyroidism-retardation-dysmorphism (HRD) may result.

Cancellous bone in hypoparathyroidism is abnormal, suggesting that PTH is required to maintain normal trabecular structure (Rubin et al, 2010). Hypoparathyroidism with hypocalcemia is one of the most common results of damage to parathyroid glands during surgery; in fact, it may be diagnosed during a workup for hypocalcemia.

Vitamin D levels may also be deficient. Intraoperative PTH levels are used widely during parathyroidectomy as an indicator of parathyroid gland function; vitamin D supplementation after surgery may be given to anticipate decreased parathyroid gland function and to avoid symptomatic hypocalcemia (Quiros et al, 2005).

Hypoparathyroidism is a chronic condition that requires lifelong treatment with large doses of calcium and vitamin D supplements. Episodes of tetany are treated with calcium given intravenously to provide quick relief of symptoms. Controlled release of physiological concentrations of PTH can be achieved using a surgically implantable controlled-release delivery system (Anthony et al, 2005).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: There is a hypoparathyroidism-deafness-renal (HDR) dysplasia syndrome which is an autosomal dominant disorder caused by mutations of the GATA3 gene (Ali et al, 2007).

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Weakness, fatigue
- Hyperreflexia
- Chvostek's sign positive (with tapping of facial muscles)
- Irritability or psychosis
- Seizures
- Muscle cramps and tetany
- Tingling of the lips or fingers
- Hair loss, dry skin
- Dental hypoplasia
- Chronic cutaneous moniliasis
- Abnormal heart rhythms on ECG

Lab Work

- PTH (low)
- Ca^{++} (serum levels <2.5–3 mg/dL)
- Urinary Ca^{++} (altered)
- Serum phosphorus (high)
- Mg^{++} (may be low)
- Na^{+}, K^{+}

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Mineral Intake

Assessment Data: Abnormal labs with low PTH, serum calcium 2.4 mg/dL, high serum phosphorus; positive for Chvostek's sign and tetany with muscle cramps and abnormal ECG. Diet history indicates no dairy intake for past several years. Medical diagnosis of hypoparathyroidism after thyroid surgery.

Nutrition Diagnosis (PES): Inadequate mineral (calcium) intake related to nutritional intake low in calcium and hypoparathyroidism as evidenced by low PTH, low serum calcium (2.4), high serum phosphorus, and signs of tetany.

Intervention: Food and nutrient delivery—provide foods rich in calcium (nondairy if necessary) and provide a calcium supplement calculated for body size and age. Educate about the role of calcium in alleviating signs of tetany and abnormal labs. Counsel about nondairy sources of calcium and tips for increasing intake throughout the day.

Monitoring and Evaluation: Resolution of low serum calcium and PTH, high phosphorus. No signs of tetany, muscle cramps, irritability. Improvement in dry skin or hair loss. Normal ECG.

INTERVENTION

OBJECTIVES

- Normalize serum and urinary levels of calcium, phosphorus, and vitamin D.
- Prevent long-term complications such as cataracts, pernicious anemia, Parkinson's disease, and bone disease.
- Prevent mental retardation or malformed teeth in affected children.
- Decrease symptoms of tetany and improve overall health status.

FOOD AND NUTRITION

- Use a high-calcium diet with dairy products, nuts, salmon, peanut butter, broccoli, and other green leafy vegetables. If tolerated, lactose should be included in the diet for better absorption of calcium.
- Oral supplements high in calcium should be used, such as calcium carbonate.
- Reduce excess use of meats, phytates (whole grains), and oxalic acid (spinach, chard, and rhubarb) if the diet contains large amounts.
- Intake of vitamin D and protein should be adequate, at least meeting recommended levels.

Common Drugs Used and Potential Side Effects

- Calcium lactate (8–12 g) may be used. Ergocalciferol (Calciferol) is a vitamin D analog that is used with calcium

supplements in this condition. Calcitriol (Rocaltrol) also may be useful.
- Diuretics sometimes are given to prevent too much calcium from being lost through the urine, which is a problem that can lead to kidney stones. Taking diuretics also reduces the amount of calcium and vitamin D supplements needed.
- Overuse of steroids may cause hypocalcemia.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Indicate which foods are good sources of calcium, phosphorus, and vitamin D.
- Indicate which foods are sources of phytates and avoided, if dietary intake is a concern.
- Discuss role of sunlight exposure in vitamin D formation and how it relates to individual needs.

Patient Education—Foodborne Illness

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

For More Information

- American Society for Bone and Mineral Research
  http://www.asbmr.org/
- Hypoparathyroidism Association
  http://www.hypoparathyroidism.org/
- Medline
  http://www.nlm.nih.gov/medlineplus/ency/article/000385.htm
- National Institutes of Health–Osteoporosis and Related Bone Diseases
  http://www.osteo.org/

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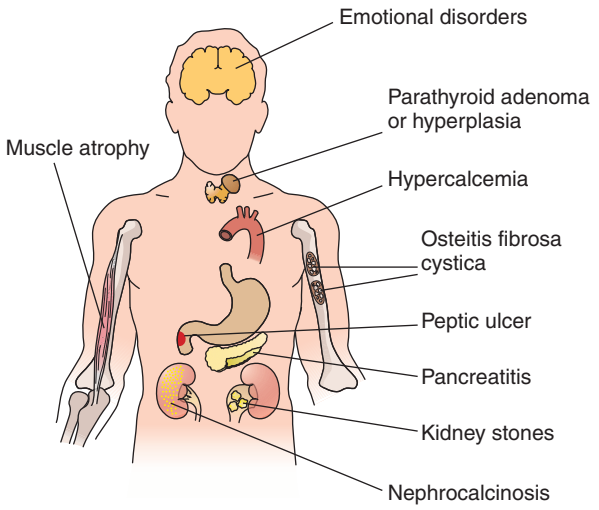
HYPERPARATHYROIDISM AND HYPERCALCEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 2



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

DEFINITIONS AND BACKGROUND

Primary hyperparathyroidism (pHPT) results from parathyroid adenoma in up to 80% of cases, hyperplasia of the parathyroid glands in 10–20% of cases, or cancer. Double parathyroid adenomas occur in 2–15% of pHPT cases (Abboud et al, 2005). pHPT has been associated with premature death in CVDs and should, therefore, be quickly managed (Nilsson et al, 2005).

Secondary hyperparathyroidism (sHPT) occurs in renal failure or even after renal transplantation. Calcitriol deficiency and phosphorus retention are involved in the pathogenesis. Parathyroid gland hyperplasia develops in azotemic patients, producing hypercalcemia and hyperphosphatemia. Secondary hyperparathyroidism in chronic kidney disease is stimulated by dietary phosphate loading and ameliorated by dietary phosphate restriction (Martin et al, 2005). The disorder is complex in that not enough phosphate is cleared from the body; phosphate is released from bone and Vitamin D is not produced. Thereafter, absorption of calcium in the gut is low and serum levels of calcium are lowered.

In children with renal failure, growth can be impaired. Postmenopausal women after Roux-en-Y gastric bypass may show evidence of secondary hyperparathyroidism with elevated bone resorption. There is an effect of early breast tumors on calcium homeostasis; subclinical hyperparathyroidism may increase the risk for breast cancer (Martin et al, 2010). Age-induced increased PTH plasma levels have been associated with cognitive decline and dementia. Increased PTH levels may become a biological marker of both dementia and osteoporosis (Braverman et al, 2009).

Parathyroidectomy can induce long-lasting improvement in regulation of BP, left ventricular diastolic function, and other signs of myocardial ischemia, with improved life expectancy (Nilsson et al, 2005). A minimally invasive procedure is available. After surgery, mild cognitive changes seem to improve, especially for depression and anxiety (Walker et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Peroxisome proliferator-activated receptor gamma PPARgamma2 has a dominant role in controlling osteoblast differentiation and numerous gene–gene interactions suggests there is a "master" regulatory process (Shockley et al, 2009). Familial multiple endocrine neoplasia type 1 and familial tumoral calcinosis account for 2% of pHPT cases. In the second form, altered GALNT3 is usually present. More studies are needed on the effects of genes on calcium and bone homeostasis.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- Weakness, fatigue
- Constipation
- Growth delay or rickets in children
- Anorexia, weight loss
- Increased thirst
- Dehydration
- Itchy skin
- Back pain
- Bone and joint pain
- Bone x-rays showing fractures or reabsorption
- X-rays, ultrasound, or Sestamibi scan of parathyroids

Lab Work

- PTH (>60 pg/mL)
- Serum Ca^{++} (>11 mg/dL)
- Urinary Ca^{++} (elevated)
- Serum phosphorus (high?)
- Calcium-phosphorus product (Ca × P)*
- Albumin-corrected calcium level (Ca corr)
- FGF23 assessment, as available
- Mg^{++}, Na^{+}, K^{+}
- Alb, BUN, Creatinine
- H & H, serum ferritin
- Gluc

*Calcium phosphate product (Ca× Pi) is a clinically relevant tool to estimate the cardiovascular risk of patients with renal failure.

INTERVENTION

OBJECTIVES

- Lower elevated serum calcium and urinary calcium levels. Maintain calcium levels between 8.4 and 9.5 mg/dL.
- Normalize serum phosphate; keep phosphorus between 3.5 and 5.5 mg/dL and calcium × phosphorus product below 55 mg/dL.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Mineral Intake

Assessment Data: Abnormal nutritional labs with PTH 65 pg/mL; serum Ca^{++} 12.5 mg/dL; urinary Ca^{++} levels elevated; elevated serum phosphorus; diagnosis of hyperparathyroidism related to early renal failure without dialysis. Signs of weakness and anorexia. Poor intake because of nausea. Unplanned weight loss of 2 lb recently.

Nutrition Diagnosis (PES): Excessive phosphorus intake related to inability of kidneys to metabolize vitamin D_3 in renal failure as evidenced by abnormal labs (PTH 65 pg/mL; serum Ca^{++} 12.5 mg/dL; urinary Ca^{++} levels elevated; serum phosphorus 6, anorexia and weight loss.

Intervention: Food-Nutrient Delivery—provide a low-phosphorus diet. Educate about foods to avoid and which to include (see Table 9-25). Counsel about dining away from home, packing a lunch, or other relevant guidelines, how to take Sevelamer and Sensipar according to doctor's orders.

Monitoring and Evaluation: Improvement in serum and urinary labs related to calcium, PTH and phosphorus. Resolution of symptoms including weight loss, nausea, anorexia. Able to state foods to avoid when PTH is elevated and when to contact the physician.

- Alleviate constipation, anorexia, weight loss, and weakness.
- Avoid clinical consequences such as renal osteodystrophy, hyperphosphatemia, cardiovascular calcification, extraskeletal calcification, endocrine disturbances, neurobehavioral changes, compromised immune system, altered erythropoiesis, renal stones, and sleep disturbances.
- Prevent rickets and growth delay in children (Sabbagh et al, 2005).
- Prepare for surgery if parathyroidectomy is necessary.

FOOD AND NUTRITION

- Use a low-calcium diet with fewer dairy products, nuts, salmon, peanut butter, and green leafy vegetables.
- Extra fluid is useful to correct or prevent dehydration, which can elevate serum calcium levels.
- Limit phosphorus-containing foods if hyperphosphatemia is present. See Table 9-25. Use alternatives such as nondairy creamer, sorbet, jams and jellies, white rice, noodles with margarine, cream cheese, whipped cream, popcorn, pretzels, gingerale or Kool-aid if extra calories are needed.
- Dietary protein 0.8 g/kg for a balanced intake of protein in adults.

Common Drugs Used and Potential Side Effects

- Vitamin D therapy sends a signal to the parathyroid gland to slow down the making of PTH. This helps to prevent many of the unwanted complications of hyperparathyroidism.

TABLE 9-25 Phosphorus Facts

Normal PO_4 levels in plasma: 2.5–4.5 mg/dL

Total body PO_4 content: 500–700 g (85% in bone)

Dietary Reference Intake: 700 mg

Typical U.S. dietary intake: 1200 mg

GI absorption—Mainly passive, through Na/Pi transporter; enhanced by vitamin D

Kidney is major regulator—mediated by brush border Na/Pi transporter; PTH increases excretion and Vitamin D decreases excretion

Phosphorus-Rich Foods

Dairy Products
- Milk
- Cheese
- Yogurt
- Ice cream
- Pudding and custard

Dried beans and lentils
- Kidney, lima, pinto
- Soy beans
- Split peas
- Black-eyed peas

Nuts and seeds
- Almonds
- Cashews
- Sunflower seeds
- Peanuts and Peanut Butter

Meats
- Liver
- Organ meats
- Sweetbreads
- Smelt, sardines, herring

Miscellaneous
- Beer
- Bran, bran flakes, wheat germ
- Chocolate
- Colas

- Treatment with active vitamin D from analogs can increase VDR expression, inhibit growth of parathyroid tumors, and reduce PTH levels (Akerstrom et al, 2005). Zemplar (paricalcitol) and Hectorol (doxercalciferol) are examples of vitamin D analogs. These products are especially useful for dialysis patients.
- Cinacalcet (Sensipar) has been approved to treat sHPT in renal patients and parathyroid cancer. It also appears to effectively treat pHPT. Cinacalcet normalizes serum calcium with only modest increases in PTH (Sajid-Crockett et al, 2008).
- Phosphate-binding agents that do not contain calcium offer therapeutic alternatives for managing renal osteodystrophy. Sevelamer (Renagel) lowers serum phosphorus and PTH levels without inducing hypercalcemia. Sevelamer binds drugs such as furosemide, cyclosporine, and tacrolimus, making them less effective. The timing of administration should allow several hours between these medicines. Standard protocols are recommended for use of phosphate binders.
- Once-yearly intramuscular cholecalciferol injections (600,000 IU) have been used to correct vitamin D deficiency; controlled trials are needed to determine the effect on PTH levels over time.
- Some antacids may contain high levels of calcium; monitor carefully.
- Bisphosphonates may be needed to decrease risks for osteoporosis.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Conjugated linoleic acid (CLA) reduces prostaglandin E_2 synthesis, which is required for PTH release. More research is needed.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss foods that are sources of calcium, phosphorus, and vitamin D. Indicate food sources of phytates and oxalates, if intake is a concern.
- Discuss role of sunlight exposure in vitamin D formation and how it relates to individual needs.
- Drink plenty of liquids.
- In renal patients, focused counseling may help to clarify misunderstanding of simple dietary facts.
- The doctor should monitor Ca^{++} and P monthly and PTH quarterly after stabilization.
- Exercise and smoking cessation may be needed.

Patient Education—Foodborne Illness

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

For More Information

- American Society for Bone and Mineral Research
  http://www.asbmr.org/
- Endocrine Web
  http://www.endocrineweb.com/hyperpara.html
- National Institutes of Health–Osteoporosis and Related Bone Diseases
  http://www.osteo.org/
- NIDDK
  http://endocrine.niddk.nih.gov/pubs/hyper/hyper.htm

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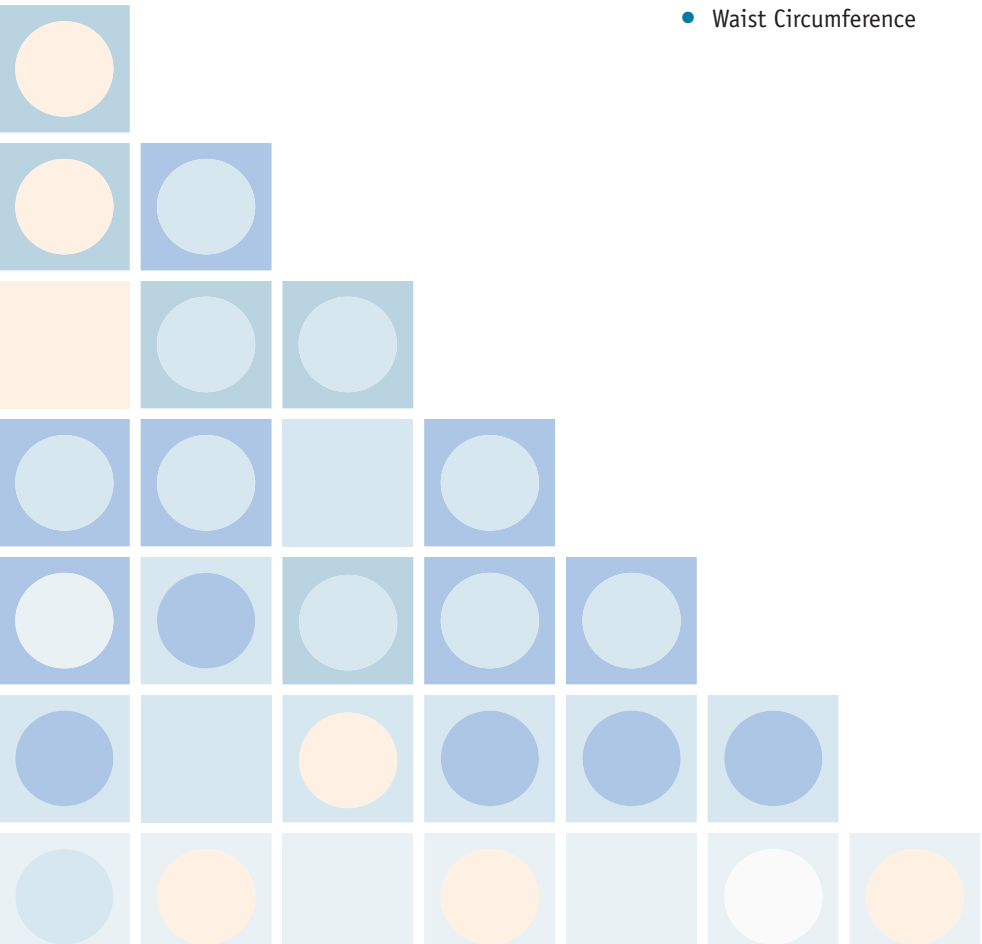
SECTION 10

Malnutrition—Obesity and Undernutrition

CHIEF ASSESSMENT FACTORS

- Anorexia, Nausea, Vomiting, Diarrhea—Frequency, Length of Time
- Blood Pressure, Either Elevated or Very Low Levels
- Body Mass Index (BMI) Using Height and Weight
- Frame Size—Small, Medium, Large
- Goal Weight and Current Percentage of Goal Weight
- Healthy Body Weight Range
- History of Usual Body Weight, Present Weight, Recent Weight Changes,% Usual Body Weight
- Laboratory Values—Glucose, Albumin/Transthyretin, Hemoglobin and Hematocrit (H & H), Lipids, C-Reactive Protein (CRP)
- Medical History—Disordered Eating; Cancer or Other Conditions or Disease States; Menstrual or Reproductive Problems in Women; Smoking and Alcohol Histories
- Physical Activity Level
- Sleep Apnea, Altered Lung Function; Sleep Disorder
- Triceps Skinfold (TSF) Measurements; Arm Muscle Circumference (AMC) Measurements—Comparing Individual Against Own Values Over Time
- Waist Circumference



TYPES OF MALNUTRITION

The malnourished state arises from a combination of inflammation and a disturbed nutrient balance in either undernutrition or overnutrition (Soeters and Schols, 2009). Malnutrition is defined by the American Society of Parenteral and Enteral Nutrition (ASPEN) as any derangement in the normal nutrition status.

Undernutrition may be *primary*—from insufficient intake—or *secondary*—from impaired utilization; see Table 10-1. Malnutrition originates not only from an imbalance of calories but also from psychological, economic, physical, and social factors. Health problems, alcoholism, depression, restricted diets, and mobility problems may play a part. Undernutrition contributes to half of the world's mortality in children, particularly in India and sub-Saharan Africa.

Nutrition Day, which originated in Europe, is now recognized in the United States. The intent is to identify the type and percentages of malnourished patients in hospitals and nursing homes in order to clarify their impact on health outcomes.

Overnutrition is caused by excessive calorie intake and/or inadequate activity; see Table 10-2. The thrifty genotype developed when food was scarce; now, an environment where food is plentiful contributes to the dilemma (Bouchard, 2007). The study of extreme human obesity caused by single gene defects has provided a glimpse into the long-term regulation of body weight through a disrupted hypothalamic leptin-melanocortin system and extremely heterogeneous factors (Ranadive and Vaisse, 2008).

In addition to the change in environment, thirst, hunger, and high calorie beverage intake have changed in the past few decades. The intake of energy-yielding beverages seems to be related to pleasure-oriented, hedonic factors rather than hunger or thirst (McKiernan et al, 2008). Pavlovian cues for rewards become endowed with incentive salience, guiding "wanting" to a learned reward (Tindell et al, 2009). Thus, the hedonic appetite has displaced genuine hunger in the current environment of plenty, perhaps in relation to conditioned stimuli. For example, salted food may be an addictive substance that stimulates the opiate and dopamine receptors in the brain's reward and pleasure center; both salted food and opiate withdrawal stimulate the appetite, increase calorie consumption, and augment the incidence of overeating, becoming overweight, and becoming obese (Cocores and Gold, 2009).

