Manual of Pediatric Nutrition

THIRD EDITION

Hendricks

Duggan

Walker

Contents

1

NUTRITIONAL ASSESSMENT

DIETARY EVALUATION

Kristy M. Hendricks, RD, MS, ScD

Nutritional assessment is the tool by which the nutritionist evaluates the patient for maintenance of normal growth and health, risk factors contributing to disease, and early detection and treatment of nutritional deficiencies and excesses. Comparison of an individual with an established norm provides a basis for objective recommendations and evaluation of nutrition therapy.[1,2] Although much information has been published on the use on increasingly sophisticated techniques, clinical judgment and perceptive history taking remain important overall components of nutritional assessment.[3] In children, this includes family history, developmental assessment, medical history including growth history, and physical examination including anthropometry. Nutritional assessment in children has special significance because undernutrition is the single most important cause of growth retardation.[4] Acute and chronic malnutrition remain common in hospitalized pediatric patients in the United States, underscoring the need for early detection and treatment of nutritional deficiency.[5] In addition, in the United States, overnutrition in the pediatric population has risen significantly,[6] and the association of obesity with chronic diseases in adulthood such as heart disease and diabetes is strong; thus, nutrition assessment is equally important for the early referral and treatment of nutrition excess.

A combination of anthropometric, biochemical, clinical, and dietary information forms the basis of evaluation. As no one parameter is completely satisfactory with regard to sensitivity and specificity, various tests monitor different aspects of nutritional status in each category. Standards that are relevant to a specific population are important, as are appropriate techniques and equipment for measurement. Throughout this manual, guidelines are provided to help determine where to begin the assessment of an individual (Table 1–1), what type of assessment is likely to yield valuable screening information, and how and when to proceed with more extensive and costly evaluation. An example of a worksheet for data collection and assessment on general pediatrics is included (Table 1–2).[7] Dietary insufficiency or excess generally precedes signs of biochemical, anthropometric, or clinical deficiency, and guidelines for dietary assessment are included in this chapter. The various indices of anthropometry, reference standards, techniques, interpretation, and classification of malnutrition are detailed in Chapter 2. Clinical evaluation is covered in Chapter 3. Biochemical parameters useful in nutritional assessment, are included in Chapter 4. This information provides guidelines related to basic nutritional assessment; recommendations for specific disease states and nutrition therapies are discussed throughout the manual. The following are excellent general reference sources for pediatric nutritional assessment: The Centers for Disease Control (Nutrition Division, Atlanta, Georgia) Anthropometric Software Package, which can be used to calculate height and weight percentile, Z score, and malnutrition category relative to the National Center for Health Statistics (NCHS) reference growth standards; the American Academy of Pediatrics *Pediatric Nutrition Handbook*;[8] and *Quality Assurance Criteria for Pediatric Nutrition Conditions*, prepared by Dietitians in Pediatric Practice Group and published by the American Dietetic Association, Chicago, Illinois.[9]

Table 1–1. An Approach to the Identification of Nutritional Problems

Screening	*Dietary*	*Clinical*	*Anthropometric*	*Biochemical*
Routine:				
To be done on all patients. If problems are indicated, additional midlevel or in-depth parameters should be evaluated	Typical dietary pattern (food pyramid/food frequency), vitamin and mineral supplement, family eating habits, subsidy support	Physical and dental history and examination, sexual maturation, use of medication(s)	Weight, length, head circumference, weight for height, and BMI	Hemoglobin, hematocrit, MCV, total cholesterol (LDL dependent on total cholesterol, see Table 29–2, page 436)
Midlevel, add:				
As indicated by routine screening or in populations at risk for chronic nutrition problems and children with special health care needs	24-hour recall and 3- to 7-day food records, developmental evaluation of feeding skills	More extensive examination (eg, skin, hair, nails)	Height and weight Z score, triceps skinfold, arm circumference, prediction of mature height	Albumin, total protein, total lymphocyte count
In-depth, add:				
As indicated in acute and chronic PCM and to monitor chronically ill patients	Same, observation in hospital	Bone mineralization (eg, epiphyseal enlargement, cranial bossing), bone age	Height velocity	Specific vitamin, mineral, and electrolyte levels or enzymes and proteins that require that nutrient; delayed cutaneous hypersensitivity (see Table 4–2, pages 71–6)

BMI = body mass index; MCV = mean corpuscular volume; LDL = low-density lipoprotein; PCM = protein calorie malnutrition.

Table 1–2. Pediatric Nutritional Assessment Data Sheet

Name _____

Date _____

Date of birth _____

History

Presenting problems _____

_____

_____

Growth history _____

_____

_____

Anthropometric Data

Weight _____ kg _____ percentile _____ % standard

Height _____ cm _____ percentile _____ % standard

Weight/height _____ percentile

BMI _____

Head circumference _____ cm _____ percentile

Skinfold thickness _____ mm _____ percentile

Arm circumference _____ cm _____ percentile

Biochemical Data

Hemoglobin _____ WBC _____

Hematocrit _____ TLC _____

MCV _____ Cholesterol _____

Albumin _____ LDL _____

Total protein _____

Clinical Data

Signs or symptoms of nutrient deficiencies or excess _____

_____

Classification of malnutrition _____

_____

Dietary Data

Estimated calorie intake from _____

Table 1–2. continued

_____ kcal/day _____ kcal/kg	
_____ g protein _____ protein/kg _____	_____% kcal
_____ g fat _____ % calories	
_____ g carbohydrate _____ % calories	

Vitamin/mineral supplement type and amount _____

Feeding skills and behavior appropriate for age: ☐ yes ☐ delayed

Use of: ☐ Food stamps ☐ WIC Other_____

Recommendations

Ideal weight for height _____ kg

Recommended _____ kcal/day

Recommended _____ protein/day

_____

_____

_____

Dietary inadequacy or excess is frequently the cause of under- or overnutrition and often precedes biochemical, anthropometric, or clinical signs; thus, evaluation of an individual's diet plays an important role in nutritional diagnosis and treatment. Quality and quantity of food intake and the macro and micronutritients provided can be measured using a variety of techniques. In addition, past dieting history, development of feeding skills, abnormal eating habits, difficulty in feeding, and activity level should be assessed.

A number of methods are available for the collection of information about food consumption.[10] Some are more appropriate for the assessment of population data on food intake. In the clinical setting, where individual information is important, more detailed and precise methods are generally used. The most common dietary assessment

tools in clinical practice are the 24-hour recall, 3- to 7-day food records, or "usual patterns" described by the patient or caregiver. A complete dietary history combines a number of methods with the gathering of medical and clinical information relative to dietary assessment.

Each method has certain weaknesses and limitations, and difficulty in quantifying and qualifying actual intake is well documented.[11-14] Patients of normal weight give the most accurate record whereas underweight patients overestimate and overweight patients underestimate actual food consumed. Similarly, assessments of dietary intake over long periods tend to overestimate actual intake, and those covering a short period tend to underestimate intake. Because of considerable differences in nutrient intake data obtained by different techniques, variability of intake from day to day, and difficulty in obtaining information on children by different care providers, it is helpful in some cases to use a combination of methods (24-hour recall with 3-day food records) to provide a more complete and accurate dietary evaluation. Emphasis should be placed on careful questioning and detailed recording of intake.

Additional limitations to the accurate assessment of intake include a wide variety of food composition tables and computerized databases for analysis and difficulty in establishing actual nutrient needs.[2,11]

References

1. Blackburn GL, Bistrian BR, Maini BS, et al. Nutritional and metabolic assessment of the hospitalized patient. J Pediatr Endocrinol Metab 1977;1:11.
2. Lee RD, Nieman DC. Introduction to nutritional assessment. In: Nutritional assessment. 2nd ed. St Louis: Mosby; 1996.
3. Baker JP, Detsky AS, Wesson DE, et al. Nutritional assessment: comparison of clinical judgment and objective measurements. N Engl J Med 1982;306:969.

4. Duggan C. Failure to thrive: management in the pediatric outpatient setting. In: Walker WA, Watkins JB, editors. Nutrition in pediatrics. Toronto: BC Decker; 1996:705–15.
5. Hendricks KM, Duggan C, Gallagher L, et al. Malnutrition in hospitalized pediatric patients: current prevalence. Arch Pediatr Adolesc Med 1995;149:1118–22.
6. Interagency Board for Nutrition Monitoring and Related Research. Third report on nutrition monitoring in the United States: executive summary. Washington (DC): US Government Printing Office; 1995:159–97.
7. Laramee SH, Hendricks KM. Development and use of pediatric nutrition and metabolic worksheet. Abstracts of the American Dietetic Association 1979.
8. American Academy of Pediatrics Committee on Nutrition. Pediatric nutrition handbook. 4th ed. Kleinman RE, editor. Elk Grove (IL): American Academy of Pediatrics; 1998.
9. Quality Assurance Committee, Pediatric Nutrition Practice Group. Quality assurance criteria for pediatric nutrition conditions: a model. Chicago (IL): American Dietetic Association; 1993.
10. Lee RD, Nieman DC. Nutritional assessment. 2nd ed. St Louis: Mosby; 1996:91–145.
11. National Research Council, Food and Nutrition Board. Recommended dietary allowance. Washington (DC): National Academy of Sciences; 1989.
12. Carter RL, Sharbaugh CO, Stapell CA. Reliability and validity of the 24-hour recall. J Am Diet Assoc 1981;79:542.
13. Karvetti RL, Knuts LR. Agreement between dietary interviews. J Am Diet Assoc 1981;79:654.
14. Stunkard AJ, Waxman M. Accuracy of self-reports of food intake. J Am Diet Assoc 1981;79:547.

2

NUTRITIONAL ASSESSMENT

ANTHROPOMETRICS AND GROWTH

Kristy M. Hendricks, RD, MS, ScD

Anthropometric Evaluation

Physical growth is, from conception to maturity, a complex process influenced by environmental, genetic, and nutritional factors. Anthropometry is the measurement of physical dimensions of the human body at different ages. Comparison with standard references for age and sex helps determine abnormalities in growth and development that may have resulted from nutrient deficiencies or excesses. Reference standards (included here) are derived from measurements of a normal population. Revised standards are expected to be available soon. Repeated measurements of an individual over time provide objective data on nutrition, health, and well-being. Errors in the comparison of measurements taken at different times can be caused by poor technique and equipment. Detailed descriptions of standardized techniques and equipment can be found in other sources.[1,2]

Weight

Body weight is a reproducible growth parameter and a good index of acute and chronic nutritional status. An accurate age, sex, and reference standard is necessary for evaluation. Weight is evaluated in three ways: weight for age, weight for height, and body mass index (BMI). Weight for age compares the individual to reference data for weight

attained at any given age whereas weight for height looks at the appropriateness of the individual's weight compared to his or her own height. For example, an infant may be at the 95th percentile weight for age but at the 50th percentile weight for height, indicating appropriate weight.

Standards. Figures 2–1 to 2–4 are National Center for Health Statistics (NCHS) growth charts.[3] All measure-

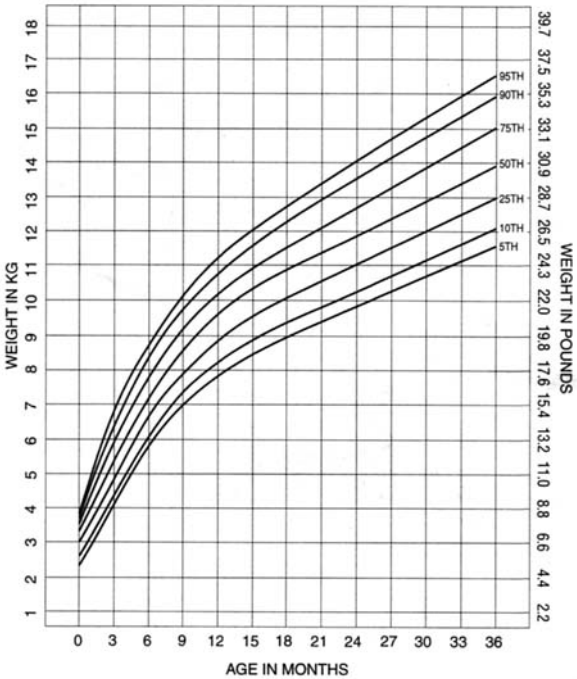


Figure 2–1. Weight by age percentiles for girls (birth to 36 months). (Figures 2–1 to 2–12 are reproduced with permission from Hamil PVV, Drizd TA, Johnson CL, et al. Physical growth: National Center for Health Statistics percentiles. Am J Clin Nutr 1979;32:607–29.)

ments were done between 1962 and 1974 by the US Public Health Service on large samples of children throughout the United States. These data represent the most comprehensive measurements available for comparison.

Interpretation. Weight below the 10th percentile or above the 90th percentile may indicate weight deficit or excess, respectively. Weight can be calculated as a percentage of standard weight (the 50th percentile for age and sex) as follows:

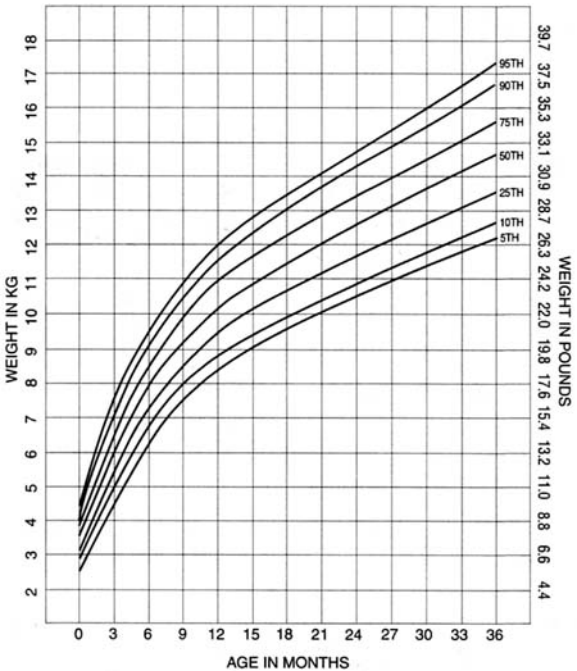


Figure 2–2. Weight by age percentiles for boys (birth to 36 months).

$$\% \text{ standard} = \frac{\text{actual weight}}{\text{standard weight}} \times 100$$

> 120% standard = excess
80 to 90% standard = marginal deficiency
60 to 80% standard = moderate deficiency
< 60% standard = severe deficiency

Recent change in weight (loss or gain) is also important to note as it is often an indicator of acute nutritional problems. A weight loss greater than 5 percent in 1 month

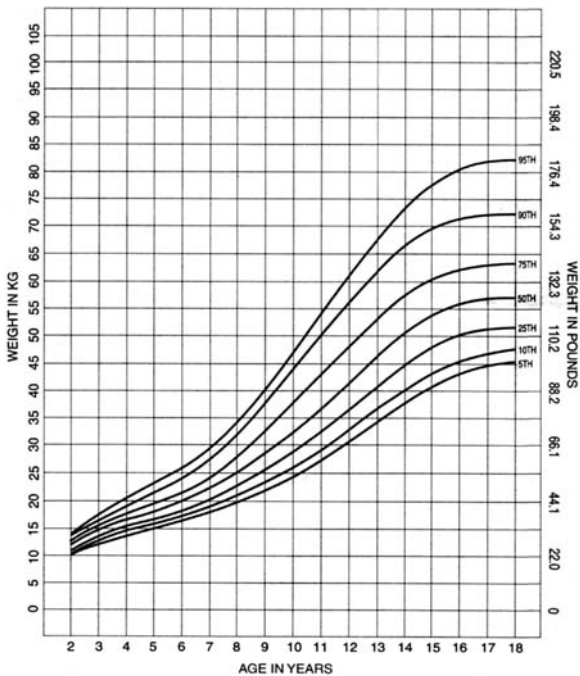


Figure 2–3. Weight by age percentiles for girls (2 to 18 years).

is considered abnormal in children.

Percent weight change can also be calculated as follows:

$$\text{percent weight change} = \frac{\text{usual weight} - \text{current weight}}{\text{usual weight}} \times 100$$

Technique. The subject stands, lies, or sits in the center of a balance scale platform. Minimal clothing and no shoes should be worn. Weight is taken to the nearest 0.1 kg or 1.0 oz.[1,2]

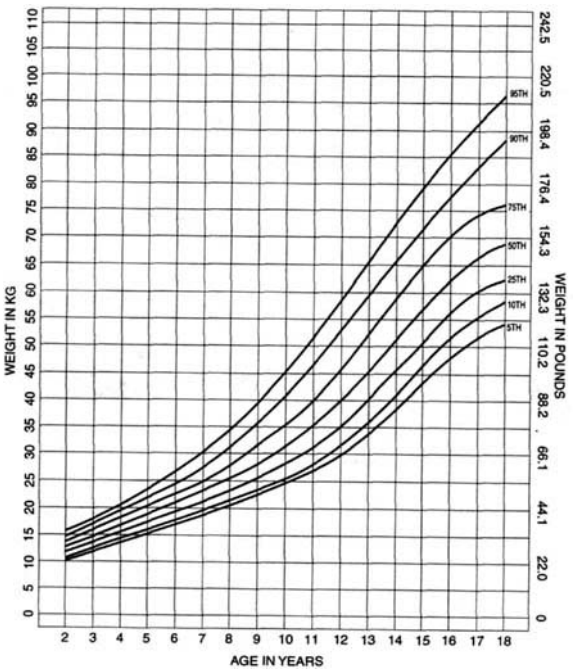


Figure 2–4. Weight by age percentiles for boys (2 to 18 years).

Length

Measured with appropriate equipment and technique, length is a simple and reproducible growth parameter that provides, in conjunction with weight, significant information.[1,2]

Standards. The NCHS growth charts (see Figures 2–5 to 2–8) are used for standards of length.[3]

Interpretation. Length for age below the 5th percentile indicates a severe deficit, and measurements that range between the 5th and 10th percentiles should be eval-

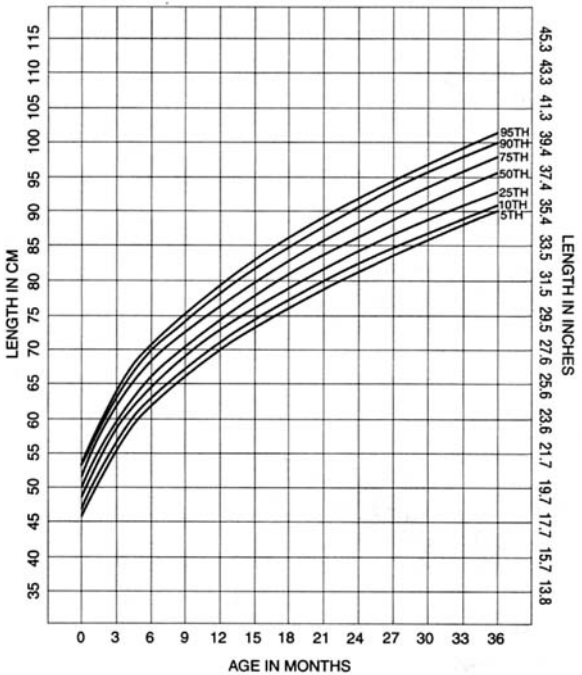


Figure 2–5. Length by age percentiles for girls (birth to 36 months).

uated further. Evaluation of growth velocity can be helpful in the determination of chronicity or constitutional short stature. Length assesses growth failure and chronic undernutrition, especially in early childhood and adolescence.

Technique. Measurement of length is frequently erroneous because of improper technique or equipment. The patient should be standing erect, without shoes, on the scale platform or on the floor. Shoulders should be straight, and the subject should look straight ahead.

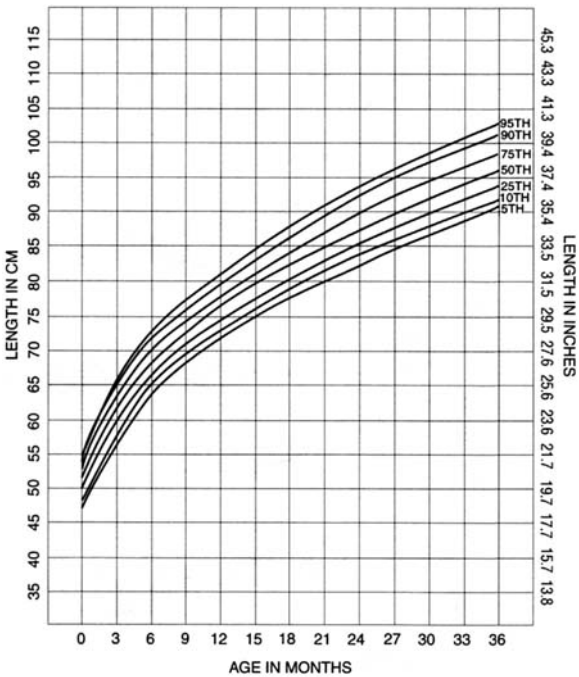


Figure 2–6. Length by age percentiles for boys (birth to 36 months).

Children younger than 2 years of age should be measured recumbent on a length board. Measurements should be to the nearest 0.5 cm or 0.125 in.[1,2]

Head Circumference

Head circumference can be influenced by nutritional status until the age of 36 months, but deficiencies are manifest in weight and height before being seen in brain growth. Routine examination also screens for other possible influences on brain growth.

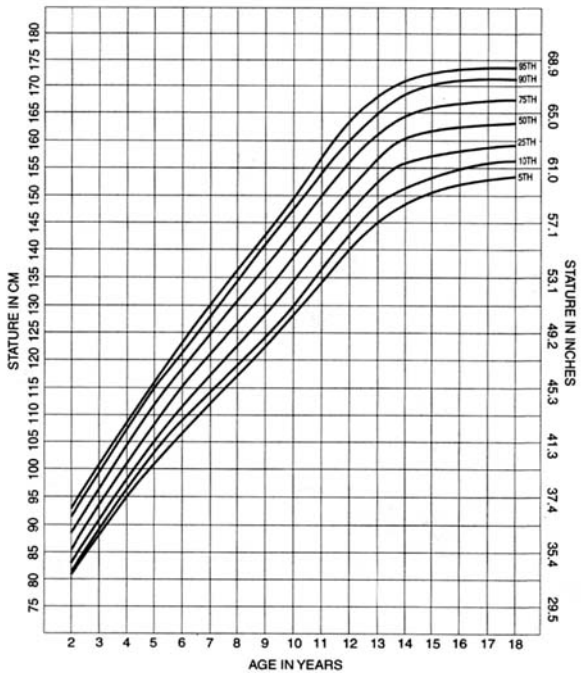


Figure 2–7. Stature by age percentiles for girls (2 to 18 years).

Standards. Figures 2–9 and 2–10 are NCHS growth charts.[3]

Interpretation. Measurements below the 5th percentile may indicate chronic undernutrition during fetal life and early childhood.

Technique. A flexible, narrow tape measure is placed firmly around the head above the supraorbital ridges and over the frontal bulge, where the circumference is greatest. Measurements should be taken to the nearest 0.5 cm or 0.25 in.[1,2]

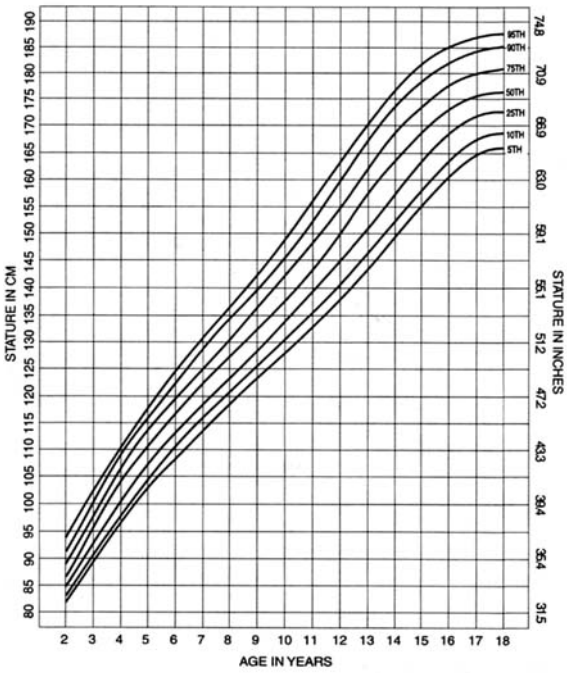


Figure 2–8. Stature by age percentiles for boys (2 to 18 years).

Weight for Height

This ratio more accurately assesses body build and distinguishes wasting (acute malnutrition) from stunting (chronic malnutrition).

Standards. Figures 2–11 and 2–12 chart weight from height for measurements taken by the NCHS.[3]

Interpretation. Measurements that fall near the 50th percentile indicate appropriate weight for height; the

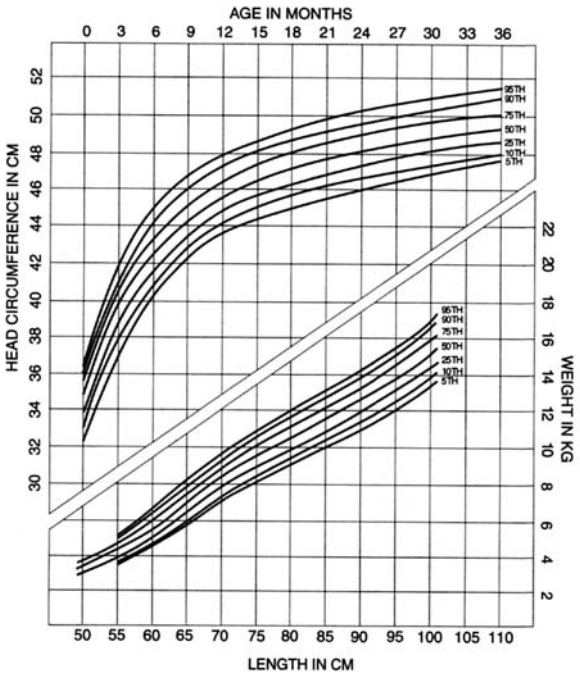


Figure 2–9. *Top*, head circumference by age percentiles for girls (birth to 36 months). *Bottom*, weight by length percentiles for girls (birth to 36 months).

greater the deviation, the more over- or undernourished the individual.

Body Mass Index

Body mass index is determined by dividing the person's weight in kilograms by their height in meters squared.[1,2] The formula for BMI is:

$$BMI = \text{weight (kg)}/\text{height}^2 \text{ (meters)}$$

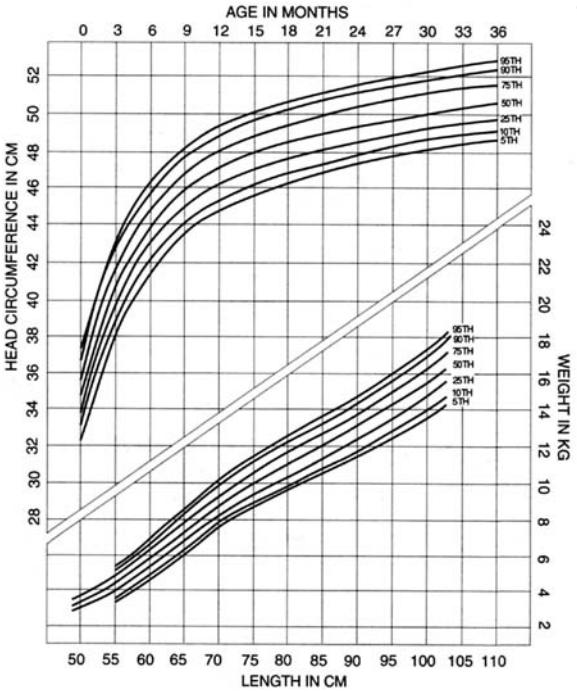


Figure 2–10. *Top,* head circumference by age percentiles for boys (birth to 36 months). *Bottom,* weight by length percentiles for boys (birth to 36 months).

BMI correlates well with adiposity in adults and because of its relative ease and accuracy of basic measures, is recommended for use in screening for obesity in adults. In the pediatric population use of BMI is still being evaluated. It is recommended that percentiles be used rather than an absolute number because this value changes throughout periods of growth.[4-7] Currently, a BMI at or above the 95th percentile for age and sex, using the NCHS growth data, indicates need for evaluation and treatment

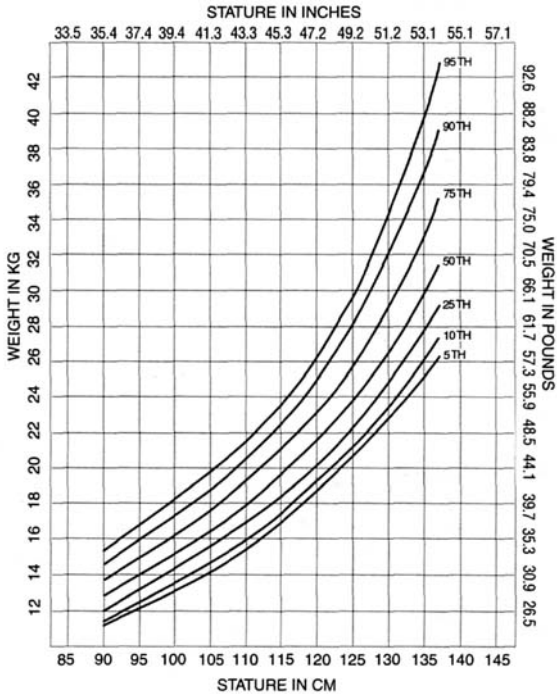


Figure 2–11. Weight by stature percentiles for prepubescent girls.

for obesity. A BMI at the 95th percentile may range from 18 to 30 depending on the age and sex of the child.[4–7]

Z Score

An alternative way to express height, weight, and weight for height is Z score, which denotes units of standard deviation from the median. It allows the clinician to locate an observation on the normal curve by the number of standard deviations it is from the center of the curve, and thus

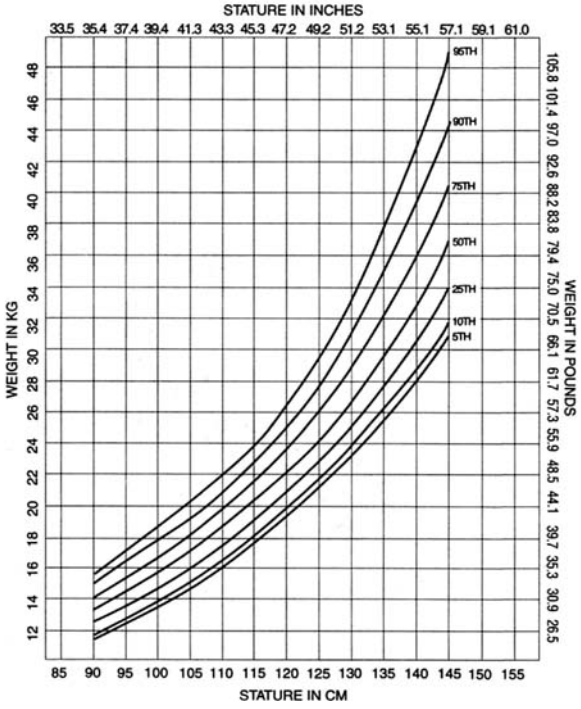


Figure 2–12. Weight by stature percentiles for prepubescent boys.

detect movement toward or away from the median, which is more sensitive than percentile changes. This is especially useful in cases that lie outside the percentiles (ie, below the 5th or above the 95th percentile). The World Health Organization recommends using Z scores, especially when describing groups of subjects.[8] The Z score is calculated as follows:

$$\text{Z score} = \frac{\text{actual anthropometric value} - \text{median reference value}}{\text{standard deviation}}$$

3% = −1.88
50% = 0.0
97% = +1.88

For example, for a 2-year-old male with cystic fibrosis whose weight is 10 kg (5th percentile):

$$\text{Z score} = \frac{10 - 12.3}{1.67 \text{ (upper limit)}} = -1.38$$

Subsequent to nutrition evaluation, a feeding gastrostomy was placed, and night enteral feedings were instituted. When seen at 1-month follow-up (age 2 years and 1 month), his weight was 10.6 kg, and his Z score is as follows:

$$\text{Z score} = \frac{10.6 - 12.5}{1.68} = -1.13$$

Although still below the median for age, his weight at age 2 years and 1 month is moving toward the median (ie, showing improvement).

Growth Velocity

Growth velocity is a simple and reproducible measure that evaluates change in rate of growth over a specified time

period; it generally is expressed in centimeters per year. It is a more sensitive way of assessing growth failure or slowed growth, and is particularly helpful in the early identification of children with undernutrition.

Standards. Figures 2–13 and 2–14 are growth velocity charts, based on growth data of North American children.[9] Additional reference data for 1-month increments

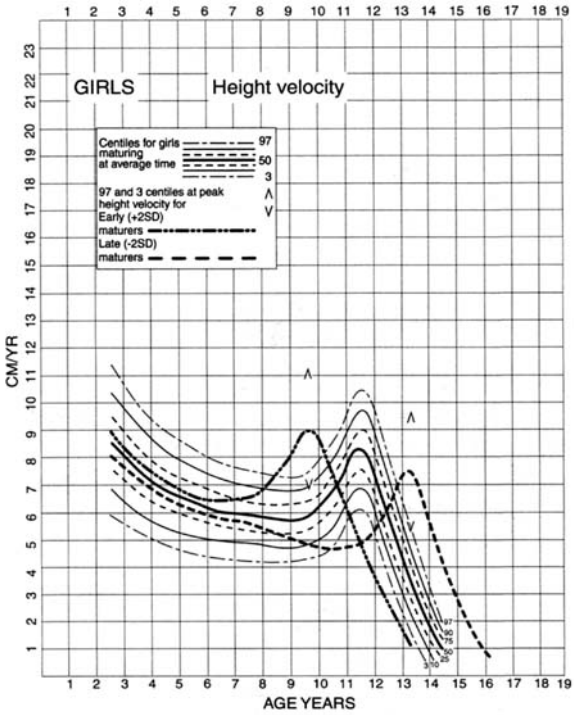


Figure 2–13. Height velocity for American girls. (Reproduced with permission from Tanner JM, Davis PSW. Clinical longitudinal standards for height and weight velocity for North American children. J Pediatr 1985;107:317–29.)

of weight and recumbent length from 1 to 12 months of age are shown in Figures 2–15 to 2–18.

Interpretation. Increments in growth may occur at different times but follow a similar sequence in most instances. Growth velocity charts are constructed from and used for longitudinally obtained incremental data. They detect changes in growth status more quickly

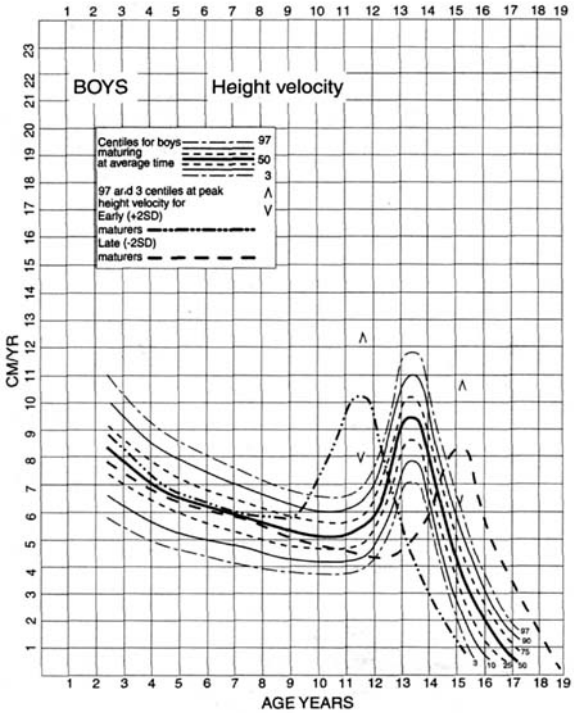


Figure 2–14. Height velocity for American boys. (Reproduced with permission from Tanner JM, Davis PSW. Clinical longitudinal standards for height and weight velocity for North American children. J Pediatr 1985;107:317–29.)

because they evaluate rate of growth. Chapter 27 discusses growth failure in more detail.

Estimation of Mature Height

Several methods are available for predicting mature height; each considers different variables and is an estimate only.[10–12]

Standards. Tables 2–1 and 2–2 are Fels parent-specific standards for height.[10] They are sex- and age-specific and are calculated by adding the biologic mother's height to the biologic father's height in centimeters and dividing by two. This parental midpoint should estimate the height of the child at specific ages. Tanner and colleagues found that height at 3 years, more so than at any other age, showed a good correlation with mature height.[11] He developed the following formulas:

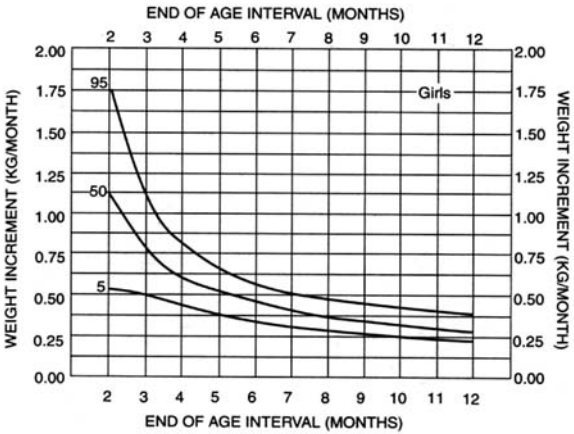


Figure 2–15. Selected percentiles of 1-mo increments in weight (kg/mo) for girls. These increments are plotted opposite the ends of the age intervals. (Figures 2–15 to 2–18 are reproduced with permission from Roche AF, Guo S, Moore WM. Weight and recumbent length from 1 to 12 months of age: reference data for 1-mo increments. Am J Clin Nutr 1989;49:599–607.)

