SECTION 16

Renal Disorders

CHIEF ASSESSMENT FACTORS

Ten Symptoms of Kidney Disease

- Changes in urination
- Confusion
- Feeling dizzy
- Headache
- High blood pressure
- Loss of appetite or change in taste
- Nausea or vomiting
- Severe itching not related to a bite or rash
- Shortness of breath
- Swelling of face, hands, and/or feet

Other Factors

- Altered Lipid and Amino Acid Levels
- Abnormal Blood Urea Nitrogen (BUN):Creatinine Ratio
- Anorexia
- Bone Pain, Altered Height, or Lean Body Mass
- Burning or Difficulty During Urination
- Changes in Glomerular Filtration Rate (GFR)
- Chronic Inflammation
- Flank Pain
- Frequent Weight Shifts
- Ghrelin and Obestatin Ratios
- Insomnia
- Itching, Dry Skin
- Leg Cramps
- More Frequent Urination and Nocturia
- Pain in Small of Back Just Below Ribs, Not Aggravated by Movement
- Passage of Bloody-Appearing Urine (Hematuria)
- Presence or History of Urinary Tract Infections or Stones
- Proteinuria, Microalbuminuria
- Protein–Energy Malnutrition or Wasting
- Puffiness Around Eyes
- Serum Creatinine 1.7 mg/dL
- Unbalanced Calcium:Phosphorus Ratios
- Uremia
- Weakness, Pallor, Anemia

OVERVIEW OF RENAL NUTRITION

According to the National Institute of Diabetes and Digestive and Kidney Diseases, more than 16% of adults 20 years and older (23 million people) have evidence of chronic kidney disease (CKD), with moderately or severely reduced glomerular filtration rate (GFR) (NIDDK, 2010). The Kidney Disease Outcomes Quality Initiative (KDOQI) classifies CKD on the basis of the level of kidney function. Simple blood tests, as follows, can be done to determine renal status but are not always ordered (Aase, 2010):

- Reduction of kidney function—eGFR $<$ 60 mL/min/1.73 m^2 and/or
- Evidence of kidney damage/persistent albuminuria—$\geq$30 mg of urine albumin per gram of urine creatinine (UACR)
- Kidney failure eGFR $<$ 15 mL/min/1.73 m^2

The kidney plays an important role in blood pressure management through the renin–angiotensin system and identifies the damage from inflammation following scarlet fever, flu, or tonsillitis. Table 16-1 gives a brief background about the kidneys.

A registered dietitian (RD) with renal experience should be a central and integral part of the management of both pediatric and adult patients (NKF, 2010). In pediatrics, the evaluation of growth as well as physical, developmental, educational, and social needs is essential. Table 16-2 lists terms and abbreviations commonly used in renal disorder management.

Renal patients need a detailed nutritional assessment by dietitians (Chauveau, 2009). Subjective Global Assessment (SGA) is used and includes weight change over the past 6 months, dietary intake, gastrointestinal (GI) symptoms, visual assessment of subcutaneous tissue and muscle mass. While weight change is assessed by evaluating weights during the past 6 months, edema might obscure a greater amount of weight loss. Dietary intake is evaluated by using a comparison of the patient's usual or recommended intakes to current intake. Duration and frequency of GI symptoms (e.g., nausea, vomiting, and diarrhea) are also assessed. The interviewer rates a 7-point scale with higher scores if the patient has little or no weight loss, a better dietary intake, better appetite, and the absence of GI symptoms. See Appendix B for the SGA questionnaire.

Nutritional interventions and specifically supplemented diets have many advantages in terms of managing the progression of renal failure, better metabolic and endocrine control, and decreased proteinuria (Chauveau, 2009). Because anorexia and weight loss are associated with wasting, morbidity, and mortality, dietitians must be aware of the impact of hormones, such as the orexigenic ghrelin and the anorexigenic obestatin, on intake (Mafra et al, 2010). In addition, the role of inflammation must be understood. Inflammation causes increased levels of cytokines (interleukin [IL]-6 and tumor necrosis factor [TNF]-α) and acute-phase proteins (C-reactive protein [CRP] and serum amyloid A); loss of muscle mass; changes in plasma composition; decreases in serum albumin, transthyretin, and transferrin; altered lipoprotein structure and function to favor atherogenesis. Knowing that fish oils may decrease the loss of renal function by affecting eicosanoid and cytokine production, altering renal dynamics, and decreasing inflammation is important for all dietitians to understand.

Renal replacement therapy (RRT) includes dialysis and renal transplantation. RRT is designed to normalize the volume and composition of the body fluids and to remove uremic toxins. But because renal disease is often related to diabetes and hypertension, a multidisciplinary approach is warranted.

Not everyone with CKD needs a strict hemodialysis (HD) diet. Appropriate, individualized use of one- and two-page information sheets on sodium, phosphorous, potassium,

TABLE 16-1 Human Kidney Functions

Waste excretion: Kidneys remove the waste products of metabolism (urea, uric acid, creatinine).

Acid–base balance: Kidneys regulate pH by eliminating excess hydrogen ion concentration and controlling composition of the blood. Blood plasma pH is maintained by the kidney at a neutral pH of 7.4. Urine is either acidic (pH 5) or alkaline (pH 8). Potassium and phosphate require renal control as well.

Blood pressure control: Sodium ions are controlled in a homeostatic process involving aldosterone, which increases sodium ion absorption in the distal convoluted tubules. When blood pressure is too low, *renin* is secreted by cells of the distal convoluted tubule. Renin acts on the blood protein, *angiotensinogen*, converting it to *angiotensin I*. Angiotensin I is then converted by the angiotensin-converting enzyme in the lung capillaries to *angiotensin II*, which stimulates the secretion of *aldosterone* by the adrenal cortex. Aldosterone stimulates increased reabsorption of sodium ions from the kidney tubules, which causes an increase in the volume of water that is reabsorbed from the tubule. This increase in water reabsorption increases the volume of blood, which ultimately raises the blood pressure.

Plasma volume and osmolality: Any rise or drop in blood osmotic pressure due to a lack or excess of water is detected by the *hypothalamus*, which notifies the *pituitary gland* via negative feedback. A lack of water causes the *posterior pituitary gland* to secrete *antidiuretic hormone*, which results in water reabsorption and an increase in urine concentration. Tissue fluid concentration thus returns to normal. Body fluid is two thirds extracellular and one third intracellular.

Hormone secretion: *Erythropoietin* is secreted for red blood cell production; deficiency is common in chronic renal anemia. *Urodilatin* is a natriuretic peptide that mediates natriuresis. *Vitamin D_3* is converted from an inactive form (D2) for calcium:phosphorus homeostasis; the final stage of conversion of vitamin D to its active form, 1,25-dihydroxyvitamin D occurs in the proximal tubule.

Carnitine synthesis: Carnitine carries fatty acids from cytoplasm to mitochondria for heart and skeletal muscle fuel. Lysine, methionine, vitamin C, iron, vitamin B_6, and niacin are needed to produce carnitine.

Glucose homeostasis: The kidney plays a role in gluconeogenesis and glucose counterregulation.

Prostaglandin E_2: It is a major renal cyclooxygenase metabolite of arachidonic acid that impacts renal hemodynamics and salt and water excretion.

TABLE 16-2 Renal Abbreviations

Abbreviation	Description	Abbreviation	Description
a1-AG	a1-Acid Glycoprotein	IPAA	Intraperitoneal Amino Acids
aBWef	Adjusted Edema-Free Body Weight	Kt/V_{urea}	A measure of dialysis; K is the dialyzing membrane clearance, t is the time of dialysis delivered in minutes, and V_{urea} is the volume of distribution of urea
AMA	Arm Muscle Area	MAC	Midarm Circumference
APD	Automated Peritoneal Dialysis	MAMA	Midarm Muscle Area
BIA	Bioelectrical Impedance Analysis	MAMC	Midarm Muscle Circumference
BUN	Blood Urea Nitrogen	MD	Maintenance Dialysis
CANUSA	Canada/United States Peritoneal Dialysis Study	MHD	Maintenance Hemodialysis
CAPD	Continuous Ambulatory Peritoneal Dialysis	NHANES	National Health and Nutrition Evaluation Survey
CCPD	Continuous Cyclic Peritoneal Dialysis	nPCR	Protein Catabolic Rate normalized to body weight
CoA	Coenzyme A	nPNA	Protein Equivalent of Total Nitrogen Appearance normalized to body weight
CPD	Chronic Peritoneal Dialysis	PCR	Protein Catabolic Rate
CPN	Central Parenteral Nutrition	PNA	Protein Equivalent of Total Nitrogen Appearance
CrCl	Urinary Creatinine Clearance	PTH	Parathyroid Hormone
CRF	Chronic Renal Failure (GFR < 20 mL/min)	RTA	Renal Tubular Acidosis
CRI	Chronic Renal Insufficiency (GFR less than normal but >20 mL/min)	SBW	Standard Body Weight
CRP	C-Reactive Protein	SDS	Standard Deviation Score
CVVHD	Continuous Venovenous Hemofiltration with Hemodialysis	SGA	Subjective Global Assessment
DXA	Dual Energy X-Ray Absorptiometry	SUN	Serum Urea Nitrogen
ESRD	End-Stage Renal Disease	TBW	Total Body Water
GFR	Glomerular Filtration Rate	TNA	Total Nitrogen Appearance
HD	Hemodialysis	TSF	Triceps Skinfold
IDWG	Interdialytic Weight Gain	UBW	Usual Body Weight
IDPN	Intradialytic Parenteral Nutrition	UNA	Urea Nitrogen Appearance
IGF-I	Insulin-Like Growth Factor-I		

and protein will enable dietitians to advise their clients more effectively than using complex documents (Aase, 2010).

Ideally, dietitians who work with renal patients are certified renal dietetic specialists ("CS-R" credential), which requires experience and a successful examination score. However, because there is a gap between the dialysis unit (where renal dietitians practice) and the doctor's office (where gradual changes in CKD may not be detected), all dietitians must be comfortable with the basics of renal intervention (Aase, 2010).

While **diabetic renal disease** (diabetic nephropathy) is the leading cause of end-stage renal failure, the process cannot be reversed by glycemic control but by control of blood pressure and protein restriction. Therefore, specific nutrition care is needed for all renal patients—whether before starting dialysis, while on dialysis, or after a kidney transplant. Early dietary intervention is most critical to reduce sodium intake, control blood pressure, manage diabetes, and reduce excessive protein intake in early stages of CKD (Aase, 2010).

Standards of professional performance and standards of practice for dietitians in nephrology care set the expectations for renal practice (Brommage et al, 2009; Joint Standards Task Force, 2009). In addition, the American Dietetic Association evidence analysis library provides regular updates on research in the field. Medicare CKD–related medical nutrition therapy (MNT) reimbursement requires use of these evidence-based guidelines (EBGs) and currently begins at approximately stage 3 (GFR between 13 and 50 mL/min/1.73 m^2).

The National Kidney Disease Education Program (NKDEP) has developed multiple tools, tear-off sheets, and guidelines that can be used (see Web site http://nkdep.nih.gov/professionals/index.htm). There are clever lists of "kidney-friendly" foods, supermarket tips, and other simplified ways to enhance patient understanding. Once NKDEP brings primary care clinicians up to speed, referrals to RDs and rates of reimbursement for qualified CKD-related MNT should increase (Aase, 2010). Go to the Medicare MNT reimbursement Web site for more details at http://www.cms.hhs.gov/MedicalNutritionTherapy/01_Overview.asp#TopOfPage.

For More Information

- American Association of Kidney Patients
  http://www.aakp.org/
- American Kidney Fund
  http://www.kidneyfund.org/

- American Society of Pediatric Nephrology
  http://www.aspneph.com/
- Cyber Nephrology
  http://www.cybernephrology.org/
- Dialysis and Transplantation
  http://www.eneph.com/
- European Dialysis and Transplant Association
  http://www.era-edta.org/
- Home Dialysis
  http://www.homedialysis.org/
- International Society of Nephrology
  http://www.isn-online.org/
- International Society for Peritoneal Dialysis
  http://www.ispd.org/
- Kidney Disease: Improving Global Outcomes
  http://www.kdigo.org/
- Kidney Options Diet and Nutrition
  http://www.kidneyoptions.com/dietnutrition.html
- Kidney School
  http://www.kidneyschool.org/
- NKF
  http://www.kidney.org/
- NKF Handouts
  http://www.kidney.org/atoz/atozTopic_Brochures.cfm
- National Kidney and Urologic Diseases Information Clearinghouse
  http://kidney.niddk.nih.gov/
- Nephron Information Center
  http://www.nephron.com/
- Northwest Kidney Centers
  http://www.nwkidney.org/
- Renal Physicians Association
  http://www.renalmd.org/
- Renal World
  http://www.renalworld.com/
- UNC Kidney Center—Links
  http://www.unckidneycenter.org/about/links.html
- Vitamin D and Vitamin D Receptors in Kidney Disease
  http://www.kidney.org/professionals/tools/pdf/Vit_D_ReceptorsTool.pdf

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Brommage D, et al. American Dietetic Association and the National Kidney Foundation standards of practice and standards of professional performance for registered dietitians (generalist, specialty, and advanced) in nephrology care. *J Am Diet Assoc.* 109:1617, 2009.

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National Kidney Foundation (NKF). Web site accessed February 20, 2010, at http://www.kidney.org/Professionals/kdoqi/.

COLLAGEN-IV NEPHROPATHIES: ALPORT SYNDROME AND THIN GLOMERULAR BASEMENT MEMBRANE NEPHROPATHY

NUTRITIONAL ACUITY RANKING: LEVEL 2–3

DEFINITIONS AND BACKGROUND

Alport syndrome (ATS) is a progressive inherited (usually X-linked) nephritis. ATS is characterized by irregular thinning, thickening, and splitting of the renal glomerular basement membrane. It is often associated with hearing loss and ocular symptoms (Longo et al, 2006). Both ATS and **thin glomerular basement membrane nephropathy (TBMN)** are genetically heterogeneous conditions with initial presentation that usually involves hematuria (Haas, 2009). In ATS, kidney function is lost, with eventual progression to end-stage renal disease (ESRD) between adolescence and age 30 years (Tan et al, 2010). Mutational analysis of the affected genes will be a valuable adjunct to the treatment options, avoiding the need for transplantation and dialysis (Thorner, 2007).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Both ATS and TBMN can be considered as genetic diseases of the glomerular basement membrane (GBM) involving the alpha3/alpha4/alpha5 network of type IV collagen (Thorner, 2007).

Clinical/History	Body mass index (BMI)	Input and output (I & O)
Height	Diet history	
Weight		

Edema-free adjusted body weight (aBWef)
Edema, ankles and feet
Abnormal urine color
Hypertension?
Hearing loss before age 30? (males)
Cataracts?
Microscopic hematuria?
Temperature
Renal biopsy, genetic testing

Lab Work

Blood urea nitrogen (BUN)
Creatinine (Creat)
Chloride (Cl_2)
GFR
Creatine clearance (CrCl)
Cholesterol (Chol)—increased?
Albumin (Alb), transthyretin
CRP
Na^+, K^+
Phosphorus
Aspartate aminotransferase (AST)
Ca^{++}
Proteinuria
Hemoglobin and hematocrit (H & H)
Serum Fe, ferritin
Total iron-binding capacity (TIBC), percentage saturation
Parathormone (PTH)
Serum Cu (decreased?)

INTERVENTION

OBJECTIVES

- Reduce renal workload. Improve or control excretion of waste products such as urea and sodium.
- Manage edema resulting from sodium and fluid retention.
- Prevent uremia from nitrogen retention.
- Adjust electrolyte intakes (sodium, potassium, and chloride) if needed.
- Prevent systemic complications, where possible, and protein catabolism, as from poor intake.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Sodium Intake

Assessment: Dx of ATS at age 11, now 20 and away from home at college. Recent admission to hospital with edema and hypertension, despite use of a diuretic each day. Diet history indicates frequent use of canned soups and a bag of chips with a sandwich at lunch every day. Normally uses a lower sodium breakfast and dinner.

Nutrition Diagnosis (PES): NI-5.10.7 Excessive sodium intake related to the use of canned soups, chips, and sandwich for lunch every day as evidenced by recent severe edema and hypertension.

Interventions:

Food-Nutrient Delivery: Low-sodium diet plan for lunch; alternatives identified that are easy to pack and take to college or campus.

Education: Review the requirements for the low sodium diet; has had previous teaching two times by a RD back in hometown.

Counseling: Supportive counseling about food choices that are lower in sodium.

Coordination of Care: Work with medical team to determine whether diuretic is indicated.

Monitoring and Evaluation: Improvement in edema and blood pressure. No further problems following the low sodium diet; change in lunch choices well received.

- Manage hypertension.
- Recommend genetic counseling for those with ATS. A family history and a renal biopsy may be needed (Thorner, 2007).

FOOD AND NUTRITION

- Determine fluid intake (measured output plus 500 mL insensible losses).
- Restrict sodium intake to 2–3 g if patient has hypertension or edema.
- In the case of renal failure, protein intake should be low, as 0.6–0.8 g/kg of adjusted edema-free BW. Use 50% high biological value proteins to ensure positive nitrogen balance. If needed, follow dialysis guidelines.
- Check need for vitamin A, which may be low.
- Decrease elevated phosphorus levels with a low-phosphate diet (5–10 mg/kg/d) as needed.
- Provide adequate energy intake (35 kcal/kg BW).
- Use fish oils to reduce inflammation.

Common Drugs Used and Potential Side Effects

- Antihypertensive, antibiotic, and immunosuppressive medications are commonly used. Monitor for specific side effects, especially with long-term use.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Discuss the varied sources of fish oil supplements and how much to take.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Ensure dietary measures are appropriate for patient's current status.
- Discuss ways to include more omega-3 fatty acids and fish in diet.

Patient Education—Food Safety

- Foodborne illness can occur when there is contamination of food at any point during the preparation process. Because renal patients are at high risk for foodborne illness, follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill.
- Details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- Alport Syndrome Foundation
  https://www.alportsyndrome.org/
- Hearing Loss
  http://www.entnet.org/

ALPORT SYNDROME AND NEPHRITIS—CITED REFERENCES

Haas M. Alport syndrome and thin glomerular basement membrane nephropathy: a practical approach to diagnosis. *Arch Pathol Lab Med.* 133:224, 2009.

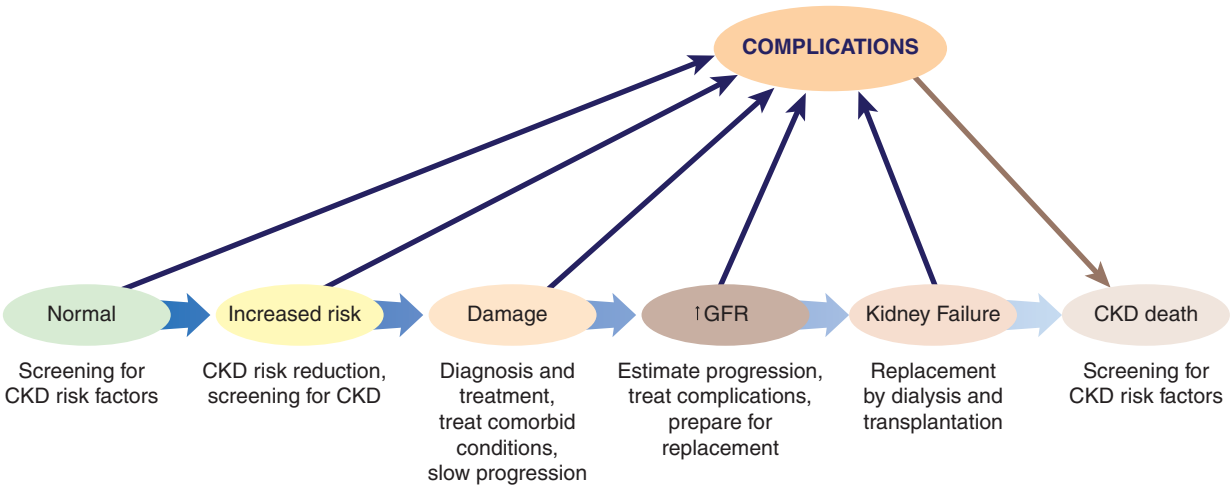
Longo I, et al. Autosomal recessive Alport syndrome: an in-depth clinical and molecular analysis of five families. *Nephrol Dial Transplant.* 21:665, 2006.

Tan R, et al. Alport retinopathy results from "severe" COL4A5 mutations and predicts early renal failure. *Clin J Am Soc Nephrol.* 5:34, 2010.

Thorner PS. Alport syndrome and thin basement membrane nephropathy. *Nephron Clin Pract.* 106:82, 2007.

CHRONIC KIDNEY DISEASE AND RENAL FAILURE

NUTRITIONAL ACUITY RANKING: LEVEL 4



DEFINITIONS AND BACKGROUND

CKD is characterized by the inability of kidney function to return to normal after acute kidney failure or progressive renal decline from disease. Excess urea and nitrogenous wastes accumulate in the bloodstream (azotemia). CKD causes permanent reduction in function, eventually leading to end-stage ESRD. Early stages of CKD are defined by GFR and urinary albumin:creatinine ratio (Castro and Coresh, 2009). Table 16-3 describes the five stages of CKD.

Approximately 23 million Americans have some degree of CKD. Fifty percent of individuals with CKD have diabetes; hypertension is the second highest risk factor. Other risk factors include cardiovascular disease, family history of CKD, use of certain medications, and age >60 years. Undiagnosed CKD is especially common in people who have diabetes. The Pima Indians of Arizona have the world's highest incidence of type 2 diabetes and ESRD. Incorporating estimated GFR into screening for CKD would identify individuals earlier and enable early effective treatment (Middleton et al, 2006).

Depending on the form of disease, renal function may be lost in a matter of days or weeks, or may deteriorate slowly and gradually over the course of decades. Potentially modifiable risk factors in CKD include proteinuria, hypertension, dyslipidemia, anemia, oxidative stress, infections, depression, hyperglycemia, bone disease, and obesity. A low-protein diet (LPD) and low phosphorus intake may retard the progression of kidney disease and should be used in the renal population (Kent, 2005). Studies have shown that LPDs can reduce patient morbidity, preserve renal function, relieve uremic symptoms, improve nutritional status, postpone the start of dialysis for 6 months, and entail substantial cost-savings (Eyre et al, 2008).

Acute renal failure (ARF) involves abrupt decline in renal function with waste retention. ARF occurs when the kidneys fail to function because of circulatory, glomerular, or tubular deficiency resulting from an abrupt cause. ARF is caused by diabetes or hypertension in most cases. Other causes include glomerulonephritis (GN), polycystic kidney disease (PKD), and other causes (burns, severe crush injuries, transfusions, antibiotics, nephrotoxicity from drugs such as tacrolimus or cyclosporine, surgery or anesthesia, cardiac transplantation, shock, or sepsis). In children, ARF has been caused by hemolytic uremic syndrome from a specific strain of *Escherichia coli* (0157:H7) that is associated with undercooked ground beef. ARF occurs also in approximately 5% of surgical or trauma cases; frequently, this is reversible. Intermittent HD and continuous RRT are equally beneficial for patients with ARF (Pannu et al, 2008). In ARF, the patient gradually improves, although some loss of function may be permanent. If toxic accumulation occurs, ARF may be fatal. The phases of ARF include the following:

- Prodromal phase—varies in duration depending on cause—urine output may be normal
- Oliguric (average 10–14 days)—output typically between 50 and 400 mL/d
- Postoliguric phase (average 10 days)—urine output gradually returns to normal
- Recovery (from 1 month or up to 1 year).

TABLE 16-3 Stages, Symptoms, and Preventive Measures for Chronic Kidney Disease (CKD)

Stage	Glomerular Filtration Rate (GFR)	Symptoms and Preventive Measures
Stage 1	≥90 mL/min/1.73 m^2	Kidney damage with normal or increased GFR occurs. Blood flow through the kidney increases (hyperfiltration), and the kidneys are larger than usual. A person with stage 1 CKD usually has no symptoms. Regular testing for protein in the urine and serum creatinine can show whether the kidney damage is progressing. Living a healthy lifestyle can help slow the progression of kidney disease.
Stage 2	60–89 mL/min/1.73 m^2	A person with stage 2 CKD has kidney damage with a mild decrease in GFR. Filtration rate remains elevated or nearly normal. Glomeruli show signs of damage. Blood pressure is usually normal. Albuminuria is <30 mg/d. If people find out they have stage 2 CKD, it is usually because they were being tested for diabetes or high blood pressure.
Stage 3	30–59 mL/min/1.73 m^2	A person with stage 3 CKD has kidney damage with a moderate decrease in the GFR. Microalbuminuria becomes constant. Losses increase to 30–300 mg/d. This can occur after about 7 years of having diabetes. As kidney function declines, uremia occurs. Complications such as high blood pressure, anemia, and early bone disease may occur. Consult a nephrologist to perform specific lab tests. Limit protein from diet to 0.8 g/kg.
Stage 4	15–29 mL/min/1.73 m^2	A person with stage 4 CKD has advanced kidney damage with a severe decrease in the GFR. Nephropathy occurs with passage of large amounts of protein in the urine (>300 mg/d); blood pressure continues to rise. Creatinine rises >1.1–1.3 mg/dL and waste products build up (uremia). New symptoms include nausea, taste changes, uremic breath, anorexia, difficulty concentrating, and numbness in fingers and toes. Visits to the nephrologist every 3 months will be needed to test for creatinine, hemoglobin, calcium, and phosphorus levels and for management of hypertension and diabetes. An arteriovenous (AV) fistula and AV graft are created surgically and need a few months or so to mature before dialysis is needed. By doing everything possible to help prolong kidney function and overall health, the goal is to put off dialysis or transplantation for as long as possible. Limit protein to 0.8 g/kg.
Stage 5	<15 mL/min/1.73 m^2	A person with stage 5 CKD has end-stage renal disease where kidney failure occurs. The kidneys have lost all their ability to do their job effectively. Renal replacement therapy (RRT) is initiated with dialysis or renal transplantation. Dialysis is started when renal Kt/V (urea) falls below 2.0/wk or when kidney function is 15% or less. New symptoms that can occur in stage 5 CKD include anorexia, nausea or vomiting, headaches, fatigue, anuria, swelling around eyes and ankles, muscle cramps, tingling in hands or feet, and changing skin color and pigmentation.

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National Kidney Foundation. Clinical practice guidelines for nutrition in chronic renal failure. Accessed February 15, 2010, at http://www.kidney.org/professionals/kdoqi/guidelines.
Nephron Online. Web site accessed February 16, 2010, at http://www.nephronline.org/management/ckd.html

Chronic renal failure (CRF) is the slow, gradual loss of kidney function. Causes include lupus, ATS, chronic hypertension, prolonged urinary obstruction, nephrotic syndrome, PKD, cystinosis, or diabetes. Diabetic nephropathy may be delayed by tightly controlling blood glucose levels and using angiotensin-converting enzyme (ACE) inhibitors.

Some forms of CRF can be controlled or slowed down but never cured. Partial loss of renal function means that some of the patient's nephrons have been scarred and cannot be repaired. In patients with progressive CRF who consume uncontrolled diets, progressive declines in spontaneous protein and energy intake, serum proteins, cholesterol, total creatinine excretion, and anthropometric values are evident below a CrCl of approximately 25 mL/min. Mortality increases greatly with serum albumin levels below 3.5 g/dL and low cholesterol level (<150–180 mg/dL).

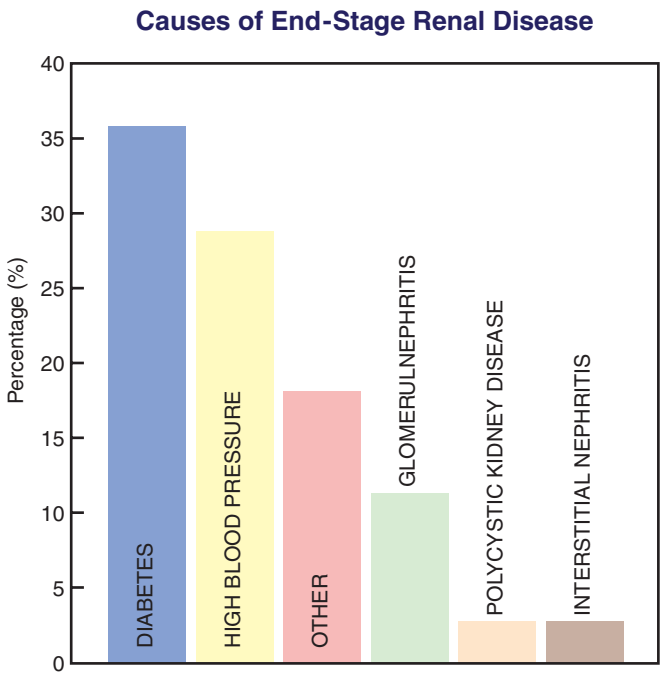
Chronic inflammation predicts mortality in the CKD population; IL-6 is a significantly better predictor of mortality than CRP, albumin, or TNF-α (Barreto et al, 2010).

Most CKD patients also have elevated serum total homocysteine (tHcy) and low selenium levels. Suggest a multivitamin supplement that contains 800 μg folic acid, adequate vitamins B_6 and B_{12} and selenium if needed. Glutathione peroxidase helps prevent generation of free radicals and decreases the risk of oxidative damage to tissues, including the kidney and its vascular supply.

The African American Study of Kidney Disease and Hypertension (AASK) supports tight blood pressure control (<130/80 mm Hg) in CKD. Adherence to practices known to be of clinical benefit not only improves patient outcomes but also reduces costs of care. A multidisciplinary effort is needed.

ESRD is stage 5 CKD. Care involves early detection of progressive renal disease, interventions to retard its progression, prevention of uremic complications, control of related conditions, adequate preparation for RRT, and timely initiation of dialysis. Higher blood pressure and lower income are associated with a higher incidence of ESRD in both white and African American men. Mortality is remarkably high in ESRD, usually from cardiovascular disease (Shen et al, 2006). Decreasing complications may reduce death rates.

Wasting and protein–energy malnutrition (PEM) are complications of ESRD. Cachexia from anorexia, acidosis, and inflammation can lead to the **Malnutrition-Inflammation Complex Syndrome**. The decline in nutritional status may be caused by disturbances in protein and energy metabolism, hormonal derangement, and spontaneous reductions in dietary energy and protein intake. Factors that can worsen PEM include poor dentition, infections or sepsis,



multiple medications, and pain. Worsening of PEM over time is associated with a greater risk for cardiovascular death form the **cardiorenal syndrome.**

Other CKD complications that should be managed include metabolic acidosis due to reduced acid (hydrogen ion) excretion, anemia due to impaired erythropoiesis and low iron stores, hyperkalemia, and dyslipidemia. In addition, secondary hyperparathyroidism (HPT) and **bone and mineral metabolism disorder** may start as early as stage 2 or 3 CKD, and dietitians must monitor vitamin D and calcium levels and restrict dietary phosphorous intake (Aase, 2010). Vitamin D receptor activation improves endothelial function in CKD, and this area of research is very promising (Wu-Wong et al, 2010).

The Kidney Early Evaluation Program (KEEP) is a free screening program designed to detect CKD early, promote follow-up evaluation with clinicians, and ultimately improve outcomes (Vassolotti et al, 2010). The program screens individuals with diabetes, hypertension, or those with a first-degree relative with diabetes, hypertension, and/or kidney disease. African Americans, Native Americans, Hispanics, and Asians/Pacific Islanders have a 3 times higher risk for CKD and anemia (McFarlane et al, 2008; Vassolotti et al, 2010). Such health disparities must be addressed (NKF, 2009).

Management of CKD is costly, yet adverse effects can be prevented. Early nutritional intervention may delay or prevent rapid progression of many complications. KDOQI guidelines recommend that patients on dialysis achieve an albumin level of 3.7 g/dL. SGA scores and other nutritional indicators, such as BMI, handgrip strength for measures of muscle mass, waist circumference, serum albumin level, and serum creatinine level are a good place to start; see Table 16-4. All patients with diabetes and CKD should receive nutritional interventions (NKF, 2007).

Current EBGs should always be followed. The NKF-KDOQI guidelines address strategies for diabetes, hypertension, dyslipidemia, bone disease, anemia, and heart disease in CKD patients. Guidelines are also available for managing CKD in children.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Potential markers of CKD include asymmetric dimethylarginine, factors involved in calcium–phosphate metabolism, adrenomedullin, A-type natriuretic peptide, N-terminal pro-brain natriuretic peptide, liver-type fatty acid–binding protein, kidney injury molecule 1, neutrophil gelatinase-associated lipocalin, apolipoprotein A-IV, adiponectin, and genetic polymorphisms (Kronenberg, 2009). Apolipoprotein E polymorphisms are relevant, with e2 allele conferring risk and e4 providing protection; these associations differ between ethnic groups (Chu et al, 2009).

Clinical/History

- Height
- Weight
- aBWef
- Pitting edema in hands and legs
- BMI
- Waist circumference
- Waist:hip ratio
- Diet history
- BP (increased)
- I & O
- Severe headaches
- Dyspnea
- Failing vision
- Poor appetite
- Nausea, vomiting
- Abdominal pain
- Mouth ulcers, hiccups
- Bone and joint pain
- Fatigue
- Pale skin
- Uremic convulsions
- Pericarditis
- Skin changes and pigmentation
- Body composition (SGA score)
- Electrocardiogram
- Renal sonography
- Renal biopsy
- Dual-energy x-ray absorptiometry (DEXA) scan

Lab Work

- Urine flow: Normal, 1–1.5 L/d
  - Nonoliguria, >500 mL/d
  - Oliguria, <500 mL/d
  - Anuria, <100 mL/d
  - Polyuria, 3 L/d
- Azotemia (excess urea and nitrogen in blood)
- Urine albumin:creatinine ratio
- Alb (goal, >4.0 g/dL)
- Transferrin saturation (goal, >20%)
- CRP
- IL-6
- BUN
- Creat
- BUN:creatinine ratio
- GFR (if <10–15 mL/min, consider dialysis)
- Glucose (goal, <140–160 mg/dL random)
- Hemoglobin A1c (HbA1c) (goal, <7%)
- Na^{+}
- K^{+} (goal, 3.5–5.5 mEq/L)
- Ca^{++} (goal, 8.5–10.2 mg/dL)
- Phosphorus (increased?)
- Uric acid (increased?)
- Mg^{++} (increased?)
- Serum bicarbonate
- PTH
- Hemoglobin (goal, 12 g/L for men; 11 g/L for women)

TABLE 16-4 Protein–Energy Malnutrition (PEM) in Renal Patients

Etiology	Comments
Blood loss	Loss of blood may be due to gastrointestinal bleeding, frequent blood sampling, and blood sequestered in the hemodialyzer and tubing.
Dialysis	The process promotes wasting by removing such nutrients as amino acids, peptides, protein, glucose, water-soluble vitamins, and other bioactive compounds, and may promote protein catabolism, due to bioincompatibility.
Endocrine changes	Endocrine disorders of uremia include resistance to the actions of insulin and IGF-I, hyperglucagonemia, and hyperparathyroidism.
Inadequate food intake	Poor intake is secondary to anorexia caused by the uremic state; altered taste sensation; intercurrent illness; emotional distress or illness; impaired ability to procure, prepare, or mechanically ingest foods; and unpalatable prescribed diets.
Inflammation and the catabolic response	Chronic inflammation from cardiovascular disease, diabetes mellitus, and other illnesses may produce anorexia, hypercatabolism, and malnutrition. Diminished appetite relates to high concentrations of proinflammatory cytokines. Inflammatory cytokines, such as tumor necrosis factor-α and interleukin-6, are associated with protein synthesis and catabolism in the body, and they downregulate albumin synthesis. Serum albumin is altered by systemic inflammation; a low serum albumin concentration is strongly associated with mortality in CKD.
Other issues	Sleep disturbances, pain, erectile dysfunction, patient dissatisfaction with care, depression, symptom burden, and perception of intrusiveness of illness may all lead to poor quality of life, resulting in poor appetite and intake. Suggest psychotherapy, antidepressants, or other treatments.
Oxidative stress	PEM and low body mass index may be associated with increased oxidative stress and impaired endothelium-dependent vasodilation with reduced bioavailability of nitric oxide. Elevated C-reactive protein is a risk factor for mortality in chronic kidney disease (Menon et al, 2005).
Toxins	There may be accumulation of endogenously formed uremic toxins or the ingestion of exogenous toxins.
Wasting	Wasting may be due to anorexia, nausea, emesis, uremia, inflammation, infections, diabetes, underdialysis, or dental problems. Supplementation with branched-chain amino acids spares lean body mass during weight loss, promotes wound healing, may decrease muscle wasting with aging, and may have beneficial effects in renal disease (Tom and Nair, 2006).
Assessments	**Comments**
Lab values—BUN: creatinine ratio	BUN:creatinine ratio is altered by catabolic stress, low urine volume, and muscle mass changes.
Lab values—creatinine	Creatinine is a waste product that comes from muscle activity. When kidneys are working well, they remove creatinine from the blood. As kidney function slows, blood levels of creatinine rise. Serum creatinine concentration reflects muscle mass, somatic protein stores, and dietary protein intake. Because it predicts outcome in CKD, it may be a useful marker of nutritional status or loss of muscle mass in CKD. However, creatinine levels are affected by inflammation, age, sex, race, residual kidney function, variation in creatinine metabolism, and dialysis. The assessment of dietary intake is more commonly used to assess protein status.
Lab values—creatinine index	The creatinine index is directly related to the normalized protein equivalent of total nitrogen appearance (nPNA) and independent of the dialysis dose. A low or declining creatinine index correlates with mortality independently of the cause of death. People with catabolic diseases may have larger and faster declines in the creatinine index before death.
Lab values—glomerular filtration rate (GFR)	GFR is a good measure of kidney function. A math formula using the person's age, race, gender, and serum creatinine is used to calculate the GFR.
Lab values—nitrogen balance	Because the nitrogen content of protein is relatively constant at 16%, the protein equivalent of total nitrogen appearance (PNA) can be estimated by multiplying total nitrogen appearance (TNA) by 6.25 (PNA is mathematically identical to the protein catabolic rate or PCR). In the clinically stable patient, PNA can be used to estimate protein intake. However, PNA approximates protein intake only when the patient is in nitrogen equilibrium (steady-state). In the catabolic patient, PNA will exceed protein intake to the extent that there is net degradation and metabolism of endogenous protein pools to form urea.
Lab values—serum cholesterol	Low serum cholesterol is an indicator of chronically inadequate protein–energy intake. It may be an indicator for mortality in maintenance hemodialysis patients.
Lab values—transthyretin (prealbumin)	Transthyretin is a good index of liver protein synthesis, but it is reabsorbed and metabolized by the proximal tubule; serum levels rise as kidney function declines. Transthyretin levels correlate strongly with serum albumin.

(continued)

TABLE 16-4 Protein–Energy Malnutrition (PEM) in Renal Patients *(continued)*

Assessment Frequency	Suggested Evaluation for PEM Status
Monthly assessments	Predialysis or stabilized serum albumin
	Percentage of usual postdialysis (MHD) or postdrain (CPD) body weight
	In hemodialysis: nPNA
Every 3–4 months	In peritoneal dialysis: nPNA
Every 4 months	Percentage of standard (NHANES II) body weight
Every 6 months	Subjective global assessment
	Dietary interview and/or diary
As needed to confirm or extend the data obtained earlier	Predialysis or stabilized serum prealbumin
	Skinfold thickness
	Midarm muscle area, circumference, or diameter
	Dual-energy x-ray absorptiometry
If low, to identify the need for more rigorous examination	Low predialysis or stabilized serum creatinine level (<10 mg/dL)
	Low or declining creatinine index
	Low predialysis or stabilized serum urea nitrogen level
	Low predialysis or declining serum cholesterol level (150–180 mg/dL)

Developed using:
- KDOQI Guidelines. Web site accessed February 23, 2010, at http://www.kidney.org/Professionals/kdoqi/guidelines_updates/doqi_nut.html.
- Menon V, et al. C-reactive protein and albumin as predictors of all-cause and cardiovascular mortality in chronic kidney disease. *Kidney Int.* 68:766, 2005.
- Tom A, Nair KS. Assessment of branched-chain amino acid status and potential for biomarkers. *J Nutr.* 136:324S, 2006.

Serum Fe, serum ferritin
Mean cell volume (MCV)
Red blood cell (RBC) folate (≥200 ng/mL)
Serum 23-hydroxyvitamin D (goal, >30 ng/mL)
pH
CO_2 (goal, 24–32 mEq/L)
Serum chloride
Chol (goal, >160–<200 mg/dL)
Triglycerides (goal, <150 mg/dL)

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Enteral Nutrition Infusion

Assessment Data: Dietary recall and I & O records, nursing flow sheets. Renal lab values: K+ 6.0 mEq/L, BUN 60 mg/dL, serum creat 1.5 mg/dL. On tube feeding with fluid restriction.

Nutrition Diagnosis (PES): NI-2.3 Inadequate intake from enteral nutrition (EN) infusion related to volume restriction as evidenced by recorded infusion of 900 mL of enteral formula over the past 24 hours providing only 70% of estimated energy and protein needs.

Interventions:

Food-Nutrient Delivery: Increase tube feeding by concentrating formula and increasing protein.

Coordinate Care: Work with the medical team to monitor for tolerance and lab changes.

Monitoring and Evaluation: Monitor by I & O intake to monitor change in enteral formula administration, output, and serum creatinine levels.

INTERVENTION

OBJECTIVES

- Postpone dialysis as long as possible. Work with high-risk conditions, such as diabetes or when urine albumin:creatinine ratio is abnormal (>30). Start working with other patients when serum creatinine is >1.5 mg/dL (women) or >2.0 mg/dL (men) to limit further renal impairment and reduce kidney workload. Patients should see a renal team, including a dietitian.
- Maintain or improve nutritional status. Protect lean body mass, minimize tissue catabolism. Consume adequate calories to spare protein.
- Keep protein intake at 0.6–0.75 g/kg/d to postpone dialysis (Eyre et al, 2008). Provide amino acids in proportion to protein status; attempt to keep serum albumin level of 4.0 g/dL.
- Control uremic symptoms and reduce complications from accumulation of nitrogenous waste. Evaluate the BUN: creatinine ratio; typically, a 10:1 ratio is desirable (urinary creatinine doubles when renal function is <50%).
- Treat hypertension aggressively. Limit dietary sodium, use moderate alcohol intake, obtain regular exercise, lose weight in those with a BMI >25, and reduce saturated fats. Blood pressure goal is <130 mm Hg systolic and <80 mm Hg diastolic for all CKD patients.
- Keep blood glucose under control with HbA1c ≤ 7%. Control carbohydrate (CHO) intake if needed.
- Include a variety of grains, especially whole grains, fresh fruits, and vegetables. Limit intake of refined and processed foods high in sugar and sodium.

- Choose low saturated fat, low cholesterol, and moderate total fat foods to normalize lipid profiles.
- Restore and maintain electrolyte balance; correct acidosis. Individualize potassium and phosphorus plans if blood levels are high.
- Consume the dietary recommended intake (DRI) for vitamins and minerals; preventing osteodystrophy.
- Have regular checkups and take medicines as prescribed. Exercise regularly, stop smoking, and eat a healthy diet.
- Aim for a healthy weight and include physical activity each day; obesity causes a decline in kidney function.
- Treat anemia, defined as a hematocrit <36% in women and <39% in men.
- Maintain growth in children with adequate calories, vitamins, and minerals.
- Correct other physiological changes such as constipation, diarrhea, blurred vision, pruritus, ecchymosis, pallor, crackles in the lungs, loss of muscle tone, and tingling of lips or fingertips.

FOOD AND NUTRITION

- Consume adequate calories. Nondialyzed patients younger than 60 years with advanced CKD (GFR <25 mL/min) need 35 kcal/kg/d; those older than 60 years need 30–35 kcal/kg.
- CHO should be 50–60% of total calories per day (Burrowes, 2008). CHO intolerance is common; fructose, galactose, or sorbitol may be better tolerated than sucrose.
- With diabetes or heart disease, limit total fat to 30% or less of total calories per day; saturated fat less than 10% of the total calories; 200 mg cholesterol per day (Burrowes, 2008).
- In stages 1, 2, or 3 of CKD, limit protein to 12–15% of calories per day. Use 0.8 g to 1.0 g protein/kg BW from high-quality protein sources.
- In stage 4 CKD, reduce protein to 10% of calories per day. For nondialyzed patients with GFR <25 mL/min, use 0.6 g protein/kg/d, 50% from high–biological value sources. Amino acid analogs (CHO skeleton of amino acids minus the amino group) may also be used.

TABLE 16-5 Spices and Condiment Substitutes for Salt

Allspice	Dry mustard	Paprika
Bay leaf	Garlic, fresh or powder	Rosemary
Black pepper	Ginger	Scallions
Caraway seeds	Green bell pepper	Shallots
Celery seed	Lemons and limes	Sugar substitute
Chili powder	Mint	Sweet basil
Chives	Mrs. Dash	Tabasco sauce
Cinnamon	Nutmeg	Thyme
Cloves	Onions	Turmeric or cumin
Curry powder	Oregano	Vanilla extract
Dill	Pan spray, nonstick	Vinegar

- Limit sodium to 2.3 g/d for those with diabetes, high blood pressure, or fluid retention. Limit intake of processed foods high in sodium. Use less salt at the table and fewer high-sodium ingredients. See Table 16-5 for seasoning ideas instead of salt.
- Phosphorus and potassium are modified on the basis of the stage of CKD (Burrowes, 2008):
  Phosphorus: stages 1–2: 1.7 g/d; stages 3–4: 0.8–1.0 g/d
  Potassium: stages 1–2: less than 4 g/d; stages 3–4: 2.4 g/d

See Table 16-6 for tips on managing these potassium and phosphorus in the diet. Limit use of salt substitutes. Liberalize K+ when there is diarrhea or vomiting.

- Limit elemental calcium to <2000 mg, including that from phosphorus binders, as needed. Initiate vitamin D therapy if PTH is high, calcium is <9.5 mg/dL, or phosphorous is <4.6 mg/dL.
- Fluid intake should be output plus 500–1000 mL for insensible losses. Monitor regularly.
- Avoid over-the-counter dietary supplements unless approved by the nephrologist. Supplement with the DRI for the water-soluble vitamin B complex, and limit vitamin C to 100 mg/d from supplemental sources. Vitamin A and iron may not be desirable, levels can build up as kidney function declines.
- EN significantly increases serum albumin concentrations and improves total dietary intake (Stratton et al, 2005). Renal-specific enteral products may be needed; monitor content according to lab data. Because EN can help infants and children overcome malnutrition and promote catch-up growth, noctural feedings may be useful. If central parenteral nutrition (CPN) is needed, avoid excesses of micronutrients.
- Work with the interdisciplinary team, as shown on page 871.

Common Drugs Used and Potential Side Effects

- See Table 16-7.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician. Some products may actually contribute to renal failure, such as aristolochic acid, found in Mu Tong or Fangchi in Taiwanese prescription medicines (Lai et al, 2010).
- Probiotic dietary supplements are being designed to metabolize nitrogenous waste that has diffused from the bloodstream into the bowel.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Counseling interventions should include aggressive blood pressure control, reduction of dietary protein to

TABLE 16-6 Tips for Managing Potassium and Phosphorous in the Diet

Most high-potassium foods come from plants (fruits and vegetables). High-phosphorus foods are mainly from animals (meats, poultry, fish, eggs, cheese, dairy foods), but dried beans and peas also tend to be high in phosphorus. Foods that are high in both potassium and phosphorus are dairy foods, nuts, seeds, chocolate, and whole grain foods.

Category	Tips and Alternatives for Lowering Potassium (K+)
Beverages	Drink ice water with sliced lemon and cucumber, instead of drinking tomato or vegetable juices.
Cooking	Cook with onion, bell peppers, mushrooms, or garlic, instead of tomatoes, tomato or chili sauces.
Desserts and snacks	Choose vanilla- or lemon-flavored desserts, instead of chocolate desserts. A plain donut is acceptable. Choose unsalted popcorn or pretzels, rice cakes, jelly beans, red licorice, or hard candies. Avoid products made with molasses and nuts.
Fruit	Choose apples, berries, canned peaches, canned pears, dried cranberries, fruit cocktail, grapes, pineapple, and plums. Avoid bananas, oranges, cantaloupe, honeydew, dried fruits, mango, or kiwi.
Fruit juice	Drink apple, cranberry, pineapple, or grape juice, instead of orange or prune juice.
Potatoes	Leach potatoes by cooking in water first to lower the potassium content. Prepare mashed potatoes or hash browns from leached potatoes instead of eating a baked potato, sweet potatoes, yams, or French fries.
Pumpkin or squash	Use summer squashes such as crookneck or zucchini, instead of pumpkin or winter squashes (acorn, banana, hubbard).
Seasonings	Season with pepper, lemon, or low-sodium herb and spice blends, instead of salt substitutes or seeds.
Vegetables	Choose green beans, wax beans, carrots, cabbage, cauliflower, or snow peas, instead of spinach, dried beans or peas.
Vegetables for dips, salads	Prepare salads and vegetable appetizers with cucumber, cauliflower, eggplant, lettuce and sweet peppers. Avoid avocado and artichokes.
High K+ and High Phosphorus	**Alternatives for a Lower K+/Phosphorus Diet**
Cheese	Vegan rella cheese, low-fat cottage cheese, sprinkle of parmesan cheese; very small amounts of extra sharp cheeses for the maximum flavor
Chocolate	Desserts made with lemon or apple, white cake, rice-crispy treats
Cream soup	Broth-based soups made with pureed vegetables or make soups with Mocha Mix® nondairy creamer, Rich's Coffee Rich®, or Dairy Delicious® milk
Dried beans and peas	Green beans, wax beans
Ice cream	Mocha Mix® frozen dessert, sorbet, sherbet, popsicles
Milk	Dairy Delicious® milk, Mocha Mix® nondairy creamer, Coffeemate®, Rich's Coffee Rich®, Rice Dream® original, unenriched rice beverage
Nuts	Low-salt snack foods including pretzels, tortilla chips, popcorn, crackers, Sun Chips®
Peanut butter	Low-fat cream cheese, jam, or fruit spread

Derived from: Davita Dialysis Centers, http://www.davita.com/diet-and-nutrition/c/diet-basics, accessed February 22, 2010.

recommended levels, weight loss, and control of hyperlipidemia. Referral to a qualified dietitian is important.

- For diabetic kidney disease (DKD), provide tips on controlling CHOs from the diet. The NKF recommends intense measures to manage hyperglycemia, hypertension, and dyslipidemia (NKF, 2007). Maintain HbA1c <7.0% regardless of the stage of CKD. Keep high blood pressure controlled (<130/80 mm Hg); low-density lipoprotein (LDL) cholesterol <100 mg/dL; dietary protein intake at 0.8 g/kg BW per day. Avoid high-protein diets with 20% of calories from protein.
- As eGFR declines, renal metabolism of insulin and oral medication is reduced, potentially causing hypoglycemia; cranberry juice cocktail, grape, apple juice, or glucose tablets may be helpful (Aase, 2010).
- Teach the Dietary Approaches to Stop Hypertension (DASH) diet that emphasizes sources of protein other than red meat for hypertension, diabetes, and CKD stages 1–2 (NKF, 2007).
- Discuss reading food labels, measuring portions, reading restaurant menus, planning box lunch foods, and dining away from home.
- LPDs are safe when properly planned. They reduce the accumulation of metabolic products and can suppress progressive loss of kidney function (Mitch, 2005). Have patient consume the designated proteins throughout the day. Low-protein wheat starch, hard candy, and jelly can be used to increase calories.
- Foods with sharp, distinct flavors may be needed. Lack of interest in red meats is common, and taste changes can occur.

INTERDISCIPLINARY NUTRITION CARE PLAN
End-Stage Renal Disease (Hemodialysis)

Client Name: _____ **#:** _____ **Initiated by:** _____ **Date:** _____

SCREENING
Nutrition Screen diagnosis: End-Stage Renal Disease (or dialysis)
Signed: _____ Date: _____

ASSESS *(Check any/all)*
Blood chemistries
❑ Serum albumin
❑ Serum transferrin, iron, or ferritin
❑ Total iron-binding capacity (TIBC)
❑ Serum ferritin
❑ Hematocrit, hemoglobin
❑ RBC indices, reticulocyte count
❑ BUN, creatinine
❑ Potassium/phospohorus, calcium
❑ Glucose
❑ Other:_____
Weight/BMI
❑ Weight loss >3 lb/wk or > 5%/mo or >10%/6 mo
❑ Weight gain >2 lb/d (fluid weight gain)
❑ BMI <20 (High Risk)
Poor Oral Intake Symptoms
❑ Complex diet order
❑ Nausea/vomiting
❑ Poor appetite/early satiety
❑ Problems chewing/swallowing
❑ Depression/anxiety
❑ GI distress
Signed: _____ Date: _____

1 or more

HIGH-RISK INTERVENTIONS *(Check any/all)*
❑ **Hemodialysis and Your Renal Diet** provided and reviewed
Obtain Dr. orders as needed:
❑ RD referral for home visit(s)
❑ Social Services referral for home visit(s)
❑ Labs: _____
❑ Monitor weight q:_____
❑ Medication adjustments
❑ BID/TID supplement or sole source
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
❑ Abnormal blood chemistries
❑ Continued Poor Oral Intake Symptoms
❑ Weight change not appropriate per goal
❑ Declining strength
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

1 or more

OUTCOMES NOT ACHIEVED
Reassess acuity/evaluate need for further nutrition support. Document on Nutrition Variance Tracking form.

GOALS (Check any/all):

❑ Maintain or improve nutritional status in ____ (goal time).

❑ Improve serum albumin in ____ (goal time).

❑ Maintain or improve adherence to renal diet in ____ (goal time).

Weight ❑ maintained, or ❑ loss/ ❑ gain of ____ lb in ____ (goal time).

None

MODERATE-RISK INTERVENTIONS *(Check any/all)*
❑ **Hemodialysis and Your Renal Diet** provided and explained
Obtain Dr. orders as needed:
❑ RD chart consult
❑ Social Services chart consult
❑ Monitor blood chemistry
❑ Monitor weight q:_____
❑ Medication adjustments
❑ BID/TID supplement or sole source
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
❑ Abnormal blood chemistries
❑ Exhibiting Poor Oral Intake Symptoms
❑ Weight change not appropriate per goal
❑ Declining strength
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

1 or more

None

OUTCOMES ACHIEVED
❑ Weight maintained or improved
❑ Adherence to renal diet
❑ Normal blood chemistries
❑ **Other:**_____
(See notes for documention.)
❑ Repeat Nutrition Screen in ____ days
Signed: _____ Date: _____

None

OUTCOMES ACHIEVED
❑ Weight maintained or improved
❑ Adherence to renal diet
❑ Normal blood chemistries
❑ **Other:**_____
❑ Repeat Nutrition Screen in ____ days
(See notes for documention.)
Signed: _____ Date: _____

Adapted with permission from www.RD411.com, Inc.