TABLE 10-1 Concerns with Undernutrition

- The lack of adequate macronutrients or selected micronutrients (zinc, selenium, iron, and the antioxidant vitamins) can lead to clinically significant immune deficiency (Cunningham-Rundles et al, 2005). Iron deficiency alters myelination, monoamine neurotransmitter synthesis, and hippocampal energy metabolism in the neonatal period (Georgleff, 2007). Zinc deficiency alters autonomic nervous system regulation and hippocampal and cerebellar development (Georgleff, 2007).
- Inflammation and oxidative stress fuel each other. Cachexia includes distinct metabolic changes that are the result of an acute-phase response (APR) mounted by the host, including increased muscle proteolysis, increased fat lipolysis, and increased hepatic production of acute-phase proteins such as C-reactive protein and fibrinogen (Gullett et al, 2009).
- Children who are undernourished may not reach their potential in many aspects of development. Stunting represents growth failure resulting from poor nutrition and health during the pre- and postnatal periods (Milman et al, 2005). Intrauterine and early neonatal life is a period during which environmental and nutritional influences may produce chronic and frequent infections, long-term effects, and disease risk in adulthood (Buckley et al, 2005).
- The effect of any nutrient deficiency or overabundance on brain development will be governed by the principle of timing, dose, and duration (Georgleff, 2007).
- Bourre (2006) reported on nutritional requirements for the brain. The use of glucose by nervous tissue requires thiamin. Vitamins B_6 and B_{12} are directly involved in the synthesis of some neurotransmitters. Supplementation with cobalamin improves cerebral and cognitive functions in the frontal lobe. Alpha-tocopherol is actively uptaken by the brain and is directly involved in the protection of nervous membranes. Vitamin K is also involved in nervous tissue biochemistry. Iron deficiency anemia is associated with apathy, depression, and fatigue. Iodine deficiency during pregnancy induces severe cerebral dysfunction, actually leading to cretinism.
- Long-chain polyunsaturated fatty acids are important for synaptogenesis, membrane function, and, potentially, myelination (Georgleff, 2007). The rise in autism, learning problems, cognitive decline with age, Alzheimer's, Parkinson's Disease, and the SIDS epidemic has a common link: dietary deficit in omega-3 brain-foods from fish and seafood (Saugstad, 2008).
- Undernutrition in adult women tends to be associated with high levels of undernutrition in children, reflecting overall food/nutrient insecurity (Nube, 2005). Prenatal exposure to famine can lead to low birth weight and cardiovascular disease in adulthood (Painter et al, 2005).
- A woman with a low body mass index (BMI) may have difficulty becoming pregnant; preconceptional undernutrition shortens gestation in those women who do become pregnant (Rayco-Solon et al, 2005).
- **Recent weight loss** appears to be the most important single indicator of nutritional status. Nutritional risk with muscle mass depletion increases lengths of stay in hospitals (Kyle et al, 2005). Undernutrition is associated with increased resting energy expenditure (REE; kcal/kg/d). Reduced respiratory quotient (RQ) and protein synthesis (g/kg/d) occur in patients with coexistent disease; refeeding normalizes this process (Winter et al, 2005).
- Involuntary weight loss can be categorized into one of three primary etiologies (Thomas, 2007): **Starvation** results in a loss of body fat and non-fat mass due to inadequate intake of protein and energy. **Sarcopenia** involves a reduction in muscle mass and strength occurring with normal aging, associated with a reduction in motor unit number and atrophy of muscle fibers; this leads to diminished strength and exercise capacity. **Cachexia** is severe wasting that accompanies disease states such as cancer or immunodeficiency disease.
- Malnutrition in pediatric hospitals ranges from 15–30% of patients, with an impact on growth, morbidity, and mortality; nutrition-support interventions can help (Agostoni et al, 2005).
- If a person has a BMI below 18, there is an increased risk for nutrition-related complications such as infections, poor wound healing, and pressure ulcer development.

(continued)

TABLE 10-1 Concerns with Undernutrition *(continued)*

- Approximately 25–50% of hospital patients have been found to be malnourished. Only 50% of malnourished patients are identified by the medical and nursing staff (Kruizenga et al, 2005).
- Over half of older adults have protein–energy malnutrition at admission or acquire nutritional deficits while hospitalized (Pepersack, 2005). Malnutrition may come from the "11 Ds": disease, drinking alcohol, drugs, deficits (sensory), desertion/isolation, dementia, delirium, depression, destitution, despair, and dysphagia. Cognitive function is particularly sensitive to glucose and insulin availability. Older individuals often have dementia, falls, mobility disorders, malnutrition, end-of-life issues, pressure ulcers, and urinary incontinence to consider.
- Nutritional screening and assessment must become routine. Subjective Global Assessment and the Mini Nutritional Assessment tools are used to detect patients who need preventive nutritional measures (Kyle et al, 2005). The short nutritional assessment questionnaire (SNAQ) is simple and may be used to screen for **appetite changes**—another important factor leading to undernutrition and its consequences (Wilson et al, 2005).
- Outcome monitoring and evaluation should include weight change, use of supplemental drinks and snacks between meals, use of tube feeding or parenteral nutrition, number of consultations by the hospital dietitian, and decreased length of hospital stay (Kruizenga et al, 2005).

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TABLE 10-2 Concerns with Overnutrition

- Obesity caused by excess nutrition or excess storage of fat relative to energy expenditure is a form of malnutrition that is increasingly seen in children (Cunningham-Rundles et al, 2005).
- Leptin is a cytokine-like immune regulator that has complex effects in both overnutrition and in the inflammatory response in malnutrition.
- BMI $>$ 30 is the initial calculated measure used to determine obesity. If a person has too high a BMI, risks increase for high blood pressure, high blood cholesterol, diabetes, orthopedic problems, gallstones, gout, osteoarthritis, sleep apnea, and cancers of the breast, colon, gallbladder, and so on.
- Critical factors that may put a person at risk for obesity, as they accumulate and interact over an individual's life span, include rapid weight gain in infancy and childhood, early puberty, and excessive weight gain in pregnancy (Johnson et al, 2006).
- Obesity in women can lead to several health challenges. Epidemiological evidence shows that being overweight contributes to menstrual disorders, infertility, miscarriage, poor pregnancy outcome, impaired fetal well-being, and diabetes mellitus. Changes in sensitivity to insulin may occur. Pregnant women who are obese are more at risk for pregnancy-induced hypertension and preeclampsia.
- Weight management programs should include a strong component of nutrition education (Klohe-Lehman et al, 2006).
- Obesity in older adults is increasing in prevalence along with related macro- and micronutrient deficiencies (Flood and Carr, 2004).
- Individualized programs with the goal of achieving modest weight reduction in obese patients are likely to result in immediate (e.g., alleviation of arthritic pains and reduction of glucose intolerance) and possibly long-term (e.g., reduction in cardiovascular risk) healthcare benefits.
- Lifestyle modifications are best. Diets based on complex carbohydrates, fibers, red wine, fresh fruit and vegetables, and nonanimal fat may protect against age-related cognitive impairment and dementia (Flood and Carr, 2004).
- Permanent 100-kilocalorie reductions in daily intake among the overweight/obese would eliminate approximately 71.2 million cases of overweight/obesity; in the long term, this could increase national productivity by $45.7 billion annually, and more aggressive diet changes of 500 kilocalories would yield benefits of $133.3 billion (Dall et al, 2009).

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Food variety is another factor with a major influence on energy intake (Epstein et al, 2009). Obese individuals prefer and consume more highly palatable foods more frequently than do persons of normal weight. There are 10 neurophysiologic pathways involved in the selection of food, which include reflexive and uncontrollable neurohormonal responses to food images, cues, and smells; mirror neurons that cause people to imitate the eating behavior of others without awareness; and limited cognitive capacity to make informed decisions about food (Cohen, 2008).

A transition in patterns and food intake occurs for immigrants. Any intervention aimed at reducing obesity must consider ethnicity, income, and degree of acculturation (Chen 2009). The intake of both sweetened drinks and meat increases among individuals emigrating to the United States (Novotny et al, 2009). The use of low-energy dense food (fruits and vegetables) and routine healthy breakfast consumption can help to maintain or lose weight (Greenwood and Stanford, 2008).

Restaurant and fast food consumption, large portion sizes, and consumption of sugar-sweetened beverages are closely associated with weight gain. Fructose from both sucrose and high fructose corn syrup is a concern (Bray, 2009). Fructose acutely increases thermogenesis, triglycerides, and lipogenesis as well as blood pressure; this leads to changes in body weight, fat storage, and triglycerides and increases inflammatory markers (Bray, 2009).

Many tools are available to assess food habits and weight status. Body mass index (BMI) standards are used to determine weight status. BMI correlates well with body fat for most individuals. Table 10-3 provides important weight and BMI calculations and guidelines. Whenever possible, use body mass index (BMI) to estimate a desirable weight range for height. Table 10-4 provides resources and calculations for estimating one's ideal body weight range; the Hamwi method (1974) is listed but is not evidence-based. Table 10-5 provides the standard BMI tables for adults. Table 10-6 provides several short methods for calculating energy needs.

TABLE 10-3 Weight Calculations and Body Mass Index (BMI) Guidelines

Calculation of BMI: (Weight [lb] ÷ Height [in]^2) × 705

BMI more often correctly predicts risks for chronic disease or malnutrition than life insurance tables.

BMI Web sites: There are many Web sites that make it easy to calculate BMI, which may be downloaded to a hand-held device, such as http://www.cdc.gov/nccdphp/dnpa/bmi/calc-bmi.htm.

For both English and metric calculations: http://www.nhlbisupport.com/bmi/bminojs.htm.

Usual Body Weight and% Change:

Calculation of % usual body weight = (Actual Weight/Usual Weight) × 100.

Calculation of % weight change = (Usual Weight − Actual Weight/Usual Weight) × 100.

Waist measurements: Waist circumference correlates with intra-abdominal adipose tissue. It is the most practical anthropometric measurement for assessing a patient's abdominal fat content before and during weight loss treatment. Computed tomography (CT) and magnetic resonance imaging (MRI) are both more accurate but impractical for routine use. Upper body obesity is defined as a waist circumference of >40 inches for men and >35 inches for women; this is the "apple shape." If more weight is around the hips, this "pear shape" has lower metabolic risks. Waist-hip ratio is often more useful in special populations such as HIV-AIDS and in older adults.

Calculation of lean body mass: Body composition is often measured using dual-energy X-ray absorptiometry (DEXA) or electrical impedance absorptiometry (BIA). BIA may underestimate body fat percentage more than DEXA.

Ethnic-specific cut-off points: Compared to Caucasians, African-Americans of the same age, gender, waist circumference, weight, and height may have lower total and abdominal fat mass when measured with DEXA bone density scanning or computed tomography (CT).

Because muscle mass may be higher in blacks, ethnic-specific cutoffs for what values represent overweight and obesity are needed. Dr. Dagogo-Jack summarized these findings at the Endocrine Society's Annual Meeting in 2009. The determination of the health status of all individuals should be based on metabolic indicators of health rather than on BMI alone. Counselors should promote healthy lifestyles that include adequate amounts of physical activity and rest as well as a nutrient-dense diet.

BMI Clinical Guidelines

<18.5	Underweight
18.5–24.9	Normal
25–29.9	Overweight
≥30	Obese
≥40	Morbidly obese

http://www.nhlbi.nih.gov/guidelines/obesity/ob_home.htm.

BMIs for Pregnant Women

The amount of weight a woman should gain during her pregnancy depends on her prepregnant BMI. To avoid complications and long-term health risks, the woman wants to gain enough weight to have a healthy baby but not too much weight. For twins, ideal weight gain is about 35–45 pounds (National Academy of Sciences, http://books.nap.edu/books/0309041384/html/220.html#pagetop).

Prepregnant BMI	Weight Gain During Pregnancy (lb)
≤19.5	28–40
19.6–26	25–35
27–29	15–25
≥30	~13–15

(continued)

TABLE 10-3 Weight Calculations and Body Mass Index (BMI) Guidelines *(continued)*

BMIs for Children

Growth charts are used for children to watch the pattern of their growth. Charts cannot be used to diagnose obesity or malnutrition; if a child is over the 85th percentile or lower than the 5th percentile on the charts, the child should see a doctor. The curves on the growth chart show the pattern of growth. Growth charts for infants and children are calculated the same as for adults but interpreted differently based on BMI. Children are not just small adults; as they grow, their BMI will change. For example, it may be healthy for a 2-year-old child to have a BMI of 16.1 and for that same child to have a BMI of 15.5 at age 6 years and then a BMI of 20 at age 15 years. Children under the 5th percentile should be examined to see if they are normal but small children or if they have a problem that prevents normal growth rate. Growth charts can be found at http://www.cdc.gov/growthcharts. Tables adapted for use in WIC Clinics are at http://www.nal.usda.gov/wicworks/Learning_Center/WIC_growthcharts.html. A calculator for estimating requirements with physical activity levels can be found at http://www.mydr.com.au/tools/child-energy-calculator.

BMIs for Adolescents

An expert consensus panel suggests that a BMI of 95% for age and gender should define obesity. BMI charts are used for the specific age and sex of the adolescent. Being over the 95th percentile on this chart is "overweight," while 85th to 95th percentiles are "at risk of overweight." Most guidance for teens is similar to that for adults. BMI calculators for teens include http://kidshealth.org/teen/food_fitness/dieting/bmi.html or http://apps.nccd.cdc.gov/dnpabmi/Calculator.aspx.

Template for Calculating Total Energy Expenditure

An easy way to calculate daily EERs for adults includes a template based on physical activity level. The estimated energy requirement (EER) equations of the Institute of Medicine DRI Committee account for all factors and measurements to determine physical activity level and energy expended from daily physical activity.

$$\text{BEE} = 293 - 3.8 \times \text{age (years)} + 456.4 \times \text{height (meters)} + 10.12 \times \text{weight (kg)}$$

For women:

$$\text{BEE} = 247 - 2.67 \times \text{age (years)} + 401.5 \times \text{height (meters)} + 8.6 \times \text{weight (kg)}$$

$$\Delta\ \text{PAL} = (\text{METs} - 1) \times [(1.15/0.9) \times \text{Duration (minutes)}]/1440)$$

$$\text{BEE}/[0.0175 \times 1440 \times \text{weight (kg)}]$$

After the Δ PAL is calculated for each physical activity, the physical activity category (PAL: sedentary, low active, active, or very active) is determined based on the basal activity impact on energy expenditure (a factor of 1.1) and the sum of all activities (sum of Δ PAL). This factor accounts for TEF and postexercise increase in energy expenditure. The PAL is automatically calculated as PAL = 1.1 + sum of Δ PALi, where Δ PALi is the list of each reported activity impact on energy expenditure. The template is available in an Excel spreadsheet at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1784117/; accessed 11/7/09.

REFERENCE

Gerrior S, et al. An easy approach to calculating estimated energy requirements. *Prev Chronic Dis*. 3:129A, 2006.

BMI and Mortality

Both general adiposity and abdominal adiposity are associated with the risk of death and support the use of waist circumference or waist-to-hip ratio in addition to BMI in assessing the risk of death (Pischon et al, 2008). Lean men and women (BMI <18 kg/m^2) experience increased all-cause mortality compared with those with a BMI between 20 and 22 kg/m^2, particularly for cardiovascular and respiratory diseases.

It is important that public health messages regarding healthy eating are aimed at maintaining a healthy body weight rather than just "losing weight" (Thorogood, 2003). The following table indicates the lower ranges of BMI, below which mortality increases (Stevens, 2000).

Ages 20–29	BMI: Men 21.4; Women 19.5
Ages 30–39	BMI: Men 21.6; Women 23.4
Ages 40–49	BMI: Men 22.9; Women 23.2
Ages 50–59	BMI: Men 25.8; Women 25.2
Ages 60–69	BMI: Men 26.6; Women 27.3

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Thorogood M. Relation between body mass index and mortality in an unusually slim cohort. *J Epidemiol Community Health*. 57:130, 2003.

Using BMI for Older Adults

TEE and physical activity level (PAL, defined as the ratio of total to resting energy expenditure) decline progressively throughout adult life in both normal weight and overweight men and women (Roberts and Dallal, 2005). In normal weight individuals (defined as BMI 18.5–25) TEE falls by approximately 150 kilocalories per decade, and PAL falls from an average of 1.75 in the second decade of life to 1.28 in the ninth decade (Roberts and Dallal, 2005). Thermic effect of food does not appear to change.

Waist-to-hip ratio (WHR) rather than BMI appears to be a more appropriate yardstick for health risk among older adults over age 70 (Srikanthan et al, 2009).

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Roberts SB, Dallal AG. Energy requirements and aging. *Public Health Nutr*. 8:1028, 2005.
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TABLE 10-4 Calculations of Ideal Body Weight Range

Estimated Ideal Body Weight (Hamwi Method)–Not a Validated Method

Medium-frame women: allow 100 lb for first 5 ft of height, plus 5 lb for each additional inch

Medium-frame men: allow 106 lb for first 5 ft of height plus 6 lb for each additional inch

Small/large frame: subtract/add 10%

REFERENCES

Hamwi GJ. Therapy: changing dietary concepts. In: Danowski TS, ed. *Diabetes mellitus: diagnosis and treatment*. New York: American Diabetes Federation, 1974:612–623.

Adjustment for Paraplegia or Quadriplegia

Paraplegic: ideal weight minus 5–10%

Quadriplegic: ideal weight minus 10–15%

REFERENCE

O'Brien RY. Spinal cord injury. In: Gines DJ, ed. *Nutrition management in rehabilitation*. Rockville, MD: Aspen Publishers, Inc., 1990:165.

Adjustment for Amputation

$Wt_E = Wt_0/1 - P$

Key: Wt_E = estimate of total body weight; Wt_0 = observed body weight; P = proportion of total body weight represented by missing limb.

REFERENCE

Himes JH. New equation to estimate body mass index in amputees. *J Am Diet Assoc*. 95:646, 1995.

Adjustment for Obese Patients

Indirect calorimetry is the "gold standard" for determining energy requirements in the obese patient, or equations can be used, such as 21 kcal/kg actual weight. Formulas for adjusted body weight for obesity [(actual body weight − ideal body weight) × 0.25 + ideal body weight] have not been validated, and predictive equations for resting metabolic rates (RMR) have many flaws. The Harris-Benedict Equation (Harris and Benedict, 1919) may lead to overfeeding, particularly in older adults. Estimated energy needs should be based on RMR using indirect calorimetry; if RMR cannot be measured, then the Mifflin-St. Jeor equation using **actual** weight is the most accurate for estimating RMR for overweight and obese individuals.

Mifflin-St. Jeor Equation:

Men: RMR = 9.99 × weight + 6.25 × height − 4.92 × age + 5

Women: RMR = 9.99 × weight + 6.25 × height − 4.92 × age − 161

For the obese elderly (Frankenfield et al, 2009): Mifflin (0.71) + T_{max} (85) + Ve(64) − 3085 (ADA, Weight Management, 2009; Dobratz et al, 2007).

Adjustment for Critically Ill Patients

In critical illness, energy needs often fluctuate substantially. Nutrition delivery may be influenced by the risk of refeeding syndrome, hypocaloric feeding regimens, inadequate access, feeding intolerance, and feeding-delay for procedures (Walker and Heuberger, 2009). No equation accurately predicts REE in hospitalized patients. Only indirect calorimetry (IC) will provide accurate assessment of energy needs (Boullata et al, 2007).

When comparing popular equations (Harris-Benedict, Ireton-Jones, Penn State 1998 and 2003; Swinamer, 1990), prediction accuracy is rarely within 10% of the measured energy expenditure (Walker and Heuberger, 2009). Thirteen studies comparing RMR and the Harris-Benedict equation without adjustments report an underestimation of energy needs in the critically ill population, by as much as 1000 kilocalories or more (ADA, 2009).

Use of hand-held indirect calorimeters have significant advantages when metabolic carts are not available (Spears et al, 2009). Prediction of metabolic rate is imperfect and requires clinical judgment. The ADA evidence analysis library has described comparisons of these estimated calculations (ADA, 2009). The Penn State equation provides the most accurate assessment of metabolic rate in critically ill patients (79%) if indirect calorimetry is unavailable (Frankenfield et al, 2009). This equation follows: RMR = BMR (0.85) + V_E (33) + T_{max} (175) − 6433

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American Dietetic Association (ADA). Position Paper on Weight Management. *J Am Diet Assoc*. 100:330, 2009.
Boullata J, et al. Accurate determination of energy needs in hospitalized patients. *J Am Diet Assoc*. 107:393, 2007.
Dobratz JR, et al. Predicting energy expenditure in extremely obese women. *JPEN J Parenter Enteral Nutr*. 31:217, 2007.
Frankenfield DC, et al. Analysis of estimation methods for resting metabolic rate in critically ill adults. *JPEN J Parenter Enteral Nutr*. 33:27, 2009.
Harris J, Benedict F. *A biometric study of basal metabolism in man*. Washington, DC: Carnegie Institute of Washington, 1919.
Spears KE, et al. Hand-held indirect calorimeter offers advantages compared with prediction equations, in a group of overweight women, to determine resting energy expenditures and estimated total energy expenditures during research screening. *J Am Diet Assoc*. 109:836, 2009.
Walker RN, Heuberger RA. Predictive equations for energy needs for the critically ill. *Respir Care*. 54:509, 2009.