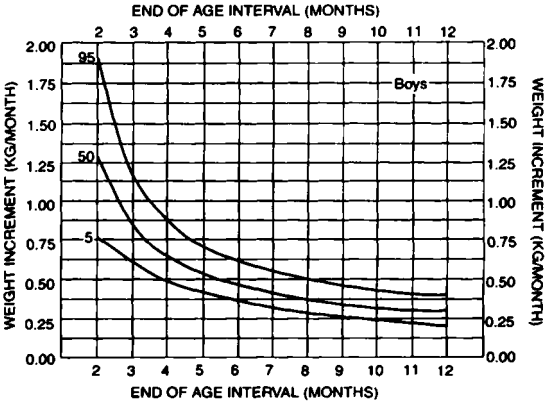


Figure 2–16. Selected percentiles of 1-mo increments in weight (kg/mo) for boys. These increments are plotted opposite the ends of the age intervals.

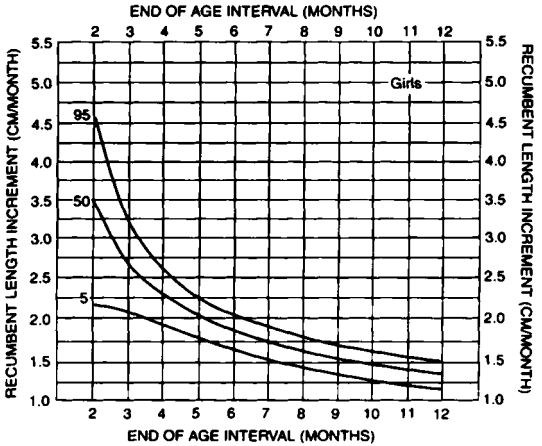


Figure 2–17. Selected percentiles of 1-mo increments in recumbent length (cm/mo) for girls. These increments are plotted opposite the ends of the age intervals.

Height (cm) at maturity (male)	=	1.27 × height at age 3 years + 54.9 cm
Height (cm) at maturity (female)	=	1.29 × height at age 3 years + 42.3 cm

Interpretation. Due to genetic influences, some children may be taller or shorter than average. Estimations may be helpful in evaluating short stature. The growth patterns of other family members may also be helpful in determining the correct diagnosis in such instances.[12] Short stature during childhood and adolescence should be evaluated to determine whether it is a normal variation or whether it indicates an underlying caloric deficiency or disease.

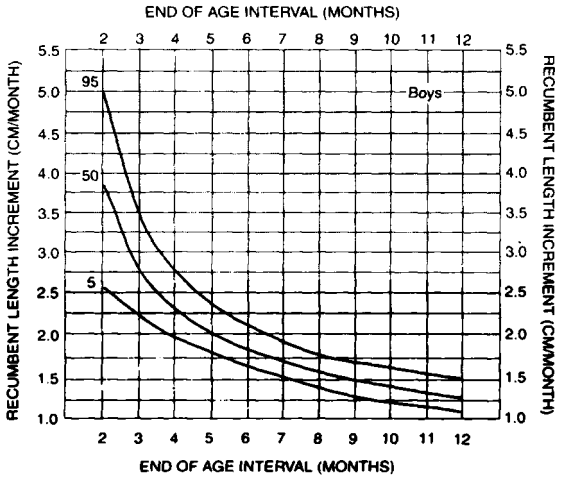


Figure 2–18. Selected percentiles of 1-mo increments in recumbent length (cm/mo) for boys. These increments are plotted opposite the ends of the age intervals.

Skinfold Thickness

Nearly half of the body fat in humans is in the subcutaneous layer, and measurements of this deposit can lead to accurate estimates of total body fat. Skinfold thickness measurements are accurate, simple, and reproducible and can be used to monitor changes in total body fat.

Standards. The measurements in Table 2–3 were compiled by Karlberg and colleagues and are based on longitudinal data of 200 Swedish children in the first 3 years of life.[13] Measurements for childhood through adult life were compiled by Frisancho, based on measurements of large samples of children throughout the United States (Table 2–4).[14]

Interpretation. Skinfold thickness measurements assess current nutritional status and body composition. They provide an index of body energy stores and can be used in conjunction with weight or height to determine chronic undernutrition and to define the athletic child who may be overweight but not "overfat." Measurement sites vary, and edema or intravenous fluids may affect accuracy. Measurements are most useful on children who can be followed over a period of time.[1,2]

Technique. Measurement of skinfold thickness is taken at the midpoint between the acromion and olecranon on the relaxed nondominant arm. The layer of skin and subcutaneous tissue is pulled away from the underlying muscle and held until measurement with the calipers at the midpoint has been taken. Readings should be taken to 0.5 mm, 3 seconds after application of calipers. Lange or Harpenden skinfold calipers are recommended for accurate measurements.[1,2]

Arm Circumference

In conjunction with triceps skinfold thickness, arm circumference can be used to determine cross-sectional midarm muscle and fat areas. As with skinfold thickness,

Table 2–1. Fels Parent-Specific Standards for Height: Girls' Stature by Age and Parental Midpoint States*

	Parental Midpoint (cm)									
Age (yr–mo)	*161*	*163*	*165*	*167*	*169*	*171*	*173*	*175*	*177*	*178*
Birth...	47.3	48.9	49.0	49.2	49.2	48.8	49.7	49.1	49.0	47.5
0–1...	53.0	53.4	54.2	52.0	53.3	53.1	53.5	53.2	55.8	52.8
0–3...	58.4	58.4	59.6	57.4	59.4	59.6	59.4	58.0	61.5	57.6
0–6...	64.4	64.7	65.6	65.7	64.6	66.5	66.6	67.4	67.3	65.8
0–9...	68.2	69.0	70.2	70.1	69.8	71.5	71.5	71.0	72.2	69.8
1–0...	72.3	73.0	73.8	74.0	74.0	75.2	75.5	74.6	77.3	73.2
1–6...	78.8	79.5	80.6	81.4	80.2	81.7	82.6	81.6	84.0	81.0
2–0...	84.6	84.0	86.5	87.4	85.5	88.8	88.7	88.2	89.5	87.6
2–6...	89.1	87.2	91.0	91.6	89.9	93.2	92.9	92.6	93.9	92.0
3–0...	93.2	90.4	94.5	95.8	93.8	97.1	96.5	96.5	98.5	96.2
3–6...	96.7	93.5	98.3	99.6	97.8	101.4	100.3	102.0	102.4	103.0
4–0...	100.1	96.8	102.4	103.5	103.9	104.9	104.0	103.8	105.8	104.3
4–6...	103.5	100.2	106.0	106.7	105.8	108.6	107.5	107.4	109.4	108.0
5–0...	106.8	103.5	108.9	109.9	109.1	111.6	110.9	111.0	112.6	111.7
5–6...	110.0	107.0	112.2	113.2	112.0	114.8	114.4	114.2	115.8	115.4
6–0...	113.2	110.2	115.0	116.2	115.0	118.2	117.8	117.3	119.1	118.8
6–6...	116.1	113.4	117.8	119.4	117.6	121.6	121.2	120.8	122.6	122.3
7–0...	118.8	116.5	120.6	122.4	120.2	124.4	124.4	124.0	125.0	125.5
7–6...	121.7	119.4	123.5	125.7	122.9	127.6	127.6	127.3	127.8	128.7

8–0...	124.6	122.4	126.3	128.8	125.8	130.7	130.8	130.2	130.8	132.0
8–6...	127.3	125.5	129.4	131.8	128.5	133.8	133.8	133.4	133.9	135.0
9–0...	130.1	128.6	132.2	134.7	131.4	137.1	136.7	136.6	137.0	138.2
9–6...	132.7	131.6	135.6	137.5	134.2	140.2	139.8	139.8	139.9	140.9
10–0...	136.0	135.1	139.0	140.3	136.9	143.8	142.9	143.1	143.8	143.6
10–6...	139.1	138.5	142.3	143.2	140.0	147.4	146.0	146.6	147.4	146.4
11–0...	141.9	141.6	145.9	146.0	143.4	150.3	149.0	149.6	151.3	149.4
11–6...	145.0	144.8	149.4	148.9	146.6	153.2	152.1	152.8	155.3	152.2
12–0...	148.0	147.8	152.8	151.8	150.3	156.4	155.2	155.8	159.0	154.9
12–6...	150.8	151.1	155.8	154.4	154.0	159.0	158.2	158.8	161.1	158.0
13–0...	152.9	154.2	158.8	157.0	157.0	161.0	161.1	161.7	162.3	160.5
13–6...	154.5	157.2	161.0	159.1	159.0	163.0	163.3	164.0	163.0	162.5
14–0...	155.4	158.8	161.7	160.9	160.4	163.7	165.0	165.9	163.9	164.1
14–6...	155.7	159.4	162.2	162.5	161.5	164.0	166.2	167.4	164.5	165.5
15–0...	155.9	159.8	162.6	163.7	162.2	164.0	167.1	168.4	165.0	166.5
15–6...	156.1	160.1	162.7	164.7	162.9	164.0	167.5	169.2	165.3	167.8
16–0...	156.0	160.5	162.8	165.5	163.4	164.1	167.8	169.7	165.5	168.7
16–6...	156.1	160.7	162.9	166.1	163.8	164.2	167.8	170.3	165.6	169.4
17–0...	156.2	160.8	163.0	166.5	164.0	164.3	167.9	170.9	165.7	170.0
17–6...	156.2	160.9	163.0	166.9	164.2	164.4	167.9	171.4	165.7	170.4
18–0...	156.2	161.0	165.0	167.2	164.3	164.4	167.9	171.8	165.7	170.8

*No attempt to eliminate sampling fluctuations.

Reproduced with permission from Garn GM, Rohman CC. Interaction of nutrition and genetics in the timing of growth and development. Pediatr Clin North Am 1966;13:353.

Table 2–2. Fels Parent-Specific Standards for Height: Boys' Stature by Age and Parental Midpoint States*

	Parental Midpoint (cm)									
Age (yr–mo)	*161*	*163*	*165*	*167*	*169*	*171*	*173*	*175*	*177*	*178*
Birth...	X	47.1	49.7	50.3	50.0	48.3	50.7	50.0	51.5	51.4
0–1...	X	52.7	54.6	54.7	57.6	53.2	53.6	52.2	55.6	55.9
0–3...	X	58.9	60.8	60.0	62.2	57.4	60.8	61.2	61.4	62.6
0–6...	X	65.1	66.2	66.8	67.4	65.8	70.2	69.0	70.2	70.3
0–9...	X	70.7	72.9	73.8	73.2	71.0	74.8	75.2	77.1	75.7
1–0...	X	73.1	75.6	75.7	75.1	73.4	76.6	77.1	79.6	77.8
1–6...	X	79.9	82.4	81.7	82.0	81.2	82.6	83.4	86.8	85.2
2–0...	X	85.4	87.2	87.0	87.4	87.8	88.0	88.9	92.0	91.3
2–6...	X	88.8	91.3	92.0	92.1	93.2	93.5	94.0	96.7	96.0
3–0...	X	93.2	94.9	96.1	96.0	97.2	98.1	98.3	100.7	99.9
3–6...	X	96.3	98.4	100.0	99.5	101.0	102.3	102.6	104.5	103.5
4–0...	X	99.5	102.2	103.5	103.1	104.6	106.0	106.3	108.0	107.0
4–6...	X	102.7	105.4	107.1	106.6	108.0	109.6	109.6	111.4	110.4
5–0...	X	105.6	108.5	110.6	110.0	111.5	113.2	112.7	114.6	113.8
5–6...	X	108.3	111.3	113.4	112.7	114.5	116.3	115.8	117.4	116.8
6–0...	X	110.9	114.1	116.4	115.4	117.4	119.4	118.7	120.4	119.8
6–6...	X	113.6	116.9	119.3	118.4	120.3	122.4	121.7	123.4	122.8
7–0...	X	116.2	119.7	122.3	121.3	123.2	125.6	124.6	126.6	125.6
7–6...	X	118.9	122.6	125.1	124.3	126.1	128.8	127.6	129.5	128.4

8–0...	X	121.6	125.0	127.8	126.8	128.8	131.6	130.4	132.8	131.6
8–6...	X	124.2	127.6	130.7	129.3	131.5	134.9	133.2	135.9	134.6
9–0...	X	126.9	130.4	133.3	131.9	134.1	138.0	136.0	138.8	137.5
9–6...	X	129.9	132.9	136.1	134.6	136.9	141.0	138.8	142.0	140.5
10–0...	X	132.5	135.8	138.8	137.4	139.8	143.8	141.5	145.3	143.2
10–6...	X	135.6	138.8	141.5	140.3	142.6	146.8	144.3	148.6	146.0
11–0...	X	138.5	141.8	144.1	143.0	145.4	149.9	146.8	151.9	148.9
11–6...	X	141.6	144.9	146.9	145.6	148.3	152.8	149.6	155.4	151.6
12–0...	X	144.7	148.0	149.7	148.4	151.4	155.7	152.4	158.8	154.5
12–6...	X	147.7	151.1	152.6	151.6	154.6	158.3	155.8	162.6	157.5
13–0...	X	151.0	154.2	155.7	154.9	158.0	161.7	159.6	166.3	160.5
13–6...	X	154.5	157.7	158.9	158.1	161.6	164.6	163.6	170.1	163.8
14–0...	X	158.8	161.7	162.3	161.6	165.7	167.6	167.8	173.4	166.9
14–6...	X	162.6	164.9	165.9	164.8	169.6	170.3	172.0	175.2	171.3
15–0...	X	165.8	168.1	169.1	167.9	172.9	173.0	174.7	176.4	175.2
15–6...	X	168.0	171.3	172.0	170.6	174.5	175.6	175.8	177.0	178.6
16–0...	X	169.4	173.3	174.3	172.8	177.3	177.5	176.6	177.4	181.2
16–6...	X	170.3	174.2	175.8	174.4	178.4	178.7	177.3	177.4	182.8
17–0...	X	170.9	174.7	176.8	175.4	179.2	179.4	177.8	177.5	184.3
17–6...	X	171.2	174.9	174.4	176.0	180.0	179.9	178.2	177.6	185.4
18–0...	X	171.5	175.0	177.9	176.2	180.6	180.2	178.6	177.6	186.3

*No attempt to eliminate sampling fluctuations.

Reproduced with permission from Garn GM, Rohman CC. Interaction of nutrition and genetics in the timing of growth and development. Pediatr Clin North Am 1966;13:353.

Table 2–3. Thickness of Triceps and Subscapular Skinfolds

Age			*Triceps (mm)*		*Subscapular (mm)*	
(mo)	*Percentiles*	*SD*	*Males*	*Females*	*Males*	*Females*
1		–2	2.9	3.5	3.1	3.8
	10		4.0	4.5	4.2	4.9
	25		4.7	5.2	4.8	5.4
	50		5.3	5.8	5.6	6.2
	75		6.2	6.7	6.5	7.0
	90		7.0	7.6	7.5	7.9
		+2	8.1	8.3	8.3	9.0
3		–2	4.5	5.0	3.5	4.7
	10		6.0	6.2	4.9	5.9
	25		6.8	7.2	5.8	6.9
	50		8.1	8.2	6.9	8.0
	75		9.2	9.2	8.1	8.6
	90		10.3	10.5	9.0	9.4
		+2	11.7	11.8	10.7	11.1
6		–2	6.3	6.7	3.8	4.0
	10		7.8	8.2	5.5	5.9
	25		8.6	9.0	6.2	6.9
	50		9.7	10.4	7.1	8.1
	75		11.1	11.3	8.4	8.9
	90		11.8	12.7	10.1	10.3
		+2	13.5	13.9	11.0	12.4
9		–2	6.0	6.7	3.4	4.7
	10		7.5	7.9	5.3	6.0
	25		8.7	8.8	6.0	6.7
	50		9.9	10.1	7.1	7.6
	75		11.2	11.3	8.5	8.8
	90		12.5	12.5	9.7	10.1
		+2	14.0	13.5	11.4	11.1
12		–2	6.2	6.4	3.8	4.5
	10		7.8	7.6	5.3	6.0
	25		8.6	8.7	6.0	6.5
	50		9.8	9.8	7.2	7.5
	75		11.1	11.2	8.6	8.7
	90		12.2	12.2	9.6	9.8
		+2	13.8	13.6	11.0	10.9

Table 2–3. continued

Age			Triceps (mm)		Subscapular (mm)	
(mo)	Percentiles	SD	Males	Females	Males	Females
18		−2	6.4	6.8	3.9	4.2
	10		7.7	7.9	5.3	5.7
	25		8.6	8.9	6.0	6.2
	50		9.9	10.3	6.8	7.1
	75		11.4	11.3	7.9	8.0
	90		12.2	12.3	9.3	9.0
	+2		13.6	13.6	10.3	10.2
24		−2	5.8	6.5	3.0	3.9
	10		7.4	8.3	4.6	5.3
	25		8.5	8.9	5.4	5.6
	50		9.8	10.1	6.5	6.5
	75		11.6	11.6	7.4	7.3
	90		13.1	12.8	8.3	8.4
		+2	14.2	14.1	10.2	9.5
36		−2	6.6	6.4	2.9	2.6
	10		7.8	8.2	4.5	4.7
	25		9.0	9.4	5.0	5.2
	50		9.8	10.3	5.5	6.1
	75		11.0	11.5	6.4	7.2
	90		12.2	12.5	7.1	8.6
		+2	13.4	14.4	8.9	10.6

Adapted from Karlberg P, Engstrom I, Lichtenstein H, Svennberg I. The development of children in a Swedish urban community: a prospective longitudinal study. III. Physical growth during the first three years of life. Acta Paediatr Scand Suppl 1968;187:48.

arm circumference correlates well with other more sophisticated and difficult measures of body composition.

Standards. Tables 2–5 and 2–6 were completed by Frisancho using NCHS measurements.[14]

Interpretation. A simple nomogram calculation requires arm circumference and triceps skinfold measures to determine muscle circumference and cross-sectional muscle and fat areas (Figures 2–19 and 2–20).[15]

Table 2–4. Percentile for Triceps Skinfold (mm^2)*

	Males								*Females*							
Age Group	*n*	*5*	*10*	*25*	*50*	*75*	*90*	*95*	*n*	*5*	*10*	*25*	*50*	*75*	*90*	*95*
1–1.9	228	6	7	8	10	12	14	16	204	6	7	8	10	12	14	16
2–2.9	223	6	7	8	10	12	14	15	208	6	8	9	10	12	15	16
3–3.9	220	6	7	8	10	11	14	15	208	7	8	9	11	12	14	15
4–4.9	230	6	6	8	9	11	12	14	208	7	8	8	10	12	14	16
5–5.9	214	6	6	8	9	11	14	15	219	6	7	8	10	12	15	18
6–6.9	117	5	6	7	8	10	13	16	118	6	6	8	10	12	14	16
7–7.9	122	5	6	7	9	12	15	17	126	6	7	9	11	13	16	18
8–8.9	117	5	6	7	8	10	13	16	118	6	8	9	12	15	18	24
9–9.9	121	6	6	7	10	13	17	18	125	8	8	10	13	16	20	22
10–10.9	146	6	6	8	10	14	18	21	152	7	8	10	12	17	23	27
11–11.9	122	6	6	8	11	16	20	24	117	7	8	10	13	18	24	28
12–12.9	153	6	6	8	11	14	22	28	129	8	9	11	14	18	23	27
13–13.9	134	5	5	7	10	14	22	26	151	8	8	12	15	21	26	30
14–14.9	131	4	5	7	9	14	21	24	141	9	10	13	16	21	26	28
15–15.9	128	4	5	6	8	11	18	24	117	8	10	12	17	21	25	32
16–16.9	131	4	5	6	8	12	16	22	142	10	12	15	18	22	26	31

17–17.9	133	5	5	6	8	12	16	19	114	10	12	13	19	24	30	37
18–18.9	91	4	5	6	9	13	20	24	109	10	12	15	18	22	26	30
19–24.9	531	4	5	7	10	15	20	22	1060	10	11	14	18	24	30	34
25–34.9	971	5	6	8	12	16	20	24	1987	10	12	16	21	27	34	37
35–44.9	806	5	6	8	12	16	20	23	1614	12	14	18	23	29	35	38
45–54.9	898	6	6	8	12	15	20	25	1047	12	16	20	25	30	36	40
55–64.9	734	5	6	8	11	14	19	22	809	12	16	20	25	31	36	38
65–74.9	1503	4	6	8	11	15	19	22	1670	12	14	18	24	29	34	36

*Data collected from whites in the United States Health and Nutrition Examination Survey 1 (1971–1974).
Reproduced with permission from Frisancho AR. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981;34:2540.

Table 2–5. Percentiles of Upper Arm Circumference and Estimated Upper Arm Muscle Circumference*

	Arm Circumference (mm)							*Arm Muscle Circumference (mm)*						
Age Group	*5*	*10*	*25*	*50*	*75*	*90*	*95*	*5*	*10*	*25*	*50*	*75*	*90*	*95*
							Males							
1–1.9	142	146	150	159	170	176	183	110	113	119	127	135	144	147
2–2.9	141	145	153	162	170	178	185	111	114	122	130	140	146	150
3–3.9	150	153	160	167	175	184	190	117	123	131	137	143	148	153
4–4.9	149	154	162	171	180	186	192	123	126	133	141	148	156	159
5–5.9	153	160	167	175	185	195	204	128	133	140	147	154	162	169
6–6.9	155	159	167	179	188	209	228	131	135	142	151	161	170	177
7–7.9	162	167	177	187	201	223	230	137	139	151	160	168	177	190
8–8.9	162	170	177	190	202	220	245	140	145	154	162	170	182	187
9–9.9	175	178	187	200	217	249	257	151	154	161	170	183	196	202
10–10.9	181	184	196	210	231	262	274	156	160	166	180	191	209	221
11–11.9	186	190	202	223	244	261	280	159	165	173	183	195	205	230
12–12.9	193	200	214	232	254	282	303	167	171	182	195	210	223	241
13–13.9	194	211	228	247	263	286	301	172	179	196	211	226	238	245
14–14.9	220	226	237	253	283	303	322	189	199	212	223	240	260	264
15–15.9	222	229	244	264	284	311	320	199	204	218	237	254	266	272
16–16.9	244	248	262	278	303	324	343	213	225	234	249	269	287	296
17–17.9	246	253	267	285	308	336	347	224	231	245	258	273	294	312

18–18.9	245	260	276	297	321	353	379	226	237	252	264	283	298	324
19–24.9	262	272	288	308	331	355	372	238	245	257	273	289	309	321
25–34.9	271	282	300	319	342	362	375	243	250	264	279	298	314	326
35–44.9	278	287	305	326	345	363	374	247	255	269	286	302	318	327
45–54.9	267	281	301	322	342	362	376	239	249	265	281	300	315	326
55–64.9	258	273	296	317	336	355	369	236	245	260	278	295	310	320
65–74.9	248	263	285	307	325	344	355	223	235	251	268	284	298	306
						Females								
1–1.9	138	142	148	156	164	172	177	105	111	117	124	132	139	143
2–2.9	142	145	152	160	167	176	184	111	114	119	126	133	142	147
3–3.9	143	150	158	167	175	183	189	113	119	124	132	140	146	152
4–4.9	149	154	160	169	177	184	191	115	121	128	136	144	152	157
5–5.9	153	157	165	175	185	203	211	125	128	134	142	151	159	165
6–6.9	156	162	170	176	187	204	211	130	133	138	145	154	166	171
7–7.9	164	167	174	183	199	216	231	129	135	142	151	160	171	176
8–8.9	168	172	183	195	214	247	261	138	140	151	160	171	183	194
9–9.9	178	182	194	211	224	251	260	147	150	158	167	180	194	198
10–10.9	174	182	193	210	228	251	265	148	150	159	170	180	190	197
11–11.9	185	194	208	224	248	276	303	150	158	171	181	196	217	223
12–12.9	194	203	216	237	256	282	294	162	166	180	191	201	214	220
13–13.9	202	211	223	243	271	301	338	169	175	183	198	211	226	240
14–14.9	214	223	237	252	272	304	322	174	179	190	201	216	232	247
15–15.9	208	221	239	254	279	300	322	175	178	189	202	215	228	244

Table 2–5. continued

	Arm Circumference (mm)							*Arm Muscle Circumference (mm)*						
Age Group	*5*	*10*	*25*	*50*	*75*	*90*	*95*	*5*	*10*	*25*	*50*	*75*	*90*	*95*
							Females							
16–16.9	218	224	241	258	283	318	334	170	180	190	202	216	234	249
17–17.9	220	227	241	264	295	324	350	175	183	194	205	221	239	257
18–18.9	222	227	241	258	281	312	325	174	179	191	202	215	237	245
19–24.9	221	230	247	265	290	319	345	179	185	195	207	221	236	249
25–34.9	233	240	256	277	304	342	368	183	188	199	212	228	246	264
35–44.9	241	251	267	290	317	356	378	186	192	205	218	236	257	272
45–54.9	242	256	274	299	328	362	384	187	193	206	220	238	260	274
55–64.9	243	257	280	303	335	367	385	187	196	209	225	244	266	280
65–74.9	240	252	274	299	326	356	373	185	195	208	225	244	264	279

*Data collected from whites in the United States Health and Nutrition Examination Survey 1 (1971–1974).
Adapted from Frisancho AR. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981;34:2540.

Table 2–6. Percentiles for Estimates of Upper Arm Fat Area and Upper Arm Muscle Area*

	Arm Muscle Area Percentiles (mm^2)							*Arm Fat Area Percentiles (mm^2)*						
Age Group	*5*	*10*	*25*	*50*	*75*	*90*	*95*	*5*	*10*	*25*	*50*	*75*	*90*	*95*
						Males								
1–1.9	956	1014	1133	1278	1447	1644	1720	452	486	590	741	895	1036	1176
2–2.9	973	1040	1190	1345	1557	1690	1787	434	504	578	737	871	1044	1148
3–3.9	1095	1201	1357	1484	1618	1750	1853	464	519	590	736	868	1071	1151
4–4.9	1207	1264	1408	1579	1747	1926	2008	428	494	598	722	859	989	1085
5–5.9	1298	1411	1550	1720	1884	2089	2285	446	488	582	713	914	1176	1299
6–6.9	1360	1447	1605	1815	2056	2297	2493	371	446	539	678	896	1115	1519
7–7.9	1497	1548	1808	2027	2246	2494	2886	423	473	574	758	1011	1393	1511
8–8.9	1550	1664	1895	2089	2296	2628	2788	410	460	588	725	1003	1248	1558
9–9.9	1181	1884	2067	2288	2657	3053	3257	485	527	635	859	1252	1864	2081
10–10.9	1930	2027	2182	2575	2903	3486	3882	523	543	738	982	1376	1906	2609
11–11.9	2016	2156	2382	2670	3022	3359	4226	536	695	754	1148	1710	2348	2574
12–12.9	2216	2339	2649	3022	3496	3968	4640	544	650	874	1172	1558	2536	3580
13–13.9	2363	2546	3044	3553	4081	4502	4794	475	570	812	1096	1702	2744	3322
14–14.9	2830	3147	3586	3963	4575	5368	5530	453	563	786	1082	1608	2746	3508
15–15.9	3138	3317	3788	4481	5134	5631	5900	521	595	690	931	1423	2434	3100
16–16.9	3625	4044	4352	4951	5753	6576	6980	542	593	844	1078	1746	2280	3041
17–17.9	3998	4252	4777	5286	5950	6886	7726	598	698	827	1096	1636	2407	2888
18–18.9	4070	4481	5066	5552	6374	7067	8355	560	665	860	1264	1947	3302	3928

Table 2–6. continued

	Arm Muscle Area Percentiles (mm^2)							*Arm Fat Area Percentiles (mm^2)*						
Age Group	*5*	*10*	*25*	*50*	*75*	*90*	*95*	*5*	*10*	*25*	*50*	*75*	*90*	*95*
						Males								
19–24.9	4508	4777	5274	5913	6660	7606	8200	594	743	963	1406	2231	3098	3652
25–34.9	4694	4963	5541	6214	7067	7847	8436	675	831	1174	1752	2459	3246	3786
35–44.9	4844	5181	5740	6490	7265	8034	8488	703	851	1310	1792	2463	3098	3624
45–54.9	4546	4946	5589	6297	7142	7918	8458	749	922	1254	1741	2359	3245	3928
55–64.9	4422	4783	5381	6144	6919	7670	8149	658	839	1166	1645	2236	2976	3466
65–74.9	3973	4411	5031	5716	6432	7074	7453	573	753	1122	1621	2199	2876	3327
						Females								
1–1.9	885	973	1084	1221	1378	1535	1621	401	466	578	706	847	1022	1140
2–2.9	973	1029	1119	1269	1405	1595	1727	469	526	642	747	894	1061	1173
3–3.9	1014	1133	1227	1396	1563	1690	1846	473	529	656	822	967	1106	1158
4–4.9	1058	1171	1313	1475	1644	1832	1958	490	541	654	766	907	1109	1236
5–5.9	1238	1301	1423	1598	1825	2012	2159	470	529	647	812	991	1330	1536
6–6.9	1354	1414	1513	1683	1877	2182	2323	464	508	638	827	1009	1263	1436
7–7.9	1330	1441	1602	1815	2045	2332	2469	491	560	706	920	1135	1407	1644
8–8.9	1513	1566	1808	2034	2327	2657	2996	527	634	769	1042	1383	1872	2482
9–9.9	1723	1788	1976	2227	2571	2987	3112	642	690	933	1219	1584	2171	2524
10–10.9	1740	1784	2019	2296	2583	2873	3093	616	702	842	1141	1608	2500	3005

11–11.9	1784	1987	2316	2612	3071	3739	3953	707	802	1015	1301	1942	2730	3690
12–12.9	2092	2182	2579	2904	3225	3655	3847	782	854	1090	1511	2056	2666	3369
13–13.9	2269	2426	2657	3130	3529	4081	4568	726	838	1219	1625	2374	3272	4150
14–14.9	2418	2562	2874	3220	3704	4294	4850	981	1043	1423	1818	2403	3250	3765
15–15.9	2426	2518	2847	3248	3689	4123	4756	839	1126	1396	1886	2544	3093	4195
16–16.9	2308	2567	2865	3248	3718	4353	4946	1126	1351	1663	2006	2598	3374	4236
17–17.9	2442	2674	2996	3336	3883	4552	5251	1042	1267	1463	2104	2977	3864	5159
18–18.9	2398	2538	2917	3243	3694	4461	4767	1003	1230	1616	2104	2617	3508	3733
19–24.9	2538	2728	3026	3406	3877	4439	4940	1046	1198	1596	2166	2959	4050	4896
25–34.9	2661	2826	3148	3573	4138	4806	5541	1173	1399	1841	2548	3512	4690	5560
35–44.9	2750	2948	3359	3783	4428	5240	5877	1336	1619	2158	2898	3932	5093	5847
45–54.9	2784	2956	3378	3858	4520	5375	5974	1459	1803	2447	3244	4229	5416	6140
55–64.9	2784	3063	3477	4045	4750	5632	6247	1345	1879	2520	3369	4360	5276	6152
65–74.9	2737	3018	3444	4019	4739	5566	6214	1363	1681	2266	3063	3943	4914	5530

*Data collected from whites in the United States Health and Nutrition Examination Survey 1 (1971–1974).
Adapted from Frisancho AR. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981;34:2540.

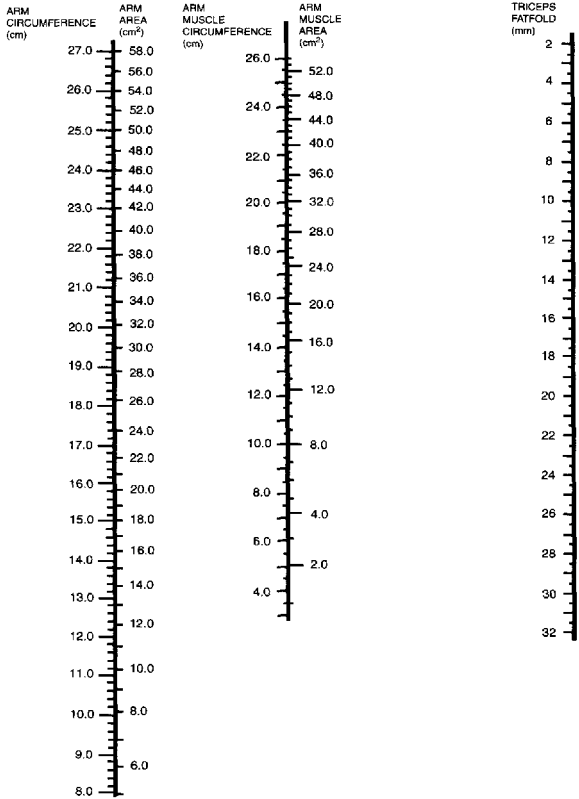


Figure 2–19. Arm anthropometry in nutritional assessment: nomogram for children. To obtain muscle circumference (1) lay ruler between values of arm circumference and fatfold, (2) read off muscle circumference on middle line. To obtain tissue areas (1) read arm and muscle areas listed to the right of their respective circumferences; (2) fat area = arm area – muscle area. (Reproduced with permission from Gurney JM, Jelliffe DB. Arm anthropometry in nutritional assessment: nomograms for rapid calculation of muscle circumference and cross-sectional muscle and fat areas. Am J Clin Nutr 1973;26:912.)

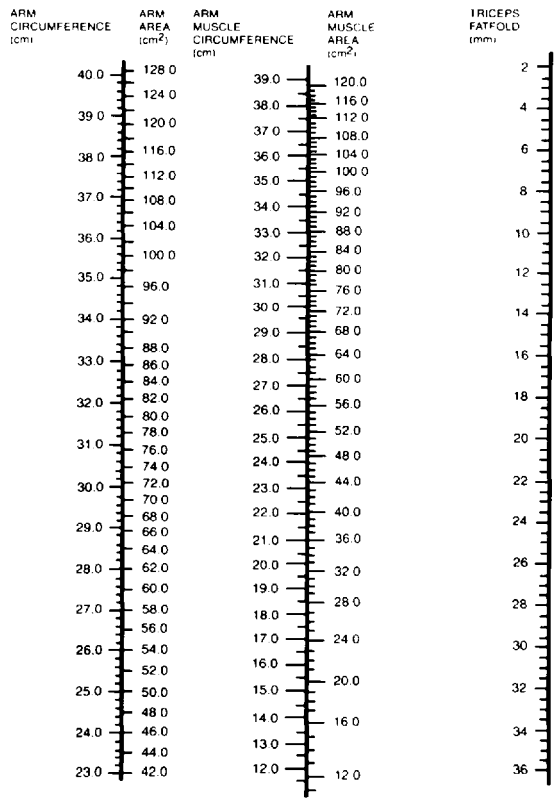


Figure 2–20. Arm anthropometry in nutritional assessment: nomogram for adults. To obtain muscle circumference (1) lay ruler between values of arm circumference and fatfold, (2) read off muscle circumference on middle line. To obtain tissue areas (1) read arm and muscle areas listed to the right of their respective circumferences; (2) fat area = arm area – muscle area. (Reproduced with permission from Gurney JM, Jelliffe DB. Arm anthropometry in nutritional assessment: nomograms for rapid calculation of muscle circumference and cross-sectional muscle and fat areas. Am J Clin Nutr 1973;26:912.)

Bone Age

Epiphyseal closure is a measure of skeletal maturation. Roentgenography of the hand and wrist is generally used for convenient determination of this measure. The percentage of maturity can be used to estimate potential for catch-up growth.

Standards. Measurements for epiphyseal closure can be found in the *Radiographic Atlas of Skeletal Development of the Hand and Wrist* by Greulich and Pyle.[16]

Interpretation. Skeletal age is generally advanced in overnutrition and retarded in any condition in which growth (measured by height) is slowed secondary to malnutrition. The success of catch-up growth depends on the length and age of slowed growth, the adequacy of nutritional repletion, and disease control. During this time, height velocity may be twice the average, and weight velocity may be four times the average. The rate of skeletal maturation also increases.

Sexual Maturation

During adolescence, growth in height and weight is accelerated. Following sexual maturation, a rapid deceleration of growth occurs. The impact of nutrition on delaying or accelerating puberty is well known.[17,18] Clinical evaluation of sexual maturation is helpful in determining the level of progression through adolescence.

Standards. Tanner's stages of sexual development provide a clinical rating scale (1 = preadolescent; 5 = mature) for comparison of development.[11] Tables 2–7 and 2–8 describe the various aspects of developmental stages.

Interpretation. Considerable variability exists as to the age at which these events occur. The sequence, however, is fairly uniform.