TABLE 16-7 Drugs Used in Chronic Kidney Disease (CKD) and Dialysis Patients

Medication	Comments
Angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker	First-line pharmacological intervention should be an ACE inhibitor or angiotensin receptor blocker in those with >200 mg protein/g creatinine on a urine sample.
Antidepressants	Depression is common in this population. Antidepressant treatment may be needed to improve appetite and intake.
Carnitine	Carnitine is formed from lysine and methionine, requiring adequate vitamin C, niacin, iron, and vitamin B_6. Red meat and dairy products are typical dietary sources. Because the kidneys cannot make it, carnitine supplements are sometimes recommended.
Cinacalcet	**Cinacalcet (Sensipar)** has been approved to treat secondary hyperparathyroidism.
	Cinacalcet can be used to lower PTH despite elevations in calcium and/or phosphorus. Parathyroidectomy may be needed to lower PTH when pharmacologic intervention fails.
Growth hormone	Pharmacological doses of recombinant human growth hormone constitute an effective anabolic therapy.
Insulin	Insulin may be needed to control blood glucose levels. It is an anabolic hormone and can protect lean body mass for some patients.
Iron supplements and epoetin	**Recombinant human erythropoietin (rHuEPO)** is used to treat anemia; an iron supplement will be necessary. Be careful not to overload with excesses, and do not take supplements at the same time as calcium.
	Retacrit (epoetin zeta) is being evaluated for intravenous administration.
Lipid-lowering medications	Lipoprotein metabolism is often impaired in CKD. Patients with low-density lipoprotein ≥100 mg/dL should be treated with diet and a statin.
Phosphate binders	Phosphate binders offer options for phosphorus reduction; newer binders are available that will not increase serum calcium. Nausea or vomiting may occur.
	Aluminum-containing binders are effective but can accumulate in tissues or may lead to bone disease, encephalopathy, or anemia.
	Calcium-containing binders (gluconate, carbonate, or lactate) may increase calcium intake, which may be otherwise inadequate if milk is limited. They are widely used but can lead to hypercalcemia or calcification.
	Lanthanum carbonate (Fosrenol) has good potency and minimal absorption; long-term studies on tolerability needed. Does not cause hypercalcemia.
	Magnesium carbonate can lead to hypermagnesemia, but long-term studies are lacking. It has the potential to minimize calcium load.
	Sevelamer (Renagel) causes less vascular calcification than calcium binders; it also reduces triglycerides and LDL cholesterol. It is costly and tolerability is variable.
Vitamin supplement	Renal multivitamins (such as Diatx) may be useful in providing B-complex vitamins (especially folic acid, B_6, and B_{12}) for hyperhomocysteinemia and CKD.
Vitamin D	In CKD, the patient's kidney is unable to convert vitamin D to its active form; osteodystrophy can result. Take the supplements with extra water if possible because constipation may be a problem.
	Calcitriol (Calcijex®, Rocaltrol®) is the active vitamin D form that increases calcium and phosphorus absorption; it loses effectiveness with high serum P. The active form should not be taken if calcium or phosphorus levels are too high; it can increase phosphorus deposits in soft tissues such as arteries, lungs, eyes, and skin.
	Vitamin D analogs lower parathyroid hormone and increase bone mineralization but may raise calcium and phosphorus. **Paricalcitol (Zemplar®) and Doxercalciferol (Hectorol®)** are active vitamin D analogs that are less calcemic.

- Check for weights daily. Offer tips for maintaining fluid balance and managing thirst; see Table 16-8.
- Discuss dietary sources of vitamin D (see Appendix A).
- Manage high levels of phosphorus by dietary changes and prescribed medicines.
- Teach the patient how to monitor for signs of hyperkalemia, including nausea, weakness, numbness, tingling, slow pulse, or irregular heartbeat.
- Exercising regularly, maintaining a healthy weight, and not smoking prolong kidney health. Patients should talk to their doctors about an exercise plan.

Patient Education—Food Safety

- Foodborne illness can occur when there is contamination of food at any point during the preparation process.

TABLE 16-8 Tips for Managing Thirst and Fluid Restrictions

Fluid Equivalents	Sample Fluid Content
30 mL = 1 fluid oz = 2 tablespoons	1 whole popsicle = 90 mL
240 mL = 8 fluid oz = 1 cup	4 oz soup = 120 mL
2 cups = 1 lb fluid weight	6 oz juice = 180 mL
2.2 lb = 1 kg fluid weight	8 oz beverage = 240 mL
1 kg fluid weight = 4 cups liquid	12 oz soda = 360 mL 16 oz milkshake = 480 ml

Tips for Managing Thirst

Control high blood glucose; thirst is a side effect of hyperglycemia.

Limit foods that contain hidden or large amounts of fluid (popsicles, soup, gravy, watermelon, and ice cream).

Reduce intake of salty and spicy foods.

Rinse often with mouthwash or suck on a lemon to decrease dry mouth.

Sipping beverages will make them last longer.

Stay cool in warm weather; drink cool instead of warm beverages.

Take medicines with applesauce instead of beverages.

Using ice instead of beverages may seem more satisfying; freeze the allotted amount of beverages (such as fruit juice) and track intake accordingly.

Derived from: Davita Dialysis Centers, http://www.davita.com/diet-and-nutrition/diet-basics/a/477, accessed February 22, 2010.

- To prevent contamination from *E. coli* bacteria (0157:H7) that is associated with undercooked meats, cook until juices run clear (155°F for ground meat or hamburger).
- Because renal patients are at high risk for foodborne illness, follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill. See the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- End-Stage Renal Disease Clinical Performance Measures (CPMs) Project http://www.cms.hhs.gov/CPMProject/
- ESRD Quality Initiative http://www.cms.hhs.gov/ESRDQualityImproveInit/downloads/ESRDOverview.pdf
- KDOQI Guidelines for Managing Diabetes and CKD http://www.ajkd.org/article/S0272–6386(06)01843–9/fulltext
- MEDLINE—ESRD http://www.nlm.nih.gov/medlineplus/ency/article/000500.htm
- NKF—CKD http://www.kidney.org/kidneydisease/ckd/index.cfm
- NKF-KDOQI Guidelines http://www.ajkd.org/content/kdoqiguidelines
- NKF-KDOQI Guidelines for Pediatrics http://www.kidney.org/professionals/KDOQI/guidelines_updates/pdf/CPGPedNutr2008.pdf

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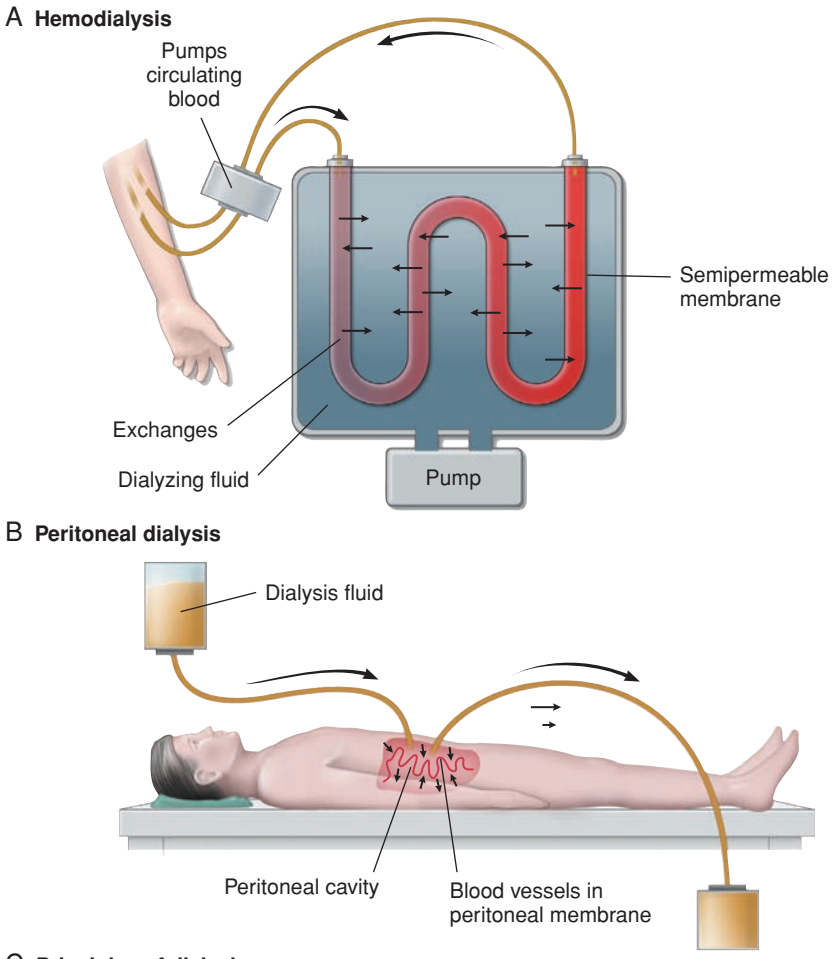
DIALYSIS

NUTRITIONAL ACUITY RANKING: LEVEL 4



Adapted from: Premkumar K. *The Massage Connection Anatomy and Physiology*. Baltimore: Lippincott Williams & Wilkins 2004.

DEFINITIONS AND BACKGROUND

Dialysis specifically involves artificial filtering of blood by a machine. It is a catabolic process. Among patients on dialysis, 33% have diabetes, and 50% of deaths are related to cardiovascular disease. Morbidity is largely related to physical fitness at the start of therapy. Chronic long-term dialysis can aggravate bone disease, anemia, endocrine disorders and can lead to malnutrition if not monitored carefully.

Peritoneal dialysis (PD) involves artificial filtering of the blood by a hyperosmolar solution (with osmosis to remove water and diffusion for glucose exchange/waste removal). The patient and doctor select continuous ambulatory PD (CAPD) or intermittent PD. CAPD does not require a machine. Continuous cycler-assisted PD (CCPD) requires a machine called a *cycler* to fill and drain the abdomen, usually during sleep; this is called *automated PD (APD)*. PD removes metabolic wastes and excess fluid from the body but not so thoroughly that diet therapy is unnecessary. Between dialysis treatments, the patient must return to a strict renal diet. Sometimes alternative methods are required. Intradialytic parenteral nutrition (IDPN) is convenient since it is administered during dialysis treatment. Overweight is more prominent in this population than malnutrition, which occurs frequently in HD patients.

HD is the more common method used to treat advanced and permanent kidney failure. In HD, blood flows through a special filter that removes wastes and extra fluids and then is returned to the body. With HD, less protein is lost than with PD; nevertheless, amino acid losses still occur. Approximately 40% of patients undergoing **maintenance HD** suffer from varying degrees of PEM. Causes of PEM include the catabolic effects of HD treatments, acidemia associated with ESRD, comorbid conditions, and uremia-induced anorexia (Ohlrich et al, 2005). Toxins accumulate with renal failure that suppress appetite and contribute to nutritional decline. Pica (mostly ice but also starch, dirt, flour, and aspirin) has also been found in many dialysis patients.

Nutritional status in these patients should be assessed by a panel of measures rather than by any single measure. Criteria often used to diagnose malnutrition in dialysis patients include serum albumin <3.4 g/dL, average BW <90% of desired goal, or documented protein intake <0.8 g/kg. The NKF Kidney Disease Guidelines for Nutrition provide many clinical practice guidelines for adults and separate guidelines for children.

Higher dietary phosphorus intake and higher dietary phosphorus:protein ratios are each associated with increased death risk in HD patients, even after adjustments for serum phosphorus, phosphate binders and their types, and dietary protein, energy, and potassium intakes (Noori et al, 2010). Control of phosphorus levels must, therefore, be a priority. Nutritional intervention can decrease both malnutrition and mortality in HD. The first step is careful evaluation of protein–energy status, followed by intensive nutrition counseling and then by oral nutrition supplementation, appetite stimulation, or enteral tube feedings (Ohlrich et al, 2005). Prealbumin concentrations <20 mg/dL are a concern, and a fall in serum prealbumin level over 6 months is independently associated with increased death risk (Rambod et al, 2008). However, albumin is more of a marker of illness severity than nutritional status (Friedman and Fadem, 2010). EN or PN support may be needed; PN should be limited to patients who do not respond to other medical, psychiatric, and nutritional interventions.

Anemia can lead to chronic fatigue and debility if not corrected. Recombinant human erythropoietin beta (rHuEPO) is often given to improve nutritional status. Patients who do not respond to erythropoietin replacement may suffer from malnutrition and inflammation (Locatelli et al, 2006). Higher serum hemoglobin levels are associated with fewer hospitalizations and fewer hospital days (O'Connor et al, 2005).

CKD disrupts calcium homeostasis and causes high PTH, low calcitriol, reduced intestinal calcium absorption, low serum calcium, and high serum phosphorus at low GFR. In this secondary HPT, not enough phosphate is cleared from the body; phosphate is released from bone. Vitamin D is not produced. Absorption of calcium in the gut is low, and blood levels of calcium are lowered. Fortunately, treatment of secondary HPT has improved in recent years, and skeletal pain, disabling fractures, tendon ruptures, and other symptoms can be avoided. Parenteral vitamin D is associated with improved survival among long-term HD patients (Shoben et al, 2008).

Dyslipidemia is common; dialysis patients have increased cardiovascular morbidity and mortality. Excessive cardiovascular mortality of dialysis patients is related to chronic inflammation and muscle wasting (Kuhlmann and Levin, 2005; Workeneh and Mitch, 2010). The ATP-dependent ubiquitin–proteasome system causes skeletal muscle wasting not only from inflammation but also from metabolic acidosis, angiotensin II, and defective insulin signaling (Workeneh and Mitch, 2010). Elevated CRP levels can predict cardiovascular mortality. Eventually, 14-kD actin levels can be a marker of excessive muscle wasting (Workeneh and Mitch, 2010).

Children need adequate protein to encourage growth, and an intensive program of dialysis and nutrition intervention can be used in children on maintenance RRT. Monitor potassium and phosphorus restrictions carefully; protein choices are often high in these nutrients. For all patients, monitor trends and not just single biochemical numbers to identify the need for more education and counseling.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Dialysis may be necessary for some of the genetic causes of renal failure.

Clinical/History

- Height
- Weight
- aBWef
- Edema
- BMI
- Diet history
- Waist circumference
- Waist:hip ratio
- BP
- I & O
- Triceps skinfold (TSF)
- Midarm circumference (MAC)
- Midarm muscle circumference (nonaccess arm in HD)

Temperature
DEXA scan

Lab Work

BUN
Creat
GFR
CrCl (on HD, it will be <10)
Urine urea nitrogen (UUN) or Kt/V
Serum phosphorus (goal, <5.5 mg/dL)
CRP (usually elevated)
Serum PTH (goal, 100–200 pg/mL)
Serum vitamin D
Serum Ca^{++} (goal, 9.2–9.6 mg/dL)
Mg^{++}
Gluc, HgbA1c
Transferrin
Serum bicarbonate (low?)
Na^{+}
K^{+} (levels >6 mg/dL can trigger heart failure)
Metabolic acidosis, hyperchloremia
Uric acid
Alb, transthyretin
Urea
Serum folacin
Serum B_{12}
Chol, Trig
AST (decreased)
Hemoglobin
Hematocrit—target of 33–36%
Serum Fe, ferritin
N balance
Serum zinc

INTERVENTION

OBJECTIVES

- Normalize the volume and composition of the body fluids and remove uremic toxins.
- Compensate for lost amino acids without causing uremic symptoms.
- Spare protein adequately to allow for tissue repair and synthesis; ensure sufficient total energy intake. Inflammation may elevate resting energy expenditure levels (Utaka et al, 2005); monitor accordingly.
- In PD only: alter calorie intake according to glucose absorption from the solution (e.g., 20 kcal/L in 1.5% solution; 60 kcal/L in 2.5% solution; and 126 kcal/L in 4.5% solution).

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Mineral Intake

Assessment Data: HD patient returning to clinic; most labs within normal range, but phosphorus level at 7 mg/dL. Diet history indicates increased intake of dairy foods in the past week.

Nutrition Diagnosis (PES): NI-5.1.6 Excessive mineral (phosphorus) intake related to high intake of dairy foods in the past days as evidenced by serum phosphorus level of 7 mg/dL.

Interventions:

Food-Nutrient Delivery: Encourage return to renal dietary protocol.

Education: Review effects of hyperphosphatemia on quality of life.

Counseling: Discuss alternatives to dairy foods and provide recipes, menus, and other tips.

Coordination of Care: Review care plan with medical team and plan follow-up for the next clinic visit.

Monitoring and Evaluation: Improvement in serum phosphorus. No complaints with renal diet or dairy substitutes. Better able to plan menus and use recipes that are suitable and acceptable.

- Promote growth in children.
- Maintain fluid balance; modify electrolytes and fluid intake according to tolerance and lab values.
- Prevent or correct anorexia, constipation, growth delay, muscle weakness, cardiac arrhythmias, and hypertriglyceridemia.
- Correct metabolic acidosis to suppress muscle protein losses and improve bone metabolism (Workeneh and Mitch, 2010).
- Prevent the consequences of mineral dysregulation, including renal osteodystrophy, hyperphosphatemia, cardiovascular calcification, extraskeletal calcification, endocrine disturbances, neurobehavioral changes, compromised immune system and altered erythropoiesis. Severe dietary restrictions to control serum phosphorus reduce protein intake and can lead to protein–energy wasting and poor survival (Shinaberger et al, 2008). Dialysate concentrations should be prescribed with reference to plasma electrolyte levels. The KDOQI goals include the following:

Serum PTH	150–300 pg/mL
Serum Ca (albumin-corrected)	8.4–9.5 mg/dL
Serum P	3.5–5.5 mg/dL
Calcium-phosphorus (Ca-P) product	<55 mg/dL

- Improve patient survival, reduce morbidity, increase efficiency of care, and improve quality of life.
- Maintain follow-up contact for consistency of care in other settings or at home.

FOOD AND NUTRITION

- In many hospitals, a standard renal diet provides 60 g protein, 2 g sodium, and 2 g potassium, but this may not be appropriate for dialysis. Most dialysis patients are required to limit their intake of phosphorus to 800–1200 mg/d and total (dietary and medication) elemental calcium to ≤ 2000 mg/d.
- Keep open communication with the nephrologist to determine how to best manage the patient. See Table 16-9 for guidelines on managing dialysis nutrition.

Common Drugs Used and Potential Side Effects

- The drugs listed in Table 16-6 are used.
- Vitamin D deficiency is a common problem. In one study, 79% of the dialysis population was vitamin D deficient (25-hydroxyvitamin D < 30 ng/mL); black race, female sex, winter season, and serum albumin level <3.1 g/dL were the strongest predictors of vitamin D deficiency (Bhan et al, 2010). Vitamin D repletion guidelines are shown in Table 16-10.

Herbs, Botanicals, and Supplements

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

TABLE 16-9 Nutrition Therapy for Dialysis Patients

Nutrient	Hemodialysis (HD)	Peritoneal Dialysis (PD)
Protein for adults (50% from high biologic value sources)	1.2 g protein/kg/d. Urea kinetic modeling may also be used to devise a protein prescription.	1.2–1.3 g protein/kg/d. If there is peritonitis, 1.5 g/kg may be needed until infection subsides.
Protein for children	For children, base initial protein intake on recommended dietary allowance (RDA) for age plus an increment of 0.4 g/kg/d.	Children undergoing PD should be given RDA levels of protein, plus increments based on anticipated losses.
Energy	35 kcal/kg/d for patients who are <60 years of age and 30–35 kcal/kg for patients ≥60 years of age.	35 kcal/kg/d for patients who are <60 years of age and 30–35 kcal/kg for patients ≥60 years of age. Include dialysate calories in calculations. Extra calories may be needed in peritonitis.
Energy for children	For children, follow RDA levels by age for energy.	Same as HD
Carbohydrate (CHO) and fat	After protein is calculated, assess patient needs (e.g., less CHO in diabetes, fewer lipids with dyslipidemia), and calculate percentages accordingly. Try oral supplements before using other modes of feeding such as enteral or parenteral nutrition (PN).	Total energy intake increases from glucose in the dialysate in continuous ambulatory PD. This extra 300–450 kcal of glucose can increase weight and triglyceride levels. CHO absorption calculations must be individualized and altered as the diet prescription changes. Limit simple sugars and saturated fats.
Fluid	With ≥1 L fluid output = 2 L fluid needed. With <1 L fluid output = 1–1.5 L fluid needed. With anuria = 1 L fluid needed.	Fluid restriction is less common in PD; 1–3 L/d is suggested. Fluid intake should be determined by patient's state of hydration; encourage or restrict according to intake and output. No more than 1 kg should be gained in 1 day.
Potassium and phosphorus	Check levels of potassium and phosphorus; modify diet accordingly.	Same as HD
	Dialysis removes very little phosphorus; use 800–1000 mg or ≤17 mg/kg ideal body weight (BW). Calculate potassium as 40 mg/kg BW.	Adjust phosphorus intake according to serum levels; 800–1000 mg phosphorus or 10–15 mg phosphorus/g protein.
Sodium	Limit sodium intake unless there are large losses in dialysate, vomiting, or diarrhea. Restriction to 2–4 g of sodium is common.	Intake of sodium should be liberal, pending assessment of hydration, blood pressure, losses in dialysate, vomiting, and diarrhea.
Vitamins	Use water-soluble vitamin supplements to replace dialysate losses. Daily replacement may not be needed.	Supplement diet with water-soluble vitamins, especially vitamin B_6 and folic acid.
	Folic acid (often need 1 μg), vitamin B_6 (1.3–1.7 mg), vitamin C (75–90 mg), and vitamin B_{12} (2.4 μg)	Active vitamin D should be monitored and replaced at recommended levels. Avoid vitamin A excesses.
	For children use DRI levels for age.	Same as HD
Minerals	Monitor serum labs as available.	Same as HD
	Individualize calcium; dialysate concentrations should be prescribed according to plasma levels.	Same as HD
Enteral nutrition	Try oral supplements first. If tube feeding is necessary, use an appropriate product to meet protein, electrolyte, and volume needs. If low volume is needed, use a product with 1.5–2.0 kcal/mL with small free-water flushes. Monitor electrolytes.	Same as HD
Parenteral nutrition	Before considering PN as an intervention in a dialysis patient, all other methods should be used. Use caution with PN, especially for zinc, and vitamins A and D.	Same as HD
Omega-3 fatty acids	Fish oil supplementation may help reduce prostaglandin synthesis and may help improve hematocrit levels.	Same as HD
Mediterranean diet	A Mediterranean dietary pattern and regular soy intake may be considered.	Same as HD

TABLE 16-10 Vitamin D_3 Repletion

Serum 25(OH)D (ng/mL)	Vitamin D Status	Dose (IU)	Route	Duration (months)[a]
<5	Severe deficiency	50,000/wk × 12 weeks; then monthly	*po*	6
		500,000 once	*im*	
5–15	Mild deficiency	50,000/wk × 4 weeks, then monthly	*po*	6
16–30	Insufficiency	50,000/mo	*po*	6

[a]Duration for all methods is 6 months, after which assay is needed.

contain pharmacologically active compounds that may be hazardous to patients with kidney problems (Burrowes and Van Houten, 2005). For example, noni juice has a high potassium content and should be avoided.
- Approximately half of dialysis and transplant patients use CAM. Mineral supplements and vitamins rank first, followed by herbal teas and other products that contain ingredients that may accumulate in renal failure (Nowack et al, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

Dialysis: General

- Public Law 92-603 provides financial assistance via Medicaid to all persons covered by Social Security who have ESRD with dialysis. Table 16-11 discusses the role of dietitians in dialysis care.
- Discuss signs of uremia (nausea, vomiting, hiccups, fatigue, and weakness).

TABLE 16-11 Role of the Renal Dietitian in Dialysis Care

Patients with end-stage renal disease often experience malnutrition as a result of decreased dietary intake; inadequate dialysis; loss of nutrients into the dialysate; abnormal protein, carbohydrate, and lipid metabolism; and concomitant diseases, which may contribute to an increase in morbidity and mortality.

Close monitoring of nutritional status is completed by evaluating serum albumin and relevant biochemical data, appetite assessments, dietary energy and protein intakes, consumption of vitamins and minerals, and intake of oral supplemental foods, tube feeding, and parenteral nutrition.

Anthropometry is performed at baseline and on a yearly basis.

Specified changes in serum albumin level or body weight trigger action by the dietitian to prevent protein–energy malnutrition.

Multiple diet parameters are necessary to provide optimal nutritional health, including monitoring of calories, protein, sodium, fluid, potassium, calcium, and phosphorus, as well as other individualized nutrients. Consider all modes of nutritional intervention; use that which is best accepted by the patient and the least invasive.

- Discuss high-energy, low-protein, electrolyte-controlled food choices and supplements. Adequate care must be taken to ingest the designated protein and energy levels, according to current lab reports.
- When patients participate in an exercise program, appetites often improve. Exercise training is a potentially beneficial approach to correct muscle wasting, but more information is needed to optimize exercise regimens (Workeneh and Mitch, 2010).
- Counsel patient regarding managing a healthy diet to prevent or control heart disease or diabetes. A Mediterranean dietary pattern and regular soy intake both have been shown to attenuate chronic inflammation, and this may be useful in CKD patients (Kuhlmann and Levin, 2005).
- If avoiding potassium-rich foods is needed, salt substitutes should be monitored closely. Use of longer cooking times and extra water may help leach out excess potassium.
- Taste alterations are common, especially distaste for red meats, fish, poultry, eggs, sweets, and vegetables. Work individually with the patient to plan meals for adequacy of protein intake.
- Discuss sodium alternatives; see Table 16-5 for spices and condiments to use instead of salt.
- Provide information about dining away from home, home-delivered meals, and meals while traveling.
- Establishing trust, mutual respect, and emotional support are essential to promote a successful set of nutritional outcomes.

Hemodialysis

- Discuss maximum fluid gain (usually 3–5% of BW) between dialysis sessions. Noncompliance with fluid restriction is common and can lead to systemic and cardiac overload.
- Teach fluid management to motivate patients to comply with their regimens; patients report that they feel better when their weight gains are within acceptable limits.

Peritoneal Dialysis

- Fluid restrictions are not always needed with PD. Patient should learn how to recognize significant changes in dry weight (adjusted edema-free BW) or food intake. Discuss actions to be taken. Usually, 3–4 lb between intermittent PD is allowed.
- Teach the patient and family about managing diet to control phosphorus levels (goal is <5.5 mg/dL). Increasing patient knowledge may enhance dietary adherence to phosphorus restriction and use of phosphate binders. Avoid use of carbonated beverages that contain phosphates, such as colas.

Patient Education—Food Safety

Foodborne illness can occur when there is contamination of food at any point during the preparation process. Because renal patients are at high risk for foodborne illness, follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill. More details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- Dialysis
  http://www.nlm.nih.gov/medlineplus/dialysis.html
- HD
  http://kidney.niddk.nih.gov/kudiseases/pubs/hemodialysis/index.htm
- Merck Manual—HD
  http://www.merck.com/mmpe/sec17/ch234/ch234b.html
- Merck Manual—PD
  http://www.merck.com/mmpe/sec17/ch234/ch234d.html
- National Guidelines Clearinghouse
  http://www.guideline.gov/summary/summary.aspx?doc_id=10016&ss=15
- NKF Dialysis Cookbook
  http://www.kidney.org/atoz/pdf/dialysis_cookbook.pdf
- NKF—HD
  http://www.kidney.org/atoz/content/dietary_hemodialysis.cfm
- NKF—PD
  http://www.kidney.org/atoz/content/peritoneal.cfm
- PD
  http://kidney.niddk.nih.gov/kudiseases/pubs/peritoneal/index.htm

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GLOMERULAR AND AUTOIMMUNE KIDNEY DISEASES

NUTRITIONAL ACUITY RANKING: LEVEL 2 ACUTE; LEVEL 3 CHRONIC

DEFINITIONS AND BACKGROUND

Glomerulonephritis is a collective term used for diseases with renal inflammation stemming from the glomeruli; immune mechanisms are involved in all of them (Segelmark and Hellman, 2010). Antigen–antibody complex reactions become trapped in the glomeruli with resulting edema, scarring, and inflamed glomeruli. Resolution can be promoted by anti-inflammatory clearance by macrophages and mesangial cells (Watson et al, 2006). Clinical trials have provided compelling evidence that omega-3 polyunsaturated fatty acid (eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) supplementation is useful, although some other studies suggest such supplementation might be without benefit (Pestka, 2010).

Acute GN occurs with damage to the glomeruli from infection, lupus, or other forms of kidney disease. GN caused by lupus is classified on the basis of lesions, extent and severity of the involvement, immune complex deposition, activity, and chronicity (Seshan and Jennette, 2009). Postinfectious GN (PIGN) may be related to *Staphylococcus* (Wen and Chen, 2010) or to untreated *Streptococcal* infections. Anuria with <400 mL/24 hours of urinary output may require temporary dialysis. Most acute GN conditions resolve after 3–12 months.

In **chronic GN**, repeated episodes of nephritis lead to the loss of renal tissue and kidney function; glomeruli disappear, and normal filtering is lost. The kidneys can no longer concentrate urine, and more urine is voided in an effort to rid the body of wastes. Protein and blood are lost in the urine. Blood pressure rises, causing vascular changes and CKD. The level and type of proteinuria (only albumin or including other proteins) will determine the extent of damage and whether a patient is at risk for developing CKD. Decreasing proteinuria indicates an improved prognosis, whereas hypertension does not.

GN is the second most common cause of renal failure. Conservative treatment uses protein restriction (0.6 g protein/kg BW) to correct metabolic and hormonal derangements; restriction is contraindicated in cases of protein malnutrition, neoplasm, growth, or infection. Sufficient energy intake is essential. Four forms of primary GN include Goodpasture or anti-GBM disease, IgA nephritis (IgAN, Berger disease), membranous nephropathy, and membranoproliferative GN (MPGN), which occurs in adults of African descent and is a common cause of nephrotic syndrome. Much of the ESRD in African Americans is attributable to variants in the gene that encodes motor protein myosin 2 a (MYH9) in this haplotype (Freedman and Sedor, 2008).

Glomerulosclerosis involves hardening (scarring or sclerosis) of the glomeruli from lupus, HIV infection, or diabetes. Significant reduction of renal mass initiates a series of events that lead to proteinuria and glomerulosclerosis

(An et al, 2009). Albumin is lost in the urine, nitrogen waste products are retained, and retinal changes occur. **Focal segmental glomerulosclerosis (FSGS)** may cause nephrotic syndrome secondary to chronic pyelonephritis or diabetes; it is more common in Caucasian adults. Omega-3 fatty acid supplementation may reduce or reverse upregulation of prooxidant, proinflammatory, and profibrotic pathways in the remnant kidney (An et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Both genetics and infection seem to play a role. MYH9 is a gene that promotes MPGN in African Americans. Genes that cause the hereditary forms of FSGS that affect Caucasians include ACTN4, TRPC6, and CD2AP.

Clinical/History

- Height
- Weight
- aBWef
- BMI
- Diet history
- Waist:hip circumference
- BP
- Edema of face, ankles, feet, legs
- Fever?
- I & O
- Shortness of breath
- Decrease in urine volume (oliguria)
- Urine smell on breath and sweat
- Itching
- Abdominal pain, vomiting
- Dark or rust-colored urine
- Foamy urine
- Yellowish–brown skin discoloration
- Renal radiographs
- Kidney biopsy

Lab Work

- Alb, transthyretin (low?)
- CRP
- GFR
- CrCl level
- Complement levels
- Anti-glomerular basement membrane antibody test
- Uremia (accumulation in the blood of wastes)
- Transferrin
- BUN, Creat
- Proteinuria
- Gluc
- Urinary ketones
- Chol (increased from proteinuria?)
- Trig
- White blood cell (WBC) count
- H & H
- MCV
- Serum Fe, ferritin
- TIBC
- Specific gravity (decreased?)
- PTH
- Na^+, K^+
- Serum phosphorus
- AST
- Ca^{++}, Mg^{++}
- Serum copper (increased?)

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Energy Intake

Assessment Data: I & O records. BMI of 21. Diet hx shows an average daily intake of 1300 kcal but requirements of 1750 kcal daily. Intake of protein (60 g) meets needs.

Nutrition Diagnosis (PES): Inadequate energy intake NI 1.4 related to poor renal status and sufficient protein intake without calories for sparing as evidenced by diet hx of 1300 kcal, 60 g protein but estimated needs of 1750 kcal/d.

Interventions:

Food-Nutrient Delivery: Increase the use of CHO and fats to spare protein.

Education: Provide instruction and education about the role of protein and the need for sufficient intake of calories from CHO and fat.

Counseling: Provide individualized tips for increasing calories to enhance total energy by using preferred foods.

Coordination of Care: Work with the medical team to assess the effectiveness of extra calories to keep lab values closer to normal and to gain a few pounds over time.

Monitoring and Evaluation: BMI improving; now 23 after 3 months of higher-energy intake. Protein intake remains approximately 60 g, calorie intake between 1700 and 1800 kcal/d. No undesirable side effects.

INTERVENTION

OBJECTIVES

- Improve renal functioning; prevent systemic complications where possible.
- Monitor abnormal protein status and serum nitrogen retention.
- Spare protein for tissue repair. Prevent further catabolism of protein to lessen production of urea and other protein waste products.
- Control hypertension and edema.
- Manage hyperglycemia or dyslipidemia.
- Correct metabolic abnormalities. Improve nutritional status and appetite.
- Prevent or correct growth failure in children.
- Reduce inflammation and attenuate oxidative damage.
- Reduce workload of circulatory system by decreasing excess weight, where needed.

FOOD AND NUTRITION

- Modify patient's diet according to disease progression; maintain sufficient levels of protein as long as kidneys can eliminate waste products of protein metabolism.
- Use sufficient energy to spare protein (60% CHOs, 30% fat). For adults, 30–40 kcal/kg adjusted edema-free BW is needed to spare protein for tissue synthesis and wound healing.
- In **uremia**, diet should include 50% high–biologic value proteins (such as from cheese, meats, fish, eggs, and dairy foods), or essential amino acids at 2–3 times the normal should be included. Limit protein to 0.6–0.8 g/kg.
- In **oliguria**, restrict fluid intake to 500–700 mL. Limit protein to 0.6–0.8 g/kg. When urinary output is reduced greatly, restrict phosphorus intakes if needed. Potassium is often controlled by medications, but monitor and

adjust as needed. Some patients will require dialysis to remove waste products.
- With **edema** or **high blood pressure**, restrict sodium intake to 2–3 g/d. In a child, a restriction of 500–1000 mg may be needed. Carefully monitor sodium levels because sodium depletion can occur during the diuretic phase of chronic GN.
- Vegetarian diets, soy products, and use of omega-3 fatty acids may be beneficial for dyslipidemia. Restrict fat and cholesterol if needed; monitor diet carefully.
- Control CHO intake with diabetes or hyperglycemia.
- If patient is obese, use an energy-controlled diet, but avoid fasting and very low–calorie diets.
- Determine vitamins and nutrients provided by therapeutic diet and supplement to meet daily requirements, especially for calcium, niacin, and other B vitamins, which are easily lost in urine. Children with uremia require vitamin D_3 replacement for growth and to improve appetite; adults will need it to maintain bone health.

Common Drugs Used and Potential Side Effects

- When diuretics such as furosemide are used to reduce edema, watch for potassium wasting or dehydration. Dehydration can elevate BUN; assess carefully.
- ACE inhibitors are useful for reducing hypertension.
- Immunosuppressants may be used.
- See Table 16-6 for more specific medications.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Omega-3 fatty acid supplementation may be beneficial (An et al, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- A renal dietitian should provide nutrition therapy and counseling. A diet controlled in protein, fluid, phosphorus, sodium, and potassium may be needed; it must be individualized and is likely to change frequently.
- Commitment from the patient and family is needed; extra expense may be incurred for low-protein foods and amino acid analogs.
- Fluid intake should be distributed carefully throughout the patient's waking hours (see Table 16-7). Check for changes needed during diarrhea or related problems.
- Edema is better controlled by sodium restriction than by fluid restriction; monitor patient carefully. Patients with edema are often thirsty; edema water is trapped and unavailable for body's use.
- Encourage frequent doctor or clinic visits to monitor renal functioning.
- Patients with ascites may become anorexic in the upright position. Position patient carefully for food intake.

Patient Education—Food Safety

- Foodborne illness can occur when there is contamination of food at any point during the preparation process.
- Because renal patients are at high risk for foodborne illness, follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill.
- Details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- Acute GN
  http://www.nephrologychannel.com/agn/
- MEDLINE—GN
  http://www.nlm.nih.gov/medlineplus/ency/article/000484.htm

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KIDNEY STONES

NUTRITIONAL ACUITY RANKING: LEVEL 1–2

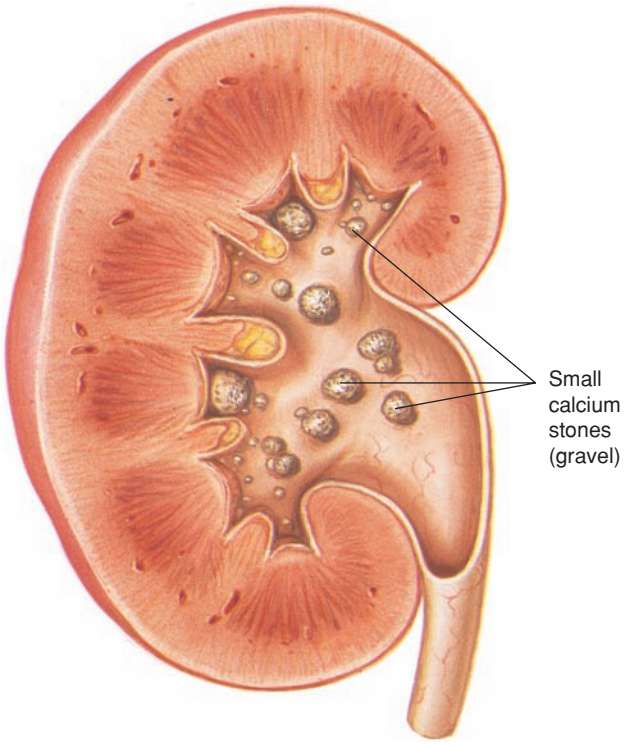

TABLE 16-12 Causes of and Predisposition to Kidney Stones

Climate	Hot climate and dehydration during summer months
Diet	Low intakes of dietary calcium and fluid, high intakes of sodium in susceptible individuals
Diminished water intake	During sleep, travel, or illness or from poor habits
Family	Family history of kidney stones
Genetic disorders	Gout, primary hyperoxaluria, hyperparathyroidism, renal tubular acidosis, cystinuria, and hypercalciuria
Gender	Three times more common in males
Intestinal changes	Inflammatory bowel disease, intestinal bypass surgery, ostomy surgery
Medications	Certain diuretics, use of the protease inhibitor indinavir, excessive intake of supplemental vitamin D
Urinary tract issues	Urinary tract infection or stagnation from blockage

REFERENCE
National Institute of Diabetes and Digestive and Kidney Diseases. Kidney stones in adults, http://kidney.niddk.nih.gov/kudiseases/pubs/stonesadults/, accessed February 22, 2010.

DEFINITIONS AND BACKGROUND

Kidney stones are masses made of crystals; when they move down through the ureters, they cause severe pain. Unfortunately, kidney stones are common due to modern lifestyles, dietary habits, and obesity (Straub and Hautmann, 2005). The process is officially known as *urolithiasis* or *nephrolithiasis*. While all humans form calcium oxalate crystals, most do not form stones. However, prevalence is rising in both sexes (Moe, 2006). Annually, approximately 400,000 people are treated for this problem. Stones affect 5% of adults but may even occur in premature infants.

Kidney stones develop when salt and minerals in urine form crystals that coalesce and grow in size. Stones are formed by progressive deposition of crystalline material around an organic nidus. Decreased fluid intake and consequent urine concentration are among the most important factors influencing stone formation. Other major causes of renal stones are urinary tract infections (UTIs), cystic kidney diseases, metabolic disorders such as HPT, and renal tubular acidosis (see Table 16-12). A person who has had kidney stones or has a family history of kidney stones is at high risk.

Fluid is a key factor. It is not the quantity of fluid consumed but that which is voided that is important. Extra fluid intake will be needed by those who live in hot, dry conditions and by those who exercise or perspire significantly. Many stones can be prevented through changes in diet (Grases et al, 2006). Apple or grapefruit juices may aggravate risk whereas wine, beer, and some diet sodas are protective against stones. Orange and lemon–lime flavors contain alkalis of citrate and malate, which inhibit calcium formation; good choices include Fresca, diet Sunkist orange, diet 7-Up, and diet Canada Dry.

Approximately 80% of stones are composed of calcium oxalate and calcium phosphate (Reynolds, 2005). Ten percent are composed of struvite (magnesium ammonium phosphate produced during infection with bacteria that possess the enzyme urease). The remaining 10% are composed of uric acid, cystine or ammonium acid urate, or melamine. Melamine contamination of infant formula has been noted by the FDA (Skinner et al, 2010).

Struvite stones are mostly found in women who have UTIs. **Cystine stones** may form in individuals who have cystinuria, a familial disorder.

Uric acid stones may result from purine metabolism, gout, leukemia, cancer, or colectomy; they are more common in men. An abnormally low urinary pH (UpH) contributes to the problem. Creating an alkaline urine is the goal; a UpH of 6.2–6.8 is desirable (Hess, 2006). A lower-purine food intake (such as sardines) is also beneficial.

Most studies show no increase in **calcium oxalate** stone risk with high calcium intake (from either diet or supplements) but a high rate of stone risk from a low calcium intake (Heany, 2008). In the Health Professionals Follow-up Study, the Nurses' Health Study I and II, spinach accounted for >40% of oxalate intake; risks were higher in men with low dietary calcium intake, but dietary oxalate was *not* a major risk factor (Taylor and Curhan, 2007). Therefore, diets need not be excessively low in oxalates (see list in Table 16-12).

Diets high in animal protein provide a high acid load; sufficient potassium and alkali are required to neutralize the effect (Straub and Hautmann, 2005). Vegetarians form

stones at one third of the rate of those eating a mixed diet. Balanced diets containing moderate amounts of meat and plant proteins (legumes, seeds, nuts, and grains) may keep urinary composition within guidelines. Untreated **calcium oxalate stones** can lead to a greater chance of forming additional stones within later decades.

Citrate inhibits calcium oxalate stone formation by complexing with calcium in the urine, inhibiting spontaneous nucleation, and preventing growth and agglomeration of crystals; hypocitraturia is a metabolic abnormality found in 20–60% of stone formers (Zuckerman and Assimos, 2009). A normal calcium, low animal-protein diet with lemonade and citrus juices is effective for these individuals.

Extracorporeal shockwave lithotripsy remains the preferred method of treatment of urinary stones. Medical expulsive therapy is under review as a new treatment.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Hypercalciuria is inherited, and it may be the cause of stones in more than half of the population who have calcium oxalate stones. Cystinuria runs in families and affects both men and women.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- I & O
- Excruciating groin or flank pain
- Fever
- Hematuria or mild pyuria
- Abnormal urine color
- Excessive nocturia
- Nausea, vomiting
- Burning and urinary frequency

- Kidney ultrasound
- Intravenous pyelogram
- Abdominal radiographs or computed tomography scan

Lab Work

- Urinalysis (crystals, RBCs)
- 24-hour urine studies:
  - Total urine volume
  - Urinary calcium (normal, 300–400 mg)
  - Urinary sodium
  - Urinary citrate
  - Uric acid
  - UpH
- Serum oxalate levels
- Gluc
- Ca^{++}
- Mg^{++}
- Alb
- CRP
- Lactose intolerance
- Serum Na^{+}, K^{+}
- BUN
- Creat
- H & H
- Serum Fe

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Fluid Intake

Assessment Data: I & O records showing low intake of fluids throughout the average day (<6 cups total). Recently hospitalized with uric acid stones; Hx of gout for 3 years.

Nutrition Diagnosis (PES): NI-3.1 Inadequate fluid intake related to poor daily habits as evidenced by uric acid stones and intake records showing <6 cups of fluid per average day.

Interventions:

Food-Nutrient Delivery: Increase fluid intake to 10–12 cups per day from water and other liquids.

Education: Discuss the importance of fluid intake and taking appropriate medicines as prescribed.

Counseling: Discuss purine-rich foods and that diet is seldom needed if the medicine is taken and works properly.

Coordination of Care: Work with pharmacist to ensure that medications will suffice.

Monitoring and Evaluation: Resolution of high urinary uric acid levels and no more renal stones. Intake of fluid averaging between 11 and 12 cups daily.

INTERVENTION

OBJECTIVES

- Determine predominant components and prevent recurrence by normalization of BMI, adequate physical activity, balanced nutrition, and sufficient daily fluid intake. Modify diet according to predominant components; there is seldom a single cause.
- To increase excretion of salts, dilute urine by increasing fluid volume to at least 2 L/24 h.
- Prevent scarring, recurrence of stones, obstruction, bone demineralization, or kidney damage.
- Promote use of a heart-healthy diet (Mitka, 2009).
- Repeat urinary studies approximately 6–8 weeks after initial metabolic testing to evaluate effectiveness of dietary changes. Once stable, where the patient's urine demonstrates decreased risk of stone formation, metabolic testing and radiographs should be performed at least annually.

FOOD AND NUTRITION

- General guidelines: Fluid intake should be high, as tolerated (2 L/d). Colorless urine is sought. Individuals with cystinuria will need 3.5 L/d fluid intake (Reynolds, 2005). Limit the use of apple or grapefruit juices.
- Consume a diet that is balanced. Fruits and vegetables may reduce the low potassium/high sodium intake (Reynolds, 2005). The DASH diet is a good recommendation as it is associated with a marked decrease in kidney stone risk (Taylor et al, 2009).
- A heart-healthy weight loss and exercise plan may be needed if the patient is obese (Mitka, 2009; Taylor et al, 2005).
- Specific guidelines by stone content are provided in Table 16-13.
- Calcium should not be restricted, except in absorptive hypercalciuria where calcium restriction remains beneficial in combination with a drug (Straub and Hautmann, 2005). Hypocitraturia should be managed through a combination of oral alkali, dietary modifications, and

TABLE 16-13 Dietary Treatment of Specific Renal Stones

Type	Treatment
Calcium oxalate stones	Calcium intake should be increased to >1000 mg/d; good sources include skim milk, yogurt, low-fat dairy products, broccoli, fortified orange juice, and ricotta or other cheese. Fortified foods and almonds are several nondairy choices. Reducing urinary oxalates has a more powerful effect on stone formation than reduction of urinary calcium. However, diet has a limited role. Omit **very high sources** from rhubarb, spinach, strawberries, beets, All Bran, Swiss chard, almonds and mixed nuts, chocolate soy milk, miso, tahini, and soybeans. Limit **high sources**: okra, sweet potatoes, tomatoes, greens, fried potatoes, navy or black beans, apricots, figs, kiwifruit, some grains, soy milk, soy ice cream, chocolate candy bars, poppy seed, and turmeric. See Web site http://www.ohf.org/docs/Oxalate2008.pdf.
Citrate stones	Hypocitraturia is found in up to 20% of stone formers and may be idiopathic or secondary to intestinal, renal, dietary, or pharmacological causes.
Cystine stones	Individuals with cystinuria will need 3.5 L of fluid daily. Use a diet low in cystine, methionine, and cysteine. Protein intake should be mildly reduced. Cystine stones usually are the result of a rare hereditary defect. Alkalize urine with agents like D-penicillamine.
Struvite stones	Struvite stones contain magnesium ammonium phosphate and may form after an infection in the urinary system. They can grow very large and may obstruct the kidney, ureter, or bladder if not removed.
Uric acid stones	Urate stones can be dissolved by urine alkalinization with citrate or bicarbonate. There is no inhibitor of uric acid crystal formation; dietary measures focus on reducing uric acid and increasing urine volume. Nonfat milk, low-fat yogurt, and other dairy products are useful. High intake of fruits such as cherries and vegetable protein may reduce serum urate levels.

lemonade or other citrus juice–based therapy (Zuckerman and Assimos, 2009). Include legumes and dried beans for their health-promoting saponins, which are useful in the treatment of hypercalciuria.

Common Drugs Used and Potential Side Effects

- Certain medications, such as triamterene (Dyrenium), indinavir (Crixivan), and acetazolamide (Diamox), are associated with urolithiasis.
- Approximately 15% of patients forming stones require specific pharmacological prevention (Straub and Hautmann, 2005).
- For **calcium oxalate** or **uric acid stones**, allopurinol (Zyloprim) and probenecid are usually used; a purine-restricted diet is not always needed (Cameron and Sakhaee, 2007). Drink 10–12 glasses of fluid daily; avoid concomitant intake of vitamin C supplements. Maintain alkaline urine (may need to use sodium bicarbonate). Thiazide diuretics may reduce hypercalciuria (Reynolds, 2005).
- **Hypocitraturia** should be managed through diet and oral alkali (Zuckerman and Assimos, 2009). Potassium citrate and potassium bicarbonate help to alkalinize the urine. Sodium restriction may be inappropriate; sodium supplementation is beneficial in these patients because it results in voluntary increased fluid intake (Stoller et al, 2009).
- For **cystine stones:** D-penicillamine use requires vitamin B_6 and zinc supplementation. Increase fluid intake. Take the drug 1–2 hours before or after meals. Stomatitis, diarrhea, nausea, vomiting, and abdominal pain may occur.
- **Struvite** stones will require use of antibiotics.
- Uric acid stones are often dissolved by using potassium citrate/bicarbonate. Acetazolamide increases UpH in patients with uric acid and cystine stones who are already taking potassium citrate; however, it may be poorly tolerated and can induce calcium phosphate stone formation (Sterrett et al, 2008).
- Potassium citrate (Polycitra K) reduces kidney stone incidence in children treated for seizures with the ketogenic diet (McNally et al, 2009).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- The use of probiotics such as *Oxalobacter formigenes* in the prevention of calcium oxalate stone disease needs further investigation.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The most important lifestyle change to prevent stones is to drink more liquids, especially water; try to produce at least 2 quarts of urine in every 24-hour period.
- Teach the principles of a heart-healthy DASH diet (Mitka, 2009; Taylor et al, 2009).
- A patient's 24-hour urine chemistry profile should guide the dietary adjustments. Use dietary measures that are appropriate for condition and content of the stone.
- Discuss occupational risks for stone formation, including working in hot, dry environments, or working with metals such as cadmium.

Patient Education—Food Safety

Foodborne illness can occur when there is contamination of food at any point during the preparation process. Follow standard practices in food safety and handling.

For More Information

- Kidney Stone Diet
  http://www.gicare.com/pated/edtgs29.htm.
- Kidney Stones in Adults
  http://www.niddk.nih.gov/health/kidney/pubs/stonadul/stonadul.htm
- National Library of Medicine
  http://www.nlm.nih.gov/medlineplus/ency/article/000458.htm
- Oxalosis and Hyperoxaluria Foundation
  http://www.ohf.org/
- Renal Stones in Adults
  http://kidney.niddk.nih.gov/Kudiseases/pubs/stonesadults/

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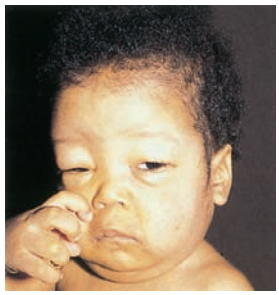
NEPHROTIC SYNDROME

NUTRITIONAL ACUITY RANKING: LEVEL 2–3



DEFINITIONS AND BACKGROUND

Nephrotic syndrome is not a disease but causes massive proteinuria, with 3.5 g or more of protein lost within 24 hours. As much as 30 g can be lost as a result. Serum albumin is especially affected. The common nephrotic syndrome in children is called "minimal change disease." In approximately 20% of children with nephrotic syndrome, kidney biopsy reveals scarring or deposits in the glomeruli.

Adults who have nephrotic syndrome usually have some form of GN, with renal failure not far behind. FSGS is the most common cause in Caucasians; MPGN is the common cause in African Americans. Other causes include DKD, lupus, amyloidosis, HIV infection, hepatitis B or C, malaria, and heart failure.

Elevated LDL cholesterol is common because of altered lipoprotein production. When the liver makes more albumin in an effort to replace that which is lost, more cholesterol and triglycerides are released.

MNT centers on the problem of salt and water retention, protein depletion, hyperlipidemia, and loss of carrier proteins for vitamins and minerals. Patients have normal anabolic responses to dietary protein restriction (decreased amino acid oxidation) and feeding (increased protein synthesis and decreased degradation). A very high–protein diet will alter GFR; limit protein to decrease hyperfiltration. A moderate-protein diet that provides adequate energy can maintain nitrogen balance in nephrotic syndrome. Immunosuppressive medication is not helpful in the genetic forms of congenital nephrotic syndrome (CNS); a high-energy diet is needed and kidney transplantation is the only curative therapy (Jalanko, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: CNS is a rare kidney disorder caused by genetic defects in the components of the glomerular filtration barrier, especially nephrin and podocin (Jalanko, 2009).

Clinical/History	Lab Work	
Height	Alb, transthyretin	Ceruloplasmin (decreased)
Weight	Proteinuria, uremia	AST (increased)
Weight gains?	CRP	Na^+
aBWef	LDL Chol and Trig (increased)	K^+ (hypokalemia?)
Edema	H & H	Serum phosphorus
BMI	Serum Fe	Hypovitaminosis D?
Diet history	TIBC, percent saturation	Ca^{++}, Mg^{++}
BP	Serum ferritin	BUN, Creat
I & O	Transferrin (increased)	GFR, CrCl
Foamy urine		
Chest pains		
Weakness		
Renal biopsy		
Renal radiographs		

INTERVENTION

OBJECTIVES

- Treat the underlying condition and take appropriate medications.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Protein Intake

Assessment Data: Dietary recall and laboratory analysis.

Nutrition Diagnosis (PES): NI-5.7.1 Inadequate protein intake, of only 40 g/d, and worsening proteinuria as evidenced by urinary losses of more than 20% of the usual status.

Intervention: Education on protein requirements and sources. Counseling about ways to increase protein without exceeding the recommended level.

Monitoring and Evaluation: Lab values and dietary intake.

- Prevent thrombotic episodes and manage infectious complications (Jalanko, 2009).
- Ensure efficient utilization of fed proteins, spared by use of adequate calories. Prevent muscle catabolism. Rarely, if protein losses are severe, albumin infusion is used.
- Correct anorexia and prevent malnutrition.
- Reduce edema, control sodium intake, prevent or control renal failure.
- Manage hyperlipidemia and elevated triglycerides.
- Monitor for potassium deficits with certain diuretics.
- Replace any other nutrients, especially those at risk (e.g., calcium, vitamin D).