TABLE 10-5 **Body Mass Index Table for Adults**

Obese (>30) | Overweight (25-30) | Normal (18.5-25) | Underweight (<18.5)

HEIGHT in feet/inches and centimeters

WEIGHT		4'8"	4'9"	4'10"	4'11"	5'0"	5'1"	5'2"	5'3"	5'4"	5'5"	5'6"	5'7"	5'8"	5'9"	5'10"	5'11"	6'0"	6'1"	6'2"	6'3"	6'4"	6'5"
lbs	(kg)	142cm		147	150	152	155	157	160	163	165	168	170	173	175	178	180	183	185	188	191	193	196
260	**(117.9)**	**58**	**56**	**54**	**53**	**51**	**49**	**48**	**46**	**45**	**43**	**42**	**41**	**40**	**38**	**37**	**36**	**35**	**34**	**33**	**32**	**32**	**31**
255	(115.7)	57	55	53	51	50	48	47	45	44	42	41	40	39	38	37	36	35	34	33	32	31	30
250	**(113.4)**	**56**	**54**	**52**	**50**	**49**	**47**	**46**	**44**	**43**	**42**	**40**	**39**	**38**	**37**	**36**	**35**	**34**	**33**	**32**	**31**	**30**	**30**
245	(111.1)	55	53	51	49	48	46	45	43	42	41	40	38	37	36	35	34	33	32	31	31	30	29
240	**(108.9)**	**54**	**52**	**50**	**48**	**47**	**45**	**44**	**43**	**41**	**40**	**39**	**38**	**36**	**35**	**34**	**33**	**33**	**32**	**31**	**30**	**29**	**28**
235	(106.6)	53	51	49	47	46	44	43	42	40	39	38	37	36	35	34	33	32	31	30	29	29	28
230	**(104.3)**	**52**	**50**	**48**	**46**	**45**	**43**	**42**	**41**	**39**	**38**	**37**	**36**	**35**	**34**	**33**	**32**	**31**	**30**	**30**	**29**	**28**	**27**
225	(102.1)	50	49	47	45	44	43	41	40	39	37	36	35	34	33	32	31	31	30	29	28	27	27
220	**(99.8)**	**49**	**48**	**46**	**44**	**43**	**42**	**40**	**39**	**38**	**37**	**36**	**34**	**33**	**32**	**32**	**31**	**30**	**29**	**28**	**27**	**27**	**26**
215	(97.5)	48	47	45	43	42	41	39	38	37	36	35	34	33	32	31	30	29	28	28	27	26	25
210	**(95.3)**	**47**	**45**	**44**	**42**	**41**	**40**	**38**	**37**	**36**	**35**	**34**	**33**	**32**	**31**	**30**	**29**	**28**	**28**	**27**	**26**	**26**	**25**
205	(93.0)	46	44	43	41	40	39	37	36	35	34	33	32	31	30	29	29	28	27	26	26	25	24
200	**(90.7)**	**45**	**43**	**42**	**40**	**39**	**38**	**37**	**35**	**34**	**33**	**32**	**31**	**30**	**30**	**29**	**28**	**27**	**26**	**26**	**25**	**24**	**24**
195	(88.5)	44	42	41	39	38	37	36	35	33	32	31	31	30	29	28	27	26	26	25	24	24	23
190	**(86.2)**	**43**	**41**	**40**	**38**	**37**	**36**	**35**	**34**	**33**	**32**	**31**	**30**	**29**	**28**	**27**	**26**	**26**	**25**	**24**	**24**	**23**	**23**
185	(83.9)	41	40	39	37	36	35	34	33	32	31	30	29	28	27	27	26	25	24	24	23	23	22
180	**(81.6)**	**40**	**39**	**38**	**36**	**35**	**34**	**33**	**32**	**31**	**30**	**29**	**28**	**27**	**27**	**26**	**25**	**24**	**24**	**23**	**22**	**22**	**21**
175	(79.4)	39	38	37	35	34	33	32	31	30	29	28	27	27	26	25	24	24	23	22	22	21	21
170	**(77.1)**	**38**	**37**	**36**	**34**	**33**	**32**	**31**	**30**	**29**	**28**	**27**	**27**	**26**	**25**	**24**	**24**	**23**	**22**	**22**	**21**	**21**	**20**
165	(74.8)	37	36	34	33	32	31	30	29	28	27	27	26	25	24	24	23	22	22	21	21	20	20
160	**(72.6)**	**36**	**35**	**33**	**32**	**31**	**30**	**29**	**28**	**27**	**27**	**26**	**25**	**24**	**24**	**23**	**22**	**22**	**21**	**21**	**20**	**19**	**19**
155	(70.3)	35	34	32	31	30	29	28	27	27	26	25	24	24	23	22	22	21	20	20	19	19	18
150	**(68.0)**	**34**	**32**	**31**	**30**	**29**	**28**	**27**	**27**	**26**	**25**	**24**	**23**	**23**	**22**	**22**	**21**	**20**	**20**	**19**	**19**	**18**	**18**
145	(65.8)	33	31	30	29	28	27	27	26	25	24	23	23	22	21	21	20	20	19	19	18	18	17
140	**(63.5)**	**31**	**30**	**29**	**28**	**27**	**26**	**26**	**25**	**24**	**23**	**23**	**22**	**21**	**21**	**20**	**20**	**19**	**18**	**18**	**17**	**17**	**17**
135	(61.2)	30	29	28	27	26	26	25	24	23	22	22	21	21	20	19	19	18	18	17	17	16	16
130	**(59.0)**	**29**	**28**	**27**	**26**	**25**	**25**	**24**	**23**	**22**	**22**	**21**	**20**	**20**	**19**	**19**	**18**	**18**	**17**	**17**	**16**	**16**	**15**
125	(56.7)	28	27	26	25	24	24	23	22	21	21	20	20	19	18	18	17	17	16	16	16	15	15
120	**(54.4)**	**27**	**26**	**25**	**24**	**23**	**23**	**22**	**21**	**21**	**20**	**19**	**19**	**18**	**18**	**17**	**17**	**16**	**16**	**15**	**15**	**15**	**14**
115	(52.2)	26	25	24	23	22	22	21	20	20	19	19	18	17	17	16	16	16	15	15	14	14	14
110	**(49.9)**	**25**	**24**	**23**	**22**	**21**	**21**	**20**	**19**	**19**	**18**	**18**	**17**	**17**	**16**	**16**	**15**	**15**	**15**	**14**	**14**	**13**	**13**
105	(47.6)	24	23	22	21	21	20	19	19	18	17	17	16	16	16	15	15	14	14	13	13	13	12
100	**(45.4)**	**22**	**22**	**21**	**20**	**20**	**19**	**18**	**18**	**17**	**17**	**16**	**16**	**15**	**15**	**14**	**14**	**14**	**13**	**13**	**12**	**12**	**12**
95	(43.1)	21	21	20	19	19	18	17	17	16	16	15	15	14	14	14	13	13	13	12	12	12	11
90	**(40.8)**	**20**	**19**	**19**	**18**	**18**	**17**	**16**	**16**	**15**	**15**	**15**	**14**	**14**	**13**	**13**	**13**	**12**	**12**	**12**	**11**	**11**	**11**
85	(38.6)	19	18	18	17	17	16	16	15	15	14	14	13	13	13	12	12	12	11	11	11	10	10
80	**(36.3)**	**18**	**17**	**17**	**16**	**16**	**15**	**15**	**14**	**14**	**13**	**13**	**13**	**12**	**12**	**11**	**11**	**11**	**11**	**10**	**10**	**10**	**9**

Note: BMI values rounded to the nearest whole number. BMI categories based on CDC (Centers for Disease Control and Prevention) criteria.

www.vertex42.com BMI = Weight[kg] / (Height[m] x Height[m]) = 703 x Weight[lb] / (Height[in] x Height[in])

For more information, see http://www.nhlbi.nih.gov/guidelines/obesity/bmi_tbl.htm.

TABLE 10-6 Short Methods for Calculating Energy Needs

Weight and Height Conversion Factors

1 kg = 2.2 lb
1 lb = 0.453 kg
1 ft = 30.48 cm
1 in = 2.54 cm
1 cm = 0.39 in
1 m = 39.37 in

	Level of Activity or Illness		
Goal	**Low**	**Moderate**	**High**
Lose weight	15 kcal/kg	20 kcal/kg	25 kcal/kg
Maintain weight	20 kcal/kg	25 kcal/kg	30 kcal/kg
Gain weight	25 kcal/kg	30 kcal/kg	35 kcal/kg

Energy Needs Based on Gender for Adults

Men, active women	15 kcal/lb body weight
Most women, sedentary men, and adults over 55 years	13 kcal/lb body weight
Sedentary women, obese adults	10 kcal/lb body weight
Pregnant women	
1st trimester	13–15 kcal/lb body weight
2nd and 3rd trimester	16–17 kcal/lb body weight
Lactating women	15–17 kcal/lb body weight

Energy Needs Based on Age for Children

There is a general decline in the calories needed per pound as a child gets older. These figures are not accurate for obese children.

0–12 months	55 kcal/lb body weight
1–10 years	45–36 kcal/lb body weight
11–15 years–young women	17 kcal/lb body weight
11–15 years–young men	30 kcal/lb body weight
16–20 years–young women	15 kcal/lb body weight
16–20 years–young men	**18 kcal/lb body weight**

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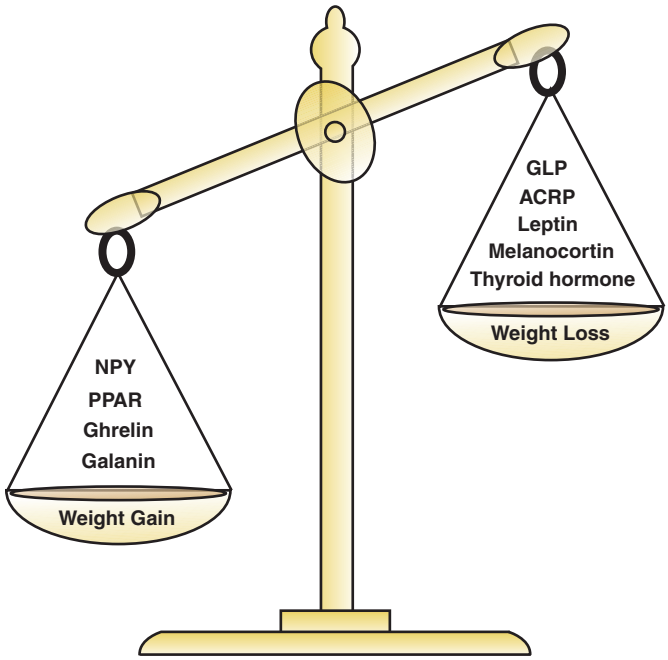
OVERNUTRITION

OVERWEIGHT AND OBESITY

NUTRITIONAL ACUITY RANKING: LEVEL 3–4



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

DEFINITIONS AND BACKGROUND

Overweight is defined as a BMI of 25–29; obesity is defined as a BMI of 30 or more. Obesity is a problem because the percentage of body fat is much greater than lean body mass. Micronutrients are often consumed at lower than desirable levels, while macronutrients are eaten in large amounts. Obesity has reached epidemic proportions in the United States, with 35.1% of adults being classified as obese (Catenacci et al, 2009). The World Health Organization (WHO) labels the increase in obesity and related syndromes as an epidemic in both industrialized countries and in developing countries (Misra and Khurana, 2008).

Obesity is a complex multifactorial chronic disease that develops from an interaction of genetic, social, behavioral, cultural, physiological, and metabolic factors (NHLBI, 2009; Thorleifsson et al, 2009). Genetics account for 30–40% of the variations in weight between individuals (Pi-Sunyer and Kris-Etherton, 2005). Indeed, weight and BMI are highly heritable (Thorleifsson et al, 2009). Environmental factors include low physical activity, energy intake, smoking cessation, overconsumption of high-fat foods.

The prevalence of overweight (BMI >25) and obesity (BMI >30) has steadily increased over the past decade. Approximately 34% of American adults are obese, and 14.6% of low-income, preschool-aged children are obese (CDC, 2009). Blacks have a 51% higher prevalence of obesity than whites, and Hispanics have a 21% higher obesity prevalence compared with whites (CDC, 2009). However, abdominal visceral adipose tissue (VAT) is significantly greater in white men and women; African American men and women have higher subcutaneous adipose tissue (SAT) than whites (Katzmarzyk et al, 2009). An increased understanding of race-based differences in adiposity in specific body depots helps to explain the differential health risks associated with obesity.

The current generation in the United States may have a shorter life expectancy than that of their parents if the obesity epidemic is not controlled (Catenacci et al, 2009). In Project EAT, researchers found that the use of low-calorie soft drinks was a marker for more general dietary behaviors and weight concerns; there was no association between sugar-sweetened beverage consumption, juice consumption, and adolescent weight gain over a 5-y period; and adolescents who consumed little or no white milk gained significantly more weight than their peers who consumed white milk (Vaneslow et al, 2009).

Recent evidence also shows a link between obesity and viral infections, particularly with human adenoviruses (Atkinson, 2007; Mitra and Clarke, 2009). The human distal gut harbors microbiota that provide important metabolic capabilities including the ability to extract energy from otherwise indigestible dietary polysaccharides (Turnbaugh et al, 2009). Metagenomic studies demonstrated that certain mixes of gut microbiota may protect or predispose the host to obesity by increasing dietary energy harvest, promoting fat deposition, and triggering systemic inflammation (Tsai and Coyle, 2009). Each person's gut microbial community varies in the specific bacterial lineages present, which may lead to differences in leanness or obesity (Turnbaugh et al, 2009).

Both overweight and obesity increase the risks of contracting chronic diseases, secondary symptoms, and impaired quality of life. Waist circumference, waist-hip ratio, and BMI may predict chronic disease and mortality. Hypertension is common in persons with central-type obesity, and women who have a BMI over 30 may have problems with fertility. Morbid obesity (BMI >40) is a strong predictor of premature death. When a patient reaches the overweight stage, he or she should be given guidance on how to avoid obesity (see Table 10-7 and figure).

Reimbursement for obesity counseling and management is a complicated issue. Not everyone loses weight easily or steadily, and there are indirect costs such as pain and suffering to consider. BMI can help predict those who may benefit from weight loss counseling, but there are no guarantees of success.

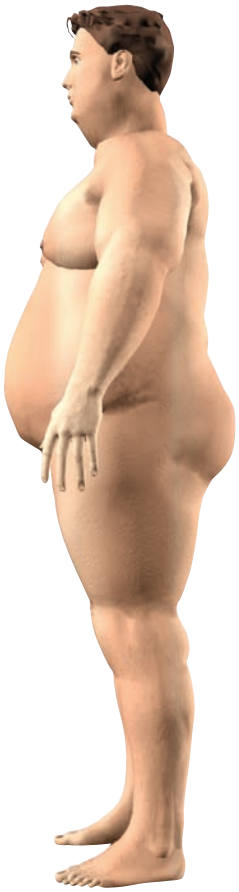
There are varied options for the management of overweight and obese patients including dietary approaches, altered physical activity patterns, behavior therapy techniques, pharmacotherapy, surgery, and combinations of these techniques. Studies have shown that small changes in weight and increases in physical activity can make significant improvements in health.

Starvation diets are not the solution. Orthostatic hypotension may complicate very low–calorie diets (VLCD) because of sodium depletion and depressed sympathetic nervous system activity. It is more desirable to calculate basal energy

TABLE 10-7 Suggested Weights for Initiation of Weight Management Counseling

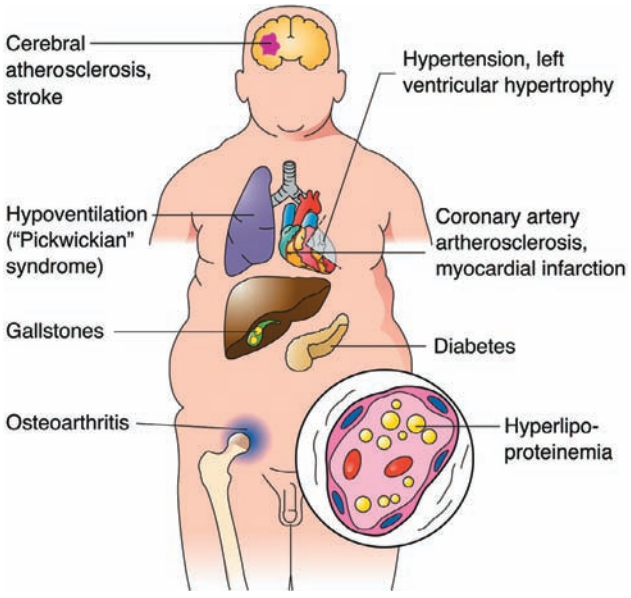
Height (in)	Overweight in lb (BMI >25)	Obese in lb (BMI >30)
58	119	143
59	124	148
60	128	153
61	132	158
62	136	164
63	141	169
64	145	174
65	150	180
66	155	186
67	159	191
68	164	197
69	169	203
70	174	207
71	179	215
72	184	221
73	189	227
74	194	233
75	200	240
76	205	246

Derived from: National Heart, Lung, and Blood Institute. Body mass index table. Accessed August, 8, 2005 at http://www.nhlbi.nih.gov/guidelines/obesity/bmi_tbl.pdf.



An obese male. LifeART image copyright © 2010 Lippincott Williams & Wilkins. All rights reserved.

requirements for the individual and to determine a reasonable energy intake accordingly. Evidence suggests that weight loss diets should include moderate carbohydrate (35–50% of energy), moderate fat (25–35% of energy), and protein as 25–35% of energy (Schoeller and Buchholz, 2005). Specifically, avoid consuming large portions of energy-dense foods or snacks, high-calorie beverages, and foods with empty calories (Pi-Sunyer and Kris-Etherton, 2005).



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

Energy-reduced, isocaloric very-low-carbohydrate, high-fat (VLCHF) diets and high-carbohydrate, low-fat (HCLF) diets produce similar weight loss results (Tay et al, 2008). However, the use of appropriate types of fat is important to protect the heart. Comparisons of the biological effect of popular diets such as Atkins, South Beach, and Ornish during weight maintenance periods show that the Atkins diet leads to the least favorable results in flow-mediated vasodilation (Miller et al, 2009). The HCLF diet has favorable effects on the blood lipid profile (Tay et al, 2008). To estimate the percentage of fat calories for a nutritional plan, review Table 10-8.

Hormones, genes, nutrients and the central nervous system converge to manage energy intake. Dysregulation in the system of fuel-sensing signals may underlie problems such as obesity and diabetes (Sandoval et al, 2008). Leptin is a mediator of long-term regulation of energy balance, suppressing food intake, and inducing weight loss. Leptin is produced within white adipose tissue, brown fat, the placenta, and in a fetal heart, bone, and cartilage. As the amount of fat stored in adipocytes rises, leptin is released into the blood and signals to the brain that the body has had enough to eat. Altered leptin receptor genes (LEPR) can lead to changes in BMI over time. Obesity markedly influences serum insulin, leptin, growth hormone (GH) secretion, and free fatty acid (FFA) levels. Most overweight people have altered levels of serum leptin.

TABLE 10-8 Calculation of Fat Grams

Divide desired body weight by 2. Examples:

120 lb/2 = 60 g	130 lb/2 = 65 g
140 lb/2 = 70 g	150 lb/2 = 75 g
160 lb/2 = 80 g	170 lb/2 = 85 g
180 lb/2 = 90 g	190 lb/2 = 95 g
200 lb/2 = 100 g	210 lb/2 = 105 g

Hormones and peptides such as melatonin, ghrelin, obestatin, and leptin perform dual functions in the pancreas by maintaining metabolic homeostasis; appetite-controlling neuropeptides such as ghrelin, orexin A, and neuropeptide Y regulate pancreatic secretions (Chandra and Liddle, 2009). Ghrelin acts quickly for meal initiation and is produced in the stomach.

Satiety signals from the pancreas and intestine include cholecystokinin, peptide YY, pancreatic polypeptide (PP), glucagon-like peptide 1 (GLP-1), oxyntomodulin (OXM), and amylin (Wren and Bloom, 2007). Cholecystokinin induces satiety by interacting through CCK-1 receptors located in the hindbrain; it also inhibits the expression of orexigenic peptides in the hypothalamus and prevents stimulation of specialized neurons by ghrelin (Chandra and Liddle, 2007). Ghrelin, peptide YY, and cholecystokinin are all influenced by macronutrient intake (Orr and Davy, 2005).

Elevated cholecystokinin levels decrease appetite and also reduce intestinal inflammation caused by parasites and bacterial toxins (Chandra and Liddle, 2007). Elevated inflammatory markers—such as tumor necrosis factor (TNF) alpha, soluble TNF receptor II (sTNF-RII), interleukin-6 (IL-6), and C-reactive protein (CRP)—are characteristically found in the serum in obese patients. Night eating syndrome (NES) is also affected by disordered neuroendocrine functioning; see Table 10-9.

It is important to discuss psychological well-being, particularly feelings about food and body image. The Power of Food Scale (PFS) assesses individual differences in appetitive responsiveness to rewarding properties of the food environment; it is a reliable and valid tool for assessing food cravings and binge eating (Lowe and Butryn, 2007). The PFS evaluates the appetite for palatable foods at three different levels of food proximity: food available, food present, and food tasted (Lowe et al, 2009). See Table 10-10 for the questions asked in the Power of Food Scale. Setting achievable goals will require individualized approaches (Nonas and Foster, 2009).

Self-esteem, body image, self-efficacy, locus of control, motivation, stress management, problem-solving and

TABLE 10-9 Night Eating Syndrome Description and Questionnaire

Night eating syndrome (NES) is a special condition that affects 1–1.5% of the general population, 6%–16% of patients in weight reduction programs, and 8%–42% of candidates for bariatric surgery (Stunkard et al, 2008). Viewed as a delay in the circadian rhythm of food intake, NES is defined by two core criteria: evening hyperphagia (ingestion of at least 25% of daily calories after supper) with awakenings and ingestions at least three times a week (Stunkard et al, 2008). Single PET scans have shown significant elevation of serotonin transporters in the midbrain of night eaters; this is from a genetic vulnerability transmitted as part of the established heritability triggered by stress (Lundgren et al, 2006). Because of elevations in serotonin transporter levels, sertraline (an SSRI) helps to alleviate impaired circadian rhythm and resulting NES (O'Reardon et al, 2006).