Table 2–7. Stages of Sexual Development: Female

Age	*Stages*	
0–12	I	Preadolescent. Female pelvic contour evident, breast flat, labia majora smooth and minora poorly developed, hymenal opening small or absent, mucous membranes dry and red, vaginal cells lack glycogen.
8–13	II	Breast: Nipple is elevated small mound beneath areola is enlarging and begins pigmentation. Labia majora: Become thickened, more prominent and wrinkled; labia minora easily identified because of increased size; clitoris, urethral opening more prominent, mucous membranes moist and pink, some glycogen present in vaginal cells.
9–14	III	Rapid growth peak is passed; menarche most often occurs at this stage and invariably follows the peak of growth acceleration. Breasts: Areola and nipple further enlarge and pigmentation becomes more evident; continued increase in glandular size. Labia minora: Well developed and vaginal cells have increased glycogen content; mucous membranes increasingly more pale. Hair: In pubic region thicker, coarser, often curly (considerable normal variation, including a few girls with early stage II at menarche). Skin: Further increased activity in sebaceous and sweat glands; beginning of acne in some girls, adult body odor.
12–15	IV	Breasts: Projection of areola above breast plane, and areolar (Montgomery's) glands apparent (this development is absent in about 20% of normal girls); glands easily palpable. Labia: Both major and minor assume adult structure, glycogen content of vaginal cells begins cyclic characteristics. Hair: In pubic area more abundant; axillary hair present (rarely present at stage II, not uncommonly present at stage III).

Table 2–7. continued

Age	*Stages*	
12–17	V	Breasts: Mature histologic morphology; nipple enlarged and erect, areolar (Montgomery's) glands well developed, globular shape. Hair: In pubic area more abundant and may spread to thighs (in about 10% of women it assumes "male" distribution with extension toward umbilicus; facial hair increased often in form of slight mustache. Skin: Increased sebaceous gland activity and increased severity of acne if present before.

Table 2–8. Stages of Sexual Development: Male

Age	*Stages*	
0–14	I	Preadolescent
10–14	II	Testes and penis: Increasing size is evident (testicle length reaches 2.0 cm or more); scrotum integument is thinner and assumes as increased pendulous appearance.
11–15	III	Rapid growth peak is passed; nocturnal emissions begin. Testes and penis: Further increase in size and pigmentation; Leydig's cells (interstitial) first appear at stage II, are now prominent in testes. Hair: More abundant pubic area and present on scrotum; still scanty and finely textured; axillary hair begins. Breasts: Button-type hypertrophy in 70% of boys at stages II and III. Larynx: Changes in voice caused by laryngeal growth begin. Skin: Increasing activity of sebaceous and sweat glands; beginning of acne, adult body odor.
12–16	IV	Testes: Further increase in size (length 4.0 cm or greater); increase in size of penis greatest at stages III and IV.

		Hair: Pubic hair thicker and coarser; in most it ascends toward umbilicus in typical male pattern; axillary hair increases, facial hair increases overlap and upper cheeks. Larynx: Voice deepens. Skin: Increasing pigmentation of scrotum and penis; acne often more severe. Breast: Previous hypertrophy decreased or absent.
13–17	V	Testes: Length greater than 4.5 cm. Hair: Pubic hair thick, curly, heavily pigmented, extends to thighs and toward umbilicus; adult distribution and increase in body hair (chest, shoulders, and thighs) continues for more than another 10 years; baldness, if present may begin. Larynx: Adult character of voice. Skin: Acne may persist and increase.

Reproduced with permission from Lowrey GH. Growth and development of children. 7th ed. Chicago: Year Book; 1978.

Classification of Malnutrition

Malnutrition is a pathologic state of varying severity; its clinical features are caused by a deficiency or by an imbalance of essential nutrients. The cause may be primary (insufficient quantity or quality of food) or secondary (increased requirements or inadequate utilization).

Development of *marasmus* occurs after severe deprivation primarily of calories, and it is characterized by growth retardation and wasting of muscle and subcutaneous fat. In *kwashiorkor*, the protein deficiency exceeds the calorie deficiency; edema accompanies muscle wasting that results from acute protein deprivation or loss of protein caused by stress or inadequate calories. Indifference, apathy, and fatigue are present in victims of both conditions, and psychologic alterations may be profound. Severe anorexia, apathy, and irritability make children with these conditions difficult to feed and manage. Many of the clin-

Table 2–9. Clinical Signs in Malnutrition

	Marasmus	*Kwashiorkor*
Growth retardation (linear)	++	+
Severely underweight	++	–
Muscle wasting	+	++
Edema	–	+
Apathy, fatigue	+	++
Irritability	+	+
Electrolyte imbalance (hypokalemia)	+	+
Hypoalbuminemia	–	+
Anemia	+	++
Fatty liver	–	+
Low body temperature	+	++
Flakey pain dermatitis	–	+

– = not seen; + = seen; ++ = seen more frequently or is more marked.

ical signs (changes in hair and skin) lack specificity and are identical to symptoms of other nutrient deficiencies listed in Table 2–9.

Malnutrition was first defined in terms of a deficit in weight for a child's age.[19] However, height for age and weight for height are often more useful tools for evaluation of acute and chronic malnutrition.[20,21] For example, a low weight for height is seen in acute malnutrition. In chronic undernutrition, there are frequently no clinical signs other than a low height for age. Children who are over 90 percent of their expected weight for height and less than 90 percent of their expected height for age are termed nutritional dwarfs, since their height has been stunted but their weight is appropriate for their height (Table 2–10).

The morbidity and mortality associated with malnutrition are more closely correlated with the degree of malnutrition than with sex, age, or specific clinical factors,

Table 2–10. Most Common Classifications of Protein-Calorie Malnutrition

	Normal	*Mild*	*Moderate*	*Severe*
Weight for height*	110–90	90–85	85–75	< 75
Weight for age†	110–90	90–81	80–61	< 60
Weight for age‡	> 90	90–75	75–61	< 60
Height for age§	> 95	98–87	87–80	< 80
Weight for height§	> 90	90–80	80–70	< 70

*Reproduced with permission from McLaren DS, Read WWC. Weight/length classification of nutrition status. Lancet 1975;2:219.
†Reproduced with permission from Jelliffe D. The assessment of the nutrition status of the community. Geneva (Switzerland): World Health Organization Monograph 53;1966.
‡Reproduced with permission from Gomez F, Galvan RR, Cravioto J, Frenk S. Malnutrition in infancy and childhood with special reference to kwashiorkor. Adv Pediatr 1955;7:131.
§Reproduced with permission from Waterlow JC. Classification and definition of protein calorie malnutrition. BMJ 1972;3:565.

although some studies show a higher mortality rate in infancy than in older age groups. Third-degree (severe) malnutrition has a range of mortality rates from 30 to 60 percent, and second-degree (moderate) malnutrition has a mortality rate of 25 percent. A higher mortality rate is seen with kwashiorkor than with marasmus; electrolyte and fluid imbalances and increased risk of infection increase the death rate significantly.[22–24]

References

1. Gibson RS. Principles of nutritional assessment. Oxford: Oxford University Press; 1990. p. 155–62.
2. Ekvall SW. Nutritional assessment and early intervention. In: Pediatric nutrition in chronic diseases and developmental disorders. Ekvall SW, editor. New York: Oxford University Press; 1993. p. 41–76.

3. Hamil PVV, Drizd TA, Johnson CL, et al. Physical growth: National Center for Health Statistics percentiles. Am J Clin Nutr 1979;32:607–29.
4. Deitz WH, Bellizzi MC. Introduction: the use of body mass index to assess obesity in children. Am J Clin Nutr 1999; 70 Suppl:1235–55.
5. Malina RM, Katzmarzyk PT. Validity of the body mass index as an indicator of the risk and presence of overweight in adolescents. Am J Clin Nutr 1999;70 Suppl:1315–65.
6. Must A, Dallal GE, Dietz WH. Reference data for obesity: 85th and 95th percentiles of body mass index (wt/ht^2) and triceps skinfold thickness. Am J Clin Nutr 1991;53:839–46.
7. Must A, Dallal GE, Dietz WH. Reference data for obesity: 85th and 95th percentiles of body mass index (wt/ht^2) a correction. Am J Clin Nutr 1991;54:773.
8. Dibley MJ, Staehling N, Nieburg P, Trowbridge FL. Interpretation of Z score anthropometric indicators derived from the International Growth Reference. Am J Clin Nutr 1987;46:749.
9. Tanner JM, Davis PSW. Clinical longitudinal standards for height and weight velocity for North American children. J Pediatr 1985;107:317–29.
10. Garn SM, Rohmann CG. Interaction of nutrition and genetics in the timing of growth and development. Pediatr Clin North Am 1966;13:353.
11. Tanner JM, Healy MJR, Lockhart RO, Aberdeen growth study. I. The prediction of actual body measurements from measurements taken each year from birth to five years. Arch Dis Child 1956;31:372.
12. Himes JH, Roche AF, Thissen D, Moore WM. Parent-specific adjustments for evaluation of recumbent length and stature of children. Pediatrics 1985;75:304.
13. Karlberg P, Engstrom I, Lichtenstein H, Svennberg I. The development of children in a Swedish urban community: a prospective longitudinal study. III. Physical growth during the first three years of life. Acta Paediatr Scand Suppl 1968;187:48.

14. Frisancho AR. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981;34:2540.
15. Gurney JM, Jelliffe DB. Arm anthropometry in nutritional assessment: nomogram for rapid calculation of muscle circumference and cross-sectional muscle and fat areas. Am J Clin Nutr 1973;26:912.
16. Greulich WW, Pyle SI. Radiographic atlas of skeletal development of the hand and wrist. 2nd ed. Stanford: Stanford University Press; 1959.
17. Mills JL, Shiono PH, Shapiro LR, et al. Early growth predicts timing of puberty in boys: results of a 14 year nutrition and growth study. J Pediatr 1986;109:543–7.
18. Lowry GH. Growth and development of children. 7th ed. Chicago: Year Book Medical Publishers Inc.; 1978. p. 326–7.
19. Gomez F, Galvan RR, Cravioto J, Frenk S. Malnutrition in infancy and childhood with special reference to kwashiorkor. Adv Pediatr 1955;7:131.
20. McLaren DS, Read WWC. Weight/length classification of nutritional status. Lancet 1975;2:219.
21. Jelliffe D. The assessment of nutritional status of the community. Geneva (Switzerland): World Health Organization Monograph 53;1966.
22. Galvan RR, Calderon JM. Death among children with third degree malnutrition. Am J Clin Nutr 1965;16:351.
23. Sommer A, Lowwenstein MS. Nutritional status and mortality. Am J Clin Nutr 1975;28:287.
24. McLaren DS, Shirajian E, Loshkajian HJ, Shadarevian S. Short-term prognosis in protein-calorie malnutrition. Am J Clin Nutr 1969;22:863.

3

NUTRITIONAL ASSESSMENT

CLINICAL EVALUATION

Kristy M. Hendricks, RD, MS, ScD

Severe nutritional deprivation is easily detectable in most instances. More subtle physical signs, which suggest less severe chronic or subacute deficiencies, are often nonspecific for individual nutrients. Thorough medical and dental histories and physical examinations that show signs suggestive of nutrient deficiency or excess should be recorded and described as precisely as possible and confirmed by biochemical, anthropometric, or dietary evaluation. Table 3–1 describes the major physiologic functions, signs of deficiency, signs of excess, important food sources, potential causes of intolerance, and detailed laboratory evaluation for essential nutrients.[1,2,3] The impact of medications and other nutrients on individual nutrient status is also significant (see Appendix B). Recommended nutrient intakes are detailed in Chapter 5.

References

1. Christakis G. Nutritional assessment in health programs. Am J Public Health (Suppl) 1973;63:1.
2. National Research Council, Food and Nutrition Board. Recommended dietary allowances. Washington (DC): National Academy of Sciences; 1989.
3. Loughrey CM, Duggan C. Assessment of nutritional status: the role of the laboratory. In: Soldin SJ, Rifai N, Hicks JMB, editors. Biochemical basis of pediatric disease. Washington (DC): AACC Press; 1998.

Table 3–1. Clinical Examination of Nutritional Deficiencies and Excesses

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Carbohydrate					
Supplies energy at an average of 4 cal/g (sparing protein) and is the major energy source for CNS function; unrefined, complex carbohydrates supply fiber that aids in normal bowel function	Ketosis	May cause diarrhea, obesity	Bread, cereals, crackers, potatoes, corn, simple sugar (sugar, honey), fruits and vegetables, milk, breastmilk, infant formula	Malabsorption	Blood sugar, OGTT
Fat					
Concentrated calorie source at an average of 9 cal/g; constitutes part of the membrane structure of every cell; supplies essential fatty acids and provides and carries fat-soluble vitamins (A,D,E,K)	Essential fatty acid deficiency; dry, scaly skin, poor weight gain, hair loss. Requirements are increased by cell turnover	Atherosclerosis may be affected by excessive intakes of certain dietary fats; altered blood lipid levels	Shortening, oil, butter, margarine, protein-rich foods (meat, dairy, nuts), breast milk, infant formula	Cystic fibrosis, biliary disease, short bowel/ hereditary lipoprotein disorders	Total cholesterol, LDL, HDL

Table 3–1. continued

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Protein					
Constitutes part of the structure of every cell; regulates body processes as part of enzymes, hormones, body fluids, and antibodies that increase resistance to infection; provides nitrogen and has a caloric density of 4 cal/g	Dry, depigmented, easily pluckable hair; bilateral, dependent edema, cirrhosis, fatty liver, decreased visceral proteins. Skin is dry with pellagroid dermatoses in severe cases	Azotemia, acidosis, hyperammonemia	Meat, poultry, fish, legumes, eggs, cheese, milk, and other dairy products, nuts, breastmilk, infant formula	Protein-losing enteropathy, liver disease, gastrointestinal disease, renal disease	Albumin, retinol-binding protein
Fat-Soluble Vitamins					
Vitamin A					
Formation and maintenance of skin and mucous membranes;	Night blindness, degeneration of the retina,	Fatigue, malaise, lethargy, abdominal pain,	Carrots, liver, green vegetables, sweet potatoes,	Liver disease, cystic fibrosis, short bowel	Plasma retinol (HPLC), plasma retinol-binding

necessary for the formation of rhodopsin (the photosensitive pigment of the rods governing vision in dim light), and regulation of membranes' structure and function; necessary for growth and normal immune function	xerophthalmia, follicular hyperkeratosis, poor growth, keratomalacia, Bitot's spots	hepatomegaly, alopecia, headache with increased intracranial pressure, vomiting	butter, margarine, apricots, melons, peaches, broccoli, cod liver oil, breastmilk, infant formula	disease, protein deficiency (alters transport)	protein, relative dose response, dark adaption test, liver biopsy concentration
Vitamin D					
Promotes intestinal absorption of calcium and phosphate, renal conservation of calcium and phosphorus	Rickets, osteomalacia, costochondral beading, epiphyseal enlargement, cranial bossing, bowed legs, persistently open anterior fontanelle	Hypercalcemia, vomiting, anorexia, diarrhea, convulsions	Cod liver oil, fish, eggs, liver, butter, fortified milk, sunlight (activation of 7-dehydro-cholesterol in the skin), infant formula	Liver disease, cystic fibrosis, short bowel disease, renal disease	Plasma 25-hydro-xy-vitamin D (HPLC), serum alkaline phosphatase, calcium and phosphate, radiography, bone densitometry

Table 3–1. continued

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Vitamin E					
Acts as an antioxidant and free radical scavenger to prevent peroxidation of polyunsaturated fatty acids in the body; *neuromuscular* function	Hemolytic anemia in the premature and newborn, enhanced fragility of red blood cells, increased peroxidative hemolysis	In anemia, suppresses the normal hematologic response to iron	Oils high in polyunsaturated fatty acids, milk, eggs, breastmilk, infant formula	Cystic fibrosis, short bowel, liver disease	Plasma tocopherol (HPLC) (corrected for total or LDL-cholesterol), hydrogen peroxide hemolysis
Vitamin K					
Necessary for prothrombin and the three blood-clotting factors VII, IX, and X. Half of the vitamin K in man is of intestinal origin, synthesized by gut flora; necessary for bone mineralization	Hemorrhagic manifestations (especially in newborns), prolonged clotting	Hemolytic anemia, nerve palsy	Green leafy vegetables, fruits, cereals, dairy products, soybeans, breast milk, infant formula	Liver disease, antibiotic therapy	Prothrombin time (prolonged), plasma phyllo-quinone, clotting factor levels

Water-Soluble Vitamins

Ascorbic Acid (C)

Forms collagen cross-linkage of proline hydroxylase, thus strengthening tissue and improving wound healing and resistance to infection; aids absorption of iron	Joint tenderness, scurvy (capillary hemorrhaging), impaired wound healing, acute periodontal gingivitis, petechiae, purpura, anemia	Documentation of a chronic high intake may result in "rebound" deficiency symptoms	Broccoli, papayas, oranges, mangoes, grapefruit, strawberries, tomatoes, potatoes, leafy vegetables, breastmilk, infant formula	Stress	Plasma level (enzyme assay/ HPLC), leukocyte concentration (longer-term), whole blood concentration, urine concentration

Biotin

Component of several carboxylating enzymes; plays an important role in the metabolism of fat and carbohydrate	Anorexia, nausea, vomiting; glossitis; depression; dry, scaly, dermatitis; thin hair	None known	Liver, kidney, egg yolk, breastmilk, infant formula	Certain inborn errors of metabolism	Plasma (microbiologic assay), plasma lactate, urine organic acids, lymphocyte carboxylase

Cobalamin (B_{12} intrinsic factor required)

Cobalamin-containing coenzymes function in the degradation of certain odd-chain fatty acids and in the recycling of tetrahydrofolate	Megaloblastic anemia, neurologic deterioration	None known	Animal products, breastmilk, infant formula, fortified soy products	Ileal disease, strict vegetarian, lack of intrinsic factor	Plasma level (RIA or microbiologic), Schilling test, plasma homocysteine, deoxyuridine suppression test

Table 3–1. continued

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Folacin					
Required for 1 carbon carbontransfer and nucleotide synthesis	Megaloblastic anemia, stomatitis, glossitis, neural tube defects in pregnancy, elevated homocysteine levels	None known	Liver, leafy vegetables, fruit, yeast, breastmilk, infant formula	Liver disease, alcoholism, celiac disease, inflammatory	Plasma level (RIA or microbiologic), red cell level bowel disease
Niacin					
Aids in energy utilization as part of a coenzyme (NAD+ and NADP+) in fat synthesis, tissue respiration, and carbohydrate utilization; aids digestion and fosters normal appetite; synthesized from the amino acid tryptophan	Pellagra (dermatitis, diarrhea, dementia, death), cheilosis, angular stomatic inflammation of mucous membranes, weakness	Dilation of the capillaries, vasomotor instability, "flushing", utilization of muscle glycogen, serum lipids, mobilization of fatty acids during exercise	Liver, meat, fish, poultry, peanuts, fortified cereal products, yeast, breastmilk, infant formula	B_6 deficiency (impairs conversion of tryptophan to niacin)	Urine ratio of metabolites (N-methylnicotinamide:2-pyridone), tryptophan load red cell NAD or NAD:NADP ratio

Pantothenic Acid					
Component of coenzyme A; plays a role in release of energy from carbohydrates and in synthesis and degradation of fatty acids	Infertility, fetal loss, slow growth, depression, vomiting, malaise, abdominal stress	Diarrhea, water retention	Meat, fish, poultry, whole grains, legumes, breast milk, infant formula	Severe malnutrition	Urine excretion, whole blood level (RIA/ microbiologic)
Pyridoxine (B_6)					
Coenzyme component for many of the enzymes of amino acid metabolism. All compounds implicated as neurotransmitters are synthesized and/or metabolized in the B_6-dependent reactions	Convulsions, loss of weight, abdominal distress, vomiting hyperirritability, depression, confusion, hypochromic and macrocytic anemia	Neuropathy	Fish, poultry, meat, wheat, breastmilk, infant formula	Elderly, high protein intake	Red cell aminotransferase activity, plasma pyridoxal phosphate (HPLC) tryptophan loading test, urine 4-pyridoxic acid
Riboflavin (B_2)					
Functions primarily as the reactive portion of flavoproteins concerned with biologic oxidations (cellular metabolism)	Cheilosis, glossitis, photophobia, angular stomatitis, corneal vascularization, scrotal skin changes, seborrhea, magenta tongue	None known	Dairy products, liver, almonds, lamb, pork, breast milk, infant formula	Alcoholism, starvation, chronic diarrhea, malabsorption	Red cell glutathione reductase activity, red cell flavine adenine dinucleotide, urine riboflavin:creatinine ratio

Table 3–1. continued

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Thiamine (B_1)					
Aids in energy utilization as part of coenzyme component to promote the utilization of carbohydrate; promotes normal functioning of the nervous system; coenzyme for oxidative carboxylation of 2-keto acids	Beriberi, neuritis, edema, cardiac failure, anorexia, restlessness, confusion, loss of vibration sense and deep tendon reflexes, calf tenderness	None known	Pork, nuts, whole grain and fortified cereal products, breast milk, infant formula	Alcoholism, refeeding after starvation, prolonged dialysis	Red cell transketolase activity, whole blood level (HPLC), urine thiamine: creatinine ratio
Minerals					
Calcium					
Essential for calcification of bone (matrix formation); assists in blood clotting; functions in normal muscle contraction	Osteomalacia, osteoporosis tremor, convulsions, hyperexcitability (hypocalcemia tetany)	Hypercalcemia vomiting, anorexia, lethargy	Dairy products (milk, cheese), sardines, oysters, salmon, herring, greens, breast milk, infant formula	Renal disease, liver disease, steroid use	Plasma total calcium, plasma free calcium in altered protein binding (eg, hypoalbuminemia),

and relaxation and in normal nerve transmission					acidosis, radiographs, or CT and photon densitometry
Magnesium					
Essential part of many enzyme systems; important for maintaining electrical potential in nerves and muscle membranes and for energy turnover	Tremor, tingling, weakness, seizures, arrhythmia	Nausea, vomiting hypertension, weakness, prolonged QT interval	Widely distributed, especially in food of vegetable origin; breastmilk, infant formula	PCM; refeeding	Plasma total or free magnesium, magnesium loading test
Phosphorus					
Important intracellular anion; involved in many chemical reactions within the body; necessary for energy turnover (ATP)	Weakness, anorexia, malaise, bone pain, growth arrest	Hypocalcemia (when parathyroid gland not fully functioning)	Dairy products, fish, legumes, pork, breast milk, infant formula	Renal disease, liver disease, refeeding syndrome	Plasma concentration, alkaline phosphatase activity, radiography densitometry, renal tubular excretion threshold

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Trace Elements					
Chromium					
Maintenance of normal glucose metabolism, cofactor for insulin	Disturbed glucose metabolism (lower glucose tolerance caused by insulin resistance)		Brewer's yeast, meat products, cheeses	PCM, elderly	Plasma chromium, glucose tolerance
Copper					
Constituent of proteins and enzymes, some of which are essential for proper utilization of iron. Immunity, skeletal development	Anemia (hemolytic), neutropenia, bone disease	Excess accumulation in the liver, brain, kidney, cornea; anemia, diarrhea	Oysters, nuts, liver, kidney, corn-oil margarine, dried legumes	Menkes' kinky-hair syndrome. Excess: Wilson's disease	Plasma copper, plasma ceruloplamin (ferrochelatase), liver biopsy concentration, superoxide dismutase activity
Fluoride					
The main target organs of fluoride in man are the enamel of teeth and	Poor dentition, caries, osteoporosis	Mottling, brown staining of teeth (in excess of 4 ppm); fluorosis	Fluoridated water. Depends on the geochemical environment;	Unfluoridated water, bottled water	

bones, where fluoride is incorporated into the crystalline structure of hydroxyapatite and produces increased caries resistance		occurs after prolonged (10–20 yr) ingestion of 20–80 mg/day	therefore, amount in food varies widely		
Iodine					
Component of thyroid hormones triiodothyronine and thyroxine, important in regulation of cellular oxidation and growth	Goiter, depressed thyroid function, cretinism	Thyroid suppression (thyrotoxicosis)	Iodized table salt, saltwater fish, shellfish (content of most other foods geographically dependent), breast milk, infant formula	Endemic goiter in low-iodine areas	Thyroid hormones, TSH, urinary iodide: creatinine ratio
Iron					
Part of hemoglobin molecule; prevents nutritional anemia, and fatigue; increases resistance to infection; functions as part of enzymes involved in tissue respiration	Anemia, malabsorption, irritability, anorexia, pallor, lethargy	Hemosiderosis, hemochromatosis	Red meats, liver, dried beans and peas, enriched farina, breastmilk, infant formula, infant cereal, fortified cereals	Protein-losing enteropathy, malabsorption, acute or chronic blood loss Excess: hemochromatosis	Plasma iron and ferritin, total iron-binding capacity, hemoglobin/ hematocrit, red cell indices, RBC zinc protoporphyrin: heme ratio, bone marrow aspirate stain

Table 3–1. continued

Nutrient and Major Physiologic Functions	*Deficiency Signs*	*Excess Signs*	*Important Food Sources*	*Potential Causes of Deficiency or Excess*	*Laboratory Assessment*
Manganese					
Essential part of several enzyme systems involved in protein and energy metabolism and in the formation of mucopolysaccharide	Impaired growth, skeletal abnormalities, lowered reproductive function, neonatal ataxia	In extremely high exposure of contamination: severe psychiatric and neurologic disorders	Nuts, whole grains, dried fruits, fruits, vegetables (leafy)		Plasma level, whole blood level, mitochondrial superoxide dismutase
Molybdenum					
Essential for the function of flavin-dependent enzymes involved in the production of uric acid and in the oxidation of aldehydes and sulfites	Not described in man	Acts as an antagonist to the essential element copper; goutlike syndrome associated with elevated blood levels of molybdenum, uric acid, and xanthin oxidase	Varies considerably, depending on growing environment; main contributions come from meat, grains, and legumes		

Selenium					
Functions as a part of the enzyme glutathione peroxidase, which protects cellular component from oxidative damage	Cardiomyopathy, probably secondary to oxidative damage	In animals, blindness, abdominal pain	Seafoods, kidney, liver, meat, grains (depending on growing areas)	Cystic fibrosis	Plasma concentration, glutathione peroxidase activity, nail/hair selenium
Zinc					
Constituent of enzymes involved in most major metabolic pathways (specifically nucleic acid synthesis for growth and repair)	Growth failure, skin changes, delayed wound healing, hypogeusia, sexual immaturity, hair loss, diarrhea	Acute gastrointestinal upset, vomiting, sweating, dizziness, copper deficiency	Whole grains, legumes, beef, lamb, pork, poultry, nuts, seeds, shellfish, eggs, some cheeses, breast milk, infant formula	Malabsorption, chronic diarrhea, liver disease, sickle cell disease	Plasma concentration, alkaline phosphatase activity, urinary excretion, leukocyte concentration

ATP = adenosine triphosphate; CNS = central nervous system; CT = computed tomography; HDL = high-density lipoprotein; HPLC = high-performance liquid chromatography; LDL = low-density lipoprotein; NAD = nicotinamide-adenine dinucleotide; NADP = nicotinamide-adenine dinucleotide phosphate; OGTT = oral glucose tolerance test; PCM = protein calorie malnutrition; RBC = red blood cell; RIA = radioimmunoassay; TSH = thyroid-stimulating hormone.

4

LABORATORY ASSESSMENT OF NUTRITIONAL STATUS

Clodagh M. Loughrey, MD, MRCP, MRCPath, and Christopher Duggan, MD, MPH

Nutritional assessment in pediatrics rests largely on the clinical, anthropometric, and dietary methods detailed in Chapters 1 through 3. In certain situations, however, confirmation by biochemical means is crucial to (1) diagnose subclinical nutrient deficiencies, (2) substantiate clinically evident over- or undernutrition, and (3) provide baseline data for monitoring response to nutritional interventions.

Nitrogen Balance

Nitrogen (N) balance is one of the oldest methods of assessing nutritional status. It has classically been used to define amino acid requirements, since negative N balance will ensue if inadequate amounts of an essential amino acid are ingested. Children or others who should actively be gaining lean body mass should normally be in positive N balance whereas healthy adults may be said to be in nitrogen equilibrium if N loss is within 5% of N intake. It should be noted, however, that the mere demonstration of positive N balance does not disclose any information about N distribution throughout the body, nor about the accumulation of lean body mass.

Urea, which is the main excretory product of N metabolism, appears in both urine and sweat; approximately 85% of the body's N is lost in the urine. Other sources of N loss include fecal losses, integumental losses (eg,

desquamating skin, sweat, hair and nail growth), and miscellaneous losses (eg, saliva, vomitus, blood drawing, and menstrual losses, etc). These losses can increase significantly in patients with wounds or burn injuries. Since most proteins contain 16% N, dietary protein intake is customarily divided by a factor of 6.25 to estimate N intake.

The equation for calculating N balance is therefore:

$$\begin{aligned}\text{N balance} &= \text{N intake} - \text{N output} \\ &= (\text{24 h dietary protein intake in grams}/6.25) \\ &\quad - \text{24 h UUN} - \text{factor}\end{aligned}$$

where UUN = urine urea nitrogen (in grams) and factor = allowance made for uncollected N loss in stool, skin, and miscellaneous sources. In adults, this factor is 2 to 4 g/d; in children, an estimate of 10 mg/kg/d may be used.

Negative N balance can result from inadequate energy intake, inadequate protein intake, or catabolic stress and lean body mass breakdown. Positive N balance implies adequate energy and/or protein intake.

Serum Proteins

Proteins synthesized by the liver have long been used to assess protein status, since decreased blood concentrations presumably reflect a reduced supply of amino acid precursors and/or decreased hepatic (and other visceral) mass. Serum proteins may also be classified according to whether their serum concentration increases or decreases in the setting of acute infection or catabolism (Table 4–1). The concentrations of positive acute phase proteins are increased in infectious or other catabolic illnesses, whereas those of negative acute phase proteins are decreased.

Albumin

Albumin is the most abundant serum protein, is the least expensive and easiest to measure, and is therefore the

Table 4–1. Serum Proteins and Acute Illness

Positive Acute Phase Proteins	*Negative Acute Phase Proteins*
C reactive protein	Albumin
Fibrinogen	Prealbumin
Ferritin	Retinol binding protein
Ceruloplasmin	Transferrin
$Alpha_1$-antitrypsin	
$Alpha_1$-glycoprotein	

most commonly used biochemical marker in assessing protein status. Since more than half of body albumin is extravascular (primarily in skin and muscle), maintenance of normal serum levels can occur from mobilization of these stores despite prolonged energy or protein inadequacy. Combined with its long half-life of 20 days, these factors make serum albumin a relatively insensitive marker of nutritional status or of the effectiveness of nutritional interventions. Nevertheless, hypoalbuminemia continues to be a reasonable predictor of morbidity and mortality in hospitalized patients.

Normal concentrations of serum albumin are 3.5 to 5.0 g/dL. Hypoalbuminemia is not necessarily a definitive indicator of malnutrition. It can also be seen in situations of decreased synthesis (eg, liver disease, age over 70 years, malignancy), increased losses (eg, nephrosis, protein-losing enteropathy, burn injuries), or increased losses to extravascular spaces (eg, acute catabolic stress with capillary leak syndrome). Fluid overload can also dilute albumin concentrations, and bedrest can decrease levels 0.5 g/dL over several days.

Prealbumin

Prealbumin, named for its proximity to albumin on an

electrophoretic strip, is a transport molecule for thyroxine; hence its alternative name, transthyretin. It circulates in plasma in a 1:1 ratio with retinol-binding protein. Its short half-life (2 days) and high ratio of essential to nonessential amino acids make it a good measure of visceral protein status and more sensitive than albumin as a measure of nutritional recovery. Normal concentrations of prealbumin are shown in Table 4–2.

Retinol Binding Protein

Retinol binding protein (RBP) has similar properties to prealbumin in that it has a small body pool and a rapid response to protein-energy depletion and repletion. Its half-life is 12 hours. Since RBP is metabolized in the kidneys, levels will be artificially high in renal failure. Retinol binding protein levels also drop in vitamin A deficiency and, as with albumin and prealbumin, with infectious or other catabolic stresses. Normal levels of RBP are 3 to 6 mg/dL.

Transferrin

Transferrin is another serum protein sometimes used to assess visceral protein status. It is synthesized primarily in the liver and has a half-life of 8 days. Transferrin concentrations are decreased in all situations depressing serum albumin (see above) as well as with steroid therapy, iron overload, and anemia of chronic disease. Increased concentrations are seen in pregnancy, oral contraceptive use, and iron deficiency anemia. Normal levels of transferrin are 220 to 350 mg/dL.

Other Serum Proteins

Other serum proteins of possible use in assessing nutritional status include insulin-like growth factor I (IGF-1), which is the mediator for the anabolic effects of growth

hormone. Although IGF-1 levels vary with liver and kidney disease, they seem to correlate reasonably well with N balance. Fibronectin, a plasma protein with a half-life of 15 hours, has also been used as a marker for nutritional repletion in some studies.

Essential Fatty Acids

Fatty acids are classified according to carbon chain length and the presence or absence of one or more double bonds. The essential fatty acids (EFA) in humans are linoleic acid (C18:2 ω-6) and linolenic acid (C18:3 ω-3), both of which are long chain fats. Two common clinical scenarios leading to essential fatty acid deficiency are (1) prolonged fasting, or reliance on lipid-free parenteral nutrition, and (2) extended use of a formula with a predominance of medium chain fats. The clinical signs of linoleic acid deficiency include poor growth and a desquamating skin rash, but it is much more common to diagnose EFA deficiency by biochemical profile. When EFA deficiency occurs, the nonessential fatty acid eicosatrienoic acid (C20:3 ω-9) increases in serum; its three double bonds make it a "triene." Conversely, arachidonic acid (C20:4 ω-9, ie, a "tetraene") is reduced. Essential fatty acid deficiencies show a triene to tetraene ratio of > 0.4.

Other Laboratory Measurements of Nutritional Status

The most useful and least expensive laboratory measure of nutritional status is a complete blood count with differential. Lymphopenia is a well-known feature of protein-energy malnutrition resulting from a reduction in circulating T lymphocytes. Total lymphocyte count (TLC) can be calculated as follows:

$$\text{TLC (cells/mm}^3\text{)} = \text{WBC count} \times \text{percentage lymphocytes}$$

With mild malnutrition, TLC < 1500 cells/mm^3, with moderate malnutrition 800 to 1200 cells/mm^3, and with severe malnutrition, TLC < 800 cells/mm^3. Total lymphocyte count is, however, a nonspecific and insensitive measure of nutritional status.

Another common functional test of immunocompetence and, therefore, of adequate nutritional status, is *delayed-type hypersensitivity testing*. Cutaneous anergy, a delayed or absent response to intradermal injection of antigens, is a consistent finding in moderate to severe malnutrition and has been associated with an increased risk of complications of surgery. Anergy is also a nonspecific measure of nutritional status, however. Radiotherapy, the use of immunosuppressive agents, and critical illness are also associated with anergy.