FOOD AND NUTRITION

- Use a diet of modest protein restriction (0.8 g/kg in adults, with 50% of high biological value). Children should be given the RDA for their age because high protein levels may worsen proteinuria and will not improve serum albumin levels.
- Diet should provide 35–40 kcal/kg/d. CHO intake should be high to spare protein for lean body mass; use high–complex CHO and high-fiber foods.
- With dyslipidemia, limit saturated fats and cholesterol; decrease intake of concentrated sugars and alcohol. Encourage use of linoleic acid and omega-3 fatty acids. A vegetarian, soy-based diet with amino acid replacements may be beneficial.
- If patient has edema, restrict sodium intake to 2–3 g. The DASH diet may be useful.
- Provide adequate sources of potassium, vitamin D, and calcium. Replace zinc, vitamin C, folacin, and other nutrients. Monitor the need for iron but do not use excesses, especially with infections.
- Fluid restrictions may be necessary if edema is refractory to diuretic therapy.
- Offer appetizing meals to increase intake. If required, use tube feedings (specialty renal products if needed). Nocturnal feedings can help children with growth.

Common Drugs Used and Potential Side Effects

- ACE inhibitors such as benazepril (Lotensin), captopril (Capoten), and enalapril (Vasotec) are used to lower blood pressure; they may help prevent protein from leaking into urine and damaging the kidneys. Angiotensin II receptor blockers losartan (Cozaar) and valsartan (Diovan) may also be prescribed.
- Antibiotics may be needed when infection is present. Evaluate effects of diet accordingly on drug effectiveness.
- Blood thinners such as warfarin (Coumadin) are prescribed if blood clots are a risk.
- Cholesterol-lowering drugs are often prescribed. If statins such as atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Altoprev, Mevacor), pravastatin (Pravachol), rosuvastatin (Crestor), and simvastatin (Zocor) are used, CoQ10 may be depleted.
- Diuretics are used to excrete excess fluid. Thiazide diuretics such as furosemide (Lasix) will deplete potassium; others may spare or retain potassium (e.g., spironolactone). Monitor closely.
- Immune system–suppressing medications, such as corticosteroids, are used to decrease renal inflammation. With prednisone, sodium restrictions may be needed and losses of potassium, nitrogen, or calcium may result. Muscle wasting, weight gain, and other side effects are common. Cyclophosphamide has fewer side effects (du Boef-Vereijken et al, 2005).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- With cyclosporine, avoid echinacea and St. John's wort because of their counterproductive effects on the drug.
- The use of soy to slow down the progression of diabetic nephropathy and proteinuria warrants further study.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Help patient plan appetizing meals. Sodium restriction is common.
- If patient has abdominal edema, careful positioning may increase comfort at mealtimes.
- Weight management plans will be needed if steroid use will be long-term.
- Nephrotic syndrome can increase the risk of infections and blood clots; medical follow-up is needed.
- In steroid-dependent nephrotic syndrome (SDNS), doses of prednisone lower than 0.75 mg/kg/d do not seem to stunt growth in children (Simmonds et al, 2010).

Patient Education—Food Safety

Foodborne illness can occur when there is contamination of food at any point during the preparation process. Because renal patients are at high risk for foodborne illness, follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill. More details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- Mayo Clinic
  http://www.mayoclinic.com/health/nephrotic-syndrome/DS01047
- NephCure Foundation
  http://www.nephcure.org/
- University of North Carolina Kidney Center
  http://www.unckidneycenter.org/kidneyhealthlibrary/nephroticsyndrome.html

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RENAL METABOLIC DISORDERS: HYPOPHOSPHATEMIC RICKETS AND HARTNUP DISORDER

NUTRITIONAL ACUITY RANKING: LEVEL 3–4

DEFINITIONS AND BACKGROUND

X-linked hypophosphatemic rickets (XLH) associated with decreased renal tubular reabsorption of phosphorous. A defect in the skeletal response to PTH contributes to HPT in XLH (Levine et al, 2009). Incidence is 1 in 20,000 births. XLH is characterized by low to normal serum levels of 1,25-dihydroxyvitamin D_3, normocalcemia, and hypophosphatemia. There is abnormal regulation of production and/or degradation of PTH. Because vitamin D is metabolized abnormally, skeletal and dental structures are affected. Hypophosphatemia is responsible for the clinical manifestations, which vary with the age of the patient and the severity of wasting (Laroche and Boyer, 2005). In poorly growing patients, growth hormone therapy improves final height, phosphate retention, and radial bone mineral density.

Hartnup disorder is an autosomal recessive abnormality of renal and GI neutral amino acids, especially tryptophan and histidine. It is a rare familial condition characterized by hyperaminoaciduria. A red, scaly, photosensitive pellagra-like skin rash is seen on the face, neck, hands, and legs. Cerebellar changes occur, including delirium. Without the absorption of tryptophan, conversions into serotonin, melatonin and niacin are inefficient. Because niacin is a precursor to nicotinamide, part of NAD+, those metabolic functions are also altered. The failure to resorb amino acids in this disorder is compensated by a protein-rich diet (Broer et al, 2005). Bone deformities, pain, and small stature can occur even in children with good compliance, requiring surgical correction and bone lengthening (Fucentese et al, 2008).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: XLH consists of a genetic alteration in the handling of phosphate in the proximal tubule. It is inherited as a sex-linked dominant trait with mutations of the metalloproteinase PHEX on chromosome Xp22.1; FGF23, DMP1, and ENPP1 may also be involved. Hartnup disorder is an autosomal recessive disorder with alterations in the neutral amino acid transporter B(0)AT1 (SLC6A19) on chromosome 5, the major luminal, sodium-dependent neutral amino acid transporter. Its expression in the kidney depends on collectrin (Tmem27), a protein related to ACE2, which maintains blood pressure and glomerular structure (Camargo et al, 2009).

Clinical/History

- Birth weight
- Present weight
- Length
- Growth percentile
- BMI
- Diet history
- BP
- For XLH:
  - Bowing of lower limbs
  - Bone pain
  - Seizures
  - Short stature, delayed growth
  - Skeletal radiograph
- For Hartnup disorder:
  - Red, scaly rash
  - Abnormal muscle tone
  - Cerebral ataxia

Lab Work

- Usual Chemistry panels plus those listed
- For XLH:
  - Serum phosphorus (low)
  - Serum vitamin D
  - Ca^{++} (normal)
  - Alk Phos (elevated with high bone turnover)
  - PTH
  - Hyperphosphaturia (rickets)
  - BUN, Creat (normal)
- For Hartnup disorder:
  - Tryptophan-loading test

INTERVENTION

OBJECTIVES

- **XLH:** Correct malabsorption of phosphorus to establish homeostasis with adequate bone and skeletal mineralization. Avoid overly aggressive therapy to avoid hypercalcemia and secondary extraskeletal calcification.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal Nutritional Labs

Assessment Data: Child with low rate of growth, percentile for height and weight at 15th percentile. Genetic testing is positive for XLH. Serum phosphate levels very low; calcium is normal, alk phos level elevated.

Nutrition Diagnosis (PES): Abnormal nutritional labs related to new diagnosis of genetic condition (vitamin D-resistant rickets, XLH) as evidenced by very low serum phosphate level with high alk phos and normal calcium levels and genetic test results.

Interventions:

Food-Nutrient Delivery: Maintain a balanced intake of calcium, phosphorus, and vitamin D-rich foods.

Education: Discuss the genetics of this disorder and how diet alone will not correct it.

Counseling: Counsel about the importance of taking all prescribed medications as ordered and not taking over-the-counter vitamins and minerals without first discussing with the doctor.

Coordination of Care: Discuss with the medical team any implications for diet. Support the importance of taking prescribed phosphate and vitamin D exactly as ordered.

Monitoring and Evaluation: Improved serum phosphate. Eventual improvement in growth, which may require growth hormone replacement.

- **Hartnup disorder:** Correct skin changes and behavioral side effects of this pellagra-like condition. Ensure adequacy of protein. Support measures such as psychiatric treatment as needed.

FOOD AND NUTRITION

- **XLH:** Diet should provide adequate amounts of calcium. Monitor to avoid mineral toxicities.
- **Hartnup disorder:** A high-protein diet with supplements will be needed.

Common Drugs Used and Potential Side Effects

- In **XLH,** ergocalciferol is a vitamin D analog that is used with phosphate supplements. Organic phosphate should be given every 4 hours along with supportive vitamin D therapy. After growth is completed, the drug is often reduced. If growth hormone therapy is used, monitor for undesirable side effects.
- **Hartnup disorder:** Patients are given oral nicotinamide therapy (40–200 mg/d) and oral neomycin. Psychiatric medications, such as antidepressants or mood stabilizers, may be needed.

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain measures that are appropriate to the specific condition. Encourage regular medical visits and nutritionist follow-up.
- Patients with **XLH** will need counseling about desired intake of phosphorus and vitamin D to ensure adequate growth and development.
- Patients with **Hartnup disorder** should use sunscreen and avoid sun exposure as much as necessary. They will need counseling about high-protein diets.

Patient Education—Food Safety

Foodborne illness tips are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- Hartnup Disorder
  http://www.nlm.nih.gov/medlineplus/ency/article/001201.htm
- Merck Manual—Vitamin D-Resistant Rickets
  http://www.merck.com/mmhe/print/sec11/ch146/ch146g.html
- Office of Rare Diseases
  http://rarediseases.info.nih.gov/
- Vitamin D-Resistant Rickets
  http://www.xlhnetwork.org/site/Diagnosis.html

RENAL METABOLIC DISORDERS—CITED REFERENCES

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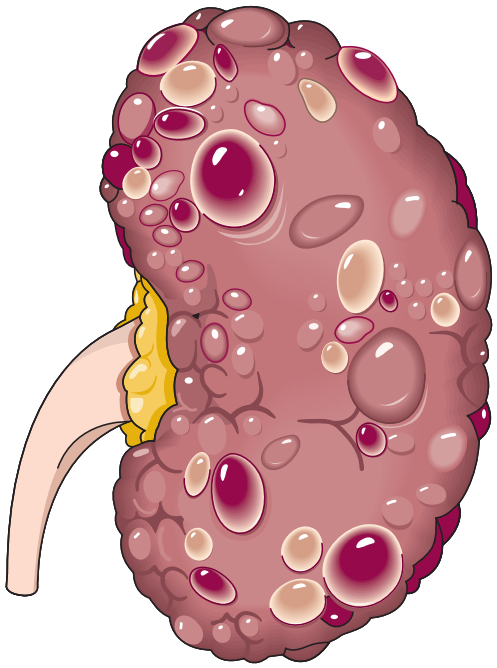
POLYCYSTIC KIDNEY DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 2



LifeART Super Anatomy Collection 9, CD-ROM. Baltimore: Lippincott Williams & Wilkins.

DEFINITIONS AND BACKGROUND

PKDs are disorders that cause multiple, fluid-filled, bilateral cysts in the kidneys and may also affect the liver, pancreas, colon, blood, and heart valves. Fluid-filled sacs or cysts of varying sizes that become larger as the disease progresses replace normal kidney tissue. Intracellular calcium signaling is important in kidney development, and defects in this signaling pathway cause the cyst formation. PKD results from loss of function of either of two proteins, polycystin-1 or polycystin-2. Increased understanding of genetic and pathophysiologic mechanisms has laid the foundation for development of more effective therapies.

PKD in its **autosomal dominant form (ADPKD)** affects 600,000 adults in the United States, at a rate of 1 in 400–1000 persons. ADPKD is one of the most common human genetic disorders. The antidiuretic hormone, arginine vasopressin (AVP), operates continuously in ADPKD patients to stimulate the formation of cyclic adenosine monophosphate (cAMP), thereby contributing to cyst and kidney enlargement and renal dysfunction (Torres et al, 2009). Patients with ADPKD remain clinically asymptomatic for decades while significant anatomic and physiologic systemic changes take place (Rizk and Chapman, 2008). Symptoms most often appear between 30 and 40 years of age. There is a close correlation between the extent of hypertension, left ventricular hypertrophy, deterioration of GFR, and progressive enlargement of the cystic kidneys in adult ADPKD (Schrier, 2009).

The rare **autosomal recessive form (ARPKD)** occurs in 1 per 44,000 births. ARPKD is the neonatal form of PKD and is associated with enlarged kidneys and biliary dysgenesis (Harris and Torres, 2009). Infants who are not treated may die before 1 month of age. Most infants with autosomal recessive PKD have unusual facial features (Potter face) and failure to thrive. Children with ARPKD experience high blood pressure, UTIs, and frequent urination. Their disease affects the liver, spleen, and pancreas, resulting in low blood cell counts, varicose veins, and hemorrhoids. Because kidney function is crucial for early physical development, children with autosomal recessive PKD are usually smaller than average size. Rare, syndromic forms of ARPKD cause defects of the eye, central nervous system, digits, or neural tube (Harris and Torres, 2009).

Treatment of PKD involves efforts to identify patients at greatest risk for disease progression, thus targeting therapy to retard disease progression and renal functional deterioration. The effect of renin–angiotensin–aldosterone system inhibition on renal volume and kidney function is being studied in the Halt Progression of Polycystic Kidney Disease (HALT PKD) trial (Schrier, 2009). Laparoscopic surgery to remove the cysts may be beneficial for some patients. Half of patients with PKD will require dialysis (Barash et al, 2010). For others, transplantation is needed.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: ADPKD is the most common life-threatening hereditary disorder (Belibi and Edelstein, 2010); it is caused by mutations in the *PKD1* gene in 85% of cases and the *PKD2* gene in the rest of the cases.

ARPKD is caused by mutation in *PKHD1*; the ARPKD protein, fibrocystin, is localized to cilia/basal body and complexes with polycystin-2 (Harris and Torres, 2009).

Clinical/History

- Length or height
- Birth weight
- Present weight
- Growth percentile
- BMI
- Diet history
- BP (abnormally high)
- Vomiting
- Chronic headaches
- UTIs
- Hematuria
- Liver or pancreatic cysts
- Abnormal heart valves
- Kidney stones
- Brain aneurysms
- Diverticulosis, hemorrhoids
- Back or side pain
- Ultrasound

Lab Work

- Proteinuria, microalbuminuria
- Serum AVP
- BUN, Creat
- H & H
- Serum Fe, ferritin
- Na^+, K^+
- Chol, Trig
- Gluc
- Serum vitamin D
- Alb, transthyretin
- CRP
- Ca^{++}, Mg^{++}
- Serum phosphorus

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Mineral Intake

Assessment Data: I & O records, nursing flow sheets. Adult 50-year-old male with ADPKD. Blood pressure very high, 190/90 mm Hg on three separate measurements. Diet hx indicates use of bacon every breakfast, luncheon meats and cheese for midday meal, and canned soups for dinner at least four times weekly.

Nutrition Diagnosis (PES): NI-5.10.7 Excessive mineral (sodium) intake related to intake of high sodium foods as evidenced by BP 200/90 mm Hg and diet hx of high sodium food choices consumed every day.

Interventions:

Food-Nutrient Delivery: Suggest alternatives for meal planning that are lower in sodium. Discuss appropriate fluid intake as well.

Education: Educate the patient and his family members about the importance of lowering blood pressure by using medications and a lower sodium diet.

Counseling: Work with family members to plan menus that are easy to prepare for daily meals; discuss tips for restaurant and shopping choices.

Coordination of Care: If high fluid intake is recommended, work with the medical team accordingly.

Monitoring and Evaluation: Blood pressure gradually down to 120/80 mm Hg. Sodium intake within 2–3 g guideline on a daily basis.

INTERVENTION

OBJECTIVES

- Prevent renal failure; manage CKD.
- Minimize or alleviate nausea, vomiting, and anorexia.
- Bring hypertension under control where present. The goal BP is 120/80 mm Hg.
- Control AVP secretion.
- Correct or alleviate proteinuria or microalbuminuria.
- Manage dialysis when and if needed. Prepare for transplantation if planned.

FOOD AND NUTRITION

- Modify diet according to symptoms; sodium restriction may be beneficial for lowering blood pressure. The DASH diet may be beneficial.
- Meet protein dietary allowance for age unless proteinuria is excessive, in which case lower levels are needed.
- Fluid intake should be tailored to the individual. Water loading, by suppressing AVP-stimulated cAMP production, is a proposed therapy for ADPKD; more trials are needed (Barash et al, 2010; Torres et al, 2009).
- Use more antioxidant-rich, anti-inflammatory foods, such as berries, apples, soy, flax oil, fish including salmon, and fish oils.

Common Drugs Used and Potential Side Effects

- ACE inhibitors are most frequently prescribed for hypertension (Jafar et al, 2005). Calcium channel blockers, diuretics, and beta-blockers may also be used. A common combined therapy is a diuretic plus an ACE inhibitor.
- Antibiotics may be used to treat infections; monitor specific medicines for nutritional side effects.
- Analgesics may be useful for pain management.
- Vasopressin V2 receptor antagonists, mTOR inhibitors, and statins that reduce cyst formation and improve renal function are being tested (Belibi and Edelstein, 2010).

Herbs, Botanicals, and Supplements

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

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain measures that are appropriate to specific condition. If high fluid intake is desirable, discuss goals and options with the patient and/or family.
- Encourage regular medical visits and nutritionist follow-up.

Patient Education—Food Safety

- Renal patients are at high risk for foodborne illness. Follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill.
- Details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- National Kidney and Urologic Diseases Information Clearinghouse http://kidney.niddk.nih.gov/kudiseases/pubs/polycystic/index.htm
- Polycystic Kidney Disease Foundation http://www.pkdcure.org

POLYCYSTIC KIDNEY DISEASE—CITED REFERENCES

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Rizk D, Chapman A. Treatment of autosomal dominant polycystic kidney disease (ADPKD): the new horizon for children with ADPKD. *Pediatr Nephrol.* 231029, 2008.

Schrier RW. Renal volume, renin-angiotensin-aldosterone system, hypertension, and left ventricular hypertrophy in patients with autosomal dominant polycystic kidney disease. *J Am Soc Nephrol.* 20:1888, 2009.

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RENAL TRANSPLANTATION

NUTRITIONAL ACUITY RANKING: LEVEL 4

DEFINITIONS AND BACKGROUND

Renal transplantation may be completed in ESRD when GFR drops to 10 mL/min. Persons with poor health or history of cancer often cannot receive a transplantation. Patient and graft survival rates, as well as long-term quality of life, have improved dramatically, a result of advances in surgical techniques, immunosuppression, and perioperative care. Laparoscopic living-donor nephrectomy has improved the possibility of live renal donations. In addition, pediatric kidney transplants are an option for children with ESRD. A child must reach a certain body surface area or weight (such as 20 kg) to receive a parent's kidney; siblings younger than 18 years are generally not allowed to donate a kidney.

Malnutrition prior to transplantation is associated with an increased risk of infection, delayed wound healing, and muscle weakness. Delaying surgery for nutrition therapy (enteral and parenteral) is indicated only if severe malnutrition is present. Preoperative nutrition therapy should be conducted prior to hospital admission to lower the risk of nosocomial infections (Weimann et al, 2009).

An adequate nutritional status may improve outcomes after transplantation. In addition, a weight loss program may be needed prior to surgery if the patient is obese. Orlistat (alli® or Xenical®) and a structured low-fat, low-calorie diet and exercise program may be beneficial (MacLaughlin et al, 2010).

After a renal transplantation, the patient has a functioning donor kidney. High doses of glucocorticoid drugs are given to prevent rejection. Elimination of donor-specific anti–human leukocyte antigen antibodies during antibody-mediated rejection (AMR) is important; proteasome inhibitor–based therapy has been shown to effectively treat refractory AMR (Walsh et al, 2010).

The acute posttransplantation phase lasts up to 2 months; the chronic phase starts after 2 months. Complications of corticosteroid use include new-onset diabetes, osteoporosis, and hyperlipidemia. In the long term, cardiovascular morbidity remains the greatest risk (Roberts et al, 2006), followed by cancer and infections. Complications are listed in Table 16-14.

Early intensive dietary advice and follow-up helps control complications after renal transplantation. Prognosis is good for transplant patients who take care of themselves and attend to medical follow-up.

TABLE 16-14 Complications After Renal Transplantation

Complication	Description
Anemia	Posttransplantation anemia is a prevalent and undertreated condition.
Cancer	Lymphomas may occur. Malignancies are rapidly becoming a major cause of mortality.
Cardiovascular complications	Hyperlipidemia is one of the consequences of long-term use of corticosteroids. Elevated triglycerides and metabolic syndrome can also persist. Renal transplantation recipients are at high risk for ischemic heart disease or heart failure and will benefit from lifestyle modifications.
Diabetes	Patients are at risk for posttransplantation diabetes because of the use of corticosteroids. Higher BMI, elevated fasting blood glucose levels, and high blood pressure should be managed carefully.
Infection	Acute meningitis or Guillain–Barré syndrome triggered by *Campylobacter jejuni* infection are examples of infections in this population.
Neurological complications	Neurotoxic immunosuppressive medications may cause mild symptoms, such as tremors and paresthesia.
Osteoporosis	Bone density declines after long-term use of corticosteroids. Tertiary hyperparathyroidism may occur after kidney transplantation.
Pulmonary complications	Infectious pulmonary complications may occur. Noninfectious pulmonary complications arise because of the toxicity of posttransplantation medications.
Stroke	Stroke may occur in a small number of renal transplant recipients.
Weight gain or obesity	Obesity decreases effectiveness of insulin receptors, increasing the tendency for glucose intolerance. It may also delay wound healing.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Renal transplantation may be needed for some of the inherited disorders, such as PKD.

Clinical/History	Lab Work	
Height	CRP	tHcy levels
Present weight	Alb	Lipid profile (LDL goal <100 mg/dL)
aBWef	Ca^{++}, Mg^{++}	Trig
Edema	Phosphorus	N balance
BMI	Na^{+}, K^{+}	GFR, CrCl
Weight changes	H & H	Serum phosphorus
Diet history	Serum Fe	AST, ALT
I & O	BUN, Creat	Bilirubin
BP	GFR	PTH
Temperature	WBC	
DEXA scan	Total lymphocyte count (TLC)	
	Gluc	

SAMPLE NUTRITION CARE PROCESS STEPS

Obesity

Assessment Data: Female patient 6 months posttransplant with weight gain of 30#; BMI now 32. Diet history indicates patient is eating small meals every 2–3 hours, consuming 300–400 kcal in excess of goal intake of 1700 kcal/d. Glucose and lipid levels slightly elevated over past reports.

Nutrition Diagnosis (PES): Obesity related to excess oral food and beverage intake as evidenced by weight gain of 30# since transplant 6 months ago and current BMI 32.

Interventions:

Education: Reduce overall energy intake to the goal range of 1700 kcal/d. Suggest lower calorie alternatives for food choices and preparation methods.

Counseling: Develop menu plans together to support goal achievement.

Coordination of Care: Discuss plans with medical team to support the patient goals during monthly clinic visits. Patient will see RD again in 3 months.

Monitoring and Evaluation: Improved weight status; loss of 10# in 3 months. Patient expresses satisfaction with meeting weight loss goal thus far. Lab values within normal limits for glucose and lipids.

INTERVENTION

OBJECTIVES

- Promote healing and prevent infections, especially during acute phase.
- Prevent or control AMR.
- Support immunity and prevent new infections.
- Food intake is recommended within 24 hours postsurgery. Modify diet according to drug therapy to enhance outcome.
- Initiate immediate postoperative EN or PN in patients who did not receive oral intake for more than 7 days perioperatively or whose oral food intake is less than 60–80% for more than 14 days (Weimann et al, 2009).
- Prevent abnormalities in calcium or phosphorus metabolism with HPT.
- Monitor for abnormal electrolyte levels (sodium, potassium). Control blood pressure carefully to prevent cardiac problems.
- Maintain good blood pressure control and near-normal fasting blood glucose levels and HbA1c levels. Manage CHO intake, but provide enough energy to spare protein for healing.
- Manage fluid intake according to intake and output. Most patients can return to a normal or increased fluid intake after transplantation.
- Help patient adjust to a lifelong medical regimen during chronic phase. Improve survival rate by supporting immune response.
- Correct or manage complications that occur, such as posttransplantation anemia.
- Minimize long-term weight gain.

FOOD AND NUTRITION

- Progress to solids as quickly as possibly postoperatively. Monitor fluid status and adjust as needed.
- Energy should be calculated as 30–35 kcal/kg. Needs may increase with postoperative complications.
- Daily intake of protein should be 1.3–2.0 g/kg in the acute phase and 0.8–1.0 g/kg in the chronic phase. Soy proteins can be used in most cases and may help lower LDL cholesterol level.
- Control CHO intake; 50% of total calories is usual. Limit concentrated sweets and encourage the use of complex CHO.
- Encourage monounsaturated fats, omega-3 fatty acids, and the Mediterranean diet. Use more fish and fish oils and olive oil.
- Fluid is not restricted unless there are problems with graft functioning.
- Daily intake of sodium should be 2–4 g until drug regimen is reduced. Careful management of sodium efficiently controls blood pressure in patients who are hypertensive (Keven et al, 2006).
- Adjust potassium levels as needed (2–4 g if hyperkalemic).
- Daily intake of calcium should be 1200–1500 mg. Children especially will need adequate calcium for growth.
- Supplement diet with vitamin D, magnesium, phosphorus, and thiamine if needed. If homocysteine-lowering multivitamin therapy is needed; folic acid and vitamins B_6 and B_{12} are needed.
- Reduce gastric irritants as necessary, if GI distress or reflux occurs.
- Encourage exercise and a weight control plan for the long-term recovery phase.

Common Drugs Used and Potential Side Effects

- See Table 16-15 for a description of immunosuppressive drugs that are used.
- Calcium or vitamin D supplements may be needed to correct osteopenia.
- Most adult renal transplantation recipients develop dyslipidemia within 1 month of the initiation of immunosuppressive therapy. Statins and diet therapy are required.
- Chronic allograft nephropathy remains a cause of graft attrition over time (Afzali et al, 2005). Switching to tacrolimus due to cyclosporine-related side effects improves disease-specific quality-of-life indicators within a short time (Franke et al, 2006).

Herbs, Botanicals, and Supplements

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

TABLE 16-15 Immunosuppressant Drugs Used After Renal Transplantation

Drugs may be either *induction agents*, powerful drugs at the time of transplant, or *maintenance agents* used for long-term antirejection. Most patients are on a combination of the drugs listed below. Many transplant centers use tacrolimus, mycophenolate mofetil, and prednisone.

Medication	Comments
Antiproliferative agents: (mycophenolate mofetil, mycophenolate sodium, azathioprine)	These drugs lower the number of T cells and may cause leukopenia, thrombocytopenia, oral and esophageal sores, macrocytic anemia, pancreatitis, vomiting, diarrhea, and other complex side effects. Folate supplementation, liquid or soft diet, and use of oral supplements may be needed.
Belatacept	This is an investigational biologic agent, injected following renal transplantation to prevent rejection, maintain kidney function, and sustain lower blood pressure. "Posttransplant lymphoproliferative disorder"—a type of lymphoma associated with organ-transplant patients is one risk.
Corticosteroids (prednisone, Solu-Cortef)	Used for immunosuppression. Side effects include increased catabolism of proteins, negative nitrogen balance, hyperphagia, ulcers, decreased glucose tolerance, sodium retention, fluid retention, and bone thinning/osteoporosis. Cushing syndrome, obesity, muscle wasting, and increased gastric secretion may result. A higher protein intake and controlled carbohydrate intake are needed.
Calcineurin inhibitors: Cyclosporine (Sandimmune, Neoral, Gengraf)	Cyclosporine does not retain sodium as much as corticosteroids. Nausea, vomiting, and diarrhea are common side effects. Hyperlipidemia, hypertension, and hyperkalemia, hyperglycemia, hyperlipidemia, hair growth, gum enlargement, or tremors may occur. The drug is also nephrotoxic; a controlled renal diet may be beneficial.
Calcineurin inhibitor: Tacrolimus (Prograf, FK506)	Being 100 times more potent than cyclosporine, smaller doses are required to suppress T-cell immunity. Side effects include gastrointestinal distress, nausea, vomiting, hyperkalemia, headaches, hair loss, tremors, and hyperglycemia.
Monoclonal antibodies (mAbs) (basiliximab, daclizumab)	Less nephrotoxic than cyclosporine but can cause nausea, anorexia, diarrhea, fever, stomatitis, and vomiting. Monitor carefully. Anti-CD25 mAbs (basiliximab and daclizumab) are well tolerated. Anti-CD52 (Campath-1 H), anti-CD20 (rituximab), anti-LFA-1, anti-ICAM-1, and anti-tumor necrosis factor-α (infliximab) show potential.
Proteasome inhibitors (bortezomib, rituximab)	Proteasome inhibitor–based therapy may reduce early graft rejection (Walsh et al, 2010).
mTOR inhibitor (sirolimus)	Side effects may include rash, bone marrow problems (anemia, low white blood cell count and low platelet count), swelling of ankles, and frothy urine because of protein leakage from urine.

REFERENCE
Walsh RC, et al. Proteasome inhibitor-based primary therapy for antibody-mediated renal allograft rejection. *Transplantation*. 89:277, 2010.

- With cyclosporine, avoid use with echinacea and St. John's wort because of counterproductive effects on the drug.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Share, as appropriate, details of the DASH diet and Mediterranean diet. Discuss food sources of protein, calcium, magnesium, potassium, and sodium.
- Increase intakes of fiber-rich foods as a primary preventive approach against metabolic syndrome and cardiovascular disease, which are very prevalent after renal transplant (Noori et al, 2010).
- Avoid smoking and keep alcohol intake to a minimum.
- Rigorous efforts should be made to optimize weight before and after solid-organ transplantation by a judicious combination of diet, exercise, minimization of steroid therapy, surgery, and psychological therapies. Encourage moderation in diet; promote adequate exercise.
- Discuss the importance of bone health and how diet affects prevention of osteoporosis. If patient does not drink milk, describe other sources of calcium.
- Patients should learn how to apply self-management and when to seek medical attention. UTIs are the most common infection after renal transplantation.

Patient Education—Food Safety

- Foodborne illness can occur when there is contamination of food at any point during the preparation process. Follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill.
- Details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- MEDLINE—Renal Transplantation
  http://www.nlm.nih.gov/medlineplus/ency/article/003005.htm
- NKF—Transplantation
  http://www.kidney.org/atoz/atozTopic_Transplantation.cfm
- Nephrology Channel
  http://www.nephrologychannel.com/rrt/transplant.shtml
- Transplant Experience
  http://www.transplantexperience.com/kidney.php
- University of Maryland Transplant Center
  http://www.umm.edu/transplant/kidney/index.html

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URINARY TRACT INFECTIONS

NUTRITIONAL ACUITY RANKING: LEVEL 1 (3 IF CHRONIC)

DEFINITIONS AND BACKGROUND

UTIs are associated with significant morbidity (Nanda and Juthani-Mehta, 2009). Single UTI episodes are very common, especially in adult women where there is a 50-fold predominance compared with adult men (Guay, 2009).

Acute pyelonephritis affects the renal parenchyma and is the most common cause of UTIs. *E. coli* most often cause upper UTIs; other organisms are found in complicated infections associated with diabetes mellitus (e.g., *Candida* spp), urinary stones, or immunosuppression. With effective antibacterial therapy, the immune response by both T and B lymphocytes supports antibodies that can eradicate the infectious agent.

There are approximately 250,000 cases of acute pyelonephritis each year. When the patient is septic, hospitalization and treatment with parenteral antibiotics may be needed. Indications for inpatient treatment include complicated infections, sepsis, persistent vomiting, failed outpatient treatment, or extremes of age (Ramakrishnan and Scheid, 2005). If patient does not improve rapidly, ultrasound and computed tomography are used to diagnose obstruction, abscess, or emphysematous pyelonephritis. Emphysematous pyelonephritis is a life-threatening infection especially seen in patients with poorly controlled diabetes mellitus. Mortality is high in that population, especially in the elderly. Most complications are treated percutaneously with broad-spectrum antibiotics and fluid resuscitation, followed by surgical therapy if needed.

In acute pyelonephritis in pregnancy, changes in innate immunity are present. Low maternal plasma concentrations of adiponectin are found in these cases; adiponectin has profound anti-inflammatory properties (Mazaki-Tovi et al, 2010). Further study is needed to identify measures that can be taken to enhance immunity during pregnancy.

Chronic pyelonephritis results from treatment failure and may be caused by resistant organisms, underlying anatomic/functional abnormalities, or immunosuppressed states (Ramakrishnan and Scheid, 2005). Fibrosis, scarring, and dilatation of the tubules impair renal function. Scarring from chronic pyelonephritis leads to loss of renal tissue and function, even progressing to ESRD. Hypertension is often present in this population.

Cystitis affects the lower urinary tract. **Interstitial cystitis (IC)** is a painful bladder syndrome with an inflamed bladder wall, pelvic discomfort, and urinary frequency and urgency. Omission of alcohol, caffeine, citrus beverages, and tomatoes gives relief to some individuals.

Cranberry inhibits the adhesion of uropathogens through anthocyanidin/proanthocyanidin as potent antiadhesion compounds (Guay, 2009). It may decrease the number of symptomatic UTIs over a 12-month period, particularly for women, but it is not clear whether juice, tablets, or capsules are the most effective source (Jepson and Craig, 208). GI intolerance, weight gain from excessive calorie load, and drug–cranberry interactions due to the inhibitory effect of flavonoids on cytochrome P450–mediated drug metabolism may occur (Guay, 2009).

Urinary incontinence requires attention, even if there are no UTIs. Check for vitamin B_{12} deficiency, decrease caffeine intake, and try bladder training (use of toilet every 2 hours). Maintain adequate fluid intake to prevent the onset of any UTIs. Women may need guidance on pelvic floor exercises to strengthen those muscles after childbirth or menopause.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: UTIs are not genetic, but many inherited conditions can aggravate them. Elevated procalcitonin is a biomarker that differentiates lower UTI from pyelonephritis in the pediatric age group (Nanda and Juthani-Mehta, 2009).

Clinical/History	Edema	BP (increased?)
Height	Diet history	Intravenous pyelogram
Weight	Temperature	
BMI	I & O	

Lab Work

Urine culture
Procalcitonin (elevated?)
Alb, transthyretin
CRP
Transferrin
H & H
Serum Fe, ferritin
Ca^{++}, Mg^{++}
Serum phosphorus
Na^{+}, K^{+}
Urinary Na^{+}
Gluc
RBP
BUN, Creat

INTERVENTION

OBJECTIVES

- Preserve kidney function.
- Control blood pressure.
- Acidify urine to decrease additional bacterial growth. Prevent bacteremia.
- Force fluids unless contraindicated.

FOOD AND NUTRITION

- Restrict protein intake only if renal function is decreased. Otherwise, use sufficient amounts of high biological–value proteins, including foods such as meat, fish, poultry, eggs, and cheese.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Oral Food-Beverage Intake

Assessment Data: I & O records, nursing flow sheets for hospitalized elderly patient, coming from long-term care facility with UTI and temperature of 102°F. Diet Hx reveals poor intake for 3 days related to fever and anorexia. Signs of dehydration.

Nutrition Diagnosis (PES): NI-2.1 Inadequate oral food and beverage intake related to UTI with fever and resulting anorexia as evidenced by temperature of 102°F and I & O records showing very low intake for 3 days.

Interventions:

Food-Nutrient Delivery: Add extra fluids to trays and encourage 4 oz intake with all medications 3 times daily; goal is 30–35 mL/kg daily. Offer preferred foods; enhance with extra calories as needed (such as extra gravy with potatoes, extra margarine on bread). Include yogurt and cranberry juice if tolerated.

Education: Discuss the need to increase intake of fluids and nutrient-dense foods to support immunity, fight the infection, and reduce fever.

Counseling: Discuss food choices and ways to enhance total intake without being overwhelmed by large quantities of food.

Coordination of Care: Support the antibiotic therapy by providing high-quality food choices.

Monitoring and Evaluation: Temperature back to normal; resolution of UTI. Improved food and nutrient intake. Able to be discharged from hospital with guidance on how to prevent further dehydration and UTIs.

- Cranberries, plums, and prunes produce hippuric acid, which acidifies urine. Corn, lentils, breads/starches, peanuts, and walnuts also acidify the urine.
- Although vitamin C is not necessarily effective in lowering the UpH, sufficient levels of intake are needed to stimulate the anti-infective process.
- Avoid an excess of caffeine because of its diuretic effect. Stimulants such as caffeine rapidly leave the bladder, a vulnerable site where additional infections may begin.
- Vitamin A may be low; encourage improved intake, especially from beta-carotene–rich foods.
- Non-antimicrobial-based approaches undergoing investigation include probiotics, vaccines, oligosaccharide inhibitors of bacterial adherence and colonization, and bacterial interference with immunoreactive extracts of *E. coli* (Guay, 2009). Offer probiotic choices, such as yogurt or kefir, to replenish good intestinal bacteria.

Common Drugs Used and Potential Side Effects

- Outpatient oral antibiotic therapy with a fluoroquinolone is successful in most patients with mild uncomplicated pyelonephritis (Ramakrishnan and Scheid, 2005).
- Ceftriaxone and gentamicin are cost-effective because only once-daily dosing is needed. With urinary anti-infectives, sufficient water and fluids should be ingested. Be careful with forced water diuresis, which impairs antibiotic effectiveness. Monitor responses to glucose changes in people with diabetes. Avoid use with alcohol.
- Liposome treatments are under study for interstitial cystitis. The interstitial cystitis-related liposomes coat the bladder and may reduce inflammation there.
- Nitrofurantoin (Furadantin, Macrodantin) should be consumed with food, milk, and a diet adequate in protein. Nausea, vomiting, and anorexia are common; diarrhea is rare.
- Penicillin products such as amoxicillin (Amoxil, Trimox, Wymox) and ampicillin may be used. With penicillin allergy, vancomycin may be used.
- Quinolones include ofloxacin (Floxin), norfloxacin (Noroxin), ciprofloxacin (Cipro), and trovafloxacin (Trovan). If Cipro (ciprofloxacin) is used, avoid taking with calcium supplements, milk, and yogurt; limit use of caffeine and monitor for nausea. If a fluoroquinolone (Floxin, Maxaquin) is used, nausea is a side effect. Take separately from vitamin supplements.
- Sulfisoxazole (Gantrisin) can deplete folacin and vitamin K. Nausea and vomiting may also occur.
- Trimethoprim (Trimpex) and trimethoprim/sulfamethoxazole (Bactrim, Septra, Cotrim) may cause diarrhea, GI distress, and stomatitis. Use adequate fluid.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Probiotics may be useful to prevent and treat recurrent complicated and uncomplicated UTIs. Blueberry, parsley, bearberry, yogurt, birch, and couch grass have been suggested, but no clinical trials have proven efficacy.

- Cranberry juice may be useful for women but not necessarily for the elderly who require catheterization (Jepson and Craig, 2008). It may be useful also for pregnant women (Wing et al, 2008).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Indicate which foods are palatable as sources of nutrients for dietary restrictions and for nutrients that tend to be low.
- Encourage appropriate fluid intake. Schedule voiding frequency and fluid intake if needed.
- Discuss the use of probiotics to support a healthy immune system, especially after antibiotic use.
- Limit caffeine and oral fluid intake at night, if needed.
- Showers, instead of baths, may be preventive.

Patient Education—Food Safety

Foodborne illness can occur when there is contamination of food at any point during the preparation process. Because renal patients are at high risk for foodborne illness, follow the four-step guidelines established by the U.S. Department of Agriculture: clean, separate, cook, and chill. More details are available at the Kidney Foundation Web site at http://www.kidney.org/atoz/content/foodsafety.cfm.

For More Information

- Interstitial Cystitis Association
  http://www.ichelp.org/
- Mayo Clinic—Pyelonephritis
  http://www.mayoclinic.com/health/kidney-infection/DS00593
- National Bladder Foundation
  http://www.bladder.org/

PYELONEPHRITIS AND URINARY TRACT INFECTIONS—CITED REFERENCES

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SECTION 17

Enteral and Parenteral Nutrition Therapy

CHIEF ASSESSMENT FACTORS

- Availability of Appropriate Lab Work
- Benefits of Nutritional Intervention Outweigh Risks?
- Inadequate Oral Intake (Mechanical, Gastrointestinal, Psychological, or Surgical Reasons)
- Indirect Calorimetry (IC)[a]
- Nitrogen Balance[b]
- Other Planned Procedures and Impact of Delayed Nutrition Therapy
- Presence or Absence of Sepsis (in EN vs. PN)
- Risk for Refeeding Syndrome in Patients with Poor Intake, Maldigestion, and Malabsorption[c]
- Serial Anthropometric Measures
- Signs of Micronutrient Deficiency (selenium, copper, zinc)
- Systemic Inflammatory Response to Injury or Infection or Protein-Energy Undernutrition
- Surgery Affecting Oral Intake, Digestion, or Absorption
- Treatment, Timing, and Condition:
  - Cancer Having Intake or GI Consequences (Radiation, Chemotherapy, Surgery)
  - Cystic Fibrosis
  - Failure to Thrive, Chronic Malnutrition
  - GI Obstruction, Chronic Diarrhea, Crohn's Disease, Short Bowel Syndrome (SBS)
  - GI Disease from HIV Infection or AIDS
  - Organ Transplantation
  - Pancreatic Disease
  - Pulmonary Aspiration, Complications, or Ventilator Use
  - Sepsis, Trauma, Burns, or Other Causes of High Rates of Catabolism

[a]Indirect Calorimetry = measure of gas exchange based on oxygen used and carbon dioxide released, using the formula RQ = VCO_2/VO_2)

[b]Nitrogen Balance = net gain or loss of nitrogen per day; helps to determine if patient is in catabolic or anabolic state. 1 g Nitrogen = 6.25-g protein. N balance calculation = 24 hour protein intake (total g)/6.25 minus (24 hour UUN + 4)

Key: 0 balance implies equilibrium. 4–6 g (+) is anabolic/positive balance. Negative (−) implies catabolism. Goals: To maintain status, use 1-g N:200–300 kcal. For anabolism, use 1-g N:150 kcal. Interpretation: renal disease, liver disease, burns, or trauma may skew the results.

[c]Patients at risk for refeeding syndrome may have low serum levels of phosphorus (<3.0 mg/dL), magnesium (<1.3 mg/d/L), and potassium (<3.5 mEq/L) from poor intake. Feeding causes increased demand for these electrolytes so the macronutrients provided can be utilized. The increased metabolic demand causes further drops in these levels

unless they are repleted BEFORE feeding is initiated. Reintroduction of carbohydrate after a period of 24–72 hours of fasting, when blood glucose and insulin levels have both declined, sends out a surge of insulin. This insulin surge pulls glucose and the electrolytes into the cells, leading to hypophosphatemia (<1.0–1.5 mg/dL), hypomagnesemia (<1 mg/dL), and hypokalemia (<2.5 mEq/dL) and life-threatening results, as it has cardiac, pulmonary, hepatic, renal, neuromuscular, metabolic, and hematological consequences (Tresley and Sheean, 2008).

Note: Jensen et al (2009) indicate that the presence or absence of the systemic inflammatory response and whether the inflammation is severe or sustained distinguishes the forms of malnutrition described here. **Sarcopenia** is a smoldering inflammatory state partially driven by cytokines and oxidative stress, cachexia overlapping with failure to thrive. **Cachexia** as a systemic proinflammatory process with associated insulin resistance, increased lipolysis, increased lipid oxidation, increased protein turnover, and loss of body fat and muscle, as in pancreatic cancer or organ failure. **Marasmus** is pure starvation with reduced food intake or assimilation with loss of body cell mass and weight, but no underlying inflammatory condition; visceral proteins are generally preserved and edema is not present. If marasmic individuals subsequently develop inflammatory conditions and edema, the term **marasmic kwashiorkor** is used.

OVERVIEW OF ENTERAL AND PARENTERAL NUTRITION THERAPY

There are about 1000 kcal available as glucose or glycogen in the muscles, liver, and bloodstream, supplying energy for only 18–24 hours. Daily glucose replacement is crucial for brain and red blood cell survival. When oral feeding is not possible or safe, nutrients should be replaced by other means. Specialized nutrition therapy includes both tube (enteral) and IV (parenteral) feeding methods and must be carefully planned and administered.

Nutrition therapy has grown steadily over the past four decades. Table 17-1 lists definitions important in nutrition therapy. Initially, central parenteral nutrition (CPN, formerly total parenteral nutrition or TPN) was the ultimate standard of care. Later, tube feeding (enteral nutrition [EN]) was found to protect gut immunity and function more effectively. A critically ill patient who has been inadequately fed for 10–14 days will manifest characteristics of both the systemic inflammatory response and protein-energy undernutrition, even loss of up to 30% of body cell mass; nutrition modulation of the systemic inflammatory response can occur with early feeding (Jensen et al, 2009). Integrated approaches are required, including anti-inflammatory diets, glycemic control, physical activity and resistance training, appetite stimulants, anabolic agents, anti-inflammatory agents, anti-cytokines, and probiotics (Jensen, 2006). Because there are over 500 types of bacteria living in the human gut, the goal is to keep the healthy ones and reduce those that are detrimental. Temperature, osmolality, pH, and substrate availability can alter the host–bacteria relationship very quickly. Probiotics may help maintain the beneficial flora and reduce

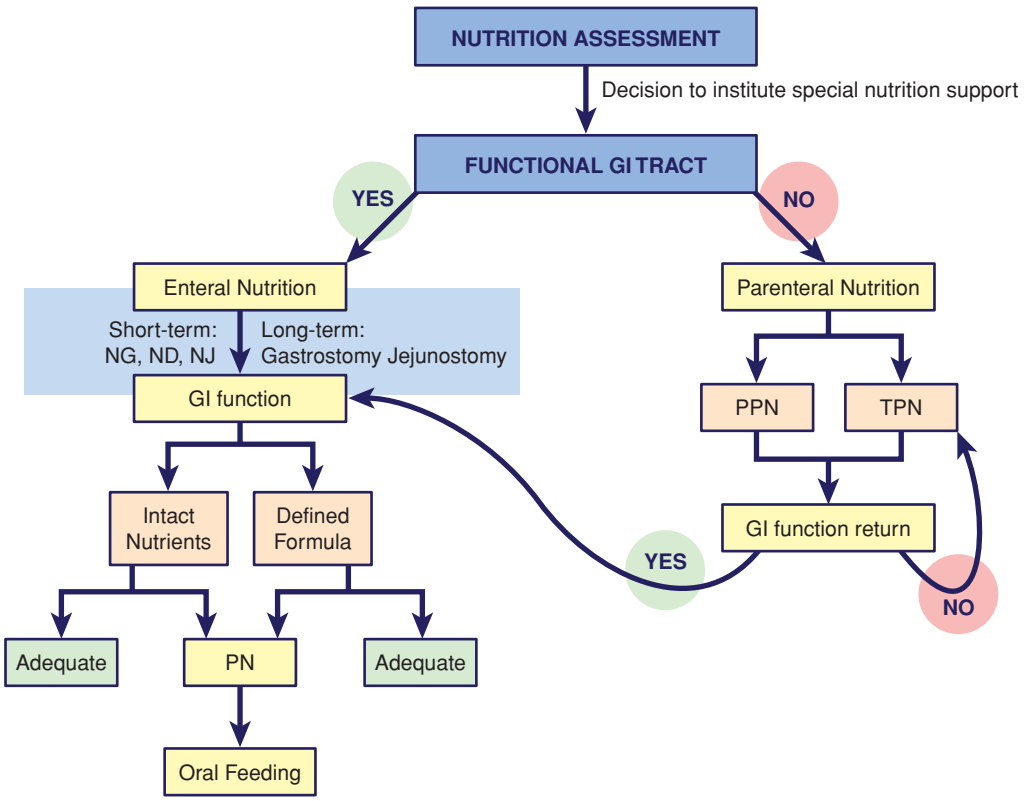


TABLE 17-1 ASPEN Definition of Terms Related to Nutrition Support

Admixture: The result of combining two or more fluids

Closed Enteral System: A closed enteral container prefilled with sterile, liquid formula by the manufacturer, which is considered ready to hang

Compatibility: The ability to combine two or more products such that the physical integrity and stability of each product is not altered when combined

Formulation: A mixture of nutrients suitable for administration to a patient

Hang Time: The length of time a formulation is considered safe for administration to the patient beginning with the time the formulation has been compounded, reconstituted, warmed, decanted, or has had the original package seal broken

Intravenous fat emulsion (IVFE): An IV oil-in-water emulsion of oils, egg phosphatides, and glycerin; the term is used in preference to lipids

Nutrition Support Process: The assessment, diagnosis, ordering, preparation, distribution, administration, and monitoring of nutrition support therapy

Nutrition Support Service (or Team): An interdisciplinary group that may include physicians, nurses, dietitians, pharmacists, and/or other healthcare professionals with expertise in nutrition who manage the provision of nutrition support therapy

Osmolality: The measured osmotic concentration of a liquid expressed in osmoles or milliosmoles per kilogram of solvent (Osmol per kg or mOsmol per kg, respectively). Osmolality indicates the osmotic pressure exerted by a liquid across a semipermeable membrane

Modular Enteral Feeding: Formulation created by combination of separate nutrient sources or by modification of existing formulations

Multichamber Bag: A container designed to promote extended stability of a parenteral nutrition formulation by separating some components (e.g., IV fat emulsion) from the rest of the formulation

Parenteral Nutrition: The IV administration of nutrients

- Central (CPN): Parenteral nutrition delivered into a large-diameter vein, usually the superior vena cava adjacent to the right atrium
- Peripheral (PPN): Parenteral nutrition delivered into a peripheral vein, usually of the hand or forearm

Preparation: A food, drug, or dietary supplement (or mixtures thereof) compounded in a licensed pharmacy or other healthcare-related facility pursuant to the order of a licensed prescriber

Product: A commercially manufactured food, drug, or dietary supplement. Drug products are accompanied by full prescribing information, which is commonly known as the Food Drug Administration-approved manufacturer's labeling or product package insert

Stability: The extent to which a product retains, within specified limits and throughout its period of storage and use (i.e., its shelf life), the same properties and characteristics that it possessed at the time of its manufacture

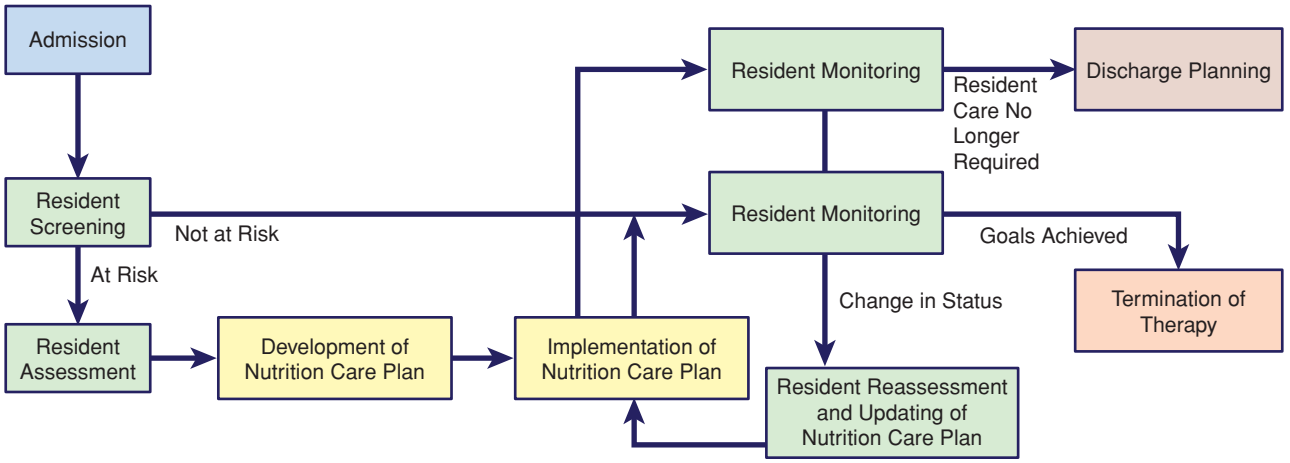
Standardized Parenteral Nutrition Formulation: A parenteral nutrition formulation intended to meet the daily maintenance requirements of a specific patient population (e.g., age-, stress-, or disease state specific) and differentiated by the route of administration (central vs. peripheral vein)

Total Nutrient Admixture: A parenteral nutrition formulation containing IV fat emulsion as well as the other components of parenteral nutrition (dextrose, amino acids, vitamins, minerals, water, and other additives) in a single container

Vascular Access Device: Catheter placed directly into the arterial or venous system for infusion therapy and/or phlebotomy

Adapted from: ASPEN, http://www.nutritioncare.org/, accessed March 15, 2010,

those that are pathogenic. This can be especially useful in patients with *Clostridium difficile* or vancomycin-resistant enterococci (VRE) colonization and may even prevent ventilator-associated pneumonia. Taking yogurt containing live *Lactobacillus* GG daily as 100 g for at least 1 month may help. More studies are warranted. The American Society for Parenteral and Enteral Nutrition (ASPEN) recently updated their Clinical Guidelines for various ages and facilities (ASPEN, 2009). ASPEN has published a series of related documents that may be referred to when using these guidelines, available at their web site at: http://www.nutritioncare.org/Library.aspx. For pediatrics, where nutrition therapy is needed for many acute or chronic conditions, various guidelines are also available from ASPEN. One of the more recent documents addresses management of the obese, hospitalized pediatric patient (ASPEN, 2010). There are ASPEN guidelines for managing critically ill patients who need nutrition therapy and will be in the ICU for 2–3 days or longer (McClave et al, 2009). Critically ill patients are at high risk for infections, organ dysfunction, and death. Feeding obese, critically ill adults is a bigger challenge; they are at high risk for infectious complications, slow healing postoperative wounds, nosocomial infections, and mortality. Protein stores are mobilized and less protein is synthesized during critical illness. The goal for feeding the critically ill obese patient is to attenuate hypermetabolism, lessen inflammation, and minimize catabolic losses. If the critically ill ICU patient is hemodynamically stable with a functional gastrointestinal (GI) tract, EN is recommended over parenteral nutrition (PN) because it has benefits such as less septic morbidity, fewer infectious complications, and significant cost savings (American Dietetic Association, 2010). Nutrition therapy is not always applied effectively or consistently, despite scientific evidence and protocols. PN must be carefully managed, especially since overfeeding may aggravate sepsis. Generally, if well managed, CPN is an effective alternative to EN in patients who cannot be fed via the GI tract. Payments for PN infusions, EN formulas, and prosthetic devices are managed under the U.S. federal guidelines. Therefore, prudent use of expensive products is expected. Generally, EN is preferred over PN where possible (American Dietetic Association, 2010; McClave et al, 2009). Advances in nutrition therapy and technology have contributed to improved quality of life for many patients in hospitals, long-term care, and home settings. Note that special guidelines should be used in home care, including an evaluation for sanitary preparation, administration, and storage of the products (Kovacevich et al, 2006). Home preparation tips for EN and parenteral (PN) therapies are available from a number



of hospitals, web sites, and formula companies. Selection of patients in long-term care who could receive tube feeding (TF) must include consideration of long-term goals and ethical issues. Because of the potential complications of permanent enteral access, percutaneous gastrostomy (PEG) or jejunostomy (PEJ) should be considered only when anticipated length of use is 1 month or longer (McMahon et al, 2005). When considering initiation of feedings, the treatment goal should be taken into account, whether palliative, curative, or rehabilitative. Additionally, the patient's wishes should be considered above all. In long-term care, the following flow chart is useful in designing protocols.