Night Eating Questionnaire

1. How hungry are you usually in the morning?
2. When do you usually eat for the first time?
3. Do you have cravings or urges to eat snacks after supper but before bedtime?
4. How much control do you have over your eating between supper and bedtime?
5. How much of your daily food intake do you consume after suppertime?
6. Are you currently feeling blue or down in the dumps?
7. When you are feeling blue, when is your mood lower?
8. How often do you have trouble getting to sleep?
9. Other than using the bathroom, how often do you get up in the middle of the night?
10. Do you have cravings or urges to eat snacks when you wake up at night?
11. Do you eat in order to get back to sleep when you awake at night?
12. When you get up in the middle of the night, how often do you snack?
13. If you snack in the middle of the night, how aware are you of your eating?
14. How much control do you have over your nighttime eating?

Allison KC, et al. The Night Eating Questionnaire (NEQ): psychometric properties of a measure of severity of the night eating syndrome. *Eat Behav.* 9:62, 2008. Accessed November 1, 2009 at http://www.med.upenn.edu/weight/nighteatingform.shtml.

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Stunkard A, et al. Issues for DSM V: Night eating syndrome. *Am J Psychiatry*. 265:424, 2008.

TABLE 10-10 Power of Food Scale

Please indicate the extent to which you agree that the following items describe you. Use the following 1–5 scale for your responses:

1 don't agree at all
2 agree a little
3 agree somewhat
4 agree
5 strongly agree

1. I find myself thinking about food even when I'm not physically hungry.
2. When I'm in a situation where delicious foods are present, but I have to wait to eat them, it is very difficult for me to wait.
3. I get more pleasure from eating than I do from almost anything else.
4. I feel that food is to me like liquor is to an alcoholic.
5. If I see or smell a food I like, I get a powerful urge to have some.
6. When I'm around a fattening food I love, it's hard to stop myself from at least tasting it.
7. I often think about what foods I might eat later in the day.
8. It's scary to think of the power that food has over me.
9. When I taste a favorite food, I feel intense pleasure.
10. When I know a delicious food is available, I can't stop myself from thinking about having some.
11. I love the taste of certain foods so much that I can't avoid eating them even if they're bad for me.
12. When I see delicious foods in advertisements or commercials, it makes me want to eat.
13. I feel like food controls me rather than the other way around.
14. Just before I taste a favorite food, I feel intense anticipation.
15. When I eat delicious food, I focus a lot on how good it tastes.
16. Sometimes, when I'm doing everyday activities, I get an urge to eat "out of the blue" (for no apparent reason).
17. I think I enjoy eating a lot more than most other people.
18. Hearing someone describe a great meal makes me really want to have something to eat.
19. It seems like I have food on my mind a lot.
20. It's very important to me that the foods I eat are as delicious as possible.
21. Before I eat a favorite food, my mouth tends to flood with saliva.

Didie ER. The Power of Food Scale (PFS): development and theoretical evaluation of a self-report measure of the perceived influence of food. A thesis submitted to the faculty of Drexel University, June 2003. Accessed November 2, 2009 at http://dspace.library.drexel.edu/bitstream/1860/205/7/didie_thesis.pdf.

decision-making, and assertiveness are important considerations. Stress may cause those who are overweight to add more pounds (Block et al, 2009). Elevated cortisol or other hormone levels may be the trigger from stress.

Traditional weight management programs can be helpful. Sustained modest weight loss by obese adults can result in substantial health and economic benefits. Successful weight management to improve overall health for adults requires a lifelong commitment to healthy lifestyle behaviors emphasizing sustainable and enjoyable eating practices and daily physical activity (Seagle et al, 2007). The American Dietetic Association has recommended eight medical nutrition therapy visits for adult weight management. (See Figure on page 613)

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Research into the genetics of obesity is extensive (Farooqi and O'Rahilly, 2007). The human Ob gene has been mapped to chromosome 7. The FTO (fat mass and obesity-associated) gene is found on chromosome 16. A genome-wide association (GWA) study has linked thousands of SNPs close to or in the FTO, melanocortin-4 receptor (MC4R,) brain-derived neurotrophic factor (BDNF), and SH2B adapter protein (SH2B1) genes (Thorleifsson et al, 2009). Other genes may play a role. The fatty-acid binding protein (FABP2) gene is associated with fat absorption and metabolism. The peroxisome proliferator-activated receptor gamma (PPARG) gene has a key role in the formation of fat cells. The adrenergic beta-2-receptor (ADRB2) gene mobilizes fat cells for energy, whereas the adrenergic beta-3-receptor (ADRB3) gene regulates the breakdown of fat from tissues in response to exercise. The glutamate decarboxylase 2 (GAD2) gene is released from pancreatic and brain cells. GAD2 codes for the GABA neurotransmitter, which regulates food intake. Clearly, these multiple hormones and genes suggest that future obesity treatment will require a genetic profile and individualized plan.

Clinical/History

- Height
- Weight
- BMI
- Desirable BMI
- Weight changes
- Percentage of excess weight
- Percentage of body fat
- Diet history
- Blood pressure (BP)
- Skinfold thickness
- Waist circumference
- Sleep apnea

Lab Work

- CRP (elevated)
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Uric acid
- Triiodothyronine (T3), thyroxine (T4)
- Glucose (Gluc)
- Cholesterol (Chol)
- Triglycerides (Trig)
- Hypoxemia
- Plasma cortisol

INTERVENTION

OBJECTIVES

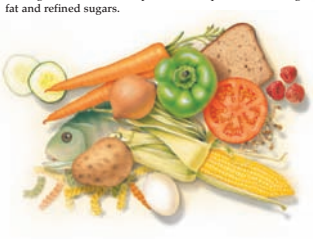
The Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (NHLBI, 2009) suggest the following objectives:

1. Reduce body weight, maintain a lower body weight over the long term, and prevent further weight gain. It is better to maintain a moderate weight loss over a prolonged period than to regain from a marked weight loss. See Figure 10-6 for tips.

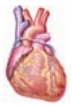
MAINTAINING A HEALTHY WEIGHT

Healthy Diet Plan

If you are concerned about your diet, no particular food plan is magical and no particular food must be either included or avoided. Your diet should consist of foods that you like or can learn to like, that are available to you, and that are within your means. The most effective diet programs for weight loss and maintenance are based on physical activity and reasonable serving sizes, with less frequent consumption of foods high in fat and refined sugars.

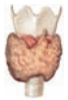


Physiological Hazards That Accompany Low-Carbohydrate Diets



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

- **High Blood Cholesterol**
  Low-carbohydrate diets can raise blood cholesterol because in these diets, fruits, vegetables, breads, and cereals are replaced by meat and dairy products, which are rich in fat and protein. High fat and protein intakes, especially from meat and dairy products, raise LDL and total cholesterol.



- **Metabolic Abnormalities**
  When carbohydrate intake is low, ketones are produced from fat to replace carbohydrates as a source of energy for the brain. Since ketones are acids, high levels can make the blood acidic, altering respiration and other metabolic processes that are sensitive to an increase or decrease in acidity.

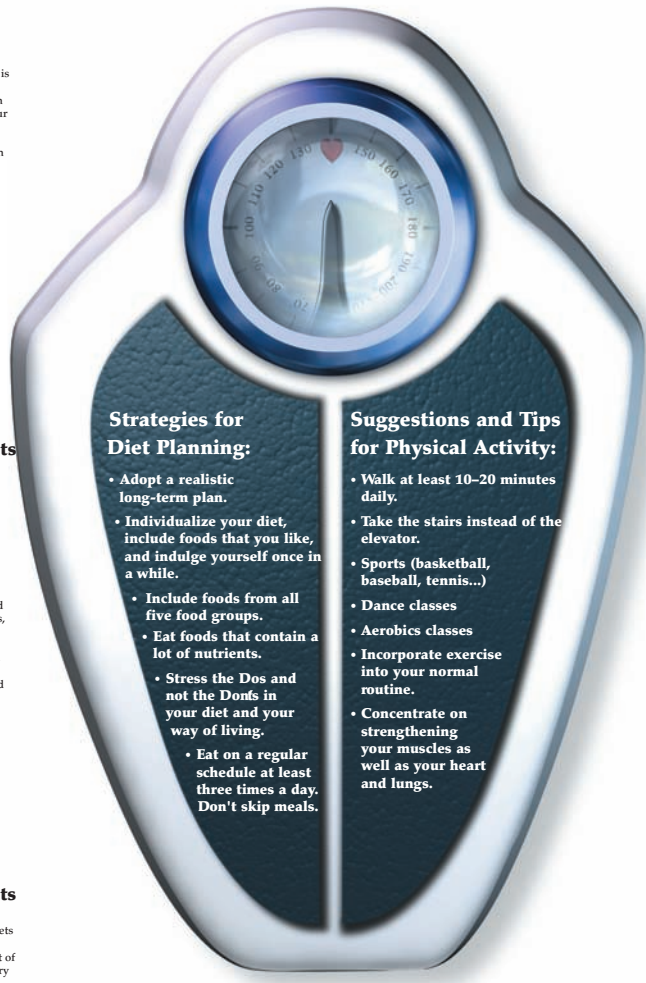
The Risk in Low-Carbohydrate Diets

Low-carbohydrate diets, especially if undertaken without medical supervision, can be dangerous. Low-carbohydrate diets are designed to cause rapid weight loss by promoting an undesirably high concentration of ketone bodies (a byproduct of fat metabolism). The sales pitch is that you'll never feel hungry and that you'll lose weight faster than you would on any "ordinary diet". Both claims are true but the low-carb diets are true but misleading. Fast weight loss means loss of water and lean tissue, which are rapidly regained when people begin eating their usual diets again. The amount of body fat lost will be the same as with a conventional low- calorie diet. Fat loss is always equal to the difference between enegy consumed in food and energy expended in activity.

Overweight Problems

As the amount of body fat increases, especially around the abdomen, so does the risk of:

- Respiratory disease
- Obstructive sleep apnea
- Complications during surgery
- Gallbladder disease
- Stroke
- Non–insulin-dependent (type 2) diabetes
- Some forms of cancer, especially breast and colon
- Coronary heart disease
- Hypertension



Strategies for Diet Planning:

- Adopt a realistic long-term plan.
- Individualize your diet, include foods that you like, and indulge yourself once in a while.
- Include foods from all five food groups.
- Eat foods that contain a lot of nutrients.
- Stress the Dos and not the Donts in your diet and your way of living.
- Eat on a regular schedule at least three times a day. Don't skip meals.

Suggestions and Tips for Physical Activity:

- Walk at least 10–20 minutes daily.
- Take the stairs instead of the elevator.
- Sports (basketball, baseball, tennis...)
- Dance classes
- Aerobics classes
- Incorporate exercise into your normal routine.
- Concentrate on strengthening your muscles as well as your heart and lungs.

What Is Your Body Mass Index?

Your body mass index (**BMI**) is your weight in kilograms divided by the square of your height in meters. It is used to indicate whether or not you are overweight or underweight.

How to Calculate Your Body Mass Index:

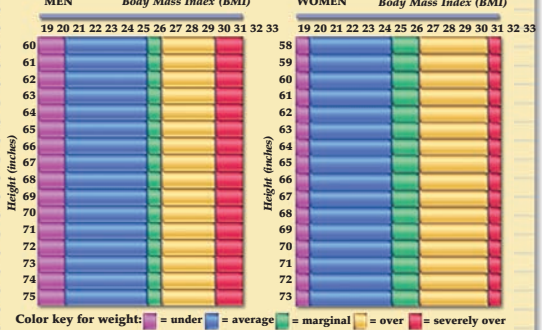
1. Convert your weight in pounds (lb) to kilograms (kg) by dividing your weight in pounds by 2.2 kg.
2. Convert your height in inches (in) to meters (m) by multiplying your height in inches by .0254 m.
3. Take your height in meters and square it by multiplying it by itself.
4. Divide your weight in kilograms by your height in meters squared (your calculated height from step 3).

Example: Mark weighs 150 lb and is 5 ft, 10 in tall (70 in).

1. 150 lb ÷ by 2.2 kg = 68.18 kg
2. 70 in x .0254 m = 1.778 m
3. 1.778 m x 1.778 m = 3.161 m^2
4. 68.18 kg ÷ 3.161 m^2 = 21.56

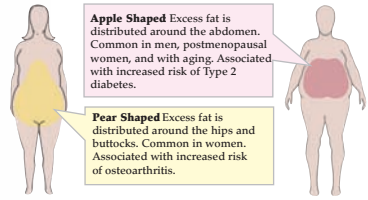
Mark's BMI is 21.56

Acceptable Weight for Height Based on Body Mass Index



Source: Compiled from Body Mass Index Table from Hamilton, Whitney, and Sizer. 1991. *Nutrition concepts and controversies*. New York. West. and Body Mass Index from 1998 Centers for Disease Control and Prevention.

Excess Fat Distribution



Apple Shaped Excess fat is distributed around the abdomen. Common in men, postmenopausal women, and with aging. Associated with increased risk of Type 2 diabetes.

Pear Shaped Excess fat is distributed around the hips and buttocks. Common in women. Associated with increased risk of osteoarthritis.

Understanding Calories

Calories are a standard measurement of heat energy. Technically, 1 calorie is 1 kilocalorie, which is the amount of heat required to raise the temperature of 1 kg. of water by 1° C.

A person's energy needs are determined by the amount of lean tissue or muscle and by the level of activity. A small, elderly, sedentary woman may need only about 1200 calories to meet her energy needs each day, whereas a tall, young, physically active man may need as many as 4000 calories daily.

How to Calculate Your Total Daily Energy (Calorie) Needs

1. Convert your weight from pounds (lb) to kilograms (kg) by dividing your weight in pounds by 2.2 lb/kg.
2. Multiply your weight in kilograms by 30 kcal/kg if you are a man and 25 kcal/kg if you are a woman.

Example

1. 150 lb ÷ by 2.2 lb/kg = 68.18 kg
2. 68.18 kg x 30 kcal/kg = 2045 kcal

Result: A 150 lb man needs approximately 2045 kcal (calories) a day to maintain her weight.

Energy Demands of Activities

Activity	Body Weight (lb) 110	125	150	175	200
	CALORIES PER MINUTE				
Aerobics	6.8	7.8	9.3	10.9	12.4
Basketball (vigorous)	10.7	12.1	14.6	17.0	19.4
Bicycling					
13 miles per hour	5.0	5.6	6.8	7.9	9.0
Cross-country skiing					
8 miles per hour	11.4	13.0	15.6	18.2	20.8
Golf (carrying clubs)	5.0	5.6	6.8	7.9	9.0
Rowing (vigorous)	10.7	12.1	14.6	17.0	19.4
Running					
5 miles per hour	6.7	7.6	9.2	10.7	12.2
Soccer	10.7	2.1	14.6	17.0	19.4
Studying	1.2	1.4	1.7	1.9	2.2
Swimming					
20 yards per minute	3.5	4.0	4.8	5.6	6.4
Walking (brisk pace)					
3.5 miles per hour	3.9	4.4	5.2	6.1	7.0

Source: Compiled from Hamilton, Whitney, and Sizer. 1991. *Nutrition concepts and controversies*. New York. West.

Underweight Problems

When body weight decreases to 15–20% below desirable weight (BMI < 18.5), the amount of energy being consumed is not sufficient to support the function of vital organs. Lean tissue is being broken down and utilized for energy to make up the deficit. The results are:

- Low body temperature
- Abnormal electrical activity in the brain
- Altered blood lipids
- Dry skin
- Impaired immune response
- Loss of digestive function
- Abnormal hormone levels
- Malnutrition
- Anemia

SAMPLE NUTRITION CARE PROCESS STEPS

Not Ready for Lifestyle Change

Assessment: Diet, weight, and physical activity histories and psychosocial issues.

Nutrition Diagnoses (PES): Not ready for diet/lifestyle change (NB 1.3) related to excessive oral food/beverage intake as evidenced by increased weight gain in spite of previous counseling sessions and statements such as, "I just don't want to lose weight; I can't see the point."

Overweight (NC-3.3) related to excess energy intake as evidenced by BMI >25, waist circumference >40 inches, and reports of overconsumption of high-fat/calorie-dense food and beverages.

Interventions: Nutrition prescription (ND-1) of 2200 kilocalories per day with approximately 25–30% of calories from fat and less than 10% of intake from saturated fat.

Initial/brief nutrition education (E-1) to address motivation to get to the next stage of change; reinforce instructions on following the diet plan; provide basic nutrition-related information; discuss the importance of physical activity and the benefits of weight loss.

Transtheoretical Model/Stages of Change (C-1.5): to help patient move from the precontemplation stage to the contemplation stage of change.

Collaboration (RC-1.3): MD and RD to collaborate on helping patient choose to lose weight.

Monitoring and Evaluation: Assess changes in motivation in one month. Evaluate for changes in weight, other lab values.

2. The initial goal of weight loss therapy should be to reduce body weight by approximately 10% from baseline. With success, further weight loss can be attempted if indicated through further assessment.
3. Weight loss should be about 1–2 lb/wk for a period of 6 months, with the subsequent strategy based on the amount of weight lost.
4. Promote use of low-calorie diets (LCDs). A diet that is individually planned to help create a deficit of 500–1000 kcal/d should be an integral part, aimed at achieving a weight loss of 1–2 lb/wk.
5. Increased physical activity is recommended since it produces some weight loss, decreases abdominal fat, and increases cardiorespiratory fitness. Physical activity is an integral part of weight maintenance. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week.
6. Behavior therapy is a useful adjunct. Practitioners need to assess the patient's motivation and readiness to implement the plan; they must provide ongoing support for positive outcomes.
7. Weight-loss drugs that are approved by the Food and Drug Administration (FDA) may be used as part of a comprehensive weight-loss program but never without additional lifestyle modifications. Continual assessment of drug therapy for efficacy and safety is necessary.
8. Weight-loss surgery is an option for patients with morbid obesity (BMI ≥40 or ≥35 with comorbid conditions) when less invasive methods of weight loss have failed.
9. Weight-loss maintenance is enhanced by an ongoing program of dietary therapy, physical activity, and behavior therapy. Drug safety and efficacy beyond 1 year of total treatment has not been established. A weight maintenance program should be a priority after the initial 6 months of weight loss therapy; a high frequency of contacts between the patient and the practitioner should be provided over the long term.
10. All smokers, regardless of their weight status, should quit smoking.
11. In seniors, restrictions on overall food intake due to dieting could result in inadequate intake of protein or essential vitamins or minerals. Involuntary weight loss could be indicative of occult disease. Proper nutritional counseling and regular body weight monitoring in older persons is desirable. Weight reduction may improve cardiovascular risk factors in older and younger adults.
12. Tailor approaches to the needs of various patients or cultural/religious preferences.

Other Advice

- Reduce exposure and heighten awareness of the "obesogenic" environment, including calorie abundance, decreasing physical activity, and increasing automation.
- Maintain a normal or slightly higher protein intake to maintain nitrogen balance and to decrease perceived hunger with energy-restricted diets (Nickols-Richardson et al, 2005).
- Have the patient set his or her own goals. Self-monitoring is important for maintaining calorie, fat gram, and physical activity goals.
- Weigh weekly on the same scale with the same clothing at about the same time of day. After reaching a desired weight, daily weighing often helps maintain motivation for continuing effective lifestyle changes.
- Intake of foods with high water content reduces subsequent energy intake (Rolls et al, 2005). The DASH diet works well because it encourages intake of more fruits and vegetables.
- Avoid or correct disordered eating (e.g., abnormal eating, diet cheating, compulsive eating, addictive or manipulative habits, eating disorders, night eating syndrome). Healthy eating patterns, not constant evaluation of body fat, are the desired end behaviors.
- Use nutrient-dense, antimicrobial spices and antioxidant foods to enhance immunity.

FOOD AND NUTRITION

- Plan a diet with moderate carbohydrate (35–50%), moderate fat (25–35%), and protein as 25–35% of energy (Schoeller and Buchholz, 2005). Total energy reduction is the goal.
- A lower glycemic load is helpful for some people. Foods that have a low glycemic index include salads with an oil and vinegar dressing, high-fat granola cereal, and most

fresh fruits and vegetables (Pittas et al, 2005). **Glycemic load = glycemic index × available carbohydrate amount.**

- Schedule 6–8 small meals at frequent intervals to prevent cheating and overeating.
- Breakfast should be emphasized (Song et al, 2005). Cereal consumption may play a role in helping to maintain a healthful BMI (Barton et al, 2005) and a high-fiber choice may curb appetite at lunch.
- Fiber-rich foods take longer to chew, are low in calories, and increase satiety. Encourage the consumption of 25–35 grams of fiber per day. For example, eating 4–6 walnuts before meals may curb appetite and intake. Oats and barley with their soluble fiber, beta-glucan, may also be particularly useful grains.
- The American Dietetic Association supports a "total diet approach," where the overall food pattern is more important than one food or meal (American Dietetic Association, 2007). If food is consumed in moderation with appropriate portion size and regular activity, a positive approach to food makes the client feel less anxious and guilty.
- Diet should provide adequate fluid intake to excrete metabolic wastes; use 30 cc/kg of body weight for an estimate.
- Decrease the overall salt intake if fluid retention is a concern. Use other spices and herbs.
- NHLBI guidelines recommend 35% kilocalories from fat, more from monounsaturated sources and less from saturated fats. Fat substitutes, when used in moderation, can be safely used to lower total energy intake. With elevated triglycerides, cut down on sugars and alcoholic beverages.
- For a modified fast, a meal replacement product for 1–2 meals per day can get the patient started. This method is not recommended for the long term unless sufficient fiber is also available.
- For obese patient in hospitals or critical care, estimating needs may be inaccurate. Indirect calorimetry is the gold standard. Most medical intensive care unit (ICU) patients require only 25 kcal/kg, with greater energy requirements needed in burn or trauma units.