Table 4–2. Normal Ranges of Select Nutritional Laboratory Values

Nutrient	*Age*	*Males*	*Either*	*Females*
Albumin (g/dL)	< 5 d (< 2.5 kg)		2.0–3.6	
	< 5 d (> 2.5 kg)		2.6–3.6	
	1–3 yr		3.4–4.2	
	4–6 yr		3.5–5.2	
	7–9 yr		3.7–5.6	
	10–19 yr		3.7–5.6	
Alkaline phosphatase (U/L)	1–3 yr	145–320		145–320
	4–6 yr	150–380		150–380
	7–9 yr	175–420		175–420
	10–11 yr	135–530		130–560
	12–13 yr	200–495		105–420
	14–15 yr	130–525		70–230
	16–19 yr	65–260		50–130
Ammonia (μmol/L)	< 30 d		21–95	
	< 1 yr		18–74	
	1–14 yr		17–68	
	> 14 yr		22–66	

Table 4–2. continued

Nutrient	*Age*	*Males*	*Either*	*Females*
Calcium	< 5 d (< 2.5 kg)		7.9–10.7	
(mg/dL)	1–3 yr		8.7–9.8	
	4–6 yr		8.8–10.1	
	7–9 yr		8.8–10.1	
	10–11 yr		8.9–10.1	
	12–13 yr		8.8–10.6	
	14–15 yr		9.2–10.7	
	16–19 yr		8.9–10.7	
Calcium,	0–1 mo		1.0–1.5	
ionized	1–6 mo		0.95–1.5	
(mmol/L)	1–19 yr		1.22–1.37	
Carnitine,	1–7 d		17–46	
total (μmol/L)	2 yr		24–66	
	> 2 yr		37–89	
Ceruloplasmin	0–5 d		50–260	
(mg/L)	1–3 yr		240–460	
	4–6 yr		240–420	
	7–9 yr		240–400	
	10–13 yr	220–360		230–430
	14–19 yr	140–340		200–450
Cholesterol	1–3 yr		44–181	
(mg/dL)	4–6 yr		108–187	
	7–9 yr		112–247	
	10–11 yr	125–230		127–244
	12–13 yr	127–230		125–213
	14–15 yr	106–224		130–213
	16–19 yr	110–220		106–217
Copper	< 5 d		9–46	
(μg/dL)	1–5 yr		80–150	
	6–9 yr		84–136	
	10–14 yr	80–121		82–120
	15–19 yr	64–171		72–160
Ferritin	1–5 yr		6–24	
(ng/mL)	6–9 yr		10–55	
	10–14 yr	23–70		6–40
	14–19 yr	23–70		6–40

Table 4–2. continued

Nutrient	*Age*	*Males*	*Either*	*Females*
Folic acid (nmol/L)	newborn		16–72	
	1–12 yr		4–20	
	adult		10–63	
Glucose (mg/dL)	0–1 mo		55–115	
	1–6 mo		57–117	
Growth hormone (μg/L)	overnight minimum			
	5–10 yr		> 3.0	
	11.7–15.5 yr		> 3.9	
	After stimulation test		> 5.0	
Hemoglobin (g/dL)	1–3 d	14.7–18.6		12.7–18.3
	4–7 d	13.4–17.9		12.2–18.7
	8–14 d	11.1–16.7		11.9–16.9
	15–30 d	9.9–14.9		10.5–14.7
	31–60 d	8.9–11.9		8.9–12.3
	2–6 mo	9.7–12.2		9.7–12.0
	6 mo–2 yr	10.3–12.4		10.4–12.4
	2–6 yr	10.5–12.7		10.7–12.7
	6–12 yr	11.0–13.3		10.9–13.3
	12–18 yr	11.5–14.8		11.2–13.6
	>18 yr	10.9–15.7		10.7–13.5
Hemoglobin A_{1C} (%)	Normals		4–6	
	Diabetic with average control		9–10	
Homocysteine, total (μmol/L)	2 mo–10 yr		3.3–8.3	
	11–15 yr		4.7–10.3	
	16–18 yr		4.7–11.3	
Insulin-like growth factor-1 (IGF-1) (ng/mL)	1–30 d	2–56		7–92
	1–6 mo	2–82		5–72
	6 mo–1 yr	2–54		8–75
	1–3 yr	17–116		12–135
	4–6 yr	21–160		13–176
	7–9 yr	65–207		53–253
	10–12 yr	69–196		75–357
	13–15 yr	80–404		66–344
	16–18 yr	96–383		99–364

Table 4–2. continued

Nutrient	*Age*	*Males*	*Either*	*Females*
Iron	1–5 yr		22–136	
(μmol/dL)	6–9 yr		39–136	
	10–14 yr	28–124		45–145
	14–19 yr	34–162		28–184
Iron-binding	1–5 yr		48–79	
capacity	6–9 yr		43–91	
(mmol/dL)	10–14 yr	54–91		57–103
	14–19 yr	52–102		52–101
Mean	1–3 d	97.3–109.8		99.4–113.8
corpuscular	4–7 d	95.5–109.3		97.9–111.6
volume (μm^3)	8–14 d	93.1–105.4		97.3–109.3
	15–30 d	88.7–101.2		91.8–102.5
	31–60 d	84.6–95.4		85.0–96.9
	2–6 mo	73.6–86.6		74.7–87.6
	6 mo–2 yr	70.5–81.2		71.5–81.8
	2–6 yr	72.7–83.6		73.8–84.3
	6–12 yr	75.9–86.5		76.8–87.6
	12–18 yr	77.9–89.9		79.4–91.0
	>18 yr	81.3–94.2		80.8–93.4
Osmolality	Birth		275–300	
(mOsm/kg)	7 d		276–305	
	28 d		274–305	
	Adult		282–300	
Phosphate	0–5 d (< 2.5 kg)		4.6–8.0	
(mg/dL)	1–3 yr		3.9–6.5	
	4–6 yr		4.0–5.4	
	7–9 yr		3.7–5.6	
	10–11 yr		3.7–5.6	
	12–13 yr		3.3–5.4	
	14–15 yr		2.9–5.4	
	16–19 yr		2.8–4.6	
Prealbumin	0–5 d		6–21	
(transthyretin)	1–5 yr		14–30	
(mg/dL)	6–9 yr		15–33	
	10–13 yr		20–36	
	14–19 yr		22–45	

Table 4–2. continued

Nutrient	*Age*	*Males*	*Either*	*Females*
Protein, total (g/dL)	0–5 d (< 2.5 kg)		3.8–6.2	
	0–5 d (>2.5 kg)		5.4–7.0	
	1–3 yr		5.9–7.0	
	4–6 yr		5.9–7.8	
	7–9 yr		6.2–8.1	
	10–19 yr		6.3–8.6	
Retinol-binding protein (RBP) (mg/dL)	0–5 d		0.8–4.5	
	1–5 yr		1.0–7.6	
	6–9 yr		2.0–7.8	
	10–13 yr		1.3–9.9	
	14–19 yr		3.0–9.2	
Selenium (μmol/dL)	0–5 d		6–9	
	1–5 yr		10–14	
	6–9 yr		10–16	
	10–14 yr		10–19	
	15–19 yr		10–19	
Transferrin (g/L)	0–5 d		1.43–4.46	
	1–3 yr		2.18–3.47	
	4–6 yr		2.08–3.78	
	7–9 yr		2.25–3.61	
	10–13 yr		2.24–4.42	
	14–19 yr		2.33–4.44	
Triglycerides (mg/dL)	1–3 yr		27–125	
	4–6 yr		32–116	
	7–9 yr		28–129	
	10–11 yr	24–137		39–140
	12–13 yr	24–145		37–130
	14–15 yr	34–165		38–135
	16–19 yr	34–140		37–140
Urea nitrogen (mg/dL)	1–3 yr		5–17	
	4–13 yr		7–17	
	14–19 yr		8–21	
Vitamin A (retinol) (μg/dL)	Preterm neonates		13–46	
	Term neonates		18–50	
	1–6 yr		20–43	
	7–12 yr		20–49	
	13–19 yr		26–72	

Table 4–2. continued

Nutrient	*Age*	*Males*	*Either*	*Females*
Vitamin B_1 (thiamine) (μg/dL)			1.6–4.0	
Vitamin B_6 (pyridoxine) (μg/L)			3.6–18.0	
Vitamin B_{12} (ng/L)			200–900	
Vitamin C (ascorbic acid) (mg/dL)			0.2–2.0	
Vitamin D (as 25-hydroxy D) (μg/L)	1–30 d	3.3–33.4		1.9–32.0
	1mo–1 yr	7.4–53.3		11.6–48.2
	1–3 yr	6.9–46.8		11.3–48.9
	4–12 yr	4.6–37.4		2.8–36.7
	13–18 yr	2.0–31.4		1.8–28.3
Vitamin D (as 1,25-dihydroxy D) (pg/mL)			15–60	
Vitamin E (μg/mL)	*Preterm neonates*		0.5–3.5	
	Term neonates		1.0–3.5	
	2–5 mo		2.0–6.0	
	6–24 mo		3.5–8.0	
	1–6 yr		3.0–9.0	
	7–12 yr		4.0–9.0	
	13–19 yr		6.0–10.1	
Zinc (μg/dL)	0–5 d		65–140	
	1–5 yr		67–118	
	6–9 yr		77–107	
	10–14 yr	76–101		79–118
	15–19 yr	64–104		60–101

Reproduced with permission from Soldin SJ, Brugnara C, Gunter KC, Hicks JM, editors. Pediatric reference ranges. 2nd ed. Washington (DC): AACC Press; 1997; and Alpers JB, editor. Laboratory handbook. 6th ed. Boston: Children's Hospital; 1988.

5

NUTRITIONAL REQUIREMENTS

DIETARY REFERENCE INTAKES

Linda Gallagher Olsen, MEd, RD

Dietary reference intakes (DRIs) represent the new approach adopted by the Food and Nutrition Board to provide quantitative estimates of nutrient intakes for use in a variety of settings. The DRIs are the result of a vast expansion in our understanding of the roles of nutrients and other food components that impact long-term health, going beyond deficiency states; they expand and replace the series of Recommended Dietary Allowances that have been published since 1941 by the Food and Nutrition Board. The DRIs consist of four reference intakes:

1. **Recommended Dietary Allowance (RDA):** a nutrient intake level used as a goal for the individual; a level sufficient to meet the nutrient requirements of nearly all (97 to 98%) healthy individuals in the group. The population recommendations are broken into gender, life cycle groups (infants, elderly, etc) with more specific age subgroups (see Tables 5–1 and 5–4).

2. **Estimated Average Requirement (EAR):** a nutrient intake level estimated to meet the requirements of half of the healthy individuals in a group. Used to assess the intake adequacy of population groups. The estimated average requirement is used along with knowledge of the distribution of requirements to develop RDAs; RDA is calculated as EAR + 2 standard deviations (see Table 5–5).

Table 5–1. Food and Nutrition Board, National Academy of Sciences: National Research Council Recommended Dietary Allowances (Revised 1989)

Designed for the maintenance of good nutrition of practically all healthy people in the United States

Category	Age (yr) or Condition	Weight* (kg)	Weight* (lb)	Height* (cm)	Height* (in)	Protein (g)	Vit A (μg RE)†	Vit E (mg α TE)†	Vit K (μg)	Vit C (mg)	Iron (mg)	Zinc (mg)	Iodine (μg)	Selenium (μg)
Infants	0–0.5	6	13	60	24	13	375	3	5	30	6	5	40	10
	0.5–1.0	9	20	71	28	14	375	4	10	35	10	5	50	15
Children	1–3	13	29	90	35	16	400	6	15	40	10	10	70	20
	4–6	20	44	112	44	24	500	7	20	45	10	10	90	20
	7–10	28	62	132	52	28	700	7	30	45	10	10	120	30
Males	11–14	45	99	157	62	45	1,000	10	45	50	12	15	150	40
	15–18	66	145	176	69	59	1,000	10	65	60	12	15	150	50
	19–24	72	160	177	70	58	1,000	10	70	60	10	15	150	70
	25–50	79	174	176	70	63	1,000	10	80	60	10	15	150	70
	51+	77	170	173	68	63	1,000	10	80	69	10	15	150	70
Females	11–14	46	101	157	62	46	800	8	45	50	15	12	150	45
	15–18	55	120	163	64	44	800	8	55	60	15	12	150	50
	19–24	58	128	164	65	46	800	8	60	60	15	12	150	55
	25–50	63	138	163	64	50	800	8	65	60	15	12	150	55
	51+	65	143	160	63	50	800	8	65	60	10	12	150	55

Pregnant		60	800	10	65	70	30	15	175	65
Lactating	0–6 mo	65	1,300	12	65	95	15	19	200	75
	6–12 mo	62	1,200	11	65	90	15	16	200	75

*Weights and heights of reference adults are actual medians for the US population of the designated age, as reported by National Health and Nutrition Examination Survey II (NHANES II). The median weights and heights of those under 19 years of age were reproduced with permission rom Hamill PVV, Drizd TA, Johnson CL, et al. Physical growth: National Center for Health Statistics percentiles. Am J Clin Nutr 1979;32:607–29. The use of these figures does not imply that the height-to-weight ratios are ideal.

†Retinol equivalents (RE). 1 RE = 1 μg retinol or 6 μg β-carotene.

‡α-Tocopherol equivalents (α-TE). 1 mg d-α-tocopherol = 1 α-TE.

3. **Adequate Intake (AI):** a level felt to meet the needs of all individuals in the group but for which there is much less clinical data than necessary to establish an RDA (underscoring the need for continued research on requirements for these nutrients) (see Table 5–2).
4. **Tolerable Upper Intake Level (UL):** the maximum level of daily nutrient intake that is unlikely to cause adverse health effects to almost all individuals of the group. These guidelines should be used to prevent consumption of too much of a nutrient.

The DRI project has been divided into seven nutrient groups:

1. Calcium, vitamin D, phosphorus, magnesium, fluoride
2. Folate and other B vitamins
3. Antioxidants (eg, vitamins C and E, selenium)
4. Macronutrients (eg, protein, fat, carbohydrates)
5. Trace elements (eg, iron, zinc)
6. Electrolytes and water
7. Other food components (eg, fiber, phytoestrogens)

At the time of this printing, two DRI reports (that include nutrients noted in groups #1 and #2, above) have been released. These and the 20th edition RDA tables for those nutrients not yet incorporated into the DRIs are included in this book for clinical reference. Also included is a table summarizing minimum requirements for selected minerals (Table 5–3).

Table 5–2. Summary of Estimated Safe and Adequate Daily Dietary Intakes of Selected Vitamins and Minerals*

		Vitamins		*Trace Elements*†				
Category	*Age (yr)*	*Biotin (μg)*	*Pantothenic Acid (mg)*	*Copper (mg)*	*Manganese (mg)*	*Fluoride (mg)*	*Chromium (μg)*	*Molybdenum (μg)*
Infants	0–0.5	10	2	0.4–0.6	0.3–0.6	0.1–0.5	10–40	15–30
	0.5–1	15	3	0.6–0.7	0.6–1.0	0.2–1.0	20–60	20–40
Children,	1–3	20	3	0.7–1.0	1.0–1.5	0.5–1.5	20–80	25–50
Adolescents	4–6	25	3	1.0–1.5	1.5–2.0	1.0–2.5	30–120	30–75
	7–10	30	4–5	1.0–2.0	2.0–3.0	1.5–2.5	50–200	50–150
	11+	30–100	4–7	1.5–2.5	2.0–5.0	1.5–2.5	50–200	75–250
Adults		30–100	4–7	1.5–3.0	2.0–5.0	1.5–4.0	50–200	75–250

Reproduced with permission from Food and Nutrition Board, National Academy of Sciences. Recommended Dietary Allowances 10e (revised 1989). Courtesy of the National Academy Press, Washington (DC).

*These figures are provided here in the form of ranges of recommended intakes since there is less information available on which to base allowances.

†Since the toxic levels for many trace elements may be only several times usual intakes, the upper levels for the trace elements given in this table should not be habitually exceeded.

Table 5–3. Summary of Estimated Sodium, Chloride, and Potassium Minimum Requirements of Healthy Persons*

Age	*Weight (kg)**	*Sodium (mg)*†*	*Chloride (mg)*†*	*Potassium (mg)*‡*
Months				
0–5	4.5	120	180	500
6–11	8.9	200	300	700
Years				
1	11.0	225	350	1,000
2–5	16.0	300	500	1,400
6–9	25.0	400	600	1,600
10–18	50.0	500	750	2,000
>18§	70.0	500	750	2,000

Reproduced with permission from Food and Nutrition Board, National Academy of Sciences-National Research Council. Recommended Dietary Allowances (revised 1989).
*No allowance has been included for large, prolonged losses from the skin through sweat.
†There is no evidence that higher intakes confer any health benefit.
‡Desirable intakes of potassium may considerably exceed these values.
§No allowance is included for growth. Values for those below 18 years assume a growth rate at the 50th percentile reported by the National Center for Health Statistics and averaged for males and females.

Table 5–4. Median Heights and Weights and Recommended Energy Intake

Category	*Age (yr) or Condition*	*Weight*		*Height*		*REE**	*Multiples of REE*	*Average Energy Allowance (kcal)*†*	
		(kg)	*(lb)*	*(cm)*	*(in)*	*(kcal/day)*		*Per kg*	*Per day‡*
Infants	0.0–0.5	6	13	60	24	320		108	650
	0.5–1.0	9	20	71	28	500		98	850

Children	1–3	13	29	90	35	740		102	1,300
	4–6	20	44	112	44	950		90	1,800
	7–10	28	62	132	52	1,130		70	2,000
Males	11–14	45	99	157	62	1,440	1.70	55	2,500
	15–18	66	145	176	69	1,760	1.67	45	3,000
	19–24	72	160	177	70	1,780	1.67	40	2,900
	25–50	79	174	176	70	1,800	1.60	37	2,900
	51+	77	170	173	68	1,530	1.50	30	2,300
Females	11–14	46	101	157	62	1,310	1.67	47	2,200
	15–18	55	120	163	64	1,370	1.60	40	2,200
	19–24	58	128	164	65	1,350	1.60	38	2,200
	25–50	63	138	163	64	1,380	1.55	36	2,200
	51+	65	143	160	63	1,280	1.50	30	1,900
Pregnant	1st tri								+0
	2nd tri								+300
	3rd tri								+300
Lactating	0–6 mo								+500
	2nd 6 mo								+500

REE = resting energy expenditure.
Reproduced with permission from Food and Nutrition Board. National Academy of Sciences-National Research Council. Recommended Dietary Allowances (revised 1989).
*Calculation based on FAO equations, then rounded.
†In the range of light to moderate activity, the coefficient of variation is ± 20%.
‡Figure is rounded.

Table 5–5. Food and Nutrition Board, Institute of Medicine-National Academy of Sciences—Dietary Reference Intakes: Recommended levels for individual intake[a]

Life-Stage Group	*Ca (mg/d)*	*P (mg/d)*	*Mag (mg/d)*	*Vit D[bc] (mcg/d)*	*Fluor (mg/d)*	*Thiamin (mg/d)*	*Riboflavin (mg/d)*	*Niacin[d] (mg/d)*	*Vit B-6 (mg/d)*	*Folate[e] (mcg/d)*	*Vit B-12 (mcg/d)*	*Pantothenic Acid (mg/d)*	*Biotin (mcg/d)*	*Choline[f] (mg/d)*
Infants														
0–6 mo	210	100	30	5	0.01	0.2	0.3	2	0.1	65	0.4	1.7	5	125
7–12 mo	270	275	75	5	0.5	0.3	0.4	4	0.3	80	0.5	1.8	6	150
Children														
1–3 y	500	**460**	**80**	5	0.7	**0.5**	**0.5**	**6**	**0.5**	**150**	**0.9**	2	8	200
4–8 y	800	**500**	**130**	5	1	**0.6**	**0.6**	**8**	**0.6**	**200**	1.2	3	12	250
Males														
9–13 y	1,300	**1,250**	**240**	5	2	**0.9**	**0.9**	**12**	**1.0**	**300**	**1.8**	4	20	375
14–18 y	1,300	**1,250**	**410**	5	3	**1.2**	**1.3**	**16**	**1.3**	**400**	**2.4**	5	25	550
19–30 y	1,000	**700**	**400**	5	4	**1.2**	**1.3**	**16**	**1.3**	**400**	**2.4**	5	30	550
31–50 y	1,000	**700**	**420**	5	4	**1.2**	**1.3**	**16**	**1.3**	**400**	**2.4**	5	30	550
51–70 y	1,200	**700**	**420**	10	4	**1.2**	**1.3**	**16**	**1.7**	**400**	**2.4[g]**	5	30	550
> 70 y	1,200	**700**	**420**	15	4	**1.2**	**1.3**	**16**	**1.7**	**400**	**2.4[g]**	5	30	550
Females														
9–13 y	1,300	**1,250**	**240**	5	2	**0.9**	**0.9**	**12**	**1.0**	**300**	**1.8**	4	20	375
14–18 y	1,300	**1,250**	**360**	5	3	**1.0**	**1.0**	**14**	**1.2**	**400[h]**	**2.4**	5	25	400
19–30 y	1,000	**700**	**310**	5	4	**1.1**	**1.1**	**14**	**1.3**	**400[h]**	**2.4**	5	30	425
31–50 y	1,000	**700**	**320**	5	4	**1.1**	**1.1**	**14**	**1.3**	**400[h]**	**2.4**	5	30	425
51–70 y	1,200	**700**	**320**	10	4	**1.1**	**1.1**	**14**	**1.5**	**400**	**2.4[g]**	5	30	425
>70 y	1,200	**700**	**320**	15	4	**1.1**	**1.1**	**14**	**1.5**	**400**	**2.4[g]**	5	30	425

Pregnancy														
# 18 y	1,300	**1,250**	**400**	5	3	**1.4**	**1.4**	**18**	**1.9**	**600[l]**	**2.6**	6	30	450
19–30 y	1,000	**700**	**350**	5	3	**1.4**	**1.4**	**18**	**1.9**	**600[l]**	**2.6**	6	30	450
31–50 y	1,000	**700**	**360**	5	3	**1.4**	**1.4**	**18**	**1.9**	**600[l]**	**2.6**	6	30	450
Lactation														
# 18 y	1,300	**1,250**	**360**	5	3	**1.5**	**1.6**	**17**	**2.0**	**500**	**2.8**	7	35	550
19–30 y	1,000	**700**	**310**	5	3	**1.5**	**1.6**	**17**	**2.0**	**500**	**2.8**	7	35	550
31–50 y	1,000	**700**	**320**	5	3	**1.5**	**1.6**	**17**	**2.0**	**500**	**2.8**	7	35	550

[a]Recommended Dietary Allowances (RDAs) are presented in bold type and Adequate Intakes (AIs) in regular type. RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97% to 98%) individuals in a group. For healthy breastfed infants, the AI is the mean intake. The AI for other life-stage and gender groups is believed to cover needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of persons covered by this intake. Source: The National Academy of Sciences. ©1998.

[b]As cholecalciferol. 1 mcg cholecalciferol = 40 IU vitamin D.

[c]In the absence of adequate exposure to sunlight.

[d]As niacin equivalents (NE). 1mg niacin = 60 mg tryptophan; 0 to 6 mo = preformed niacin (not NE).

[e]As dietary folate equivalent (DFE). 1DFE = 1 mcg food folate = 0.6 (g folic acid (from fortified food or supplement) consumed with food = 0.5 µg synthetic (supplemental) folic acid taken on an empty stomach.

[f]Although AIs have been set for choline, there are few data to assess whether a dietary supply of choline is needed at all stages of the life cycle, and it may be that the choline requirement can be met by endogenous synthesis at some of these stages.

[g]Because 10% to 30% of older people may malasorb food-bound vitamin B-12, it is advisable for those older than 50 years to meet their RDA mainly by consuming foods fortified with vitamin B-12 or a supplement containing vitamin B-12.

[h]In view of evidence linking folate intake with neural tube defects in the fetus, it is recommended that all women capable of becoming pregnant consume 400 mcg synthetic folic acid from fortified foods and/or supplements in addition to intake of food folate from a varied diet.

[i]It is assumed that women will continue consuming 400 mcg folic acid until their pregnancy is confirmed and they enter prenatal care, which ordinarily occurs after the end of the periconceptional period—the critical time for formation of the neural tube.

6

BREASTFEEDING

Jill Kostka Fulhan, MPH, RD

It is now widely recognized that breastmilk is the optimal choice of feeding for both full-term and premature newborn infants. The nutritional, immunologic, physiologic, health, psychologic, and socioeconomic benefits to baby and mother are many (Table 6–1), prompting a recent increase in the number of mothers initiating breastfeeding postpartum. Despite an increase to nearly 60% of mothers breastfeeding their newborns in 1995, however, only 21.6% continued to breastfeed infants aged 5 to 6 months.[1] While promising, these numbers fall far below the Healthy People 2000 goals of a minimum 75% of mothers breastfeeding initially and 50% continuing to breastfeed infants up to the age of 5 to 6 months.[2]

The American Academy of Pediatrics, in conjunction with the World Health Organization, strongly recommends that infants receive breastmilk solely through the first 6 months of life and continue to receive breastmilk with complementary foods through at least the first year of life.[3,4] To achieve this goal, the breastfeeding family should be provided with support and education from the health and medical profession as well as the community, workplace, and media. Emotional support, combined with the knowledge of how to establish and continue breastfeeding, will help the breastfeeding dyad succeed, despite the many barriers existing today.

Nearly all mothers can successfully breastfeed their newborn infants. The health care professional should ensure that all women of child-bearing age understand the

Table 6–1. Benefits of Breastfeeding

Infant Nutrition

Protein
- Whey predominant ratio to casein (70:30), easily digestible
- Promotes rapid gastric emptying
- High biologic value protein

Fat
- Provides 40–50% calories
- Bile salt–stimulated lipase and lipoprotein lipase readily break down triglycerides
- Contains essential fatty acids
- LCFAs: DHA and arachidonic acid may improve vision and cognition
- Cholesterol: essential for CNS development, may influence cholesterol metabolism

Carbohydrate
- Lactose: enhances calcium absorption, breaks down into galactose and glucose for energy to the brain
- Amylase: aids in the digestion of starch, may aid with glucose polymers as well

Nonprotein nitrogen: free amino acids for growth

Iron: increased iron absorption

Immunologic

Protection against infection and allergy
- Secretory IgA: passive immunologic protection via the enteromammary immune system
- Lactoferrin: iron-binding protein, reduces the iron-binding sites available for iron-dependent pathogens
- Lysozyme: antimicrobial factor
- *Lactobacillus bifidus*: promotes the growth of beneficial bacteria, inhibits growth of enteropathogens
- Leukocytes: includes macrophages and lymphocytes to fight infection

Low risk of contamination of feed if fed directly from breast

Reduced incidence and/or severity of:
- Diarrhea
- Otitis media
- Lower respiratory infection

Table 6–1. continued

- Bacteremia
- Bacterial meningitis
- Botulism
- Urinary tract infection
- Necrotizing enterocolitis

Possible protection against:

- SIDS
- IDDM
- Crohn's disease
- Ulcerative colitis
- Lymphoma
- Allergic diseases
- Other chronic digestive diseases

Physiologic

Water content: 87.5% of volume, to maintain hydration

Low renal solute load: aids in kidney function

Aids in intestinal maturation (via growth factors, hormones)

Infant self-regulates intake based on need, via "feeding on demand"

Breastmilk composition changes based on infant need:

- Colostrum, transitional, and mature milk, preterm vs. term milk
- Changes occur during each feeding, from morning to evening, throughout entire breastfeeding course

Psychologic

Bonding between mother and infant

Mutual caregiving

Maternal Health Benefits

Increased levels of oxytocin, leading to less postpartum bleeding, rapid uterine involution

Earlier return to prepregnancy weight

Delayed resumption of ovulation, increased child spacing

Improved bone remineralization postpartum

Reduction in hip fractures in the postmenopausal period

Reduced risk of ovarian cancer

Reduced risk of premenopausal breast cancer

Socioeconomic
Parents have more time available for infant/siblings (no need to obtain and prepare formula)
Reduced employee absenteeism secondary to decreased infant/child illness
Reduced lost family income secondary to less absenteeism at work
Savings of > $400 per child on total annual food purchases per family
Federal health care cost savings, secondary to less infant/child morbidity, decreased WIC formula distribution
Recent expansion of employee breastfeeding programs to help meet the working mother's needs

Adapted in part from American Academy of Pediatrics, Work Group on Breastfeeding. Breastfeeding and the use of human milk. Pediatrics 1997;100:1035–9.

LCFAs = long chain fatty acids; IDDM = insulin-dependent diabetes mellitus; DHA = docosahexaenoic acid; CNS = central nervous system; IgA = immunoglobulin A; SIDS = sudden infant death syndrome; WIC = Special Supplemental Food Program for Women, Infants, and Children.

benefits of breastfeeding and the steps involved in successfully initiating it (Table 6–2).

Accurate assessment of the mother/infant dyad is essential in determining an infant's nutritional and hydration status, especially for the hospitalized infant. Breastfeeding is too often prematurely discontinued, because health care staff are concerned about nourishment but are unable to determine whether the baby is getting enough milk. Despite good intentions, mothers often receive discouraging messages about their ability to feed their child. To improve assessment of feeding and identify areas in need of intervention, breastfeeding assessment tools designed for clinicians, such as one described in Tables 6–3 and 6–4, may be employed.

Guidelines for expression and storage of breastmilk are outlined in Tables 6–5 and 6–6. Common concerns are

Table 6–2. Steps to Successful Breastfeeding: Guidelines for Health Care Professionals

Prenatal

Plan ahead, encourage mother to:
- Attend breastfeeding classes and read about breastfeeding
- Observe another mother feeding her infant at breast (to help understand how it works)
- Identify a support person(s): family, friends with positive breastfeeding experience, lactation consultant
- Obtain quality breast pump if she plans to return to work while breastfeeding, learn how to use it

Choose an obstetrician, hospital, and pediatrician who are "baby friendly" (see Table 6–9):
- Medical professionals who are knowledgeable and supportive of breastfeeding
- Lactation support available 24 hours/day in hospital and through doctor's office referral
- Breastfeeding encouraged as soon as possible after delivery
- Rooming-in of mother and infant is practiced
- Hospital staff will not provide formula unless medically necessary

Breast/nipple examination:
- Examine breasts for potentially inverted or flat nipples (will impede latch)
- If discovered, mother may benefit from wearing breast shells prior to delivery
- Refer mother to lactation consultant (in anticipation of potentially difficult latch)

Postpartum

Initiation of breastfeeding:
- Should commence as soon as possible after delivery, preferably within 1 hour
- Encourage feeding "on demand," as frequently as every hour, with no more than 4 hours between feeds
- Explain that first days are crucial to let-down and establishment of the milk supply
- Discuss infant hunger cues—increased activity, alertness, rooting—and emphasize to not wait until infant is crying

Assess mother/infant dyad:
- Intermittent assessment is essential

Table 6–2. continued

- Observe positioning, infant latch, suck, and swallow (see Tables 6–3 and 6–4 on LATCH guidelines)
- Remedy any problems that arise and/or have resources for further assistance

Initiation of breastmilk expression:
- Begin as soon as possible if infant must be separated from mother
- Recommend mother pump every 3 hours, including throughout the night, to establish milk supply
- Instruct mother on proper storage and handling techniques (see Tables 6–5 and 6–6)

Encouragement of breastfeeding:
- Support from father of baby, family, and friends toward mother's decision to breastfeed is crucial
- Reinforcement from doctor, nurse, dietitian, IBCLC also important

Alternative methods of feeding breastmilk:
- Recommend appropriate method when mother not available for every feeding at breast
- Use judiciously: cup feeding, syringe feeding, finger feeding to prevent nipple confusion
- Aids transition from tube feeding to feeding at breast

Establishing the Milk Supply

The law of supply and demand:
- The more the infant feeds, the more milk the mother will produce
- Infant should breastfeed 8–12 times a day
- Infant must breastfeed during the night
- Infant should breastfeed q 1–3 hours at first, may go longer between feeds when older
- Mother should alternate initial breast offered at each feeding
- Equal emptying of breasts is important (more vigorous suck at first breast)
- Inform mother that breastfed infants may nurse more often than formula-fed infants take the bottle

Positioning:
- Optimal milk production requires an effective latch, which requires proper positioning
- Types of positions: tummy-to-tummy cradle hold, football hold, lying on side

Table 6–2. continued

- Important to bring baby to level of breast
- May need to support breast: C-hold, scissor hold, dancer hold
- Ensure mother is as comfortable and relaxed as possible, offer pillow and footrest for support

Latch:
- Assure proper positioning
- Mother can tickle infant's bottom lip with nipple to help infant open mouth
- As infant opens mouth wide, mother should draw infant to breast
- Baby should have all of nipple and as much areola in mouth as possible
- If latch is not right the first time, mother should release infant and try again
- To release, place finger in corner of baby's mouth to break seal, then draw infant away slowly

Inadequate milk production may be due to:
- Insufficient, infrequent feeding (the most common and preventable cause)
- Hesitation/failure to wake sleeping baby to feed
- Excessive use of a pacifier
- Baby reluctant to feed (can be a sign of inadequate feeding as well)
- Mother unaware of necessity of frequent feeds, especially immediately postpartum
- Intermittent bottle feeding: leads to missed opportunities to feed and produce more milk
- Bottle feeds not recommended until 4–6 weeks, when milk supply established

Follow-Up Care

Early discharge from hospital:
- Many newborns and mothers are home < 48 hours after delivery, before breastmilk "comes in"
- AAP recommends follow-up at 24–48 hours after discharge and again within 2 weeks after discharge
- Doctor, visiting nurse, or other health care professional recommended for follow-up care

Clinical assessment includes:
- Infant hydration status
- Breastmilk intake, use of supplements/formula

- Weight gain
- Presence of jaundice
- See LATCH assessment guidelines (Tables 6–3 and 6–4)

Maternal needs:

- Address questions, concerns, possible anxieties of mother
- Refer to lactation consultant if necessary

Returning to Work

Best time to plan for return to work is during pregnancy. Need to consider:

- Length of maternity leave: the longer the leave, the more success with continued breastfeeding
- Workplace: times and locations available to express breastmilk, employee program/IBCLC available?
- Daycare: proximity to work to allow breastfeeding visit, feelings of staff toward handling and provision of breastmilk
- Method of breastmilk expression: double pump set-up available for most efficient pumping
- Emotional needs: expect and try to prepare for emotional highs and lows, separation from baby

IBCLC = International Board Certified Lactation Consultant; AAP = American Academy of Pediatrics.

addressed in Table 6–7, with contraindications to breastfeeding shown in Table 6–8. Guidelines for breastfeeding support and promotion in hospitals are provided in Table 6–9. Further reading is suggested as is networking with an International Board Certified Lactation Consultant (IBCLC). See "Additional Resources" at the end of this chapter for contact information.

Breastfeeding Promotion

The World Health Organization (WHO) and United Nations International Children's Emergency Fund (UNICEF) published a joint statement in 1989 entitled *Protecting, Promoting and Supporting Breastfeeding: The Special Role of Maternity Services*, with a view to increasing global awareness of the impact of health care services

Table 6–3. LATCH Assessment Guidelines for Lactation

Goal:
- Systematic assessment of the breastfeeding dyad
- Identifies areas requiring intervention and/or education
- Serves as a communication and documentation tool among health care professionals
- Increases consistency of evaluation
- Repeated scoring identifies changes in the breastfeeding experience

Charting system:
- Based on the Apgar scoring system
- A numerical score is assigned to each component for each session observed
- Scores are added, total score determines "success" of infant feeding
- Higher score (9–10) indicates successful breastfeeding with minimal assistance
- Lower score (4–5) indicates greater assistance required, impaired feeding
- Individual low score for particular component shows where assistance is needed

Adapted from Jensen D,Wallace S, Kelsay P. LATCH: a breastfeeding charting system and documentation tool. J Obstet Gynecol Neonatal Nurs 1994;23:27–32.

on breastfeeding advocacy. From their statement came the "Ten Steps to Successful Breastfeeding," a guideline for hospitals and other health care providers to follow to ensure that breastfeeding is promoted and adopted in their facility. Proven practice of these steps, listed in Table 6–9, will, in part, earn the facility a "baby friendly" designation. The Baby Friendly Hospital Initiative (BFHI) began in 1992, and health care facilities working toward certification now span the globe.

Table 6–4. LATCH Scoring Table

		0	*1*	*2*
L	Latch	Baby too sleepy	Repeated attempts	Grasps breast
		No latch achieved	Holds nipple in mouth	Lips flanged out
			Stimulation to suck	Tongue down
				Rhythmic sucking
A	Audible swallow	None	A few with stimulation	Spontaneous
				Intermittent to frequent
T	Type of nipple	Inverted	Flat	Everted after stimulation
C	Comfort	Breasts engorged	Filling breasts	Soft breast
		Cracked nipples	Reddened nipples	Tender
		Severe discomfort	Mild/moderate discomfort	
H	Hold	Staff assists with	Minimal assistance	No assistance
		holding of infant	Mother takes over	Mother able to position and hold

Adapted from Jensen D,Wallace S, Kelsay P. LATCH: a breastfeeding charting system and documentation tool. J Obstet Gynecol Neonatal Nurs 1994;23:27–32.

Table 6–5. Expression of Breastmilk

Why express breastmilk?
- Separation of mother and infant due to maternal or infant illness, prematurity, return to work
- Infant unable to feed by mouth (eg, neurologic problems, ventilator dependency, difficulty with cleft lip/palate)
- Increases or maintains breastmilk supply

Methods of breastmilk expression
- Most appropriate choice is based on individual pumping needs, number of times/day needed to pump
- *Manual expression, battery-operated pump, electric pump* (single and double setup)

Obtaining a pump
- Rental stations, check local breastfeeding support group for locations
- Manufacturer (see names/addresses in Additional Resources on p. 104–105)
- Pharmacy/medical supply store (usually just sell/rent pump, no assistance provided)
- Check for established pumping rooms in hospital and how to obtain supplies

Expression of breastmilk
- Each pump is different; read all instructions prior to use, including how to clean the pump
- Wash hands, find a clean area to pump, have bottles ready to collect milk
- Begin with hand expression to start milk flowing (if needed), progress to pump, start with low suction
- Massage breasts while pumping to increase flow, decrease pumping time
- *Pump each breast 10–20 minutes or until flow decreases*
- If double pumping, may simultaneously pump both breasts in 10–15 minutes
- Label each bottle of breastmilk with baby's name, and date and time pumped

Storing breastmilk
- Always provide infant with fresh pumped milk first, then refrigerated, then frozen
- If unable to use fresh breastmilk, follow storage guidelines in Table 6–6

Preparation of stored breastmilk
- Choose oldest pumped milk first, check date for possible expiration
- To thaw and warm breastmilk, run under lukewarm water or submerge in a basin of it; may need to replace warm water in basin until milk is at room temperature
- *Never* microwave breastmilk as uneven heating may cause hot spots and damage immune properties

Table 6–6. Guidelines for the Storage of Breastmilk*

	Room Temperature	*Refrigerator (Do Not Store in Door)*	*Freezer < -20°C (Average Top or Side Freezer Unit)*	*Deep Freeze < -70°C (Separate Unit)*
Fresh breast milk	6 hours (refrigerate immediately if not to be used within 6 hours)	48 hours (can be safely stored up to 5 days; if not to be used in 48 hours, then freeze)	4 months (do not store in door, place toward back)	6–12 months (not available in hospital. Send excess milk home for freezing, or consider donation to human milk bank)
Thawed breastmilk	1 hour	24 hours (re-label after thawing, with date and time thawed. Discard after 24 hours if not used)	*Do not re-freeze*	*Do not re-freeze*

Table 6–7. Addressing Common Concerns

"Is my baby getting enough milk?"