In a **coma (persistent vegetative state)**, the patient is completely unaware of self and the environment. This is accompanied by normal sleep–wake cycles and hypothalamic/brainstem autonomic functions. Coma may occur as a complication of an underlying illness or as a result of injury. Everyone who is in a coma has the nutrition diagnosis of "inadequate oral food and beverage intake" and requires some form of nutrition therapy to prevent malnutrition. While medically assisted nutrition can maintain life, it is considered futile if it cannot improve the prognosis, comfort, or general status of health of an individual (Andrews and Marian, 2006). Nutrition therapy has not necessarily improved outcomes in end-stage cancer, dementia, or other terminal illnesses. Many healthy elderly persons do not desire to be tube fed, especially with advanced disease or dementia. EN is of questionable benefit for nursing home residents with advanced dementia, yet two thirds of U.S. nursing home residents on EN had their feeding tube inserted during an acute care hospitalization; this practice should be discouraged (Teno et al, 2010). Quality of life must be considered. Some patients have "nutritional distress" if they cannot eat orally (Winkler, 2010). Other individuals receiving long-term EN or home PN have expressed anger, anxiety, or depression resulting from losses of independence and control of body functions and from the altered psychological and emotional aspects of eating (Winkler, 2007). Dietitians must help the patient, family, or guardian in deciding whether to initiate, withhold, or withdraw nutrition therapy (Table 17-2). The social, legal, emotional, and ethical needs and wants must be considered

TABLE 17-2 Ethics of Nutrition Support Therapy and End-of-Life Care

ASPEN Guidelines on Withholding or Withdrawing Nutrition Support
Withholding or withdrawing nutrition support therapy often involves different considerations than other life-sustaining therapies, in part because of emotional, religious, and symbolic meanings
Legally and ethically, nutrition support therapy should be considered a medical therapy
The decision to receive or refuse nutrition support therapy should reflect the autonomy and wishes of the patient. The benefits and burdens of nutrition support therapy, and the interventions required to deliver it, should be considered before offering this therapy
Care providers should be familiar with current evidence of the benefits and burdens of nutrition support therapy
Patients should be encouraged to have living wills and/or advance directives and to discuss with their loved ones their wishes in the event of a serious or terminal accident or disease. This directive should include nutrition support therapy
Competent patients or the legal surrogate of incompetent patients shall be involved in decisions regarding withholding or withdrawing of treatment
Incompetent patients' wishes (as documented in advance directives) shall be considered in making decisions to withhold/withdraw nutrition support therapy.
If there is no family and the patient is not competent, consider a conference with the physician, nursing director, social worker, dietitian to discuss the feeding options
Nutrition support therapy should be modified or discontinued when there are disproportionate burdens or when benefit can no longer be demonstrated
Institutions should develop clear policies regarding the withdrawal or withholding of nutrition support therapy and communicate these policies to patients in accordance with the Patient Self-Determination Act

(continued)

TABLE 17-2 Ethics of Nutrition Support Therapy and End-of-Life Care *(continued)*

Artificial Nutrition at the End of Life
Treat each case individually
When oral feeding is medically appropriate as per swallowing examination, do not artificially feed. Minimize suffering and discomfort; provide comfort foods without dietary restrictions
In dysphagia without complications, recommend as patient usually benefits
In dysphagia with complications, determine if patient will equivocally benefit. Will the benefits outweigh potential risks?
In persistent vegetative state, do not recommend as patients are unable to experience satisfying quality of life
The Dying Process, Palliative Care, Hospice
Dickinson Law School: Starvation is a long, drawn out, and painful process that can take anywhere from 30 to 60 days. Dying patients who stop taking in food and fluids *do not* starve to death. While the body can sustain itself for up to 2 months without food, it can sustain itself no more than about 2 weeks (at most) without fluid intake. Unlike starvation, dehydration is typically not a painful or even an uncomfortable process, especially when good comfort care measures are undertaken. Many patients report less discomfort, and there is less request for pain medication as dehydration runs its course
Patients who stop taking food and fluids drift into a state of unconsciousness. This phase of the process may take 5–8 days if the patient is fully hydrated when food and fluid intake is stopped. Patients will typically die peacefully several days after that. If the patient is already partly dehydrated when fluid intake is stopped, the dying process will be compressed and may only last a couple of days or less
American Academy of Hospice and Palliative Medicine: While most patients with complex medical conditions at the end of life do not experience hunger even with low energy intake, they do experience dry mouth
American Nurses Association: It is not common to hydrate in the presence of symptoms of edema or vomiting
Pediatrics: There is a need for more ethics education and more interdisciplinary discussion of inherently complex and stressful pediatric end of life cases. Appropriate goals of care and use of medically supplied nutrition and hydration are part of this educational process (Solomon et al, 2005).

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with each individual. Full authorization to write diet, EN, and CPN orders expedites patient-centered care and expands the dietitian's responsibilities beyond traditional dietetic practice (Braga et al, 2006). When dietitians are granted full authority to implement nutritional recommendations, they write orders on the physician's order sheets, change existing orders, and implement new orders immediately. While state licensure boards differ on their defined scopes of practice, several have approved order writing capacity by dietitians.

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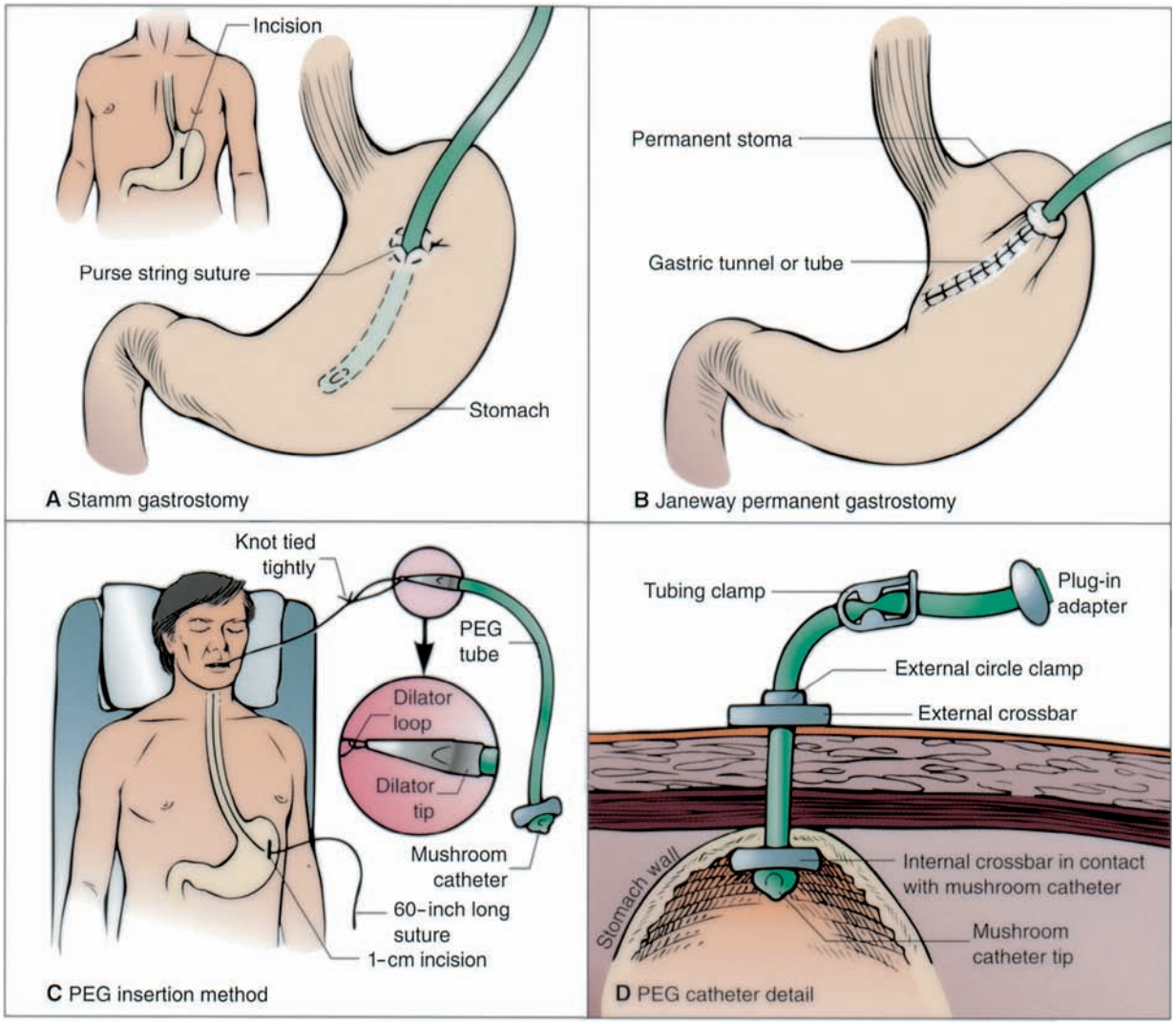
ENTERAL NUTRITION

NUTRITIONAL ACUITY RANKING: LEVEL 4 IF AT HOME



Adapted from: Smeltzer SC, Bare BG, *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 9th ed. Philadelphia, Lippincott Williams & Wilkins, 2000.

DEFINITIONS AND BACKGROUND

EN makes it possible to provide important substrates for those who cannot or will not meet daily requirements via oral intake but who have an intact digestive system (Chen and Peterson, 2009). EN involves nutrition therapy via nasogastric tube, orogastric tube, gastrostomy, nasoduodenal or nasoenteric feeding, or jejunostomy. PN is reserved for conditions in which EN is contraindicated, unsuccessful, or inadequate. EN is economical, yields effective nutrient utilization, and maintains gut mucosal integrity. Trophic stimulation of the gut occurs with EN but not with PN. Trophic feedings of 10–30 mL/hr may prevent some gut atrophy but will not yield as much benefit as using a more sufficient quantity for the individual. Prevention of intestinal permeability, faster return of cognitive function, and better outcomes have been noted with EN versus PN. EN has also been associated with a reduction in infection rates, hospital-associated infectious complications, ventilator dependency, and intensive care use. Immunoglobulin A (IgA) prevents absorption of enteric antigens; IgA levels increase with EN but not with PN. Immunoenhancement occurs when arginine, glutamine, long-chain polyunsaturated fatty acids (PUFAs), omega-3 fatty acids, vitamins A, C, E, and ribonucleic acids have pharmacological effects. Arginine is an intermediary in the urea cycle, making urea and ornithine via arginase. It is also useful for ammonia detoxification and the synthesis of glutamate and nitric oxide. With infection, the body catabolizes more protein to provide substrate (citrulline) for do novo arginine synthesis. Thus, in stress, trauma, or catabolic states, arginine becomes conditionally essential. Most people consume 5–6 g daily of arginine from dairy, beef, poultry, seafood, soybeans, nuts, seeds, or wheat germ. Enteral formulas vary in content, with an average of 1–2 g/L; enriched formulas may be 12 g/L or higher. Improved wound healing and net nitrogen retention have

been noted with arginine supplementation. Glutamine is a primary substrate for the GI tract and lymphocytes; it helps to maintain acid–base balance. Because glutamine is a heat shock protein regulator, pneumonia, sepsis, and bacteremia are less frequent among patients who receive supplemented feedings. Glutamine is useful in attenuating the systemic inflammatory response syndrome (SIRS) response in highly stressed, critically ill patients (such as major elective surgery, trauma, burns, head and neck cancer, or ventilator patients). While its supplementation can decrease infectious complications, routine use is not warranted (American Dietetic Association, 2010).

There are some disadvantages of EN. Judicious use of EN should be considered in low birth weight or premature infants because feeding intolerance is common. Although EN is often started to prevent aspiration pneumonia from oral intake in stroke or demented patients, it may also increase the risk if positioning is poor or if tube placement is wrong. Tube dislodgement or occlusion, infection at the gastrostomy or jejunostomy site, hyperglycemia, azotemia, and fluid and electrolyte imbalances may also occur if not managed carefully. In some patients, maldigestion, malabsorption, abdominal distention, high residuals, nausea, vomiting, constipation, and other signs of GI intolerance are present. Home EN (HEN) for SBS, bowel obstruction, chronic pancreatitis, enterocutaneous fistula, cancer, or severe dysphagia requires careful and regular monitoring from the team.

The traditional assessments using albumin, transthyretin, and anthropometry are not useful in critical care settings. Weight loss prior to admission, disease severity, comorbidities or complications, and GI tract function are more useful. When possible, a nutritionally focused physical examination is needed, including state of hydration, abdominal examination for possible GI intolerance, state of consciousness, general overall appearance, body composition, presence of respiratory distress, nausea, vomiting, abdominal distention, diarrhea, abdominal cramping, constipation, weight changes, hydration status, and abnormal laboratory values. As appropriate, malnourished patients at risk can be fed early without observed negative clinical consequences.

Early EN is beneficial if the patient is hemodynamically stable, depending on where the tube must be placed. Improved clinical outcomes, lower rates of infection, and decreased hospital stays have been observed when EN was initiated within 24 hours and advanced to goal within 48–72 hours. A review of 13 trials with 1173 GI surgical patients found that early commencement of feeding (within 24 hours) reduces risk of postsurgical complications, length of hospital stay, and mortality (Lewis et al, 2009).

Either gastric or small bowel feedings work well; the latter used when aspiration or intolerance is noted. After mechanical ventilator extubation, oral intake is often low and nutrition therapy may be needed (Peterson et al, 2010). When patients are hemodynamically unstable, EN is usually held or combined with PN as needed. Use of an ICU nutrition protocol increases the likelihood of patients receiving the proper nutrition therapy.

Pediatric nutrition therapy protocols are not readily available for critically ill children (Joffe et al, 2009). After intestinal failure with SBS, children are often dependent on PN. However, this dependency can lead to intestinal failure-associated liver disease (IFALD) unless transition to trophic feeds and, eventually, full EN is attempted (Le et al, 2010). Use of omega-3 fatty acids is under study for this population, and more research is needed for pediatric critical care.

Because tube-fed patients in long-term acute-care facilities may be routinely over- or underfed, monitoring how much feeding is actually given to a patient should be done to determine if needs are being met. Both underfeeding and overfeeding affect ventilatory status. Therefore, it is ideal to measure a patient's energy requirements using indirect calorimetry (IC) at least once, especially for the critically ill,

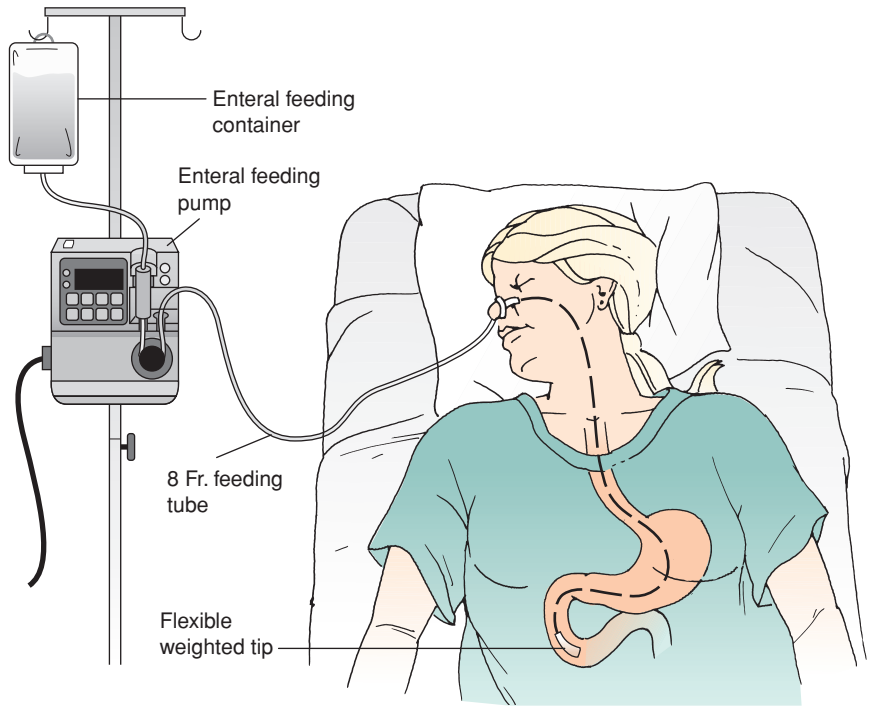


TABLE 17-3 Consequence Statement: Not Feeding a Resident/Patient When Oral Intake Is Inadequate

"The clinical manifestations of protein–energy undernutrition are related to length of time and extent of nutritional deprivation and the prior health status of the person. On the basis of both animal and human studies, there are serious detrimental effects on the function of every organ system, including the **heart, respiratory muscles, and the brain.** 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, D. The role of nutrition in increased morbidity and mortality. Clin Geriatr Med. 11:663, 1995.*

Other documented consequences of not tube feeding a resident/patient who *will not* or *cannot* eat enough orally:

- **Dehydration** with increased risk of urinary tract infections, fever, swollen tongue, sunken eyeballs, decreased urine output, constipation, nausea and vomiting, decreased blood pressure, mental confusion, electrolyte disturbances
- **Decreased awareness** of environment from decreased glucose availability for the brain
- **Development of 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 and rapid, involuntary weight loss** that are highly predictive of illness and imminent death. (Note: seniors are especially unable to regain weight after a stress situation)

I, the undersigned, understand and acknowledge that these consequences have been reviewed with me. I am deciding not to tube feed _____ (resident/patient) and accept the outcome of this decision.

Signed:_____Date:_____ Witness:_____ Date_____

Power of Attorney, Guardian or Other Representative Witness:_____ Date_____

obese, or in whom estimation of requirements are difficult (American Dietetic Association, 2010).

For terminally ill individuals, consider the patient's advance directives. When a patient's wishes are not known, TF is viewed as humane by many internists. The ramifications of not feeding a patient should be discussed with all parties (see example form in Table 17-3).

The definition of what constitutes gastric residual volumes as indicators of EN tolerance will vary. Volume of gastric residuals, which prompts holding or cessation of feeding, varies from one facility to the next. One high volume should probably not prompt the clinician to stop TF but to monitor carefully and recheck frequently. Optimal patient positioning, use of prokinetic agents to improve gastric emptying, continuous infusion, and abdominal examinations to evaluate for distention are helpful.

Medical nutrition therapy (MNT) for patients who are tube fed saves thousands of dollars per case each year. Nutrition support teams (NSTs) are associated with improved quality and cost-effective care. Teams are often able to decrease complications, decrease lengths of stay, and decrease readmission rates. While it is not beneficial to use EN in the first week for dysphagic stroke patients who are not malnourished, there is reasonable evidence for using it in low birth weight infants (trophic feeding), malnourished geriatric patients, perioperative patients, patients with chronic liver disease, and critically ill patients (Koretz et al, 2007). Fortunately, EN results in clinically relevant, statistically significant risk reduction for infectious complications, pancreatic infections, and mortality in patients with severe acute pancreatitis (Petrov et al, 2008).

The "CNSC" credential indicates basic competency for managing EN therapy. The National Board of Nutrition Support Certification (NBNSC) is responsible for awarding Certified Nutrition Support Clinician (CNSC), to those who pass the exam. The CNSC Registered Dietitian has clinical expertise in nutrition therapy obtained through education, training, and experience. The CNSC assures optimal therapy for the nutrition diagnosis of "inadequate oral food and beverage intake" from a variety of etiologies. Interventions usually include alternative feeding methods for delivery of nutritional requirements. The CNSC practices in a variety of settings (acute facilities, subacute facilities, ambulatory/outpatient clinics, rehabilitation centers, long-term care facilities, home care, and hospice) and cares for patients of all age groups and types of illness.

Registered dietitians with special training and demonstrated competency in nutrition support therapy are able to evaluate, write, or recommend TF orders. A multidisciplinary approach works best for consideration of all medical, nutritional, and ethical issues. ASPEN published EN Practice Recommendations in 2009 that provide excellent, detailed recommendations (ASPEN, 2009). Standardized forms, protocols, clinical practice guidelines or pathways help provide predictable nutrition therapy outcomes for patients (Table 17-4). The American Dietetic Association (ADA) recommends four or more MNT services for adults who are receiving EN. The ADA Coding and Coverage Committee (CCC) is working diligently to expand coverage and reimbursement.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The use of EN is not specific to genetic conditions.

Clinical/History	Lab Work	As Needed:
Height	Initially and once weekly thereafter:	C-reactive protein (CRP)
Weight	Glucose (Gluc)	Partial pressure of carbon dioxide (pCO_2)
Body mass index (BMI)	Na^+ and K^+	Partial pressure of oxygen (pO_2)
Intake and output (I & O)	Phosphorus	Cholesterol (Chol)
Blood pressure (BP)	Magnesium	Triglycerides (Trig)
Diarrhea	Blood urea nitrogen (BUN)	
Temperature	Creatinine (Creat)	
Nausea, vomiting	Albumin (Alb)	
Chest x-ray	Ca^{++}	
Residuals	Hemoglobin and hematocrit (H & H)	
	Transferrin	
	Chloride	
	Respiratory quotient (RQ) (>1.0, energy delivery is excessive)	
	Prothrombin time (PT)	
	International normalized ratio (INR)	

INTERVENTION

OBJECTIVES

- Prevent or reverse malnutrition, cachexia, impaired immunity, and loss of lean body mass related to inadequate oral food and beverage intake. If the gut works, use

TABLE 17-4 Clinical Practice Guidelines for Nutrition Support

Issue	Considerations or Evidence
EN vs. PN	When considering nutrition support in critically ill patients, strongly recommend that EN be used in preference to PN
Early vs. late EN	Recommend that standard, polymeric enteral formula be initiated within 24–48 hours after admission to an ICU
Formula composition of EN	Standard formula is acceptable for most patients
Positioning of patient for EN	When clinically feasible, patients should be placed in a 30–45° head of bed elevation during gastric feedings to decrease reflux of gastric contents into pharynx and esophagus and possibly to decrease pneumonia
Dose and actual delivery of EN	Actual delivery of threshold intake of approximately 14–18 kcal/kg/d or 60–70% of enteral feeding goal in the first week of ICU admission is associated with improved outcomes (e.g., length of hospital stay, time on ventilator, infectious complications), particularly when initiated within 48 hours of injury or admission
EN in combination with supplemental PN	When initiating EN, strongly recommend that PN not be used in combination with EN
Optimize delivery of EN	Start at the target rate, promotility agents (metoclopramide) or postpyloric feeding should be considered to reduce the gastric residual volume
Minimize risks in EN	Manage rate of advancement, check residuals, use bedside algorithms, consider motility agents, use small bowel vs. gastric feedings when needed, elevate head of the bed, use closed delivery systems, consider use of probiotics, and evaluate bolus administration
Dose of PN and composition of PN	Calculate needs for protein, carbohydrates, IV lipids, additives, vitamins, trace elements, and immune-enhancing substances
Insulin therapy	With elevated glucose levels (as in diabetes, infection, and sepsis), insulin therapy will help to achieve better control
Outcomes	Length of stay (ICU and hospital), quality of life, and specific complications should be considered: EN is associated with decreased infectious complications in comparison to PN
Tube placement	Placement of the tip of the feeding tube in the postpyloric position is associated with decreased gastric residual volume, a factor associated with reduced reflux of formula. Postpyloric feeding tube placement may not be necessary or feasible for all patients but may be useful in patients with large gastric residual volumes
Protocol	An enteral feeding protocol should address the following • When to use enteral vs. parenteral feeding • When to initiate enteral feeding • Positioning of patient • Energy goal per kilogram BW per day • Policy not to use blue dye • Indications for holding feedings • Tube placement • Prokinetic/promotility agents

Adapted from: American Dietetic Association. Critical Illness Nutrition Evidence Analysis Project. Evidence Analysis Library, http://www.adaevidencelibrary.com/topic.cfm?cat=1032, accessed March 11, 2010.

SAMPLE NUTRITION CARE PROCESS STEPS

Swallowing Difficulty

Assessment: 71-year-old male recently discharged from hospital for GI bleeding. Status post-cerebral infarction (CVA) resulting in left hemiparesis, moderate dementia, HTN. Admitted to long-term care unit with PEG tube placed secondary to new-onset dysphagia.

Medical HX: GI hemorrhage, dementia with behavioral disturbances, hx of aspiration, dysphagia.

Current Medications: Aricept, Lasix.

Height: 67 in. Current Weight: 138.1 lb (62 kg). IBW Range: 133–162 lb. IBW: 100%. BMI: 21.6.

Most laboratories are within normal limits (WNL) except for slightly elevated BUN (25) and low albumin (2.8).

Estimated Needs: Energy (25–30 kcal/kg): 1550–1860 kcal; protein (1.0–1.25 g/kg): 62–77 g; fluids (30 mL/kg): 1860 mL.

TF and Flush Order: Enteral formula with 1.5 kcal/mL; 1 can or 240 mL, 5 times a day (6 AM, 10 AM, 2 PM, 6 PM, 10 PM). Bolus feeding via syringe. Flush tube with 50-mL water before and after medications. Flush tube with 100-mL water every shift.

Nutrition Diagnoses: Swallowing difficulty (NC-1.1) related to recent stroke as evidenced by abnormal video swallow study done by speech language pathologist upon admission to long-term care facility.

Increased need for protein (NI-5.1) related to inflammation and depleted visceral protein status as evidenced by a low albumin level of 2.8.

Nutrition Interventions: Administer enteral feeding as ordered (ND-2). Implement nutrition-related medication management (ND-6) of protein additive three times daily (TID).

Goal 1: no significant weight changes; no signs of dehydration.

Goal 2: no episodes of nausea, vomiting, diarrhea, constipation, or aspiration.

Goal 3: albumin level of 3.0 and other laboratories WNL.

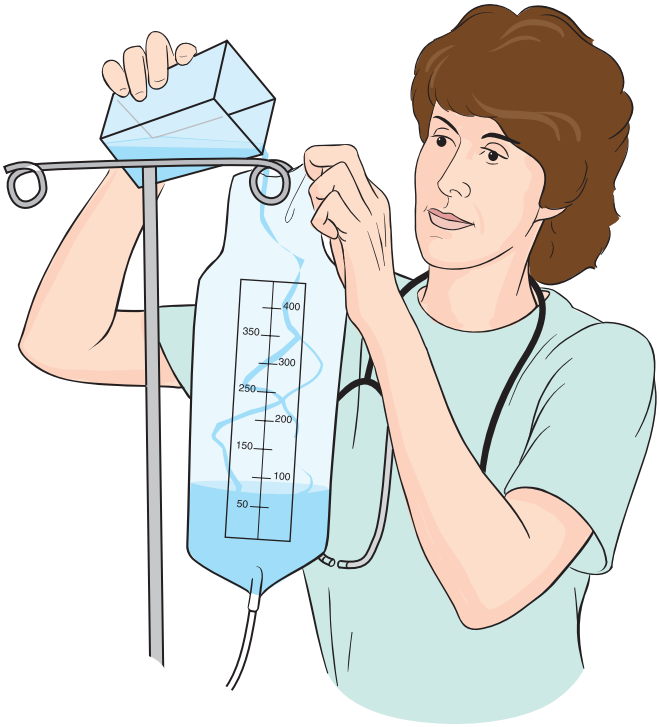
Monitoring and Evaluation: Tolerating TF well; currently meeting daily estimated nutrition and hydration needs. Monitor monthly weights. Monitor tolerance to TF order. Monitor albumin and significant laboratories as ordered.

it and check tube placement, laboratories, electrolytes, and fluid boluses to be sure the patient is tolerating the product well.

- Write or edit **nutrition prescription** to provide adequate protein, carbohydrate, fat, vitamins, minerals, and water. Nitrogen needs will increase for burn, pressure ulcer, or trauma patients; the percentage of total kilocalories from protein should be increased in these cases. Aim for 100% of dietary reference intakes (DRIs) for vitamins and minerals; provide explanation if order is excessive or insufficient. Additional liquid vitamins and minerals may be needed if formula volume does not meet micronutrient needs. Alter TF prescriptions as appropriate according to results of the nutritionally focused physical examinations.
- Choose **timing** wisely. If the critically ill patient is adequately fluid resuscitated, EN should be started within 24–48 hours following injury or admission to the ICU (American Dietetic Association, 2010).
- Recommend or select **feeding tube** and **site/location** on the basis of clinical condition, GI anatomy, and anticipated length of treatment. For patients at high risk for aspiration, who will be fed longer than 1 month, try a PEG tube with continuous feeding. If aspiration persists, try a transjejunal tube with lower placement in the GI tract. Gastrostomy buttons have minimal complications, acceptable longevity, and good tolerance in children and adults.
- **Formula selection** includes type of feeding needed by individual, the disorder, viscosity, and kcal/mL. Elemental formulas should be limited to specific conditions in which digestion or absorption is impaired or in which polymeric diets have failed. See Table 17-5 for types of formula selections.
- Monitor patient **positioning.** The head of the bed should be elevated 30–45° during feeding. To decrease the incidence of aspiration pneumonia and reflux of gastric contents into the esophagus and pharynx, patients should be placed in a 45-degree head of bed elevation, if not contraindicated (American Dietetic Association, 2010). For the unconscious patient, turn to the side to help with gastric emptying. For intermittent or bolus feedings, keep patient on his/her right side or keep head of bed elevated for 30 minutes after feeding to prevent aspiration.
- Ensure **adequate free water** is provided (usually 30 mL/kg in young adults with normal renal function). Determine percentage of free water in the formula (usually 70–85%), and subtract this amount from estimated needs; flushes should provide the difference.
- Monitor for **signs of intolerance;** adjust formula type, volume, or concentration as needed:
  - With diabetic gastroparesis, tube feeding may not be well tolerated; insulin adjustments may be required.

TABLE 17-5 Sample Types of Formulas

Formula Type	Macronutrient Content
Blenderized	Whole protein, carbohydrates, and fats from regular foods and liquids blended together
Polymeric—1 kcal/mL	Intact proteins, complex carbohydrates, and mainly LCTs
Added fiber	Varies but usually polymeric
Energy Dense—1.5–2.0 kcal/mL	Varies but usually polymeric
Disease specific	Varies by condition (liver, renal, diabetes, HIV-AIDS)
Peptide based (semi-elemental; oligomeric)	Peptides of varying chain length, simple sugars, glucose polymers or starch and fat, combination of LCT/MCT
Elemental (monomeric)	Individual amino acids, glucose polymers, typically low in fat. Some with only 2–3% of fat as LCT
Immune enhancing	Varies but usually polymeric. Usually has arginine, glutamine, or RNA added
High nitrogen	May be polymeric or monomeric
Critical care	May be polymeric or monomeric



- Monitor fluid carefully in organ failure where fluid restriction is needed; a more concentrated product can achieve energy and protein goals.
- Evaluate medications or infection as potential causes of diarrhea or feeding intolerance.
- Fiber-added formulas may be appropriate with diarrhea or constipation.
- Optimal oral health, tight glycemic control, minimal use of narcotics, and continuous feedings are recommended for patients at high risk for aspiration.

- Evaluating **gastric residual volume (GRV)** is an optional part of a monitoring plan to assess tolerance; EN should be held when a GRV greater than or equal to 250 mL is documented on two or more consecutive occasions (American Dietetic Association, 2010). Replace aspirate for the electrolytes and gastric juice. Do NOT use blue dye added to the formula to test for aspiration (American Dietetic Association, 2010).
- **Weights** are also important. Patient should be weighed on same scale at regular intervals, wearing similar clothing. Check twice if weights differ significantly from previous weights.

FOOD AND NUTRITION

- Calculate energy requirements and protein, fluid, and nutrient needs according to age, sex, and medical status. See Table 17-6 for considerations.
- For weight loss, 20 kcal/kg is recommended. Use 25 kcal/kg to maintain weight, 30 kcal/kg with mild stress factors, and 35–40 kcal/kg in moderately stressed patients.
- Protein is generally 0.8–1.0 g/kg to maintain status, 1.25 g/kg for mild stress, 1.5 g/kg for moderate stress, and 1.75–2.0 g/kg for severe stress, trauma, or burns. The critically ill may need more calories and protein than other patients.
- Estimate fluid needs at 30–35 mL free H_2O/kg body weight (BW) or 1 mL/kcal.
- Check patient's tolerance and side effects; alter formula content as appropriate. Sample new products to determine costs and convenience for home or institutional use. Numerous EN formulas are currently available, with a large portion of them targeting specific disease conditions (Chen and Peterson, 2009).
- Flush tubing with water (25–100 mL) every 3–6 hours for tube patency and before/after medications are given.
- With a gastrostomy tube, bolus or intermittent feeding is possible. For postpyloric or transpyloric placement, cyclic feedings may be better tolerated than continuous feedings.
- If the patient is in transition back to oral diet or works during the day, night feeding may be used. It may be more energy efficient than continuous feeding over 24 hours.
- Interdisciplinary teamwork is a crucial factor. The following guidelines are useful for managing tube feeding and transitioning from tube to oral feeding. (See figure on page 909)

Common Drugs Used and Potential Side Effects

Drug–nutrient interactions are complex and can cause malabsorption of either the drug or the nutrient in tube feedings. Depending on the physical properties of a drug, it may be absorbed in a limited area of the GI tract or along much of the entire length. Monitor carefully for toxicity. Flush with 5- to 10-mL water after each medication is administered to prevent clogging.

- Antibiotics, H_2-receptor antagonists, and sorbitol elixirs alter gut flora and can cause diarrhea because of their high osmolality.
- Antidiarrheal drugs can be used to slow GI motility. Their use should not preclude a carefully planned fiber intake. Dry mouth is one common side effect.
- Metoclopramide (Reglan) has been used to prevent reflux and aspiration in patients who are tube fed. Administration 10 minutes before tube insertion seems to increase success rate of tube passage. Gastric motility and relaxation of the pyloric sphincter are improved with this drug. However, chronic use may dislodge gastrostomy tubes; monitor closely.
- Phenytoin (Dilantin) administration should be separated from TF by 1–2 hours to prevent decreased medication absorption. TF rate may need to be recalculated, accounting for time TF is held before and after phenytoin administration, and adjusted accordingly.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be mixed with enteral feedings without discussing with the physician and the pharmacist.
- Gastric feeding supplementation with ginger extract might reduce delayed gastric emptying, but more research is needed.

TABLE 17-6 Key Enteral Issues

Issue	Comments
Feeding site selection	Consider any GI impairments or inability to absorb nutrients, vomiting, severe and persistent diarrhea, respiratory disease or skull surgery/fracture, or tendency to remove tubes by choice or inadvertently
Nasogastric	Often used for temporary needs; tube placed into stomach from the nose
Nasoenteric	For patients with impaired gastric emptying or in whom a gastric feeding is contraindicated
Nasojejunal	For patients at risk of pulmonary aspiration or with GI problems that preclude stomach placement such as mechanical problems or problems with gastric emptying or tolerance
Gastrostomy	Surgical incision or endoscopically placed PEG. Allows long-term feeding. A low-profile device (button) can be used for long-term feedings or for improved body image
Jejunostomy	Surgical incision into the jejunum to bypass inaccessible areas of the duodenum such as with SBS or obstructions from cancer, adhesions, stricture, or inflammatory disease. PEJ is a PEG with a transjejunal limb. A jejunostomy tube may cause some bowel necrosis; monitor carefully
Gastrojejunostomy	Good for small bowel feeding when stomach must be decompressed
Contraindications for tube feeding	Severe malabsorption; severe, intractable diarrhea
Formula selection	It is generally more cost effective to have an enteral formulary established, including multiple products, but one main brand of each category (e.g., standard/isotonic, isotonic with fiber, high-nitrogen isotonic, elemental, high-protein/high calorie for stress, critical care products, concentrated for patients with volume intolerance, malabsorption, specialty products for pulmonary or diabetes or immunocompromised, renal, or hepatic patients)
Elemental vs. intact formulas	No superiority has been documented for elemental. Peptides may be used for most patients
Substrates	Carbohydrate, protein, and fat (consider patient's ability to digest and absorb nutrients)
Tolerance factors	Osmolality, calorie, and nutrient densities. In general, more free water is needed with a more concentrated formula
Fluid needs	Generally, 1 mL/kcal is recommended, unless patient needs fluid restriction; 30 mL/kg is most common for adults. Elderly individuals may require slight alterations, depending on organ function. Children must receive adequate fluid, calculated by body weight
Organ failure	Heart failure, renal failure, or liver failure with ascites; 20 mL/kg can also be used initially, progressing to 25 mL/kg as tolerated
Risk for dehydration	Risk of abnormal losses due to GI drainage, diarrhea, and dehydration, and for those with other needs for extra water, 35–40 mL/kg may be used
Delivery methods	Patient tolerance is a key. Goal: meet needs without complications such as nausea, vomiting, diarrhea, or glucosuria
Bolus	Set amount given every 3–4 hours as a rapid syringe feeding; this closely resembles an oral diet for patients who are ambulatory or with long-term and well-established feedings
Continuous	Controlled delivery of feeding over 24 hours. Less nausea and diarrhea are likely. Once stable, most patients may transfer to intermittent
Cycled	Controlled delivery over 8–16 hours, allowing some rest periods for patient during 24 hours. Cyclic is well tolerated by ambulatory patients
Intermittent	Prescribed amount given every 3–4 hours by drip over 20–30 minutes
Complications	Evaluate for metabolic complications. Electrolyte shifts and elevated glucose may occur; an insulin regimen may be needed until hyperglycemia is resolved
Aspiration	Proper positioning greatly reduces risks of pulmonary aspiration
GI side effects	For GI concerns, check for residuals and hold feedings for amounts greater than 150 mL; stop for 4 hours and recheck. For diarrhea, check osmolality of feeding, rate, albumin level, and medications (e.g., sorbitol, magnesium)
Mechanical ventilation	Use enteral nutrition with H_2 antagonists to facilitate gastric emptying
PEG tube problems	Possible complications include pain at the PEG site, leakage of stomach contents around the tube site, dislodgment or malfunction of the tube, aspiration, bleeding, and perforation
Tube clogging	Small-bore tubes are associated with clogging. To prevent or correct mechanical clogging in small-bore tubes, flush regularly with water before and after all medications

See also: ASPEN Guidelines for enteral nutrition at Web site http://www.nutritioncare.org/WorkArea/showcontent.aspx?id=3128.

INTERDISCIPLINARY NUTRITION CARE PLAN
Transitioning from Tube Feeding to Oral Diet

Client Name: _____ **#:** _____ **Initiated by:** _____ **Date:** _____

SCREENING
Nutrition Screen diagnosis: Transition From Enteral Tube Feeding to Oral Intake
Signed: _____ Date: _____

ASSESS *(Check any/all)*
❑ **Oral feeding readiness**
❑ **Dehydration**
❑ **Poor strength**
Weight/BMI
❑ Weight change >3 lb/wk, >5%/mo, or >10%/6mo
❑ BMI <20
❑ BMI <27
Poor Oral Intake Symptoms
❑ Lack of appetite
❑ Complex diet order
❑ Vomiting
❑ Decreased ability to chew/swallow
❑ Nausea
❑ Depression/anxiety

Signed: _____ Date: _____

None

1 or more

HIGH-RISK INTERVENTIONS *(Check any/all)*
❑ **4 Ways to Improve Nutrition** provided and explained
❑ **Food Record** provided and explained
❑ **Fluid intake stressed**
Obtain Dr. orders as needed:
❑ RD referral for home visit/nutrient analysis
❑ Speech Language Pathologist (SLP) referral for oral feeding readiness/problems
❑ Tube feeding
❑ Monitor weight q:_____
❑ Monitor I & O q:_____
❑ BID/TID supplements
❑ **Other:**_____
(See notes for documention.)

Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
❑ Further weight loss
❑ Continued dehydration
❑ Continued loss of strength
❑ Cannot tolerate oral feeding
❑ Exhibiting Poor Oral Intake Symptoms
❑ **Other:**_____
(See notes for documention.)
Signed: _____ Date: _____

None

1 or more

OUTCOMES NOT ACHIEVED
Reassess/evaluate need for further enteral feeding. Document on Nutrition Variance Tracking form.

GOALS (Check any/all):

❑ Maintain or improve nutritional status in ____ (goal time).

❑ Increase weight by ____ lb in ____ (goal time).

❑ Successful transition from enteral tube feeding in ____ (goal time).

❑ Maintain or improve hydration status in ____ (goal time).

❑ Increase oral intake to meet nutritional needs in ____ (goal time).

MODERATE-RISK INTERVENTIONS
(Check any/all)
❑ **H4 Ways to Improve Nutrition** provided and explained
❑ **Food Record** provided and explained
❑ **Fluid intake discussed and encouraged**
Obtain Dr. orders as needed:
❑ RD chart consult
❑ SLP chart consult
❑ Monitor Weight q:_____
❑ BID/TID supplements
❑ **Other:**_____
(See notes for documention.)

Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
❑ Further weight loss
❑ Dehydration
❑ Poor strength
❑ Cannot tolerate oral feeding
❑ Exhibiting Poor Oral Intake Symptoms
❑ **Other:**_____
(See notes for documention.)

Signed: _____ Date: _____

None

OUTCOMES ACHIEVED
❑ Oral diet tolerated
❑ Weight gained
❑ Hydration status maintained or improved
❑ Absence of Poor Oral Intake Symptoms
❑ Strength maintained or improved
❑ **Other:**_____
(See notes for documention.)
❑ Repeat Nutrition Risk Screen in ____ days
Signed: _____ Date: _____

OUTCOMES ACHIEVED
❑ Oral diet tolerated
❑ Weight gained
❑ Hydration status maintained or improved
❑ Absence of Poor Oral Intake Symptoms
❑ Strength maintained or improved
❑ **Other:**_____
(See notes for documention.)
❑ Repeat Nutrition Risk Screen in ____ days
Signed: _____ Date: _____

Adapted with permission from www.RD411.com, Inc.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Patient/caretaker should be taught to review signs and symptoms of intolerance, how to manage simple problems, when to call for registered dietitian (RD) guidance versus when to call the physician.
- At least one follow-up phone call or home visit should be made to patients on HEN.
- Patient should be allowed and encouraged to maintain social contacts at mealtime.
- When transitionally weaning young children to an oral diet, oral–motor, sensory, and developmental feeding problems may occur. Check for feeding readiness via oral stimulation and develop a feeding plan.

Patient Education—Formula Safety

- Safe preparation of TF is essential. Homemade feedings are not recommended in most cases.
- Table 17-7 describes a Hazard Analysis Critical Control Points (HACCP) procedure for maintaining a safe enteral feeding system in the hospital setting. Hang time for open-system containers (feeding bags) is 4 hours; hang time for closed-system containers is 24–48 hours.
- Always wash hands carefully and sanitize counter before handling the equipment or preparing the formula. Use a clean tube each time; wash in dishwashing liquid and rinse well between uses.
- Wash feeding bags with water. Do not use soap as it will stick to the inside of the bag and get into the formula and may lead to diarrhea and other unpleasant consequences.
- Temperature standards for refrigeration and storage of enteral feeding product must be met.
- Open cans of formula could be kept in the refrigerator and discarded if not used within 24 hours. Take out and allow to warm up to room temperature 15–20 minutes before a feeding. Feedings should be given at room temperature to minimize risk of cramping or diarrhea. Unopened commercially prepared formulas do not require refrigerated storage.
- Always flush the feeding tube with water after a feeding to prevent dehydration and prevent the food from getting clogged. The RD should always monitor that the feeding

TABLE 17-7 Critical Control Point Checklist for Tube Feedings

Purchasing, Receiving, and Storage

- Enteral feeding product(s) received according to specification
- Temperature standards for refrigeration and dry storage of enteral feeding product(s) met
- Product usage according to FIFO (first in, first out); those exceeding expiration date returned/discarded
- Liquid protein module and frozen shakes (labeled with date) placed in refrigerator for thawing
- Open cartons discarded after 48 hours
- Unopened and unused thawed liquid protein module discarded after 5 days
- Unopened and unused thawed shakes discarded after 12 days
- Medium-chain triglyceride oil, dry carbohydrate powder, and protein powder stored at room temperature; labeled with date opened. Opened cases and unused bottles/cans discarded after 1 month
- Inventory of reconstituted, mixed enteral formulas and portioned protein, fat, and carbohydrate modules reveals none are past expiration date
- Prepared enteral feedings kept separate from raw or processed food items and cleaning compounds

Preparation and Delivery

- Employees wash hands prior to preparing enteral feedings or modular components
- Cleaned and sanitized surface and equipment used to prepare enteral feedings or modular components
- Enteral formula prepared according to recipe
- Tap water used to reconstitute pediatric powdered formulas and Ceralyte; distilled or sterile water used in the preparation of enteral formulas upon specific order
- Reconstituted mixed enteral formulas and portioned protein, fat, and carbohydrate modules sealed and labeled (formula, rate of administration, patient name and room number, date prepared)
- Temperature standards for refrigerated storage of reconstituted, mixed enteral formulas and portioned protein, fat, and carbohydrate modules met
- Nursing staff washes hands prior to handling feedings and administration systems
- Nursing staff avoids touching any part of the container or administration system that will come in contact with the feeding
- Nursing staff assembles feeding system on a disinfected surface and inspects seals/reservoirs for damage
- Medications are not added to feeding unless necessary. If added, tube is flushed with tap water (or as specified) after administration
- Date/time each component of feeding system; also feeding bag is labeled with patient name and formula
- Hang time of feeding limited to 4 hours
- Feeding bags completely emptied of product prior to pouring newly opened product into the bag
- Disconnected sets are capped
- Container is positioned to prevent reflux of feeding up set
- Feeding tube is irrigated with tap water (or as specified)
- Administration sets changed every 24 hours

Adapted with permission from New York-Presbyterian Hospital/New York Weill Cornell Medical Center.

and flushes provide sufficient water. If the tube clogs, try 100 mL of cola or a small amount of meat tenderizer to rinse.

For More Information

- American Society for Gastrointestinal Endoscopy
  http://www.askasge.org/pages/brochures/peg.cfm
- Enteral Formula Selection
  http://www.healthsystem.virginia.edu/internet/digestive-health/nutritionarticles/MaloneArticle.pdf
- Home Enteral Nutrition
  http://www.mayoclinic.org/gi-jax/enteral.html
- Home Enteral Nutrition Self-Help Guide
  http://www.copingwell.com/copingwell/HENCopingManual.pdf
- University of Washington TF Guidelines
  http://healthlinks.washington.edu/nutrition/section5.html

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Chen Y, Peterson S. Enteral nutrition formulas: which formula is right for your adult patient? *Nutr Clinical Practice.* 24:344, 2009.

Joffe A, et al. Nutritional support for critically ill children. *Cochrane Database Syst Rev.* 2:CD005144, 2009.

Koretz RL, et al. Does enteral nutrition affect clinical outcome? A systematic review of the randomized trials. *Am J Gastroenterol.* 102:412, 2007.

Le HD, et al. Innovative parenteral and enteral nutrition therapy for intestinal failure. *Semin Pediatr Surg.* 19:27, 2010.

Lewis SJ, et al. Early enteral nutrition within 24 h of intestinal surgery versus later commencement of feeding: a systematic review and meta-analysis. *J Gastro Surg.* 13:569, 2009.

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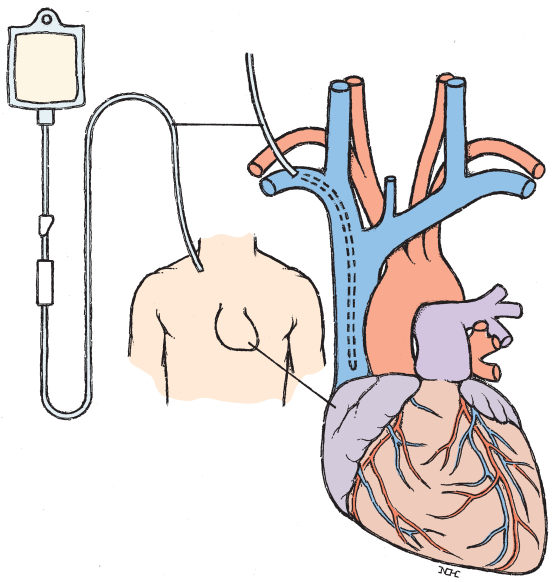
Petrov MS, et al. Enteral nutrition and the risk of mortality and infectious complications in patients with severe acute pancreatitis: a meta-analysis of randomized trials. *Arch Surg.* 143:1111, 2008.

PARENTERAL NUTRITION

NUTRITIONAL ACUITY RANKING: LEVEL 4 (HOME)



Adapted from: Neil O. Hardy, Westpoint, CT.

DEFINITIONS AND BACKGROUND

PN refers to IV nutrient admixture administered into the blood with a catheter placed in a vein. PN contains protein, carbohydrate, fat, vitamins, minerals, or other nutrients and is referred to as CPN if it meets the needs of the patient.

CPN can meet estimated nutritional needs, promote nitrogen balance, and improve anabolism. When there is a risk of malnutrition and EN is not tolerated or where there is gut failure, CPN is safe and encouraged. Start CPN only if EN attempts have failed; delay for at least a week unless malnourished before admission to a critical care unit. Short-term CPN is not recommended, especially for 1–2 days. Some medications can be infused into a parenteral solution, such as heparin, insulin, ranitidine; Y-site coinfusion saves time for the nursing staff.

Disadvantages of PN include pneumothorax, infection from the central line, metabolic complications, and the potential for overfeeding. Overfeeding may lead to sepsis. Finally, PN is more expensive than EN and oral diets and does not provide support for healthy gut immunity.

CPN and peripheral PN (PPN) are options for IV feeding. Indications for PPN include temporary losses of GI function (e.g., acute ileus) and occasions when short-term use is indicated such as after minor GI surgery. CPN may be most useful in patients undergoing surgery for esophageal or stomach cancers, in preoperative GI patients who are severely malnourished, and in patients with prolonged GI tract failure. Other indications for adults are listed in Table 17-8.

PN is used judiciously in metastatic cancer or chronic obstructive pulmonary disorder (COPD) because of complications, costs, and decreased quality of life (Ferreira et al, 2005; Joque and Jatoi, 2005). The limited nutritional response of cancer patients to PN reflects the metabolic derangements of cachexia; in nonsurgical, well-nourished oncologic patients, routine PN is not recommended because it offers no advantage and is associated with increased morbidity (Bozzetti et al, 2009). It may sometimes be used in long-term home care to manage radiation enteropathy.

Initiation of PN and metabolic complications are best managed by an expert in nutrition therapy. The skills that distinguish them from other practitioners include competency in fluid and electrolyte monitoring, acid–base monitoring, metabolic monitoring, management of refeeding syndrome, and related areas. Specially trained and certified registered

TABLE 17-8 Candidates for Central Parenteral Nutrition (CPN) in Adults

1. Someone with a nonfunctional or inaccessible gastrointestinal tract
   a. Massive bowel resection or SBS
   b. Radiation enteritis
   c. Ischemic bowel or bowel obstruction
   d. Chronic, severe malabsorption
   e. Inflammatory bowel disease, gastrointestinal obstruction, inflammatory adhesions
   f. Severe diarrhea as in AIDS when enteral feeding is not successful
   g. Enterocutaneous fistula—high-output, proximal fistula
2. Someone who cannot be adequately nourished with an oral diet or enteral nutrition (EN)
   a. After bone marrow transplantation, specifically in cases of graft vs. host disease accompanied by inadequate oral intake
   b. Intractable vomiting such as hyperemesis gravidarum
   c. In pregnancy when oral intake is compromised and when EN is not tolerated, as with SBS
   d. After major surgery when enteral access cannot be established
3. Someone with inadequate oral and enteral intake anticipated to persist for at least 7–14 days
   a. Severely catabolic patients whose gut cannot be used within 3–5 days (such as closed head trauma, fractures, burns)
   b. Cases with a high risk of aspiration
   c. Severe acute necrotizing pancreatitis when EN fails after a trial of 5–7 days
   d. Home parenteral nutrition (HPN) may be suggested for malnourished cancer patients who have a reasonable prognosis and cannot have EN

dietitians may write PN orders if granted clinical privileges by their institution or facility. To determine energy and nutrient requirements accurately, IC and use of programmed calculation software are recommended to prevent administration of excessive glucose and energy. The main consideration when administering fat and carbohydrates in PN is to prevent overfeeding the patient. (Braga et al, 2009).

Perioperative nutritional therapy can minimize negative protein balance by avoiding starvation; maintaining muscle, immune, and cognitive function; and enhancing postoperative recovery. Giving 7–10 days of preoperative PN improves postoperative outcome in patients with severe undernutrition who cannot be adequately orally or enterally fed (Braga et al, 2009).

Adequate nutrition therapy criteria include reaching a nutritional goal within 72 hours after initiation. PN may be given by continuous or cyclic infusion, altered according to patient tolerance. In most cases, gradual transition from PN to EN or oral nutrition is required to prevent periods of inadequate nutrition. The energy deficit accumulated by underfed ICU patients during the first days of stay may play an important role in outcomes for long-staying patients; how to reach calorie requirements by PN without harming the patient is a subject of debate (Singer et al, 2010).

With proper training and monitoring, CPN may be performed safely at home. CPN will drip through a needle or catheter placed into the central vein for 10–12 hours, once a day or five times a week. However, dependence on PN significantly impacts quality of life. Travel, sleep, exercise, and leisure activities are altered by home PN; quality of life issues must be addressed for each individual (Winkler, 2010).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: PN is used for a variety of conditions; some of which have a genetic origin.

Clinical/History

- Height
- Weight (measure daily)
- BMI
- Resting energy expenditure (REE)
- BP
- I & O (monitor continuously)
- Edema
- Skin turgor
- Chest x-ray
- Physical signs of malnutrition

Lab Work

Daily:
- Glucose (Gluc)—every 6 hours until stable
- CBC, Na^+ and K^+, Blood urea nitrogen (BUN)—daily for in-patients

Initially and once weekly thereafter:
- Creatinine (Creat)
- Transthyretin
- Triglycerides (if receiving IVFEs)
- Urinary Gluc

Twice Weekly at First, Then As Needed:
- Albumin (Alb)
- Calcium (Ca^{++})
- Magnesium (Mg^{++})
- Phosphate
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)

As Needed:
- C-reactive protein (CRP)
- Partial pressure of carbon dioxide (pCO_2)
- Partial pressure of oxygen (pO_2)
- Cholesterol (Chol)
- Hemoglobin and hematocrit (H & H)
- Transferrin
- Chloride
- Respiratory quotient (RQ) (>1.0, energy delivery is excessive)
- Prothrombin time (PT)
- International normalized ratio (INR)
- Serum and Urine Osmolality
- N balance
- Serum selenium
- Amylase, lipase
- Bilirubin
- Serum ammonia
- Serum Fe
- Serum folacin, B_{12}
- White blood cell count, TLC

INTERVENTION

OBJECTIVES

- If early EN is not feasible in first 7 days of hospitalization for a malnourished patient, consider PN to replete lean body mass.
- Assess estimated needs using IC, where possible. Determine appropriate patient requirements for calories, protein, vitamins, minerals, and fluid.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Oral Food and Beverage Intake

Assessment: BMI: 23. Loss of 12# in past year from inflammatory bowel disease and recent intestinal surgery to remove a small segment of the colon. Status 1 week postoperation; not tolerating enteral feedings at this time. Central line placed for CPN feedings. Most laboratories are WNL.