Common Drugs Used and Potential Side Effects

- Some medications cause weight gain, particularly those affecting the neurological system. See Table 10-11.
- Phenylpropanolamine (PPA) produces dose-related, life-threatening cardiovascular and central nervous toxicity from adrenergic overstimulation. Because of these effects and the risk of hemorrhagic stroke, the FDA stopped the use of PPA in cold medicines and prescription diet aids.
- Pharmacotherapy alone is not sufficient for permanent weight loss. Behavior modification must be taught for long-term success. See Table 10-12 for approved weight management medications. Many products on the market are either not safe or not efficacious.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing it with a physician.
- Because supplements are often taken for stimulating weight loss, dietitians should check for information on efficacy and safety (Dwyer et al, 2005). Health providers must also disclose a real or perceived conflict of interest if they promote or sell supplements to their clients (American Dietetic Association, 2009).
- Bitter orange, chitosan, conjugated linolenic acid (CLA, calcium, and hydroxycitric acid have not shown efficacy (Dwyer et al, 2005).
- Chromium picolinate may improve glucose and lipid metabolism. There is little evidence for benefit in weight loss, but there are few adverse effects (Dwyer et al, 2005).
- Ephedra (ma huang) contains ephedrine and works as an anorexiant. The FDA removed it from the market because it elevates blood pressure and can cause significant problems.
- Garcinia is not effective; it has cytotoxic effects and is toxic in animals.
- Green tea contains polyphenols and catechins, which help to promote weight loss (Nagao et al, 2005).
- Melatonin may improve glucose control by restoring the vascular action of insulin; more studies are needed.
- Modulation of gut microbiota using probiotics or prebiotics will be possible in the future for obesity management (Tsai and Coyle, 2009). Antimicrobial spices such as oregano, thyme, cinnamon, bay leaf, palmarosa, clove bud, lemongrass, and allspice may be safely used.
- Stanols (Take Control) and sterols (Benecol) do not affect weight; they are safe when used as directed to lower elevated cholesterol levels.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- A multidimensional program is best for weight management; it should include the formulation of reasonable goals, the prevention of unnecessary weight loss or gain, weight loss when necessary, prevention of relapse, and the acceptance of physique for "health at every size."
- Individualize according to psychosocial, behavioral, and biological factors. Obesity is a chronic condition that requires chronic care with varying levels of intensity (Nonas and Foster, 2009). Emphasize a balance of foods rather than any one food or meal (American Dietetic Association, 2007).
- Identify the mind–body connection. Teach the patient about physical hunger and how to identify true hunger from emotional "hunger." Practical advice and hunger rating scales are available.
- Low-energy diets tend to be higher in nutritional value if carefully planned using water-rich fruits, vegetables, cooked grains, and soups (Rolls et al, 2005). Noncalorie sweeteners and chewing sugarless gum are useful; chewing gum may help fight cravings for sweets and suppress the appetite.
- Develop reduced-calorie eating plans that meet personal food preferences and provide satisfying food portions (Rolls et al, 2005).
- Instruct the patient on how to plan menus and use recipes. Recipe modifications are useful for replacing fat in recipes (e.g., use applesauce or pear puree in muffins or sponge cake; prune or black bean puree in brownies or spice cake or chocolate cake; or white bean puree in cookies).

TABLE 10-11 Medications That Cause Weight Gain

Medications that affect the central nervous system can cause clinically relevant resting metabolic rate (RMR) effects (from 262–680 kcal/d) in all age groups (Dickerson and Roth-Yousey, 2005a). Sedation or analgesia may produce temporary reductions in RMR (Dickerson and Roth-Yousey, 2005a). Medications that may cause weight gain are listed here.

Medication	Description	Alternatives to Consider
Antianxiety Agents: benzodiazepine (alprazolam [Xanax], chlordiazepoxide [Librium])	Psychotropic drugs can cause weight gain.	
Antidepressants: Selective Serotonin Reuptake Inhibitors (SSRIs)—fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil), fluvoxamine (Luvox), Celexa	SSRIs cause weight loss at first and then weight gain within 6 months. Paxil causes the most weight change. Prozac is often used for patients with bulimia nervosa; it may cause hyperglycemia.	buproprion (Wellbutrin, Zyban)
Antidepressants: Tricyclics (TCAs)—amitriptyline (Elavil, Vanatrip), doxepin (Sinequan), imipramine (Tofranil), nefazodone (Serzone), nortriptyline (Aventyl; Pamelor), trimipramine (Surmontil), mirtazapine (Remeron)	General gains of 0.4–4.12 kg/month; minority of patients gain 15–20 kilograms in 2–6 months from slowed metabolism and increased carbohydrate cravings.	buproprion (Wellbutrin, Zyban)
Antidepressants: Monoamine Oxidase Inhibitors (MAOIs)—isocarboxazid [Marplan], phenelzine [Nardil], tranylcypromine [Parnate]	MAOIs cause more weight gain. Less profound than tricyclics; some gain with phenylzine.	MAOI selective reversible drugs (RIMAs), such as moclobemide (Aurorix, *Manerix*) or toloxatone (*Humoryl*) are not as weight-enhancing as other antidepressants.
Mood Stabilizers: lithium (Eskalith, Lithobid); Moban, Clozaril, Serlect, and Zeldox	Mood stabilizers made with lithium can cause weight gain in 11% to 65% of treated patients; up to 10 kilograms or more in 6 to 10 years.	
Antipsychotics: haloperidol [Haldol], perphenazine [Trilafon], thiothixene [Navane], thioridazine HCl (Mellaril], olanzapine [Zyprexa], risperidone [Risperdal]	Antipsychotics often cause weight gain.	ziprasidone (Geodon); quetiapine (Seroquel)
Anticonvulsants: valproic acid (Depakote/Depakene), gabapentin (Neurontin), carbamazepine (Tegretol)	These medications can increase appetite and insulin levels, and variable gains of up to 15 to 20 kilograms.	topiramate (Topamax), lamotrigine (Lamictal), zonisamide (Zonegran)
Antihistamines: diphenhydramine (Benadryl, Nytol)	Used for allergy management. If used to induce sleep, may mask sleep apnea.	Decongestants or inhalers
Cardiovascular Agents: beta-adrenergic blockers—Propranolol (Inderal), atenolol (Tenormin,) carvedilol, bisoprolol, metoprolol (Lopressor) alpha-adrenergic blockers—prazosin (Minipress), doxazosin (Cardura), terazosin (Hytrin)	These medications can reduce RMR by 4–12% over time (Dickerson and Roth-Yousey, 2005a).	ACE inhibitors such as enalapril (Vasotec) Angiotensin II Inhibitors such as candesartan (Atacand) Ca^{++} channel blockers such as amlodipine (Norvasc), verapamil (Calan, Isoptin), diltiazem (Cardizem)
Chemotherapy	Chemotherapy can decrease metabolic rate by 6–11% in patients with leukemia, breast cancer, and some solid tumors (Dickerson and Roth-Yousey, 2005b).	Treatment is short term.
Diabetes Agents: insulin (Humalog)	Weight gain of up to 8 kg with intensive treatment.	metformin (Glucophage; glucophage XR)
Diabetes Agents: thiazolidinediones	rosiglitazone (Avandia) pioglitazone Actos	acarbose (Precose) miglitol (Glyset)
Diabetes Agents: sulfonylureas	Glipizide (Glucotrol) may reduce RMR by 3.5% (Dickerson and Roth-Yousey, 2005b). Weight gain ≤5 kilograms over 3–12 months.	metformin (Glucophage; Glucophage XR); acarbose (Precose); miglitol (Glyset)
Steroid Hormones: corticosteroids	Hormones can cause weight gain if taken over a long time. cortisone, prednisolone, human growth hormone, somatotropin [Serostim]	NSAIDs if appropriate
Steroid Hormones: progestational agents; androgens	medroxyprogesterone acetate [Provera, Depo-Provera], oxymetholone [Anadrol-50], testosterone [Androderm, Testoderm])	—

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DIABETES TREATMENTS (Up to 8 kilograms gained in an intensive 3-month treatment course)

TABLE 10-12 Medications Used for Weight Reduction in the United States

Diethylpropion (Tenuate)	Appetite suppressant. Dry mouth and GI upset can occur; short-term use is recommended. Average weight 4–6%, drug alone.
Orlistat (Xenical)	Decreases pancreatic lipase and decreases fat absorption. Soft stools and anal leakage may occur, particularly if a diet high in fat (>90 g) is consumed. Orlistat helps minimize weight regain after weight loss and appears to be well-tolerated for the long term. Fat-soluble vitamins may be required during chronic therapy because absorption may be decreased. Patients on orlistat should have a BMI over 27 with risk factors or a BMI over 30 without risk factors; the drug is not for everyone. Weight loss of 8% with behavioral approaches. Alli is an over-the-counter version, which contains a lower dose of orlistat; weight loss is slower.
Phentermine (Adipex, Ionamin)	Appetite suppressant; may cause excitability, gastrointestinal (GI) distress, and insomnia, as well as dry mouth. Average weight 4–6%, drug alone.
Sibutramine (Meridia)	Increases energy expenditure and satiety; it acts via the central nervous system (CNS) as a serotonergic and noradrenergic reuptake inhibitor. It can be used long term. Elevates plasma epinephrine, plasma glucose, and blood pressure. Weight loss of about 16% with behavioral approaches.

Adapted from: Nonas CA, Foster GD. *Managing obesity: a clinical guide*. 2nd ed. Chicago: American Dietetic Association, 2009.

- Steps to normalize eating include awareness training, changing thoughts and beliefs about food, handling issues of deprivation and guilt, and refocusing on areas other than food and weight. Self-efficacy is particularly important (Wamsteker et al, 2005). Correcting unrealistic weight goals is also an issue; it is better to correct them before treatment begins (Wamsteker et al, 2009).
- Provide guidance on how to eat at parties, events, and restaurants. Low-calorie snacking and the selection of low calorie food preparation methods are needed. Avoid buffets when possible.
- Teach portion control. Sometimes measuring and weighing foods can be useful. Table 10-13 provides a handy portion adjustment guide using everyday objects.
- Behavior therapy may be helpful in self-monitoring (food diaries, weights, activity). Teach stimulus control of cues, family intervention, slowing down while eating, and monitoring of intake while at parties and during work breaks.
- To delay automatic eating, drink a glass of water and wait 20 minutes. If the sensation persists, it is probably hunger. Make meals last 20 minutes or longer. Eat slowly; chew well.
- The best diet is "don't buy it" to reduce temptations later.
- The use of meal replacements for one meal a day can be beneficial for improving cholesterol levels, plasma glucose, and diastolic blood pressure.
- Avoid an energy level that is too low, which causes hypophagia and could lead to sudden death syndrome. Low energy intake (<1200 kilocalories for women or <1500 kilocalories for men) requires the use of a multivitamin supplement.
- Physical activity is integral to weight loss maintenance; see Table 10-14. Encourage moderate levels of physical activity for 30–60 minutes daily whenever possible. It is reasonable to encourage the expenditure of 1000 kcal/wk in some type of physical activity. Resistance training increases muscle mass; aerobic exercise should be directed at 70% of maximal oxygen consumption.
- Cultural emphasis on thinness may lead to unhealthy weight loss efforts. Instead, focus on enhancement of self-esteem.
- The real challenge involves keeping weight off after it has been lost. Teach the patient to splurge by plan and not by impulse. Special considerations are important in specific age groups.
- Pregnant mothers should follow established recommendations for diet, exercise, and weight gain during pregnancy. A weight gain of 15–25 pounds in an overweight or <15 pounds in an obese mother leads to better outcomes (Crane et al, 2009).
- New mothers should breastfeed, and then provide wholesome, nourishing foods with low energy density for their children (Melanson, 2009).
- Between pregnancies, women with a history of GDM who gain more than 10 pounds are at higher risk for C-section and adverse outcomes (Paramsothy et al, 2009).
- Parents of young children must help them to develop good eating habits early in life.
- In puberty, it is common for young women to begin gaining weight. Women should not assume that premenstrual fluid weight gain is permanent; this may vary from 2–5 pounds per month.
- Encourage adequate sleep; deprivation increases a hunger hormone that can trigger overeating. Table 10-15 discusses sleep apnea and Pickwickian syndrome; a sleep study may be needed.
- Smoking cessation can lead to weight gain; see Table 10-16.
- Multiple and relentless forms of marketing, poor foods promoted in schools, and a variety of other conditions undermine personal resources, individual responsibility, and parental authority for preventing obesity among children and teens (Brownell et al, 2009). The current environment tends to promote overeating and minimal activity, yet there are social stigmas against being overweight.
- Obesity is increasing across all socioeconomic groups and educational levels (Cohen, 2008). Overall, fat intake tends to be higher in poorer neighborhoods (Keita et al, 2009). Children who live with overweight or obese individuals are often unaware of their desirable body weight range.
- Reduced activity and metabolism are factors that should be addressed to manage weight changes among older adults.
- Permanent lifestyle changes are better than popular diets or trends. Avoid bizarre fad dieting, skipping meals, or

TABLE 10-13 Portion Adjustments Using Everyday Objects

Bread, Cereal, Rice, Pasta

1 cup potatoes, rice, or pasta = 1 tennis ball or 1 ice cream scoop or a fist

1 pancake = 1 compact disc (CD)

1/2 cup cooked rice = a cupcake wrapper full

1 piece cornbread = bar of soap

1 slice bread = audiocassette tape

2 oz of pretzels = 2 handfuls

Vegetables

1 cup green salad = 1 baseball or a fist

1 baked potato = a fist

1 cup of vegetables = a fist

1/2 cup tomato juice = small Styrofoam cup

1/2 cup broccoli = 1 scoop ice cream or one light bulb

1/2 cup serving = 6 asparagus spears, 7–8 baby carrots, or 1 ear of corn on the cob

Fruit

1/2 cup grapes (15) = a light bulb

1/2 cup fresh fruit = 7 cotton balls

1 medium size fruit = a tennis ball or a fist

1 cup cut-up fruit = a fist

1/2 cup raisins = 1 large egg

Milk, Yogurt, Cheese

1½ oz cheese = one 9-volt battery or 3 dominos

1 oz cheese = pair of dice or your thumb

1 cup ice cream = 1 large scoop the size of a baseball

Meat, Poultry, Fish, Dry Beans, Eggs, Nuts

2 tbsp peanut butter = ping pong ball

1 tsp peanut butter = fingertip

1 tbsp peanut butter = thumb tip

3 oz cooked meat, poultry, fish = your palm; a deck of cards

3 oz grilled or baked fish = a checkbook

3 oz cooked chicken = one chicken leg, thigh, or breast

1 oz of nuts = 1 handful

1 oz of cheese = your index finger

Fats, Oils

1 tsp butter or margarine = size of a stamp, the thickness of your finger or thumb tip

2 tbsp salad dressing = 1 ping pong ball

Adapted from: NIDDK Web site. Accessed November 7, 2009 at http://win.niddk.nih.gov/publications/just_enough.htm.

TABLE 10-14 Physical Activity Equivalents

According to the National Weight Control Registry, people who have lost weight and kept it off for 2 years or longer, limit their intake to about **1800 kilocalories daily** and **walk about 4 miles a day.** MET is a practical means of expressing the intensity and energy expenditure of physical activities.

Physical Activity	MET
Light Intensity Activities	<3
Sleeping	0.9
watching television	1.0
writing, desk work, typing	1.8
walking, less than 2.0 mph (3.2 km/h), level ground, strolling, very slow	2.0
Moderate Intensity Activities	3 to 6
bicycling, stationary, 50 watts, very light effort	3.0
sexual activity (position dependent)	3.3
calisthenics, home exercise, light or moderate effort, general	3.5
bicycling, <10 mph (16 km/h), leisure, to work or for pleasure	4.0
bicycling, stationary, 100 watts, light effort	5.5
Vigorous Intensity Activities	>6
jogging, general	7.0
calisthenics (e.g. pushups, sit-ups, pull-ups, jumping jacks), heavy, vigorous effort	8.0
running jogging, in place	8.0

Other beneficial activities that burn kilocalories include the following:

Warm Weather	Calories/hour	Cold Weather
Jogging 6 mph	450	Jumping rope
Hiking on steep hills	400	Indoor rappelling
Aerobics (low impact)	400	Snow shoveling, light
Rowing	400	Rowing machine
Swimming	400	Skiing cross-country
Tennis, singles	390	Racquetball
Cycling 10 mph	300	Stationary bike 10 mph
Golf with walking	300	Splitting logs
Gardening	280	Window cleaning
Mowing lawn	275	Mopping floors
Tennis, doubles	235	Indoor basketball
Badminton	250	Indoor volleyball
Walking 3 mph	**250**	**Mall walking**

Other good resources:
CDC: http://www.cdc.gov/physicalactivity/everyone/measuring/index.html.
Harvard School of Public Health: http://www.hsph.harvard.edu/nutritionsource/staying-active/.
Physical Activity Guide for Americans: http://www.health.gov/paguidelines/guidelines/summary.aspx.

TABLE 10-15 Weight Management for Sleep Apnea and Pickwickian Syndrome

Obstructive sleep apnea (OSA)	Short-duration (<1 minute), repetitive episodes of impaired breathing during sleep. It mostly occurs in obese individuals, which contributes to pharyngeal obstruction. OSA acutely impacts the cardiovascular system and can increase morbidity or mortality. Morbid obesity can be associated with excessive daytime sleepiness even in the absence of sleep apnea. See Section 5 for more details.
Pickwickian syndrome	The individual is obese and hypersomnolent with cor pulmonale, polycythemia, nocturnal enuresis, and personality changes. Even mild obesity can affect lung function, particularly in men. Weight loss is desirable.

emphasis on any one nutrient. A "no fad diet" approach encourages healthy eating patterns. Table 10-17 describes some weight loss programs, several popular diets, and available Web sites.

- Large permanent changes are required to keep weight off. Promote small changes in diet and physical activity to initially prevent further weight gain (Hill, 2009). America On the Move (AOM) is a national initiative that promotes an increase in walking by 2000 steps/d and reductions in energy intake by about 100 kcal/d; people seem to be able to make positive changes in response to these messages (Stroebele et al, 2009). Greater self-reported physical activity was the strongest correlate of weight loss in the Look-Ahead Trial, followed by treatment attendance and consumption of meal replacements (Wadden et al, 2009).
- Discourage weight cycling. Cyclers tends to have a larger body fat percentage, lower metabolic rates, and higher BMI than non-cyclers (Strychar et al, 2009).
- Group counseling or corporate wellness programs may be effective.
- Electronic feedback, one-on-one advice from a nutrition counselor, phone or mail follow-ups can also help to maintain positive momentum in weight loss or maintenance.
- Given that people have a limited ability to shape the food environment individually and have no ability to control automatic responses to unconscious food-related cues, society as a whole may need to regulate the environment through the number and types of food-related cues, portion sizes, food availability, and food advertising (Cohen, 2008).
- Overweight adults diagnosed with type 2 diabetes experience significant improvement in health-related quality of life (HRQOL) by enrolling in a weight management program that yields significant weight loss, improved physical fitness, and reduced physical symptoms (Williamson et al, 2009).

Patient Education—Food Safety

There are no specific food handling techniques that are unique to overweight or obesity.

TABLE 10-16 Smoking Cessation and Weight Gain

Nicotine addiction is a chronic, relapsing condition.

Smoking cessation lowers cardiovascular and cancer risks even when compensating for possible weight gain; cessation lengthens life by several years and is worth the effort.

Weight gain is common after cessation because of increased caloric consumption, changes in activity levels, and a decrease in the basal metabolic rate. Past smokers are more likely to be obese than current smokers.

Contemplation of and experimentation with smoking is often related to weight concerns among boys and girls. Women fear weight gain more than men.

Women who quit smoking typically gain 6–12 pounds in the first year after quitting.

Interventions that address weight control are recommended. Moderate to heavy smokers who attempt to quit may need to reduce intake by 100–200 kcal/d just to maintain weight.

Behavioral weight control counseling helps to slow the rate of weight gain. Fruit often helps relieve the craving for sweets.

Exercise often helps relieve the desire to smoke. Walking is a great stress reliever.

Sustained-release bupropion (bupropion SR) was the first non-nicotine pharmacological treatment approved for smoking cessation. Others are now available.

Teach good principles of weight management:

- Weight gain is a problem requiring lifelong, multidisciplinary management.
- Weight is regulated by a complex set of biologic/environmental factors, not by a lack of willpower.
- Modest weight loss has benefits.
- Work to alter fundamental thoughts and assumptions versus unrealistic expectations.
- Emphasize slow, steady loss, followed by maintenance.
- Focus on long-term outcomes/sustained changes.

For more tips, see http://www.smokefree.gov/pubs/FFree3.pdf.

TABLE 10-17 Diet Program Comparisons

Programs	Description	Web Address	Pro	Con
Diet Center	Personal counseling	http://www.dietcenter.com	Consider "Exclusively You" option	
eDiets.com	Online dieting	http://www.ediets.com	Bargain	Must be self-motivated
Jenny Craig	Personal counseling; at home	http://www.jennycraig.com	Well-structured	Expensive
LA Weight Loss	Personal counseling	http://www.laweightloss.com	Good exercise centers	Costly foods
NutriSystem	Online dieting	http://www.nutrisystem.com	Healthy choices; good structure	
Overeaters Anon	Group support	http://www.oa.org	Physical, emotional, spiritual support	
Registered Dietitian	Expert personal counseling	http://www.eatright.org	Most flexible; personalized concerns	
Take Off Pounds Sensibly	Group or online support	http://www.tops.org	Inexpensive	Not personalized
Weight Watchers	Group or online support	http://www.weightwatchers.com	Comprehensive and sound principles	

Popular diets such as Weight Watchers, Atkins, the Zone, and Ornish achieve weight loss and reductions in LDL cholesterol, CRP, and insulin as long as calories are reduced and adherence continues for up to a year (Dansinger et al, 2005). In another study of the popular diets, Gardner et al (2007) found that premenopausal overweight and obese women assigned to follow the low CHO Atkins diet lost more weight at 12 months than women assigned to follow the Zone diet and experienced comparable or more favorable metabolic effects than those assigned to the Zone, Ornish, or LEARN diets. In general, any diet or program that achieves macronutrient reduction should yield acceptable results.