This is mother's greatest fear! Reassure adequate milk is being received if the following is occurring:

- 6–8 soaked wet diapers per day
- consistent stooling pattern (breastfed babies rarely get constipated)
- weight gain is meeting goal
- audible suck is heard for majority of feeding

Refer to LATCH guidelines (Tables 6–3 and 6–4)

Vitamin and Mineral Supplements

In most cases, the healthy, full-term, breastfed infant requires little or no supplementation

Vitamin K

All infants, breastfed or not, should receive vitamin K at birth

Vitamin D

Recommended for infants who are dark skinned with little exposure to sunlight

Recommended for infants whose mothers are deficient

Fluoride

Per AAP, no supplementation < 6 months; 3 months to 6 years dependent on local water fluoride content[6]

Iron

Absorption from breastmilk is excellent

Term infant should have sufficient stores for first 6 months of life

Preterm infant may require supplement (see Chapter 34, Prematurity)

Additional iron should be received via iron-rich solids in the second 6 months of life

Water

Generally *not* recommended, may be required in hot/humid climates

Breastmilk is 87.5% water, will provide adequate hydration with adequate volume

Table 6–7. continued

Advancing the Breastfed Infant's Diet

Breastmilk is complete nutrition up to 6 months

May initiate cup feeding at 7 months for other liquids

See Chapter 7, Development of Feeding Skills/Introduction of Solids

Jaundice

Early Onset/Breastfeeding Jaundice	*Late Onset/Breastmilk Jaundice*
Occurs after 24 hours of age	Occurs on day of life 5–10
Peaks on day of life 3 or 4	Peaks on day of life 10, may persist
Associated with inadequate/infrequent feeding, decreased stooling, water/D_5 supplements	Infant is healthy, growing, breastfeeding well, stooling well
Treatment	*Treatment*
Determine cause of poor breastfeeding	Rule out other causes (eg, hemolytic disease, G6PD deficiency, hypothyroidism)
Provide necessary intervention	May require phototherapy
Encourage frequent feeding to increase milk supply	May have to discontinue breast milk 12 to 48 hours to rule out as cause of jaundice
Monitor stooling, stimulate if necessary	Substitute formula until bilirubin level drops
May require phototherapy	Encourage and assist mother with pumping to maintain milk supply

Prematurity

Breastmilk is ideal, given above benefits

Fortification likely required given premature infant's suboptimal nutritional reserves

Mother may need lactation consultant for long-term pumping support and transition of infant to breast

May benefit from alternative feeding choice prior to feeding at breast to avoid nipple confusion

See Chapter 34, Prematurity

AAP = American Academy of Pediatrics; G6PD = glucose-6-phosphate dehydrogenase.

Table 6–8. Contraindications

Galactosemia

Infant is unable to metabolize lactose, the primary sugar found in breastmilk. The infant is given a lactose-free formula

Phenylketonuria (PKU)

Infant is unable to properly metabolize phenylalanine. Breastmilk is low in phenylalanine but must be alternated with a phenylalanine-free formula to control levels

Tuberculosis

Women with untreated, active tuberculosis should not breastfeed

Initiate pumping and discard milk until 1–2 weeks of treatment is completed, then resume feeding at breast

Monitor for isoniazid (INH) accumulation, hepatotoxicity

Human Immunodeficiency Virus (HIV) / Acquired Immunodeficiency Syndrome (AIDS)

When safe breastmilk substitutes are available, women with HIV should *not* breastfeed their infants to prevent mother-to-child transmission of the HIV virus via breastfeeding

Replacement feeding (the process of feeding a child who is not receiving any breastmilk with a diet that provides all the nutrients the child needs) must be safe, to outweigh the risks of not breastfeeding

Replacement feeding must be available, attainable, and acceptable to the mother providing it

Education and resources are necessary for HIV+ mothers regarding feeding alternatives and the risk of HIV transmission[7]

Hepatitis C

Breastfeeding is contraindicated in new mothers with hepatitis C

Controversial in mothers who are diagnosed once lactation has been established

Drug Abuse

Women who use the following should *not* breastfeed:

- Amphetamines
- Cocaine

Table 6–8. continued

- Heroin
- Marijuana
- Phencyclidine (PCP)

Nicotine/cigarette smoking: contraindicated
Nicotine passes easily into milk
May impair maternal let-down, decrease milk supply
May result in infant colic, restlessness
If mother continues to smoke, suggest:

- Limit to < 6 cigarettes/day
- Smoke 2–3 hours before feeding
- Do not smoke while feeding the baby
- Smoke right after feeding to increase time between smoking and next feeding

Alcohol: contraindicated
Passes into breastmilk
May impair infant growth and development
Increased amounts can affects mother's ability to care for infant
If mother desires one drink, take after feeding and allow 2 hours to pass until next feeding

Maternal Medications

Most medications are considered compatible with breastfeeding. Refer to the AAP article,[8] and texts by Hale[9] and Briggs, Freeman, and Yaffe.[10] Of note, the PDR is *not* a good source of information regarding medications and lactation

Contraindicated	*Effect Unknown/May Be of Concern*
Bromocriptine	Chloramphenicol
Cyclophosphamide	Metoclopramide
Cyclosporine	Metronidazole
Doxorubicin	Tinidazole
Ergotamine	Antianxiety medications
Lithium	Antidepressant medications
Methotrexate	Antipsychotic medications
Phenindione	
Radioactive compounds*	

AAP = American Academy of Pediatrics; PDR = Physician's Desk Reference.

* Requires temporary cessation of breastfeeding. Pump and discard breastmilk until radioactivity is no longer present in milk.

Table 6–9. The Baby Friendly Hospital Initiative (BFHI)

The Ten Steps to Successful Breastfeeding[11]

1. Have a written breastfeeding policy that is routinely communicated to all health care staff.
2. Train all health care staff in skills necessary to implement this policy.
3. Inform all pregnant women about the benefits and management of breastfeeding.
4. Help mothers initiate breastfeeding within an hour of birth.
5. Show mothers how to breastfeed and how to maintain lactation, even if they should be separated from their infants.
6. Give newborn infants no food or drink other than breastmilk, unless medically indicated.
7. Practice "rooming in" by allowing mothers and infants to remain together 24 hours a day.
8. Encourage breastfeeding on demand.
9. Give no artificial teats, pacifiers, dummies, or soothers to breastfeeding infants.
10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or birthing center.

References

1. Ryan AS. The resurgence of breastfeeding in the United States. Pediatrics 1997;99(4). URL: http://www.pediatrics.org/cgi/content/full/99/4/e12.
2. Healthy People 2000: National Health Promotion and Disease Prevention Objectives. Washington (DC): Dept. of Health and Human Services (US); 1990. Publication PHS 91-50212.
3. American Academy of Pediatrics, Work Group on Breastfeeding. Breastfeeding and the use of human milk. Pediatrics 1997;100:1035–9.
4. World Health Organization/UNICEF. Innocenti Declaration on the Protection, Promotion and Support of Breastfeeding. Breastfeeding in the 1990s: Global Initiative WHO/UNICEF Sponsored Meeting, 1990; Florence, Italy.

5. Jensen D, Wallace S, Kelsay P. LATCH: a breastfeeding charting system and documentation tool. J Obstet Gynecol Neonatal Nurs 1994;23:27–32.
6. American Academy of Pediatrics, Committee on Nutrition. Fluoride supplementation for children: interim policy recommendations. Pediatrics 1995;95:777.
7. World Health Organization. HIV and infant feeding: guidelines for decision makers. Geneva: World Health Organization; 1998.
8. American Academy of Pediatrics Committee on Drugs. The transfer of drugs and other chemicals into human milk. Pediatrics 1994;93:137–50.
9. Hale T. Medications and mothers milk. 8th ed. Amarillo (TX): Pharmasoft Medical Publishing; 1999.
10. Briggs GG, Freeman RK, Yaffe SJ. Drugs in pregnancy and lactation. 5th ed. Baltimore: Williams & Wilkins; 1998.
11. Protecting, Promoting and Supporting Breastfeeding: The Special Role of Maternity Services. A Joint WHO/UNICEF Statement. Geneva: World Health Organization; 1989.

Additional Resources

Books and Journals

1. Riordan J, Auerbach K. Breastfeeding and human lactation. Boston (MA): Jones and Bartlett Publishers; 1999.
2. Lawrence R. Breastfeeding: a guide for the medical profession. St. Louis (MO): Mosby-Year Book, Inc.; 1998.
3. Journal of Human Lactation
   Imprint Publications, Inc. 230 E Ohio Street, Suite 300, Chicago, IL 60611
   Official journal of ILCA, quarterly

Support Groups

ILCA
International Lactation Consultant Association
4101 Lake Boone Trail
Raleigh, NC 27607
919-787-5181 www.ILCA.org

HMBANA
Human Milk Banking Association of North America
8 Jan Sebastian Way
Sandwich, MA 02563 508-888-4041

LLLI
La Leche League International
1400 North Meacham Road
Schaumburg, IL 60173
847-519-7730 www.LaLecheLeague.org

WABA
World Alliance for Breastfeeding Advocacy
NABA (North America contact)
254 Conant Road
Weston, MA 02193-1756
www.elogica.com.br/waba

Lactation Education/Certification

IBLCE
International Board of Lactation Consultant Examiners
PO Box 2348
Falls Church, VA 22042
703-560-7330 www.iblce.org/

Breastfeeding Support Consultants
228 Park Lane
Chalfont, PA 18914
215-822-1281

Breast Pump Rental Information

Ameda Egnell (Hollister, Inc.)
2000 Hollister Drive
Libertyville, IL 60048
800-323-8750 www.hollister.com

Medela, Inc.
4610 Prime Parkway
McHenry, IL 60050
800-435-8316 www.medela.com

7

INTRODUCTION OF SOLIDS

Lauren R. Furuta, MOE, RD

Progression of the infant diet from breastmilk or formula to solid foods is dependent on multiple factors, including the development of oral motor skills. Table 7–1 outlines the normal development of sucking, swallowing, biting, and chewing skills and how they impact on the infant's ability to consume liquids and solids. Texture, consistency, type, and amount of food should complement the infant's feeding skills and development of the gastrointestinal tract. The infant feeding guide (Table 7–2) outlines the volume of fluids and amounts and textures of food categories which provide the optimal nutrition at various intervals during the first year of life. Initially, single ingredient foods should be introduced. The introduction of 1 new food every 3 to 4 days is recommended so that potential food intolerances may be easily identified.

Table 7–1. Normal Development of Oral-Motor Feeding Skills

	Feeding Behavior	*Sucking Skills*	*Swallowing Skills*	*Biting Skills*	*Chewing Skills*
Birth	• Bottle or breast fed liquids • Rooting reaction • Fed reclined < 45 degrees • Rarely drools • Physiologic flexion	• 1 to 2 sucks-swallow-breathe • Front to back tongue movement with slight tongue protrusion • Flat, thin tongue with central groove. Sucking pads evident. • Loses some liquid • Sucking cued by touch	• Strong gag reflex • Reflexive swallow	• Phasic bite reflex upon stimulation of the gums	
3 mo	• Bottle or breastfed • Fed reclined 45 to 90 degrees • Soft or pureed food may be given • Some food may be pushed out of mouth	• Sequential sucking 20+ sucks followed by swallowing • Primitive suck-swallow pattern used to remove food from spoon • No assistance from	• Suck-swallow reflex • Infrequent pauses for breathing • Occasional choking, gagging, or vomiting can occur with introduction of		

Table 7–1. continued

	Feeding Behavior	*Sucking Skills*	*Swallowing Skills*	*Biting Skills*	*Chewing Skills*
3 mo	• Taste buds begin to develop and salivary glands become active by 4 months • Vocalizes	lips to clean off spoon • Tongue thrust reflex diminishes • Wide jaw excursion	solid foods • Swallowing disorders become more easily identified as anatomy no longer protects airway		
6 mo	• Cereal and pureed foods are introduced (4–6 mo) • Fed in a semi-sitting position with external support • Cup drinking introduced • Self-feeds finger foods • Rooting reaction diminishes • Drooling noted	• True suck pattern emerges with bottle, breast, cup, and spoon feedings • Long sequences of sucking, swallowing and breathing • Up-down tongue movements emerge	• No observable tongue tip elevation during cup drinking. Lips may be open during the swallow with liquid loss noted • Front-to-back tongue movements or simple protrusion characterizes swallowing of semi-solid, solid and liquids by cup	• Primitive phasic bite and release pattern with a regular rhythmn on soft cookie "munching" • May revert to sucking cookie • Diminished phasic bite reflex	• Poorly controlled munching emerges interspersed with sucking pattern • Diagonal rotary chewing stimulated when food placed on side of mouth • Tongue lateralization emerges as gross rolling movements or shifts when

	during babbling, reaching, pointing, manipulating, and teething • Anticipates food and opens mouth for spoon. Becomes excited • Bangs cup on table. Holds bottle • Babbling emerges		• Uncoordinated swallowing during longer cup drinking sequences may result in liquid loss and coughing and choking episodes • Diminished gag reflex		food placed on side of mouth
9 mo	• Liquids, pureed foods, ground or junior foods, and mashed table food are presented • Fed sitting with no external support other than high chair frame and safety belt	• More refined sucking skills evident through front-to-back and up-down tongue movements • No longer loses any liquid by bottle or breast • Upper lip assists in food removal from spoon by	• During swallowing of semisolids an up-down sucking pattern is used with simple tongue protrusion and intermittent front-to back tongue movements • Longer sequences of continuous sucking during cup	• Holds soft cookie between teeth, but can't bite through • Jaw stability emerges for holding food • A phasic bite may be reverted to during biting of harder foods	• Vertical chewing with variations in amount and speed • Diagonal rotary chewing emerges during tongue movement to the sides of the mouth • Phasic bite and release pattern noted occasionally when chewing

Table 7–1. continued

	Feeding Behavior	*Sucking Skills*	*Swallowing Skills*	*Biting Skills*	*Chewing Skills*
	• More and more finger foods are offered and self-fed easily • Diminished spitting-up unless eating too fast or too much • Drooling no longer noted during newly learned gross motor tasks. Noted only *during* teething. Central incisors are in	moving downward and forward. Lower lip moves downward, forward, and inward around 10 mo	drinking: 3 sucks-swallow-breathe pattern with some continued difficulty coordinating swallowing • Incisors used to clear lower lip		with the front teeth • Lateral transfer of food emerges • Lips actively move with the jaw during chewing with downward, forward, and inward movements noted
12 mo	• Liquids and soft-mashed or coarsely chopped table foods and soft meats • 32 oz milk/formula daily	• Holds and drinks skillfully from cup with a sucking pattern • Tongue may protrude under cup	• Intermittent tongue tip elevation during swallowing of semi-solids, solids, and liquids by cup • No loss of food	• Soft cookie easily bitten with a controlled sustained bite • Phasic bite or sucking may be	• Lips and teeth are active during chewing. The upper teeth/gums assist in food removal off lower lip

• Introduce whole milk and eggs • Self-fed finger foods • First words emerge	with some liquid loss. Jaw excursions are up-down and back-to-front	during eating • Lips may be open during cup drinking but are closed during swallowing of food • Front-to-back tongue movements noted only during cup drinking • Simple tongue protrusion continues during swallowing of food • No pause in suck swallow sequences during cup drinking with 3 suck-swallows when thirsty • Choking rarely occurs • Good coordination of suck-swallow pattern with rare occurrences of coughing and choking (15 mo)	reverted to during biting of harder foods

Table 7–1. continued

	Feeding Behavior	*Sucking Skills*	*Swallowing Skills*	*Biting Skills*	*Chewing Skills*
18 mo	• Sits unsupported at table • Liquids, coarsely chopped table foods including most meats and raw vegetables • No longer drools while walking, running, or early fine motor tasks such as dressing or self-feeding Drools if teething • Teeth are in	• Jaw stabilizes under cup with upper lip showing a better seal on edge. No more tongue protrusion under cup	• Sequences 3 suck-swallows drinking 1 oz or more with no pause • More consistent tongue tip elevation during swallowing of semisolid foods • Lip closure	• Teeth are in • Controlled sustained bite used on hard cookie • Extraneous motor movements and slight head extension may accompany biting	• Coordinated and smooth diagonal rotary chewing • Chews with lips closed but may loose food or saliva
24 mo	• No longer drools while drawing, manipulating or speaking • Spoon feeding refined	• Internal jaw stability emerging with cup drinking • Up-down sucking with cup held between the lips		• No more overflow of extraneous movement in arms or legs • Chews with mouth closed	• Food is transferred directly from one side of the mouth to the other • Front-to-back tongue movements only with more difficult food

					• Lip control mastered with no loss of food or saliva
24+ mo	• No longer drools as the child learns to consciously swallow saliva • Good use of utensils	• Active internal jaw stability • Slight up-down jaw motions and holding edge of cup with teeth may be noted • Sweeping motion of tongue to clean food from lips by 3 yr • Independent tongue elevation and depression with skillful tongue tip action • Straw drinking refined by 3 yr	• Consistent tongue tip elevation • Easy lip closure as needed with no loss of liquid, food, or saliva • Skillfully swallows food with a combination of textures • Adult-like swallow	• Controlled, sustained bite with grading of jaw for a variety of thicknesses	• Continues to be a combination of nonstereotypic and diagonal rotary movements • Circular rotary chewing occurs during food transfer from side to side within the mouth • Can transfer food in a variety of ranges within the mouth • Continued refinement of sequencing and precision movements

Reproduced with permission from Puelzl M, Sinden N, Hendricks K. Developmental aspects of weaning. Int Semin Paediatr Gastroenterol Nutr 1993;2:10–1.

Table 7–2. Infant Feeding Guide*

Foods	*0–4 mo*	*4–6 mo*	*6–8 mo*	*8–10 mo*	*10–12 mo*
Breast milk OR	Frequent feedings 8 or more per day	Frequent feedings 5 or more per day	On demand 5 or more feedings	On demand	On demand
Iron-fortified formula	6–32 ounces	24–40 ounces	24–32 ounces	16–32 ounces	16–24 ounces
Cereals and bread	None	†Boxed rice, oatmeal or barley (spoon-fed) Mix 2–3 tsp with formula, water, or breastmilk	Most varieties of boxed infant cereals 1–4 tbsp, twice a day	Infant cereals or other hot cereals 8–12 tbsp/day Toast, bagel, or crackers	Hot or cold unsweetened cereals 1/4 cup/day Bread 1/4 slice Rice noodles or spaghetti 1/4 c. 4 servings/day
Fruit	None	None	Fresh, cooked, mashed, or strained fruits One 4 ounce jar or 1/2 cup/day	Peeled, soft, mashed fruit or wedges 1/3–1/2 cup/day Fruit juice (optional) no more than 8 ounces per day	All fresh fruits peeled and seeded 1/2 cup/day

Vegetables	None	None	Strained or mashed vegetables Dark yellow or orange (avoid corn) Dark green	Cooked and mashed, fresh or frozen vegetables 1/3–1/2 cup/day	Cooked vegetable pieces 1/2 cup/day
Protein	None	None	None	Lean meat, chicken, and fish (strained, chopped, or small, tender pieces) 3–4 tbsp/day Egg yolk Yogurt Mild cheese Cooked dried beans	Small tender pieces of meat, fish, or chicken 4–5 tbsp/day Whole egg Cheese Yogurt Cooked dried beans Peanut butter

*Adapted from Infant feeding guidelines, WIC Form #47.

†American Academy of Pediatrics recommends that exclusive breastfeeding is optimal for approximately the first 6 months of life.

8

FEEDING GUIDELINES FOR CHILDREN AND ADOLESCENTS

Isabel M. Vazquez, MS, RD, and Jan P. Hangen, MS, RD

It is important for children to eat a variety of foods that can provide them with the proper intake of carbohydrates, protein, fat, minerals, and vitamins. Each child grows at a unique rate and therefore has unique nutritional needs. Children's behaviors and food preferences are also unique. The nutritional needs of children should be balanced with their food likes and dislikes. The following are general guidelines to follow when feeding infants, children, and adolescents; they should not be viewed as precise. It is important to recognize that children eat differently every day and that they should be allowed to follow their own internal hunger and satiety cues.

Ellyn Satter describes "normal eating" in the following manner: "Normal eating is being able to eat when you are hungry and continue eating until you are satisfied. It is being able to choose food you like and eat it and truly get enough of it—not just stop eating because you think you should. Normal eating is being able to use some moderate constraint on your food selection to get the right food, but not being so restrictive that you miss out on pleasurable foods. Normal eating is giving yourself permission to eat sometimes because you are happy, sad, or bored or just because it feels good. Normal eating is three meals a day,

or it can be choosing to munch along. It is leaving some cookies on the plate because you know you can have some again tomorrow, or it is eating more now because they taste so wonderful when they are fresh. Normal eating is overeating at times: feeling stuffed and uncomfortable. It is also undereating at times and wishing you had more. Normal eating is trusting your body to make up for your mistakes in eating. Normal eating takes up some of your time and attention, but keeps its place as one important area of your life. In short, normal eating is flexible. It varies in response to your emotions, your schedule, your hunger and your proximity to food."[1] This principle of "normal eating" is what we should follow in our daily life. Remember to always prepare a variety of nutritious foods in ways the child will eat, leaving the child to decide how much of which food to eat.

Healthy Eating for Children

Children require adequate amounts of a balanced variety of foods. These include fruits, vegetables, whole and enriched grains and cereals, milk and other dairy products, and meat, fish, poultry, and other protein products.[1]

Fruits and vegetables are the primary sources of vitamins A and C and contain other nutrients such as B vitamins, trace minerals, and fiber.[1] Breads and cereals are excellent sources of B vitamins and, if enriched, iron. Whole-grain breads and cereals are also good sources of fiber, vitamin E, and trace elements such as magnesium. It is recommended that half the breads and cereals in the diet be whole grain.[1] Dairy products, especially milk, provide protein and serve as primary dietary sources of calcium and vitamin D. Milk provides the primary source of protein in early childhood. Protein in general provides dietary satiety. When the child makes the transition to

table foods toward the end of the first year, more protein will be obtained from meat, fish, poultry, or other protein foods in the diet. These foods then begin to supplement milk as a source of protein and should thus be offered on a regular basis.[1,2] Foods from the meat group are good sources of protein, iron, and trace elements such as zinc.

When planning meals, a variety of foods should be offered at each meal. The meal should provide protein (in the form of meat, fish, poultry, eggs, or legumes), bread and/or cereal, fruit and/or vegetables, and milk (Table 8–1).

Children become hungry between meals, making snacks an important part of their daily intake. Snacks should be planned, so the child does not continually graze. They should be spaced to ensure the child is hungry at meals, with the interval between meals and snacks tailored to the child's hunger and satiety cues.[1]

Meals should provide adequate amounts of protein, carbohydrates, and fat (Tables 8–2 to 8–6).[3,4] Fat in moderate amounts is an essential component of any diet. It is recommended that no less than 30% of the calories in a child's diet come from fat, unless the child is on a special high or low fat diet.[3] If the child is eating a lot of fast and/or junk food, it is likely that their dietary fat intake is too high.[1]

Carbohydrates provide a feeling of fullness and substance in a meal. Children generally enjoy starchy foods and thus do not have to be persuaded to eat them. The diet that is too high in carbohydrates may be too low in fat, however, and is likely to be unsatisfying.[1]

Nutritious Snacks

Filling and satisfying snacks should include all three macronutrients: carbohydrates, protein, and fat. Carbohydrates provide bulk and some energy value, while proteins and fat provide satiety value. Snacks are important for children as they tide them over from one meal to the next, help-

Table 8–1. Nutritious Choices within Food Groups

Protein Foods
- Natural cheeses
- Milk
- Cooked turkey, chicken, beef, ham
- Plain yogurt
- Peanut butter
- Cottage cheese
- Unsalted nuts and seeds
- Tuna
- Hard-cooked egg

Breads and Cereals
- Whole-grain breads
- Whole-grain cereals
- Whole-grain, low-fat crackers
- Tortillas (corn or flour)
- English muffin
- Rice cakes or crackers
- Pita bread
- Popcorn (plain)
- Pretzels
- Bagels

Fruits (any, but children's preferences listed)
- Apples
- Bananas
- Pears
- Berries
- Oranges or other citrus
- Melon
- Grapes
- Unsweetened canned fruits (eg, applesauce, pears, fruit cocktail, peaches, etc.)

Vegetables (any, but children's preferences listed)
- Carrots
- Celery
- Green, red, or yellow peppers
- Radishes
- Cucumbers
- Cauliflower
- Broccoli
- Tomatoes

Table 8–1. continued

Snack Combinations
Apple wedges with peanut butter
Egg salad sandwich
Deviled eggs
Raw vegetables with yogurt or cottage cheese dip
Plain yogurt with unsweetened applesauce and cinnamon
Tortilla with melted cheese
Pita bread with tuna salad
Popcorn with parmesan cheese
Frozen banana rolled in plain yogurt and chopped nuts
Rice cakes with peanut butter or cheese

ing to stabilize energy levels and moods.[1] Table 8–1 lists ideas for snack combinations. It is suggested that protein foods be combined with a choice from the breads and cereals, fruits, or vegetables also listed in Table 8–1. This will enhance the macro- and micronutrient balance of the snacks.

Table 8–2. Feeding Recommendations for Infants

Age	*Appropriate Foods*	*Amount/Serving Size*	*Comments*
0–4 months	Breastmilk or iron-fortified formula	21–29 oz formula (4–6 feedings/d) or 6–8 nursings	Breastfeeding is ideal for all infants Breastmilk or infant formula should be the sole source of nutrition until age 4–6 mo
4–6 months	Breastmilk or iron-fortified formula	29–32 oz formula (4–6 feedings/d) or 4–5 nursings	At age 4–6 mo babies can move their tongues back and forth and swallow foods given to them on a spoon Never feed solid foods from a bottle with enlarged nipple holes
	Iron-fortified infant cereal	1–2 tsp 1–2×/d	Mix infant cereal with breastmilk or formula
	Strained foods and vegetables	1–2 tsp 1–2×/d	Do not add sugar, honey, or syrup to infant cereal Honey and corn syrup can cause food poisoning (botulism) in children < 1 yr Give one new food at a time Begin with single ingredient foods rather than food mixtures Do not add butter or salt to baby's food
6–9 months	Breastmilk or iron-fortified formula	30–32 oz formula (3–5 feedings/d) 3–5 nursings/d	Introduce your child to a cup when you introduce solid foods Offer juice in a cup
	Iron-fortified infant cereal	2–3 tbsp 2×/d	

Table 8–2. continued

Age	*Appropriate Foods*	*Amount/Serving Size*	*Comments*
	Fruit juice (high in vitamin C)	3–4 oz/d	
	Mashed vegetables and fruits	2–3 tbsp 2×/d	
	Strained meats	1–2 tbsp 2×/d	
	Plain toast or teething biscuit	1/2 to 1 serving toast	
9–12 months	Breastmilk or iron-fortified formula	24–30 oz formula (2–4 feedings/d) or 3–4 nursings	Introduce whole cow's milk in a cup at 12 mo During this period you may begin finger foods or cooked cut-up table meat, poultry, fish, pieces of cheese 1/2 to 1 oz/d
	Iron-fortified infant cereal	2–4 tbsp 2×/d	Appropriate finger foods are: pieces of banana, graham crackers, strips of cheese, or bagels
	Fruit juice (high in vitamin C)	3–4 oz/d	Encourage your child to sit down to eat or drink
	Chopped vegetables	3–4 tbsp 2×/d	Whole milk, cottage cheese, and plain yogurt may be also introduced
	Chopped fruits	3–4 tbsp 2×/d	
	Strained meats	2–3 tbsp 2×/d	
	Bread and bread products	1/2 to 1 serving	

Table 8–3. Feeding Recommendations for Toddlers

Age	*Appropriate Foods*	*Amount/Serving Size*	*Comments*
1–2 years	Whole milk Yogurt Cheese Custard or pudding Cottage cheese Ice cream Meat Fish Poultry Cooked or dried beans Bread Cereals Starches	1/4 to 1/2 cup serving size (total = 2 cups/d) 1 oz = 1 egg, 1 slice of cheese, 1/2 cup cooked dried beans, peas, or lentils; 2 tbsp peanut butter, 1 slice bologna, 1 hot dog, or 1/8 cheese pizza (total = 2–3 oz/d) 1 serving = 1 slice bread, 1/2 bagel, 1/2 English muffin, 5–7 average crackers, 1/2 cup hot cereal, 3/4 cup cold cereal, 1/2 cup rice, or noodles, or	Offer a variety of foods Provide nutritious choices Establish a regular schedule for meals and snacks Serve small portions and allow the child to ask for seconds Provide a comfortable chair, and a seating arrangement with food support for the child Do not use food as a reward, punishment, or bribe Reinforce eating behavior with praise and positive, pleasant interaction Establish a quiet time before meals to help set the atmosphere for attention to eating Raisins and other small dried fruits are not generally recommended for children under age 3 because of the risk of choking A new food may be rejected at first but this does not mean the toddler will always dislike it. Some new foods need to be introduced in small portions several times before a toddler will accept or even

Table 8–3. continued

Age	*Appropriate Foods*	*Amount/Serving Size*	*Comments*
		potatoes, or 1 average pancake or waffle (total = 4 servings)	taste them Parents should be a good role model
	Fruits	1 serving fruit = 1/2 piece or 1/2 cup	Children eat better if they are not being pressured
	Vegetables	1 serving vegetable = approx 2 tbsp (minimum of 3 servings/wk)	

Table 8–4. Feeding Recommendations for Children Aged 2 to 5 Years

Age	*Appropriate Foods*	*Amount/Serving Size*	*Comments*
3–5 years	Whole milk Yogurt Cheese Custard or pudding Cottage cheese Ice cream	1/2 to 3/4 cup serving size (total = 2 cups/d)	Milk is an important food for children aged 2–5 yr mainly because of its calcium content. Drinking too much milk or any liquid may reduce appetite for other important foods, especially those rich in iron. After 2 yr, milk and milk products can be changed to a low fat alternative
	Meat Fish Poultry	1 oz = 1 egg, 1 slice of cheese, 1/2 cup cooked dried beans, peas, or lentils, 2 tbsp peanut	For preschool children who have a varied diet, vitamin supplements are not necessary

Cooked or dried beans	butter, 1 slice bologna, 1 hot dog, or 1/8 cheese pizza (total = 2–3 oz/d) 1 serving = 2–4 oz of cooked meat 3 oz = 1/4 chicken (1/2 breast, or leg and thigh), 1 medium pork chop, 3/4 inch thick, 1 lean ground beef patty, or 1 piece of meat or fish the size and thickness of the palm of the hand	Children need to snack between meals Offer nutritional snacks Offer snacks about 2 hours before a meal Use salt sparingly, if at all, in cooking and at the table Offer fresh fruits or fruits processed without syrup or with light rather than heavy syrup Water is important. Offer water several times a day Keep serving foods that are not accepted at first. Prepare them in different ways and try again If a child refuses to eat vegetables, offer fruits that contains many of the same vitamins and minerals. Prepare vegetables in different ways and try again
Bread Cereals Starches	1 serving = 1 slice bread, 1/2 bagel, 1/2 English muffin, 5–7 average crackers, 1/2 cup hot cereal, 3/4 cup cold cereal, 1/2 cup rice, noodles, or potatoes, or 1 average pancake or waffle (total = 4 servings)	
Fruits	1 serving fruit = 1/2 piece or 1/2 cup	
Vegetables	1 serving vegetable = approx 2 tbsp	

Table 8–5. Feeding Recommendations for Children Aged 6 to 12 Years

Age	*Appropriate Foods*	*Amount/Serving size*	*Comments*
6–12 years	Whole milk Yogurt Cheese Custard or pudding Cottage cheese Ice cream	1 cup serving size (total = 3 cups/d) 1 oz cheese, 1 cup yogurt	Children begin to be more independent and make more food choices Involve children in food planning and preparation Take children grocery shopping Encourage children to try a wide variety of foods, including foods of other cultures
	Meat Fish Poultry Cooked or dried beans	1 oz = 1egg, 1 slice of cheese, 1/2 cup cooked dried beans, peas, or lentils, 2 tbsp peanut butter, 1 slice bologna, 1 hot dog, or 1/8 cheese pizza 1 serving = 2–4 oz cooked meat 3 oz = 1/4 chicken (1/2 breast, or leg and thigh); 1 medium pork chop, 3/4 inch thick, 1 lean ground beef patty, or 1 piece of meat or fish the size and thickness of the palm of the hand (total = 2 servings/d)	

Bread Cereals Starches	1 serving = 1 slice bread, 1/2 bagel, 1/2 English muffin, 5–7 average crackers, 1/2 cup hot cereal, 3/4 cup cold cereal, 1/2 cup rice, noodles, or potatoes, or 1 average pancake or waffle (total = 6 or more servings)
Fruits	1 serving fruit = 1/2 piece or 1/2 cup (2 servings/d)
Vegetables	1 serving vegetable = 1/4 to 1/2 cup cooked or chopped raw vegetables

Table 8–6. Feeding Recommendations for Adolescents

Age	*Appropriate Foods*	*Amount/Serving Size*	*Comments*
12–18 years	Milk, yogurt, cheese	3–4 servings/d 1 serving = 1 cup milk or yogurt, or 1 oz cheese	Adolescents require about 1.3 mg Ca/d Recommendations to follow when working with adolescents:
	Meat, poultry, fish, beans/peas, egg, nuts	2–3 servings/d 1 serving = 3 oz meat or poultry	• Be flexible, nonjudgmental, uncritical in your approach • Recommended small increments of change, one step at a time
	Bread, cereal, rice, pasta	6–11 servings 1 serving = 3/4 cup cereal or 1/2 cup rice or pasta	• For young adolescents, talk in simple and concrete terms
	Vegetables	3–5 servings/d 1 serving = 1 cup raw, 1/2 cup cooked, or 3/4 cup vegetable juice	• Discuss food choices, ways to prepare foods, and portion sizes • Discuss food concept rather than nutrients • Talk about positive aspects of their actual diet, eg, "It is great that you are eating breakfast"
	Fruits	2–4 servings/d 1 serving = 1 medium fruit 1/2 cup fruit	• Talk about moderation instead of diet or food restriction
	Fats, oils, sweets	Use sparingly	• Explore and discuss problems such as lack of time financial resources, or a nonsupportive family • Take into consideration cultural and socioeconomic influences[4]

Reproduced with permission from Alton J, Story M. Guidelines for adolescent nutrition. Chicago, IL: Department of Health and Human Services, 1993.

Adolescent Nutrition

Adolescence is a period of transition from childhood to adulthood. It is also a period of biologic, physical, emotional, and cognitive change. Teenagers want to be more independent, have an active lifestyle, and find their identity. They are frequently sensitive to criticism. These factors can put the adolescent at nutritional risk. In addition to growth and greater demand for calories and nutrients, their change in lifestyle affects food choices. Adolescents may skip meals, eat away from home, increase snacking, eat more convenience fast foods, and generally be more responsible for their food intake.[4,5,6]

The typical adolescent may display the following nutritional habits:[5]

- Derives over 30% of calories from fat
- Skips breakfast (20%)
- Skips lunch (22%)
- Snacks heavily from 3:00 pm to bedtime (50%)

Adolescent nutritional needs are influenced by age, gender, state of puberty development, and activity level. The Recommended Dietary Allowances (RDA) for adolescents are categorized by chronologic development rather than degree of maturation. Practitioners should exercise clinical judgment in applying these.[6] Wait and colleagues support the use of calories per unit height as the best index for determining caloric needs.[7] Table 8–7 shows kcal/cm and protein/cm requirements based on the RDA. Protein and energy needs correlate more closely with growth pattern than with chronologic age. Spear recommends the use of the RDA for protein in relation to height as the most useful method of estimating needs.[6] Servings by food group are shown in Table 8–6.

Table 8–7. Estimated Daily Calorie and Protein Needs for Adolescents

	Age	*Calories (kcal/cm)*	*Protein (g/cm)*
Males	11–14	15.9	0.29
	15–18	17.1	0.34
	19–24	16.4	0.33
Females	11–14	14.0	0.29
	15–18	13.5	0.27
	19–24	13.4	0.28

Adapted from Recommended dietary allowances, 10th edition. Washington, DC: Food and Nutrition Board, National Academy of Sciences, National Research Council, 1989.

References

1. Satter E. How to get your child to eat...but not too much. Palo Alto (CA): Bull Publishing Co.; 1983. p. 69–70.
2. Satter E. Child of mine: feeding with love and good sense. Palo Alto (CA): Bull Publishing Co.; 1983.
3. Position of the American Dietetic Association: dietary guidance for healthy children aged 2 to 11 years. J Am Diet Assoc 1999;99:93–101.
4. Alton I, Story M. Guidelines for adolescent nutrition. Chicago: Dept. of Health and Human Services (US); 1993.
5. Beach R. Priority health behaviors in adolescents health promotion in the clinical setting. Adolesc Health Update 1991; 3(2).
6. Spear B. Adolescent growth and development. In: Adolescent nutrition assessment and management. 1996. p. 3–24.
7. Wait B, Blair R, Roberts LJ. Energy intake of well-nourished children and adolescents. Am J Clin Nutr 1969;22:1383.