Estimated Needs: Energy (30–35 kcal/kg): 1700–1920 kcal; Protein (1.25 g/kg): 75 g. Fluids (30 mL/kg): 1700 mL. PN Order: Start CPN as directed by RD consultation.

Nutrition Diagnoses: NI-2.1 Inadequate oral food and beverage intake related to inability to eat orally because of IBD, GI distress, intestinal surgery as evidenced by weight loss of 12# in past year and intolerance for enteral feeds.

Nutrition Interventions: Write and administer CPN infusion to meet energy, protein, and fluid requirements without overfeeding.

Goal 1: very gradual increase in weight; no signs of dehydration or refeeding syndrome

Goal 2: no sepsis, fever

Goal 3: laboratories WNL

Monitoring and Evaluation: Tolerating CPN well; currently meeting daily estimated nutrition and hydration needs. Monitor weekly weights, tolerance to CPN order, significant laboratories as ordered.

- Fat and carbohydrate (CHO) make the balance of nonprotein calories after protein needs are estimated. When hypertonic dextrose is provided in substantial amounts without protein, mortality is high; CPN should provide balanced macronutrients and micronutrients (Jensen et al, 2009).
- Formulate solutions according to individual needs. For renal patients, review fluid and electrolytes and monitor essential amino acids (EAAs) in the solution. For hepatic patients, evaluate needs for fluid, electrolytes, and specialty amino acid solutions. For pulmonary conditions and diabetes, increased fat content and decreased dextrose may be needed.
- Maintain aseptic technique in all procedures for safe parenteral therapy. IVFEs support rapid growth of microorganisms, and PN solutions containing these fat emulsions should be changed every 12 hours. All PN solutions regardless of IVFE content should be used within 24 hours.
- Avoid substrate shifts. Because glucose abnormalities occur in >90% of patients, monitor plasma glucose often and adjust the insulin dose as needed.
- Avoid suddenly stopping constant concentrated dextrose infusions, which can cause hypoglycemia.
- Prevent refeeding syndrome. The hallmark sign is hypophosphatemia, along with neurologic, pulmonary, cardiac, neuromuscular, and hematologic complications. Dextrose infusion followed by increased insulin release causes shifts in phosphorus, potassium, and magnesium, often within 2–4 days of starting to feed. This sudden shift from fat to carbohydrate metabolism increases the basal metabolic rate and may lead to confusion, coma, convulsions, or death.
- Prevent or correct other complications associated with PN: weight gain over 2 lb or 1 kg daily, indicating syndrome of inappropriate antidiuretic hormone; mouth sores; skin changes; poor night vision; fluid overload; cardiac arrhythmias; metabolic bone disease. Catheter-related sepsis occurs in about 50% of patient cases. Manage liver complications that are common in young children.
- Select patients carefully. The incidence of inappropriate PN prescription is low when multidisciplinary NSTs are closely involved and when written guidelines are used (Kohli-Seth et al, 2009).
- Maintain fluid requirements. Avoid fluid overload that is more common than dehydration.
- Prevent essential fatty acid deficiency (EFAD); need 2–4% linoleic acid (LA) (as from soybean, corn, sunflower, or safflower). Avoid excesses.
- For home PN, support patient goals to continue usual activity, employment, and a pleasant daily life.
- Transition back to EN or oral intake, when and if feasible.

FOOD AND NUTRITION

- Calculate needs for PN related to requirements for energy, protein, fluid, vitamins, and minerals. The commonly used formula of 25 kcal/kg ideal body weight furnishes an approximate estimate of daily energy expenditure and requirements; in severe stress, requirements may approach 30 kcal/kg (Braga et al, 2009). CPN usually provides water (30–40 mL/kg/d), energy (30–60 kcal/kg/d, depending on estimated needs), amino acids (1–2.0 g/kg/d, depending on catabolism), essential fatty acids, vitamins, and minerals.
- Children need energy (up to 120 kcal/kg/d) and amino acids (up to 2.5 or 3.5 g/kg/d). They may also need a different fluid requirement. For neonates, use lower dextrose concentrations (17–18%).
- In older patients, lower glucose tolerance, electrolyte and micronutrient deficiencies, and lower fluid tolerance are common (Sobotka et al, 2009). In heart or renal failure, patients may need a fluid restriction.
- Dextrose monohydrate in CPN yields 3.4 kcal/g, not 4 kcal/g. For unstable blood glucose, use fat at 10–20% of energy intake; avoid overfeeding and consider use of insulin. Maximum rate of glucose infusion should not exceed 5–6 mg/kg/min, the rate of glucose oxidation or utilization.
- Amino acid infusions should not include enhanced arginine in sepsis, whereas it can be beneficial in postsurgical patients. Glutamine deficiency is common in critical illness, inflammatory bowel disease and stress; it is available for IV use but not in the United States. Glutamine infusion should not be used in hepatic encephalopathy. For liver failure or non-dialyzed renal insufficiency, reduce protein content and use a high percentage of EAAs.
- Fat should be given daily as an energy source. A 10% IVFE generally yields 1.1 kcal/mL. IVFE should not exceed 2.5 g/kg in adults or 1 g/kg in septic patients.

- Give 2–4% total kilocalories as LA to prevent EFAD. Avoid excessive use of LA, which is metabolized to the immunosuppressive and proarrhythmic arachidonic acid and to prostaglandins, thromboxanes, and leukotrienes. The omega-3 fatty acids (alpha-linolenic [ALA], docosapentanoic acid [DPA], DHA, EPA, gamma linolenic acid [GLA]) promote vasodilation, enhanced immunity, decreased inflammation and platelet aggregation, and antiarrhythmic effects.
- Medium-chain triglyceride (MCT) oil is made from coconut or palm oils but contains no EFA. In sepsis, transport of long-chain triglycerides (LCT) is impaired; MCT may be useful because no carnitine or lipases are needed.
- Structured lipids that contain a mixture of fish oil (Omegaven) and olive oil (Clinoleic) may provide better tolerance and fewer infections (Hardy and Puzovic, 2009). They are useful for decreasing the time needed for ventilator support in respiratory patients. Fish oil IV lipids are available in Europe and Asia but not necessarily in the United States or Canada. EPA and DHA help to stabilize NFκB in the cytoplasm, which decreases an excessive inflammatory response; they also increase vagal tone that improves GI motility.
- Sample nutrient requirements are listed in Table 17-9. Antioxidants are important; assure sufficient infusion of vitamins. IV vitamin A is only 25–33% available because it attaches to the plastic bags; vitamin E may also be less available. Vitamin E may be given as 1000 IU every 8 hours, sometimes orally. IV vitamin C is often given as 1000 mg every 8 hours; vitamin D is given as ergocalciferol. Vitamin K is part of the multivitamin infusate. Choline is important in metabolic pathways but is not generally given daily.
- Zinc, glutathine (from selenium), beta carotene, lipoic acid, melatonin, and *N*-acetylcysteine are other antioxidants that are important. However, avoid toxic IV mineral levels (e.g., >50 mg/d zinc; >1000 μg/d selenium for more than 10 days).
- Iodine deficiency has adverse effects on thyroid hormone production, and because most PN formulations do not contain iodine, monitoring is recommended (Zimmerman and Crill, 2009).
- Osmolality is important to monitor to prevent dehydration and other complications. For example, D5 W 5 has an osmolality of 252 mOsm/L, whereas D20 W 5 has an osmolality of 1008 mOsm/L.
- Special considerations will vary according to the patient's condition. Examples include:
  1. Short-chain fatty acids, soy, and fermentable fiber may be needed to reduce CPN-induced bowel atrophy.
  2. For respiratory failure, suggest a lipid emulsion that provides most of the nonprotein calories to lower the RQ.
- Follow practice guidelines, especially for children and home care patients (Kovacevich et al, 2005; Wessel et al, 2005). The increasing numbers of older (>65 years) Americans will increase the demand for home health services including support services (nursing, physical therapy, occupational therapy, durable medical equipment, respiratory therapy), infusion therapies, palliative care, and hospice; certification in gerontological care is encouraged and ASPEN Guidelines should be followed for optimal outcomes (Fuhrman, 2009).
- For management of complications of PN, see Table 17-10.

TABLE 17-9 Sample Basic Adult Daily Requirements for CPN

Nutrient	Amount
Water (/kg body wt/d)	30–40 mL
Energy[a] (/kg body wt/d)	
Medical patient	30 kcal
Postoperative patient	30–45 kcal
Hypercatabolic patient	45–60 kcal
Amino acids (/kg body wt/d)	
Medical patient	1.0 g
Postoperative patient	2.0 g
Hypercatabolic patient	3.0 g
Minerals	
Acetate/gluconate	90 mEq
Calcium	15 mEq
Chloride	130 mEq
Chromium	15 μg
Copper	1.5 mg
Iodine	120 μg
Magnesium	20 mEq
Manganese	2 mg
Phosphorus	300 mg
Potassium	100 mEq
Selenium	100 μg
Sodium	100 mEq
Zinc	5 mg
Vitamins	
Ascorbic acid	100 mg
Biotin	60 mcg
Cobalamin	5 μg
Folate (folic acid)	400 μg
Niacin	40 mg
Pantothenic acid	15 mg
Pyridoxine	4 mg
Riboflavin	3.6 mg
Thiamin	3 mg
Vitamin A	4000 IU
Vitamin D	400 IU
Vitamin E	15 mg
Vitamin K	200 μg

[a]Requirements for energy increase by 7% per 1°F or 12% per 1°C of fever.
Reference: Merck–Total Parenteral Nutrition, http://www.merck.com/mmpe/sec01/ch003/ch003c.html, accessed March 11, 2010.

- Do not overfeed; hyperglycemia, fatty liver, and excessive CO_2 production may occur. Monitor phosphate levels to assess levels of anabolism, risk for refeeding syndrome, and malnutrition.
- Provide weaning when patient is ready; use TF for interim nourishment if necessary. Progress to liquids and solids when patient is ready. Infusion of PN nutrients may suppress appetite, excessively prolonging PN use.

TABLE 17-10 Complications in Parenteral Nutrition (PN)

Complication	Comments
Catheter occlusion, venous thrombosis, phlebitis, sepsis	Contact physician or designated member of health care team for evaluation, diagnosis, and treatment of lines. For air embolism, place patient on his or her left side and lower head of the bed until resolved. Monitor for pneumothorax and ensure that trained staff handles catheters
Dehydration	Elevated BUN is noted. MD gives free water as 5% dextrose via peripheral vein
	Calculate needs as 30 mL/kg body weight or as 1 mL/kcal given. Alter as needed for diarrhea, medications used, ostomy, and losses from exudates such as burns or pressure ulcers
Electrolyte abnormalities	Monitor fluid status, organ system function, and serum sodium, potassium, phosphorus, calcium and magnesium regularly. Determine relevant cause or mechanism
Fluid overload	Greater than 1-kg gain per day is noted. Calculate needs and decrease volume to meet needs; diuretics or dialysis may be needed; a higher concentration of dextrose or lipids may be needed if fluid restriction is required
Gallbladder disorder	Cholelithiasis, gallbladder sludge, or cholecystitis can be caused or worsened by prolonged gallbladder stasis. Stimulate contraction by providing about 20–30% of calories as fat and stop glucose infusion several hours a day. Oral or enteral intake also helps. Treatment with metronidazole, ursodeoxycholic acid, phenobarbital, or cholecystokinin helps some patients with cholestasis
Hypocalcemia	Consider endocrine causes. Evaluate for hypoalbuminemia. Add additional calcium if needed
Hypercalcemia	Consider endocrine causes. Evaluate vitamin D, use isotonic saline, and add inorganic phosphate to solution until normal
Hypoglycemia	Administer more dextrose; reduce or discontinue insulin use; gradually taper infusion rate during weaning
Hyperglycemia	Reduce total grams of dextrose in the solution; add or increase insulin; consider use of lipids as partial substrate; advance feedings more slowly. Sometimes use of PN with a small amount of EN or oral intake can be helpful. Blood glucose >220 mg/dL can cause hyperinsulinemia, increased intracellular transport of potassium and phosphate with hypokalemia and hypophosphatemia. Impaired phagocytosis and neutrophil clearance may also occur
Hypokalemia	Increase potassium in solution and monitor potassium-depleting diuretic use such as furosemide. Add additional potassium if needed
Hyperkalemia	Evaluate renal function; decrease potassium in solution and evaluate medications used; reduce exogenous supplements
Hypomagnesemia	Increase magnesium in solution and monitor refeeding. Consider if magnesium-wasting medications are being used. Additional magnesium may be needed
Hypermagnesemia	Decrease magnesium in solution
Hyponatremia	Sometimes this occurs with fluid overload and total body water excess. Only occasionally is added sodium required
Hypernatremia	Replace fluids with a more dilute CPN solution; decrease sodium
Hyperphosphatemia	Evaluate renal function; decrease phosphate in solution and use phosphate binders if necessary
Hypophosphatemia	Increase phosphate in solution and monitor for refeeding syndrome
Lipid abnormalities	Decrease lipids if triglycerides are higher than 300 mg/dL; infuse over a longer time period; calculate that total kilocalories are not greater than 60% from lipids. Lipids over 2 g/kg/d can increase congestion of reticuloendothelial system and impair clearance of triglycerides. Some studies suggest use of omega-3 fatty acids to reverse cholestasis
Liver function changes	Altered liver function from excess energy may cause fatty infiltration and increased alkaline phosphatase. Liver dysfunction may be transient, evidenced by increased transaminases, bilirubin, and alkaline phosphatase; it commonly occurs when CPN is started. Delayed or persistent elevations may result from excess amino acids. Pathogenesis is unknown, but cholestasis and inflammation may contribute. Progressive fibrosis occasionally develops. Reducing protein delivery may help. For painful hepatomegaly, reducing CHO infusion may be needed
Metabolic Bone Disease	Bone demineralization develops in some patients given CPN for >3 months. Temporarily or permanently stopping CPN is the only known treatment
Pulmonary complications	Calculate needs and avoid overfeeding. Minimal kilocalories may be best, e.g., 20–25 kcal/kg. CHO provision should not exceed 4–5 mg/kg/min; avoid fluid excesses. Respiratory failure may occur in patients with limited pulmonary reserve. Prolonged mechanical ventilation can occur with carbon dioxide retention associated with overfeeding
Renal function changes	Protein excesses >2 g/kg daily can increase ureagenesis and decrease renal function or cause dehydration

Adapted from: Merck–Total Parenteral Nutrition, http://www.merck.com/mmpe/sec01/ch003/ch003c.html, accessed March 11, 2010.

Common Drugs Used and Potential Side Effects

- Basic CPN solutions are prepared using sterile techniques, usually in liter batches. Normally, 2 L/d of the standard solution is needed for adults. An effective, standardized PN process uses standardized formulations and products, ordering, labeling, screening, compounding, and administration (Kochevar et al, 2007).
- Contact the pharmacy for a list of drugs that are stable and compatible with PN solutions or nutrient additives. Often, H_2-blockers, steroids, and insulin are added to PN solutions.

- Intestinal tropic factors, such as recombinant human growth hormone (r-hGH), are used for SBS patients as part of intestinal rehabilitation regimens (Matarese et al, 2005).
- There are potential therapeutic roles for growth hormone, testosterone, oxandrolone, and megestrol acetate (Gullett et al, 2010).
- Loss of muscle mass occurs from increased protein degradation (cachexia), decreased rate of muscle protein synthesis (inactivity or bed rest), or both (sarcopenia). Nutrition therapy with an emphasis on high-quality protein plus use of an anabolic agent can slow or prevent muscle loss (Evans, 2010).
- Parenteral omega-3 fatty acids, such as Omegaven, may benefit patients with SBS who develop PN-associated liver disease (PNALD) with their CPN (Diamond et al, 2009).

Herbs, Botanicals, and Supplements

- Obesity creates a low-grade SIRS with lipotoxicity and cytokine dysregulation that may respond to arginine, fish oil, and carnitine at the molecular level (Cave et al, 2008).
- Herbs and botanical supplements should not be added to any IV feedings.
- Prescription and nonprescription medications are commonly used together; nearly 1 in 25 individuals may be at risk for a major drug–drug interaction (Qato et al, 2008).
- Prebiotics or probiotics may be useful for the prevention of infection.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss with patient/caretakers the goals of the PN, especially if home CPN will be used. Discuss aseptic technique, input and output records, CPN pump use, medications, additives, and complications. Long-term consequences should be discussed, such as trace element deficiencies and metabolic problems.
- Teach transition processes when and if patient is ready. Nutrition therapy is challenging; the objective is to restore enteral autonomy to a patient with a complex medical and surgical history, and a coordinated team effort is needed to wean from PN eventually (Weseman and Gilroy, 2005). Assistance from a registered dietitian is recommended.
- Wean from CPN to TF if patient tolerates one third to one half of kilocalorie needs by that route.
- To wean from CPN to oral diet, start with sips of clear liquids and advance, if tolerated, to lactose-free full liquids by the second day. When intake is >500 kcal orally, reduce CPN by 50%. When patient is consuming two thirds to three fourths of estimated needs orally, discontinue CPN by tapering (first hour by 50%, second hour by 75%, and third hour 100%). Nausea and vomiting may occur; eating some type of concentrated CHO during transition to oral diet is helpful.
- Discuss potential problems and when to call the doctor, the dietitian, the pharmacist, or the nurse.
- Discuss psychosocial issues related to adaptation to PN, oral deprivation, sense of loss, grief, and lifestyle changes. Quality of life tends to decline with long-term CPN use (Winkler, 2010). Encourage the patient to participate in favorite activities and physical activity as much as possible (Oz et al, 2008).
- Promote positive communications and collaboration among members of the health care team. A safe PN system must minimize procedural incidents; clinicians with nutrition therapy expertise are essential team members (Kochevar et al, 2007).

Patient Education—Infusion Safety

- Solutions must always be prepared and handled under sterile conditions. A standardized process for PN must be explored to improve patient safety and clinical appropriateness and to maximize resource efficiency (Kochevar et al, 2007).
- Home CPN requires aseptic technique and meticulous catheter care. Discuss infection control measures because catheter-related bloodstream infections are serious, critical complications.
- Change bag, tubing, and cassette every 24 hours or as recommended by the facility or agency.

For More Information

- American Society for Nutrition
  http://www.nutrition.org/
- ASPEN
  http://www.nutritioncare.org/
- Certification in Nutrition Therapy
  http://www.nutritioncare.org/nbnsc/
- NIH
  http://www.nlm.nih.gov/medlineplus/druginfo/meds/a601166.html

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APPENDIX A

Nutritional Review

RECOMMENDED DIETARY ALLOWANCES AND DIETARY REFERENCE INTAKES

The Dietary Reference Intakes (DRIs) are nutrient-based reference values for use in planning and assessing diets and for other purposes (IOM, 2010). DRIs replaced older tools first published by the National Academy of Sciences in 1941. The DRI values comprise seven reports with more detailed guidance than the old system. The DRIs are composed of the following:

- **Estimated average requirements** (EAR), expected to satisfy the needs of 50% of the people in that age group.
- **Recommended dietary allowances** (RDA). After computing the EARs for each age/gender category, the Food and Nutrition Board (FNB) then established RDAs to meet the nutrient requirements of each category.
- **Adequate intake** (AI), where no RDA has been established
- **Tolerable upper intake levels** (UL), to caution against excessive intake of nutrients (like vitamin D) that can be harmful in large amounts.

REFERENCE

IOM. Institute of Medicine, National Academy of Sciences. Dietary Reference Intakes, 2001. Web site accessed 3/14/10. http://www.iom.edu/Object.File/Master/7/294/0.pdf

Reader Please Note: All tables in this Appendix A are derived from the ARS Nutrient Database for Standard Reference, Release 17.
Foods are from single nutrient reports, sorted either by food description or in descending order by nutrient content in terms of common household measures. Mixed dishes are not included here. The food items and weights in these reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods.

MACRONUTRIENTS

Acceptable macronutrient distribution ranges for individuals have been set for carbohydrate, fat, *n*-6 and *n*-3 polyunsaturated fatty acids, and protein on the basis of evidence through the Institute of Medicine.

Carbohydrates and Fiber

Carbohydrates (CHO) are essential for life and to provide energy to the body. The brain and central nervous system (CNS) require a continuously available glucose supply. When it is necessary, lean body mass is metabolized to provide glucose for these tissues. Generally, 90% of carbohydrates are absorbed from a mixed diet. The RDA for carbohydrate is set at 130 g/d for adults and children, based on the average minimum amount of glucose used by the brain. The median intake of carbohydrates is approximately 220–330 g/d for men and 180–230 g/d for women. To plan a balanced intake between 45 and 65% of kcal/d, it would equal 135 to 195 g carbohydrate on a 1200 kcal diet; 202–292 g CHO on an 1800 kcal diet; or 270–390 g CHO for a 2400-kcal diet.

No recommendations based on glycemic index have been made. Intensely flavored artificial sweeteners that are approved for use in the United States include aspartame (NutraSweet), acesulfame-K (Sunette), neotame, saccharin (Sweet n Low), sucralose (Splenda), and stevia (Truvia). These are many times sweeter than natural sugars. Sugar replacers are natural sugar-free sugar alcohols such as mannitol, erythritol, isomalt, lactitol, maltitol, xylitol, and sorbitol. Unlike artificial sweeteners, these are used in the same amount as sugars and have the same bulk and volume. Labels indicate "sugar free" or "no sugar added. Sorbitol has a sweetness value of 60; mannitol has sweetness value of 50, or half that of table sugar.

Both soluble and insoluble fiber play an important role in maintenance of health. Except in a few therapeutic situations, fiber should be obtained from food sources (20 and 35 g/d for adults). Men up to age 50 need 38 g/d; over age 50, 30 g/d. Women up to age 50 need 25 g fiber per day, women over age 50, 21 g fiber per day. All dietary fibers, regardless of type, are readily fermented by microflora of the small intestines, producing short-chain fatty acids (acetate, propionate, and butyrate). An overview of carbohydrate and fiber classifications is provided in Table A-1. A food list of fiber sources can be found in Table A-2.

TABLE A-1 Carbohydrate and Fiber

Class	Type	Food Sources	Comments
Monosaccharides	Glucose (dextrose)	Corn syrup, honey, fruits, vegetables	Most important; most widely distributed. Sweetness value = 74.
	Fructose (levulose, fruit sugar)	High-fructose corn syrup, honey, fruits, vegetables	Sweetest, especially when fruit ripens. Most fruits contain 1–7%. Makes up 40% of the weight of honey. Sweetness value = 173.
	Galactose	Milk sugar	Part of the lactose molecule. Sweetness value = 32.
	Mannose	Found in poorly digested fruit structures	Of little nutritional value
Disaccharides	Sucrose (table sugar)	Cane or beet sugars, maple sugar, grape sugar, some natural fruits and vegetables	Formed when glucose and fructose are linked together. Honey is a form of sucrose known as an invert sugar. Sweetness value = 100.
	Lactose (milk sugar)	Milk, cream, whey	Glucose + galactose molecules in the mammary glands of lactating mammals. 7.5% of the composition of human milk, 4.5% of cow's milk. Sweetness value = 16.
	Maltose (malt sugar)	Malt, sprouting grains, partially digested starch	Made of two glucose molecules. Sweetness value = 32.
Polysaccharides—digestible	Starch (amylose and amylopectin)	Modified food starch, potatoes, beans, breads, rice, pasta, starchy products such as tapioca	Most starches require cooking for best digestion. When cooked and cooled, they are effective thickeners (such as modified food starch).
	Glycogen	No foods	Muscle and liver storage form of glucose; only 18 hours can be stored in one day.
Polysaccharides—indigestible (fiber)	**Insoluble fibers:** Cellulose	Soybean hulls, fruit membranes, legumes, carrots, celery, broccoli, and many other vegetables. The most abundant organic compound in the world.	Insoluble fibers increase fecal volume (bulk) and decrease colonic transit time by their ability to increase water-holding capacity; excess may deplete mineral status.
	Insoluble hemicelluloses (xylan, galactan, mannan, arabinose, galactose)	Corn hulls, wheat and corn brans, brown rice, cereal	The predominant sugar is used to name it.
	Algal polysaccharides (carrageenan)	Processed foods such as baby food, ice cream, sour cream	Extracted from seaweed and algae.
	Lignin (noncarbohydrate)	Wheat straw, alfalfa stems, tannins, cottonseed hulls	Polymer made of phenylpropyl alcohols and acids. Flaxseed lignin is an excellent antioxidant. Lignin may lower serum cholesterol levels.
	Cutin (noncarbohydrate)	Apple or tomato peels, seeds in berries, peanut or almond skins, onion skins	
	Soluble fibers: Pectin (polygalacturonic acid)	Citrus pulp, apple pulp, strawberries, sugar beet pulp, banana, cabbage and *Brassica* foods, legumes (such as kidney beans), sunflower heads	Soluble fibers decrease serum cholesterol by decreasing the enterohepatic recycling of bile acids and increasing use of cholesterol in bile synthesis. This stabilizes blood glucose levels and maintains mineral nutriture. Little effect on fecal bulk or transit time.
	Beta-glucans (glucopyranose)	Oat and barley bran; soy fiber concentrate.	Lowers serum cholesterol
	Gums (galactose and glucuronic acid)	Oats, guar gum, legumes, barley, xanthan from prickly ash trees	Useful for gel formation which slow digestion and slows down transit time; helpful in diarrhea.
	Psyllium	Extracted from psyllium seeds or plantains	High water binding capacity (choking hazard)
Oligosaccharides	Raffinose and stachyose	Beans and other legumes	Low-molecular-weight polymers containing 2–20 sugar molecules.
	Fructans [fructooligosaccharides (FOSs), inulin, oligofructose]	Wheat, onions, garlic, bananas, chicory, tomatoes, barley, rye, asparagus, and Jerusalem artichokes	They add flavor and sweetness to low-calorie foods and provide prebiotics to stimulate the growth of intestinal bacteria, especially *bifidobacteria*. May be used as a fat replacer.
Miscellaneous carbohydrates	Chitin (Glucopyranose)	Supplement made from crab or lobster shells	May reduce serum cholesterol
	Polydextrose, polyols (sorbitol, mannitol, etc.)	Synthesized	Bulking agent or sugar substitute.
	Algal polysaccharides (carrageenan)	Isolated from algae and seaweed	Forms a gel used as thickening agent. Can be toxic.

Sources: Byrd-Brenner C, et al. *Wardlaw's perspectives in nutrition*. 8th ed. New York: McGraw-Hill, 2008; Gallagher M. *Macronutrients in Krause's food and nutrition therapy*. Mahan LK, Escott-Stump S, editors. 12th ed. St Louis, MO: Elsevier, 2008.

TABLE A-2 Food Sources of Dietary Fiber[a]

Food, Standard Amount	Dietary Fiber (g)	Calories	Food, Standard Amount	Dietary Fiber (g)	Calories
Navy beans, cooked, 1/2 cup	9.5	128	Potato, baked, with skin, one medium	3.8	161
Bran ready-to-eat cereal (100%), 1/2 cup	8.8	78	Soybeans, green, cooked, 1/2 cup	3.8	127
Kidney beans, canned, 1/2 cup	8.2	109	Stewed prunes, 1/2 cup	3.8	133
Split peas, cooked, 1/2 cup	8.1	116	Figs, dried, 1/4 cup	3.7	93
Lentils, cooked, 1/2 cup	7.8	115	Dates, 1/4 cup	3.6	126
Black beans, cooked, 1/2 cup	7.5	114	Oat bran, raw, 1/4 cup	3.6	58
Pinto beans, cooked, 1/2 cup	7.7	122	Pumpkin, canned, 1/2 cup	3.6	42
Lima beans, cooked, 1/2 cup	6.6	108	Spinach, frozen, cooked, 1/2 cup	3.5	30
Artichoke, globe, cooked, one each	6.5	60	Shredded wheat ready-to-eat cereals, various, ~1 ounce	2.8–3.4	96
White beans, canned, 1/2 cup	6.3	154	Almonds, 1 ounce	3.3	164
Chickpeas, cooked, 1/2 cup	6.2	135	Apple with skin, raw, one medium	3.3	72
Great northern beans, cooked, 1/2 cup	6.2	105	Brussels sprouts, frozen, cooked, 1/2 cup	3.2	33
Cowpeas, cooked, 1/2 cup	5.6	100	Whole-wheat spaghetti, cooked, 1/2 cup	3.1	87
Soybeans, mature, cooked, 1/2 cup	5.2	149	Banana, one medium	3.1	105
Bran ready-to-eat cereals, various, ~1 ounce	2.6–5.0	90–108	Orange, raw, one medium	3.1	62
Crackers, rye wafers, plain, two wafers	5.0	74	Oat bran muffin, one small	3.0	178
Sweet potato, baked, with peel, one medium (146 g)	4.8	131	Guava, one medium	3.0	37
Asian pear, raw, one small	4.4	51	Pearled barley, cooked, 1/2 cup	3.0	97
Green peas, cooked, 1/2 cup	4.4	67	Sauerkraut, canned, solids, and liquids, 1/2 cup	3.0	23
Whole-wheat English muffin, one each	4.4	134	Tomato paste, 1/4 cup	2.9	54
Pear, raw, one small	4.3	81	Winter squash, cooked, 1/2 cup	2.9	38
Bulgur, cooked, 1/2 cup	4.1	76	Broccoli, cooked, 1/2 cup	2.8	26
Mixed vegetables, cooked, 1/2 cup	4.0	59	Parsnips, cooked, chopped, 1/2 cup	2.8	55
Raspberries, raw, 1/2 cup	4.0	32	Turnip greens, cooked, 1/2 cup	2.5	15
Sweet potato, boiled, no peel, one medium (156 g)	3.9	119	Collards, cooked, 1/2 cup	2.7	25
Blackberries, raw, 1/2 cup	3.8	31	Okra, frozen, cooked, 1/2 cup	2.6	26
			Peas, edible-pod, cooked, 1/2 cup	2.5	42

[a]Food sources of dietary fiber are ranked by grams of dietary fiber per standard amount; also calories in the standard amount. All are ≥10% of AI for adult women, which is 25 g/d.

Fatty Acids and Lipids

The usual American diet contains 35–40% fat kcal. Fats are carriers for fat-soluble vitamins and essential fatty acids (EFAs). Fat is essential for cell membranes (especially the brain) serves as an insulating agent for organs and is a rich energy source. Generally, 95% of fat from the diet is absorbed. One to two percent of calories should be available as linoleic acid to prevent EFA deficiency. People with low body fat stores, very malnourished persons, psychiatric patients, and premature low birth weight (LBW) infants are at risk for EFA deficiency.

Lipase is needed to metabolize long-chain triglycerides (LCT) into free fatty acids (FFAs). When parenteral fat emulsions contain LCT, large doses may compromise immune function, elevate serum lipids, impair alveolar diffusion capacity, or decrease reticular endothelial system function. Medium-chain fatty acids (MCFAs) are produced from medium-chain triglycerides (MCTs). MCTs are transported to the liver via the portal vein and, therefore, do not require micelle or chylomicron formation.

Omega-3 fatty acids reduce inflammation and help prevent certain chronic diseases. These essential fatty acids are highly concentrated in the brain. Two to three servings of fatty fish per week (about 1250 mg EPA and DHA per day) are beneficial for most people. If using fish oil supplements, 3000–4000 mg standardized fish oils would be needed per day. Safe and effective doses of omega-3 fatty acid supplements have not been established in children. EPA and arachidonic acids are transformed into eicosanoids for prostaglandin, leukotriene, thromboxane, and prostacyclin synthesis. Prostaglandins are used in many diverse hormone-like compounds. Omega-3 fatty acids should be used cautiously by people who have a bleeding disorder, take

blood thinners, or are at an increased risk for hemorrhagic stroke.

It is very important to maintain a balance between omega-3 and omega-6 fatty acids in the diet. The Mediterranean diet consists of a healthier balance between omega-3 and omega-6 fatty acids than the typical American diet. Omega-6 fatty acids play a role as proinflammatory agents and are useful when initiating a stress response.

Sphingolipids are hydrolyzed throughout the GI tract to regulate growth, differentiation, apoptosis, and other cellular functions. They reduce low-density lipoprotein (LDL) levels and increase high-density lipoprotein (HDL) levels. Dietary constituents such as cholesterol, fatty acids, and mycotoxins alter their metabolism.

The recommended intakes of total fat per day equal 20–35% of kcal. Of this, consume 1–2% from omega-3 fatty acids and 5–8% from omega-6 fatty acids. Table A-3 describes more details.

Proteins and Amino Acids

Amino acids and proteins are the building blocks of life. All growth and repair functions of the body require utilization and availability of amino acids in the proper proportion and amounts. Average total essential amino acid needs are as follows: infants, 40% essential; children, 36% essential; and adults, 19% essential. This translates into 52 g protein for teenaged boys aged 14–18 years and 46 g for teenaged girls aged 14+, and women. Pregnant or nursing women need 71 g/d. Men aged 19+ need 56 g protein per day. Need increase with injury, trauma, and certain medical conditions.

TABLE A-3 Fats and Lipids

Class	Fatty Acid Component	Key Food Sources	Comments
Simple lipids *Neutral fats (acyl-glycerols)*	Mono- and diglycerides	Additive in low-fat foods	Glycerol with one or two esterified fatty acids
	Triglycerides (triacylglycerols)	Fats and oils	Glycerol with three esterified fatty acids
Fatty acids, polyunsaturated Omega-3 family	Alpha linolenic acid (ALA)	Vegetable oils (soybean, canola, or rapeseed), flaxseeds, flaxseed oil, soybeans, pumpkin seeds, pumpkin seed oil, purslane, perilla seed oil, walnuts, walnut oil. Flaxseed and flaxseed oil should be kept refrigerated; whole flaxseeds should be ground within 24 hours of use.	**An essential fatty acid.** Once eaten, the body converts ALA to EPA and DHA, the two types of omega-3 fatty acids more readily used by the body. The adequate daily intake of ALA should be 1.6 and 1.1 g for men and women, respectively.
	Eicosapentaenoic acid (EPA) and Docosahexanoic acid (DHA)	Cold-water fish such as salmon, mackerel, eel, tuna, herring, halibut, trout, sardines, and herring. Fish oil capsules should be refrigerated.	EPA and DHA (1 g/d) from fatty fish can reduce CHD and some types of cancer. Infants who do not receive sufficient DHA from their mothers during pregnancy are at risk for nerve or vision problems.
Omega-6 family	Linoleic acid	Safflower, sunflower, corn, soybean, peanut oils.	**An essential fatty acid**
	Conjugated linoleic acid (CLA)	Naturally rich in milk fat	Group of isomers produced by rumen bacteria. CLA may have a role in weight management and other functions.
	Arachidonic acid	Animal tissues (very low). There is no good dietary source.	
Monounsaturated Omega-9 family	Oleic acid	Olive oil, canola oil, peanut oil, sunflower oil, and sesame oil. Avocados, olives, nut butters, such as peanut butter, nuts and seeds, such as macadamia nuts, pecans, and almonds	
Compound lipids *Phospholipids*	Glycerophosphatides (lecithins, cephalins, plasmologens)	Egg yolks, liver, wheat germ, peanuts	Glycerol with two fatty acids and a nitrogen
	Glycosphingolipids (ceramides, sphingomyelins)	Low in the food supply, mainly in dairy products, eggs, soybeans. Fruit has a tiny bit.	Sphingosine with one esterified fatty acid (a ceramide). Found in myelin of nerve tissue.
Glycolipids	Cerebrosides, gangliosides	Low in the food supply.	Found in nerve and brain tissue. A ceramide linked to a monosaccharide or an oligosaccharide

(continued)

TABLE A-3 Fats and Lipids *(continued)*

Class	Fatty Acid Component	Key Food Sources	Comments
Lipoproteins	Protein–lipid combination	Made by the liver	VLDL, LDL, HDL have various roles in health maintenance.
Miscellaneous lipids *Sterols*	Cholesterol	Liver, heart, and other organ meats	Steroid nucleus (synthesized from acetyl-coenzyme A). Technically a wax.
Fat-soluble vitamins	A,D, E, and K	Various food sources	Lipid soluble; some are esterified with 1 fatty acid
Trans fatty acids		Margarines, some cookies and crackers. The new food label includes the amount of trans fat in a serving.	These are made by hydrogenation of vegetable oils to form more solid products (i.e., margarine).The process increases the amount of saturation and converts natural *cis* double bonds to *trans* double bonds. To decrease intake, use leaner cuts of meat, fewer cold cuts. Choose skim milk, fat-free products in food choices and in cooking.
Fat-related substances	Carnitine	Produced in the liver from essential amino acids lysine and methionine.	Carnitine transports fatty acids into mitochondria, where they undergo beta-oxidation. When in short supply, production of ATP slows down or halts altogether, affecting heart or skeletal muscle. In renal failure, it is a useful additive to improve fatty acid metabolism.
	Myo-inositol	Found as phospholipid in animals, phytic acid in plants.	Inositol phosphates liberated from glycerophosphatides acts as a secondary messenger in the release of intracellular of calcium, which in turn causes activation of certain cellular enzymes and produces hormonal responses. Possible role in diabetes mellitus or in renal failure.

The main protein source in the American diet is animal protein from beef and poultry.

To produce the nonessential amino acids from dietary intake of the essentials, it is recommended that the limited amino acids be consumed within a 24-hour period of each other. Protein synthesis requires all amino acids; an insufficient amount of any one may impede or slow formation of the polypeptide chain. For valine, leucine, and isoleucine, the requirement of each is increased by excess of the other branched-chain amino acids (BCAAs).

Protein requirement is inversely related to calories when the latter are deficient. Generally, more than 90% of protein is absorbed from the diet. Foods of high biological value (HBV) contain approximately 40% EAAs. Table A-4 indicates which amino acids are essential and which can be made by the body (nonessential). Eight amino acids are generally regarded as essential for humans: threonine, valine, tryptophan, isoleucine, leucine, lysine, phenylalanine, methionine (names of these amino acids are easy to remember by the phrase "**TV TILL PM**"). Foods that contain all of the essential amino acids are called complete proteins. Incomplete proteins tend to be deaminated for sources of energy rather than being available for new tissue, healing, and growth. For biological value of proteins see Table A-5 and see Table A-6 for sources of protein.

MICRONUTRIENTS

Minerals

Minerals are inorganic compounds containing no carbon structures. There are 22 essential minerals known to be needed from the diet. Macrominerals (needed in large amounts) include calcium, phosphorus, magnesium, potassium, sodium, chloride, and sulfur. Trace minerals include iron, copper, selenium, fluoride, iodine, chromium, zinc, manganese, molybdenum, cobalt, and others.

Major Minerals

Minerals that are needed at levels of 100 mg daily or more are known as macrominerals.

Calcium: Calcium absorption is dependent upon the calcium needs of the body, foods eaten, and the amount of calcium in foods eaten. Vitamin D, whether from diet or exposure to the ultraviolet light of the sun, increases calcium absorption. Calcium absorption tends to decrease with increased age for both men and women. *RDA for calcium is 1000 mg for most adults, 1300 mg for teenagers, and 1200 mg for those over 50 years.*

TABLE A-4 Amino Acids

Essential Amino Acids	Type	Food Sources	Comments: Must be Consumed
Histidine	Aromatic	Dairy, meat, poultry, fish.	Precursor of histamine. Important for red and white blood cells. An important source of carbon atoms in the synthesis of purines. Histidine is needed to help grow and repair body tissues, and to maintain the myelin sheaths that protect nerve cells. It also helps manufacture red and white blood cells, and helps to protect the body from heavy metal toxicity. Histamine stimulates the secretion of the digestive enzyme gastrin. Extra is needed by infants and renal failure patients.
Isoleucine	Neutral	Meats, fish, cheeses, nuts	Belongs to the branched-chain amino acid group (BCAAs), which help maintain and repair muscle tissue. Important for stabilizing and regulating blood sugar and energy levels. Needed for synthesis of hemoglobin and energy regulation. Need 20 mg/kg body weight
Leucine	Neutral	Soybeans, cowpeas, lentils, beef, peanuts, pork salami, salmon, shellfish, chicken, eggs	Belongs to the branched-chain amino acid group (BCAAs). Second most common amino acid found in proteins; necessary for the optimal growth of infants and for the nitrogen balance in adults. Needed for wound healing and healthy skin and bones. Need 39 mg/kg body weight
Lysine	Basic	Limited in corn, wheat, and rice.	It is an essential building block for all protein, and is needed for proper growth and bone development in children. Lysine helps the body absorb and conserve calcium, form collagen and muscle protein, and synthesize enzymes and hormones. Need 30 mg/kg body weight
Methionine	Sulfur	Limited in soy products.	Helps to initiate translation of messenger RNA by being the first amino acid incorporated into the N-terminal position of all proteins. Methionine supplies sulfur and other compounds required by the body for normal metabolism and growth. Reacts with ATP to form *S*-adenosyl methionine (SAM) which is the principal methyl donor in the body. It contributes to the synthesis of epinephrine and choline. Need 10.4 + 4.1 (15 total) mg/kg body weight
Phenylalanine	Aromatic	Soybeans, lentils, nuts, seeds, chicken, fish, eggs.	Exhibits ultraviolet radiation absorption properties with a large extinction coefficient. Phenylalanine is part of the composition of aspartame, a common sweetener found in prepared foods (particularly soft drinks, and gum). Key role in the biosynthesis of other amino acids and some neurotransmitters. Healthy nervous system, memory and learning. Useful against depression. Need, along with tyrosine, 25 (total) mg/kg body weight
Threonine	Neutral	Soybeans, pork, lentils, beef, fish.	Important component in the formation of protein, collagen, elastin and tooth enamel. It is also important for production of neurotransmitters and health of the nervous system and the immune system. Need 15 mg/kg body weight
Tryptophan	Aromatic	Rich in flaxseed, salami, lentils, turkey, nuts, eggs. Limited in corn.	Formed from proteins during digestion by the action of proteolytic enzymes. Tryptophan is also a precursor for serotonin (a neurotransmitter) and melatonin (a neurohormone). Precursor of niacin. Tryptophan may enhance relaxation and sleep, relieve minor premenstrual symptoms, sooth nerves and anxiety, and reduce carbohydrate cravings. Need 4 mg/kg body weight.
Valine	Neutral	Meat, eggs, milk, cereal proteins.	Belongs to the branched-chain amino acid group (BCAAs). Constituent of fibrous protein in the body. Useful in treatments involving muscle, mental, and emotional upsets, and for insomnia and nervousness. Valine may help treat malnutrition associated with drug addiction. Needed for muscle development. Need 26 mg/kg body weight

(continued)

TABLE A-4 Amino Acids *(continued)*

Conditional Amino Acids	Type	Food Sources	Comments: Must be Consumed in the Diet During Stress or Illness
Arginine, citrulline, ornithine	Basic	Peanuts, almonds, seeds	These three amino acids are part of urea acid cycle. Arginine plays an important role in cell division, wound healing, removing ammonia from the body, immune function, and the release of hormones. Extra needed in growing children. Citrulline supports the body in optimizing blood flow through its conversion to L-arginine and then nitric oxide (NO). Ornithine is a precursor of citrulline, glutamic acid, and proline; it is an intermediate in arginine biosynthesis.
Cysteine	Sulfur	Can be made from homocysteine but cannot be synthesized on its own. Limited in soy products.	Spares methionine. Component of nails, skin and hair. Antioxidant when taken with vitamin E and selenium. Extra needed in infancy, liver failure. Naturally occurring hydrophobic amino acid with a sulfhydryl group; found in most proteins. *N*-acetyl cysteine (NAC) is the most frequently used form and it helps toxify harmful substances in the body. Both cysteine and NAC increase levels of the antioxidant glutathione.
Glutamine	Acidic	Beef, chicken, milk, eggs, what.	One of the 20 amino acids generally present in animal proteins; the most abundant amino acid in the body. Over 61% of skeletal muscle tissue is glutamine. It contains two ammonia groups, one from precursor glutamate, and the other from free ammonia in the bloodstream. Glutamine is converted to glucose when more glucose is required by the body as an energy source. Glutamine assists in maintaining the proper acid/alkaline balance in the body, and is the basis for the synthesis of RNA and DNA.
Glycine	Neutral	Abundant in fish, meat, dairy products, beans.	The simplest amino acid; it has no stereoisomers. The body uses it to help the liver in detoxification of compounds and for helping the synthesis of bile acids. Glycine is essential for the synthesis of nucleic acids, bile acids, proteins, peptides, purines, adenosine triphosphate (ATP), porphyrins, hemoglobin, glutathione, creatine, bile salts, one-carbon fragments, glucose, glycogen, and L-serine and other amino acids. Beneficial for skin and wound healing. It has a sweet taste.
Hydroxyproline			Derived from proline and used almost exclusively in structural proteins including collagen, connective tissue in mammals, and in plant cell walls. The nonhydroxylated collagen is commonly termed procollagen.
Proline	Cyclic	A diet low in ascorbic acid may lead to low levels of hydroxyproline. Derived from L-glutamate.	Needed for intracellular signaling. Involved in the production of collagen and in wound healing. Proline is the precursor for hydroxyproline, which the body incorporates into collagen, tendons, ligaments, and the heart muscle. Important component in certain medical wound dressings that use collagen fragments to stimulate wound healing.
Serine	Neutral; also an alcohol	Synthesized from glycine	Important for healthy brain and immunity. Variety of biosynthetic pathways including those involving pyrimidines, purines, creatine, and porphyrins. Serine has sugar-producing qualities, and is very reactive in the body. It is highly concentrated in all cell membranes, aiding in the production of immunoglobulins and antibodies. Need extra for hemodialysis patients.
Taurine		Found in seafood and meat.	Functions with glycine and gamma-aminobutyric acid as a neuroinhibitory transmitter. Also needed for brain function and metabolism of magnesium, calcium and potassium. Extra needed for infants and in trauma, infection, renal failure.
Tyrosine	Aromatic	Soy, poultry, dairy and other high protein foods.	Metabolically synthesized from phenylalanine to become the para-hydroxy derivative. Tyrosine is a precursor of the adrenal hormones epinephrine, norepinephrine, and the thyroid hormones L-tyrosine, through its effect on neurotransmitters, is used to treat conditions including mood enhancement, appetite suppression, and growth hormone (HGH) stimulation. Extra is needed for infants and in chronic renal failure.

(continued)

TABLE A-4 Amino Acids *(continued)*

Nonessential and Other Amino Acids/Proteins	Type	Food Sources	Comments: Made by the Body if Enough Nitrogen is Available
Alanine	Neutral		Removes toxic substances released from breakdown of muscle protein during intensive exercise. Part of glucose–alanine pathway. Alanine comes from the breakdown of DNA or the dipeptides, anserine and carnosine, and the conversion of pyruvate for carbohydrate metabolism.
Asparagine	Acidic	Seafood, meat, casein, dairy products, eggs, beans, seeds, nuts, corn.	Healthy CNS. Asparagine is synthesized from aspartic acid and ATP. One of the principal and frequently the most abundant amino acids involved in the transport of nitrogen and in amination/transamination. It serves as an amino donor in liver transamination.
Aspartic acid	Acidic		Used for immunity and removal of toxins and ammonia from the body. Aspartic acid is alanine with one of the β hydrogens replaced by a carboxylic acid group. It supports metabolism during construction of asparagine, arginine, lysine, methionine, threonine, isoleucine, and several nucleotides.
Carnitine			Produced in the liver, brain and the kidneys from methionine and lysine; responsible for the transport of long-chain fatty acids into the energy-producing mitochondria. Carnitine helps maintain a healthy blood lipid profile and promote fatty acid utilization within heart muscle.
Creatine			Synthesized in the liver, kidneys and pancreas out of arginine, methionine and glycine; increases the availability of cellular ATP. Donates a phosphate ion to increase the availability of ATP. Stored in muscle cells as phosphocreatine; helps generate cellular energy for muscle contractions.
Glutamic acid	Acidic		Gamma-aminobutyric acid (GABA) is formed from glutamic acid with the help of vitamin B_6, is found in almost every region of brain, and is formed through the activity of the enzyme glutamic acid decarboxylase (GAD). GABA serves as an inhibitory neurotransmitter to block the transmission of an impulse from one cell to another in the CNS. The crystalline salt of glutamic acid, monosodium glutamate, contributes to the flavor called umami.
Glutathione		Seaweed	Glutathione (GSH) is a tripeptide composed of three different amino acids, glutamate, cysteine and glycine, which has numerous important functions within cells. Glutathione plays a role in such diverse biological processes as protein synthesis, enzyme catalysis, transmembrane transport, receptor action, intermediary metabolism, and cell maturation. Glutathione acts as an antioxidant used to prevent oxidative stress in most cells and help to trap free radicals that can damage DNA and RNA.
Theanine			L-theanine is the predominant amino acid in green tea and makes up 50% of the total free amino acids in the plant; the main component responsible for the taste of green tea. L-theanine is involved in the formation of the inhibitory neurotransmitter, gamma amino butyric acid (GABA). GABA influences the levels of two other neurotransmitters, dopamine and serotonin, producing a relaxation effect.

Adapted from: Furst P, Steele P. What are the essential elements needed for the determination of amino acid requirements in humans? The American Society for Nutritional Sciences *J Nutr* 134:1558, 2004;
Shils M, et al. *Modern nutrition in health and disease*. 9th ed. Baltimore: Lippincott Williams & Wilkins, 1999.

TABLE A-5 Biological Value of Proteins

Food Source	Biological Value	Food Source	Biological Value
Whey protein	104	Casein	77
Egg	100	Soybean	74
Cow's milk	95	Rice, white	67
Cottonseed	81	Wheat, whole	53
Beef	80	Sesame	50
Fish	79	Corn	49

From the Joint FAO/WHO Ad Hoc Expert Committee. Energy and protein requirements. WHO technical report no. 522. Geneva: World Health Organization, 1973, p. 67.

TABLE A-6 Protein Sources

Food	Serving	Weight (g)	Protein (g)
Hamburger, extra lean	6 ounces	170	48.6
Chicken, roasted	6 ounces	170	42.5
Fish	6 ounces	170	41.2
Tuna, water packed	6 ounces	170	40.1
Beefsteak, broiled	6 ounces	170	38.6
Cottage cheese or ricotta cheese	1 cup	225	28.1
Beef, fish, pork, chicken or turkey	3 ounces	85	21
Cheese pizza	2 slices	128	15.4
Yogurt, low fat	8 ounces	227	11.9
Tofu or vegetarian burger patty	1/2 cup	126	10–15
Lentils, cooked	1/2 cup	99	9
Skim milk	1 cup	245	8.4
Split peas, cooked	1/2 cup	98	8.1
Whole or chocolate milk	1 cup	244	8
Peanut or other nut butters	2 tablespoons	Varies	8
Kidney beans, cooked	1/2 cup	87	7.6
Cheddar cheese	1 ounce (1 slice)	28	7.1
Macaroni, cooked	1 cup	140	6.8
Soy milk	1 cup	245	6–9
Egg	1 large	50	6.3
Whole wheat bread	2 slices	56	5.4
White bread	2 slices	60	4.9
Rice, cooked	1 cup	158	4.3
Broccoli, cooked	5-inch piece	140	4.2
Nuts or seeds	2 tablespoons	Varies	3–5
Baked potato	2 × 5 inches	156	3
Corn, cooked	1 ear	77	2.6
Bread, cooked cereal or rice or pasta	1 slice	Varies	2

From: U. S. Department of Agriculture. Nutrient database, http://www.ars.usda.gov/SP2UserFiles/Place/12354500/Data/SR22/nutrlist/sr22w203.pdf, accessed January 22, 2010.

Roles. For strong bones and teeth, nerve irritability, muscle contraction, heart rhythm, blood coagulation, enzymes, osmotic pressure, intercellular cement, maintenance of cell membranes, and protection against high blood pressure. About 60% is bound to protein, mostly albumin. About 30–60% absorption occurs with intakes of 400–1000 mg.

Sources. Milk and cheese products remain the primary source of calcium for Americans. Phytates and excessive protein or zinc decrease absorption. Calcium from soy milk is absorbed only 75% as efficiently as from cow's milk. Calcium is lost in cooking some foods, even under the best conditions. To retain calcium, cook foods in a minimal amount of water and for the shortest possible time. Table A-7 lists both dairy and nondairy sources of calcium.

Signs of Deficiency. In hypocalcemia, there may be tetany, paresthesia, hyperirritability, muscle cramps, convulsions, and stunting in growth. No consistent data suggests that a high-protein diet depletes calcium.

Signs of Excess. With hypercalcemia, milk alkali syndrome, kidney stones or renal insufficiency can occur. Recommending excessive calcium use among the general, unsupervised public is not advisable. For bone health, vitamin D is the priority and should be given along with calcium.

Chloride: Chloride constitutes about 3% of total mineral content in the body. It is the main extracellular ion, along with sodium. *No RDA levels have been established for chloride. The upper limit (UL) for adults is 3.6 g/d.*

Roles. Digestion (HCl in stomach), acid–base balance, O_2/CO_2 exchange in red blood cells (RBCs), and fluid balance.

Sources. Table salt, salt substitutes containing potassium chloride, processed foods made with table salt, sauerkraut, snack chips, and green olives.

Signs of Deficiency. Hypochlorhydria and disturbed acid–base balance.

TABLE A-7 Dairy Sources of Calcium[a]

Dairy Food, Standard Amount	Calcium (mg)	Calories
Plain yogurt, nonfat (13 g protein/ 8 ounces), 8-ounce container	452	127
Romano cheese, 1.5 ounces	452	165
Pasteurized process Swiss cheese, 2 ounces	438	190
Plain yogurt, low-fat (12 g protein/ 8 ounces), 8-ounce container	415	143
Fruit yogurt, low-fat (10 g protein/ 8 ounces), 8-ounce container	345	232
Swiss cheese, 1.5 ounces	336	162
Ricotta cheese, part skim, 1/2 cup	335	170
Pasteurized process American cheese food, 2 ounces	323	188
Provolone cheese, 1.5 ounces	321	150
Mozzarella cheese, part-skim, 1.5 ounces	311	129
Cheddar cheese, 1.5 ounces	307	171
Fat-free (skim) milk, one cup	306	83
Muenster cheese, 1.5 ounces	305	156
Pudding, ready to eat, one cup	300	Varies
Macaroni and cheese, one cup prepared	300	Varies
1% low-fat milk, one cup	290	102
Low-fat chocolate milk (1%), one cup	288	158
2% reduced fat milk, one cup	285	122
Reduced fat chocolate milk (2%), one cup	285	180
Buttermilk, low-fat, one cup	284	98
Chocolate milk, one cup	280	208
Whole milk, one cup	276	146
Yogurt, plain, whole milk (8 g protein/ 8 ounces), 8-ounce container	275	138
Ricotta cheese, whole milk, 1/2 cup	255	214
Blue cheese, 1.5 ounces	225	150
Mozzarella cheese, whole milk, 1.5 ounces	215	128
Feta cheese, 1.5 ounces	210	113
Cottage cheese, one cup	150	
Frozen yogurt or ice cream	100	Varies
Non-food, standard amount	**Calcium (mg)**	**Calories**
Fortified ready-to-eat cereals (various), 1 ounce	236–1043	88–106
Soy beverage, calcium fortified, one cup	368	98
Sardines, Atlantic, in oil, drained, 3 ounces	325	177
Fortified orange juice, one cup	300	120
Tofu, firm, prepared with calcium, 1/2 cup	253	88
Bread, calcium-fortified, one slice	200	70
Pink salmon, canned, or sardines, with bone, 3 ounces	181	118
Collards, cooked from frozen, 1/2 cup	178	31
Molasses, blackstrap, one tbsp	172	47
Spinach, cooked from frozen, 1/2 cup	146	30
Soybeans, green, cooked, 1/2 cup	130	127
Turnip greens, cooked from frozen, 1/2 cup	124	24
Ocean perch, Atlantic, cooked, 3 ounces	116	103
Oatmeal, plain and flavored, instant, fortified, one packet prepared	99–110	97–157
Cowpeas, cooked, 1/2 cup	106	80
Almonds, 1 ounce	100	164
White beans, canned, 1/2 cup	96	153
Broccoli or kale, cooked from frozen, 1/2 cup	90–100	20
Okra, cooked from frozen, 1/2 cup	88	26
Soybeans, mature, cooked, 1/2 cup	88	149
Blue crab, canned, 3 ounces	86	84
Beet greens, cooked from fresh, 1/2 cup	82	19

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.