Type of Diet	Total Calories	Fat grams (% calories)	Carb grams (% calories)	Protein grams (% calories)	Nutrition Adequacy
Typical American diet	2200	85 (35%)	275 (50%)	82.5 (15%)	Varies by food choice and nutrient density.
High fat Low-carbohydrate diet • Dr. Atkins Diet • Zone Diet • Sugar Busters • Protein Power	1414	96 (60%) Fat level range: 35–65%	35 (10%)	105 (30%)	Low in several nutrients: Vitamins A, B_6, D, E, thiamin, folate, calcium, magnesium, iron, zinc, potassium, and dietary fiber. This type of diet also contains excess amounts of total fat, saturated fat, and dietary cholesterol. Nutritional supplementation is highly recommended.
Moderate fat diet • USDA Food Guide Pyramid • DASH Diet • American Diabetes Association • Weight Watchers • Jenny Craig	1450	40 (25%) Fat level range: 21–34%	218 (60%)	54 (15%)	Usually a nutritionally balanced eating plan assuming the dieter eats a variety of foods from all food categories. Limiting any food categories can lead to deficiencies in certain nutrients particularly calcium, iron, and zinc. Weight watchers scores are the highest here for long-term adherence.
Low- and very low-fat diet • Volumetrics • Dean Ornish's Eat More, Weigh Less • New Pritikin Program	1450	20 (13%) Fat level varies: 10–20%	235–271 (70%)	54–72 (17%)	Deficient in zinc and vitamin B12 due to infrequent meat consumption. This type of diet can be inadequate in vitamin E—a nutrient found in oils, nuts, and other foods rich in fat. Volumetrics rates the highest in this group for helping people lose weight effectively.

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  http://www.eatright.org/ada/files/wmnp.pdf
- California Adolescent Nutrition and Fitness Program
  http://www.canfit.org/
- Canadian Physical Activity Guide
  http://www.csep.ca/
- CDC, Genetics of Obesity
  http://www.cdc.gov/Features/Obesity/
- CDC, Healthy Weight Web site
  http://www.cdc.gov/healthyweight/
- CDC, Report of the Surgeon General on Physical Fitness
  http://www.cdc.gov/nccdphp/sgr/summ.htm
- Center for Weight and Eating Disorders
  http://www.med.upenn.edu/weight/foster.shtml
- DASH Diet in Weight Management
  http://www.nhlbi.nih.gov/hbp/prevent/h_weight/h_weight.htm
- Dietary Guidelines, Calories
  http://www.health.gov/dietaryguidelines/dga2005/healthieryou/html/chapter5.html
- Federal Trade Commission
  http://www.ftc.gov/bcp/edu/pubs/consumer/health/hea03.shtm
- Food Diary Format
  http://www.cdc.gov/healthyweight/pdf/Food_Diary_CDC.pdf
- Genome Studies of Obesity
  http://www.ncbi.nlm.nih.gov/disease/Obesity.html
- National Association to Advance Fat Acceptance (NAAFA)
  http://www.naafa.org/
- National Weight Control Registry
  http://www.nwcr.ws/
- NHLBI Obesity Education Initiative and Evidence Guidelines
  http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.htm
- Physical Activity Format
  http://www.cdc.gov/healthyweight/pdf/Physical_Activity_Diary_CDC.pdf
- Physical Activity Guidelines
  http://www.cdc.gov/physicalactivity/everyone/guidelines/index.html
- Shape Up America
  http://www.shapeup.org/
- Surgeon General
  http://www.surgeongeneral.gov/topics/obesity/
- Weight Watchers International
  http://www.weightwatchers.com/

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UNDERWEIGHT AND MALNUTRITION

UNDERWEIGHT AND UNINTENTIONAL WEIGHT LOSS

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

Underweight Problems

When body weight decreases to 15–20% below desirable weight (BMI < 18.5), the amount of energy being consumed is not sufficient to support the function of vital organs. Lean tissue is being broken down and utilized for energy to make up the deficit. The results are:

- Low body temperature
- Abnormal electrical activity in the brain
- Altered blood lipids
- Dry skin
- Impaired immune response
- Loss of digestive function
- Abnormal hormone levels
- Malnutrition
- Anemia

DEFINITIONS AND BACKGROUND

Underweight is defined as having a BMI below 18.5; approximately 8–9% of the population is underweight. Weight gain may be difficult for some healthy individuals because of a genetic tendency toward leanness, excessive activity, or routine eating patterns. Being underweight may or may not be associated with pathology. There are serious health risks associated with very low weight and with efforts to maintain an unrealistically lean body mass. Identification and treatment of disordered eating can be important for improving the health status of underweight individuals at any age. See Section 4 for eating disorder guidelines.

Body storage of glycogen is approximately 1100 kilocalories or about a 12- to 16-hour supply. Body storage of protein equals approximately 40,000 kilocalories of muscle tissue; the loss of 30–50% of lean body mass is incompatible with survival. Body fat is the remainder of calories in fuel storage; it varies depending on the weight of the individual compared with BMI tables for height and sex and by level of physical fitness.

Low BMI is a significant predictor of mortality among young as well as older hospitalized patients (Flegal et al, 2005; Sergi et al, 2005). A BMI value of 20 kg/m^2 seems to be a reliable threshold for defining underweight in older adults at high risk for short-term mortality (Sergi et al, 2005). The Women's Health Initiative Observational Study evaluated 40,657 women aged 65–79 years at baseline and measured frailty including muscle weakness, impaired walking, exhaustion, low physical activity, and unintended weight loss; hip fractures, activities of daily living disability, hospitalizations, and deaths were also tracked (Fugate Woods et al, 2005).

Underweight is often correlated with frailty and poor outcomes. Underweight is associated with significantly increased mortality from non-cancer, non-CVD causes (Flegal et al, 2007). Indeed, a low BMI tends to have the most risk for mortality (Heather et al, 2009).

The Ancel Keys studies (Keys et al, 1950) demonstrated that starvation results in food preoccupation, unusual eating habits, increased use of caffeine and tea, binge eating, depression, anxiety, social withdrawal, poor judgment, apathy, egocentrism, edema, sleep disturbances, hypothermia, gastrointestinal disturbances, and lowered basal metabolism. Death related to starvation is often from decreased respiratory muscle function and terminal pneumonia. In addition, patients who have chronic obstructive pulmonary disease (COPD) are more likely to have additional exacerbations when weight loss and poor intake occur (Hallin et al, 2006).

The most common cause of a person being underweight is inadequate access to food. While this problem is most significant in sub-Saharan Africa and south Asia, many Americans go to bed hungry every night. Financial resources, politics, geography, and economics play a role; food inadequacy may also relate to high prices and limited healthy choices at the local grocery store.

Unintentional weight loss with debility, cachexia, and loss of lean body mass suggests some undesirable condition or pathology, particularly among chronically ill or institutionalized individuals. Malnutrition in older adults is generally characterized by faulty or inadequate nutritional status, insufficient dietary intake, poor appetite, muscle wasting, and weight loss. Blunted responsiveness to neuropeptide Y (NPY)—a feeding stimulant—often occurs concurrently with age-related anorexia and hypophagia.

TABLE 10-18 Strengthening Tips

In healthy young people, 30% of body weight is muscle, 20% is adipose tissue, and 10% is bone; muscle accounts for 50% of lean body mass and 50% of total body nitrogen. By age 75, about half the body's muscle mass has disappeared: 15% of body weight is muscle, 40% is adipose tissue, and 8% is bone (Merck, 2009). Hospitalized or bedridden elderly people require early physical therapy and individualized exercise regimens (Merck, 2009). With restricted mobility, loss of muscle mass and strength (deconditioning) occurs, mostly in the antigravity muscles–those used to sit up, stand up, and pull up (Merck, 2009).

Up to 1.5%/d of muscle mass can be lost; for 1 day of absolute bed rest, up to 2 wks of reconditioning may be necessary to return to baseline function (Merck, 2009). Patients who can and will comply with a proper exercise program gain muscle protein mass, strength, and endurance and are often more capable of performing activities of daily living (NIA, 2009).

1. Start with a weight that can be lifted 5 times without too much effort.
2. When that is easy, rest a few minutes, and do it again (2 sets).
3. Increase to 3 sets.
4. Lift weight 10 times in each set.
5. Lift weight 15 times in each set.
6. Slowly increase weight and sets.

Sources:
Merck Manual of Geriatrics. Accessed November 7, 2009 at http://www.merck.com/mkgr/mmg/sec7/ch48/ch48d.jsp.
NIA. Exercise: a guide from the National Institute on Aging. Accessed November 7, 2009 at http://www.nia.nih.gov/HealthInformation/Publications/ExerciseGuide/chapter04a.htm.

Nutritional frailty includes sarcopenia with the loss of lean body mass, which leads to a failure to thrive and functional disability. Cytokines contribute to lipolysis, anorexia, muscle protein breakdown, and nitrogen loss. Patients with cardiac cachexia or chronic obstructive pulmonary disease (COPD) are often debilitated if they have lost weight rapidly. In older adults, micronutrient deficiency is a common result and should be addressed. Table 10-18 provides tips for helping debilitated persons to strengthen their muscles.

Carefully monitor for unintentional weight loss in adults following their admission into residential health care facilities. Red flags include early satiety, bloating, anorexia, dyspnea, fatigue, constipation, and dental problems.

Protocols must emphasize thorough assessment, interventions, frequent weighing and effective communications between all involved parties. Other populations who are at risk for weight loss and poor intake should be monitored (e.g., persons with chronic renal insufficiency, tuberculosis, HIV-AIDS, and cancer).

The American Dietetic Association Unintended Weight Loss (UWL) in Older Adults Evidence-Based Nutrition Practice Guideline was published in 2009. Qualified dietitians help to identify nutritional risk factors and recommend interventions based on the medical condition, needs, goals and desires of the individual. Studies support an association between increased mortality and underweight (BMI <20 kg/m^2 or current weight compared with usual or desired body weight) and/or unintended weight loss, such as 5% in 30 days (American Dietetic Association, 2009).

In long-term care, usual body weight is often more meaningful than comparison with an ideal body weight chart. Quality of life and resident-directed care plans ("I Care" plans) must be considered when designing interventions. The move from institutional to person-centered dining is a positive step in the right direction. The American Dietetic Association has recommended at least three MNT visits for adults who have had unintentional weight loss; it also has a Long-Term Care Toolkit for use during the nutrition care process with standardized language for that setting.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Underweight may be part of a genetic disorder or inherited naturally.

Clinical/History

- Height
- Weight
- Usual weight
- Recent weight changes
- BMI
- Diet history
- BP
- Intake and output (I & O)
- Dehydration?
- DETERMINE checklist, MNA or other screening tool
- Exercise tolerance
- Parasitic infections?
- Sarcopenia?
- Xerostomia, mucositis?
- Inflammatory bowel disease?
- Cancer or HIV infection?
- Loss of teeth or dental pain?
- Nausea or frequent emesis?
- Amenorrhea, anemia, hair loss?
- Osteoporosis or fractures?
- Stress, medication overuse?
- Cognitive decline or depression?
- Eating dependency?
- Skinfold measurements (over time)

Lab Work

- Hemoglobin and hematocrit (H & H)
- Serum ferritin, if anemia is suspected
- CRP (elevated?)
- T3, T4, TSH
- Albumin (Alb) below 3.5 g/dL
- Transthyretin below 17 g/dL
- Chol (may be low)
- Trig
- Fasting glucose
- Blood urea nitrogen (BUN)
- Creatinine (Creat)
- Nitrogen (N) balance
- Na^+, K^+
- Alkaline phosphatase (Alk phos)
- Ca^{++}, Mg^{++}

SAMPLE NUTRITION CARE PROCESS STEPS

Underweight

Assessment Data: Diet, weight, and physical activity histories. Medical history, frequent infections.

Nutrition Diagnosis (PES): Underweight related to a mismatch of energy intake and expenditure as evidenced by a 6-lb loss in 1 month without change in the usual oral intake.

Intervention: Address appropriate matching of energy intake and physical activity. Weight training may be beneficial. Counsel on food choices and ways to increase intake of energy-dense foods.

Monitoring and Evaluation: Ask patient to return in 2–4 weeks to assess weight. Review diet and activity logs, changes in BMI over time, fewer infections.

INTERVENTION

OBJECTIVES

- Increase body weight gradually if indicated. Encourage weight gain of approximately 1 lb weekly.
- Keep a food diary or log to determine changes that have been made and whether they are successful.
- Provide a 3-meal, 3-snack regimen as a baseline. Encourage increased consumption of calorie and nutrient-dense foods and beverages.
- In the case of recent acute illness or general chronic disease, provide diet as tolerated to improve nutritional status. Progress slowly; it may take several days to stimulate the patient's appetite.
- If confusion is present, dehydration may be a factor; evaluate carefully and rehydrate if appropriate.
- Try anabolic agents, exercise/physical activity, and cytokine inhibition.

FOOD AND NUTRITION

- Calculate patient's goal weight: basal energy requirements plus kilocalories according to activity or stress factors. Each pound of fat requires 3500 kilocalories; therefore, diet should be increased by 500 kcal/d to promote a weight gain of 1 lb/wk.
- Use 15–20% protein, 60–70% CHO diet with frequent feedings, adequate micronutrients, and supplements as needed. Include healthy fats from avocado, nuts, fatty fish, olive, and canola oils.
- Plan meals and snacks according to appetite and preferences; encourage a small snack approximately every 2–3 hours. Patients who have cancer or other chronic illnesses may not want to eat large meals.
- Try serving the high calorie foods first if satiety is a problem; consume beverages between rather than with meals. Smaller, more frequent meals are recommended; avoid rushing mealtimes. Prepare items with fats, oils, extra sweets if feasible. Choose beverages with added calories such as a milkshake with chocolate syrup and peanut butter.
- Offer something to eat or drink every few hours, or keep snacks close at hand. If necessary, set an alarm to remember to eat something.
- With dyspnea, rest before meals and use bronchodilators in advance. Eat slowly and use pursed lip breathing between bites. Keep food and snacks within easy reach.
- For easy fatigue, rest before meals. Serve pre-prepared meals that are readily available. Use oral supplements between meals if intake is poor at mealtime.
- For constipation, a stool softener and adding extra fiber and fluid may be useful.
- For dental problems, change the food textures as needed (grind, chop, mash). Fix the problem with a dental appointment, or repair dentures if needed.
- For swallowing difficulty, work with the Speech and Language Pathologist (SLP) to identify appropriate solid and liquid consistencies.
- Provide enteral feeding if needed and appropriate. In some cases, feeding at night is well tolerated.

Common Drugs Used and Potential Side Effects

- Appetite may be stimulated through use of medications such as megestrol acetate, dronabinol, prednisone, or oxandrolone. Not everyone responds positively with an increased appetite and weight gain. Side effects vary and are most concerning with prednisone.
- Antidepressants may be warranted when a qualified professional has documented depression. Monitor for dry mouth and other side effects specific to ordered medication.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- When a registered dietitian, as part of the healthcare team, provides medical nutrition therapy (MNT) for older adults with unintentional weight loss, improved outcomes are seen for increased energy, protein and nutrient intakes, improved nutritional status, improved quality of life, and weight gain (American Dietetic Association, 2009).
- Help patient make meals in a simple manner, using attractive foods.
- Identify spices, seasonings, and other flavor enhancements that will stimulate the senses.
- The use of high-caloric density foods may be useful in programs where the patient refuses to eat or to take supplements. Adding them by "hiding" calories and extra protein in food may also be feasible (extra dry milk powder in soups, shakes, or mashed potatoes).
- Offer tips on weight gain such as eating a small snack every 2–3 hours. A high-calorie bedtime snack is often beneficial (for example, a milkshake or sandwich). If homemade items are not available, high calorie commercial supplements can be used.

- Dietary restraint and inadequate weight gain in pregnancy is not desirable. There are tools to assess restraint that may identify women at risk so that they can be counseled early in pregnancy (Mumford et al, 2008).
- Malnutrition often presents with loss, loneliness, dependency, and chronic illness, and it impacts morbidity, nutrition quality of life, and morbidity. Coordinate referrals such as home-delivered meals.
- Promote lean body mass development through strength training where appropriate. Increased physical activity appropriate for the clinical condition can help to improve appetite and intake in many cases. Exercise training has been successful for the treatment of wasting associated with sarcopenia, cancer, and some other conditions.
- Coordinate care with other health team members, as shown in this flow chart:

INTERDISCIPLINARY NUTRITION CARE PLAN
Involuntary Weight Loss

Client Name: _____ **#:** _____ **Initiated by:** _____ **Date:** _____

SCREENING
Nutrition Screen indicates Involuntary Weight Loss
Signed: _____ Date: _____

GOALS (Check any/all):

❑ Prevent nutritional decline/adverse events in ____ (goal time).

❑ Encourage improvement as evidenced by: wt gain/increased strength in ____ (goal time).

❑ Correct causes of involuntary weight loss where possible in ____ (goal time).

ASSESS ACUITY *(Check any/all)*
❑ Poor oral intake
❑ Complex diet order
❑ BMI <20 (high risk)
❑ Wt loss: >3 lb/wk or >5%/mo or >10%/6 mo
❑ Nausea/diarrhea/vomiting
❑ Infection (e.g., pneumonia, UTI, URI)
❑ Pressure ulcers, wounds
❑ Poor Strength
Signed: _____ Date: _____

None

MODERATE RISK INTERVENTIONS *(Check any/all)*
❍ **Ways to Improve Nutrition** provided and reviewed
❍ **Food Record** provided and reviewed
❑ RD chart consult
❑ Weight monitoring q:_____
❑ BID/TID supplements
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

KEY
❍ **Recommended intervention for best practice:**
• Patient Education Materials provided and assessed for comprehension

❑ **Optional intervention:**
• Nutrition support initiated (oral/enteral)
• Referrals
• Monitoring

1 or more

HIGH-RISK INTERVENTIONS *(Check any/all)*
❍ **Ways to Improve Nutrition** provided and reviewed
❍ **Food Record** provided and reviewed
Obtain Dr. orders as needed:
❑ Weight monitoring q:_____
❑ RD referral for home visit(s)
❑ BID/TID supplements
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
❑ Further wt loss
❑ Wt gain < goal
❑ New onset infection
❑ Declining strength
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

1 or more

None

OUTCOMES ACHIEVED
Document improved:
❑ Strength
❑ Weight
❑ **Other:**_____
❑ Repeat Nutrition Screen in ____ days
(See notes for documention.)
Goals met
Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
❑ Further wt loss
❑ Wt gain < goal
❑ New onset infection
❑ Declining strength
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

None

OUTCOMES ACHIEVED
Document improved:
❑ Strength
❑ Weight
❑ **Other:**_____
❑ Repeat Nutrition Screen in ____ days
(See notes for documention.)
Goals met
Signed: _____ Date: _____

1 or more

OUTCOMES NOT ACHIEVED
Reassess acuity/evaluate need for EN/PN (refer to Tube Feeding Nutrition Care Plan). Document on Nutrition Variance Tracking form.

Adapted with permission from www.RD411.com, Inc.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Health Line
  http://www.healthline.com/symptomsearch?addterm=Unintentional%20Weight%20Loss
- Unintentional Weight Loss
  http://www.nlm.nih.gov/medlineplus/ency/article/003107.htm
- Wrong Diagnosis
  http://www.wrongdiagnosis.com/u/underweight/symptoms.htm

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UNDERNUTRITION AND PROTEIN-ENERGY MALNUTRITION

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

The undernourished category of malnutrition leads to the loss of body cell mass, which, together with inflammation, diminishes host response and quality of life (Soeters and Schols, 2009). Protein–energy malnutrition (PEM) decreases cardiac output, blood pressure, oxygen consumption, total lymphocyte count (TLC), number of T cells, and glomerular filtration rate (GFR). Undernutrition is associated with increased infection rates, emphysema and pneumonia, GI tract atrophy, intestinal bacterial overgrowth, hepatic mass losses, and anemia. In PEM, anemia is a result of ineffective erythropoiesis (Borelli et al, 2007).

Physical assessment and thorough clinical history are essential in determining the etiology of the PEM and appropriate interventions; see Table 10-19. Malnutrition may be assessed by estimating nutrient balance but, subsequently, to measure body composition (muscle mass), inflammatory activity (plasma albumin and C-reactive protein), and muscle endurance and force (Soeters and Schols, 2009).

Malnutrition is the leading cause of death among children in developing countries. Clinically, in children, PEM has three forms: dry (thin, desiccated), wet (edematous, swollen), or a combination; grading is mild, moderate, or severe. Grade is determined by calculating weight as a percentage of expected weight for length using international standards (normal 90–110%; mild 85–90%; moderate 75–85%; severe <75%). The dry form, **marasmus**, results from near starvation with a deficiency in protein and nonprotein nutrients. The wet form is called **kwashiorkor**—an African word meaning "first child–second child"—because the first child often develops malnutrition after the second child arrives and nutrient-poor foods replace breast milk. The combined form of malnutrition is called **marasmic kwashiorkor**; these children have some edema and more body fat than those with marasmus.