9

THE US DEPARTMENT OF AGRICULTURE'S FOOD GUIDE PYRAMID

Laurie A. Higgins, RD

The US Department of Agriculture (USDA) developed the Food Guide Pyramid (Figure 9–1) and released it in 1992 with the view that most Americans have diets that are too high in fat. In addition to the pyramid, the Dietary Guidelines for Americans include the following:

- Eat a variety of foods.
- Balance the food you eat with physical activity.
- Maintain or improve your weight.
- Choose a diet low in fat, saturated fat, and cholesterol.
- Choose a diet moderate in sugars.
- Choose a diet moderate in salt and sodium.
- If you drink alcohol beverages, do so in moderation.

Food pyramids and food labels are tools designed by the USDA to help Americans incorporate the Dietary Guidelines into their daily lives. Each of the food groups provide some of the nutrients we need. No one food group is more important than another, but we are encouraged to primarily choose from the bottom half of the pyramid. The pyramid enables the clinician to make a quick assessment of a client's intake to determine if it is nutritionally adequate. Variety and portion sizes should be taken into consideration when using this as a tool. The pyramid suggests we choose the foods by a range of servings, with the

smaller number providing 1,600 calories a day and the larger providing 2,800 calories a day.

The Food Guide Pyramid for Young Children (Figure 9–2) was released in March 1999, adapted from the original Food Guide Pyramid, and designed for children between the ages of 2 to 6 years. The new pyramid presents the foods in realistic style, showing single servings of foods commonly eaten by young children, and emphasizing variety. The pyramid also demonstrates graphically the importance of physical activity, symbolizing how healthy eating and activity work together for good health. In addition, a 16-page booklet, *Tips for Using the Food Pyramid for Young Children 2 to 6 Years Old*, was designed to illustrate the pyramid and pro-

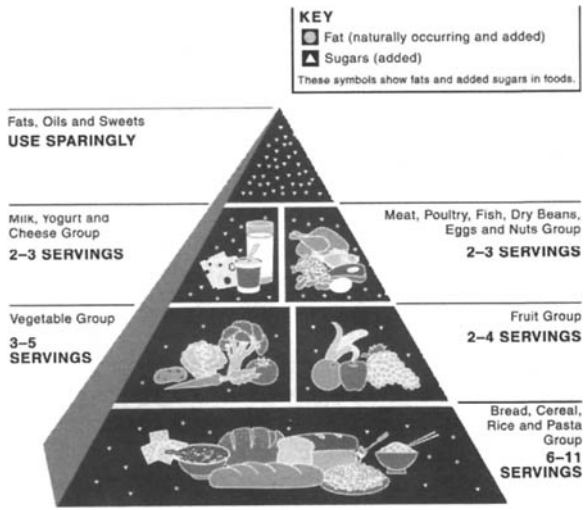


Figure 9–1. The Food Guide Pyramid (from US Department of Agriculture, http://www.na/usda.gov/fnic/Fpyr/pymid.gif)

vide guidelines for parents and educators on how to use the pyramid to educate the young child about food and eating healthy. Shown below is a table from the booklet (Table 9–1) that shows how to use the pyramid to plan a healthy day of meals and snacks. The booklet is available to the public and can be obtained by downloading it from the USDA's home page at URL: http://www.usda.gov/cnpp/index.htm/ or by calling the Government Printing Office at (202) 512-1800 and asking for stock number 001-00004665-9.

Figure 9–2. Tips for using the Food Guide Pyramid for young children 2 to 6 years old. Source: US Department of Agriculture and US Department of Health and Human Services.

Table 9–1. Sample Day Meals and Snacks for 4- to 6-Year-Old Children*

	Grain	*Vegetable*	*Fruit*	*Milk*	*Meat*
Breakfast					
100% fruit juice, 3/4 cup			1		
Toast, 1 slice	1				
Fortified cereal, 1 oz	1				
Milk, 1/2 cup				1/2	
Midmorning Snack					
Graham crackers, 2 squares	1				
Milk, 1/2 cup				1/2	
Lunch					
Meat, poultry, or fish, 2 oz					2 oz
Macaroni, 1/2 cup	1				
Vegetable, 1/2 cup		1			
Fruit, 1/2 cup			1		
Milk, 1/2 cup				1/2	
Midafternoon Snack					
Whole grain crackers, 5	1				
Peanut butter, 1 tbsp					1/2 oz
Cold water, 1/2 cup					
Dinner					
Meat, poultry, or fish, 2 1/2 oz					2 1/2 oz
Potato, 1 medium		1			
Broccoli, 1/2 cup		1			
Cornbread, 1 small piece	1				
Milk, 1/2 cup				1/2	
Total Food Group Servings	6	3	2	2	5 oz

Reprinted with permission from the US Department of Agriculture. Tips for using the food pyramid for young children 2 to 6 years old. Washington (DC): US Government Printing Office; 1999.

*2- to 3-year-old children should have the same variety but smaller portions, approximately 2/3.

10

VEGETARIAN DIETS

Heidi Schauster, MS, RD

Vegetarian diets are increasingly found among the pediatric and adolescent population as a growing number of Americans adopt this eating style. Plant-based diets have been shown to significantly lower rates of chronic diseases including heart disease, diabetes, and some cancers.[1] While most adherents are motivated by health concerns, many families and individuals choose to follow a vegetarian diet for philosophic, religious, or environmental reasons. Such diets center around plant-based foods; foods of animal origin, if consumed at all, play a minor role. Important distinctions between the different types of vegetarian diets practiced are shown in Table 10–1.

Table 10–1. Types of Foods Consumed within Different Vegetarian Diets

	Beef and Pork	*Fish and Poultry*	*Dairy*	*Eggs*	*Vegetables, Grains, Nuts, Seeds, Soyfoods*
Traditional diet	X	X	X	X	X
Semivegetarian		X	X	X	X
Lacto-ovovegetarian			X	X	X
Lactovegetarian			X		X
Ovovegetarian				X	X
Vegan*					X

*The macrobiotic vegetarian typically eats similarly to the vegan, although seafood may be consumed. Other foods eliminated from the macrobiotic diet include vegetables of the nightshade family, tropical fruits, and processed sweeteners.

Many health professionals become concerned when they discover that a child or adolescent consumes a vegetarian diet. Although misinformation exists about what constitutes a healthy vegetarian eating style, well-planned vegetarian and semivegetarian diets can completely satisfy all nutrient requirements for growth and development in infants, children, and adolescents. When assessing vegetarian diets, the clinician should ascertain whether the diet (1) meets the patient's caloric and other nutritional needs, (2) is well-balanced and varied, and (3) is excessive in any nutritional component that might pose a risk to the patient.[2]

Protein

One of the major concerns cited by health professionals and parents in relation to vegetarian diets is that the child will not receive adequate protein for growth and total health, as animal products excluded from the diet are significant sources of protein. Protein is abundant, however, in many plant and dairy foods, as shown in Table 10–2. Contrary to popular belief, if one eats a variety of plant foods and consumes adequate calories to meet energy needs, it is almost impossible to receive inadequate protein. At one time, it was believed that vegetarians had to carefully combine certain plant-based foods to receive the

Table 10–2. Vegetarian Sources of Protein

Eggs*	Dairy products*
Grains and grain products	Legumes (dried beans and peas)
Seeds	Nuts
Nut butters	Bean soups, stews, and chili
Hummus	Vegetarian burgers
Tofu	Vegetables
Soy milk and soy products	

*May not be consumed by some types of vegetarians.

appropriate amino acids comprising a complete protein. Although it is correct that some plant proteins are limited in one or more essential amino acids, it is now known that eating a reasonable variety of grains, vegetables, and legumes over the course of a day will provide appropriate amino acids and total protein.[3]

Minerals: Calcium and Iron

Calcium is the major mineral of concern of clinicians caring for vegans or ovovegetarians who do not include dairy products in their diets. Although predominant in dairy foods, calcium is found in many plant-based foods, as illustrated in Table 10–3. There is evidence that calcium is better absorbed and retained from vegetarian diets, which partly relates to the more moderate protein content of these diets. Less calcium is lost in the urine of vegetarians than in those consuming the traditional high-protein Western diet.[3]

Iron is most plentiful and biologically available in red meats, which are excluded from vegetarian or semivegetarian diets. It is possible, however, to glean adequate iron from such diets. Consuming the iron-rich foods listed in

Table 10–3. Nondairy Vegetarian Sources of Calcium

Green leafy vegetables (kale, broccoli, turnip greens, mustard greens, collard greens, bok choy)
Tofu (if processed with calcium)
Dried beans (garbanzo, kidney, navy)
Sesame seeds
Dried figs
Almonds
Blackstrap molasses
Tahini
Calcium-fortified orange juice or soy milk

Table 10–4 with foods rich in vitamin C, such as citrus fruits and juices, enhances absorption of this important mineral. Including a variety of foods in the diet as well as adequate calories should help prevent iron-deficiency in both vegetarian and nonvegetarian children.

Vitamin B_{12}

Most plant-based foods are rich in vitamins. The only vitamin of major concern for vegetarians–vegans in particular–is vitamin B_{12}. Although some plant foods may be contaminated with this vitamin, there exists no reliable nonanimal source of B_{12}. All vegans should obtain a regular, reliable source of the vitamin, either from fortified foods (some cereals, meat alternatives, soy or vegetable milks, or nutritional yeast) or in a supplement. Note that many sea vegetables, often thought to be rich in B_{12}, have not been proven to be reliable sources of the vitamin, despite package claims. The nutritional requirement for B_{12} in children > 11 years of age is a minimum of 2 μg per day.[4]

If the nutritional adequacy of a patient's vegetarian diet and the potential need for supplementation are in ques-

Table 10–4. Vegetarian Sources of Iron

Dark-green leafy vegetables (spinach, collard greens, kale, Swiss chard)
Dried beans (garbanzo beans, lentils, pinto beans)
Raisins
Figs
Prune juice
Watermelon
Pumpkin and sesame seeds
Blackstrap molasses
Iron-fortified cereals, especially cream of wheat
Soy nuts

tion, a registered dietitian should be consulted. Table 10–5 demonstrates a sample 1800-calorie daily meal plan for a balanced, vegan diet.

Special Consideration: Infant and Toddler Feeding

Children 2 years of age and older, adolescents, and adults may be healthily maintained on a carefully planned vegetarian diet. Adolescents and pregnant women may need special attention, given the special nutritional needs of these populations. The significant period of growth and development between 4 to 6 months and 2 years of age in children requires that an infant consume adequate ratios of

Table 10–5. Sample Day: Balanced 1800-Calorie Meal Plan for a Vegan Diet

Breakfast	1 cup oatmeal with 2 tbsp raisins and 2 tbsp wheat germ 1 cup soy milk 2 slices whole wheat toast with jelly 6 oz orange juice
Lunch	1 cup lentil soup Mixed green salad with tomatoes and dressing Carrot and green pepper sticks with salsa 1 whole grain muffin Water with fresh lemon
Dinner	1/2 cup marinated bean salad (kidney, garbanzo, and green beans) 1 cup pasta with 1 tsp olive oil, garlic, and basil, with a sprinkling of nutritional yeast 1/2 cup stewed tomatoes and okra 1/2 cup steamed broccoli with lemon juice 1 slice Italian bread 1/2 cup fresh fruit salad
Snack	Flavored seltzer water Bagel with jam

Reproduced with permission from The American Dietetic Association. Being vegetarian. Minneapolis: Chronimed Publishing; 1996. p 73–4.

macro- and micronutrients. While a semi-, lacto-ovo-, or lactovegetarian diet may be appropriate for these infants, a strict vegan diet is not recommended during this important time of growth. Caregivers should suspend their practice of the vegan diet and make the child's growth and healthy maturation a priority during this time. The vegan diet may be resumed at 2 years of age, if desired by the caregiver, with little effect on the baby's ultimate food preferences.[5] When balanced and varied, a vegetarian diet can be an appropriate and healthy option for children and adolescents.

References

1. World Health Organization Study Group on Diet, Nutrition and Prevention of Noncommunicable Diseases. Diet, nutrition and the prevention of chronic diseases. Nutr Rev 1991; 49:291–301.
2. Jacobs C, Dwyer JT. Vegetarian children: appropriate and inappropriate diets. Am J Clin Nutr 1988;48:811–8.
3. The American Dietetic Association. Being vegetarian. Minneapolis (MN): Chronimed Publishing; 1996.
4. Vitamins. In: Messina M, Messina V, editors. The dietitian's guide to vegetarian diets: issues and applications. Gaithersburg (MD): Aspen Publishing; 1996.
5. Ivens BJ, Weil WB. Teddy bears and bean sprouts: the infant and vegetarian nutrition. Fremont (MI): Gerber Products Company; 1997

11

SPORTS NUTRITION

Susan E. Frates, MS, RD

The number of American children and adolescents participating in organized sports is on the rise, with roughly half (49.5%) participating in organized sport teams nationwide.[1] Young athletes are bombarded with mixed messages from the media, coaches, and even professional athletes. These messages often focus on fads and false claims concerning nutrition, and unrealistic body images.

Sports nutrition begins with a base of sound nutrition. Eating well includes a balanced diet, which provides appropriate amounts of vitamins, minerals, protein, fat, and carbohydrate. Following the Recommended Dietary Allowance (RDA) for age while utilizing the US Department of Agriculture's Food Guide Pyramid can help achieve this goal.[2] A well-proportioned diet should contain 55 to 75 percent calories from carbohydrate, 25 to 30 percent calories from fat, and 15 to 20 percent calories from protein.[2]

Athletes do have special nutrition needs. Primarily, these include increased energy needs, provided mostly from carbohydrate-rich food sources. The physical activity demands of sports create additional energy needs. Table 11–1 identifies calorie demands with particular sports.[3] A carbohydrate-rich diet serves to optimally fuel muscles for endurance and strength training. Athletes can usually meet their increased energy needs by following a nutrition plan of three meals and well-timed snacks. Table 11–2 identifies carbohydrate-rich food selections. It is recommended that a carbohydrate-based pre-practice or pre-event snack

Table 11–1. Energy Expenditure Caloric Equivalent of Child's Activities*

	Body Weight (kg)									
Activity	*20*	*25*	*30*	*35*	*40*	*45*	*50*	*55*	*60*	*65*
Basketball	34	43	51	60	68	77	85	94	102	110
Calisthenics	13	17	20	23	26	30	33	36	40	43
Cycling										
10 km/hr	15	17	20	23	26	29	33	36	39	42
15 km/hr	22	27	32	36	41	45	50	55	60	65
Figure skating	40	50	60	70	80	90	100	110	120	130
Ice hockey (on-ice time)	52	65	78	91	104	117	130	143	156	168
Running										
8 km/hr	37	45	52	60	66	72	78	84	90	95
10 km/hr	48	55	64	73	79	85	92	100	107	113
Soccer (game)	36	45	54	63	72	81	90	99	108	117
Swimming (30 m/min)										
Breast	19	24	29	34	38	43	48	53	58	62
Front crawl	25	31	37	43	49	56	62	68	74	80
Back	17	21	25	30	34	38	42	47	51	55
Tennis	22	28	33	39	44	50	55	61	66	72

*In kilocalories per 10 minutes of activity.

Adapted from Bar-Or O. Pediatric sports medicine for the practitioner. 1983.

be eaten approximately 2 to 3 hours before exercise activity.[4] Carbohydrate loading may be obtained by following a carbohydrate-rich diet during training and resting the

Table 11–2. Carbohydrate-Rich Foods

	Serving Size	*CHO (g)*	*Kcal*
Fruits			
Apple	1 medium	20	80
Orange	1 medium	15	65
Banana	1 medium	25	105
Raisins	1/3 cup	40	150
Vegetables			
Corn (canned)	1/2 cup	15	70
Peas	1/2 cup	10	60
Carrot	1 medium	10	40
Grains			
Bagel	1 small	31	165
English muffin	1	25	130
Pita	1 small	21	105
Graham crackers	2 squares	10	70
Cereals			
Raisin bran	3/4 cup	30	120
Granola (low fat)	1/2 cup	45	210
Oatmeal (instant)	1 packet	30	165
Entree items			
Baked potato	1 large	50	220
Pasta	1 cup	40	200
Spaghettios	1 cup	36	200
Rice (cooked)	1 cup	45	200
Desserts			
Frozen yogurt	1 cup	44	240
Oreo cookie	1	8	50
Fig Newton	1	11	60
Pop Tart	1	30	195

CHO = carbohydrate.

Adapted from Pennington's Bowes & Church's food values of portions commonly used. 17th ed, Philadelphia (PA): Lippincott Company; 1998.

body and muscles prior to the sports event.[5] For more information on carbohydrate loading, a consultation with a registered dietitian is suggested.

Protein needs of child and adolescent athletes are not increased due to sport participation. Following RDA age-based guidelines for protein is recommended. An increased supply of protein is not indicated for muscle building, endurance, or other possible benefits.[3] Some athletes believe intense training tears and breaks down muscle tissue and thus requires additional dietary protein sources.[2] This is a belief unfounded in scientific research.[6] Excessive protein intake may lead to impairment of kidney function or other serious medical complications. Additional energy intake, provided to maintain the energy needs of exercise, will sufficiently meet most athletes' protein needs.

Fat intake also remains essentially unchanged. Appropriate intake of dietary fat is helpful in providing a sense of satiety with meals and snacks.[7] Combining some dietary fat with a carbohydrate-based pre-event snack or meal is suggested.

Fluid needs of athletes do increase. Adequate pre-event hydration may help alleviate symptoms of dehydration, including cramping, dizziness, and (in severe cases) heat stroke. Table 11–3 identifies characteristics and treatment of dehydration. Water is generally recommended as the optimal exercise fluid.[2] Water functions to regulate body temperature, transport waste products and nutrients, and assist with many biochemical reactions for energy production.[2]

During sports activities associated with excessive fluid loss in sweat, sports drinks may be useful. Sports drinks such as Gatorade provide additional electrolytes, which may be lost during intense exercise. During most exercise, small amounts of electrolytes (mostly sodium and chlorine) are lost in sweat. Sweat is relatively dilute, with an

Table 11–3. Symptoms and Treatment of Heat Disorders

Disorder	*Symptoms*	*Treatment*
Heat cramps	Thirst; nausea; chills; clammy skin; throbbing heart; muscle pain; spasms	Have athete drink 4–8 oz of cold water every 10–15 min Move athlete to shade and remove any excess clothing
Heat exhaustion	Reduced sweating; dizziness; headache; shortness of breath; weak, rapid pulse; lack of saliva; extreme fatigue	Stop exercise, move athlete to a cool place Take off athlete's wet clothes, place ice bag on head Have athlete drink 16 oz of water for every pound of weight lost
Heat stroke	Lack of sweat, urine; dry, hot skin; swollen tongue; hallucinations; rapid pulse; unsteady gait; fainting; low blood pressure	Call for emergency medical treatment Place ice bags on back of head Remove wet clothing If athlete is conscious, help him or her take a cold shower If in shock, elevate feet

Reproduced with permission from Berning J, Steen S. Nutrition for sport and exercise. 2nd ed. Gaithersburg (MD): Aspen Publishers, Inc.; 1998. p. 237.

average sodium concentration of 30 to 50 mEq. The body typically re-adjusts electrolyte balance on its own, with equal fluid replacement for fluid sweat losses. Beverages are the best vehicle to replenish lost fluids and electrolytes. Salt tablets are not recommended since they contribute to further dehydration by causing extra water to enter the stomach, pulling water away from other tissue.

Table 11–4 illustrates dextrose and electrolyte components of common fluids. Fluid replacement should begin before an event (drinking 4 to 8 oz 1 to 2 hours before and 4 to 6 oz 10 to 15 minutes before), with continued replacement of 4 to 8 oz every 15 to 20 minutes. It is important to continue to drink fluids after sporting events (16 oz for every pound of weight lost).[2,8]

Nutritional supplements, such as sports energy bars, amino acid preparations, vitamin-mineral combinations, and ergogenic aids (substances that claim to produce

Table 11–4. Composition of Common Fluids*

Fluid	*CHO (g)*	*PRO (g)*	*Fat (g)*	*Kcal*	*Na (mg)*	*K (mg)*	*mOsm/kg H_2O (osmolality approximation)*
Gatorade	14	0	0	56	110	25	280
Pedialyte	6	0	0	24	244	179	270
Cola	25	0	0	96	9	0	650
Orange juice	27	1.7	0.1	112	2	474	1600
Apple juice	29	0.2	0.3	116	7	296	1300
Skim milk	12	8.4	0.4	86	126	406	275
Whole milk	11	8.0	8.2	150	120	370	285

CHO = carbohydrate; PRO = protein.

* All values per 8 oz (236 mL).

Reproduced with permission from Berning J, Steen S. Nutrition for sport and exercise. 2nd ed. Gaithersburg (MD): Aspen Publishers, Inc. 1998. p. 150.

enhanced athletic performance) should be viewed with caution. In 1994, the Dietary Supplement Health and Education Act (DSHEA) created a classification of "dietary supplements," which includes vitamins, minerals, amino acids, herbs, and other botanical preparations that do not fall under current Food and Drug Administration (FDA) approval.[3] This legislation has allowed manufacturers to publish limited health and nutrition claims on these products. Products like L-carnitine, ginseng, creatine, and chromium picolinate are commonly found with specific claims of health enhancement such as improved muscle definition, increased energy, and subcutaneous fat loss. Few if any scientific studies have been performed to evaluate these claims. Please see Table 11–5 for the nutritional composition of popular sports bars.

In an age in which adult professional athletes are revealing their own use of controversial supplements, additional pressure is placed on younger athletes. Parents, coaches, and all health educators should consider the use of nutritional supplements inadvisable. Permanent damage to organ systems or stunting of height in children and adolescents may occur. Studies examining the conclusive long-term effects of such supplements on children and adolescents have not been documented to date.[9] Promotion of food as the optimum health fuel for endurance sports for athletes is recommended.

Special nutrition concerns of female athletes include amenorrhea, impaired bone density, and anemia. The prevalence of adolescent females with amenorrhea has been reported to range from 79 percent of ballet dancers to 12 percent of cyclers and swimmers.[10] Recent research has also linked a higher prevalence of eating disorders with female athletes. The "female athlete triad" examines the connection between overexercise, amenorrhea, and eating disorders, particularly with gymnasts, dancers, and

Table 11–5. Composition of Sports Supplements*

	CHO		*PRO*		*Fat*			
Bar	*g*	*% total cal*	*g*	*% total cal*	*g*	*% total cal*	*Kcal*	*Comments*
Balance Bar (chocolate, 1.76 oz)	22	46	14	29	6	28	190	High-fructose corn syrup is a primary ingredient Low in lactose
Boulder Bar (chocolate chip, 2.4 oz)	40	80	8	16	3	13.5	200	No high-fructose corn syrup Made with natural fruit juices and brown rice syrup No wheat or dairy products Added chromium picolinate
Clif Bar (chocolate chip, 2.4 oz)	51	82	4	6	3	11	250	No high-fructose corn syrup All-natural Wheat free Dairy free
Met Rx (fudge brownie, 3.3 oz)	52	65	29	36	2.5	7	320	High-fructose corn syrup as primary ingredient Added chromium picolinate and selenium Added protein formula "metamysin"

Power Bar (chocolate, 2.3 oz)	45	78	10	17	2	8	230	No high-fructose corn syrup Lactose free All-natural ingredients
Tiger's Milk Bar (milk chocolate, 2.3 oz)	24	74	4	12	2.5	17	130	High-fructose corn syrup primary ingredient Partially hydrogenated oils

CHO = carbodydrate; PRO=protein.

*All values per one-bar serving.

Data from nutrition facts food labels on products, compiled by S. Frates, MS, RD and K. Grimes, MS, RD.

ice-skaters.[11] Subclinical eating disorders have been rising, increasingly afflicting female athletes. Female athletes with subclinical eating disorders typically present with distorted body image, increased use of dieting behaviors, and excessive exercise. They have intakes of energy, protein, carbohydrate, and certain micronutrients that are below recommended levels.[12]

These dietary practices are not consistent with established nutritional guidelines and probably lessen athletic performance. Iron deficiency anemia is also a health concern of adolescent females. This concern had been attributed to a reduced intake of red meats and a higher prevalence of female vegetarians.[8] Appropriate use of a multivitamin supplement with iron is therefore recommended in anemic patients.

Nutritional assessment of athletes must begin with an evaluation of growth, including weight, height, weight for height, and standard height for age, using National Center for Health and Statistics (NCHS) growth charts. Weight and body composition may also be examined. Instruments such as fat calipers or bioelectrical impedance may be used to monitor changes in body composition with training. Difficulties in estimating appropriate body fat percentage ranges for children and adolescents due to normal prepubertal and pubertal changes in fat-free mass prompted the creation of specific age-appropriate equations by Lohman.[3] Health professionals should use caution when monitoring and discussing percentage of body fat as these age groups are sensitive to overemphasis on body image and to restrictive dieting.

Although many young Americans participate in organized sport, not all engage in regular physical activity. A survey by the Centers of Disease Control and Prevention (CDC) noted that 48 percent of girls and 26 percent of boys do not exercise vigorously on a regular basis.[13]

During the past decade, the number of overweight children has more than doubled.[13] Encouraging all youth to participate in a varied, enjoyable fitness routine is suggested. The "Fitness Pyramid for Kids" (Figure 11–1) illustrates various options and corresponding aerobic intensities.

Health professionals, including pediatricians, school-based nurses, coaches, personal trainers, and gym, fitness, and dance instructors, should promote food as the optimal fuel for exercise. These professionals must monitor for

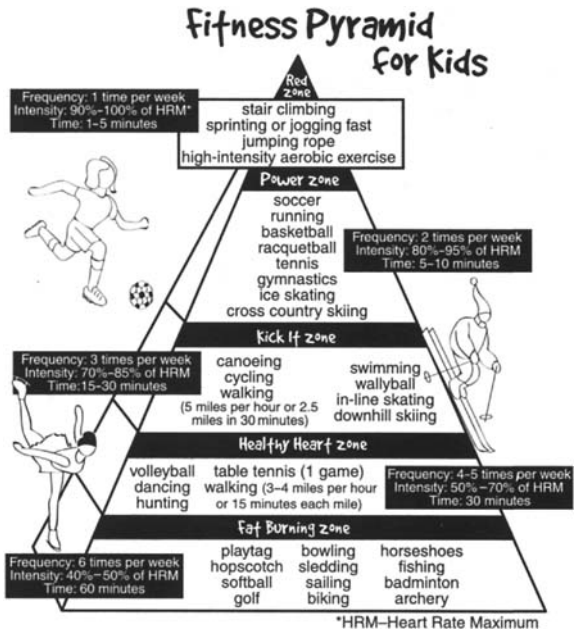


Figure 11–1. Fitness Pyramid for Kids. Reproduced with permission from American Dietetic Association. Position of the American Dietetic Association: dietary guidance for healthy children aged 2–11 years. J Am Diet Assoc 1999;99(1):97.

improper use of supplements, subclinical eating disorders, and overuse injuries related to poor nutrition. It is recommended that a registered dietitian be consulted for individual nutrition concerns of competitive athletes. A dietitian can address concerns regarding body weight, create specialized nutrition plans, and make specific fluid recommendations. It is also suggested that all health professsionals remain updated on the nutrition practices of young athletes. Continuing education in the controversial use of nutritional supplements, elevated protein diets, and subclinical eating disorders should be followed. Youth should be encouraged to follow a lifelong regimen of balanced exercise and eating rather than relying on nutritional supplements or dietary fads.

References

1. Centers for Disease Control and Prevention. Youth risk behavior surveillance—United States, 1997. Morbidity Mortality Wkly Rpt 1998;47(SS-3):1–89.
2. American Academy of Pediatrics Committee on Nutrition. Pediatric nutrition handbook. 4th ed. Klish W, editor. Elk Grove (IL): American Academy of Pediatrics; 1997.
3. Berning J, Steen S. Nutrition for sport and exercise. Gaithersburg (MD): Aspen Publishers; 1998.
4. Peterson M. Eat to compete. St. Louis: Mosby Year Book, Inc; 1996.
5. Clark N. Nancy Clark's sports nutrition guidebook. Champaign (IL): Human Kinetics; 1997.
6. American Dietetic Association. Timely statement on the nutrition guidance for child athletes in organized sports. J Am Diet Assoc 1996;96(6):610–2.
7. Jennings D, Steen S. Play hard eat right, a parent's guide to sports nutrition for children. Minneapolis (MN): Chronimed Publishing, American Dietetic Association; 1995.
8. Benardot D. Sports nutrition: a guide for the professional working with active people. Chicago (IL): American Dietetic Association; 1993.

9. Coleman E, Steen S. The ultimate sports nutrition handbook. Palo Alto (CA): Bull Publishing Co.; 1996.
10. Warren M, Shangold M. Sports gynecology: problems and care of the athletic female. Cambridge (MA): Blackwell Science, Inc.; 1997.
11. Skolnick A. Female athlete triad risk for women. JAMA 1993;270:921–3.
12. Beals K, Manore M. Nutritional status of female athletes with subclinical eating disorders. J Am Diet Assoc 1998; 98(4):419–25.
13. American Dietetic Association. Position of the American Dietetic Association: dietary guidance for healthy children aged 2–11 years. J Am Diet Assoc 1999;99(1):93–101.

Additional Resources

Sports Nutrition Organizations

American College of Sports Medicine
P.O. Box 1440
Indianapolis, IN 46206

American Dietetic Association
SCAN (Sports & Cardiovascular Nutritionists)
216 W. Jackson Blvd., Suite 800
Chicago, IL 60606-6995
800-366-1655

President's Council on Physical Fitness
701 Pennsylvania Ave., NW, Rm. 250
Washington, DC 20004

Sports Nutrition Newsletters

International Journal of Sport Nutrition
Human Kinetics Publishers, Inc.
P.O. Box 5076
Champaign, IL 61825-5076
1-800-747-4457

Penn State Sports Medicine Newsletter
PSU Center for Sports Medicine
P.O. Box 6568
Syracuse, NY 13217-9976
1-800-825-0061

Sports Science Exchange
Gatorade Sports Science Institute
P.O. Box 049005
Chicago, IL 60604-9005
312-222-7704

12

TEETH AND GUM CARE

Nancy S. Spinozzi, RD

Nutrition plays a vital role in oral health. It not only affects the integrity of oral-cavity development and maintenance but also provides protection from dental plaque and bacteria. The formation of healthy teeth, beginning at about 4 months in utero and continuing until 15 to 16 years of age, requires an adequate dietary intake of calcium, phosphorus, and vitamin D, as per the Recommended Dietary Allowance (RDA).

Fluoride plays a key role in ensuring strong enamel composition as well as providing an antibacterial effect to deter the development of dental caries. The fluoride content of drinking water varies from town to town; it is therefore recommended that parents contact their local town office for specific fluoride levels before considering fluoride supplementation. Excessive ingestion of fluoride results in mottled enamel (chronic endemic dental fluorosis). Toothpaste should not be used in children less than 2 years of age, and then only in the size of a pea. Fluoride supplementation should be administered ideally at bedtime. Table 12–1 lists the schedule for fluoride supplementation as recommended by the American Academy of Pediatric Dentistry.[1]

Dental caries remain problematic for many children. Baby-bottle caries syndrome is a common problem in children who are put to bed with a bottle containing formula or juice. Saliva flow is much reduced during sleep, leaving tooth surfaces exposed to fermentable carbohydrates (sugar and starch found in soda, candy, cereals, etc).

Likewise, children prone to gastroesophageal reflux or in whom bulimia is suspected should be carefully monitored for tooth and gum disease. Stomach acid demineralizes tooth enamel, slowly dissolving the tooth structure.

Malnutrition adversely affects tooth mineralization and saliva composition and flow. Saliva functions in a buffering capacity to prevent plaque formation. It follows that optimal nutrition provides frontline protection against caries. Riboflavin, niacin, and vitamins A, C, and D are critical for normal tooth formation; the RDA for these vitamins should be met daily. Furthermore, several foods such as cheese actually contain anticarie properties while those foods containing fermentable carbohydrate can increase cavity formation. Foods that increase masticatory action (such as fruits and vegetables) also increase salivary flow and function. Sugar exposure, when not accompanied by a frequent cleaning regimen, promotes the buildup of dental plaque.[2,3]

Several vitamin deficiencies, though rare in the United States, may be diagnosed by examination of the oral cavity. These include deficiencies of vitamin A (reduced saliva flow, enamel hypoplasia), vitamin C (swollen, bleeding

Table 12–1. Dietary Fluoride Supplementation Schedule

Age	*Less than 0.3 ppm F*	*0.3–0.6 ppm F*	*More than 0.6 ppm F*
Birth–6 mo	0	0	0
6 mo–3 yr	0.25 mg	0	0
3–6 yr	0.50 mg	0.25 mg	0
6 yr–max 16 yr	1 mg	0.50 mg	0

Ppm = parts per million; F = fluorine.
Reproduced with permission from American Academy of Pediatric Dentistry Reference Manual 1997–98.

gingival and periodontal tissue), vitamin K (bleeding gingiva), vitamin B complex (hypertrophy and atrophy of tongue papillae, angular cheilosis), thiamine (oral hypersensitivity), folic acid (ulcerations of tongue, pharynx, esophagus), and cobalamin (tongue ulcerations).[3]

Routine daily cleaning with a moist cloth or infant toothbrush should begin as soon as any tooth erupts, by 6 months of age. In older children and adolescents, daily brushing with fluoridated toothpaste is necessary, accompanied by the use of fluoride-containing oral rinses.

In summary, optimal nutrition and oral hygiene are critical to the prevention of oral diseases and to the development and maintenance of oral health.

References

1. American Academy of Pediatric Dentistry. Reference manual 1995–96. Pediatr Dent 1995;17(6):24.
2. Shaw JH. Nutritional aspects of pediatric dental-oral health problems. In: Walker WA, Watkins JB, editors. Nutrition in pediatrics. 2nd ed. Hamilton: B.C. Decker, Inc.; 1997. p. 680–1.
3. Nutrition and oral health. In: American Academy of Pediatrics Committee on Nutrition. Kleinman RE, editor. Pediatric nutrition handbook. 4th ed. Elk Grove (IL): American Academy of Pediatrics; 1998.

13

VITAMIN AND MINERAL SUPPLEMENTS

Kathleen M. Gura, PharmD, BCNSP

The tables that follow summarize the composition of oral multivitamin and multivitamin/mineral products in common use in pediatrics, as well as any inactive ingredients and other information. When taken according to the manufacturer's guidelines, these agents provide or exceed the RDA for most of the nutrients listed. Each table notes the dose for the appropriate age group. Table 13–1 lists the fat-soluble vitamins, while Table 13–2 summarizes the water-soluble components. Table 13–3 lists the mineral content of each product as well as any inactive ingredients and miscellaneous information that may be of value to the practitioner. Table 13–4 lists common parenteral and oral electrolyte supplementation products. Information regarding the composition of parenteral multivitamin and trace element products may be found in Chapter 17 (Parenteral Nutrition).