[a]Food sources of calcium ranked by milligrams of calcium per standard amount; also calories in the standard amount. All are ≥20% of AI for adults 19–50, which is 1,000 mg/d. Those containing 300 mg or more are highlighted in salmon, those with 200–299 mg are shaded lighter, and those with the lightest shading have 80–199 mg.

Note: Both calcium content and bioavailability should be considered when selecting dietary sources of calcium. Some plant foods have calcium that is well absorbed, but the large quantity of plant foods that would be needed to provide as much calcium as in a glass of milk may be unachievable for many. Many other calcium-fortified foods are available, but the percentage of calcium that can be absorbed is unavailable for many of them.

Signs of Excess. Disturbed acid–base balance.

Magnesium: Magnesium is a mineral needed by every cell of the body; half of the stores are found inside cells of body tissues and organs, and half are combined with calcium and phosphorus in bone. Only 1% of magnesium in the body is found in blood. The body works hard to keep blood levels of magnesium constant. Results of two national surveys indicated that the diets of most adult men and women do not provide the recommended amounts of magnesium. Adults aged 70 years and over eat less magnesium than younger adults, and non-Hispanic black subjects consume less magnesium than either non-Hispanic white or Hispanic subjects. Despite poor intakes, magnesium deficiency is rarely seen in the United States in adults. *RDA varies by age but is typically 420 mg for men and 320 mg for women. UL for supplemental magnesium for adolescents and adults is 350 mg/d.*

Roles. Magnesium is needed for more than 300 biochemical reactions including normal muscle contraction, nerve transmission and function, heart rhythm, energy metabolism and protein synthesis, enzyme activation (ADP, ATP), glucose utilization, bone matrix and growth, and normal Na^+/K^+ pump. Maintaining an adequate magnesium intake is a

TABLE A-8 Food Sources of Magnesium[a]

Food, Standard Amount	Magnesium (mg)	Calories
Pumpkin and squash seed kernels, roasted, 1 ounce	151	148
Brazil nuts, 1 ounce	107	186
Bran ready-to-eat cereal (100%), ~1 ounce	103	74
Halibut, cooked, 3 ounces	91	119
Quinoa, dry, 1/4 cup	89	159
Spinach, canned, 1/2 cup	81	25
Almonds, 1 ounce	78	164
Spinach, cooked from fresh, 1/2 cup	78	20
Buckwheat flour, 1/4 cup	75	101
Cashews, dry roasted, 1 ounce	74	163
Soybeans, mature, cooked, 1/2 cup	74	149
Pine nuts, dried, 1 ounce	71	191
Mixed nuts, oil roasted, with peanuts, 1 ounce	67	175
White beans, canned, 1/2 cup	67	154
Pollock, walleye, cooked, 3 ounces	62	96
Black beans, cooked, 1/2 cup	60	114
Bulgur, dry, 1/4 cup	57	120
Oat bran, raw, 1/4 cup	55	58
Soybeans, green, cooked, 1/2 cup	54	127
Tuna, yellowfin, cooked, 3 ounces	54	118
Artichokes (hearts), cooked, 1/2 cup	50	42
Peanuts, dry roasted, 1 ounce	50	166
Lima beans, baby, cooked from frozen, 1/2 cup	50	95
Beet greens, cooked, 1/2 cup	49	19
Navy beans, cooked, 1/2 cup	48	127
Tofu, firm, prepared with nigari,[b] 1/2 cup	47	88
Okra, cooked from frozen, 1/2 cup	47	26
Soy beverage, one cup	47	127
Hazelnuts, 1 ounce	46	178
Oat bran muffin, 1 ounce	45	77
Great northern beans, cooked, 1/2 cup	44	104
Oat bran, cooked, 1/2 cup	44	44
Brown rice, cooked, 1/2 cup	42	108
Haddock, cooked, 3 ounces	42	95

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.
[a]Food Sources of Magnesium ranked by milligrams of magnesium per standard amount; also calories in the standard amount. (All are ≥ 10% of RDA for adult men, which is 420 mg/d).
[b]Calcium sulfate and magnesium chloride.

positive lifestyle modification for preventing and managing high blood pressure. Elevated blood glucose levels increase the loss of magnesium in the urine, which in turn lowers blood levels of magnesium; this explains why low blood levels of magnesium are seen in poorly controlled type 1 and type 2 diabetes.

Sources. Magnesium is present in many foods but usually in small amounts. Eating a wide variety of foods, including five servings of fruits and vegetables daily and plenty of whole grains, helps to ensure an AI. The center of the chlorophyll molecule in green vegetables contains magnesium. Intake from a meal may be 45–55% absorbed. Beware of excess phytates. Water can provide magnesium, but the amount varies according to the water supply. "Hard" water contains more magnesium than "soft" water. Dietary surveys do not estimate magnesium intake from water, which may lead to underestimating total magnesium intake and its variability (see Table A-8).

Signs of Deficiency. When magnesium deficiency occurs (hypomagnesemia), it is usually due to excessive loss of magnesium in urine from diabetes, antibiotics, diuretics, or excessive alcohol use; gastrointestinal (GI) system disorders; chronically low intake of magnesium; chronic or excessive vomiting, diarrhea, and fat malabsorption. Magnesium deficiency can cause metabolic changes that may contribute to heart attacks, strokes, and postmenopausal osteoporosis. Poor growth, confusion, disorientation, loss of appetite, depression, tetany with muscle contractions and cramps, tingling, numbness, abnormal heart rhythms, coronary spasm, abnormal nerve function with seizures or convulsions, hyperirritability, and even death can occur.

Signs of Excess. Magnesium toxicity (hypermagnesemia) is often associated with kidney failure, when the kidney loses the ability to remove excess magnesium. Mental status changes, nausea, osmotic diarrhea, appetite loss, muscle weakness, difficulty breathing, respiratory failure, extremely low blood pressure, and irregular heartbeat can occur. High doses of magnesium from laxatives can promote diarrhea, even with normal kidney function. The elderly are at risk of magnesium toxicity; kidney function declines with age and use of magnesium-containing laxatives and antacids is common.

Phosphorus: Phosphorus is second only to calcium in quantity in the human body. About 80% is in the skeleton and teeth as calcium phosphate; 20% is in extracellular fluid and cells. About 10% is bound to protein. *RDA for adult men and women is 700 mg/d. UL for adults varies from 3–4 g/d.*

Roles. Energy metabolism (ADP, ATP); fat, amino acid, and carbohydrate metabolism; calcium regulation; vitamin utilization; bones and teeth; osmotic pressure; DNA coding; buffer

salts; fatty acid transport; oxygen transport and release; leukocyte phagocytosis; and microbial resistance.

Sources. Protein-rich foods such as meat, poultry, fish, egg yolks, dried beans and nuts, whole grains, enriched breads and cereals, milk, cheese, and dairy products. Also found in peas, corn, chocolate, and seeds. Excessive intake of soft drinks increases phosphorus intake, often causing an unbalanced intake of calcium. About 70% of oral intake is absorbed. In the United States, milk and cheese products contribute the main sources of phosphorus for infants, toddlers, and adolescents; meat, poultry, and fish products contribute more to the diets of adults.

Signs of Deficiency. Hypophosphatemia leads to neuromuscular, renal and hematological changes, as well as rickets or osteomalacia. Deficiency is rare but may occur in those persons who take phosphate binders, persons receiving total parenteral nutrition without phosphate, and prematurity.

Signs of Excess. Hyperphosphatemia is especially problematic in renal failure. Nutritional secondary hyperparathyroidism may occur, with fragile bones and fractures.

Potassium: Potassium constitutes about 5% of total mineral content in the body. It is the main intracellular ion. *No specific RDA exists.*

Roles. Nerve conduction, muscle contraction, glycolysis, glycogen formation, protein synthesis and utilization, acid–base balance, cellular enzyme functioning, and water balance.

Sources. Fruits and vegetables, dried beans and peas, whole grains, and whole and skim milk. In the United States, milk and cheese products, meat, poultry, and fish products contribute the most; vegetables follow. Table A-9 provides a list of foods that contain over 500 mg potassium per serving.

Signs of Deficiency. Hypokalemia includes muscle weakness, cardiac arrhythmia, paralysis, bone fragility, decreased growth, weight loss, and even death.

Signs of Excess. Hyperkalemia promotes paralysis, muscular weakness, arrhythmias, heart disturbances, and even death.

Sodium: Sodium constitutes about 2% of total mineral content in the body. It is the main extracellular ion, along with chloride. *No specific RDA is set; 2300 mg/d is the UL for adults.*

Roles. Nerve stimulation, muscle contraction, acid–base balance, regulation of blood pressure, and glucose transport into cells. Sodium is the major extracellular fluid cation.

Sources. Milk, cheese, eggs, meat, fish, poultry, beets, carrots, celery, spinach, chard, seasoned salts, baking powder and soda, table salt (NaCl), many drugs and preservatives, some drinking water. High-sodium processed foods include: salty snack foods; ketchup and mustard; cured and processed meats; processed cheese; canned soups, vegetables, beans, and meats; soup, rice, and pasta mixes; soy sauce or hoisin sauce; monosodium glutamate (MSG); garlic salt, onion salt, celery salt, and seasoned salts. More than 95% of sodium from a mixed diet is absorbed.

TABLE A-9 Food Sources of Potassium[a]

Food, Standard Amount	Potassium (mg)	Calories
Sweet potato, baked, one potato (146 g)	694	131
Tomato paste, 1/4 cup	664	54
Beet greens, cooked, 1/2 cup	655	19
Potato, baked, flesh, one potato (156 g)	610	145
White beans, canned, 1/2 cup	595	153
Yogurt, plain, nonfat, 8-ounce container	579	127
Tomato puree, 1/2 cup	549	48
Clams, canned, 3 ounces	534	126
Yogurt, plain, low-fat, 8-ounce container	531	143
Prune juice, 3/4 cup	530	136
Carrot juice, 3/4 cup	517	71
Blackstrap molasses, one tbsp	498	47
Halibut, cooked, 3 ounces	490	119
Soybeans, green, cooked, 1/2 cup	485	127
Tuna, yellowfin, cooked, 3 ounces	484	118
Lima beans, cooked, 1/2 cup	484	104
Winter squash, cooked, 1/2 cup	448	40
Soybeans, mature, cooked, 1/2 cup	443	149
Rockfish, Pacific, cooked, 3 ounces	442	103
Cod, Pacific, cooked, 3 ounces	439	89
Banana, one medium	422	105
Spinach, cooked, 1/2 cup	419	21
Tomato juice, 3/4 cup	417	31
Tomato sauce, 1/2 cup	405	39
Peaches, dried, uncooked, 1/4 cup	398	96
Prunes, stewed, 1/2 cup	398	133
Milk, nonfat, one cup	382	83
Pork chop, center loin, cooked, 3 ounces	382	197
Apricots, dried, uncooked, 1/4 cup	378	78
Rainbow trout, farmed, cooked, 3 ounces	375	144
Pork loin, center rib (roasts), lean, roasted, 3 ounces	371	190
Buttermilk, cultured, low-fat, one cup	370	98
Cantaloupe, 1/4 medium	368	47
1–2% milk, one cup	366	102–122
Honeydew melon, 1/8 medium	365	58
Lentils, cooked, 1/2 cup	365	115
Plantains, cooked, 1/2 cup slices	358	90
Kidney beans, cooked, 1/2 cup	358	112
Orange juice, 3/4 cup	355	85
Split peas, cooked, 1/2 cup	355	116
Yogurt, plain, whole milk, 8-ounce container	352	138

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods.

[a]Food sources of potassium ranked by milligrams of potassium per standard amount, also showing calories in the standard amount. The AI for adults is 4700 mg/d potassium.

Signs of Deficiency. Hyponatremia occur from water intoxication with resulting anorexia, nausea, muscle atrophy, poor growth, weight loss, confusion, coma, and even death.

Signs of Excess. Hypernatremia may cause confusion, high blood pressure, calcium excretion from bones, heart failure, edema, and coma.

Sulfur: Sulfur exists as part of the amino acids methionine, cystine, and cysteine and as part of the antioxidant glutathione peroxidase and other organic molecules. *No specific RDA is set.*

Roles. Amino acids (methionine, cystine, cysteine), thiamin molecule, coenzyme A, biotin and pantothenic acid, connective tissue metabolism, penicillin, sulfa drugs, insulin molecule, heparin, and keratin of skin, hair, and nails.

Sources. Meat, poultry, fish, eggs, dried beans and legumes, *Brassica* family vegetables (broccoli, cabbage, cauliflower, Brussels sprouts), and wheat germ.

Signs of Deficiency. Not specific but likely to occur with hypoalbuminemia.

Signs of Excess. Uncommon because excess is excreted in the urine as sulfate, usually in combination with calcium. This may result in hypercalciuria (often after a high-protein meal).

Trace Minerals

Trace minerals are elements that are found in minute amounts in body tissues and are specific to the function of certain enzymes. They are typically not found in free ionic state but are bound to other proteins.

Copper: Copper is an antioxidant. Concentrations of copper are highest in the liver, brain, heart, and kidney; skeletal muscle also contains a large percentage because of total mass. About 90% of copper is bound as ceruloplasmin and is transported to other tissues mainly by albumin. Absorption occurs in the proximal jejunum. *RDA is 900 mg* daily for men and women. UL was set at 10 mg/d.

Roles: Skeletal development, immunity, formation of RBCs and leukopoiesis, phospholipid synthesis, electron transport, pigmentation, aortic elasticity, connective tissue formation, and CNS and myelin sheath structure. Part of metalloenzymes including cytochrome c-odixase.

Sources: Barley and whole grains, oysters and shellfish, nuts, dried beans and legumes, cocoa, eggs, prunes, and potatoes. Note: Milk is low in copper. Daily intake of copper in the United States is 2–5 mg; however, many persons have a lower intake than this because fresh foods are low in copper. Meat, poultry, and fish are primary sources in the United States. Approximately 30–60% of oral intake is absorbed, enhanced by acid and decreased by calcium. 94% of copper is tightly bound to ceruloplasmin.

Signs of Deficiency. Hypochromic anemia, cardiomyopathy, leukopenia, neutropenia, skeletal abnormalities and osteoporosis, decreased skin and hair pigmentation, Menke's steel hair (kinky-hair) syndrome, and reduced immune response. Deficiency is rare in adults, except with celiac disease, protein-losing enteropathies, and nephrotic syndrome. Requirement is increased by excessive zinc intake.

Signs of Excess. Excess is rare, but liver cirrhosis, biliary cirrhosis, and Wilson's disease may contribute to the retention of copper. Abnormalities in RBC formation, copper deposits in the brain, diarrhea, tremors, and liver damage occur. Eye rings are present in Wilson's disease. Excesses may decrease vitamin A absorption.

Fluoride: Fluoride is found in nearly all drinking waters and soils. The American Dietetic Association affirms that fluoride is an important element for all mineralized tissues and that appropriate consumption aids bone and tooth health (American Dietetic Association, 2005). Eighty to ninety percent of oral intake is absorbed. *RDA is 3–4 mg/d for men and women. UL is set at 10 mg/d.*

Roles.: Calcium uptake; some role in prevention of calcified aortas; resistance to dental caries, collapsed vertebrae, and osteoporosis; formation of hydroxyapatite; and enamel growth.

Sources. Fluoridated water, tea, mackerel, salmon with bones, infant foods to which bone meal has been added.

Signs of Deficiency. Dental caries, calcification of aorta, and anemia. Possibly bone thinning and osteoporosis.

Signs of Excess. Bony outgrowths at the spine. Tooth mottling, pitting, and discoloration (fluorosis) occur at doses of greater than 2–3 ppm in the drinking water. Excess can result in neurological problems; this feature is used in rat poison, for example.

Iron

Functional iron is found in hemoglobin (two-thirds), myoglobin (almost one-third), and enzymes. Storage iron is found in ferritin, hemosiderin, and transferrin. Iron is conserved and reused at a rate of 90% daily; the rest is excreted, mainly in bile. Dietary iron must be consumed to meet the 10% gap to prevent deficiency. To increase iron absorption, use sulfur amino acids and vitamin C (especially with nonheme foods); cook foods in an iron skillet; choose iron-enriched rice and do not rinse before cooking. Take iron separately from calcium supplements. Excessive calcium intake and oxalic, tannic, and phytic acids can reduce absorption. Avoid coffee and tea for 1 hour after eating; the tannic acid blocks iron absorption. Serum iron is largely bound to transferrin. About 5–15% is absorbed as ferrous iron; 15–25% of oral intake of heme iron is absorbed (meat, fish, and poultry); 2–20% of oral intake of nonheme iron is absorbed (legumes, grains, and fruit). *The RDA is 8 mg for men and postmenopausal women; 18 mg for premenopausal women. Pregnant women need 27 mg/d and breastfeeding women need 9 mg. Teenage girls (ages 14–18 years) need 15 mg iron per day (27 mg if pregnant; 10 mg if breastfeeding). Teenage boys (ages 14–18 years) need 11 mg iron per day The UL for iron is 45 mg/d.*

Roles. Responsible for carrying oxygen to cells through hemoglobin and myoglobin, skeletal muscle functioning, cognitive functioning, leukocyte functions and T-cell immunity, cellular enzymes, and cytochrome content for normal cellular respiration.

Sources: Beans, beef, dried fruit, enriched grains, fortified cereals, pork. In the United States, grain products provide the highest amount of dietary iron (see Table A-10).

Signs of Deficiency. Hypochromic and microcytic anemia, fatigue and weakness, pallor, pale conjunctiva, koilonychia (thin, spoon-shaped nails), impaired learning ability, cheilosis, glossitis, pica, tachycardia. Deficiency is defined as having an abnormal value for two of three laboratory tests of iron status (erythrocyte protoporphyrin, transferrin saturation, or serum ferritin). Iron deficiency anemia is defined as iron deficiency plus low hemoglobin. Iron deficiency anemia is the most common nutrient deficiency in the world, especially among toddlers, teenage girls, and women of childbearing age.

Signs of Excess. Iron deposits (hemosiderosis); vomiting or diarrheas with GI distress, hemochromatosis, drowsiness. Excess may occur from taking iron supplements daily or from consuming multiple sources, including transfusion overload with sickle cell anemia or thalassemia major.

Zinc: Zinc is distributed in the body with proteins such as albumin, transferrin, ceruloplasmin, and gamma-globulin.

TABLE A-10 Food Sources of Iron[a]

Food, Standard Amount	Heme (H) or Nonheme (N)	Iron (mg)	Calories
Clams, canned, drained, 3 ounces	H	23.8	126
Fortified ready-to-eat cereals (various), ~1 ounce	N	1.8–21.1	54–127
Oysters, eastern, wild, cooked, moist heat, 3 ounces	H	10.2	116
Organ meats (liver, giblets), various, cooked, 3 ounces[b]	N	5.2–9.9	134–235
Fortified instant cooked cereals (various), one packet	N	4.9–8.1	Varies
Soybeans, mature, cooked, 1/2 cup	N	4.4	149
Pumpkin and squash seed kernels, roasted, 1 ounce	N	4.2	148
White beans, canned, 1/2 cup	N	3.9	153
Blackstrap molasses, one tbsp	N	3.5	47
Lentils, cooked, 1/2 cup	N	3.3	115
Spinach, cooked from fresh, 1/2 cup	N	3.2	21
Beef, chuck, blade roast, lean, cooked, 3 ounces	H	3.1	215
Beef, bottom round, lean, 0 inch fat, all grades, cooked, 3 ounces	H	2.8	182
Kidney beans, cooked, 1/2 cup	N	2.6	112
Sardines, canned in oil, drained, 3 ounces	H	2.5	177
Beef, rib, lean, 1/4 inches fat, all grades, 3 ounces	H	2.4	195
Chickpeas, cooked, 1/2 cup	N	2.4	134
Duck, meat only, roasted, 3 ounces	H	2.3	171
Lamb, shoulder, arm, lean, 1/4 inches fat, choice, cooked, 3 ounces	H	2.3	237
Prune juice, 3/4 cup	N	2.3	136
Shrimp, canned, 3 ounces	H	2.3	102
Cowpeas, cooked, 1/2 cup	N	2.2	100
Ground beef, 15% fat, cooked, 3 ounces	H	2.2	212
Tomato puree, 1/2 cup	N	2.2	48
Lima beans, cooked, 1/2 cup	N	2.2	108
Soybeans, green, cooked, 1/2 cup	N	2.2	127
Navy beans, cooked, 1/2 cup	N	2.1	127
Refried beans, 1/2 cup	N	2.1	118
Beef, top sirloin, lean, 0 inch fat, all grades, cooked, 3 ounces	H	2.0	156
Tomato paste, 1/4 cup	N	2.0	54

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.

[a]Food Sources of iron ranked by milligrams of iron per standard amount; also calories in the standard amount. All are ≥ 10% of RDA for teen and adult females, which is 18 mg/d.

[b]High in cholesterol.

Animal sources are better utilized; vegetarian diets must be monitored for zinc deficiency. Ten to forty percent of zinc from meals is absorbed in the duodenum and the jejunum. Vitamin D can increase bioavailability. Phytates, excess copper, and fiber form complexes and reduce absorption, as do calcium and phosphate salts. Zinc absorption is affected by level of zinc in the diet and interfering substances, such as phytates, calcium, cadmium, folic acid, copper, or excessive fiber. Albumin is the major plasma carrier. *RDA is 11 mg for men and 8 mg for women. UL is 40 mg.*

Roles. ACTH-stimulated steroidogenesis in adrenals; sexual maturation; fatty acid, carbohydrate, protein, and nucleic acid metabolism; CO_2 transport; amino acid breakdown from peptides; oxidation of vitamin A; reproduction; growth; enzymes including alcohol dehydrogenase, alkaline phosphatase, and lactic acid dehydrogenase; wound healing; catalyst for hydrogenation; immunity; night vision; alcohol detoxification in the liver; heme synthesis; taste and smell acuity; synthesis of glutathione; and collagen precursors. Zinc supplementation has been shown to be effective in reducing morbidity and mortality from diarrhea, malaria, HIV infection, sickle cell anemia, renal disease, and GI disorders. Zinc gluconate lozenges do not always alleviate cold symptoms in children and adolescents but may be somewhat helpful.

Sources. Beans, seafood (e.g., lobster, shrimp, oysters), poultry, meat (red meat such as beef and liver especially), eggs, milk, peanuts, oatmeal, whole grains (e.g., whole wheat, rye bread, wheat germ), and yeast. The average American diet contains 10–15 mg/d. Meat, poultry, and fish are the primary sources of zinc.

Signs of Deficiency. Zinc is so prevalent in cellular metabolism that even minor impairment in supply is likely to have multiple biological and clinical effects. Deficiency reduces antibody responses and cell-mediated immunity and may cause dermatitis, skin lesions, growth failure (dwarfism), hypogonadism, decreased taste acuity (hypogeusia), alopecia, diarrhea, glossitis, stomatitis, and impaired wound healing. Zinc deficiency has a significant impact on development and on immune function; premature infants and children are at greatest risk. In children with zinc deficiency, severe growth depression is seen. Strict vegetarians, preschoolers who do not eat meat, adolescent females, and those on a chronically high-phytate diet may also be at risk, especially if other disease states are present.

Signs of Excess. Vomiting, diarrhea, low levels of serum copper, hyperamylasemia may result. Excessive zinc intake is probably self-limiting because of GI distress that occurs. Zinc toxicity can occur in renal dialysis if not carefully monitored.

Ultra-Trace Minerals

Boron: Boron can be found in the brain and the bone; it is also found in the spleen and thyroid. It is found in foods such as sodium borate; it is absorbed at a rate of 90%. *No RDA has been set, but a UL of 20 mg was established.*

Roles. Mineral metabolism in animals and humans, cell membrane functioning. It may function in a role similar to estrogen in bone metabolism and strengthening.

Sources. Drinking water, wine, cider and beer, noncitrus fruits, potatoes, peanuts, and legumes. Note: Protein foods and grains are low in boron. Infant foods, fruits and fruit juices, milk and cheese foods, and beverages provide the most.

Signs of Deficiency. None known at this time.

Signs of Excess. Reproductive and developmental effects are possible.

Chromium: Chromium is closely related to insulin action. Absorption ranges from 0.5 to 2%. Chromium needs transferrin for distribution. *RDA is 20 mg/d for women and 35 mg/d for men.* No UL has been set.

Roles. Insulin molecule (part of glucose tolerance factor known as chromodulin); fatty acid, triglyceride, and cholesterol metabolism; normal glucose metabolism; nucleic acid stability; regulation of gene expression; and peripheral nerve functioning.

Sources. Oysters, liver, potatoes, eggs, vegetable oil, brewer's yeast, whole grains and bran, shortening, nuts, and peanuts. Dairy products, fruits, and vegetables are low in chromium. Phytates and oxalates can decrease absorption.

Signs of Deficiency. Hyperglycemia refractory to insulin, weight loss, glucosuria, peripheral neuropathy, encephalopathy. Deficiency may be found in severe malnutrition, in diabetes, or in elderly patients with cardiovascular disease.

Signs of Excess. Dermatological allergy, nausea, vomiting, convulsions, irritation, gastrointestinal ulcers.

Cobalt: Most cobalt is in vitamin B_{12} stores in the liver. Cobalt may share intestinal transport with iron and is increased in patients with low iron intake and iron stores. *No specific RDA is set.*

Roles. Treatment of some anemias, part of structure of cobalamin in vitamin B_{12}, role in immunity, and healthy nerves and RBCs.

Sources. Seafood (such as oysters and clams), meats (such as liver), poultry, some grains, and cereals. Note: Cow's milk is very low. More than 50% of dietary cobalt is absorbed.

Signs of Deficiency. Weakness, anemia, and emaciation. Deficiency is usually in conjunction with vitamin B_{12} deficiency and low intake of protein foods. Lack of intrinsic factor, gastrectomy, or malabsorption syndromes may also cause deficiency.

Signs of Excess. Polycythemia, bone marrow hyperplasia, reticulocytosis, and increased blood volume may result.

Iodine: With the iodization of salt, iodine deficiency has been almost eliminated in the United States and Western nations. Fortification results in fewer goiters and less cretinism, stillbirths, spontaneous abortions, and mental or

growth retardation. Millions of people are at risk in other nations. The thyroid gland maintains 75% of the body's iodine; the rest is in the gastric mucosa and bloodstream. Iodide content of vegetables varies by the content of the local soil. Absorption is 50–100% from the gut. *RDA is 150 mg*/d for both men and women. UL is 1.1 mg/d.

Roles. Energy metabolism, proper thyroid functioning, normal growth and reproduction, prevention of goiter, and regulation of cellular metabolism and temperature. Iodine is found with T3 and T4 distribution.

Sources. Iodized salt, seaweed, and seafood (clams, oysters, sardines, lobster, and saltwater fish). Other lesser sources may include cream (in milk), eggs, drinking water in various areas, plant leaves (broccoli, spinach, and turnip greens), cranberries, and legumes. Iodized salt should be encouraged for pregnant women. Goitrogens in cabbage, turnips, rapeseeds, peanuts, cassava, and soybeans may block uptake of iodine by body cells; heating and cooking inactivate them.

Signs of Deficiency. Enlarged thyroid gland or related goiter; hypercholesterolemia; weight gain; cold intolerance; thinning hair; cognitive impairment; deafness; decreased metabolic rate; neuromuscular impairments. In infants, abnormal fetal growth, brain development, and cretinism can result.

Signs of Excess. Excess may depress thyroid activity and elevate thyroid-stimulating hormone (TSH) levels. High levels of thyroid hormone with goiter or myxedema can occur.

Manganese: Manganese affects reproductive capacity, pancreatic function, and carbohydrate metabolism. Less than 5% is absorbed from diet. It is transported bound to a macroglobulin, transferrin, or transmanganin. Manganese, cobalt, and iron compete for pathways. Human milk tends to be low in manganese levels. *An AI level has been set at 2.3 mg for men and 1.8 mg for women. The UL has been established at 11 mg.*

Roles. Polysaccharide and fatty acid metabolism, enzyme activation, tendon and skeletal development, possible role in hypertension, fertility and reproduction (role in squalene as a precursor of cholesterol and sex hormones), melanin and dopamine production, energy and glucose production.

Sources. Tea, coffee, whole grains, wheat germ and bran, blueberries, peas, beans and dried legumes, nuts, spinach, and cocoa powder. Sources of manganese are plant foods, not animal foods. In the United States, grain products are primary sources.

Signs of Deficiency. Nausea, vomiting, transient dermatitis, weight loss, ataxia, skeletal and cartilage abnormalities occur. Beware of excess calcium, phosphorus, iron, or magnesium supplementation.

Signs of Excess. Excesses accumulate in the liver and CNS. Parkinsonian tremors, difficulty in walking, facial muscle spasms, ataxia, hyperirritability, or hallucinations can occur.

Molybdenum: Molybdenum is important mostly for its role in xanthine oxidase. About 40–100% of intake is absorbed from the duodenum in protein-bound form. It is readily absorbed from the stomach and small intestine and excreted in the urine. *RDA is 45 mcg for both men and women. The UL is 2 mg.*

Roles. Flavoproteins; copper antagonist; component of sulfite oxidase, aldehyde oxidase, and xanthine oxidase; iron storage; energy metabolism; and degradation of cysteine and methionine through sulfite oxidase.

Sources. Legumes, whole-grain breads and cereals, dark green leafy vegetables, milk and dairy products, and organ meats. Milk and cheese products and grains provide the most from diet.

Signs of Deficiency. Tachycardia, tachypnea, visual and mental changes, elevated plasma methionine, low-serum uric acid, headache, lethargy, nausea, and vomiting. Long-term parenteral nutrition that is deficient in molybdenum is a concern.

Signs of Excess. Hyperuricemia and gout-like syndrome can occur. Excesses are rare.

Selenium: Cellular and plasma glutathione are the functional parameters for measuring selenium status. Selenium intake in the United States is generally very good; deficiency is rare. More than 50% dietary intake is absorbed (average range, 35–85%). It is transported via albumin from the duodenum. *RDA level for selenium was set at 55 mg*/d; *UL was set at 400 mg.*

Roles. Selenium functions within mammals primarily as selenoproteins. Glutathione catalyzes the reduction of peroxides that can cause cellular damage. Protein biosynthesis (selenomethionine and selenocysteine), sparing of vitamin E, protection against mercury toxicity; some role in thyroid function and immunity.

Sources. Seafood and fish, chicken, egg yolks, meats (especially kidney and liver), whole-grain breads and cereals, wheat germ, foods grown in selenium-rich soil including garlic, dairy products, Brazil nuts, and onions. Dietary selenium is found with protein in animal tissue (muscle meats, organ meats, and seafood). Grains and seeds have variable amounts dependent on the soil.

Signs of Deficiency. Muscle weakness and pain, carcinogenesis, and cardiomyopathy. Keshan's disease with cardiomyopathy is a selenium deficiency in China where soil levels are quite low. Kashin-Beck's disease occurs in preadolescents and adolescents; it has effects similar to osteoarthritis with stiffness and swelling of the elbows, knees, and ankles.

Signs of Excess. Selenosis causes garlicky odor on the breath and decaying of teeth. Nausea, vomiting, hair and nail loss, fatigue, headache, and chronic dermatitis can also occur.

Less-Studied Ultra-Trace Minerals

Aluminum, Arsenic, Cadmium, Lead, Lithium, and Tin: Not much is known about the roles, functions, or purpose in the human body of these minerals. For all age groups, grain products (mainly cornbread, pancakes, biscuits, muffins, and yellow cake) provide the highest amount of aluminum in the

diet. Organic arsenic is found in dairy products, meats, poultry, fish, grains and cereal. Inorganic arsenic is toxic.

Nickel: There may be roles in iron and zinc metabolism, hematopoiesis, DNA and RNA, and enzyme activation. Less than 10% of nickel is absorbed. It is transported by serum albumin. Nickel hypersensitivity can occur from prior dermal contact. *No RDA has been set, but UL was established at 1 mg/d.*

Sources. Nuts, legumes, cereals, sweeteners, chocolate candy, and chocolate milk powders.

Silicon: There may be roles in normal bone growth and calcification; collagen and connective tissue formation in the presence of calcium.

Natural food and water sources do not seem to cause adverse health effects. *No RDA or UL have been established.*

Sources. Most plant-based foods. Grains and beer are good sources.

Vanadium: Vanadium seems to have a role in thyroid metabolism. High doses may cause biochemical changes that precede cancer. *No RDA has been set. UL was established at 1.8 mg/d.*

Sources. Shellfish, whole grains, mushrooms, black pepper, parsley, and dill seed.

VITAMINS

Vitamins were first named "vital amines" in 1912 because they seemed to be important to life. Once it was known that they contain few amine groups, the "e" was dropped. There are 13 known vitamins (four fat-soluble and nine water-soluble vitamins). They are organic compounds, containing carbon structures. The best nutritional strategy for promoting optimal health and reducing the risk of chronic disease is to obtain adequate nutrients from a wide variety of foods. Eating more whole grains, fruits and vegetables is the recommendation, rather than taking supplements. Supplements are needed in special cases, such as in pregnancy and in some medical conditions. Supplements are most useful when taken as a multivitamin supplement that meets 100% of the daily recommended values. Although fat-soluble vitamins should not be consumed above UL doses, meeting 100% DRI levels is safe.

Fat-Soluble Vitamins

Vitamin A (Retinol, Retinal, Retinoic Acid): Vitamin A is best known for its role in vision and skin integrity. From 7–65% of vitamin A from the diet is absorbed in the mucosal cells of the small intestine. There, dietary retinyl esters are hydrolyzed by pancreatic triglyceride lipase (PTL), and the intestinal brush border phospholipase. Once in the cell, retinol is complexed with cellular retinol-binding protein type 2 (CRBP2), a substrate for reesterification of the retinol by the enzyme lecithin/retinol acyltransferase (LRAT). Retinol-binding protein (RBP) is used for transport. Retinyl esters are incorporated into chylomicrons. Stress can increase excretion; zinc or protein deficiency can decrease transport. Dietary vitamin A is transported via chylomicrons; 90% of vitamin A is stored in the liver. Its provitamins include beta-carotene and cryptoxanthin. *AI for infants is based on the amount of retinol found in human milk. RDA is 900 mg for men and 700 mg for women, adjusted for differences in average body size. UL is 3000 mg/d.*

Functions. Vision (especially night), gene regulation, growth, prevention of early miscarriage, immunity against infection (measles and many others), corticosterones, weight gain, proper bone, tooth, and nerve development, membrane functions, and epithelial tissue integrity in lungs and trachea especially. Vitamin A supplementation is used to treat some forms of cancer and degenerative retinitis pigmentosa.

Sources. See Table A-11 for sources.

TABLE A-11 Food Sources of Vitamin A[a]

Food, Standard Amount	Vitamin A (μg RAE)	Calories
Organ meats (liver, giblets), various, cooked, 3 ounces[b]	1490–9126	134–235
Carrot juice, 3/4 cup	1692	71
Sweet potato with peel, baked, one medium	1096	103
Pumpkin, canned, 1/2 cup	953	42
Carrots, cooked from fresh, 1/2 cup	671	27
Spinach, cooked from frozen, 1/2 cup	573	30
Collards, cooked from frozen, 1/2 cup	489	31
Kale, cooked from frozen, 1/2 cup	478	20
Mixed vegetables, canned, 1/2 cup	474	40
Turnip greens, cooked from frozen, 1/2 cup	441	24
Instant cooked cereals, fortified, prepared, one packet	285–376	75–97
Various ready-to-eat cereals, with added vitamin A, ~1 ounce	180–376	100–117
Carrot, raw, one small	301	20
Beet greens, cooked, 1/2 cup	276	19
Winter squash, cooked, 1/2 cup	268	38
Dandelion greens, cooked, 1/2 cup	260	18
Cantaloupe, raw, 1/4 medium melon	233	46
Mustard greens, cooked, 1/2 cup	221	11
Pickled herring, 3 ounces	219	222
Red sweet pepper, cooked, 1/2 cup	186	19
Chinese cabbage, cooked, 1/2 cup	180	10

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.
[a]Food Sources of vitamin A ranked by micrograms. Retinol Activity Equivalents (RAE) of vitamin A per standard amount; also calories in the standard amount. (All are ≥20% of RDA for adult men, which is 900 mg/d RAE).
[b]High in cholesterol.

Signs of Deficiency. Impaired vision is the first sign. Xerophthalmia, night blindness (nyctalopia), follicular hyperkeratosis or thickening of skin around hair follicles, drying of the whites of the eyes, eventual blindness, spots on the whites of the eyes, infections, and death.

Vulnerable Populations. Anorexia nervosa, burns, biliary obstruction, cancer, cirrhosis, celiac disease, cystic fibrosis, drug use (cholestyramine, mineral oil, and neomycin), hookworm, hepatitis of infectious origin, giardiasis, kwashiorkor, malaria, measles, pancreatic disease, pneumonia, pregnancy, prematurity, rheumatic fever, tropical sprue, and zinc deficiency.

Signs of Excess. Headache, anorexia, abdominal pain, blurred vision, muscle weakness, drowsiness, irritability, peeling of skin, peripheral neuritis, papilledema, serum vitamin A >100 μg/dL. Long-term supplementation with retinol (such as 25,000 IU/d) may have adverse effects on blood lipid levels and bone health. Vitamin A is known for its teratogenic affects; pregnant women should consider taking it in the form of beta-carotene. Caution should be given to supplement use in women of childbearing age because many women don't know they are pregnant in the earliest stages; supplements with retinol should be avoided during the first trimester.

Carotenoids: There are more than 500 natural carotenoids. Two beta-carotene molecules are equivalent to one molecule of vitamin A. The bioavailability of carotenoids from vegetables is low; fat is required for adequate absorption. Between 9 and 17% of dietary carotenes are absorbed. Dietary carotenoids may prevent some types of cancer through enhancement of immune response, inhibition of mutagenesis, and protection against oxidative damage to cells; alpha-carotenes, beta-cryptoxanthin, lutein, zeaxanthin, and lycopene contribute to these important functions. Persons at risk for developing lung cancer (i.e., current smokers and workers exposed to asbestos) should *not* take beta-carotene supplements. *No specific RDA recommendations were made for beta-carotene.*

Sources. Beta-carotene is found in deep yellow, orange, or dark green fruits and vegetables such as pumpkin, sweet potato, carrots, spinach, kale, turnip greens, cantaloupe, apricots, romaine lettuce, broccoli, papaya, mango, and tangerine.

Signs of Excess. Hypercarotenodermia involves yellowing of skin and the whites of eyes.

Vitamin D (Calcitriol, D_3): Vitamin D for humans is obtained from sun exposure, food and supplements. It is biologically inert and has to undergo two hydroxylation reactions to become active as Calcitriol (1,25-dihydroxycholecalciferol), or 1,25-dihydroxyvitamin D_3. Steps in metabolism include 7-dehydrocholesterol → previtamin D_3 → cholecalciferol (D_3) → 25-hydroxycholecalciferol → calcitriol (1,25-dihydroxycholecalciferol, the active form). Bile salts are required for absorption; 90% of dietary intake is absorbed. Dietary vitamin D is absorbed with lipid into the intestine by passive diffusion; it is synthesized in the skin from cholesterol that enters the capillary system.

Transport occurs via chylomicrons to the liver. Although five forms of vitamin D have been discovered, the two forms that matter are vitamins D_2 (ergocalciferol) and D_3 (cholecalciferol). There is decreased production from the skin with aging. *2.5 mcg of vitamin D is considered sufficient to prevent vitamin D-deficiency rickets, higher levels of 5* μg/d are recommended for infants and children throughout the period of skeletal development. The AI increases to 10 μg/d for adults aged 51 and older and to 15 μg/d for adults 71 years and older. The UL for infants is 25 μg/d and for children and adults, 50 μg/d.

Functions. The main functions of vitamin D are bone metabolism, calcium homeostasis, and expression of hundreds of genes. Calcitriol plays a key role in the maintenance of many organ systems; the volume and acidity of gastric secretions; growth of soft tissues including skin and pancreas; bone calcification, growth and repair; tooth formation; parathormone (PTH) management; and renal/intestinal phosphate absorption.

Sources. Brief and casual exposure (10–20 minutes daily) to natural sunlight can be encouraged. Check nutrition labels for vitamin D fortification when using soy or rice milks. Food sources of vitamin D are listed in Table A-12.

TABLE A-12 Food Sources of Vitamin D

Foods with Naturally Occurring Vitamin D	International Units
Herring, 3 ounces	1384
Cod liver oil, one tablespoon	1350
Halibut, 3 ounces	510
Catfish, 3 ounces	425
Salmon, canned, 3 ounces	390
Mackerel, 3 ounces	306
Sardines, canned in oil, 1.75 ounces	250
Tuna, canned in oil, 3 ounces	200
Egg yolk, one yolk	20
Beef liver, 3 ounces	15
Swiss cheese, 1 ounce	12
Vitamin D–Fortified Foods	
Soy milk, one cup	119
Milk, one cup	100
Fortified orange juice, one cup	100
Fortified breakfast cereal, one cup	20–100
Fortified yogurt, one cup	80
Fortified margarine, one tablespoon	60

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.

Signs of Deficiency. Hypocalcemic tetany is a low calcium condition in which the patient has overactive neurological reflexes, spasms of the hands and feet, cramps and spasms of the voice box (larynx). Vitamin D from sunlight exposure is low during the winter months. Hypovitaminosis D is related to lowered vitamin D intake, less exposure to ultraviolet light (UVB), anticonvulsant use, renal dialysis, nephrotic syndrome, hypertension, diabetes, winter season, and high PTH and alkaline phosphatase levels. Hypovitaminosis D is common in general medical in-patients. Bowed legs, rickets in children, and osteomalacia in adults may occur.

Vulnerable Populations. The prevalence of vitamin D deficiency is high. The National Health and Nutrition Examination Survey (NHANES) team found that 9% of U. S. children were vitamin D deficient (<15 ng/mL of blood) and another 61% were vitamin D insufficient (15–29 ng/mL). Those individuals with dark skin or who wear protective clothing may also not absorb sufficient vitamin D from sun exposure alone. Living near the North or South Pole in winter is a risk for deficiency. Biliary obstruction, celiac disease, cystic fibrosis, drug use (bile salt binders, glucocorticoids, phenobarbital, primidone, and mineral oil), end-organ failure, Fanconi's disease, hepatic disease, hypertension, primary hypophosphatemia, hypoparathyroidism, inflammatory bowel disease, intestinal malabsorption, lymphatic obstruction, multiple sclerosis, nephrotic syndrome, neurological and psychiatric conditions such as depression and bipolar disorders, pancreatitis, parathyroid surgery, postmenopausal status, prematurity, renal disease and dialysis, small bowel resection, and tropical sprue. Pregnant women should enjoy a few minutes in the sun each day, if possible. Low levels may increase the risk of breast, colon, and prostate cancers; depression, poor brain function, severe dementia in older adults; tuberculosis; pneumonia; bacterial infections and gum disease; autoimmune diseases, such as multiple sclerosis and type 1 diabetes.

Signs of Excess. Patients often complain of headache and nausea, and infants given excessive amounts of vitamin D may have GI upset, bone fragility, and retarded growth. Stupor, confusion, anorexia, nausea, vomiting, constipation, weakness, hypertension, renal stones, polyuria, polydipsia, cardiac arrhythmias, itchy skin, increased bone density on x-ray, calcium deposits throughout the body, increased serum calcium, increased calcium in the urine, elevated serum 25-hydroxycholecalciferol or elevated serum 1,25-dihydroxycholecalciferol levels. In the absence of sufficient vitamin K, excessive vitamin D can cause soft tissue calcification.

Vitamin E (Tocopherol, Tocotrienols): Tocotrienols plus other forms affect cholesterol metabolism, carotid arteries, and immunity against cancer. The natural form is D-alpha-tocopherol (need 22 IU). The DL-alpha-tocopherol form is synthetic and less effective. Vitamin E is absorbed in the upper small intestine by micelle-dependent diffusion. Overall, 20–40% is absorbed with meals; bile and pancreatic secretions are needed and VLDLs and LDLs carry it to tissues. An IU of vitamin E is equal to 0.67 mg of RRR-alpha-tocopherol and 1 mg of all-rac-alpha-tocopherol. *RDA is 15 mg for adults. UL is 1000 mg.*

Functions: The main function of vitamin E is as a lipid-soluble membrane antioxidant, along with vitamin C and selenium. It protects unsaturated phospholipids of the membrane from oxidative degradation through free-radical scavenging. Other roles include anticoagulant and vitamin K antagonist; intracellular respiration; hemopoietic agent; roles in muscular, vascular, reproductive, and CNS systems; some role in reproduction; neutralizes free radicals; protects against cataracts; may relieve discomforts of rheumatoid arthritis; protects against effects of the sun, smog, and lung disease; protects brain cell membranes.

Sources. Tocopherols and tocotrienols are synthesized only by plants so plant products, especially oils, are the best sources of Vitamin E.

Requirements increase with use of PUFAs; if normal needs are 15 mg/d, normal requirements double daily with high PUFA intakes. Gamma-tocopherol is more common in the U. S. food supply (as from soybean oil); it is less useful to the body (see Table A-13).

Signs of Deficiency. Symptoms of deficiency in humans are rare, but can manifest clinically as loss of deep tendon reflexes, impaired vibratory and position sensation, changes in balance and coordination, muscle weakness and visual disturbances. Other targets of deficiency include the vascular and reproductive systems. Rupture of RBCs, impaired vitamin A storage, and prolonged blood coagulation may also occur. For supplementation, pharmacists evaluate equivalent vitamin E dosing as follows:

DL-alpha-tocopheryl acetate	1 mg	1 IU
DL-alpha-tocopherol	0.91 mg	1.1 IU
DL-alpha-tocopheryl acid succinate	1.12 mg	0.89 IU
D-alpha-tocopheryl acid succinate	0.826 mg	1.21 IU

Vulnerable Populations. Alzheimer's disease, arthritis, biliary cirrhosis, bronchopulmonary dysplasia, cardiovascular diseases, cystic fibrosis, drug use (cholestyramine, clofibrate, oral contraceptives, and triiodothyronine), high intake of PUFA in diet, malabsorption syndromes, malnutrition, musculoskeletal disorders, pancreatic diseases, pregnancy, prematurity, pulmonary diseases, and steatorrhea.

Signs of Excess. Vitamin E is one of the least toxic vitamins, but at high doses, it can antagonize the utilization of other fat-soluble vitamins. Excessive intake has caused isolated cases of dermatitis, fatigue, pruritus ani, acne, vasodilation, hypoglycemia, GI symptoms, increased requirement for vitamin K, impaired coagulation, and muscle damage.

Vitamin K: First isolated from alfalfa, the forms of vitamin K are **phylloquinone (K_1)** in green plants; **menaquinone (K_2)** from human bacterial synthesis and **menadione (K_3)** in pharmaceutical form. Vitamin K absorption is optimal with bile and pancreatic juice; 10–70% of dietary intake is usually absorbed. The phylloquinones are absorbed by and energy-dependant process in the small intestine; the menaquinones and menadione are absorbed in the small intestine and colon by passive diffusion. Because intestinal bacteria make about 50% of the bodily requirement, a sterile gut or malabsorption can create deficiency. Vitamin E excesses can reduce absorption of vitamin K. Warfarin (Coumadin) blocks regeneration of active, reduced vitamin K, thus prolonging clotting time,

TABLE A-13 Food Sources of Vitamin E[a]

Food, Standard Amount	AT (mg)	Calories
Fortified ready-to-eat cereals, ~1 ounce	1.6–12.8	90–107
Sunflower seeds, dry roasted, 1 ounce	7.4	165
Almonds, 1 ounce	7.3	164
Sunflower oil, high linoleic, one tbsp	5.6	120
Cottonseed oil, one tbsp	4.8	120
Safflower oil, high oleic, one tbsp	4.6	120
Hazelnuts (filberts), 1 ounce	4.3	178
Mixed nuts, dry roasted, 1 ounce	3.1	168
Turnip greens, frozen, cooked, 1/2 cup	2.9	24
Tomato paste, 1/4 cup	2.8	54
Pine nuts, 1 ounce	2.6	191
Peanut butter, two tbsp	2.5	192
Tomato puree, 1/2 cup	2.5	48
Tomato sauce, 1/2 cup	2.5	39
Canola oil, one tbsp	2.4	124
Wheat germ, toasted, plain, two tbsp	2.3	54
Peanuts, 1 ounce	2.2	166
Avocado, raw, 1/2 avocado	2.1	161
Carrot juice, canned, 3/4 cup	2.1	71
Peanut oil, one tbsp	2.1	119
Corn oil, one tbsp	1.9	120
Olive oil, one tbsp	1.9	119
Spinach, cooked, 1/2 cup	1.9	21
Dandelion greens, cooked, 1/2 cup	1.8	18
Sardine, Atlantic, in oil, drained, 3 ounces	1.7	177
Blue crab, cooked/canned, 3 ounces	1.6	84
Brazil nuts, 1 ounce	1.6	186
Herring, Atlantic, pickled, 3 ounces	1.5	222

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.
[a]Food sources of vitamin E ranked by milligrams of vitamin E per standard amount; also calories in the standard amount. (All provide ≥10% of RDA for vitamin E for adults, which is 15 mg a-tocopherol [AT]/d).

which is best monitored through international normalized ratio (INR) where the timing level of 2–3 is desired. *AI is 120 mg for men and 90 mg for women. No UL has been established, but data are limited and one should not assume that high vitamin K consumption is harmless.*

Functions. Vitamin K is important for normal blood clotting, calcium metabolism, and bone mineralization. Low vitamin K intakes are associated with an increased incidence of hip fractures. Genetic loss of less critical vitamin K–dependent proteins, dietary vitamin K inadequacy, human polymorphisms or mutations, and vitamin K deficiency induced by chronic anticoagulant (warfarin/Coumadin) therapy are all linked to age-associated conditions: bone fragility after estrogen loss (osteocalcin) and arterial calcification linked to cardiovascular disease (McCann and Ames, 2009). Vitamin K is also linked with sphingolipid metabolism in the brain.

Sources. Plant foods such as green leafy vegetables are better sources of vitamin K than animal foods. Fish, liver, meat, eggs, cereal, and some fruits contain smaller amounts (see Table A-14).

Signs of Deficiency. Hemorrhage with prolonged clotting time is the key sign of deficiency, which might even lead to a fatal anemia. Vitamin K deficiency can be found in lipid malabsorption, chronic antibiotic therapy, and liver disease where

TABLE A-14 Food Sources of Vitamin K

Food and Serving Size	Vitamin K (μg)
Kale, cooked 1/2 cup	531
Spinach, cooked 1/2 cup	444
Collard greens, cooked 1/2 cup	418
Beet greens, cooked 1/2 cup	348
Swiss chard, cooked 1/2 cup	286
Turnip greens, cooked, 1/2 cup	265
Mustard greens, cooked 1/2 cup	210
Spinach, raw one cup	145
Broccoli 1/2 cup	110 cooked, 45 raw
Brussels sprouts, cooked 1/2 cup	109
Scallions (including bulb and green tops), raw 1/2 cup	104
Cabbage, cooked 1/2 cup	81
Lettuce (green leaf), raw one cup	97
Prunes, stewed one cup	65
Okra, cooked one cup	64
Parsley one tablespoon	62
Cucumber, raw one large	49
Asparagus, cooked four spears	48
Tuna, canned 3 ounces	37
Celery, raw one cup	35
Black-eyed peas, cooked 1/2 cup	31
Kiwi one medium	30
Blackberries or blueberries, raw one cup	29
Artichoke, cooked one cup	25
Peas 1/2 cup	24
Grapes (red or green) one cup	23
Strawberries, sliced one cup	23
Green beans, cooked 1/2 cup	20
Soy beans or mung beans, cooked 1/2 cup	16–17
Oil, canola, or olive one tablespoon	8–10

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.

bleeding and hypoprothrombinemia can result. Giving an injection of vitamin K upon birth prevents hemorrhagic disease of newborns. In addition, there are 16 known vitamin K–dependent (VKD) proteins; five of them have critical functions with preferential distribution of dietary vitamin K1 to the liver to preserve coagulation function when vitamin K1 is limiting (McCann and Ames, 2009).

Vulnerable Populations. Individuals who practice pica or have calcium disorders, use certain medications (anticoagulants, cholestyramine, mineral oil, phenytoin, neomycin or other antibiotics, or large doses of aspirin), hepatic biliary obstruction, hepatocellular disease, malabsorption syndromes, postmenopausal women at risk for hip fractures, prematurity, and small bowel disorders. Individuals who take large doses of vitamin A or E may also acquire a vitamin K deficiency.

Signs of Excess. Prolonged bleeding time. Menadione can be toxic if given in excessive dose; severe jaundice in infants or hemolytic anemia may result. Seek consistency in vitamin K intake while using warfarin; aim for 120 μg/d for men and 90 μg/d for women. Although dietary sources do not appear to be dangerous, excessive menadione can be toxic and may cause hemolytic anemia.

REFERENCE

McCann JC, Ames B. Vitamin K, an example of triage theory: is micronutrient inadequacy linked to diseases of aging? *Am J Clin Nutr.* 90:889, 2009.