Nutrients and growth factors regulate brain development during fetal and early postnatal life. The rapidly developing brain is more vulnerable to nutrient insufficiency yet also demonstrates its greatest degree of plasticity (Georgleff, 2007). Chronic protein energy malnutrition (stunting) affects the ongoing development of higher cognitive processes during childhood and may also result in long lasting cognitive impairments (Kar et al, 2008). The nutrients that have greater effects on brain development than do others include protein, energy, certain fats, iron, zinc, copper, iodine, selenium, vitamin A, choline, and folate (Georgleff, 2007).

Adult malnutrition syndromes differ from those in underdeveloped countries; an understanding of the systemic inflammatory response should help guide assessment, diagnosis, and treatment (Jensen et al, 2009). In adult patients with acute illnesses, the prevalence of malnutrition is high, particularly related to age and metabolic stress (Martinez Olmos et al, 2005; Pirlich et al, 2006). Of patients admitted to hospitals, 35–55% are malnourished on admission and 25–30% more become malnourished during stay. Problems are common in gastrointestinal (GI) patients, particularly patients with inflammatory bowel disease (IBD); ventilator, radiation, or chemotherapy patients; burn and surgical patients; and patients with renal failure. Even with dialysis, renal patients are at high risk (Kopple, 2005), mostly from cardiovascular disease with markers of a malnutrition–inflammation complex syndrome (Colman et al, 2005).

Tissue catabolism usually begins with lowered plasma proteins, red blood cells, and leukocytes; later, wasting of organs, skeletal muscle, bone, skin, and subcutaneous tissue

TABLE 10-19 Complicating Effects of Chronic Malnutrition on Body Systems

Cardiac and Hematological System—Anemia; altered clotting time; decreased heart size; decreased amount of blood pumped; slow heart rate; decreased blood pressure; heart failure; decreased number of blood cells.

Digestive Tract—Frequent, chronic, or even fatal diarrhea; bacterial translocation in gut; low HCl production in stomach; progressive weight loss; gastrointestinal mucosal or villous atrophy with loss of immune function.

Endocrine System—Decreased body temperature (hypothermia); fluid accumulation in skin from lower subcutaneous fat and decreased albumin levels; vitamin and mineral deficiencies.

Immune System—Depressed cell-mediated immunity; increased infection, particularly gram-negative sepsis; impaired wound healing; more wound infections or disruption; impaired ability to fight infections; delayed response to cancer chemotherapy or radiation therapy.

Muscular System—Decreased activity; delayed physical rehabilitation; decreased muscle size and strength; delayed hospital discharge and ability to perform work.

Nervous System—Irritability, weakness, and apathy even if intellect remains intact.

Pulmonary System—Depressed ventilatory response to hypoxia; decreased lung capacity; slow breathing; pneumonia and eventually respiratory failure.

Quality of Life—Increased and prolonged use of hospitals, critical care units, and expensive drugs; excessive requirements of hospital support.

Renal System—Fluid, electrolyte, and acid–base malfunctioning; increased frequency of urinary tract infections; elevated blood urea nitrogen from muscle and tissue breakdown; decreased glomerular filtration rate.

Reproductive System—Decreased size of ovaries or testes; decreased libido; cessation of menstruation.

Skin and Skeleton—Pale, thin, dry inelastic skin; pressure ulcers; decreased subcutaneous fat; loss of bone density.

will occur. Chronic undernutrition of protein, calories, and micronutrients (particularly zinc) will compromise cytokine response and affect immune cell functioning. Nutrients act as antioxidants and as cofactors in cytokine regulation (Cunningham-Rundles et al, 2005). The central nervous system is the last system to be catabolized, and magnetic resonance imaging (MRI) scans taken of children who have moderate to severe malnutrition show cerebral atrophy (Odabas et al, 2005). Total starvation is fatal in 8–12 weeks.

Certain types of stress and surgery can lead to malnutrition; pancreatic surgery is one specific example (Schnelldorfer and Adams, 2005). Because malnutrition and involuntary weight loss are common problems in seniors, there is a significant increase in infection rate, a decrease in the rate of healing, and an increase in length of stay in older, malnourished burn patients when compared with those who are well nourished (Demling, 2005).

Common causes of malnutrition in the elderly involve decreased appetite, dependency on help for eating, impaired cognition and/or communication, poor positioning, frequent acute illnesses with GI losses, medications that decrease appetite or increase nutrient losses, polypharmacy, decreased thirst response, decreased ability to concentrate urine, intentional fluid restriction because of fear of incontinence or choking if dysphagic, psychosocial factors such as isolation and depression, monotony of diet, and higher nutrient density requirements, along with the demands of age, illness, and disease on the body (Harris and Fraser, 2004).

Poor nutritional state may be associated with an increase in postoperative complication rate; low serum albumin levels can be reflected by a higher rate of infectious complications as well as increased intensive care unit stays (Schnelldorfer and Adams, 2005). The Subjective Global Assessment (SGA) is a tool for nutritional screening that can be used on hospital admission. The SGA score, disease category, presence of malignancy, serum albumin level, percent triceps skinfold thickness, and percent arm muscle circumference are significant predictive parameters for hospital stay in patients with digestive diseases (Wakahara et al, 2007). SGA has also been found to be useful in chronic kidney disease, hemodialysis, and cardiac disorders as well as liver diseases and cancer.

It is important to use designated descriptions or codes for malnutrition in the medical record so that proper attention is given. Table 10-20 describes several types of undernutrition that are common in hospitals. Table 10-21 describes a malnutrition universal screening tool (MUST). The MUST tool has been developed to screen all adults, even if weight and/or height cannot be measured. This tool provides more complete information on malnutrition prevalence; MUST screening predicts clinical outcome in the hospitalized elderly where malnutrition is common in over half of the population (Stratton et al, 2006). Other tools are available in Appendix B.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: While genetics are typically not responsible for malnutrition, some conditions make an individual prone to poor nutriture. Examples include inherited disorders such as cystic fibrosis, inflammatory bowel disease, and celiac disease. Recent studies have shown that vitamin D_3 and calcitriol impact over 1000 genes, and deficiency contributes to rickets, cancers, hypertension, stroke, heart attack, diabetes, bone fractures, multiple sclerosis, and periodontal disease (Edlich et al, 2010). Vitamin D up-regulates cathelicidin—a naturally occurring broad-spectrum antibiotic—and may even play a role in protecting against viral respiratory infections including the common cold and influenza (Cannell and Hollis, 2009).

Clinical/History

Height, arm length, or knee length
Stunting in a child
Weight
BMI
Recent weight; weight changes
Usual weight
Desirable BMI
Diet history—poor appetite
I & O
BP
Edema
Muscle wasting
Sarcopenia?
Cachexia?
TSF
MAMC, MAC

Lab Work

CRP
Alb, transthyretin (may be altered)
Chol, Trig (decreased)
Serum Fe, ferritin
Alk phos (decreased)
Gluc
H & H
Oxygen saturation levels
Total iron-binding capacity (<250 mg/dL)
Urine acetone
T3, T4
Ca^{++}, Mg^{++}
BUN (decreased)
Creat
White blood cell count (decreased)
Na^{+}, K^{+}, Cl^{-}
Serum B_{12}, folate, B_6
Serum D_3 [25(OH)D]*

*Natural vitamin D levels in humans living in a sun-rich environment are between 40 and 70 ng/mL (Cannell and Hollis, 2009).

INTERVENTION

OBJECTIVES

- Provide adequate macronutrients and micronutrients. Work up to 100% of estimated needs over several days.
- Correct weight loss, weakness, infections, and poor wound healing. Improve signs of apathy and irritability.
- Reduce the costs of care. International studies suggest that disease-related malnutrition increases hospital costs by 30–70%; with this large cost of disease-related malnutrition, intervention can result in substantial absolute cost savings (Elia, 2009).
- Avoid hazards of refeeding (hypophosphatemia, low magnesium, and potassium). Fluid administration must be monitored carefully. Prevent sepsis, overfeeding, hyperglycemia, heart failure, or other organ failure by refeeding slowly.
- Allow normal growth of brain and prevent permanent IQ deficits in children.
- Correct malnutrition in patients who have dysphagia as it may contribute to further declines in the capacity for rehabilitation.
- Provide sufficient nutrients for gene expression.
- Prevent complications, which can include dehydration, electrolyte imbalances, infections, vitamin-mineral deficiencies, and other biochemical changes (Table 10-22).

TABLE 10-20 Indicators of Malnutrition

Category	Criteria for Nutrition Diagnosis of Malnutrition
Severe malnutrition	At least one of the following criteria must be met:
Protein-energy malnutrition	– BMI ≤16.0 – Weight loss of ≥26% of UBW within ≤6 months
Moderate malnutrition	At least one of the following criteria must be met:
Protein-energy malnutrition	– BMI 16.0–16.9 – Weight loss of 16–25% of UBW within ≤6 months
Mild malnutrition	At least one of the following criteria must be met:
Protein-energy malnutrition	– BMI 17–18.4 – Weight loss of 5–15% of UBW within ≤6 months – Weight loss of ≥5% of UBW within ≤1 month
Malnutrition, unspecified	Albumin must be below 3.4 and at least one of the following criteria must be met:
Disease/injury related malnutrition	– One indicator of malnutrition as described above – Peripheral edema or anasarca – Delayed wound healing – History of acute or chronic disease or trauma

Albumin should not be used as the sole indicator of visceral protein status in fluid imbalance, liver disease, post-operative states, infection, and nephrotic syndrome. Serum albumin levels drop when there is inflammation; when inflammation is corrected, levels may rise again regardless of nutritional intake. Albumin is a marker of severity of illness, not as a marker of protein nutriture.
Adapted from the Nutrition Support Committee, Emory Hospitals, Atlanta GA; 2008.

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TABLE 10-21 Malnutrition Universal Screening Tool (MUST)

MUST is a 5-step screening tool to identify adults who are malnourished, at risk of malnutrition (undernutrition), or obese. It includes management guidelines for care planning. It is used in hospitals, community care settings and other care settings and can be used by all care workers. It was established by the British Association for Enteral and Parenteral Nutrition (BASPEN). The guide contains the following:

- A flow chart showing 5 steps to use for screening and management
- BMI chart
- Weight loss tables
- Alternative measurements when BMI cannot be obtained by measuring weight and height

A more detailed MUST explanatory booklet should be used for procedures when weight and height cannot be measured and when screening with more interpretation is needed (e.g., those with fluid disturbances, plaster casts, amputations, or critical illness, or pregnant or lactating women).
The 5 MUST steps are as follows.

Step 1: Measure height and weight to get a BMI score using the chart provided.

If height cannot be measured: use recently documented or self-reported height (if reliable and realistic). If the subject does not know or is unable to report their height, use one of the alternative measurements to estimate height (ulna, knee height, or arm span).

If height and weight cannot be obtained: use mid upper arm circumference (MUAC) measurement to estimate BMI category.

If BMI cannot be determined: use subjective clinical impression—thin, acceptable weight, overweight. Obvious wasting (very thin) and obesity (very overweight) can also be noted.

BMI	Score
>20	☐ 0
18.5–20	☐ 1
<18.5	☐ 2
BMI score = _____	

Alternative procedures are available from the guide http://www.baspen.org.uk/the-must.htm.

Step 2: Note percentage of unplanned weight loss, and score using tables in the screening tool.

If recent weight loss cannot be calculated: use self-reported weight loss (if reliable and realistic).

Unplanned weight loss: clothes and/or jewelry have become loose fitting (weight loss); history of decreased food intake, reduced appetite or swallowing problems over 3–6 months; underlying disease or psychosocial/physical disabilities likely to cause weight loss.

Record presence of obesity. Control underlying conditions before treating obesity.

% of Unplanned weight loss in past 3–6 months	Score
<5%	= 0
5–10%	= 1
>10%	= 2
Weight loss score = _____	

Step 3: Establish the acute disease effect and score.

No nutritional intake or likelihood of no intake for more than 5 days = score 2.

Acute disease effect score = _____

Step 4: Add scores from steps 1, 2, and 3 to obtain the overall risk of malnutrition.

0 = Low Risk
1 = Medium Risk
≥2 = High Risk

(continued)

TABLE 10-21 Malnutrition Universal Screening Tool (MUST) *(continued)*

Step 5: Develop care plan and treat.

Observe and document dietary intake for 3 days if patient is in hospital or long-term care (LTC) facility.

If improved or adequate intake—little clinical concern. If no improvement—clinical concern; follow local policy. Repeat screening: Hospital, weekly; LTC facility, at least monthly; community, at least every 2–3 months.

Treat unless detrimental, no benefit is expected from nutritional support, or death is imminent:

Record need for special diets and follow local policy.

Record malnutrition risk category; as needed, refer to dietitian or nutritional support team or implement local policy.

Treat underlying condition and provide help and advice on food choices, eating, and drinking when necessary.

Improve and increase overall nutritional intake.

Monitor and review care plan: hospital, weekly; LTC facility, monthly; community, monthly.

SAMPLE NUTRITION CARE PROCESS STEPS

Malnutrition

Assessment Data: Diet, intake records, weight, medical history, frequency of infections, serum levels of vitamins such as vitamin A, lipids such as total cholesterol.

Nutrition Diagnoses (PES):

Malnutrition (NI-5.2) related to lack of appetite and poor food selection as evidenced by diet recall showing consumption of only 1200 kcal/d and 25 grams PRO, percent wt change of > 10% over the past 6 months, BMI of 18, serum cholesterol of 130, and albumin of 2.5 g/dL.

Involuntary weight loss (NC 3.2) related to inadequate dietary intake as evidenced by reported dietary recall and daily intake of 500–700 kilocalories was less than estimated needs.

Interventions:

Food and Nutrient Delivery: ND 1.3 Increase fiber and fluid intake

ND 1.2 High calorie, high protein diet

ND 5.7 Initiate meals on wheels

Education: E-1.2 Provide examples of high fiber, high protein foods, and high calcium foods

ND – 6.5 Develop regular schedule for taking iron supplements

Counseling: C-2.5 Discuss social support

C-2.4 Discuss fluid needs and how to practically incorporate them into daily meal plans

Coordination of Care: Contact psychologist to review ongoing depression and meds, make arrangements for dental clinics, refer to social worker for community and financial resources

Monitoring and Evaluation: Assess po intake of kilocalories, PRO, and fluid. Monitor weight and labs. Review intake and activity logs. Monitor for changes in BMI over time, fewer infections, and improved quality of life.

- Develop a targeted therapeutic approach to skeletal muscle loss and muscle strength in older persons (Thomas, 2007).
- Establish a nutritional plan according to patient prognosis (Table 10-23).

FOOD AND NUTRITION

- Monitor physical exam and clinical status to determine needed dietary changes.
- **Mild malnutrition:** Provide sufficient calories and protein, gradually increasing to meet needs. Diet should provide adequate carbohydrate (CHO) and caloric intake to spare protein and correct weight loss. Use tube feeding or CPN, if appropriate (see Section 17). Vitamin-mineral supplementation is recommended.
- **Severe malnutrition or cachexia:** Start treatment with intravenous glucose. Gradually add lactose-treated milk and soft, easily tolerated solids. Provide high-biologic value proteins with sufficient calories that are adequate to use nitrogen effectively. Avoid overfeeding (use 20–25 kcal/kg, progressing gradually to 35–40 kcal/kg). Add a vitamin–mineral supplement, particularly including thiamin. Provide enteral feeding, if needed; start with continuous versus intermittent or bolus feedings at a slow rate until serum electrolyte levels are stable.
- Practical suggestions for improving intake in debilitated patients include liberalizing previous diet restrictions where safe and appropriate, addressing impaired dentition and swallowing, addressing physical and cognitive deficits, encouraging family and friends to provide favorite foods, addressing poor consumption of specific foods, and providing appropriate nutrient supplements (Harris and Fraser, 2004).
- Oral nutritional supplements are feasible in nutritionally depleted patients, particularly between meals.

TABLE 10-22 Selected Biochemical Changes Observed in Severe Protein–Energy Malnutrition (PEM)

Body Composition	Energy Malnutrition	Protein Malnutrition/ Edema
Total body water	High	High
Extracellular water	High	Higher
Total body potassium	Low	Lower
Total body protein	Low	Low
Serum or plasma		
Transport proteins (transferrin, ceruloplasmin, retinol-, cortisol-, and thyroxine-binding proteins, beta-lipoproteins)[a]	Normal or low	Low
Enzymes such as amylase, alkaline phosphatase	Normal	Low
Transaminase	Normal or high	High
C-reactive protein	Varies by condition	Varies
Liver		
Glycogen	Normal or low	Normal or low
Urea cycle enzymes and other enzymes	Low	Lower
Amino acid synthesizing enzymes	High	Not as high

[a]Note. Inflammatory processes and their effects on hepatic protein metabolism (albumin, transferrin, and transthyretin) have been identified. Serum hepatic protein levels correlate with *severity* of illness but do not accurately measure effectiveness of nutritional repletion (Fuhrman et al, 2004). Evaluate their value cautiously.

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- Assure adequate intake of vitamin D_3 from sunlight exposure, diet, and supplements to support gene expression and health.

Common Drugs Used and Potential Side Effects

- Medications that are often used to increase intake by stimulating appetite include oxandrolone (Oxandrin), megestrol acetate (Megace), cyproheptadine HCl (Periactin), and dronabinol (Marinol).
- Experimental therapies for treating cachexia include nonsteroidal antiinflammatory drugs, tumor necrosis factor alpha antagonists, tetrahydrocannabinol, growth hormone, ghrelin, oxandrolone, and omega-3 fatty acids (Gullett et al, 2009). Comparing oxandrolone, strength training, and nutrition alone, strength training is most cost effective and improves quality of life more than nutrition alone or oxandrolone (Shevitz et al, 2005).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing it with the physician.
- Treatment of vitamin D deficiency in otherwise healthy patients with 2000–7000 IU vitamin D per day should be sufficient to maintain year-round 25(OH)D levels in the range of 40–70 ng/mL (Cannell and Hollis, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Emphasize the importance of gradual refeeding.
- Discuss the complicating effects of malnutrition. Unless nutritional therapy is aggressive, infection and sepsis are major risks, and surgery becomes life threatening. PEM can increase fistula formation, reduce recovery and wound healing after surgery, and lead to pneumonia or poor drug tolerance.
- Allow patients to participate in feeding decisions. Set goals and help plan together with family.

Patient Education—Food Safety

Use of tube feeding or CPN at home warrants training to prevent foodborne illnesses. Handwashing, counter sanitation, and sterile techniques are important in handling these products and the administration kits.

TABLE 10-23 Poor Prognosis and Consequences of Not Feeding a Patient

Clinical manifestations of PEM relate to length of time, extent of nutritional deprivation, and prior health status. There are serious detrimental effects on every organ. "When maintained on a prolonged semi-starvation diet, otherwise healthy individuals experience a **loss of heart tissue** that parallels their loss of body mass. Respiratory rate, vital capacity, and minute volume of ventilation also decrease. These **changes in pulmonary function** are thought to result from reduced basal metabolic rate that accompanies starvation. In addition, **liver function declines, kidney filtration rates decline, and nearly every aspect of the immune system is compromised.** Defective ability to fight bacterial and viral infections occurs. Starvation therefore leads to **increased susceptibility to infection, delayed wound healing, reduced rate of drug metabolism, and impairment of both physical and cognitive function.** If starvation is prolonged, complications develop, **leading eventually to death**" (Sullivan, 1995).

Other consequences of not feeding an individual who *will not* or *cannot* eat orally in sufficient amounts include

- **Dehydration** with increased risk of urinary tract infections, fever, swollen tongue, sunken eyeballs, decreased urine output, constipation, nausea, vomiting, decreased blood pressure, mental confusion, and electrolyte disturbances.
- **Decreased awareness** of environment from decreased glucose availability for the brain.
- **Development of new or additional pressure ulcers** over bony prominences from lack of sufficient protein, calorie, vitamin, and mineral intakes and decreased body fat.
- **Decreased ability to participate in activities of daily living** (self-feeding, dressing, bathing, toileting).
- **Low body weight or rapid, involuntary weight loss,** which are highly predictive of illness and imminent death. The elderly are particularly unable to regain weight after a stress situation.

REFERENCE

Sullivan D. The role of nutrition in increased morbidity and mortality. *Clin Geriatr Med.* 11:663, 1995.

Poor prognosis may be seen in individuals with the following conditions who also have PEM:

Age <6 months

Cachexia from chronic renal failure

Clinical jaundice or elevated serum bilirubin level

Circulatory collapse: cold hands and feet, weak radial pulse, diminished consciousness

Deficit in weight for height >30% or in weight for age >40%

Dehydration and electrolyte disturbances, particularly hypokalemia and severe acidosis

Extensive exudative or exfoliative cutaneous lesions or deep pressure ulcers

Hypoglycemia

Hypothermia

Infections, particularly bronchopneumonia or measles

Petechiae or hemorrhagic tendencies (purpura is usually associated with septicemia or a viral infection)

Persistent tachycardia, signs of heart failure, or respiratory difficulty

Severe anemia with clinical signs of hypoxia

Stupor, coma, or other alterations in awareness

Cachexia is characterized by maladaptive responses such as anorexia, elevated basic metabolic rate, wasting of lean body tissue, and underutilization of fat tissue for energy. Inflammation secondary to cytokines is significant.