Table 13–1. Vitamin and Mineral Supplements

Product	*Age Group*	*Dose*	*A*	*D*	*E*	*K*
Tri-Vi-Sol drops	0–3 yr	1 mL qd	1500 IU palmitate	400 IU		
Poly-Vi-Sol drops	0–3 yr	1 mL qd	1500 IU	400 IU	5 IU	
ADEKS drops	0–1 yr	1 mL qd	1500 IU total	10 µg (400 IU)	27 mg (40 IU)	0.1 mg
	1–3 yr	2 mL qd	450 µg palmitate 1 mg beta carotene			
Bugs Bunny Complete tablets	2–4 yr > 4 yr	½ tab po qd 1 tab po qd	5000 IU as acetate and beta carotene	400 IU	30 IU	
ADEKS tablets	4–10 yr > 10 yr	1 tab po qd 2 tabs po qd	4000 IU	400 IU	150 IU	150 µg
Prenatal S tablets (Goldline)	> 12 yr	1 tab po qd	4000 IU	400 IU	11 IU	
MVI capsules (Numark)	> 12 yr	1 cap po qd	5000 IU	400 IU	10 IU	
Theragran-M tablets	> 12 yr	1 cap po qd	5000 IU	400 IU	30 IU	28 µg
Theragran Liquid	> 12 yr	5 mL po qd	5000 IU	400 IU		
Centrum Liquid	> 12 yr	15 mL po qd	2500 IU	400 IU	30 IU	
Nephrocaps capsules	> 12 yr	1 cap po qd				

Table 13–2. Vitamin and Mineral Supplements

Product	*Age Group*	*Dose*	*B_1 Thiamine*	*B_2 Riboflavin*	*B_3 Niacin*
Tri-Vi-Sol drops	0–3 yr	1 mL qd			
Poly-Vi-Sol drops	0–3 yr	1 mL qd	0.5 mg	0.6 mg	8 mg
ADEKS drops	0–1 yr 1–3 yr	1 mL qd 2 mL qd	0.5 mg	0.6 mg	6 mg
Bugs Bunny Complete tablets	2–4 yr > 4 yr	½ tab po qd 1 tab po qd	1.5 mg	1.7 mg	20 mg
ADEKS tablets	4–10 yr > 10 yr	1 tab po qd 2 tabs po qd	1200 µg	1300 µg	10 mg
Prenatal S tablets (Goldline)	> 12 yr	1 tab po qd	1.84 mg	1.7 mg	18 mg
MVI capsules (Numark)	> 12 yr	1 cap po qd	2.5 mg	2.5 mg	20 mg
Theragran-M tablets	> 12 yr	1 cap po qd	3 mg	3.4 mg	20 mg
Theragran Liquid	> 12 yr	5 mL po qd	10 mg	10 mg	100 mg
Centrum Liquid	> 12 yr	15 mL po qd	1.5 mg	1.7 mg	20 mg
Nephrocaps capsules	> 12 yr	1 cap po qd	1.5 mg	1.7 mg	20 mg

B_5 Pantothenic Acid	B_6 Pryridoxine	B_{12} Cyanocobalamin	C	Folic Acid	Biotin
			35 mg		
	0.4 mg	2 µg			
3 mg	0.6 mg	4 µg	45 mg		15 µg
10 mg	2 mg	6 µg	60 mg	400 µg	
10 mg	1.5 mg	12 µg	60 mg	200 µg	50 µg
	2.6 mg	4 µg	100 mg	800 µg	
	0.5 mg	2 µg	50 mg		
10 mg	3 mg	9 µg	90 mg	400 µg	30 µg
21.4 mg	4.1 mg	5 µg	200 mg		
10 mg	2 mg	6 µg	60 mg		300 µg
5 mg	10 mg	6 µg	100 mg	1 mg	150 µg

Table 13–3. Vitamin and Mineral Supplements

Product	*Age Group*	*Dose*	*Minerals*
Tri-Vi-Sol drops	0–3 yr	1 mL qd	
Poly-Vi-Sol drops	0–3 yr	1 mL qd	
ADEKS drops	0–1 yr 1–3 yr	1 mL qd 2 mL qd	Zinc 5 mg
Bugs Bunny Complete tablets	2–4 yr > 4 yr	½ tab po qd 1 tab po qd	Elemental iron 18 mg zinc 15 mg calcium 100 mg magnesium 20 mg iodine 150 µg copper 2 mg phosphorus 100 mg
ADEKS tablets	4–10 yr > 10 yr	1 tab po qd 2 tabs po qd	Zinc 7.5 mg
Prenatal S tablets (Goldline)	> 12 yr	1 tab po qd	Elemental iron 60 mg zinc 25 mg calcium 200 mg

Inactive Ingredients	*Taste*	*Miscellaneous*
Glycerin, polysorbate 80, **SUGAR FREE,** caramel color	Fruity	Occasional deepening of color doesn't effect potency; may be given undiluted or mix with formula, juice, or other food
Glycerin, polysorbate 80, ferrous sulfate (stabilizer for B_{12}), caramel color, **SUGAR FREE**	Fruity	Store away from direct light; may be given undiluted or mix with formula, juice, or other food
Glycerin, propylene glycol, simethicone emulsion, sodium saccharin, sodium hydroxide		Shake well before each use
Sorbital, gelatin, fruit acids, starch, hydrogenated vegetable oil, aspartame, monoammonium glycyrrhizinate, carrageenan **SUGAR FREE** **contains phenylalanine** FD&C yellow #6 Lake FD&C red #40 Lake FD&C blue #1 Lake	Orange cherry grape bubble gum fruit punch	Chewable tablet
Fructose, dextrates, stearic acid, silicon dioxide, magnesium stearate, glycyrrizic acid		Chewable tablet; dye free; no artificial sweetners
Microcrystalline cellulose croscmellose sodium stearic acid hydroxypropylmethyl cellulose FD&C yellow #6 Lake FD&C red #40 Lake FD&C blue #1 Lake **SODIUM AND SUGAR FREE**		

Table 13–3. continued

Product	*Age Group*	*Dose*	*Minerals*
MVI capsules	> 12 yr	1 cap po qd	
Theragran-M tablets	> 12 yr	1 cap po qd	Elemental iron 18 mg zinc 15 mg calcium 40 mg magnesium 100 mg manganese 3.5 mg chromium 26 µg iodine 150 µg selenium 21 µg copper 2 mg phosphorus 31 mg nickel 5 µg silicon 2 mg boron 150 µg tin 10 µg vanadium 10 µg molybdenum 32 µg
Theragran Liquid	> 12 yr	5 mL po qd	
Centrum Liquid	> 12 yr	15 mL po qd	Elemental iron 9 mg zinc 3 mg manganese 2.5 mg chromium 25 µg iodine 150 µg molybdenum 25 µg
Nephrocaps capsules	> 12 yr	1 cap po qd	

Inactive Ingredients	*Taste*	*Miscellaneous*
Lactose, sucrose, polyethylene glycol, FD&C red #40 Lake, FD&C blue #2 Lake microcrystalline cellulose hydroxyprophyl methylcellulose silica gel		
Sucrose, glycerin, propylene glycol, sodium benzoate, methlyparaben lactose carboxymethylcellulose sodium		
Sucrose, ethyl alcohol (5.4% w/v), glycerin, polysorbate 80, BHA, natural and artificial flavours, food starch, edetic acid		

Table 13–4. Comparison of Mineral and Electrolyte Products

Mineral	*Salt Form*	*Cation %*	*mEq/gm*	*Available Products*
Calcium	Acetate	25	12.6	Tablets: 667 mg (169 mEq calcium) Calphron, PhosLo
	Carbonate	40	20	Tablets: 650 mg (260 mg Ca^{++}) 667 mg (266.8 mg Ca^{++}) 1.25 g (500 mg) 1.5 g (600 mg) OsCal, Caltrate Chewable tablets: 750 mg (300 mg Ca^{++}) 1.25 g (500 mg Ca^{++}) Tums, Calci-Chew Suspension: 1.25 g/5mL (500 mg Ca^{++})
	Chloride	27	13.5	Injection: 100 mg/mL (1.36 mg Ca^{++}/mL) (27 mg Ca^{++}/mL)
	Citrate	21	12	Tablets: 950 mg (200 mg Ca^{++}) Citracal Effervescent tablets: 2376 mg (500 mg Ca^{++}) Citracal Liquitabs
	Glubionate	6.5	3.3	Syrup: 1.8 g/5 mL (115 mg/5 mL Ca^{++}) Neo-Calglucon
	Gluconate	9.3	4.6	Tablets: 500 mg (45 mg Ca^{++}) 650 mg (59 mg Ca^{++}) 975 mg (87 mg Ca^{++}) 1 g (89 mg Ca^{++})

Table 13–4. continued

Mineral	*Salt Form*	*% Cation*	*mEq/gm*	*Available Products*
Calcium	Gluconate (cont'd)			Injection: 100 mg/mL (0.45 mEq/mL Ca^{++}) (9 mg Ca^{++}/mL)
	Lactate	13	9.2	Tablets: 325 mg (42.5 mg Ca^{++}) 650 mg (84.5 mg Ca^{++})
Magnesium	Chloride	11.8	9.8	Sustained release tablet: 535 mg (64 mg Mg^{++}) Slo-Mag
	Gluconate	5.9	4.8	Tablet: 500 mg (27 mg Mg^{++}) Almora, Magonate
				Liquid: 500 mg Magtrate, Magonate
	Oxide	60	50	Tablet: 400 mg (241.3 mg Mg^{++}) Mag-Ox 400
				Capsule: 140 mg (84.5 mg Mg^{++})
	Sulfate	9.9	8.12	Injection: 500 mg/mL (4 mEq/mL)
Potassium	Acetate	39.8	10.19	Injection: 2 mEq/mL
	Bicarbonate	39.1	9.99	Tablet for oral solution: 6.5 mEq K^{+} 25 mEq bicarbonate K-lyte, K-Gen
	Chloride	52.4	13.41	Capsules, sustained release: 8 mEq, 10 mEq
				Injection: 2 mEq/mL
				Powder packets: 15 mEq, 20 mEq, 25 mEq

Table 13–4. continued

Mineral	*Salt Form*	*Cation* %	*mEq/gm*	*Available Products*
Potassium	Chloride (cont'd)			Oral solution: 1 mEq/mL
				Effervescent tablets: 20 mEq, 25 mEq, 50 mEq
				Tablets, sustained release: 6.7 mEq, 8 mEq, 10 mEq
	Phosphate			Injection: 3 mM/mL (4.4 mEq K^+)
				Capsules: 250 mg (8mM) phosphate with 14.25 mEq K^+ Neutra-Phos K
Sodium	Chloride	39 3	17.1	Tablets: 650 mg (11.3 mEq) 1g (17 mEq) 2.25 g (38.5 mEq)
				Slow release tab: 600 mg (10.3 mEq)
				Enteric coated tab: 1 g (17 mEq)
				Injection: 2.5 mEq/mL 4 mEq/mL
	Lactate	20.5	8.92	Injection: 0.167 mEq/mL
	Phosphate			Injection: 3 mM/mL phosphate (4 mEq/mL Na^+)
				Solution: 4.1 mM/mL phosphate (4.82 mEq/mL Na^+) Fleet Phospho-Soda

14

FOOD ASSISTANCE AND NUTRITION EDUCATION PROGRAMS

Marilyn Bernard, MS, RD

Knowledge of age-appropriate feeding practices and good nutrition concepts cannot be implemented unless a caregiver has access to food or to financial resources to buy food. A number or programs have been created over the years to assist families in obtaining foods, many developed by the US Department of Agriculture (USDA). Table 14–1 describes USDA-directed programs for children. In addition, many state and local agencies, neighborhood health centers, and local schools and universities offer programs and services to promote the nutritional health of children.

Local food assistance programs are typically found in churches or other places of worship or are listed in the yellow pages under food pantries, food assistance, and food banks, or under social and human services. Nationwide programs such as Worldshare, Inc. (www.worldshare.org; 1-888-742-7372) and Second Harvest (www.secondharvest.org; 1-312-263-2303) have regional programs throughout the country. Additional North American programs are listed on the Winnipeg Harvest Website (www.winnipegharvest.org/links).

Table 14–1. United States Department of Agriculture Food Assistance Programs

Program	*Description*	*Eligibility Components*
Food Stamp Program	Provides food coupons or electronic benefit cards to buy food in approved stores	Must meet income criteria
Special Supplemental Nutrition Program for Women, Infants, and Children (WIC)	Provides health referrals, nutrition education, and food assistance to women, infants, and children	Women: pregnant or postpartum Children: < 5 years old Must meet income criteria
National School Lunch Program and National School Breakfast Program	Provides low-cost or free lunches and breakfasts to children in public and nonprofit schools and residential child care institutions	Must meet income criteria
Special Milk Program	Provides milk to children in schools and child care institutions that do not participate in other federal child nutrition meal service programs	Must meet income criteria
Summer Food Service Program	Provides free, nutritious meals to low-income children during school vacations	Must live in a low-income area; may be required to be enrolled in a program at the site where the meals are served
Child Care Food Program	Provides low-cost or free healthy meals and snacks in child care facilities	Must meet income criteria

Homeless Children Nutrition Program (HCNP)	Provides free food throughout the year to homeless children under the age of 6 years in emergency shelters	Must be a child under 6 years of age living in a shelter
Commodity Supplemental Food Program	Provides nutrition education and monthly packages of USDA foods to low-income infants and children up to 6 years of age. (Postpartum and breastfeeding women and elderly persons aged 60 and older are also eligible)	Must meet income criteria
Food Distribution Program Indian Reservations (FDPIR)	Provides a monthly package of USDA foods to low-income households on Indian reservations and to low-income Native Americans living near Indian reservations	Must meet income criteria

USDA = US Department of Agriculture.

References

1. Mahan LK, Escott-Stump. Food, nutrition, and diet therapy. Philadelphia: W.B. Saunders Company; 1996.
2. Committee on Nutrition, American Academy of Pediatrics. Pediatric nutrition handbook. 4th ed. The Academy; 1998.
3. Child nutrition programs. US Department of Agriculture. Available from: URL: www.usda.gov/fcs/cnp.htm

15

NUTRITIONAL ASSESSMENT IN SICK OR HOSPITALIZED CHILDREN

Christopher Duggan, MD, MPH

Hospitalized children who are acutely and/or chronically ill are at increased risk for malnutrition (Table 15–1),[1] and malnourished hospitalized patients suffer disproportionately from both infectious and noninfectious complications.[2] Nutritional assessment provides the foundation for rational and effective nutritional support (see Chapter 1), but, unfortunately, this process is especially difficult in hospitalized patients. Table 15–2 lists standard methods of nutritional assessment and difficulties in applying them to hospitalized patients.

As a result of these difficulties in interpreting standard nutritional assessment techniques, multiple, admittedly

Table 15–1. Undesirable Nutritional Practices in the Hospitalized Patient

Failure to record and accurately plot anthropometric data
Failure to recognize increased nutritional needs
Withholding meals because of diagnostic tests
Frequent rotation of staff
Failure to help feed those in need of assistance
Failure to provide food in a timely and attractive manner
Delay in nutritional evaluation
Diffusion of responsibility of nutritional care of patient

Adapted from Butterworth CE. The skeleton in the hospital closet Nutr Today 1974;2:4.

imperfect, steps are recommended. Foremost is careful physical examination of the patient for evidence of edema or dehydration, changes in body water distribution that will impact on body weight. Edema can also falsely elevate anthropometric measures of body composition such as triceps skinfold. Physical examination findings and lab-

Table 15–2. Assessment of Nutritional Status in Hospitalized Patients

Methodology	*Usual Indication*	*Problem in Applying to Hospitalized Patients*
History of weight loss	Standard risk factor for malnutrition	None
Weight for age	vs. Standard curves	Difficult to obtain; falsely affected by acute hydration changes
Height for age	vs. Standard curves	Difficult to obtain
Absolute lymphocyte count	Immune function	Falsely elevated with infection or other causes of leukocytosis
Hemoglobin	Iron status	Falsely low with phlebotomy, anemia of chronic disease
Anergy	Immune function	Many confounders (eg, renal failure, burns, sepsis)
Serum albumin	Visceral protein stores	Falsely low due to bedrest, capillary leak syndrome, renal or gastrointestinal losses, or hepatic disease
Serum prealbumin	Visceral protein stores	Falsely low in hepatic disease
Serum retinol	Vitamin A status	Acute phase response depresses blood levels and may increase urine loss

oratory evidence of gastrointestinal and hepatic disease should also be documented. Finally, concurrent laboratory assessment of the acute phase response (eg, serum C reactive protein, erythrocyte sedimentation rate) can help facilitate the interpretation of visceral protein and select micronutrient levels.

Nutritional Requirements of Hospitalized Patients: Energy

Unlike the clinical situation of prolonged fasting (eg, anorexia nervosa) or other states in which malnutrition is due solely to energy deficiency, the protein-energy malnutrition that occurs in the face of catabolic injury is less amenable to simple repletion of energy, protein, or other nutrients. Nutritional requirements of patients who are stressed by critical illness may be significantly different than those of healthy individuals (see Chapter 5, USRDA and DRIS) or other patients who are less ill. Important mechanisms by which infections or critical illness impact on these requirements are: anorexia associated with infection, decreased absorption of ingested nutrients, increased requirements of energy and protein (especially with fever), drug-nutrient interactions, and decreased energy requirements due to absence of body growth and reduced physical activity.

When considering energy requirements in the hospitalized patient, it is helpful to review the components of total energy expenditure (TEE):

$$TEE = BMR + SDA + E_{activity} + E_{growth} + E_{losses}$$

where BMR = basal metabolic rate (the amount of energy required by the body at rest and while fasted); SDA = the specific dynamic action or thermic effect of food (the energy produced as heat during digestion and metabolism of food); $E_{activity}$ = energy required for physical activity;

E_{growth} = energy needed for somatic growth; and E_{losses} = obligatory energy lost in urine and stool due to inefficiencies of absorption and metabolism.

Basal metabolic rate is the largest component of TEE, and several equations have been published to calculate BMR from readily available anthropometric data, age, and sex. The oldest and best known of these are the Harris-Benedict equations for adults (Table 15–3). In pediatrics, it has been reported that the correlation between measured and predicted BMR is highest for the equations of Schofield.[3] These data are therefore commonly referred to in two different forms: BMR reported for children based on the equations of Schofield et al[4] (Table 15–4) or based on weight (Tables 15–5 and 15–6).

Pediatric patients show significant age-related changes in the components of TEE (Figure 15–1). The 1-month-old infant has relatively low energy requirements for activity (10 kcal/kg/d) but significant needs for body growth (40–50 kcal/kg/d). In just 5 months, however, a 6-month-old infant's growth rate has slowed considerably but her activity level is much higher.

Table 15–3. Harris-Benedict Equations for Calculating Basal Metabolic Rate in Adults

Males
BMR = 66 + (13.7 × weight [kg]) + (5 × height [cm]) − (6.9 × age [yr])
Females
BMR = 665 + (9.6 × weight [kg]) + (1.8 × height [cm]) − (4.7 × age [yr])

Adapted from Harris JA, Benedict FG. A biometric study of basal metabolism. Washington (DC): Carnegie Institution of Washington; 1919. Publication No. 279.

Table 15–4. Schofield Equations for Calculating Basal Metabolic Rate in Children

Males	
0–3 years	REE = 0.167W + 15.174H – 617.6
3–10 years	REE = 19.59W + 1.303H + 414.9
10–18 years	REE = 16.25W + 1.372H + 515.5
> 18 years	REE = 15.057W + 1.004H + 705.8
Females	
0–3 years	REE = 16.252W + 10.232H – 413.5
3–10 years	REE = 16.969W + 1.618H + 371.2
10–18 years	REE = 8.365W + 4.65H + 200
> 18 years	REE = 13.623W + 23.8H + 98.2

REE = kcal/day; W = weight (kg); H = height (cm).
Adapted from Schofield W. Predicting basal metabolic rate, new standards and review of previous work. Hum Nutr Clin Nutr 1985; 39C Suppl 1:5–41.

With some exceptions (discussed below), hospitalized pediatric patients generally have lower energy requirements than healthy children; a review of the different components of TEE reveals why. Of the five components of TEE, four are often significantly reduced in the seriously ill, especially in those receiving parenteral nutrition (PN). Energy required for physical activity is usually reduced in inpatients due to bedrest and, occasionally, use of paralytic agents. Energy required for growth can also be reduced since the catabolic nature of major illness means that anabolism (the accretion of lean body mass) cannot proceed. The thermic effect of food is minimized in patients receiving parenteral as opposed to enteral nutrition. Finally, obligatory gastrointestinal losses of nutrients are less in parenterally fed patients.

Table 15–5. Assessment of Energy Requirements in Hospitalized Pediatric Patients: Step 1. Estimating Basal Metabolic Requirements

	Kcal/d			*Kcal/d*	
Body Wt (kg)	*Male*	*Female*	*Body Wt (kg)*	*Male*	*Female*
3.0	120	144	36.0	1270	1173
4.0	191	191	38.0	1305	1207
5.0	270	274	40.0	1340	1241
6.0	330	336	42.0	1370	1274
7.0	390	395	44.0	1400	1306
8.0	445	448	46.0	1430	1338
9.0	495	496	48.0	1460	1369
10.0	545	541	50.0	1485	1399
11.0	590	582	52.0	1505	1429
12.0	625	620	54.0	1555	1458
13.0	665	655	56.0	1580	1487
14.0	700	687	58.0	1600	1516
15.0	725	718	60.0	1630	1544
16.0	750	747	62.0	1660	1572
17.0	780	775	64.0	1690	1599
18.0	810	802	66.0	1725	1626
19.0	840	827	68.0	1765	1653
20.0	870	852	70.0	1785	1679
22.0	910	898	72.0	1815	1705
24.0	980	942	74.0	1845	1731
26.0	1070	984	76.0	1870	1756
28.0	1100	1025	78.0	1900	1781
30.0	1140	1063	80.0	1935	1805
32.0	1190	1101	82.0	1970	1830
34.0	1230	1137	84.0	2000	1855

Adapted from Schofield W. Predicting basal metabolic rate, new standards and review of previous work. Hum Nutr Clin Nutr 1985; 39C Suppl 1:5–41.

Table 15–6. Assessment of Energy Requirements in Hospitalized Pediatric Patients: Step 2. Multiplying Basal Metabolic Demands by Stress Factor According to Illness Severity

Clinical Condition	*Stress Factor*
Starvation	0.9
Fever	12% per degree > 37°C
Cardiac failure	1.15–1.25
Major surgery	1.20–1.30
Sepsis	1.40–1.50
Catch-up growth	1.5–2.0
Burns	1.5–2.0
Example: 18-month-old, 12 kg male child admitted with sepsis and respiratory distress. Intubated and heavily sedated.	
Basal metabolic demands: 625 kcal/d	
Stress factor: × 1.4 = 875 kcal per day = estimated energy requirement	

Note: Recommended dietary allowance for an 18-month-old is 102 kcal/kg/d. Use of this estimate would lead to 1224 kcal/d being calculated, almost 40% more than estimated above.

As a result of these metabolic factors, it is generally recommended that the energy needs of hospitalized patients be estimated by calculating or measuring BMR (see below for discussion of indirect calorimetry) then estimating a stress factor by which BMR should be multiplied to achieve an estimated TEE. Table 15–6 provides suggested stress factors in critical illness, and an example of a critically ill patient's estimated energy needs. These factors are substantially less than the activity factors cited in Table 5–4, ranging from 1.6 to 2.0, that are used to estimate the daily energy needs of healthy children.

The "gold standard" for energy balance is weight gain or loss over time. Serial monitoring of body weight is

therefore the most effective measure of whether energy requirements are being met.

Indirect Calorimetry

The application of indirect calorimetry in measuring a patient's resting energy expenditure (REE) has shed much light on the subject of caloric requirements in the hospital setting. As the name implies (calor is the Latin word for heat), indirect calorimetry is the determination of heat production of a biochemical reaction by measuring uptake of oxygen and liberation of carbon dioxide. (This is in contrast to direct calorimetry, wherein the heat produced by the body at rest is measured.) Oxygen consumption (VO_2) and carbon dioxide production (VCO_2) measured by the calorimeter are entered into the Weir equation to calculate REE, as follows:

$$REE = (3.94 \times VO_2) + (1.06 \times VCO_2) - (2.17 \times UUN)$$

where UUN = urinary N excretion, used as a correction factor for protein oxidation.

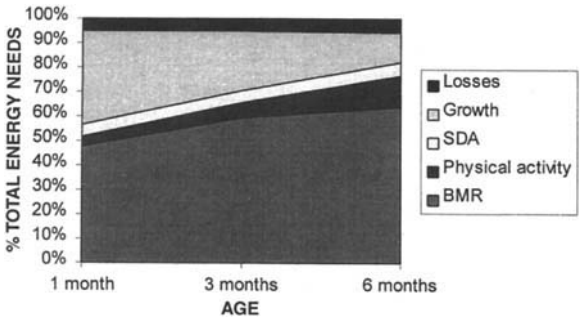


Figure 15–1. Factorial estimates of energy requirements. SDA = specific dynamic action or thermic effect of food; BMR = basal metabolic rate. Adapted from Tsang RC, Nichols BL, editors. Nutrition during infancy. C.V. Mosby Co.; 1988. p. 6.

Indirect calorimetry can also help determine whether a patient is being overfed. The ratio of VCO_2 to VO_2 is termed the "respiratory quotient" (RQ) and is used to estimate substrate oxidation. For example, in the case of pure glucose oxidation, 1 mole of carbohydrate reacts with 6 moles of oxygen to create 6 moles each of carbon dioxide and water:

$$C_6H_{12}O_6 + 6\ O_2 \rightarrow 6\ CO_2 + 6\ H_2O$$

The RQ would then be 6/6 = 1.0. When long chain fat such as palmitic acid is oxidized with 23 moles of oxygen, 16 moles each of carbon dioxide and water are produced:

$$CH_3(CH_2CH_2)_7COOH + 23\ O_2 \rightarrow 16\ CO_2 + 16\ H_2O$$

The RQ is then 16/23 = 0.695.

Thus, the RQ in a fasted state is normally 0.70 to 1.00, a range that usually represents a mixed substrate oxidation. The lower RQ noted for lipid oxidation has been used as a rationale for feeding patients with advanced lung disease a diet higher in fat than in carbohydrate so as to avoid an increased carbon dioxide load to excrete. This remains controversial, however.[5]

When excess energy is provided, lipogenesis results as follows:

$$9\ C_6H_{12}O_6 + 8\ O_2 \rightarrow 2\ CH_3(CH_2CH_2)_7COOH + 22\ CO_2 + 22\ H_2O$$

The resulting RQ = 22/8 = 2.75.

Therefore, the finding of an RQ significantly greater than 1.0 is consistent with energy intake in excess of energy requirements. Other reasons would include hyperventilation (CO_2 excreted at high rates) or failure to achieve a steady state in gas measurement.

While REE measurements are usually taken to approximate BMR, REE actually includes BMR, plus nonshivering thermogenesis, and stress hypermetabolism. The difference between REE and BMR is estimated to be 10%.

An alternative to indirect calorimetry in calculating REE is the Fick equation:

$$REE = CO \times Hgb \times (SaO_2 - SvO_2) \times 95.18$$

where CO = cardiac output (L/minute) as measured with a thermodilution pulmonary catheter; Hgb = hemoglobin concentration (g/dL); SaO_2 = oxygen saturation in arterial blood; SvO_2 = oxygen saturation in mixed venous blood.

This equation can obviously only be applied to patients whose cardiac output is measured with a pulmonary artery catheter, which is not a routine pediatric intensive care unit (ICU) procedure.

Studies have underlined the standard practice not to use the published US Recommended Dietary Allowances (USRDAs) to estimate a catabolic patient's energy requirement. Indeed, other estimates of basal metabolic rate can either overestimate or underestimate measured REE.[6] Many studies that directly or indirectly measure energy expenditure via calorimetry or other means have validated this approach of estimating energy needs. Moreover, they have emphasized that the use of the USRDAs and other standard formulas may substantially overestimate caloric requirements in the ICU setting.

Two common clinical scenarios among hospitalized pediatric patients in which energy needs are substantially higher than might be predicted are thermal injuries and nutritional rehabilitation (ie, catch-up growth). Nutritional therapy of burn patients is discussed in Chapter 19, and that of patients recovering from malnutrition/growth failure in Chapter 27.

Refeeding Syndrome

Refeeding syndrome refers to a constellation of fluid, electrolyte, and metabolic abnormalities that occur upon

aggressive nutritional support in the malnourished host.[7] During chronic malnutrition, lean body mass is broken down and total body stores of nitrogen, phosphorus, magnesium, and potassium are depleted. Nevertheless, their serum levels are usually maintained in the normal range. Upon refeeding, however, intracellular protein synthesis and insulin released due to carbohydrate provision combine to increase cellular uptake of these cations, leading to precipitous drops in serum concentrations. The clinical manifestations of hypophosphatemia include hemolytic anemia, muscle weakness (especially diaphragmatic muscle), and decreased cardiac output. In conjunction with hypokalemia and hypomagnesemia, cardiac failure and fluid overload may occur.

Pediatric patients at highest risk of refeeding syndrome include those with severe weight loss (eg, patients with anorexia nervosa, cancer cachexia, and other cases of severe malnutrition) as well as those patients having been on prolonged intravenous hydration. Serial monitoring of serum electrolytes once to twice per day in the early stages of nutritional recovery are indicated in these patients, with supplementation titrated to serum concentrations. Routine phosphate supplementation is usually recommended in the initial inpatient treatment of anorexia nervosa patients (see Chapter 24, Eating Disorders.)

Nutritional Requirements of Hospitalized Patients: Protein

Protein requirements in disease are generally thought to be higher than in health due to the increased urinary nitrogen losses characteristic of the catabolic state, gastrointestinal and skin losses, and the increased requirement for protein synthesis. As reviewed in Chapter 4, Laboratory Assessment of Nutritional Status, measurement of nitrogen balance is the most direct way to measure whether the nutri-

tion provided to an individual patient is adequate in protein. In the absence of nitrogen balance data, one often relies on serum concentrations of visceral proteins such as albumin, prealbumin, and retinol binding protein. As a general rule, protein intakes roughly 100 to 150% of the USRDA for age are used for hospitalized pediatric patients.

Ideally, the protein provided to a patient in parenteral solutions should not be used as a fuel source per se but as amino acid substrates for enzyme synthesis and lean body mass accretion. In cases where the parenteral nutrition is providing far less than the BMR for energy, the amino acids will be used as a substrate, which is why this form of PN is referred to as an "expensive" and ineffectual manner of nutrition. Some centers therefore do not commonly include protein in summing up energy intake from PN.

Instead, the ratio of nonprotein energy (kcal) to protein intake (grams of nitrogen) is estimated as a measure of adequate energy and protein balance; ratios between 150 and 250:1 are acceptable. See Chapter 17, Parenteral Nutrition, for details.

Micronutrients and Other Essential and Conditionally Essential Nutrients

The concept that certain nutrients, synthesized endogenously in adequate amounts in conditions of health, may become essential in conditions of catabolism continues to be an exciting area in the field of clinical nutrition. The significant nitrogen loss in the urine of catabolic patients in the ICU setting certainly makes it understandable that some amino acids may be "conditionally essential." Among the amino acids studied in this regard are glutamine and arginine. Other micronutrients essential in health but more so in illness include vitamin A and zinc. It is likely that the clinical nutritionist of the future will have a large variety of

disease-specific nutrient mixtures from which to choose to optimally nourish the hospitalized patient.[8]

References

1. Hendricks KM, Duggan C, Gallagher L, et al. Malnutrition in hospitalized pediatric patients: current prevalence. Arch Pediatr Adolesc Med 1995;149:1118–22.
2. Naber TH, Schermer T, de Bree A, et al. Prevalence of malnutrition in nonsurgical hospitalized patients and its association with disease complications. Am J Clin Nutr 1997; 66(5):1232–9.
3. Kaplan A, Zemel B, Neiswender K, Stallings V. Resting energy expenditure in clinical pediatrics: measured versus predicted equations. J Pediatr 1995;127:200–5.
4. Schofield W. Predicting basal metabolic rate, new standards and review of previous work. Hum Nutr Clin Nutr 1985; 39C Supp 1:5–41.
5. Silberman H, Silberman A. Parenteral nutrition, biochemistry, and respiratory gas exchange. J Parenteral Enteral Nutr 1986;10:151–4.
6. Cross-Bu J, Jefferson L, Walding D, et al. Resting energy expenditure in children in a pediatric intensive care unit: comparison of Harris-Benedict and Talbot predictions with indirect calorimetry values. Am J Clin Nutr 1998;67:74–80.
7. Solomon S, Kirby D. The refeeding syndrome: a review. J Parenteral Enteral Nutr 1990;14:90–7.
8. Furst P. Old and new substrates in clinical nutrition. J Nutr 1998;128:789–96.

16

ENTERAL NUTRITION

Kattia M. Corrales, RD,
Lori J. Bechard, MEd, RD, CNSD,
Kelly A. Kane, MS, RD, and
Deanne K. Kelleher, RD

Nourishment provided through the alimentary tract, either naturally by oral feeding or artificially through use of a feeding tube, is known as enteral nutrition. The alimentary tract should be the first choice for nutritional support. It offers several advantages over intravenous provision of nutrients, otherwise known as parenteral nutrition (Table 16–1).

Tube feeding is indicated when a child or infant is unable to meet nutritional needs orally (Table 16–2). Tube feeding can provide either total or supplemental nutrition. It can be used for short-term rehabilitation or long-term nutritional management. In the child with anorexia, a differential diagnosis can help identify the cause of anorexia and the anticipated duration of tube feeding (Table 16–3). This chapter offers guidelines for choosing the delivery

Table 16–1. Advantages of Enteral Versus Parenteral Nutrition

- Reduced risk of infection and metabolic abnormalities
- Maintains and can help restore the integrity of gastrointestinal mucosa
- May facilitate restoration of digestive enzymes
- Less expensive than parenteral nutrition
- Mimics standard human nutrition

route and equipment for tube feeding, selecting formulas, initiating and advancing feedings, monitoring and evaluating response to therapy, initiating transitional feeding, and home tube feeding.

Table 16–2. Indications for Tube Feeding

Decreased ability to ingest nutrients by mouth
Neurologic disorders
Coma
Severe mental retardation
Cerebral palsy affecting oral motor skills
Anatomic abnormalities
Facial trauma
Congenital anomalies, ie, TEF
Tumor or other mass
Prematurity (< 34 weeks)
Inability to meet full nutrient needs orally
Increased metabolic needs
Burns
Sepsis
Trauma
Congenital heart disease
Bronchopulmonary dysplasia
Anorexia (see Table 16–3)
Psychosocial disorders
Anorexia nervosa
Nonorganic growth failure
Altered absorption or metabolism requiring modification of diet
Chronic diarrhea
Short bowel syndrome
Inflammatory bowel disease
Glycogen storage disease (types I and III)
Chronic intestinal pseudo-obstruction
Pancreatitis
Amino or organic acidopathies

TEF = tracheoesophageal fistula.

Adapted from Davis A. Indications and techniques for enteral feeds. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994. p. 68.

Routes and Equipment

Small bore silicone or polyurethane tubes are placed nasally for anticipated usage of 3 months or less;[1] larger bore tubes for extended use are placed endoscopically or surgically. Nasogastric tubes are easily placed and often the first consideration for enteral nutrition therapy. Bolus feeding into the stomach can mimic typical meal patterns; nocturnal feedings supplement oral intake. Transpyloric feeding directly into the small bowel allows for use of the gastrointestinal tract despite poor tolerance to gastric feedings

Table 16–3. Differential Diagnosis of Anorexia

Acquired immunodeficiency syndrome (AIDS)
Acute or chronic infection
Cancer
Chronic disease
eg, cystic fibrosis, liver disease, sickle cell disease
Cyanotic heart disease
Drugs
Aminophylline
Amphetamines
Antihistamines
Antimetabolites
Chemotherapy
Digitalis
Narcotics
Endocrine disease
Esophagitis/gastroesophageal reflux
Iron deficiency
Lead poisoning
Pregnancy
Psychosocial deprivation (neglect/abuse)
Psychosocial factors
Chronic mental/environmental stress
Depression
Zinc deficiency

(Table 16–4).[2] Whereas the stomach can expand to accommodate a large bolus, the small intestine cannot. Therefore, continuous feedings are indicated when small bowel feeding is used. Methods of gastric feeding and small bowel feeding are described in Table 16–5 and Table 16–6.

Enteral feeding pumps are employed for slow drip feedings. Pumps are often attached to a pole in the hospital setting and can be programmed to the appropriate rate and volume. Portable pumps are also available for ease of mobility and travel.

Formula Selection

To appropriately select a formula, a complete nutritional evaluation must be conducted. The patient's energy and protein requirements, fluid and electrolyte status, digestive capacity, and organ system function must be assessed, and any food allergies or macronutrient sensitivity noted. Age is also an important consideration in formula selection as certain formulas are specifically designed to meet the needs of children at specific ages (eg, < 34 weeks, up to 1 year, 1 to 10 years, and > 10 years). These formulas may differ in their nutrient composition as well as in their vitamin and mineral content (Tables 16–7, 16–8, 16–9, 16–10, and 16–11).

Table 16–4. Types of Enteral Feeding

	Indications	*Contraindications*
Gastric feeding	Dysphagia Anorexia Supplement to oral intake	Severe gastroesophageal reflux Poor gastric motility
Small bowel feeding	Delayed gastric emptying Increased risk of aspiration	Nonfunctioning GI tract Inability to access intestine

Table 16–5. Gastric Feeding

	Advantages	*Disadvantages*
Orogastric	Avoids nasal passage obstruction Appropriate for infants < 34 weeks gestational age	Not appropriate for patients with gag reflex
Nasogastric	Easy intubation	Nasal or esophageal irritation Easily dislodged
Percutaneous endoscopic gastrostomy (PEG)	Fewer occlusions with larger bore tube Appearance can be hidden under clothing Open surgery not required	Invasive technique for placement Site at risk for infection Appropriate anatomy required
Surgical gastrostomy	Endoscopy not required for placement Procedure directly accesses stomach	Risks of anesthesia/surgery Open surgical wound at risk for infection

Table 16–6. Small Bowel Feeding

	Advantages	*Disadvantages*
Nasoduodenal/nasojejunal	Temporary access for small bowel feeding pH-guided placement available	Easily dislocated; may require radiographic evidence of appropriate placement
Gastrostomy-jejunostomy	Transpyloric tube may be passed through existing gastrostomy	Requires healing of gastrostomy tract prior to jejunostomy tube placement
	Intestinal access for feeding and gastric access for decompression and medications	Meticulous care of both ports necessary
Jejunostomy	Direct access to small bowel	Easily occluded

Adapted from Warman KY. Enteral nutrition: support of the pediatric patient. In: Hendricks KM, Walker WA, editor. Manual of pediatric nutrition. 2nd ed. Toronto: B.C. Decker, Inc.; 1990.