Water-Soluble Vitamins

Thiamin (Vitamin B_1): Known as the "morale" vitamin, thiamin is beneficial for nerve and heart function. Thiamin is absorbed by the proximal small intestine by active transport in low doses and passive diffusion in high doses. High-CHO intakes, pregnancy, lactation, increased basal metabolic rate, and antibiotic use will all increase needs. As energy intake from protein and fat increases, thiamin requirement decreases. The extent of absorption varies widely. Thiamin hydrochloride is the common supplemental form. The DRIs for thiamin include AIs for infants as found in human milk; RDAs are based on levels of energy intake. *RDA is 1.2 mg/d for men and 1.1 mg/d for women. No UL was established.*

Functions. Thiamin is mainly a coenzyme for decarboxylations of 2-keto acids and transketolations. It prevents beriberi and has a role in cell respiration, RNA and DNA formation, protein catabolism, growth, appetite, normal muscle tone in cardiac and digestive tissues, neurologic functioning. It is a coenzyme in the energy-producing Krebs' cycle, with thiamin pyrophosphate (TPP) at the pyruvic acid step. Magnesium, manganese, riboflavin, and vitamin B_6 are synergists. Thiamin is essential for conversions derived from the amino acids methionine, threonine, leucine, isoleucine, and valine. Acetylcholine synthesis also requires thiamin.

Sources. Liver, fortified cereals, dried legumes such a split peas, brown rice, organ and lean meats, whole grains such as oats and whole wheat, nuts, cornmeal, enriched flour or bread, dried milk, wheat germ, dried egg yolk or whole egg, green peas, and seeds. Note: Two slices of bread or one slice of bread and one serving of cereal will provide 15% of the daily RDA. Some nutrients are thiamin sparing; others destroy the nutrient. Thiamin is spared by fat, protein, sorbitol, and vitamin C; antagonists include raw fish, tea, coffee, blueberries, and red cabbage. Avoid cooking with excessive water and alkaline products such as baking soda; thiamin is lost readily.

Signs of Deficiency. Anorexia, calf muscle weakness, weight loss, cardiac and neurologic signs. Eventually, beriberi occurs with mental confusion, muscular wasting, edema, peripheral neuropathy, and tachycardia. Energy deprivation and inactivity are causes of dry (nonedematous) beriberi. Wernicke's encephalopathy is due to thiamin deficiency and often associated with malnutrition and alcoholism. TPN without multivitamin use can lead to symptoms of Wernicke's encephalopathy.

Vulnerable Populations. Individuals who have alcoholism, cancers, cardiomyopathies, CHO (high intakes), celiac disease, children with congenital heart disease before and after surgery, congestive heart failure, fever, high parenteral glucose loading, lactation and pregnancy, tropical sprue, and thyrotoxicosis are at risk.

Signs of Excess. Respiratory failure and death with large doses (1000 times nutritional needs). With 100 times the normal dose, headache, convulsions, muscular weakness, cardiac arrhythmia, and allergic reactions have been noted. Massive doses greater than 1000 times the estimated needs suppress the respiratory center and lead to death.

Riboflavin (Vitamin B_2): Riboflavin is important in CHO metabolism and maintenance of healthy mucous membranes.

Riboflavin is absorbed in the free form by a carrier-mediated process in the proximal small intestine. The DRIs for riboflavin include AIs for infants and RDAs based on the amount required to maintain normal tissue reserves based on urinary excretion, RBC riboflavin contents, and erythrocyte glutathione reductase activity. *RDA is 1.3 mg/d for men and 1.1 mg/d for women.* Requirements are higher during pregnancy and lactation. *No UL was established.*

Functions. It is the main coenzyme in redox reactions of fatty acids and the tricarboxylic acid (TCA) cycle. Cell respiration, oxidation reduction, conversion of tryptophan to niacin, component of retinal pigment, involvement in all metabolisms (especially fat), purine degradation, adrenocortical function, coenzyme in electron transport as flavin adenine dinucleotide (FAD) and flavin mononucleotide (FMN), healthy mucous membranes, skin and eyes, growth, and proper functioning of niacin and pyridoxine.

Sources. Milk, yogurt, cheese, egg whites, liver, beef, chicken, fish, legumes, peanuts, enriched grains and fortified cereals. Body size, growth, activity excesses, and fat metabolism affect daily requirements. Avoid excesses of niacin and methylxanthines. Light destroys riboflavin; buy milk in opaque cartons. As protein intake increases, the need for riboflavin decreases. Riboflavin is spared by dextrins and starch and is found in greater amounts in protein foods. Cheilosis causes a magenta-colored tongue.

Signs of Deficiency. Deficiencies are first evident in the tissues that have rapid cellular turnover such as the skin and epithelia. Seborrheic dermatitis of the nasolabial folds, ears, and/or eyelids; itchy and burning eyes or lips, mouth, and tongue; cheilosis (fissures and scaling of lips); angular stomatitis; glossitis with purplish-red, swollen tongue; peripheral neuropathy; dyssebacea (sharkskin); corneal vascularization; photophobia; lacrimation; normochromic normocytic anemia with erythroid hypoplasia; pancytopenia due to generalized marrow hypoplasia. Riboflavin deficiency is most commonly found in developing countries like India and can lead to deficiency of vitamins B_6 and B_2 and impairment of psychomotor function. Riboflavin metabolism is affected by infections, drugs, and hormones.

Vulnerable Populations. Alcoholism, cancers, chronic infections, drug use (broad-spectrum antibiotics and chloramphenicol), gastrectomy, and low oral intake during childhood, pregnancy, or lactation.

Signs of Excess. There is no known toxicity.

Niacin (Nicotinic Acid, Nicotinamide): Niacin is absorbed in the stomach and small intestine by carrier-mediated facilitated diffusion. Niacin requirements are related to protein and calorie intake. Intake for infants is established as AIs. *RDA is 16 mg/d for men and 14 mg/d for women. UL was established at 35 mg for men and women.*

Functions. Niacin serves as coenzyme for several dehydrogenases. It is part of nicotinamide adenine dinucleotide (NAD) and NADH in over 200 enzymes involved with metabolism of CHO, protein, and fat. It is involved with intracellular reactions and biosynthetic pathways; energy metabolism; growth; conversion of vitamin A to retinol; metabolism of fatty acids, serum cholesterol, and triglycerides. It has a role in DNA repair and gene stability, thus affecting cancer risk. It prevents pellagra (along with other B-complex vitamins). It is needed to treat tuberculosis as Isoniazid. Nicotinic acid is used as a vasodilator; nicotinamide is less vasodilating.

Sources. Beef and lean meats, organ meats, poultry, tuna and salt water fish, peanut butter, peanuts and legumes, enriched breads and fortified cereals. Diet supplies 31% of niacin intake as tryptophan. Milk and eggs are good sources of tryptophan but not niacin. Sixty milligrams of tryptophan are needed to equal 1 mg of niacin.

Signs of Deficiency. Signs start with beefy tongue, stomatitis with swelling and soreness of the oral mucosa; esophagitis with gastritis; diarrhea with proctitis; inflammation and erythema of the mucosal surfaces of the genitourinary tract; symmetric pigmented rash with scaling and hyperkeratosis; worsening of the skin rash in areas of sun exposure or trauma; headaches, insomnia, depression, anxiety; tremors or rigidity of limbs; loss of tendon reflexes; numbness and paresthesias in limbs. Severe deficiency leads to **pellagra** with Casal's collar, a rough, red **d**ermatitis, **d**iarrhea, **d**ementia (the three Ds of pellagra).

Vulnerable Populations. Alcoholism, cancers, chronic diarrhea, cirrhosis, diabetes, and tuberculosis. Undernourished people in Africa.

Signs of Excess. Toxicity is generally low but may cause histamine release, flushing of skin (when using 1–2 g of nicotinic acid daily in an effort to lower cholesterol levels), or even liver toxicity. Megadoses should be avoided.

Pantothenic Acid (Vitamin B-5): Pantothenic acid is a coenzyme in fatty acid metabolism. Pantothenic acid is available "from everywhere." It is absorbed by passive diffusion and active transport in the jejunum. Fifty percent is bioavailable from diet. Needs increase by one-third in pregnancy and lactation. *AI for adult men and women is 5 mg/d. UL is not established.*

Functions. Coenzyme A is essential in the formation of acetyl CoA and energy production from the macronutrients. It also affects synthesis of cholesterol and fatty acids, adrenal gland activity, acetyl transfer, antibodies, normal serum glucose, electrolyte control and hydration, heme synthesis, and healthy RBCs. It changes choline to acetylcholine and prevents premature graying in some animals.

Sources. Pantothenic acid is present in all plant and animal tissues. The most important sources are meats, particularly heart and liver, but mushrooms, avocados, broccoli, egg yolks, yeast, skim milk, and sweet potatoes are also good sources.

Signs of Deficiency. Deficiency is rare but can impair lipid synthesis and energy production. Symptoms include paresthesia in the toes and soles of the feet, burning sensations in the feet, depression, fatigue, insomnia, and weakness.

Vulnerable Populations. Alcoholism, elderly women, liver disease, chronic ulcerative colitis, and pregnancy.

Signs of Excess. Rarely, excess causes mild intestinal distress or diarrhea.

Vitamin B_6 (Pyridoxine): This group includes numerous 2-methyl-3,5-dihydroxymethylpyridine derivatives exhibiting the biologic activity of PN, including pyridoxol, pyridoxal, and pyridoxamine. Vitamin B_6 is absorbed by passive diffusion of the dephosphorylated forms PN, PL, or PM, primarily in the jejunum and ileum. Pyridoxal phosphate (PLP) is the active form. Some studies suggest that a higher RDA is desirable for some individuals. About 96% of dietary vitamin B_6 is absorbed. High-protein intakes may deplete vitamin B_6 levels, thus needs increase with increased protein intake. Needs decrease with fatty acids or with other B-complex vitamins. The DRIs for vitamin B_6 include AIs for infants. Infants need three times as much vitamin B_6 as adults. *RDA is 1.3–1.7 mg/d for men and 1.3–1.5 mg/d for women. The UL was established at 100 mg/d for adult men and women.*

Functions. Vitamin B_6 is primarily known for its role in amino acid metabolism, histamine synthesis, gene expression, and hemoglobin synthesis. PLP is involved in practically all reactions in the metabolism of amino acids and in several aspects of the metabolism of neurotransmitters, glycogen, sphingolipids, heme, steroids, coenzymes (-ases), conversion of tryptophan to niacin, fat metabolism (changing linoleic to arachidonic acid), synthesis of folic acid, homocysteine metabolism, glandular and endocrine functions, antibodies, dopamine and serotonin metabolism, glycogen phosphorylase, and immunity.

Sources. Wheat and whole-grain cereals (such as oatmeal), legumes and nuts (garbanzo beans, soybeans, and peanuts) are the highest sources.

Signs of Deficiency. The vitamin is widely distributed throughout the diet; deficiency is rare but may present as convulsions in infants; depression, confusion, irritability; hypochromic, microcytic anemia. Peripheral neuropathies; glossitis, cheilosis, angular stomatitis; impaired immunity; seborrheic dermatitis involving the face and neck; blepharitis may also occur.

Vulnerable Populations. Alcoholism, use of certain medications (cycloserine, Dilantin, hydralazine, isoniazid, oral contraceptives, and penicillamine), elderly status, pregnancy, schizophrenia, isoniazid treatment and no vitamin replacement for tuberculosis.

Signs of Excess. Toxicity is relatively low. Sensory neuropathy with gait changes, peripheral sensations, and muscle incoordination.

Biotin (Vitamin B-7): Biotin is a coenzyme for carboxylations. *RDA is 30 mg/d for adult men and women. UL is not established.*

Functions. Coenzyme in CO_2 fixation, deamination, decarboxylation, synthesis of fatty acids, CHO metabolism, oxidative phosphorylation, leucine catabolism, and carboxylation of pyruvic acid to oxaloacetate.

Sources. Liver, kidney, pork, milk, egg yolk, yeast, cereal, nuts, legumes, and chocolate. Note: Biotin can be synthesized by intestinal bacteria. Be wary of extended antibiotic use or prolonged unsupplemented TPN use. Probably, 50% of dietary biotin is absorbed from the small intestine. Biotin is called the "anti–raw egg white" factor; avidin in raw egg white decreases biotin availability.

Signs of Deficiency. Inflammation of the skin and lips. Other symptoms include dermatitis, alopecia, paralysis, depression, nausea, hepatic steatosis, hypercholesterolemia, and glossitis.

Vulnerable Populations. Individuals who consume excessive intake of avidin from raw egg whites. People with genetic conditions (beta-methylcrotonylglycinuria and propionic acidemia), or inadequate provision with long-term parenteral nutrition.

Signs of Excess. There are no known toxic effects.

Folic Acid (Vitamin B-9): Folate generally refers to pteroylmonoglutamin acid and its derived compounds. Folate is found naturally in food; folic acid is the supplemental form. Pteroylglutamic acid is the pharmacological form. Folic acid works primarily as a coenzyme in single-carbon metabolism. Some folic acid can be made in the intestines with help from biotin, protein, and vitamin C. Synthetic folic acid increases serum levels more effectively than food sources of folate. Only 25–50% of dietary sources are bioavailable. Absorption occurs by active transport mainly in the jejunum, but can also be absorbed by passive diffusion when ingested in large amounts.

The DRIs express folate in "dietary folate equivalents or DFEs," which account for the difference between food sources and the more bioavailable supplemental sources. DFEs from fortified foods provide 1.7 times the micrograms of natural folic acid. Fortification of more commonly eaten foods has been implemented to provide adequate folic acid for vulnerable populations. About 90% of circulating folacin is bound to albumin. Drugs that interfere with utilization include aspirin, sulfasalazine (Azulfidine), methotrexate, antacids, anticonvulsants such as phenytoin (Dilantin), oral contraceptives, pyrimethamine, trimethoprim. *The DRI is described as AIs for infants. RDA is 400 mcg for adults. Pregnant women need 600 mcg and lactating women need 500 mcg. UL is 1000 mcg/d.*

Functions. Needed for growth; hemoglobin; amino acid metabolism. Prevents excessive buildup of homocysteine to protect against heart disease and some forms of cancer. Prevents megaloblastic and macrocytic anemias. Reduces the incidence of neural tube defects. Folate is required for synthesis of phospholipids, DNA, proteins, new cells, and neurotransmitters. Choline is used as a methyl donor (to convert homocysteine to methionine) when folate intake is low. Folic acid is needed during pregnancy to prevent spina bifida, cleft palate, some heart or other birth defects.

Sources. Foods with highest folate content include fortified cereals, pinto and navy beans (cooked), lentils, beets, asparagus, spinach, romaine lettuce, broccoli, and oranges. Analysis of foods for their folate content is complex and difficult. Folate exists in 150 different forms, and losses of 50–90% typically occur during storage, cooking, or processing at high temperatures (see Table A-15).

Signs of Deficiency. Decrease in total number of cells (pancytopenia) and large RBCs with a macrocytic or megaloblastic anemia. Deficiency is common, especially during pregnancy, with oral contraceptive use, in malabsorption syndromes, in alcoholics, in teens, and in elderly individuals. Neural tube defects such as spina bifida or anencephaly may result from impaired biosynthesis of DNA and RNA.

Vulnerable Populations. Alcoholics, cancer, medication use (aspirin, cycloserine, Dilantin, methotrexate, oral contraceptives, primidone, and pyrimethamine), hematological diseases (pernicious anemia, sickle cell anemia, and thalassemia), vitamin B_{12} deficiency, malabsorption syndromes, and pregnancy.

Signs of Excess. No adverse effects of high oral doses of folate have been reported. Although parenteral administration of amounts some 1000 times the dietary requirement produce epileptiform seizures in the rat.

Vitamin B_{12} (Cobalamin, Cyanocobalamin): Vitamin B_{12} is known for its role as a coenzyme in metabolism of propionate, amino acids, and single-carbon fragments. It is known as a blood cell and nerve cell growth stimulator, along with folic acid. It is the "extrinsic factor" needed from diet to complement the "intrinsic factor" found in the stomach. Many people with achlorhydria or over age 50 lose the ability to absorb vitamin B_{12} from foods and should consider using more fortified foods. *RDA is 2.4 mcg for adults. Pregnant women need 2.6* μg and lactating women need 2.8 μg. No UL was established.

TABLE A-15 Food Sources of Folic Acid

Food	Folic Acid or Folate (μg)
Cereal, ready to eat, one cup	100–400
Cereal, cooked (oatmeal, farina, grits), one cup	75–300
Turkey giblets, cooked, one cup	486
Lentils or black-eyed peas, cooked, one cup	358
Pinto beans, cooked, one cup	294
Chickpeas (garbanzo beans), one cup	282
Okra, frozen, cooked, one cup	269
Spinach, cooked, one cup	263
Black beans, cooked, one cup	256
Enriched long-grain white rice, cooked, one cup	238
Beef liver, cooked, 3 ounces	221
Enriched egg noodles, cooked, one cup	221
Spinach, canned, one cup	210
Collards, cooked, one cup	177
Turnip greens, cooked, one cup	170
Broccoli, cooked, one cup	168
Enriched spaghetti, cooked, one cup	167
Brussels sprouts, cooked, one cup	157
Artichokes, cooked, one cup	150
Beets, cooked, one cup	136
Peas, cooked, one cup	127
Papaya, one whole	116
Cream-style corn, canned, one cup	115
Orange juice, one cup	110

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.

Functions. Coenzymes, blood cell formation, nucleoproteins and genetic material, nutrient metabolism, growth, nerve tissue, thyroid functions, metabolism, transmethylation, myelin formation, homocysteine metabolism and prevention of heart disease.

Sources. Vitamin B_{12} is not found in plant foods; monitor vegetarian diets. For best absorption, riboflavin, niacin, magnesium, and vitamin B_6 are also needed (see Table A-16).

Signs of Deficiency. Constipation, poor balance, loss of appetite, numbness and tingling in hands and feet, megaloblastic anemia. Some psychiatric disturbances such as depression, poor memory, dementia, or confusion. Biochemical deficiency such as serum cobalamin level <150–170 pg/mL or serum methylmalonic acid (MMA) <0.35 μg/dL; a return in MMA to normal after cobalamin therapy indicates prior deficiency.

Vulnerable Populations. Adolescents with poor diet with low intake of meat/fish/dairy products or strict vegetarian (vegan) diets are at risk. Senior citizens and those with severe, chronic malnutrition are also at risk. Others at risk include those with pernicious anemia (autoimmune atrophic gastritis), disorders of the gastric mucosa, genetic defects (apoenzymes, absence of transcobalamin II, absence of ileal receptors), extensive disease affecting the stomach, intestinal infections, malabsorption (tropical sprue, celiac sprue, pancreatic insufficiency, Crohn's disease, fish tapeworm, HIV infection, or ileal resection), prolonged exposure to nitrous oxide or megadoses of folic acid. Monitor persons after total gastrectomy for megaloblastic anemia because intrinsic factor is not available. Some gastric bypass patients may also become deficient. Individuals who take zantac, pepcid, tagamet, metformin, omeprazole (Prilosec), lansoprazole (Prevacid) may also be at risk.

Signs of Excess. No toxicity is known.

Choline: Choline is a methyl-rich nutrient that is required for phospholipid synthesis and neurotransmitter function. Internal synthesis of phosphatidylcholine is insufficient to maintain choline status when intakes of folate and choline are low. *AI is set at 550 mg/d for adult men and 425 mg/d for adult women. UL level has been set at 3.5 g/d.*

Functions. Lipotropic agent, some role in muscle control and in short-term memory with the neurotransmitter acetylcholine, component of sphingomyelin, emulsifier in bile, and component of pulmonary surfactant (CO_2/O_2 exchange). It helps the body absorb and use fats, especially for cell membranes. Choline is used as a methyl donor to convert homocysteine to

TABLE A-16 Food Sources of Vitamin B_{12}

Food	Vitamin B_{12} (μg)
Boneless lamb chop, cooked, 3 ounces	2.7
Fortified cereals, one cup	1–6 (varies)
Light tuna canned in water, drained, 3 ounces	2.5
Salmon, cooked, 3 ounces	2.4
Ground beef, 90% lean, cooked, 3 ounces	2.3
Eye round roast and steak, cooked, 3 ounces	1.4
Plain yogurt, low fat, one cup	1.4
Roast turkey, 3 ounces	1.3
Milk, skim, one cup	1.3
Beef hot dog, cooked	0.9
Cottage cheese, low-fat, 4 ounces	0.7
Boneless top loin pork roast, cooked, 3 ounces	0.6
Fortified soy milk or rice milk, one cup	0.6
Chicken breast, cooked, one cup	0.5
Dark meat chicken, cooked, one cup	0.4
Cheddar cheese, one slice	0.2
American cheese, one ounce	0.2

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods. Mixed dishes and multiple preparations of the same food item have been omitted from this table.

methionine when folate intake is low. Choline magnesium trisalicylate is used to relieve pain, inflammation and tenderness caused by arthritis as a form of nonsteroidal anti-inflammatory medications

Sources. Eggs, high-protein animal products such as liver, dairy foods, soybeans, peanuts, cauliflower, lettuce, and chocolate. Lecithin is a choline precursor, as is phosphatidylcholine. Liver can synthesize or resynthesize. Average daily intake is 400–900 mg. High-fat intake accelerates deficiency.

Signs of Deficiency. Insufficient phospholipid synthesis occurs; neurotransmitter function and liver damage might occur.

Signs of Excess. Anorexia, fishy body odor, upset stomach.

Vitamin C (Ascorbic Acid, Dehydroascorbic Acid): Vitamin C is needed via an exogenous source by all humans and is found in fruits and vegetables. About 90% of dietary intake is absorbed. Concentration of vitamin C in plasma and other body fluids does not increase in proportion to increasing daily doses of vitamin C. Saturation is complete at 200 mg. *RDA for vitamin C is 75 mg for women and teen boys. Pregnant women need 85 mg and lactating women need 120 mg. Teen girls need 65 mg; 80 if pregnant or 115 if lactating.*

Men need 90 mg. Smokers need an extra 35 mg daily. The UL for vitamin C is 2000 mg/d.

Functions. Hydroxylation (lysine and proline) in collagen formation and wound healing, norepinephrine metabolism, tryptophan to serotonin transformation, folic acid metabolism, antioxidant as a scavenger of superoxide radicals and to protect vitamins A and E, changing ferric iron to ferrous iron, healthy immunity and prevention of infection, intracellular respiration, tyrosine metabolism, intercellular structures of bone, teeth, and cartilage, prevention of scurvy. It may defer aging through the collagen turnover process. Vitamin C also serves as a reducing agent, elevates HDL cholesterol in the elderly, and lowers total serum cholesterol. It is also needed for biosynthesis of carnitine and for metabolism of drugs and steroids. Dietary antioxidants, including vitamins C and E, may protect against atherosclerotic disease and cognitive impairment.

TABLE A-17 Food Sources of Vitamin C[a]

Food, Standard Amount	Vitamin C (mg)
Guava, raw, 1/2 cup	188
Red sweet pepper, raw, 1/2 cup	142
Red sweet pepper, cooked, 1/2 cup	116
Kiwi fruit, one medium	70
Orange, raw, one medium	70
Orange juice, 3/4 cup	61–93
Green pepper, sweet, raw, 1/2 cup	60
Green pepper, sweet, cooked, 1/2 cup	51
Grapefruit juice, 3/4 cup	50–70
Vegetable juice cocktail, 3/4 cup	50
Strawberries, raw, 1/2 cup	49
Brussels sprouts, cooked, 1/2 cup	48
Cantaloupe, 1/4 medium	47
Papaya, raw, 1/4 medium	47
Kohlrabi, cooked, 1/2 cup	45
Broccoli, raw, 1/2 cup	39
Edible pod peas, cooked, 1/2 cup	38
Broccoli, cooked, 1/2 cup	37
Sweet potato, canned, 1/2 cup	34
Tomato juice, 3/4 cup	33
Cauliflower, cooked, 1/2 cup	28
Pineapple, raw, 1/2 cup	28
Kale, cooked, 1/2 cup	27
Mango, 1/2 cup	23

Source: Nutrient values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17. Foods are from ARS single nutrient reports, sorted in descending order by nutrient content in terms of common household measures. Food items and weights in the single nutrient reports are adapted from those in 2002 revision of USDA Home and Garden Bulletin No. 72, Nutritive Value of Foods.
[a]Food sources of vitamin C ranked by milligrams of vitamin C per standard amount; also calories in the standard amount. (All provide ≥ 20% of RDA for adult men, which is 90 mg/d).

Sources. For details see Table A-17. No more than 1 g/d is stored in liver tissue. An excretion of 50% of intake is normal. Men are found to have lower serum levels than women. Smoking decreases serum levels; increased intake removes greater amounts of nicotine. Avoid high levels of pectin, iron, copper, or zinc from the diet and do not cook fruits or vegetables in copper pans.

Signs of Deficiency. The first symptom of deficiency is fatigue; treatment with vitamin C results in quick recovery and alleviation of symptoms. Insufficient (depletion) is 11–28 μmol/L, considered latent scurvy with mood changes; mild but distinct fatigue and irritability; vague, dull, aching pains. Deficient is <11 μmol/L with failure of wounds to heal, petechial hemorrhage, follicular hyperkeratosis, bleeding gums, eventual tooth loss, weak bones or cartilage and connective tissues, rheumatic pains in the legs, muscular atrophy, skin lesions, and psychological changes including depression and hypochondria. High blood lead levels will also deplete serum vitamin C.

Vulnerable Populations. Those with achlorhydria, Alzheimer's disease, burns, cancers, chronic diarrhea, nephrosis, pregnancy, severe trauma, surgical wounds, and tuberculosis. Older and very young are more likely to have weaker immune systems and need more vitamin C. Moderate-to-high alcohol intake reduces immune effectiveness; alcoholics are at risk of scurvy. High blood pressure and cholesterol can lead to immune problems and heart disease. Disease agents originating in the mouth may travel in addition to causing dental problems. Diabetes, high glucose, other abnormal labs can stress the immune system; pollutant exposure, steroid use, and radiation can deplete vitamin C levels. Individuals with gout, arthritis, bladder cancer, reflex pain after hip fracture, pneumonia, risk for stroke or macular degeneration or hip fracture, renal oxalate stones, pregnancy, *Helicobacter pylori* infection, or prolonged proton pump use should be evaluated closely.

Signs of Excess. GI distress and diarrhea are most common.

APPENDIX B

Dietetic Process, Forms, and Counseling Tips

INTRODUCTION TO THE PRACTICE OF DIETETICS

The American Dietetic Association (ADA) is responsible for establishing the expectations for required education, practice guidelines, and standards of professional performance for dietetic professionals. The Commission on Dietetic Registration maintains credentialing authority for the roles of Registered Dietitian (RD) and Dietetic Technician Registered (DTR). A scope of practice framework can be used to assist dietetic professionals in defining their responsibilities and refining practice according to professional job analyses that are conducted every few years.

Dietitians and dietetic technicians work in a variety of settings but tend to be **concentrated in healthcare settings:** acute care, long-term care, dialysis units, psychiatric facilities, community sites, and home health. They perform a variety of tasks including clinical services, food systems management, nutrition education, and public health functions. See Figure B-1.

There are over 59 core dietetics position descriptions including not only the roles of traditional clinical dietitian; outpatient dietitian; or Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) nutritionist but also covering areas such as consulting, sales, and communications. Generally, dietetics professionals aim to improve the health of their clientele by influencing their food and nutrition decisions. Many dietetics practice groups have now established standards of practice that are specific to their area of expertise. The following guidelines are useful in consideration of an effective practice in dietetics.

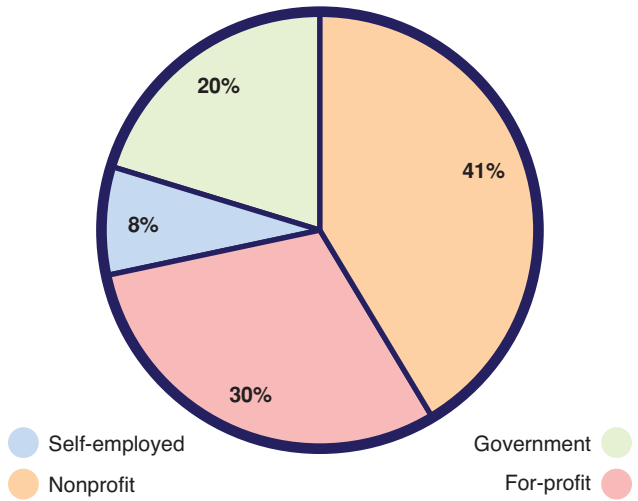

Figure B-1 Employment sector of dietetics practitioners (n = 8115) (Image courtesy of Compensation & Benefits Survey of the Dietetics Profession 2009.)

1. **Standards** from The Joint Commission and other organizations set the expectation that quality care will lead to positive outcomes. Effective leadership and continuous performance improvement are critical for maintaining quality.
2. **Regulations,** such as those from the Centers for Medicare and Medicaid Services (CMS), have been written to protect vulnerable patients/residents from inadequate care. For example, in long-term care, the F325 tag related to **§483.25 Nutrition** states that, based on a resident's comprehensive assessment, the facility must ensure that a resident (1) maintains acceptable parameters of nutritional status, such as body weight and protein levels, unless the resident's clinical condition demonstrates that this is not possible; and (2) receives a therapeutic diet when there is a nutritional problem. All of the guidelines and directives are to be followed by dietitians and their supporting staff members accordingly.
3. Most dietitians work in settings that are supportive of **interdisciplinary teamwork.** Dietitians must be proactive, take on new skills, and overcome traditional stereotypes. The roles are expanding, for example, in bariatric surgery, in functional medicine, and in home care.
4. Many dietetics positions now include **enhanced responsibilities** in nutritionally focused physical assessment, certification in cardiopulmonary resuscitation, assessment for the use of adaptive feeding equipment, worksite wellness, swallowing evaluations, exercise prescription and education, functional nutrition assessments, and home care services.
5. For **basic nutrition education**, advice can be provided by many healthcare professionals. However, **medical nutrition**

therapy (MNT) is an intensive approach to the nutritional management of chronic disease and requires significantly more education in food and nutrition science than is provided by other professional curricula. The RD is the single identifiable healthcare professional to have the most standardized education, supervised clinical training, and national credentialing, who meets the continuing education requirements for reimbursement as a direct provider of MNT.

6. **State licensure laws** protect the public from unscrupulous practices. Each licensure board establishes a relevant scope of practice for dietitians and nutritionists who practice in that state. Scopes of practice detail what can or cannot be performed by a person using the title of dietitian or nutritionist.
7. Application of the American Dietetic Association's **Nutrition Care Process** with **standardized terminology** enables dietitians to perform at a higher level of comprehensive care than in the past. See Figure B-2.
8. The best patient care emphasizes **coordinated, comprehensive care along the continuum of the healthcare delivery system**. Multidisciplinary teams are best suited to develop, lead, and implement evidence-based disease management programs. Evidence-based care integrates research into practice, regardless of the setting. Indeed, research is the basis of the profession (Pavlinac, 2010).
9. Dietitians must take an **evidence-based approach to care** based on their education, training, skill level, and work experiences. Science and evidence-based education can help to overcome dangerous perceptions of consumers or misunderstandings by members of the medical community (Krebs and Primak, 2006).
10. **Unique nutritional care guidelines** should be used in specialty areas such as cancer, diabetes, cardiology and pulmonary, gastroenterology, geriatrics, HIV-AIDS, liver disease, nutrition support, pancreatic disease, renal failure, surgical or transplantation units, intensive care, and functional nutrition therapy. See Figure B-3 for the Functional Medicine Matrix Model™ that was used as part of a holistic assessment format.
11. **Medical classification systems** are used for a variety of purposes including statistical analyses, reimbursement, decision-support systems, and procedures.
12. **Diagnostic-related groups (DRGs)** classify hospital cases into similar categories for reimbursement, assuming a similar use of resources. In 2007, Medical Severity DRG (MS-DRG) codes were added to enhance payment for conditions that involve greater morbidity or risk for mortality. In 2008, Hospital Acquired Condition (HAC) was described, and some conditions were no longer considered to be complications if they were not present upon admission (POA).
13. Use correct **nutrition diagnosis nomenclature** for malnourished adults in clinical practice settings: *"starvation-related malnutrition"* when there is chronic starvation without inflammation; *"chronic disease–related malnutrition"* when a mild to moderate inflammation is chronic; and *"acute disease or injury–related malnutrition"* for acute, severe inflammation (Jensen et al, 2009).
14. For the **coding of malnutrition** in the medical record, use ICD-9 code 262.
15. **Sentinel events** are defined by The Joint Commission as unexpected occurrences leading to serious physical or psychological injury or death. The risk for a sentinel event requires an immediate investigation and response. Issues such as changes in functional status, the severity of a nutrition diagnosis, unintended weight changes (>10% in 6 months), and changes in body composition such as the loss of subcutaneous fat are risks that should be examined.
16. **Order-writing privileges** by dietitians allow orders for nutritional prescriptions for enteral and parenteral nutrition to be implemented quickly and effectively. Order-writing must be established by the local facility with medical staff approval.
17. **Age-specific competencies** are needed to serve various populations. For example, children and adolescents require different meal plans and interventions from those for adults (Stang et al, 2006). Residents in long-term care facilities may need extra time and attention (Simmons and Schnelle, 2006). Individuals on nutrition support require monitoring while using age-specific guidelines (Durfee et al, 2006).
18. **Evaluation of outcomes** is significant. Customer satisfaction must meet or exceed basic patient expectations. **Health-related quality of life (HRQL)** is another important outcome measure. For example, the quality of life and nutritional status of older residents in long-term care facilities may be enhanced by a liberalization of diet prescriptions and person-centered care. This care involves residents in decisions about schedules, menus, and dining locations; weight loss, undernutrition, and other effects of poor nutrition and hydration are then decreased (Neidert, 2005).

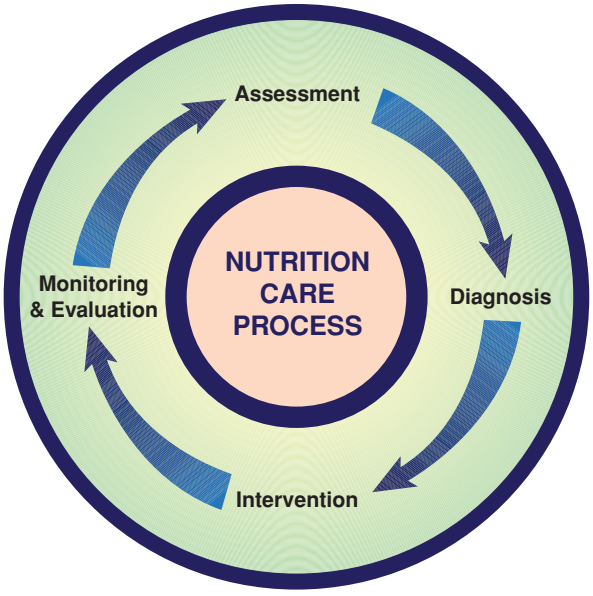

Figure B-2

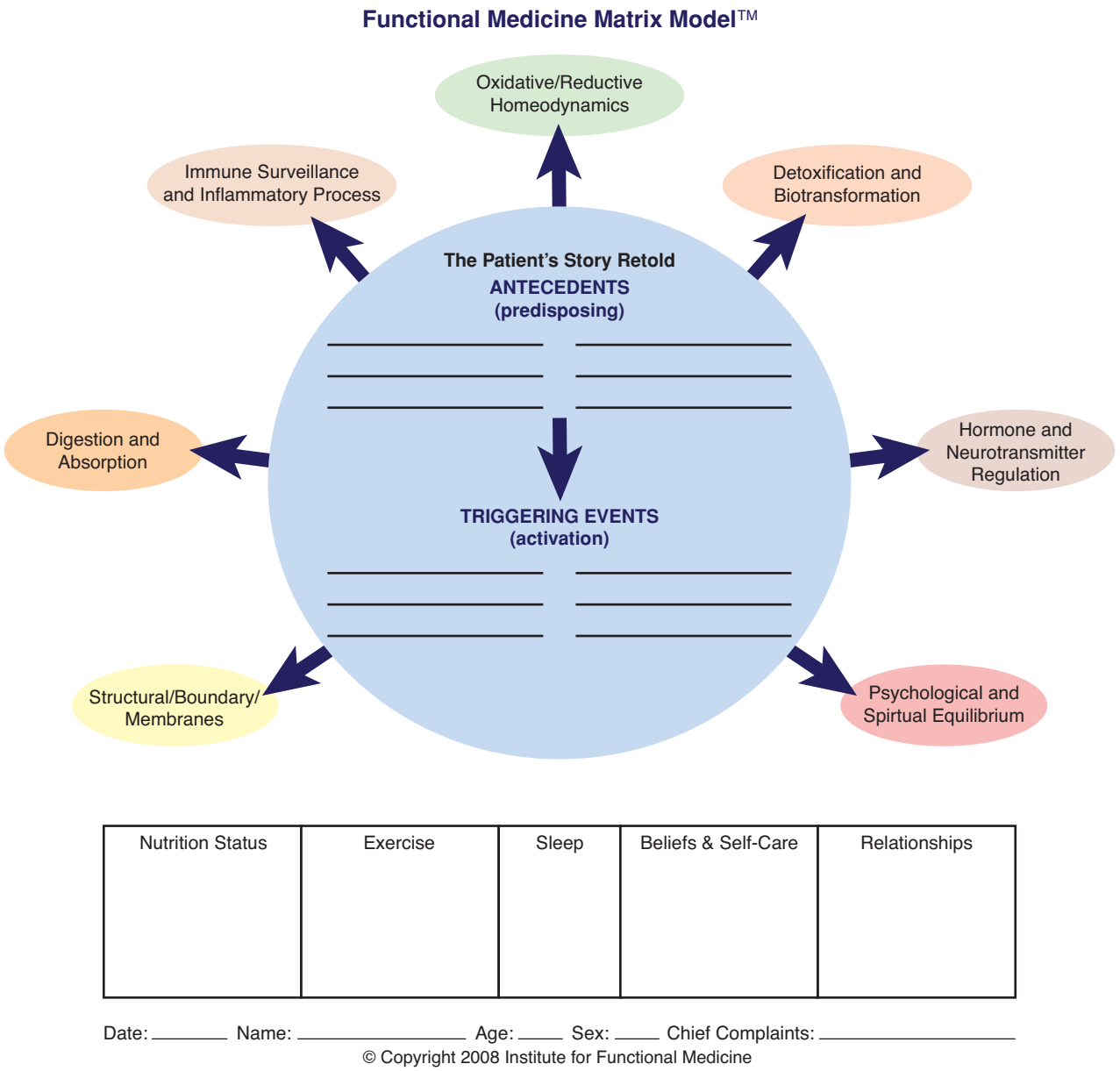

Figure B-3 Used with permission from The Institute for Functional Medicine.

19. **Ongoing review** is needed to monitor progress. Difficulty in obtaining current laboratory values makes it hard to follow patient outcomes after discharge. With electronic medical records and careful maintenance of confidentiality, monitoring can be more effective.
20. Dietetics professionals are likely to advance to **management positions**, such as food service director, consultant, or patient case manager, because they possess strong nutrition knowledge and management skills. Nearly half of all RDs and DTRs supervise other employees (Ward, 2010).
21. Having supervisory responsibility is strongly associated with higher wages (Ward, 2010). In addition, having a masters degree or PhD and one or more specialty certifications (e.g., CDE, CNSD, and the various Certified Specialist credentials offered by the Commission on Dietetic Registration) leads to an increased median wage (Ward, 2010).
22. **Food and nutrition managers** are in opportune positions to influence other managers, acquire resources, identify opportunities, and achieve desirable outcomes.
23. **Levels of responsibility** tend to increase with years of experience and with business skills such as leadership, marketing, and negotiation. Dietitians are encouraged to include management and leadership development as part of their continuing education portfolio.

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NUTRITION CARE PROCESS, FORMS, AND DOCUMENTATION TOOLS

The nutrition care process involves four steps and a comprehensive understanding of factors related to nutritional intake. Following a thorough **nutrition assessment** with patient history and current status, identify key problems (**nutrition diagnoses**). Use the latest edition of the nutrition diagnostic language from the American Dietetic Association, which is available to members at http://www.eatright.org/Members/content.aspx?id=5477. Write a PES statement (problem, etiology, and signs and symptoms) for each problem. Determine the plan of care, with **intervention** goals and actions, designed according to the etiology of the problem. Finally, plan how **monitoring and evaluation** will occur. The final step should be measurable and should be related to the identified signs and symptoms. **Sample tools and forms** are provided here; all tools should be adapted for the facility and for the age and complexity of patients in the population.

Table B-1 provides a sample scope of practice policy.

Table B-2 provides a nutrition intake history form for adults.

Table B-3 lists the advantages and disadvantages of different forms of nutrition histories.

Table B-4 provides a physical assessment checklist.

Table B-5 shows a method for estimating energy requirements in adults.

Table B-6 shows a method for estimating protein requirements in adults.

Table B-7 provides a sample pediatric nutrition assessment tool.

Table B-8 provides a chart that lists the biochemical analyses used in nutritional assessments.

Table B-9 provides a summary of food–drug interactions.

Table B-10 provides a sample worksheet for using the nutrition care process.

Table B-11 is a form for a clinical case review and for audit purposes.

Table B-12 provides tips for adult education and counseling.

Table B-13 describes various health promotion intervention models.

Table B-14 provides sample monitoring and evaluation audits for patient education.

TABLE B-1 Sample Hospital Nutrition Department Scope of Services

PURPOSE

In order to provide care consistent with the mission of the hospital system, the nutrition department has defined its scope of services and goals.

POLICY

It is the policy of the hospital system for each department to provide care and/or services based on the defined scope of services and goals and to implement policies and procedures based on the scope of services and goals.

DEPARTMENT MISSION

The nutrition department commits itself to enhancing the quality of life throughout the patient's life cycle and promoting and restoring health through the provision of quality nutritional care services in an environment that ensures dignity and respect for each person. The nutrition department has an obligation to ensure continuous quality improvement in the care and services it provides.

PROCEDURE

Care/Services Provided

The department provides timely nutrition assessment, monitoring, counseling, education, discharge planning, and diet instruction, as defined in department policies, to meet the needs of patients' various backgrounds, which affect nutritional preferences and habits. Administration evaluates the activities of the department with regard to the provision of quality dietary intervention, patient treatment and outcomes, and the quality of meal service.

Types of Patients/Customers Served

Nutrition services are available for all segments of the hospital population—newborn, infant, child, adolescent, adult, and geriatric—including outpatient services, cardiopulmonary rehabilitation, emergency department, corporate wellness, and community events.

Timeliness

Clinical dietitians are scheduled for Monday through Saturday, with shift availability at all meal periods. One dietitian is scheduled for weekend coverage; Sunday coverage is an on-call assignment. When appropriate, formula calculations, calorie counts or nutrient analyses, and basic nutrition assessments can be completed via the computer system and any telephone within the system to facilitate timely treatments.

STAFFING

The clinical nutrition manager sets a staffing plan to meet the needs of the patient population. A staffing assessment is completed every 2 years or when deemed necessary. Dietitian schedules are prepared in advance, including hours of duty and on-call schedules.

(continued)

TABLE B-1 Sample Hospital Nutrition Department Scope of Services *(continued)*

STAFF CREDENTIALS AND REQUIREMENTS

Dietitians are registered through the Commission on Dietetic Registration; each must maintain their registration through continuing education. The education requirement is 75 hours during each 5-year period. Competency standards are developed, updated, and reviewed regularly by the clinical nutrition manager to meet the specific, changing needs of the facility.

ASSIGNMENTS

Full-time equivalents of clinical dietitians are assigned to patient care and ambulatory clinics. One full-time equivalent of a clinical nutrition manager leads the clinical/outpatient staff. Staff are assigned to community education programs upon request and scheduling allows. Based on the daily census and acuity, clinical and outpatient dietitians may adjust the daily staffing pattern to meet needs according to changes in patient acuity.

STANDARDS OF CARE

Clinical dietitians apply the hospital-approved standards of nutrition care and the evidence-based guidelines of the American Dietetic Association.

SCREENING

All inpatients are screened by the nursing department within 24 hours of admission to determine the need for further nutrition assessment. Clinical dietitians develop the screening triggers, which are approved by the medical staff. Patients receive nutrition intervention based on the priority/acuity levels assigned by the dietitians. The clinical dietitians perform subsequent re-screening according to policy and regulatory guidelines.

Priority for Nutrition Services	High Priority	Moderate Priority	Low Priority
Definitions/Indications	Nutrition interventions that warrant frequent comprehensive patient reassessment to document the impact of the intervention on critical nutrition outcomes, medical status (i.e., labs, GI tolerance, etc.), and/or clinical progress. Examples: New or modified EN/PN or use of medical food supplements. Comprehensive nutrition education or counseling.	Nutrition interventions that warrant early evaluation, which can be sufficiently tracked via brief f/u notes. Less frequent comprehensive patient reassessment is needed. Examples: Stable EN or PN. Brief nutrition education for survival skills. Coordination of care.	Nutrition interventions that bring closure and/or immediately resolve a patient's nutrition diagnosis/problem OR when no nutrition intervention is needed. F/U is expected in the form of rescreening with reassessment as indicated by a change in status. Examples: Brief nutrition education. No nutrition diagnosis. Palliative care only.
Plan of care review standard	Update POC when reassessing patient.	Within 5 days, based on a brief chart review, communication w/ other patient care providers as appropriate and patient contact. State the nutrition diagnosis, response to intervention, and the new or modified plan.	Per rescreening standards
Reassessment standard	Within 3 days	Within 7 days	Per rescreening standards

ASSESSMENT AND NUTRITION DIAGNOSES

The clinical dietitians assess patients using the guidelines in the standards of care approved by the medical staff. Following the profession's nutrition care process, a nutrition diagnosis is established as needed. The etiology of the diagnosis selected determines the type and extent of intervention that will be provided. The most common nutrition diagnoses in this facility are as follows:

Inadequate oral food/beverage intake (NI-2.1)
Inadequate protein-energy intake (NI-5.3)
Increased nutrient needs (NI-5.1)
Evident malnutrition (NI-5.2)
Inadequate energy intake (NI-1.4)
Swallowing difficulty (NC-1.1)
Involuntary weight loss (NC-3.2)
Inadequate intake from enteral/parenteral nutrition (NI-2.3)
Food and nutrition-related knowledge deficit (NB-1.1)
Underweight (NC-3.1)
Overweight/obesity (NC-3.3)
Excessive energy intake (NI-1.5)

INTERVENTIONS: FOOD AND NUTRIENT DELIVERY

According to the patient's ability to take in sufficient energy, protein, carbohydrate, fat, vitamins, minerals and water, the clinical dietitian will approve or recommend alterations in the nutrition prescription, the addition or discontinuation of enteral/parenteral nutrition, and the use of specific supplements or bioactive substances (e.g., fish oil, lycopene, lutein, ginger, and multivitamin–mineral supplements).

Most common food–nutrient delivery interventions in this facility are as follows:

General/healthful diet (ND- 1.1)
Commercial beverages (ND-3.1.1)
Initiate EN or PN (ND-2.1)
Modify rate, concentration, composition or schedule (ND-2.2)
Modify distribution, type, or amount of food and nutrients within meals or at specified times (ND-1.2)
Modified beverages (ND-3.1.3)
Modified food (ND-3.1.4)
Multivitamins/minerals (ND-3.2.1)
Nutrition-related medication management (ND-6.1)

(continued)

TABLE B-1 Sample Hospital Nutrition Department Scope of Services *(continued)*

INTERVENTIONS: EDUCATION

The clinical dietitian performs a nutrition education needs assessment to determine patient/family knowledge and skill level. The patient's age, barriers to learning, assessed needs, abilities, readiness, and length of stay will determine the level of education to provide. Because lifestyle factors, financial concerns, patient/family expectations, and food preferences affect the individualized education plan, most patients will be referred to ambulatory clinics if extensive education is required to resolve a nutrition diagnosis. The most common education intervention in this facility is Nutrition Education—Survival Information (E-1.3).

INTERVENTIONS: COUNSELING

The clinical dietitian provides individualized guidance and nutritional advice to the patient and/or family members. Techniques to promote behavioral change are used, as appropriate, and documented accordingly. Readiness to change is noted. Follow-up counseling includes an evaluation of the effectiveness of counseling given and recommendations for additional follow-up. There are no specific counseling interventions most often noted for the in-patient setting; this type of intervention is handled in the ambulatory clinics.

INTERVENTIONS: COORDINATION OF CARE

The clinical dietitians participate in both weekly and patient care meetings on assigned units. Discharge planning needs are identified throughout the assessment, re-assessment, and education processes. These needs are referred to the social worker or case manager and communicated to the team during meetings. The dietitian communicates with the referring agency, facility, or next dietitian provider when a specialized care plan is needed. The most common coordination of care interventions in this facility are as follows:

Team meeting (RC-1.1)

Referral to an RD with different expertise (RC-1.2)

Collaboration/referral to other providers (RC-1.3)

MONITORING AND EVALUATION

The clinical dietitian selects the appropriate measures to monitor and evaluate the effectiveness of interventions. The top nutrition monitors used in this facility are as follows:

Total energy intake (FI-1.1)

Enteral/Parenteral nutrition intake formula/solution (FI-3.1.2), initiation (FI-3.1.4), rate/schedule (FI-3.1.5)

Nutrition physical exam findings: skin (S-3.1.6), gastrointestinal (S-3.1.3)

Weight/weight change (S-1.1.4)

DEPARTMENT GOALS

In support of the hospital mission to provide high-quality healthcare through continuous improvement and to exceed the expectations of patients and customers, the clinical nutrition staff of the food and nutrition department have the following goals:

1. Staff qualified, progressive Registered Dietitians who exceed baseline knowledge proficiency standards set by the American Dietetic Association. To accomplish this, all Registered Dietitians must set annual professional development goals on their performance evaluations. These goals are supported by the clinical nutrition manager, funded by Food and Nutrition Services, and tracked through the professional development portfolio of the Commission on Dietetic Registration (CDR).
2. Provide timely nutrition therapy, which results in measurable improvements to patient health from the time of admission to the time of discharge from therapy. Dietitians will follow outcomes of therapy via objective measures (e.g., weight, verbalization of learning, and blood glucose improvements) and chart them in their documentation.
3. Ensure that outpatient medical nutrition therapy and diabetes education programs, combined, will break-even financially. Billing and accounts receivable will be monitored by the program coordinator. Two or three free community education nutrition programs will be offered yearly to local county residents.
4. Ensure that patients will indicate >90% satisfaction rating with their inpatient nutrition care and ambulatory services.
5. Review and administer the expanded dietitian scope of practice for those staff members who have demonstrated competency and have certification (where available) in their specialty area.

EXPANDED SCOPE OF PRACTICE

Because malnutrition is a key factor in determining the prognosis of a patient, protein, energy, vitamin and mineral deficiencies can be detected and corrected earlier through a better utilization of RD skills by assigning order-writing and prescribing privileges to the qualified dietitian specialists on staff. Prescribing authority boosts patient satisfaction by offering quicker interventions, reduces costs with shorter lengths of stays, improves nutrition outcomes, and promotes greater professional satisfaction by those qualified dietitians. Currently, there are three levels of prescriptive authority for dieticians, which are implemented in healthcare facilities and defined as follows:

- **No prescriptive authority:** RDs recommend supplements, diets or diet changes, nutrition-related tests, or procedures to the physician. Recommendations may be written in the medical record or discussed with the physician, but a physician must write an order before recommendations are implemented.
- **Dependent prescriptive authority:** RDs have the authority to order diets, nutritional supplements, nutrition-related tests, or procedures according to protocols, algorithms, policies, or preapproved criteria that specify the type and magnitude of change. Physician approval of the nutritional treatment is not required as long as the orders and treatments provided fall within the guidelines of the protocol, algorithm, policy, or criteria approved in advance by the appropriate institutional authority (e.g., patient-care committee, medical executive committee). However, the RD can discuss the nutritional care provided with the physician and document the order appropriately in the medical record.
- **Independent prescriptive authority:** The RD may act autonomously to provide nutritional services that include ordering diets, nutrition-related tests, or procedures authorized by the clinical privileges granted to the practitioner by the appropriate institutional authority. Physician approval is not required; however, the RD can discuss the nutritional care provided with the physician and document the order appropriately in the medical record.

Steps for Expanded Nutrition Scope of Practice with Dietitian Order-Writing Privileges:

- Evaluate state licensure laws.
- Investigate the level of prescriptive authority permitted at the facility.
- Assure that current licensure and board certifications are accurate in the staff database.
- Discuss plans with each RD staff member with regard to credentialing, graduate study, fellowships, and other measures of basic and continuing competency.

(continued)

TABLE B-1 Sample Hospital Nutrition Department Scope of Services *(continued)*

- When RDs are allowed to have complete order-writing privileges, patient care and outcome are better than they would be if a physician must first sign off on orders.
  - A literature review conducted on the clinical nutrition management list-serv noted that 42–57% of nutrition recommendations were actually put into action by the physician.
  - An average of 17 hours was required before orders were implemented by the MD.
  - Length of stay decreased by up to 5 days for patients who were given nutrition interventions earlier.
- Document success stories, cost savings, improved timeliness of care, shorter LOS than average related to early detection and intervention by the RD.
- Detail the desired scope of dietetics practice using the ADA evidence analysis library, the appropriate standards of professional practice, and other evidence-based guidelines.
- Draft new nutrition policies and procedures. Define the desirable steps for expanded scope of practice (e.g., ordering nutrition-related lab tests; prescribing basic diet orders; changing therapeutic diet orders according to measures such as weight loss or gain, decreased functional ability, significant appetite change; developing standing orders for common nutrition problems and interventions; and writing or updating enteral/parenteral infusion orders).
- Construct an **approval packet with letters of support** for the nutrition committee to establish medical review/consent. When approved by the nutrition committee, obtain signatures from the executive board, chief of medical staff, director of nursing, and hospital director. Include the time period for the scope and a date of review.

TABLE B-2 Dietary Intake Assessment and Nutrition History

Methods for Assessing Dietary Intake	Advantages	Disadvantages
24-Hour Recall: An informal, qualitative method in which the patient recalls all of the foods and beverages that were consumed in the last 24 hours, including the quantities and methods of preparation.	Dietary information is easily obtained. It is also good during a first encounter with a new patient in which there are no other nutritional data. Patients should be able to recall all that they have consumed in the last 24 hours.	It is very limited and may not represent an adequate food intake for the patient. Data achieved using this method may not represent the long-term dietary habits of the patient. Estimating food quantities and food ingredients may be difficult, especially if the patient ate in restaurants.
Usual Intake/Diet History: This method asks the patient to recall a typical daily intake pattern, including amount, frequencies, and methods of preparation. This intake history should include all meals, beverages, and snacks. Include discussion of usual intake and lifestyle recall. This consists of asking the patient to run through a typical day in chronological order, describing all food consumption as well as activities. This method is very helpful because it may reveal other factors that affect patient's nutritional and overall health.	This method evaluates long-term dietary habits and is quick and easy to do. Components usually include the following: Ability to secure and prepare food Activity pattern Alcohol or illicit drug use Appetite Bowel habits—diarrhea, constipation, steatorrhea Chewing/swallowing ability Diet history—usual meal pattern Disease(s) affecting use of nutrients Food allergies/intolerances Medications Nausea or vomiting NPO status or dietary restrictions Pain when eating Satiety level Surgical resection or disease of the GI tract Taste changes or aversions Vitamin/mineral or other nutritional supplements Weight changes	A limited amount of information on the actual quantities of food and beverages is obtained. Also, this method only works if a patient can actually describe a "typical" daily intake, which is difficult for those who vary their food intake greatly. In these patients, it is advisable to use the 24-hour recall method. Another disadvantage is that patients may not include foods that they know are unhealthy.