For More Information

- Abbott Health Nutrition Institute
  http://abbottnutritionhealthinstitute.org/
- Mayo Clinic
  http://www.mayoclinic.com/health/senior-health/HA00066
- Nestle Nutrition
  http://www.nestlenutrition.com/en
- Nutrition Day Worldwide
  http://www.nutritionday.org/
- World Health Organization
  http://www.wpro.who.int/health_topics/protein_energy/

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REFEEDING SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

The effects of starvation are extensive and negatively affect the pituitary gland, thyroid gland, adrenal glands, gonads, and bones (Usdan et al, 2008). Refeeding syndrome (RFS) refers to these various metabolic abnormalities that may complicate carbohydrate and protein administration in undernourished patients, as in anorexia nervosa or head and neck cancer. Other conditions that can lead to refeeding syndrome can be found in Table 10-24.

During refeeding, insulin release is stimulated by the presence of carbohydrate and protein in the gut. Insulin plays a key role in the switch from using up body stores to using food; it stops the release of fat from stores and the production of glucose from protein. In refeeding, the increase in insulin lowers glucagon levels. With gluconeogenesis, glycogenolysis, and fatty acid mobilization, glucose is taken up rapidly into the cells. Phosphorus is driven inside the cells; the result is a dangerous hypophosphatemia. Adenosine triphosphate (ATP) levels drop, with major effects on the cardiac, pulmonary, CNS, hematological, and muscular systems. This process also stops sodium excretion and causes fluid retention in the first few days of refeeding or when caloric intake is increased.

TABLE 10-24 Conditions with High Risk for Refeeding Syndrome

Alcoholism
Anorexia nervosa
Chronic underfeeding
Hepatic failure
Malabsorption from GI damage or chronic use of phosphate binders such as aluminum-containing antacids or sucralfate
Morbid obesity with massive weight loss from fasting
Protein–energy malnutrition
Prolonged fasting
Prolonged parenteral nutrition
Respiratory alkalosis

The typical patient who experiences RFS has been malnourished for days to weeks and develops hypophosphatemia and, occasionally, hypokalemia and hypomagnesemia when administered a carbohydrate load in the form of glucose-concentrated fluids, total parenteral nutrition, tube feedings, or an oral diet (Marinella, 2009). Overwhelming cardiovascular and pulmonary manifestations can accompany refeeding with carbohydrate in chronically malnourished patients (Miller, 2008). Increases in heart rate, blood pressure, oxygen consumption, cardiac output, and an expansion of plasma volume are seen. The response is dependent on the amount of calories, protein, and sodium administered, and the malnourished heart can easily be given a metabolic demand that is too high for it to supply.

Stressed, critically ill patients may be at risk of refeeding following short periods of fasting (Miller, 2008). RFS usually occurs within 4 days of starting to feed again. The respiratory muscle, reduced in mass and ATP content by malnutrition, is unable to respond to the increased workload imposed by aggressive nutrition support. Excess carbon dioxide production and increased oxygen consumption can result from giving too much glucose and overfeeding. A person with malnutrition-induced respiratory muscle wasting can get short of breath and cannot sustain an increased ventilatory drive. Pulmonary edema may develop in some due to increased water load, and this may lead to respiratory failure. Clearly, RFS syndrome is a life-threatening, underdiagnosed but treatable condition (Gariballa, 2008).

Precipitous falls in electrolyte levels in persons with diabetes that occur due to intracellular shifts are a result of the anabolic effects of insulin doses (Parrish, 2009). This form of refeeding is common but not always recognized.

Problems with sodium derangements may lead to heart failure. When potassium shifts into cells, hypokalemia and arrhythmias can occur. When magnesium shifts intracellularly, tetany and seizures may be seen. Thiamin deficiency must be prevented during refeeding, because it is a cofactor in carbohydrate metabolism, important for both the heart and the brain. With thiamin deficiency, there can be signs such as mental confusion, ataxia, muscle weakness, edema,

muscle wasting, tachycardia, and cardiomegaly. Wernicke's encephalopathy can be precipitated by carbohydrate feeding in thiamine-deficient patients.

The gut is also affected by malnutrition, where it begins to atrophy. Activity of the brush border enzymes and pancreatic enzyme secretion return to normal with refeeding, but it requires a period of readaptation to food to minimize GI complaints such as diarrhea, nausea, and vomiting.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Refeeding syndrome is not genetically derived.

Clinical/History

Height
Weight
BMI
Desirable BMI
Percentage of usual weight
History of weight changes
Diet history
I & O
Anorexia
Bone pain
Edema
Tachycardia
Temperature
Rhabdomyolysis
pCO_2, pO_2
Respiratory insufficiency or failure
Dizziness
Spontaneous diarrhea

Lab Work

Serum phos (low?)
Mg^{++} (low?)
K^{+} (low?)
Gluc
Na^{+}
Chol, Trig
H & H
Serum Fe
Red blood cell dysfunction
Ca^{++}
BUN, Creat
Partial pressure of carbon dioxide (pCO_2)
Partial pressure of oxygen (pO_2)

INTERVENTION

OBJECTIVES

- Correct starvation without overloading the system with nutrients of any type. Use less than full levels of calorie and fluid requirements. Weight gain is not a goal during the first week.
- Advance calories and volume with careful monitoring of cardiac and respiratory side effects.
- Increase nutrition support slowly while assuring adequate amounts of electrolytes. Check electrolytes 2–3 times daily until stable. Organ function and fluid balance also need to be monitored daily during the first week and less frequently after that time.
- Distinguish between constitutional thinness and malnutrition since the causes are different, as are risks for RFS.
- Monitor for neurological, hematological, and metabolic complications of hypokalemia, hypophosphatemia, and hyperglycemia. Prevent sudden death.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Energy Intake—Refeeding Syndrome

Assessment Data: A 60-year old female with a 4-month history of poor dietary intake; connective tissue disease leading to myositis and dysphagia; respiratory failure needing mechanical ventilation. Low baseline electrolyte concentrations, including potassium, magnesium, calcium, and phosphate, and low serum albumin. Twelve hours after starting nasogastric tube feeding, she developed a cardiac arrest from which she was successfully resuscitated. Repeated attempts to wean her from the ventilator failed. BMI low at 16. Clinical nutrition team made the diagnosis of refeeding syndrome.

Nutrition Diagnosis: Excessive energy intake related to 4-month history of poor oral food and beverage intake as evidenced by diet history and respiratory failure with need for ventilator dependency.

Intervention: Food-nutrient delivery: high-protein, high-fat, low-carbohydrate diet, multivitamin and trace-element supplements, and electrolyte infusion. Education: role of proper pace of refeeding in someone who has been starving recently.

Monitoring and Evaluation: Monitor labs 2–3 times daily until labs improve (potassium, magnesium, calcium, and phosphate). Evaluate readiness for ventilator weaning after intake is managed and gradually increased over 14–21 days. Monitor for improvements in weight and BMI.

FOOD AND NUTRITION

Refeeding an Adult

- Avoid malnutrition—intervene early and use D5 (5% dextrose) with any saline infusion when patients are designated NPO or are on limited diets for procedures/surgery (Parrish, 2009).
- Start patients at 15–20 cal/kg for the first 3 days, but also start enteral nutrition or total parenteral nutrition at low infusion rates (Parrish, 2009). Feeding can be gradually increased and be up to desired levels by day 7.
- Protein should be started slowly (1.2 g/kg actual weight) and increased gradually to 1.5 g/kg to protect and restore some lean body mass.
- At first, restrict carbohydrate (CHO) intake to 150–200 g/d to prevent a rapid insulin surge. CHO in parenteral nutrition (PN) should be initiated at 2 mg/kg/min (about 150–200 mg/d). Provide insulin as needed to keep blood glucose within a normal range and to protect nutritional stores.
- Fat calories should make up the difference.
- Refeeding results in expansion of the extracellular space, and fluid must be given carefully during the first few days to weeks of refeeding. Weight gain greater than 1 kilogram in the first week is due to fluid retention. Fluid may need to be restricted to 800 to 1000 cc/d. Increases in blood pressure, heart rate, and respiratory rate may be early signs of fluid excess. Adjust when edema exists (e.g., fluid restriction according to I & O, tachycardia, peripheral edema).
- Rather than using boluses of potassium, phosphate, and magnesium via the enteral access, intravenous replacement can be slower and better tolerated (Parrish, 2009).

- Adjust electrolytes depending on laboratory values. Sodium must be given carefully to prevent overexpansion of the extracellular fluid. Additional phosphorus is required; 250–500 mg/d for up to 5–7 days may be needed to replenish. Potassium serum levels should be in the high normal range with 80 to 120 mEq/d needed.
- Magnesium and thiamin should also be given. Supplement with other vitamins and minerals as needed. Excesses are not required.

Refeeding a Child (Tips from the World Health Organization)

- Refeeding in a child should be through oral or tube feeding, not intravenous feedings. Use 100 kcal/kg actual weight; protein as 1–1.5 g/kg; 100–130 ml/g fluid daily. Breastfeeding may continue, but formula may be given first.
- Starter formula may be made with 300 milliliters of cow's milk, 100 grams of sugar, 20 milliliters of oil, 20 milliliters of electrolyte/mineral solution, and water to make 1000 milliliters. Feed the child every 2 hours at first, gradually decreasing to every 3 hours over the first week. If intake does not reach the 80 kcal/kg goal, then night tube feeding may be needed.
- Return of an appetite is often a first sign that the rehabilitative phase has begun; this may take a week. Continue a gradual increase to avoid heart failure. Increase each feed by 10 milliliters until some remains uneaten, usually around intakes of 200 ml/kg/d.
- After a gradual transition, give frequent feeds with unlimited amounts; 150–220 kcal/kg/d and 4–6 g/kg/d of protein are reasonable estimates. Catch-up formula can be made using 880 milliliters of milk, 75 grams of sugar, 20 milliliters of oil, 20 milliliters of electrolyte/mineral solution, and water to make 1000 milliliters.
- Sensory stimulation and emotional support is also a part of the therapy, so there is a need to provide tender loving care and a cheerful, stimulating environment. Structured play therapy for 15–30 minutes a day and physical activity as soon as the child is well enough are also important.
- The World Health Organization promotes healthy pregnancy in order to avoid chronic disease later in life (WHO, 2009). There is an association between low growth in the first year and an increased risk of CHD. Blood pressure has been found to be highest in those with retarded fetal growth and greater weight gain in infancy.
- Studies of children and health risk have found that the thinnest children, if they became obese as adults, have a greater risk of developing chronic diseases (WHO, 2009). With anorexia nervosa, short stature, osteoporosis, and infertility may be long-lasting complications (Usdan et al, 2008). Short stature may be associated with an increased risk of CHD, stroke, and even diabetes.

Common Drugs Used and Potential Side Effects

- Replacement of phosphorus, potassium, and magnesium may be needed if serum levels are depleted. ORS is responsible for saving the lives of millions of children worldwide; it is an inexpensive solution of sodium and glucose used to treat acute diarrhea.
- Monitor specific medications used and their side effects (e.g., gastrointestinal distress).
- Insulin is used to correct hyperglycemia levels. Monitor blood glucose levels as refeeding occurs.
- In clinical practice, it is not uncommon to give patients a 100-mg thiamine bolus daily for 3 days when they are at risk for refeeding syndrome (Francini-Pesenti, 2009; Parrish, 2009). Give the other B-complex and vitamins as well (Mehanna et al, 2009).
- Advances in leptin, ghrelin, and endocannabinoid systems may provide therapeutic breakthroughs. Ghrelin facilitates nutritional restoration and lowering serum leptin may be needed to avoid RFA (Stoving et al, 2009).

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Provide nutrition education to focus on adequate nutrient intake.
- Consider referral if food insecurity is a concern.
- Offer guidelines according to a discharge intervention plan for use at home or elsewhere. The physician may suggest long-term medication use or therapies.
- Encourage research in this area since evidence is lacking (Mehanna et al, 2009).

Patient Education—Food Safety

There are no specific food handling techniques that are unique to malnutrition. However, use of tube feeding or CPN at home warrants training to prevent infections.

For More Information

- Critical Care Tutorials
  http://www.ccmtutorials.com/
- Hypophosphatemia
  http://emedicine.medscape.com/article/767955-overview
- Refeeding Syndrome
  http://www.ccmtutorials.com/misc/phosphate/page_07.htm

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Usdan LS, et al. The endocrinopathies of anorexia nervosa. *Endocr Pract.* 14:1055, 2008.

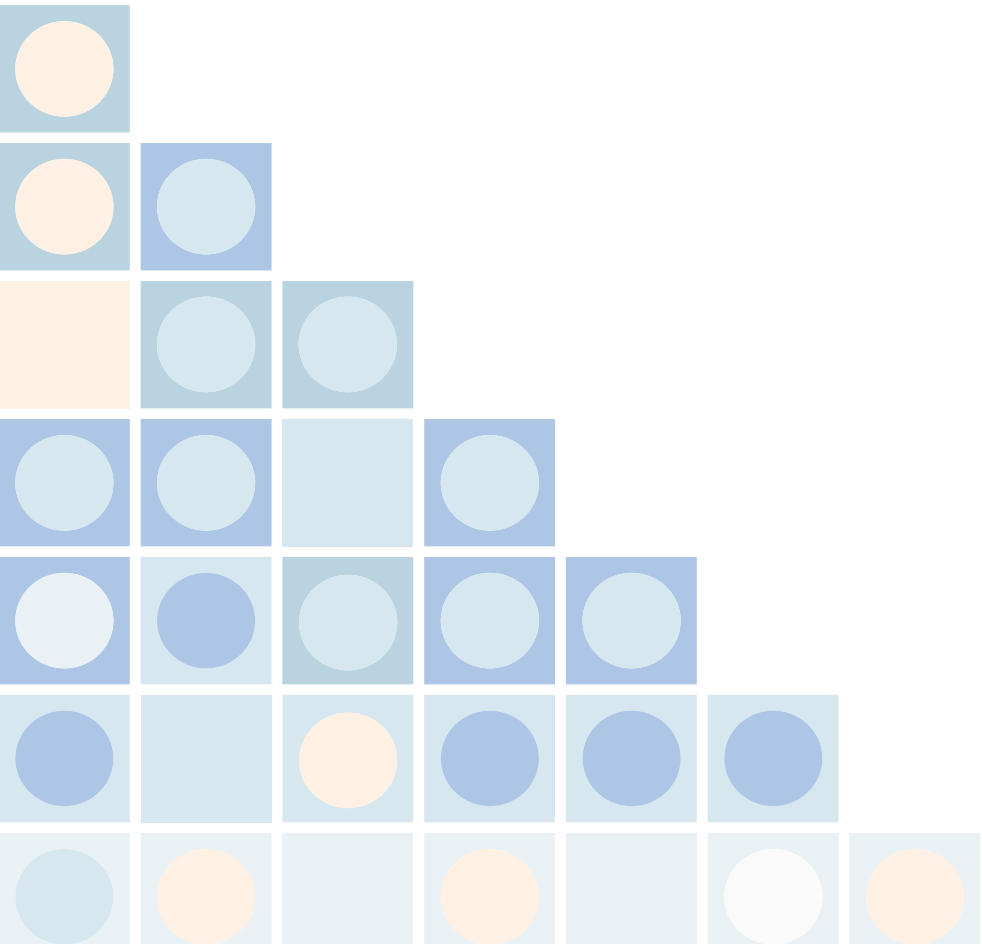
World Health Organization (WHO). Diet, nutrition and the prevention of chronic diseases. Accessed November 10, 2009 at http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf.

SECTION 11

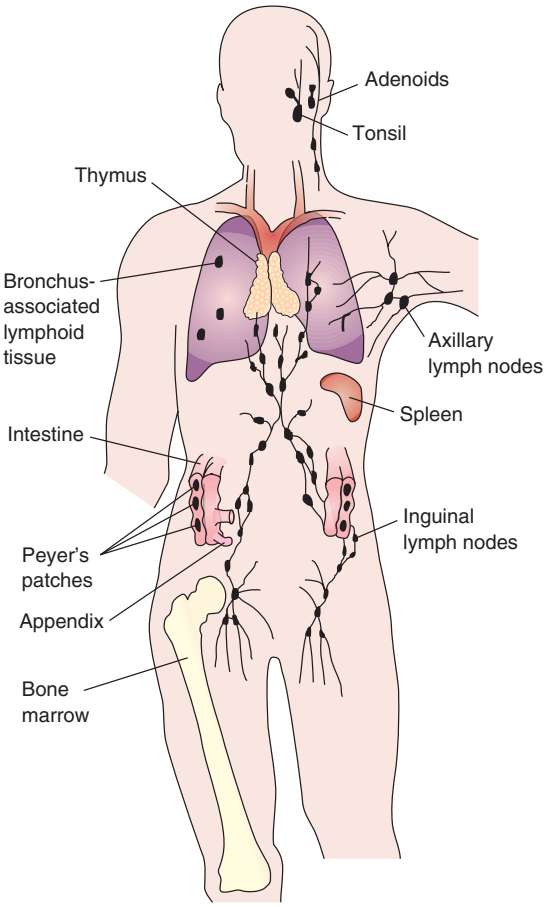
Musculo-Skeletal and Collagen Disorders

CHIEF ASSESSMENT FACTORS

- Actual Height, Measured Annually for Height Loss
- Arthritis—Warning Signs and Symptoms >2 Weeks: Early Morning Stiffness; Swelling in One or More Joints; Redness and Warmth in a Joint; Unexplained Weight Loss, Fever, or Weakness Combined with Joint Pain
- Bone Density Assessment
- Bone-Wasting Medications
- Contractures
- Easy Fatigue
- Edema
- Extremity Weakness
- Inflammation of Joints
- Movement Problems, Stiffness
- Pain in Muscles, Joints, Bones, Spine
- Psoriasis
- Unsteady Gait and Propensity to Fall
- Weight Loss, Anorexia, Depression, Insomnia
- Vitamin D_3 status (serum 25-OHD)



OVERVIEW—RHEUMATIC DISORDERS



Adapted from: Porth CM. *Pathophysiology: Concepts of altered health states,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 1998.

TABLE 11-1 Autoimmune Rheumatic Disorders[a]

Blood and blood vessels	Lupus
	Polyarteritis nodosa
	Temporal arteritis and Polymyalgia rheumatica
Digestive tract and mouth	Scleroderma
	Sjögren's syndrome
Eyes	Sjögren's syndrome
	Uveitis
Heart	Ankylosing spondylitis
	Lupus
	Rheumatic fever
	Scleroderma
Joints	Ankylosing spondylitis
	Lupus
	Osteoarthritis
	Rheumatoid arthritis
Kidneys	Gout
	Lupus
Lungs	Lupus
	Rheumatoid arthritis
	Scleroderma
Muscles	Polymyositis
Nerves and brain	Lupus
Skin	Lupus
	Scleroderma

[a]When the immune system does not work right, the immune cells can mistake the body's own cells as invaders and attack them; these are called autoimmune diseases. In this table a sample list of body systems affected by autoimmune rheumatic disorders.
Adapted from: National Institutes of Health (NIH). NIH Publication No. 02–4858. Available at http://www.niams.nih.gov/hi/topics/autoimmune/autoimmunity.htm.

Some rheumatic diseases involve connective tissues and others may be caused by autoimmune disorders, where the body attacks its own healthy cells and tissues. See Table 11-1. Rheumatic disorders include osteoarthritis (OA), rheumatoid arthritis (RA), juvenile RA, bursitis, tendonitis, infectious arthritis, spondyloarthropathies, polymyositis, psoriatic arthritis, systemic lupus erythematosus, scleroderma, polymyalgia rheumatica, polyarthritis nodosa, giant cell arteritis, gout, and fibromyalgia. Typically, treatment of these disorders includes a rheumatologist who specializes in the treatment of disorders that affect joints, soft tissue, bones and connective tissues.

Arthritis represents a group of more than 100 different rheumatic diseases that cause stiffness, pain, swelling in the joints, muscles, ligaments, tendons or bones. Over 15% (40 million) of Americans have some form of arthritis. *Spondylosis* is OA of the spine. *Infectious arthritis* is caused by bacterial invasion spread from nearby joints following chickenpox, rubella, or mumps. Autoimmune disorders, Crohn's disease, and psoriasis may cause *seronegative arthritis. Mixed connective tissue disease* shows features of RA, cutaneous systemic sclerosis, inflammatory myopathies and Raynaud's syndrome.

Mast cells and basophils are involved in several inflammatory and immune events and are known to produce a broad spectrum of cytokines (Rasheed et al, 2009). The activation of nuclear transcription factor-κB is linked with arthritis, osteoporosis, and psoriasis. The cytokine tumor necrosis factor alpha (TNFα) plays a key role in chronic inflammatory and rheumatic diseases.

Early recognition and treatment of these disorders are important. RA, juvenile idiopathic arthritis, the seronegative spondyloarthropathies, and lupus may have skeletal pathology (Walsh et al, 2005) and an inflammatory atherosclerosis. A multidisciplinary, multipronged approach is best. Physical and occupational therapies are beneficial for maintaining as much independence as possible in these conditions.

Most rheumatic conditions are managed by use of nonsteroidal anti-inflammatory drugs (NSAIDs) and TNFα antagonists. Etanercept, infliximab, and adalimumab significantly reduce symptoms and improve both functionality and quality of life (Braun et al, 2006; Nash and Florin, 2005). Fortunately, research is on-going for the autoimmune diseases. Gene profiling is helpful, especially in pediatrics (Jarvis, 2005). Osteoimmunology is a new branch of medical science, and anti-inflammatory therapies promise new treatments.

Role of Inflammation and Fatty Acids

Excessive and inappropriate inflammation contributes to acute and chronic human diseases. It is characterized by the production of inflammatory cytokines, arachidonic