(continued)

TABLE B-2 Dietary Intake Assessment and Nutrition History *(continued)*

Methods for Assessing Dietary Intake	Advantages	Disadvantages
Food Frequency Questionnaire: This method makes use of a standardized written checklist where patients check off the particular foods or type of foods they consume. It is used to determine trends in patients' consumption of certain foods. The checklist puts together foods with similar nutrient content, and frequencies are listed to identify daily, weekly, or monthly consumption.	It is possible to identify inadequate intake of any food group so that dietary and nutrient deficiencies may be identified. The questionnaire can be geared to a patient's pre-existing medical conditions.	Patient error may occur in filling out how foods are prepared. Patients may over- or underestimate food quantities.
Dietary Food Log: Patient records all food, beverage, and snack consumption for a 1-week period. Specific foods and quantities should be recorded. The data from the food log may later be entered into a computer program, which will analyze the nutrient components of the foods eaten according to specific name brands or food types. Patients are asked to enter data into the food log immediately after food is consumed.	A computer can objectively analyze data obtained. Data on calorie, fat, protein, and carbohydrate consumption can be obtained. Also, since patients are asked to enter data immediately after eating, the data are more accurate than other methods.	Patients may err in entering accurate food quantities. In addition, it is possible that the week-long food log does not accurately represent a patient's normal eating habits since he or she knows that the foods eaten will be analyzed, and thus, he or she may eat healthier for that week.

TABLE B-3 Adult Nutrition History Questionnaire

Patient Name _____ Date_____

1. Height _____feet _____inches
2. How much weight have you gained or lost in the past month? ____ 3 months? ____ 1 year? _____ Present weight _____ BMI _____ Usual weight _____ Desirable weight for height _____
3. Have you ever had problems with your weight? Overweight Underweight Comment:_____
4. Describe your eating habits: Good Fair Poor
5. How often do you skip meals? Daily Seldom Never
6. Describe your appetite recently: Hearty Moderate Poor
7. If you snack between meals, describe your typical snack _____ How often? _____
8. When you chew your food, do you have problems or take a long time? Yes No
9. Do you wear dentures at mealtime? Yes No. If yes, do they fit comfortably? Yes No
10. List the vitamin/mineral supplements/herbs and botanicals you take:_____
11. How many alcoholic drinks do you consume in a day? None 1–2 drinks More than 2 drinks
12. Describe foods you **DO NOT** tolerate: _____ Why? _____
13. List foods that you especially dislike:_____
14. List your favorite foods:_____
15. List foods that you are **ALLERGIC** to: _____What symptoms do you experience? _____
16. What type of modified diet do you follow? _____ What types in the past? _____ Who prescribed or suggested this diet for you? Doctor Friend Self-selected
17. How many meals do you eat at home each week? _____ at school(s)? ____ in restaurants? ____
18. What is your current occupation? _____
19. How active are you on a daily basis? Sedentary ___ Moderate ___ Active ____

TABLE B-3 Adult Nutrition History Questionnaire *(continued)*

Usual Daily Intake

Time	Meal	Food/Method of Preparation	Amount Eaten	RD Calculations
	Morning			
	Snack			
	Noon			
	Snack			
	Dinner			
	Snack			
	During the night			

24-Hour Diet Recall Form

Please be as specific as possible. Include all beverages, condiments, and portion sizes.

Time	Food Item and Method of Preparation	Amount Eaten	Where

Dietitian Comments:

Calculations: Estimated energy needs: ___kcal/kg Est protein needs: ___g/kg Est fluid needs: __ml/kg

Signed: _____RD Date:_____

TABLE B-4 Physical Assessment for Clinical Signs of Malnutrition

Certain risk factors and signs of nutrient deficiency or excess can be identified during the physical examination. One sign is rarely diagnostic; the more signs present, the more likely it is that they reflect a malnourished individual.

Nutrient	Physical Signs of Deficiency
Energy	Severe undernutrition, emaciation
Protein	Dull, dry depigmented hair; easily pluckable
	Edematous extremities
	Poor wound healing; pressure ulcers
	Pneumonia
Carbohydrate	Irritability, fatigue
	Respiratory or neurological changes
Fat	Skin rashes
	Dry, flaky skin
	Loss of subcutaneous fat
Vitamin A	Hair follicle blockage with a permanent "goose-bump" appearance
	Dry, rough skin
	Small, grayish, foamy deposits on the conjunctiva adjacent to the cornea
	Drying of the eyes and mucous membranes
Vitamin K	Small hemorrhages in the skin or mucous membranes
	Prolonged bleeding time
Thiamin	Weight loss
	Muscular wasting
	Occasional edema (wet beriberi)
	Malaise
	Tense calf muscles
	Distended neck veins
	Jerky eye movement
	Staggering gait and difficulty walking
	Infants may develop cyanosis
	Round, swollen (moon) face
	Foot and wrist drop
Riboflavin	Tearing, burning, and itching of the eyes with fissuring in the corners of the eyes
	Soreness and burning of the lips, mouth, and tongue
	Cheilosis (fissuring and/or cracking of the lips and corners of the mouth)
	Purple swollen tongue
	Seborrhea of the skin in the nasolabial folds, scrotum, or vulva
	Capillary overgrowth around the corneas
Niacin	Dermatitis or skin eruptions
	Tremors
	Sore tongue
	Skin that is exposed to sunlight develops cracks and a scaly form of pigmented dermatitis
	May also show signs of riboflavin deficiency
Vitamin B_6	Tongue inflammation
	Inflammation of the lining of the mouth
	Fissures in the corners of the mouth
Folate	Weakness, fatigue, and depression
	Pallor
	Dermatologic lesions
Vitamin B_{12}	Lemon-yellow tint to the skin and eyes
	Smooth, red, thickened tongue
Vitamin C	Impaired wound healing
	Edema
	Swollen, bleeding, and/or retracted gums or tooth loss; mottled teeth; enamel erosion
	Lethargy and fatigue
	Skin lesions
	Small red or purplish pinpoint discolorations on the skin or mucous membranes (petechiae)
	Darkened skin around the hair follicles
	Corkscrew hair
Chromium	Corneal lesions
Copper	Hair and skin depigmentation
	Pallor
Iodine	Goiter
Iron	Skin pallor
	Pale conjunctiva
	Fatigue
	Thin, concave nails with raised edges
Magnesium	Tremors, muscle spasms, tetany
	Personality changes
Potassium	Severe hypertension
	Arrhythmias
Zinc	Delayed wound healing
	Hair loss
	Skin lesions
	Eye lesions
	Nasolabial seborrhea
	Pressure ulcers

Adapted from: Halsted C, et al. Preoperative nutritional assessment. In Quigley E, Sorrell M, eds. *The gastrointestinal surgical patient: preoperative and postoperative care.* Baltimore: Williams & Wilkins, 1994; pp. 27–49.

TABLE B-5 Calculation of Adult Energy Requirements

For adults, body mass index is a useful guide. BMI = Weight (kg)/Height ^{2} (m) [Where 1 kg = 2.2 lb; 1 in = 2.54 cm] or [Weight (lb) × 705/Height (in)]. Basal metabolic rate (BMR) or basal energy expenditure (BEE) is energy expenditure at rest. It involves energy required to maintain minimal physiological functioning (i.e., heart beating, breathing). Various methods are used to determine BEE and energy needs. Resting energy expenditure (REE) includes specific dynamic action for digestion and absorption (slightly above basal) and is considered relatively equivalent to BEE in a clinical context. Factors affecting energy requirements and BMR include the following:

1. Age. Infants need more energy per square meter of body surface than any other age group. BMR declines after maturity.
2. Body Size. Total BMR relates to body size; large persons require more energy for basal needs and for activity.
3. Body Composition. Lean tissue is more active than adipose tissue.
4. Climate. A damp or hot climate often decreases BMR, and a cold climate increases BMR slightly.
5. Hormones. Thyroxine increases BMR; sex hormones and adrenaline alter BMR mildly; low zinc intake may lower BMR.
6. Fever. BMR increases by 7% for each degree above normal in Fahrenheit; 10% for each degree Centigrade.
7. Growth. BMR increases during anabolism in pregnancy, childhood, teen years, and the anabolic phase of wound healing.

REE can be from normal to below normal in mild starvation, such as that which occurs in the hospital setting. **Unplanned weight loss** can affect morbidity and mortality. Problematic % weight change in adults is considered to be: >2% in 1 week, >5% in 1 month, >7.5% in 3 months, > 20% in any period of time. >40% is incompatible with life.

Indirect calorimetry is useful for the critically ill where the calculation of energy expenditure is measured by gas exchange (VO_2 and VCO_2 represent intracellular metabolism). However, note that the energy needs of critically ill or injured adults are lower than previously thought; estimates are based on body mass index (BMI) if indirect calorimetry is not available. Overfeeding can cause fluid overload, increased CO_2 production, and hepatic aberrations. Defer weight repletion until a patient's critical episode subsides; needs generally normalize again in 3–6 weeks. Feeding programs should support a patient being within 10–15 pounds of desirable BMI range. Use these guidelines:

For Adults	Sedentary (kcal/kg)	Moderate	Active (kcal/kg)	Critically Ill (kcal/kg)
Overweight BMI >30	20–25	30	35	15–20
Normal weight BMI 20–29	30	35	40	20–25
Underweight BMI 15–19	30	40	45–50	30–35

(continued)

TABLE B-5 Calculation of Adult Energy Requirements *(continued)*

Mifflin – St. Jeor Equation Sheet

Weight^			Height				Age	
Pounds	Kilograms	MSJ*	Feet and Inches	Inches	Centimeters	MSJ*	Years	MSJ*
85	38.64	**386.36**	4′ 9″	57	144.78	**904.88**	70	**350**
90	40.91	**409.09**	4′ 10″	58	147.32	**920.75**	72	**360**
95	43.18	**431.82**	4′ 11″	59	149.86	**936.63**	74	**370**
100	45.45	**454.55**	5′	60	152.4	**952.50**	76	**380**
105	47.73	**477.27**	5′ 1″	61	154.94	**968.38**	78	**390**
110	50.00	**500.00**	5′ 2″	62	157.48	**984.25**	80	**400**
115	52.27	**522.73**	5′ 3″	63	160.02	**1000.13**	81	**405**
120	54.55	**545.45**	5′ 4″	64	162.56	**1016.00**	82	**410**
125	56.82	**568.18**	5′ 5″	65	165.1	**1031.88**	83	**415**
130	59.09	**590.91**	5′ 6″	66	167.64	**1047.75**	84	**420**
135	61.36	**613.64**	5′ 7″	67	170.18	**1063.63**	85	**425**
140	63.64	**636.36**	5′ 8″	68	172.72	**1079.50**	86	**430**
145	65.91	**659.09**	5′ 9″	69	175.26	**1095.38**	87	**435**
150	68.18	**681.82**	5′ 10″	70	177.8	**1111.25**	88	**440**
155	70.45	**704.55**	5′ 11″	71	180.34	**1127.13**	89	**445**
160	72.73	**727.27**	6′	72	182.88	**1143.00**	90	**450**
165	75.00	**750.00**	6′ 1″	73	185.42	**1158.88**	91	**455**
170	77.27	**772.73**	6′ 2″	74	187.96	**1174.75**	92	**460**
175	79.55	**795.45**	6′ 3″	75	190.5	**1190.63**	93	**465**
180	81.82	**818.18**					94	**470**
185	84.09	**840.91**					95	**475**
190	86.36	**863.64**					96	**480**
195	88.64	**886.36**					97	**485**
200	90.91	**909.09**					98	**490**
205	93.18	**931.82**					99	**495**
210	95.45	**954.55**					100	**500**
215	97.73	**977.27**					101	**505**
220	100.00	**1000.00**					102	**510**
225	102.27	**1022.73**					103	**515**

*REE for males = (MSJ weight + MSJ Height − MSJ age) + 5
*REE for females = (MSJ weight + MSJ Height − MSJ age) − 161
^Always use actual body weight. Activity factor is 1.2 if confined to bed; 1.3 if ambulatory.

Body Mass Index (BMI)
Weight (lb)

Height	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205
5′0″	20	21	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40
5′1″	19	20	21	22	23	24	25	26	26	27	28	29	30	31	32	33	34	35	36	37	38	39
5′2″	18	19	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	36	37	37
5′3″	18	19	19	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	35	36
5′4″	17	18	19	20	21	21	22	23	24	25	26	27	27	28	29	30	31	32	33	33	34	35
5′5″	17	17	18	19	20	21	22	22	23	24	25	26	27	27	28	29	30	31	32	32	33	34
5′6″	16	17	18	19	19	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32	33
5′7″	16	16	17	18	19	20	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32
5′8″	15	16	17	17	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30	31
5′9″	15	16	16	17	18	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30
5′10″	14	15	16	17	17	18	19	19	20	21	22	22	23	24	24	25	26	27	27	28	29	29
5′11″	14	15	15	16	17	17	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	29
6′0″	14	14	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28
6′1″	13	14	15	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27
6′2″	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	22	23	24	24	25	26	26
6′3″	12	13	14	14	15	16	16	17	17	18	19	19	20	21	21	22	22	23	24	24	25	26
6′4″	12	13	13	14	15	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	24	25

TABLE B-6 Calculations of Adult Protein Requirements

It requires 6.25 g of dietary protein to equal 1 g of nitrogen. Therefore, estimated nitrogen requirements × 6.25 = estimated protein needs in grams. If energy is not provided in adequate amounts, protein tissues become a substrate. Extra protein intake may be needed to compensate for excess protein loss in specific patient populations such as those with burn injuries, open wounds, and protein-losing enteropathy or nephropathy. Lower protein intake may be necessary in patients with chronic renal insufficiency who are not treated by dialysis and certain patients with hepatic encephalopathy. The following table provides an estimate for protein needs in adults.

Clinical Condition	Daily Protein Requirement (g/kg body weight)
Normal	0.8
Metabolic stress (illness/injury)	1.0–1.5
Acute renal failure (undialyzed)	0.8–1.0
Hemodialysis	1.2–1.4
Peritoneal dialysis	1.3–1.5

Adapted from the American Gastroenterological Association. Medical position statement: parenteral nutrition. *Gastroenterol.* 121:966, 2001.

TABLE B-7 Pediatric Nutrition Assessment

Date of Birth _____ Hx of LBW or other birth problems _____ Present Illness: _____
Medical History: _____ Social History: _____ Family Medical Hx: _____
Height (Length): _____(cm) Height for Age: _____(%ile) % Height for Age: _____ Interpretation: _____
Current weight: _____(kg) Weight for Age: _____(%ile) % Weight for Height: _____ Interpretation: _____
Ideal Weight for Height: _____(kg) Ideal Height for Age: ____(cm) Weight change? (days, weeks, or months) _____
Head circumference: _____(cm) _____(%ile) _____ (For children <3 years old, use the growth chart.)
Review of Systems for Nutritional Deficiencies:
General: _____
Skin: _____ Hair: _____ Nails: _____ Head: _____
Eyes: _____ Mouth: _____ GI/Abdomen: _____ Cardiac: _____
Extremities: _____ Neurological: _____ Musculoskeletal: _____
Laboratory Test Results: _____
Child allergic to any food or drinks? Yes / No If yes, allergic to what? _____ Rash or eczema? Yes / No
Does the child avoid any specific foods such as milk or meats? Yes / No If yes, which ones? _____
Does the child take any vitamins, minerals, or food supplements? Yes / No If yes, which? _____ With fluoride Yes / No
If not taking a vitamin, does the water supply contain fluoride? Yes / No
Using formula? _____ How much given and how much water is added? _____ Put to bed with a bottle? Yes / No
What type of milk? _____ # ounces/day? _____ Other beverages during the day? Iced tea Soda

Diet soda Kool-aid Juice Water Other _____

If eating foods, at what age were solids introduced into the diet? _____ How many meals are eaten during the day? ___
How many snacks are eaten during the day? _____ What types? _____
Does the child usually eat the food that is prepared for the family? Yes / No
Does the child chew on any of the following: Dirt Clay Paint chips Woodwork Ice Plaster Newspaper
How old is the house? Are there lead pipes? Yes / No Has the water been tested for lead? Yes / No
Estimated energy needs: _____ Protein needs: _____ Fluid needs: _____

Patient Age	Energy Requirements	Fluid
Infants, up to 6 months	108 kcal/kg	1–10 kg = 100 cc/kg body weight
Infants, 6 months to 1 year	98 kcal/kg	11–20 kg = 1000 cc plus 50 cc/kg body weight >10 kg
Children	Requirements increase to 102 kcal/kg from 1–3 years of age.	≥21 kg = 1500 cc plus 20 cc/kg body weight >20 kg
	Requirements gradually decrease with age to 70 kcal/kg at age 10.	—
Adolescents	45–55 kcal/kg male; 40–47 kcal/kg female	Can use 30 cc/kg body weight; increase with fever, illness, diseases

Dietitian Comments:
Calculations: Estimated energy needs: ___kcal/kg Estimated protein needs: ___g/kg Estimated fluid needs: __ml/kg
Signed: _____ RD Date: _____

Adapted from: University of California, Los Angeles Nutrition Department. Pediatric Nutrition Assessment Checklist. Accessed April 8, 2010 at http://apps.medsch.ucla.edu/nutrition/chklst2.htm.

TABLE B-8 Interpretation of Lab Values

Many lab tests can provide useful information on patients' nutritional status along with information on their medical status. Age-specific criteria should be used to evaluate data. Common tests include the following.

Allergy Antibody Assessment

Allergy blood and skin testing
C-reactive protein
Helicobacter pylori Antibodies Test
Immunoglobulins: IgG, IgM, IgA, IgD, and IgE; specific IgE for molds, IgG for spices and herbs
Rheumatoid factor (RF)

Blood Chemistry and Renal Tests

Albumin
Blood urea nitrogen (BUN)
Creatinine clearance
Glomerular filtration rate (GFR)
Nitrogen balance
Serum creatinine
Serum proteins, total protein

Cardiopulmonary System

Arterial blood gases
Cardiac enzymes
Creatine kinase
Homocysteine
Lactate dehydrogenase, aspartate transaminase (AST)
Lipid profile: Cholesterol, lipoproteins, triglycerides

Celiac Assessment

IgA-antitissue transglutaminase (tTG)
IgA-antiendomysial antibodies (IgA-EMA)
IgA-antigliadin antibodies (IgA-AGA)

Coagulation Tests

Bleeding or coagulation time
International normalized ratio (INR)
Prothrombin time (PT)

Endocrine System

Aldosterone
Antidiuretic hormone
Blood glucose
C-peptide
Calcium
Calcitonin
Glucose tolerance test (GTT)
Insulin
Parathormone (PTH)
Phosphorus
Thyroxine (T4)
Thyroid-stimulating hormone (TSH)
Triiodothyronine (T3)

Gastrointestinal System and Stool Tests

Bacterial overgrowth of small intestine breath test
Comprehensive digestive stool analysis
Gastric analysis
Lactose intolerance breath test
Parasitology assessment
Serum gastrin

Hepatic System

Albumin, gamma-globulin, A-G ratio
Alanine aminotransferase (ALT)
Aspartate aminotransferase (AST)
Alkaline phosphatase (ALP)
Ammonia
Bilirubin
Gamma-glutamyl transpeptidase (GGT)
Hepatitis virus
Prothrombin time (PT)
Partial thromboplastin time (PTT)
Total protein
Urobilinogen

Musculoskeletal System

Alkaline phosphatase (ALP)
Acid phosphatase
Creatine phosphokinase (CPK)
Enzymes, isoenzymes
Lactate dehydrogenase (LDH)

Pancreatic Tests

Serum amylase
Serum lipase

Red Cell Indices

Carbon dioxide
Mean cell volume (MCV)
Mean cell hemoglobin (MCH)
MCH concentration
Reticulocyte cell count
Serum ferritin
Serum iron and total iron-binding capacity
Sodium, potassium, chlorine

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Vitamin and Mineral Assessment

Calcium
Zinc
Vitamin A
Vitamin C
Vitamin B_6
Vitamin B_{12}
Folate

Urinary System

Physical observation: Color, clarity, odor
Microscopic examination: Cells, casts, crystals, bacteria, yeast
Urine volume
Chemical tests: Specific gravity, pH, protein, glucose, ketones, blood, bilirubin, urobilinogen, nitrite, leukocyte esterase

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Hematology: Coagulation Tests and Bleeding Time				
Prothrombin time (PT), international normalized ratio (INR)	11–16 seconds control; 70–110% of control value Patients on anticoagulant drugs should have an INR of 2.0–3.0 for basic "blood thinning" needs. For some patients who have a high risk of clot formation, the INR needs to be higher—about 2.5–3.5.	Since PT and INR evaluate the ability of blood to clot properly, they can be used to assess both bleeding and clotting tendencies. One common use is to monitor the effectiveness of blood thinning drugs such as warfarin (Coumadin). Anticoagulant drugs must be carefully monitored to maintain a balance between preventing clots and causing excessive bleeding.	A prolonged or increased prothrombin time means that blood is taking too long to form a clot. Antibiotics, aspirin, and cimetidine can increase the PT/INR.	Too much anticoagulation (warfarin, for example). Barbiturates, oral contraceptives, hormone-replacement therapy (HRT), and vitamin K (either in a multivitamin or liquid nutrition supplement) can decrease PT. Beef and pork liver, green tea, broccoli, chickpeas, kale, turnip greens, and soybean products contain large amounts of vitamin K and can alter PT results if consumed in large amounts.
Hematology: Blood Cell Values				
Erythrocyte count (red blood cells [RBC])	4.5–6.2 million/mm^3 in males; 4.2–5.4 million/mm^3 in females	Made in bone marrow; production controlled by erythropoietin.	Polycythemia, dehydration, severe diarrhea	Anemias such as vitamin B_{12}, iron, folic acid, and protein; chronic infections; hemorrhage
Erythrocyte sedimentation rate	0–15 mm/hour in males; 0–25 mm/hour in females	Sedimentation rate measures how quickly RBCs (erythrocytes) settle in a test tube in 1 hour. Often used with C-reactive protein (CRP) for testing of inflammation.	Inflammation, pneumonia, appendicitis, pelvic inflammatory disease, lymphoma, multiple myeloma, lupus, rheumatoid arthritis (RA), osteomyelitis, temporal arteritis	
Ferritin	20–300 mg/mL in males; 20–120 mg/mL in females	Chief iron storage protein in the body. Reflects reticuloendothelial iron storage. Usually 23% of total iron stores.	Hemochromatosis, leukemias, anemias other than iron deficiency, Hodgkin's disease, liver diseases	Iron deficiency anemia
Folate, serum	0.3 μg/dL (7 nmol/L)	Important for DNA functioning	Blind loop syndrome	Pregnancy, lactation, macrocytic anemia, intestinal malabsorption syndrome, alcoholism, use of goat's milk in childhood, use of anticonvulsants, cycloserine, methotrexate, and oral contraceptives

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Iron, serum	75–175 mg/dL in males; 65–165 mg/dL in females; 40–100+ mg/dL in children up to 2 years old; 50–120 mg/dL in children >2 years.	Iron found in blood, largely in hemoglobin	Hemochromatosis, acute or chronic liver disease, hemolytic anemia, leukemia, lead poisoning, thalassemia, vitamin B_{12} or folate deficiency, dehydration, small bowel surgery	50 μg/dL (9 mmol/L) may indicate iron deficiency anemia or blood loss. Chronic diseases, pregnancy, cancer, end-stage renal disease (ESRD) or chronic renal disease, malnutrition or poor dietary intake, sickle cell disease, viral hepatitis, thalassemia
Iron-binding capacity, total (TIBC)	240–450 mg/dL	Measure of the capacity of serum transferrin. 18–59% transferrin saturation is normal. As serum iron decreases, TIBC goes up, at least initially.	Iron deficiency, blood loss, later in pregnancy, oral contraceptive use, hepatitis, low serum iron, gastrectomy	High serum iron, renal disease, hemochromatosis, pernicious anemia, cancer, thalassemia, hemolytic diseases, small bowel surgery, sepsis, liver disease; may be low in malnutrition
Hematocrit (% packed cell volume)	40–54% in males; 37–47% in females; 33–35% in children; 37–49% in 12- to 18-year-old males; 36–46% in 12- to 18-year-old females	Cell volume % of RBCs in whole blood. Elevated when iron level is low.	Polycythemia	Anemias, prolonged dietary deficiency of protein and iron, sepsis, small bowel surgery, gastrectomy, renal or liver disease, blood loss
Hemoglobin, whole blood	14–17 g/dL in males; 12–15 g/dL in females; ≥10 g/dL in babies 6–23 months old; ≥11 in children 2–5 years old; 11.5–15.5 g/dL in children 6–12 years old; 13.0–16.0 g/dL in 12- to 18-year-old males; 12.0–16.0 g/dL in 12- to 18-year-old females	Oxygen carrier from lung to tissues. Binds CO_2 on return to the lung.	Polycythemia, dehydration, hemolysis, sickle cell anemia, recent blood transfusions, chronic obstructive pulmonary disease (COPD), heart failure, high altitude, burns, dehydration	Anemias, prolonged dietary deficiency of iron, excessive bleeding, cancer, lupus, overhydration, Hodgkin's disease, malnutrition, renal or liver disease, pregnancy, lead poisoning, sepsis, small bowel surgery, gastrectomy
Mean corpuscular hemoglobin (MCH)	26–32 pg; concentration is 32–36%	High concentration of individual RBCs (Hgb/RBC)	Macrocytic anemia	Hemoglobin deficiency, hypochromic anemia
Mean corpuscular volume (MCV)	80–94 cu/μm	Individual RBC size	High MCV or macrocytosis: folate, vitamin B_{12} deficiency. Alcoholic or liver disease, blood loss, hypothyroidism, small bowel surgery	Low MCV or microcytosis: iron, copper, pyridoxine deficiency. Thalassemia, anemia of chronic disease, cancer, blood loss
Transferrin (siderophilin)	170–370 mg/dL (1.7–3.7 g/L)	Glycoprotein in blood plasma that transports iron to liver and spleen for storage and to bone marrow for hemoglobin synthesis	Iron deficiency, acute hepatitis, oral contraceptive use, pregnancy, chronic blood loss, dehydration, gastrectomy	Acute or chronic inflammation, chronic liver disease, lupus, zinc deficiency, sickle cell anemia, pernicious anemia with vitamin B_{12} or folate deficiency, chronic infection and inflammatory diseases, malnutrition, burns, iron overload, nephrotic syndrome, sepsis, small bowel surgery

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
White blood cells (WBC), leukocytes	4.8–11.8 thousand/mm^3	Highly variable lab value; protect against disease or infection	Metabolic acidosis, acute hemorrhage, acute bacterial infections, leukemias, burns, gangrene, exercise, stress, eclampsia	Chemotherapy, ABT. Neutropenia is often seen in deficiency states. Some viral conditions, cachexia, anaphylactic shock, bone marrow suppression, pernicious or aplastic anemia, diuretic use
Hematology and Lymphatic System: Differential				
Lymphocytes	Total lymphocyte count (TLC) = normal, 12,000/mm^3; deficient, $<$900/mm^3; 24–44% total WBC count	Made in thymus and lymph nodes; produce antibodies	Infectious mononucleosis, mumps, German measles, convalescence from acute infections	Infections, malnutrition
Leukocytes: basophils	0–1.5% total WBC count	They release substances that cause smooth muscle contraction, vasoconstriction, and an increased permeability of small blood vessels. Basophils are stimulated by allergens.	Postsplenectomy, chronic myelogenous leukemia, polycythemia, Hodgkin's disease, chicken pox	Hyperthyroidism, acute infections,pregnancy, anaphylaxis
Leukocytes: eosinophils	0.5–4% total WBC count	Eosinophils are stimulated by parasites and some bacteria. They release substances that cause vasoconstriction, smooth muscle contraction, and an increased permeability of small blood vessels. Related to allergies.	Allergies, parasitic infections, pernicious anemia, ulcerative colitis, Hodgkin's disease	Use of β-blockers, corticosteroids; stress, and bacterial and viral infections including trichinosis
Leukocytes: monocytes	4–8% total WBC count	Phagocytosis. Produced in bone marrow.	Monocytic leukemia, lipid storage disease, protozoan infection, chronic ulcerative colitis	
Leukocytes: neutrophils	60–65% total WBC count	Phagocytosis	Wound sepsis in burns, bacterial infections, inflammation, cancer, traumas, stress, diabetes, acute gout	Folate or vitamin B_{12} deficiency, sickle cell anemia, steroid therapy, postsurgical status
Platelets (thrombocytes)	125,000–300,000 mm^3	Largely polysaccharides and phospholipids. Role in coagulation.	Malignancy, polycythemia vera, splenectomy, iron deficiency anemia, cirrhosis, chronic pancreatitis	Thrombocytopenic purpura
General Serum Values and Enzymes				
Amylase	60–180 Somogyi units/dL	Pancreatic enzyme for hydrolysis of starch and glycogen	Increased in perforated peptic ulcer, acute pancreatitis, mumps, cholecystitis, renal insufficiency, alcohol poisoning, partial gastrectomy	Decreased in hepatitis, severe burns, pancreatic disease, advanced cystic fibrosis, toxemia of pregnancy, hepatitis
Bicarbonate	22–28 mEq/L	Acid–base balance	Metabolic alkalosis, large intake of sodium bicarbonate, excessive vomiting, potassium deficiency, respiratory acidosis, emphysema	Diabetic acidosis, starvation, chronic diarrhea, renal insufficiency, respiratory alkalosis (hyperventilation), fistula drainage

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Bilirubin	Direct: ≤0.3 mg/dL; total: ≤1 mg/dL	Hemoglobin is converted to bilirubin when RBCs are destroyed.	Hepatitis, jaundice, biliary obstruction, drug toxicity, hemolytic disease, prolonged fasting	Seasonal affective disorder
Calcium	9–11 mg/dL (2.3–2.8 mmol/L); 3.9–5.4 mg/dL serum ionized	50% is protein bound, so protein intake affects serum calcium level more than dietary calcium. Regulated by parathormone (PTH) and thyrocalcitonin. Coma above 13 mg/dL; death below 7 mg/dL.	Hyperparathyroidism, renal calculi, vitamin D excess, osteolytic disease, milk-alkali syndrome, immobilization, tuberculosis, Addison's disease, antibiotic therapy, cancer	Steatorrhea, renal failure, malabsorption, vitamin D deficiency, hypoparathyroidism, sprue, celiac disease, overhydration, hypoalbumenia, hyperphosphatemia
Ceruloplasmin	27–37 mg/dL	Form of copper found in the bloodstream	Leukemias, anemias, cirrhosis of the liver, hypo/hyperthyroidism, collagen diseases, pregnancy	Wilson's disease, severe copper deficiency, nephrosis, leukemia remission, prolonged total parenteral nutrition (TPN) without copper, cystic fibrosis
Copper	70–140 μg/dL in males; 80–155 μg/dL in females	Found bound to albumin or as ceruloplasmin	Leukemias, anemias, cirrhosis of the liver, hypo/hyperthyroidism, collagen diseases, pregnancy	Wilson's disease, severe copper deficiency, nephrosis, leukemia remission, prolonged TPN without copper, cystic fibrosis
Creatine phosphokinase (CPK)	0–145 IU/L	Catalyst for phosphorylation of creatine by adenosine triphosphate (ATP). Mostly found in skeletal and cardiac muscle. Increases 2–4 hours after myocardial infarction (MI), returning to normal after 3 days.	Hepatic or uremic coma, striated muscle disease, muscular dystrophy, MI, cerebrovascular accident (CVA), trauma, alcoholic liver disease, encephalitis	
D-xylose, 25-g dose	30–40 mg/dL	This test measures the intestines' ability to absorb D-xylose—a simple sugar—as an indicator of whether nutrients are being properly absorbed.		Xylose malabsorption
Gamma-glutamyl transpeptidase (GGT)	5–40 IU/L	GGT participates in the transfer of amino acids across the cellular membrane and in glutathione metabolism. Used to detect diseases of the liver, bile ducts, and kidney and to differentiate liver or bile duct (hepatobiliary) disorders from bone disease.	Coronary heart failure (CHF), MI, cholecystitis, liver disease, alcoholism, hepatic biliary disease, pancreatitis, nephritic syndrome	
Lactic acid dehydrogenase (LDH)	200–680 IU/mL	Catalyzes conversion between pyruvate and lactic acid in glycolytic cycle. Has 5 isoenzymes. Increases 8–10 hours after MI, returning to normal after 7–14 days.	Untreated pernicious anemia, acute MI, heart failure, malignancy, alcoholic liver damage, cardiovascular surgery, hepatitis, pulmonary embolus, leukemia, cancer, renal failure, hemolytic or megaloblastic anemia, muscular dystrophy, nephrotic syndrome	Radiation therapy
Lipase	0.2–1.5 IU/mL	Synthesized by the pancreas	Pancreatic disease, acute pancreatitis, perforated ulcer, pancreatic duct obstruction, biliary tract infection, renal insufficiency	

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Phenylalanine, serum	<3 mg/dL	An amino acid of importance in phenylketonuria (PKU) patients	Levels might be high in PKU	Severe protein deficiency and malnutrition
Phosphatase, acid	0.5–2 Bodansky units	A blood test that measures prostatic acid phosphatase (an enzyme found primarily in men in the prostate gland and semen) to determine the health of the prostate gland	Prostate dysfunction results in the release of prostatic acid phosphatase into the blood.	
Phosphatase, alkaline (ALP)	2–4.5 Bodansky units (30–135 IU/L); children = 3× that of adults	ALP is an enzyme found in all tissues. Tissues with particularly high concentrations of ALP include the liver, bile ducts, placenta, and bone. Indirect test for calcium, phosphorus, vitamin D nutriture. Used to determine the presence of liver or bone cell disorders.	Anemia, Paget's disease, biliary obstruction, leukemia, hyperparathyroidism, rickets, bone disease, healing fracture	Malnutrition, protein deficiency
Sulfate, inorganic	0.5–1.5 mg/dL	Usual sulfur intake is 0.6–1.6 grams on a mixed diet containing 100 grams of protein.	Supplemental overdose; high sulfate intake from water supply	Protein deficiency; injury to the bones, cartilage, or tendons
Transaminase: aspartate aminotransferase (AST)	5–40 IU/mL	Found in the liver, muscle, and the brain. Functional measure of vitamin B_6 nutriture. Released in tissue injury. Formerly SGOT (serum glutamic oxaloacetic transaminase).	Hepatic cancer, shock/trauma, cirrhosis, neoplastic disease, MI	Uncontrolled diabetes mellitus; Beriberi
Transaminase: alanine aminotransferase (ALT)	4–36 IU/L	Found in the liver. Generally parallels AST levels. Functional measure of vitamin B_6 nutriture. Formerly SGPT (serum glutamic pyruvic transaminase).	Hepatitis, cirrhosis, trauma, hepatic cancer, shock, mononucleosis	Wilson's disease
Glucose Control				
Glucose, fasting serum	70–110 mg/dL (3.9–6.1 mmol/L)	Principle fuel for cellular function, particularly for the brain and RBCs.	DM, hyperthyroidism pancreatitis, MI, hyperfunction of endocrine, stress, CHF infection, surgery, CVA, hepatic dysfunction, cancer, Cushing's syndrome, burns, steroids, chromium deficiency	Postprandial hypoglycemia, sepsis, cancer, malnutrition, hypothyroid, gastrectomy, liver damage or disease, Addison's disease
Glucose tolerance test (GTT)	2-hour post load glucose <200 mg/dL	Test done when blood glucose levels are >120 mg/dL.	Diabetes, hyperthyroidism, pancreatic cancer, Cushing's syndrome, acromegaly, pheochromocytoma.	Hypoglycemia, hypothyroidism, malabsorption, malnutrition, insulinoma, hypopituitarism
Glycosylated hemoglobin–HbA1 C	4–7% (nondiabetic); good, <9%; fair, 9–12%; poor, >12%	May reflect poor glucose control over the past 2–4 months	Poor blood sugar control, newly diagnosed diabetes, pregnancy	Low RBC (chronic blood loss, hemolytic anemia), chronic renal failure (CRF), sickle cell anemia
Protein Factors				
Albumin	3.5–5 g/dL (35–50 g/L); 3.2–5.1 g/dL in infants up to 1 year old; 3.2–5.7 in children aged 1–2 years; 3.5–5.8 for ages 2 years to adult. Albumin is usually 40% of proteins.	Nonspecific protein nutriture measure. Mostly synthesized in the liver. Transports fatty acids, thyroxin, bilirubin, and many drugs. Albumin–globulin ratio (A:G) is 1.2–1.9; low in renal or liver diseases.	Dehydration, multiple myeloma	Decreased with inflammation and severity of illness, acute stress, starvation, malabsorption of protein, cirrhosis, nephritis with edema, liver disease, malnutrition

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
C-reactive protein (CRP)	0	Elevated in inflammation	Systemic inflammation, obesity, diabetes, heart disease, arthritis, smoking	Vitamin C supplements may lower an elevated CRP. Marathon runners may have very low levels.
Creatinine	0.6–1.2 mg/dL	A basic creatine anhydride, nitrogenous end product of skeletal muscle metabolism. Indirect measure of renal filtration rate.	Hyperthyroidism, CHF, diabetic acidosis, dehydration, muscle disease, some cancers, nephritis, urinary obstruction	Overhydration, multiple sclerosis (MS), pregnancy, eclampsia, increased age, severe wasting
Creatinine–height index (CHI)	CHI = (measured 24-hour creatine excretion × 100)/predicted 24-hour creatine excretion	Estimates skeletal muscle mass. CHI values of 60–80% represent mild marasmus. CHIs of 40–59% represent moderate marasmus. CHIs of <40% represent severe marasmus.	High doses of creatine supplementation	Marasmus, malnutrition
Globulin	2.3–3.5 g/dL	5 fractions; transport antibodies	Infections, leukemia, dehydration, shock, tuberculosis (TB), chronic alcoholism, Hodgkin's disease	Malnutrition, immunological deficiency
Nitrogen balance	Goal of 1–4 g/24 hours urinary urea nitrogen (UUN)	Nitrogen balance is the result of nitrogen intake (from the diet) and nitrogen losses, which consist of nitrogen recovered in urine and feces and miscellaneous losses.	Severe liver disease	Nephrosis
Prealbumin (transthyretin)	16–35 mg/dL	Reflects the past 3 days of protein intake but is affected by inflammation	Renal failure, Hodgkin's disease, pregnancy, dehydration	Acute catabolic states, hepatic disease, stress infection, surgery, low protein intake, malnutrition, hyperthyroidism, nephritic syndrome, overhydration, inflammation
Proteins, total serum	6–8 g/dL; 5.6–7.2 in infants to 1 year; 5.4–7.5 in children 1–2 years old; 5.3–8.0 in ages 2 years to adult	Amount of protein in the bloodstream. Reflects depletion of tissue proteins. Act as buffers in acid–base balance. Plasma proteins equal approximately 7% of total plasma volume.	Dehydration, shock	Hepatic disease, leukemia, malnutrition, infection, pregnancy, malabsorption, severe burns
Nutrient Values				
Ascorbic acid (vitamin C)	0.2–2.0 mg/dL	Serum levels of vitamin C	Oxalate stones, diarrhea, high uric acid	Large doses of aspirin, barbiturates, stress, anemia, tetracycline, oral contraceptives, cigarette smoking, alcoholism, dialysis
Essential fatty acids (EFA) (triene to tetraene [T/T] ratio)	0.2 is normal; a ratio >0.4 in serum phospholipids indicates EFA deficiency in healthy people	Ratio 20:39/20:46 (T/T ratio) is measured. T/T ratios are assessed in RBCs, RBC phospholipids, and serum phospholipids.		EFA deficiency occurs in starvation, marasmus, and strict low-fat diets.
Niacin: *N*-methylnicotinamide	5.8 μmol/d	For niacin assessment. Usual dietary intake is 16–33 mg/d.	Large doses of nicotinic acid	Pregnancy, lactation, Hartnup disease, pellagra, schizophrenia
Riboflavin: glutathione reductase, erythrocyte	1.2 IU/g hemoglobin	Dietary intake is related to CHO and total energy intake.		Tetracycline or thiazide diuretic use, probenecid, oral contraceptives, sulfa drugs, pellagra, cheilosis, some forms of glossitis, vegetarians using no dairy products or supplements

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Selenium: glutathione peroxidase	Serum enzyme levels	Works with vitamin E to protect RBCs as part of the enzyme.	Excessive lipid peroxidation	PCM, hypoproteinemia
Thiamin: transketolase activity, erythrocyte	1.20 μg/mL/hr	Dietary requirements increase when CHO intake is high.		Use of baking soda in cooking, high CHO intake, infantile beriberi, dry or edematous (cardiac) beriberi, excessive intake of caffeic or tannic acids, pellagra, chronic alcoholism, lactic acidosis in metabolic disorders such as maple syrup urine disease (MSUD)
Vitamin B_6: pyridoxal 5′—phosphate, plasma	5 ng/mL (20 nmol/L)	Usual dietary intake is 2 milligrams, assuming 100 grams of protein is consumed.		Acute celiac disease, chronic alcoholism, pellagra, high protein intake, pregnancy, oral contraceptive use, isoniazid (INH) and other TB drug use, use of anticonvulsants, malarial drugs
Vitamin A: carotene; retinol, serum	48–200 mg/dL; 10–60 μg/dL	Serum levels of carotenoids	Excessive intake, such as from carrots; postprandial hyperlipidemia; diabetes; hypothyroidism	High fever, liver disease, malabsorption syndrome
Retinol-binding protein	2.6–7.6 mg/dL	Used less often to determine status of retinol	May be high in renal failure	Protein deficiency
Serum vitamin A	125–150 IU/dL or 20–80 mg/dL		Hypervitaminosis A	Cirrhosis, infectious hepatitis, myxedema, night blindness, malabsorption, starvation, infections such as measles
Vitamin B_{12}: serum B_{12}	24.4–100 ng/dL (180 pmol/L)	Vitamin B_{12} is less well absorbed in the elderly and in persons who have less intrinsic factor or the ability to reabsorb in the intestines.	Leukemia such as acute myelogenous leukemia (AML) or CML, leukocytosis, polycythemia vera, liver metastasis, hepatitis, cirrhosis	Macrocytic anemia, iron or vitamin B_6 deficiency, gastritis, dialysis, congenital intrinsic factor deficiency, pernicious anemia, tapeworm, ileal resection, strict vegetarian diets, pregnancy
Vitamin D: 1,25-HCC, blood	0.7–3.3 IU/dL	Indirect measures are available from serum alkaline phosphatase, calcium levels, and serum phosphorus.	Hypervitaminosis D	Rickets; osteomalacia; steroid therapy; fracture; poorly calcified teeth; drug therapy such as anticonvulsants, cholestyramine, and barbiturates
Vitamin E: alpha-tocopherol; plasma vitamin E	<18 μmol/g (41.8 μmol/L); 0.5–2.0 mg/dL	Usual dietary intake is 14 milligrams of d-alpha-tocopherol	High vitamin E intake (this affects coagulation because it works against vitamin K)	High polyunsaturated fatty acid (PUFA) intake, premature infants, cystic fibrosis, hemolytic anemia
Vitamin K: INR or PT	PT, 10–15 seconds	Combined low levels of prothrombin activity, serum calcium, and serum carotene may indicate abnormal fat and fat-soluble vitamin absorption.	Menadione use, intravenous (IV) administration of vitamin K, parenchymal liver disease	PT is prolonged in salicylates, sulfa, and tetracycline use. Liver disease, fat malabsorption, prematurity, small bowel disorders.
Zinc: serum zinc	0.75–1.4 mg/mL or up to 79 μg/dL in plasma; 100–140 μg/dL in serum	Large amounts are found in liver, skeletal muscle, and bone. Part of the insulin molecule. Usual intake is 10–15 mg/d.	Eating foods stored in galvanized containers. Excesses from supplements.	Wounds, geophagia, high-calcium or high-phytate diets, growth, stress, skin lesions, poor taste or olfactory acuity, upper respiratory infections, MI, oral contraceptive use, cancer, pregnancy, cirrhosis, sickle cell or pernicious anemias

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Electrolytes				
Chloride	95–105 mEq/L	Acid–base balance. Major anion. Follows sodium passively in its transport.	Eclampsia, metabolic acidosis, dehydration, pancreatitis, anemia, renal insufficiency, Cushing's syndrome, head injury, hyperventilation, hyperlipidemia, hypoproteinemia	Diabetic acidosis, metabolic alkalosis, gastroenteritis, fever, potassium deficiency, excessive sweating, heart failure, hyponatremia, infection, diuretics, overhydration
Magnesium	1.8–3 mg/dL	Influences muscular activity. Coenzyme in CHO and protein metabolism.	Renal insufficiency, uncontrolled diabetes, Addison's disease, hypothyroidism, ingestion of magnesium-containing antacids or salts	Malnutrition, potassium-depleting diuretics, malabsorption, alcohol abuse, starvation, renal disease, acute pancreatitis, severe diarrhea, ulcerative colitis
Phosphorous phosphate (PO_4)	2.3–4.7 mg/dL	Influenced by diet and absorption; regulated by kidneys.	Liver disease, bone tumors, hypervitaminosis D, end-stage renal disease, renal insufficiency, hypoparathyroidism, diabetic ketoacidosis (DKA), Addison's disease, childhood	Malnutrition, gout, hyperparathyroidism, osteomalacia, hyperinsulinism, hypovitaminosis D, alcoholism, overuse of phosphate-binding antacids, rapid refeeding after prolonged starvation
Potassium, serum	3.5–5.5 mEq/L (16–20 mg/dL)	Intracellular. Cellular metabolism; muscle protein synthesis. Enzymes.	Renal insufficiency or failure, overuse of potassium supplements, Addison's disease, dehydration, acidosis, cell damage, poorly controlled diabetes	Decreased potassium intake, renal disease, burns, trauma, diuretics, steroids, vomiting, stress, diarrhea, crash diet, overhydration, malnutrition, estrogen, steroid therapy, cirrhosis, hemolysis, fistula drainage
Sodium, serum	136–145 mEq/L	Absorbed almost 100% from gastrointestinal (GI) tract. Major cation; extracellular ion. Controls osmotic pressure. Acid–base balance.	Vitamin K deficiency, vomiting, heart failure, hypervitaminosis, dehydration, diabetes insipidus, Cushing's disease, primary aldosteronism, diarrhea, steroids	Decreased sodium intake, diuretic use, burns, diarrhea, vomiting, nephritis, diabetic acidosis, hyperglycemia, overhydration
Lipids				
Total serum cholesterol (TC)	Adults: Desirable: 120–199 mg/dL; borderline high: 200–239 mg/dL; high: ≥240 mg/dL Child: Desirable: 70–175 mg/dL; borderline: 170–199 mg/dL; high: ≥200 mg/dL	Fat-related compound; component of plaque. Need fasting sample. Usually 30% high-density lipoprotein (HDL) and 70% low-density lipoprotein (LDL).	High: >200 mg/dL. Hyperlipidemia, diabetes, MI, hypertension (HTN), high-cholesterol diet, nephrotic syndrome, hypothyroidism, pregnancy, cardiovascular disease (CVD)	Low: <160 mg/dL. Low dietary fat ingestion, malnutrition, malabsorption or starvation, fever, acute infections, liver damage, steatorrhea, hyperthyroidism, cancer, pernicious anemia
High-density lipoprotein (HDL)	Males: >45 mg/dL; females: >55 mg/dL	Usually higher in females. Low levels may indicate risk for heart disease.	Vigorous exercise, weight reduction, liver disease, alcoholism	Starvation, obesity, liver disease, DM, smoking, hyperthyroidism
Low-density lipoprotein (LDL)	Desirable: <130 mg/dL; borderline high: 130–159 mg/dL; high: ≥160 mg/dL	May indicate cardiac risk when elevated.	Familial hyperlipidemia, diet high in saturated fat and cholesterol, hypothyroidism, MI, DM, nephrotic syndrome, pregnancy, hepatic disease	Nephritic syndrome

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Phospholipids	60–350 mg/dL	Measure of EFAs, indirect. Includes lecithin, sphingomyelins, cephalins, and plasmogens.		EFA deficiency
Triglycerides (TG)	Desirable: 10–190 mg/dL; borderline high: 200–400 mg/dL; high: 400–1000 mg/dL; very high: >1000 mg/dL	Neutral fats are the main transport form of fatty acids. Need fasting sample.	High-carbohydrate diet, alcohol abuse (secondary), hyperlipoproteinemias, nephrotic syndrome, CRF, MI, HTN, DM, respiratory distress, pancreatitis, hypothyroidism	Malnutrition, COPD, malabsorption, hyperthyroidism, hyperparathyroidism, brain infarct
Osmolality and pH				
pH, arterial, plasma	7.36–7.44	Hydrogen ion concentration. Enzymes work within narrow pH ranges.	Alkalosis (uncompensated), hyperventilation, pyloric obstruction, HCl losses, diuretic use	Acidosis (uncompensated), emphysema, diabetic acidosis, renal failure, vomiting, diarrhea, intestinal fistula
Osmolality, serum	270–280 mOsm/L	Maintaining normal serum level is desirable.	Elevated in dehydration	Low in overhydration
Respiratory Factors				
Respiratory quotient (RQ)	0.85 from mixed diet	1.0 from CHO; 0.80 from protein; 0.70 from fat	High-CHO diet	High-fat diet
Oxygen: partial pressure of oxygen (pO_2)	80–100 mm Hg	Reflects hemoglobin concentration. Arterial blood is used.	Hyperoxia	Hypoxia (anemic, stagnant, chronic, anoxic)
Carbon dioxide: partial pressure of carbon dioxide (pCO_2)	35–45 mm Hg; 24–30 mEq/L		Pulmonary problems, metabolic alkalosis due to ingestion of excess sodium bicarbonate, protracted vomiting with potassium deficiency, Cushing's syndrome, heart failure with edema	Diabetic ketosis or ketoacidosis, starvation, renal insufficiency, persistent diarrhea, lactic acidosis, respiratory alkalosis, diarrhea
Renal Values				
Glomerular filtration rate (GFR)	110–150 mL/min in males; 105–132 mL/min in females	GFR reflects kidney function		Renal failure: <90 mL/min/1.73 m^2 indicates renal decline
Urea clearance	40–65 mL/min standard; 60–100 mL/min maximum	Part of the assessment of renal status		Uremia
Blood urea nitrogen (BUN) to creatinine ratio	>10:1	Measure of impaired renal function.	Renal disease, excess protein intake, bleeding in small intestine, burns, high fever, steroid therapy, decreased renal blood flow, urinary tract obstruction	Low-protein intake, repeated dialysis without repletion, severe vomiting and diarrhea, hepatic insufficiency
BUN	8–18 mg/dL (3–6.5 mmol/L); slightly higher in males	Urea is the end product of protein metabolism. Varies directly with dietary intake. Formed in the liver from amino acids and other ammonia-containing compounds.	Renal failure, azotemia, DM, burns, dehydration, shock, heart failure, infection, chronic gout, excessive protein intake, catabolism, GI bleed, MI, urinary obstruction, starvation, steroid therapy, trauma	Hepatic failure, malnutrition, malabsorption, overhydration, pregnancy, acromegaly, low-protein diet

(continued)

TABLE B-8 Interpretation of Lab Values *(continued)*

Value	Normal Range	Purpose or Comment	Increased In	Decreased In
Uric acid	4.0–9.0 mg/dL in men; 2.8–8.8 mg/dL in women	Metabolite from purine metabolism. Excreted by kidney. Serum level reflects balance between production and excretion.	Higher in winter, stress, gout, leukemia, hypoparathyroidism, total fasting, toxemia of pregnancy, elevated triglycerides, DKA, hypertension, hemolytic or sickle cell anemia, renal disease, alcoholism, multiple myeloma, polycythemia, use of vincristine or mercaptopurine	Acute hepatitis, Wilson's disease, celiac disease, Fanconi's syndrome, Hodgkin's disease, use of allopurinol or large doses of Coumadin, folic acid anemia, burns, pregnancy, malabsorption, lead poisoning
Hormones				
Adrenocorticotropic hormone (ACTH)	5–95 pg/mL at 9 AM; 0–35 pg/mL at midnight		Cushing's syndrome, secondary hypoadrenalism	Pituitary, Cushing's syndrome, primary adrenal insufficiency
Cortisol	5–25 μg/dL at 8 AM; 10 μg/dL at 8 PM	Lower in women	Extreme stress, elevated in patients with night-eating syndrome	
Gastrin	0–200 pg/mL		Zollinger–Ellison syndrome, pyloric obstruction, short bowel syndrome, pernicious anemia, atrophic gastritis	
Growth hormone (GH)	<6 ng/mL in men; <10 ng/mL in women	Being administered in some medical conditions		
Insulin	6–26 IU/mL, fasting	Serum levels are useful.	Untreated obese patients who have diabetes, metabolic syndrome, insulinoma	Severe diabetic acidosis with ketosis and weight loss
Iodine, total	8–15 mEq/L	Affects the activity of the thyroid gland; usual intake with 10 grams of NaCl is 1000 micrograms of iodine.	Hyperthyroidism	Cretinism, simple goiter, low-iodine diet or high-goitrogenic dietary intake
Protein-bound iodine (PBI)	3.6–8.8 mg/dL	Most iodine is protein-bound in the thyroid hormones.	Hyperthyroidism, thyroiditis, pregnancy, oral contraceptive use, hepatitis	Cretinism, simple goiter, low-iodine diet or high-goitrogenic dietary intake
Serotonin (5-HIAA)	0.05–0.20 μg/mL	Neurotransmitter		Depression
Thyroxine (T4); triiodothyronine (T3)	T4: 4–12 μg/100 mL; T3: 75–95 μg/100 mL	Tests of thyroid function	Myasthenia gravis, nephrosis, pregnancy, preeclampsia, Graves' disease, hyperthyroidism	Increased thyroid-stimulating hormone (TSH), decreased T4, T3 (hypothyroidism); malnutrition; hypothyroidism; nephrosis; cirrhosis; Simmonds' disease
Thyroid-stimulating hormone (TSH)	≤0.2 μU/L	Thyroid function tests (TSH, T4, T3)	Primary untreated hypothyroidism, post subtotal thyroidectomy	Hyperthyroidism
Stool Values				
Fat, fecal	<7 g/24 hr		Fat malabsorption	
Nitrogen	<2.5 g/d			
Urinalysis	Normal is pale golden yellow	Abnormal color changes: orange = high level of bile; red = blood, porphyria, urates, or bile or ingestion of beets, blackberries, or food dyes; brown = blood; melanin may turn black on standing.	Dehydration: dark golden color	Overhydration: clear color (diluted)

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