Table 16–7. Infant Formula Selection

Indications	*Formula Description*	*Age*	*Examples*
Premature	Rapidly growing preterm infants High in protein MCT containing Added glucose polymers Ca:P ratio of 2:1 Additional Ca, P, vit A, vit D, folate, Zn	< 34 weeks gestational age and/or < 2 kg	Breastmilk + Enfamil Human Milk Fortifier Breastmilk + Similac Natural Care Enfamil Premature 20 & 24 Similac Special Care
		Former premies now > 2 kg or > 40 weeks corrected age*	Similac Neosure Enfacare
Normal GI tract	Cow's milk protein Lactose containing Long chain fats	Full term infants	Breastmilk Enfamil Similac Carnation Good Start
		4–6 months eating cereal and other solids*	Carnation Follow-Up Formula
Primary or secondary lactose intolerance	Milk protein isolate or Soy protein isolate Long chain fats	Full term infants	Enfamil Lactofree Similac Lactose Free Prosobee Isomil
		4–6 months eating cereal and other solids*	Carnation Follow-Up Soy

Intact protein sensitivity	Whey protein hydrolysate Long chain fats Lactose free	Full term infants	Nutramigen
Severe protein allergy	Free amino acids Long chain fats Lactose free	Full term infants	Neocate
Malabsorption Intractable diarrhea Steatorrhea	Whey protein hydrolysate Medium chain triglycerides and long chain fats Lactose free	Full term infants	Pregestimil Alimentum
Impaired fat absorption Chylous effusion Lymphatic disorder	Intact protein 86% of fat from MCT 3.5% calories from linoleic acid	Full term infants	Portagen
Decreased renal function	Intact protein Long chain fat Low iron Lower in Ca, P, maintaining 2:1 ratio Lower in K	Full term infants	Similac PM 60/40

MCT = medium chain triglycerides.

*Use may be controversial.

Table 16–8. Non-Infant Formula Selection: Need for Supplemental Nutrition or Meal Replacement

Indication	*Formula Description*	*Age*†	*Examples (listed alphabetically)**
Intact gut	Blenderized Homogenized food	1–10 years	Compleat Pediatric
	Contains fiber	>10 years	Compleat Modified
	Polymeric Most isotonic	1–10 years	Kindercal, Nutren Jr., PediaSure, ReSource Just for Kids
	Intact CHO, protein, fat 1–1.2 cal/cc May contain lactose	>10 years	Boost, Carnation Instant Breakfast, Ensure, Healthshake, Isocal, IsoSource, Meritene, NuBasics, Nutren 1.0, Osmolite, ReSource Standard, Scandi-Shake
Lactose intolerance	Polymeric Most isotonic	1–10 years	Kindercal, Nutren Jr., PediaSure, ReSource Just for Kids
	Intact CHO, protein, fat 1–1.2 cal/cc Lactose free	>10 years	Boost, Ensure, Isocal, IsoSource, NuBasics, Nutren 1.0, Osmolite, ReSource Standard, Scandi-Shake Lactose Free
Diarrhea, constipation or expected long-term use	Polymeric Most isotonic	1–10 years	Kindercal, Nutren Jr. with Fiber, PediaSure with Fiber
	Intact CHO, protein, fat 1–1.2 cal/cc Lactose free Fiber containing	>10 years	Boost with Fiber, Ensure with Fiber, FiberSource, Jevity, NuBasics with Fiber, Nutren 1.0 with Fiber, ProBalance, Ultracal

Increased protein needs	Polymeric Most isotonic Intact CHO, protein, fat 0.95–1.2 cal/cc Lactose free Contains approximately 30% more protein than standard versions	>10 years	Boost High Protein, Ensure High Protein, Entrition HN, Isocal HN, IsoSource HN, IsoSource VHN, Isotein HN, NuBasics VHP, Osmolite HN, Osmolite HN Plus, Promote, Replete
	Fiber containing		FiberSource HN, IsoSource VHN, Jevity Plus, Promote with Fiber, Protain XL, Replete with Fiber
Volume restriction	Polymeric May have higher osmolalities Most contain intact CHO and protein May contain MCT oil Lactose free May contain increased protein 1.5 cal/cc	>10 years	Boost Plus, Comply, Ensure Plus, Ensure Plus HN, IsoSource 1.5, NuBasics Plus, Nutren 1.5, Resource Plus
	2 cal/cc		Deliver 2.0, Magnacal, NovaSource 2.0, NuBasics 2.0, Nutren 2.0, TwoCal HN

Table 16–8. continued

Indication	*Formula Description*	*Age*[†]	*Examples (listed alphabetically)**
Organ system dysfunction *(Note: Efficacy of formulas may be unproven; product use may be controversial; specialized formulas are often costly)*	**Glucose Intolerance** Polymeric Isotonic Contains reduced CHO content May contain increased fat May contain increased monounsaturated fatty acids 1–1.06 cal/cc Lactose free Fiber containing	>10 years	Choice dm, DiabetiSource, Glucerna, Glytrol, ReSource Diabetic *(see also "Diarrhea, constipation, or expected long-term use" for other fiber-containing products)*
	Fat Malabsorption **Bile Acid Deficiency** **Lymphatic Disorder** Polymeric Isotonic Intact CHO, protein Increased MCT content 1.35 cal/cc Lactose free	>10 years	Lipisorb

HIV/AIDS Contains protein hydrolysate Low fat content 1.28 cal/cc Lactose free Fiber containing Contains deodorized sardine oil	>10 years	Advera
Liver Polymeric or elemental Contains increased amounts of branched chain amino acids May or may not contain MCT oil 1.2–1.5 cal/cc Lactose free	> 10 years	Hepatic-Aid, L-Emental Hepatic, NutriHep
Pulmonary Polymeric Contains reduced CHO content May contain increased fat 1.5 cal/cc Lactose free	> 10 years	NovaSource Pulmonary, NutriVent, Pulmocare, Respalor

Table 16–8. continued

Indication	*Formula Description*	*Age*[†]	*Examples (listed alphabetically)*[*]
	ARDs Contains fish oil thought to have anti-inflammatory properties		Oxepa
	Renal Polymeric Increased osmolality May contain MCT oil 2 cal/cc Lactose free Low Na, K, PO_4, Mg Increased protein content	>10 years	Magnacal Renal, Nepro, NovaSource Renal
	Decreased protein content		Amin-Aid, Renalcal, Suplena
Hyper-metabolism	Polymeric May have higher osmolalities May be low fat May contain MCT 1–1.5 cal/cc	>10 years	Glutasorb, Immun-Aid, Impact, Impact with fiber, Impact 1.5, Perative, Traumacal

	Lactose free May contain fiber May contain increased protein May contain some amino acids thought to be conditionally essential (eg, glutamine, arginine) May contain fish oil		
	Elemental		AlitraQ, Criticare HN, Crucial
Impaired digestion or impaired gut perfusion	Elemental May have higher osmolalities May contain MCT oil May be low fat Lactose free Most are fiber free May contain increased protein	1–10 years	Elecare, L-Emental Pediatric, Neocate 1+, Pediatric Vivonex
	0.8–1.0 cal/cc Contains free amino acids	>10 years	Elemental 028 Extra, L-Emental, Tolerex, Vivonex Plus, Vivonex T.E.N.

Table 16–8. continued

Indication	*Formula Description*	*Age*†	*Examples (listed alphabetically)**
	Hydrolyzed protein source	1–10 years	Peptamen Jr., Pro-Peptide for Kids
	1.0–1.5 cal/cc	>10 years	Optimental, Peptamen, Peptamen 1.5, Peptamen VHP, Pro-Peptide, Pro-Peptide VHN, Reabilan, Reabilan HN, Sandosource Peptide, Subdue, Vital HN

CHO = carbohydrate; MCT = medium chain triglycerides; ARDs = acute respiratory distress syndrome.
*Formulas commercially available as of January 2000.
†In special circumstances, some adult formulas (those indicated for >10 years) may be used when a pediatric alternative (indicated for ages 1–10) is not available.

Table 16–9. Enteral Product References: Infant (per 100 mL)

		Protein		*Fat*		*Carbohydrate*						
Formula (Manufacturer)	*Kcal/cc*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*mEq* / *Na/K*	*mg* / *Ca/P*	*mOsm/kg Water*	*mg Fe*	*Comments*
Human breastmilk	0.67	1.00 5	Whey and casein	3.90 55	Long chain fatty acids high in palmitic, oleic, linoleic and linolenic	7.20 38	Lactose	0.78 1.35	28 14	255	0.04	Preferred nutrition in human infants
Alimentum (Ross Products)	0.67	1.86 11	Casein hydrolysate, L-cystine, L-tryptophan, L-methionine, L-tyrosine	3.74 48	Safflower oil, MCT oil, soy oil	6.89 41	Sucrose, modified tapioca starch	1.29 2.04	71 51	370	1.22	
Carnation Follow Up (Nestle Clinical Nutrition)	0.67	1.76 10	Nonfat milk	2.77 37	Palm olein oil, soy oil, coconut oil, high-oleic safflower oil	8.92 53	Corn syrup solids, maltodextrin	1.15 2.33	91 61	326	1.28	
Carnation Good Start (Nestle Clinical Nutrition)	0.67	1.62 10	Reduced mineral whey protein concentrate	3.45 46	Palm olein oil, soy oil,coconut oil, high-oleic safflower oil	7.43 44	Lactose and corn maltodextrin	0.71 1.70	43 24	265	1.01	
Enfamil AR (Mead Johnson Nutritionals)	0.67	1.69 10	Whey and nonfat milk	3.42 46	Palm olein oil, soy oil, coconut oil, high-oleic sunflower oil	7.40 44	Rice starch lactose maltodextrin	1.74 1.36	53 36	Powder 230 Liquid 240	1.21	

Table 16–9. continued

		Protein		*Fat*		*Carbohydrate*						
Formula (Manufacturer)	*Kcal/cc*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*mEq* / *Na/K*	*mg* / *Ca/P*	*mOsm/kg Water*	*mg Fe*	*Comments*
Enfacare (Mead Johnson Nutritionals)	0.74	2.10 / 11	Whey and nonfat milk	3.90 / 46	Soy oil, high-oleic sunflower oil, coconut oil, MCT oil	7.90 / 43	Lactose Maltodextrin	1.13 / 2.0	89 / 49	Powder 260 Liquid 230	1.33	Formerly known as Enfamil 22
Enfamil Lactofree (Mead Johnson Nutritionals)	0.67	1.43 / 9	Milk protein isolate	3.60 / 48	Palm olein oil, soy oil, coconut oil, high-oleic sunflower oil	7.40 / 43	Corn syrup solids	0.87 / 1.9	55 / 37	200	1.22	
Enfamil with Iron 20 (Mead Johnson Nutritionals)	0.67	1.45 / 9	Whey and nonfat milk	3.60 / 48	Palm olein oil, soy oil, coconut oil, high-oleic sunflower oil	7.30 / 43	Lactose	0.80 / 1.87	53 / 36	300	1.22	
Enfamil with Iron 24 (Mead Johnson Nutritionals)	0.80	1.74 / 9	Whey and nonfat milk	4.30 / 48	Palm olein oil, soy oil, coconut oil, high-oleic sunflower oil	8.80 / 43	Lactose	0.96 / 2.25	63 / 43	360	1.46	
Enfamil Premature with Iron 20 (Mead Johnson Nutritionals)	0.67	2.00 / 12	Whey and nonfat milk	3.50 / 44	MCT oil, soy oil, coconut oil	7.50 / 44	Corn syrup solids, lactose	1.13 / 1.79	112 / 56	260	1.22	

Enfamil Premature with Iron 24 (Mead Johnson Nutritionals)	0.80	2.4 — 12	Whey and nonfat milk	4.10 — 44	MCT oil, soy oil, coconut oil	9.00 — 44	Corn syrup solids, lactose	1.39 — 2.15	134 — 67	310	1.46	
Human Milk Fortifier (Mead Johnson Nutritionals)	*Per packet 3.5	0.15 — 20	Whey and casein	0.02 — 4	From caseinate	0.68 — 76	Corn syrup solids, lactose	0.08 — 0.11	23 — 11	N/A		
Isomil (Ross Products)	0.67	1.65 — 10	Soy protein isolate, L-methionine	3.69 — 49	High-oleic safflower oil, coconut oil, soy oil	6.96 — 41	Corn syrup, sucrose	1.29 — 1.87	71 — 51	230	1.22	
Isomil DF (Ross Products)	0.67	1.80 — 11	Soy protein isolate, L-methionine	3.69 — 49	Soy oil, coconut oil	6.82 — 40	Corn syrup, sucrose	1.29 — 1.87	71 — 51	240	1.22	0.6 gm fiber/100mL
Neocate (Scientific Hospital Supplies)	0.67	2.06 pro. eq. — 12	100% free amino acids	3.00 — 41	Hybrid safflower oil, refined vegetable oil (coconut oil, soy oil)	7.80 — 47	Corn syrup solids	1.07 — 2.67	83 — 62	342	1.23	
Nutramigen (Mead Johnson Nutritionals)	0.67	1.90 — 11	Casein hydrolysate and added amino acids	3.40 — 45	Palm olein oil, soy oil, coconut oil, high-oleic safflower oil	7.40 — 44	Corn syrup solids, modified corn starch	1.39 — 1.89	64 — 43	320	1.22	

Table 16–9. continued

		Protein		*Fat*		*Carbohydrate*						
Formula (Manufacturer)	*Kcal/cc*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*mEq* / *Na/K*	*mg* / *Ca/P*	*mOsm/kg Water*	*mg Fe*	*Comments*
Pregestimil (Mead Johnson Nutritionals)	0.67	1.90 / 11	Casein hydrolysate, L- cystine, L-tyrosine, L-tryptophan	3.80 / 48	MCT oil, corn oil, soy oil, high-oleic safflower oil	6.90 / 41	Corn syrup solids, dextrose and modified corn starch	1.13 / 1.90	78 / 51	320	1.22	
Pregestimil 24 (Mead Johnson Nutritionals)	0.80	2.30 / 12	Casein hydrolysate, L-cystine, L-tyrosine, L-tryptophan	4.50 / 48	MCT oil, corn oil, soy oil, high-oleic safflower oil	8.30 / 41	Corn syrup solids, dextrose and modified corn starch	1.39 / 2.28	93 / 61	320	1.54	
Prosobee (Mead Johnson Nutritionals)	0.67	1.73 / 10	Soy protein isolate and L-methionine	3.70 / 48	Palm olein oil, soy oil, coconut oil, high-oleic sunflower oil	7.30 / 42	Corn syrup solids	1.04 / 2.08	71 / 56	200	1.22	
RCF (Ross Products)	0.40	2.00 / 20	Soy protein isolate, L-methionine	3.60 / 80	Soy oil, coconut oil	N/A* / N/A*	To be selected by practitioner	1.29 / 1.87	70 / 50	N/A (will depend on type of CHO selected)	1.22	*If applicable add carbohydrate source *Nutrients based on 1:1 dilution of concentrate

Similac with Iron 20 (Ross Products)	0.67	1.40 — 8	Nonfat milk and whey protein concentrate	3.70 — 49	High-oleic safflower oil, coconut oil, soy oil	7.30 — 43	Lactose	0.71 — 1.80	53 — 28	300	1.22	
Similac with Iron 24 (Ross Products)	0.80	2.19 — 11	Nonfat milk	4.25 — 47	Soy oil, coconut oil	8.47 — 42	Lactose	1.19 — 2.72	73 — 57	380	1.45	
Similac Lactose Free (Ross Products)	0.67	1.45 — 9	Milk protein isolate	3.65 — 49	Soy oil, coconut oil	7.23 — 43	Corn syrup solids, sucrose	0.88 — 1.85	57 — 38	230	1.22	
Similac Natural Care (Ross Products)	0.80	2.19 — 11	Nonfat milk, whey protein concentrate	4.38 — 47	MCT oil, soy oil and coconut oil	8.55 — 42	Corn syrup solids, lactose	1.51 — 2.70	169 — 94	280	0.30	
Similac Neosure (Ross Products)	0.75	1.94 — 10	Nonfat milk and whey protein concentrate	4.10 — 49	Soy oil, high-oleic safflower oil, MCT oil, coconut oil	7.69 — 41	Corn syrup solids, lactose	1.07 — 2.71	78 — 46	250	1.34	*Formerly known as Neocare
Similac PM 60/40 (Ross Products)	0.67	1.50 — 9	Whey protein concentrate, sodium caseinate	3.78 — 50	Corn oil, coconut oil, soy oil	6.89 — 41	Lactose	0.71 — 1.50	38 — 19	280	0.15	
Similac Special Care 20 (Ross Products)	0.67	1.83 — 11	Nonfat milk, whey protein concentrate	3.67 — 49	MCT oil, soy oil, coconut oil	7.16 — 42	Corn syrup solids, lactose	1.26 — 2.20	121 — 68	235	1.22	
Similac Special Care 24 (Ross Products)	0.80	2.19 — 11	Nonfat milk, whey protein concentrate	4.38 — 49	MCT oil, soy oil and coconut oil	8.55 — 42	Corn syrup solids, lactose	1.51 — 2.66	145 — 81	280	1.45	

MCT = medium chain triglycerides; CHO = carbohydrate; N/A = not available.

Table 16–10. Enteral Product References: Non-Infant (per 1000 mL)

		Protein			*Fat*		*Carbohydrate*					
Formula (Manufacturer)	*Kcal/cc*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*mEq* / *Na/K*	*mg* / *Ca/P*	*mOsm/Kg Water*	*Comments**
Advera (Ross Products)	1.28	60 / 19	Soy protein, hydrolysate, sodium caseinate	23 / 16	Canola oil, MCT oil, refined deodorized sardine oil	216 / 66	Maltodextrin, sucrose, soy fiber	46 / 73	1098 / 1098	680	• Fortified with β-carotene and omega-3 fatty acids • 20% fat as MCT • 8.9 g fiber/L • Vanilla, chocolate available
Alitraq (Ross Products)	1.0	53 / 21	Soy hydrolysate, whey protein concentrate, lactalbumin hydrolysate	16 / 13	MCT oil, safflower oil	165 / 66	Maltodextrin, sucrose, fructose	43 / 31	733 / 733	575	• Low fat elemental formula • Contains 14 g Gln/L • 53% fat as MCT oil
Amin-Aid (B. Braun McGaw)	2.0	19 / 4	Free amino acids	46 / 21	Partially hydrogenated soybean oil, soy lecithin, mono- and diglycerides	365 / 75	Maltodextrin, sucrose	< 15 / < 15	– / –	700	• Low protein formula • Designed for acute or chronic renal failure • Minimal electrolyte content • Multiple flavors available
Boost (Mead Johnson Nutritionals)	1.0	43 / 17	Milk protein concentrate	18 / 15.5	Canola oil, high-oleic sunflower oil, corn oil	173 / 67.5	Corn syrup solids, sucrose	24 / 43	1270 / 1060	610	• Multiple flavors available • High calorie version available • Fiber-containing version available

Boost High Protein (Mead Johnson Nutritionals)	1.01	61 — 24	Sodium and calcium caseinates, soy protein isolate	23 — 21	Canola oil, high-oleic sunflower oil, corn oil	139 — 55	Sucrose, corn syrup solids	40 — 54	1010 — 930	690	• Formerly known as Sustacal • High protein version of Boost
Choice DM (Mead Johnson Nutritionals)	1.06	45 — 17	Milk protein concentrate, casein	51 — 43	Canola oil, high-oleic sunflower oil, corn oil, MCT oil	106 — 40	Maltodextrin, sucrose, soy fiber	37 — 42	1060 — 1060	300–440	• Low carbohydrate content • 10% fat as MCT • 14 g fiber/L • vanilla, chocolate available
Compleat Modified (Novartis Nutrition)	1.07	43 — 16	Beef, calcium caseinate	37 — 31	Canola oil, beef	140 — 53	Maltodextrin, fruits, vegetables	43 — 36	670 — 870	300	• Blenderized formula • 4.3 g fiber/L • Unflavored
Compleat Pediatric (Novartis Nutrition)	1.0	38 — 15	Sodium and calcium caseinates, beef	39 — 35	High-oleic sunflower oil, soybean oil, MCT oil, beef	130 — 50	Hydrolyzed cornstarch, apple juice, vegetables, fruits	30 — 38	1000 — 1000	380	• Blenderized formula for children aged 1–10 • 18% fat as MCT oil • 4.4 g fiber/L • Unflavored
Deliver 2.0 (Mead Johnson Nutritionals)	2.0	75 — 15	Calcium potassium caseinates	101 — 45	Soy oil, MCT oil	200 — 40	Corn syrup	35 — 43	1010 — 1010	640	• 30% fat as MCT oil
Diabetisource (Novartis Nutrition)	1.0	50 — 20	Calcium caseinate, beef	49 — 44	High-oleic sunflower oil, canola oil, beef fat, emulsifiers	90 — 36	Maltodextrin, fructose, vegetables, fruits	40 — 36	670 — 800	360	• Moderate carbohydrate content • Contains fructose, beef, vegetables, and fruits • 4.3 g fiber/L • Unflavored

Table 16–10. continued

		Protein		*Fat*		*Carbohydrate*					
Formula (Manufacturer)	*Kcal/cc*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*mEq* / *Na/K*	*mg* / *Ca/P*	*mOsm/Kg Water*	*Comments**
EleCare (Ross Products)	1.0	30 / 15	Free amino acids	48 / 42 soy oil	High-oleic safflower oil, MCT oil,	107 / 43	Corn syrup solids	20 / 38	1082 / 808	596	• Free amino acids for protein sensitivities and allergy for children aged 1–10 • 33% fat as MCT oil • Unflavored
Elemental 028 Extra (SHS)	1.0	34 / 11	Free amino acids	40 / 36	MCT oil, canola oil, hybrid safflower oil	133 / 53	Dried glucose syrup, sucrose	30 / 27	423 / 452	502–632	• 3.4 g Gln/L • 2.3 g Arg/L • Orange, unflavored available
Ensure (Ross Products)	1.06	37 / 14	Sodium and calcium caseinates, soy protein isolate, whey protein concentrate	26 / 22	High-oleic safflower oil, canola oil, corn oil, soy lecithin	169 / 64	Corn syrup, maltodextrin, sucrose	36 / 40	1272 / 1273	555	• Multiple flavors available • Fiber-containing version available • High protein version available
Ensure Plus (Ross Products)	1.5	54 / 15	Sodium and calcium caseinates, soy protein isolate	53 / 32	Corn oil, soy lecithin	200 / 53	Corn syrup, maltodextrin, sucrose	46 / 50	705 / 705	690	• High calorie version of Ensure • Multiple flavors available
Ensure Plus HN (Ross Products)	1.5	63 / 17	Sodium and calcium caseinates, soy protein isolate	50 / 30	Corn oil, soy lecithin	200 / 53	Maltodextrin, sucrose	51 / 47	1060 / 1060	650	• High calorie, high protein version of Ensure • Vanilla, chocolate available

Fiber Source (Novartis Nutrition)	1.2	43 — 14	Soy protein isolate, soy protein concentrate	39 — 29	Canola oil, MCT oil	170 — 57	Corn syrup, soy fiber, partially hydrolyzed guar gum	48 — 46	1000 — 940	490	• 50% fat as MCT oil • 10 g fiber/L • Higher protein version available
Glucerna (Ross Products)	1.0	42 — 17	Sodium and calcium caseinates	54 — 49	High-oleic safflower oil, canola oil, soy lecithin	96 — 34	Maltodextrin, fructose, soy fiber	40 — 40	705 — 705	355	• Low carbohydrate content • 14 g fiber/L
Glutasorb (Galagen Nutrition Medical)	1.0	52 — 21	Enzymatically hydrolyzed wheat protein, free amino acids	7 — 6	Soybean oil	186 — 73	Maltodextrin, modified cornstarch	26 — 26	560 — 560	575	• Low fat formula • 10 g Gln/L • 5 g Arg/L • Unflavored
Glytrol Diet (Nestle Clinical Nutrition)	1.0	45 — 18	Calcium potassium caseinate	47.5 — 42	Canola oil, high-oleic safflower oil, MCT oil, soy lecithin	100 — 40	Maltodextrin, cornstarch, fructose, pectin, gum arabic	32 — 36	720 — 720	380	• Low carbohydrate content • 15 g fiber/L
Hepatic Aid II (B. Braun McGaw)	1.2	44 — 15	Amino acids	36 — 28	Partially hydrogenated soybean lecithin, mono- and diglycerides	169 — 57	Maltodextrin, sucrose	< 15 — < 0.01	– — –	560	• Free amino acid formula with increased BCAAs designed for chronic liver disease
Immun-Aid (B. Braun McGaw)	1.0	80 — 32	Lactalbumin, L-arginine, L-glutamine, BCAAs	22 — 20	Canola oil, MCT oil	120 — 48	Maltodextrin	25 — 27	500 — 500	460	• High protein formula with BCAAs • 12.5 g Gln/L • 15.4 g Arg/L • 50% fat as MCT oil • Custard flavored

Table 16–10. continued

Formula (Manufacturer)	Kcal/cc	Protein g / % kcal	Protein Source	Fat g / % kcal	Fat Source	Carbohydrate g / % kcal	Carbohydrate Source	mEq Na/K	mg Ca/P	mOsm/Kg Water	Comments*
Impact (Novartis Nutrition)	1.0	56 / 22	Sodium and calcium caseinates, L-arginine	28 / 25	Structured lipid from palm kernel and sunflower oil, menhaden fish oil	130 / 53	Hydrolyzed cornstarch	48 / 36	800 / 800	375	• High protein formula • Contains dietary nucleotides and fish oil • 14 g Arg/L • Unflavored • Fiber-containing version available • Higher calorie version available
Isocal (Mead Johnson Nutritionals)	1.06	34 / 13	Sodium and calcium caseinates, soy protein isolate	44 / 37	Soy oil, MCT oil	135 / 50	Maltodextrin	23 / 34	630 / 530	270	• 20% fat as MCT oil • Higher protein version available
IsoSource Standard (Novartis Nutrition)	1.2	43 / 14	Soy protein isolate	39 / 29	Canola oil, MCT oil	170 / 57	Corn syrup	48 / 43	1200 / 1100	490	• 50% fat as MCT oil • Higher protein version available • Higher calorie version available
Jevity (Ross Products)	1.06	44 / 17	Sodium and calcium caseinates	35 / 29	High-oleic safflower oil, canola oil, MCT oil, soy lecithin	154 / 54	Maltodextrin, corn syrup, soy fiber	40 / 40	910 / 760	300	• 20% fat as MCT oil • 14.4 g fiber/L • Higher calorie version available

Kindercal (Mead Johnson Nutritionals)	1.06	34 — 13	Sodium and calcium caseinates, milk protein concentrate	44 — 37	Canola oil, MCT oil, corn oil, high-oleic sunflower oil	135 — 50	Maltodextrin, sucrose, soy fiber	16 — 34	850 — 850	310	• 20% fat as MCT oil • 6.3 g fiber/L
L'Emental (Galagen Nutrition Medical)	1.0	38 — 15	Free amino acids	3 — 3	Safflower oil	205 — 82	Maltodextrin, modified cornstarch	20 — 20	500 — 600	630	• Free amino acids • High carbohydrate, low fat formula • Unflavored
L'Emental Hepatic (Galagen Nutrition Medical)	1.2	44 — 15	Free amino acids	36 — 28	Soybean oil, lecithin, mono- and diglycerides	168.5 — 57	Maltodextrin, sucrose	< 15 — < 15	– — –	560	• Free amino acid formula with increased BCAAs • Custard flavored; flavor packets available
L'Emental Pediatric (Galagen Nutrition Medical)	0.8	24 — 12	Free amino acids	24 — 25	MCT oil, soybean oil	130 — 63	Maltodextrin, modified cornstarch	17 — 31	970 — 800	360	• Free amino acid formula for children aged 1–10 • 3.1 g Gln/L • 68% fat as MCT oil • Unflavored; flavor packets available
Lipisorb (Mead Johnson Nutritionals)	1.35	57 — 17	Sodium and calcium caseinates	57 — 35	MCT oil, soy oil	161 — 48	Maltodextrin, sucrose	59 — 43	850 — 850	630	• MCT oil containing formula for fat malabsorption • 85% fat as MCT oil
Magnacal Renal (Mead Johnson Nutritionals)	2.0	75 — 15	Sodium and calcium caseinates	101 — 45	Canola oil, high-oleic sunflower oil, MCT oil, corn oil	200 — 40	Maltodextrin, sugar	35 — 32	1010 — 800	570	• Moderate protein content formula designed for dialysis patients • 20% fat as MCT oil

Table 16–10. continued

Formula (Manufacturer)	Kcal/cc	Protein g / % kcal	Protein Source	Fat g / % kcal	Fat Source	Carbohydrate g / % kcal	Carbohydrate Source	mEq Na/K	mg Ca/P	mOsm/Kg Water	Comments*
Neocate 1+ (SHS)	1.0	30 / 10	L-amino acids	35 / 32	MCT oil, canola oil, hybrid safflower oils	146 / 58	Corn syrup solids	9 / 24	620 / 620	610–835	• Free amino acids • Powder contains 4.4 g Arg/L, 3.45 g Gln/L • Liquid contains 5 g Arg/L, No Gln • 35% fat as MCT oil • Orange-pineapple RTF liquid, unflavored powder available • Flavor packets available
Nepro (Ross Products)	2.0	70 / 14	Calcium potassium magnesium sodium caseinates, milk protein isolate	96 / 43	High-oleic safflower oil, canola oil, soy lecithin	222 / 43	Corn syrup, sucrose, FOS	37 / 27	1370 / 685	665	• Moderate protein content formula designed for dialysis patients
NuBasics Drink (Nestle Clinical Nutrition)	1.0	35 / 14	Calcium potassium caseinate	37 / 33	Canola oil, corn oil, soy lecithin	132 / 53	Corn syrup solids, sucrose	38 / 32	500 / 500	500–520	• Vanilla, chocolate, strawberry available • Fiber-containing version available • High calorie versions available

Nutren Junior (Nestle Clinical Nutrition)	1.0	30 12	Isolated casein and whey proteins	42 37	Soybean oil, MCT oil, canola oil, soy lecithin	128 51	Maltodextrin, sucrose	20 34	1000 800	350	• Designed for children aged 1–10 • 25% fat as MCT oil • Fiber-containing version available
Nutren 1.0 (Nestle Clinical Nutrition)	1.0	40 16	Calcium potassium caseinates	38 33	Canola oil, MCT oil, corn oil, soy lecithin	127 51	Maltodextrin, corn syrup solids	38 32	668 668	300–360	• 25% of fat as MCT oil • Vanilla, unflavored available • Fiber-containing version available
Nutren 1.5 (Nestle Clinical Nutrition)	1.5	60 16	Calcium potassium caseinates	68 39	MCT oil, canola oil, corn oil, soy lecithin	169 45	Maltodextrin	51 48	1001 1001	430–520	• 50% fat as MCT oil • Vanilla, unflavored available
Nutren 2.0 (Nestle Clinical Nutrition)	2.0	80 16	Calcium potassium caseinate	106 45	MCT oil, canola oil, soy lecithin, corn oil	196 39	Corn syrup solids, maltodextrin, sucrose	57 49	1340 1340	720	• 75% of fat as MCT oil
Nutrihep (Nestle Clinical Nutrition)	1.5	40 11	L-amino acids, whey protein	21 12	MCT oil, canola oil, soy lecithin, corn oil	290 77	Maltodextrin, modified corn starch	14 34	1001 1001	690	• Increased BCAA content • Low fat • 66% fat as MCT oil • Unflavored; flavor packets available
Nutrivent (Nestle Clinical Nutrition)	1.5	68 18	Calcium potassium caseinate	94 55	Canola oil, MCT oil, corn oil, soy lecithin	100 27	Maltodextrin	51 48	1200 1500	330–450	• Low carbohydrate content • 40% of fat as MCT oil • Vanilla and unflavored available

Table 16–10. continued

Formula (Manufacturer)	Kcal/cc	Protein g / % kcal	Protein Source	Fat g / % kcal	Fat Source	Carbohydrate g / % kcal	Carbohydrate Source	mEq Na/K	mg Ca/P	mOsm/Kg Water	Comments*
Optimental (Ross Products)	1.0	51 / 20.5	Soy protein hydrolysate, partially hydrolyzed sodium caseinate	28 / 25	Interesterified sardine oil, MCT oil, canola oil, soy oil	139 / 54.5	Maltodextrin, sucrose, FOS	46 / 45	1060 / 1060	540–580	• Increased antioxidant content • 5 g Arg/L • 28% fat as MCT oil • Vanilla, chocolate available
Osmolite (Ross Products)	1.06	37 / 14	Sodium and calcium caseinates, soy protein isolate	35 / 29	High-oleic safflower oil, canola oil, MCT oil, soy lecithin	151 / 57	Maltodextrin	28 / 26	535 / 535	300	• 20% fat as MCT oil • Unflavored
Osmolite HN (Ross Products)	1.06	44 / 17	Sodium and calcium caseinates, soy protein isolate	35 / 29	High-oleic safflower oil, canola oil, MCT oil, soy lecithin	144 / 54	Maltodextrin	40 / 40	760 / 760	300	• High protein version of Osmolite • 20% fat as MCT oil • Unflavored
Oxepa (Ross Products)	1.5	63 / 17	Sodium and calcium caseinates	94 / 55	Canola oil, MCT oil, sardine oil, borage oil	106 / 28	Sucrose, maltodextrin	57 / 50	1060 / 1060	493	• Low carbohydrate formula • Fortified with vitamin E, vitamin C, β-carotene • 25% fat as MCT oil • Unflavored
Pediasure (Ross Products)	1.0	30 / 12	Sodium and calcium caseinates, whey protein concentrate	50 / 44	High-oleic safflower oil, soy oil, MCT oil	110 / 44	Maltodextrin, sucrose	17 / 34	970 / 800	335	• Designed for children aged 1–10 • 19.5% fat as MCT oil • Multiple flavors available

Pediasure with Fiber (Ross Products)	1.0	30 12	Sodium and calcium caseinates, whey protein concentrate	50 44	High-oleic safflower oil, soy oil, MCT oil	114 44	Maltodextrin, sucrose, soy fiber	12 34	970 800	345	• Fiber containing formula for children aged 1–10 • 19.5% fat as MCT • 5 g fiber/L
Peptamen (Nestle Clinical Nutrition)	1.0	40 16	Enzymatically hydrolyzed whey	39 33	MCT oil, soybean oil, soy lecithin	127 51	Maltodextrin, cornstarch	24 39	800 700	270	• Hydrolyzed protein formula • 70% fat as MCT • Vanilla, unflavored, and flavor packets available • High protein version available
Peptamen Junior (Nestle Clinical Nutrition)	1.0	30 12	Enzymatically hydrolyzed whey protein	39 33	MCT oil, soy oil, canola oil, soy lecithin	138 55	Maltodextrin, cornstarch	20 34	1000 800	260–360	• Hydrolyzed protein formula for children aged 1–10 • 60% fat as MCT oil • Vanilla, unflavored, and flavor packets available
Peptamen 1.5 (Nestle Clinical Nutrition)	1.5	60 16	Enzymatically hydrolyzed whey protein	58 33	MCT oil, soy oil, soy lecithin	190 51	Maltodextrin, cornstarch	44 48	1000 1000	450	• High calorie version of Peptamen • 70% fat as MCT • Vanilla, unflavored, and flavor packets available
Probalance (Nestle Clinical Nutrition)	1.2	54 18	Calcium potassium caseinate	40 30	Canola oil, MCT oil, corn oil, soy lecithin	156 52	Maltodextrin, soy, polysaccharides, gum arabic	33 40	1250 1000	350–450	• 20% fat as MCT oil • 10 g fiber/L • Vanilla, unflavored available

Table 16–10. continued

Formula (Manufacturer)	Kcal/cc	Protein g / % kcal	Protein Source	Fat g / % kcal	Fat Source	Carbohydrate g / % kcal	Carbohydrate Source	mEq Na/K	mg Ca/P	mOsm/Kg Water	Comments*
Promote (Ross Products)	1.0	62.5 / 25	Sodium and calcium caseinates, soy protein Isolate	26 / 23	High-oleic safflower oil, canola oil, MCT oil, soy lecithin	130 / 52	Maltodextrin, sucrose	43 / 51	1200 / 1200	340	• High protein formula • 19% fat as MCT oil • Fiber-containing version available
ProPeptide For Kids (Galagen Nutrition Medical)	1.0	30 / 12	Enzymatically hydrolyzed whey protein	39 / 33	Soybean oil, MCT oil, canola oil, coconut oil	138 / 55	Maltodextrin, sucrose, cornstarch	20 / 34	1000 / 800	360	• Hydrolyzed protein formula for children aged 1–10 • 40% fat as MCT oil
Protain XL (Mead Johnson Nutritionals)	1.0	57 / 22	Sodium and calcium caseinates	30 / 26	Canola oil, high-oleic safflower oil, MCT oil	129 / 52	Hydrolyzed cornstarch, sucrose, oat fiber, soy fiber	40 / 45	800 / 800	340	• High protein, fiber containing formula • 20% fat as MCT oil • 9.1 g fiber/L
Pulmocare (Ross Products)	1.5	63 / 17	Sodium and calcium caseinates	93 / 55	Canola oil, MCT oil, corn oil, high-oleic safflower, soy lecithin	106 / 28	Sucrose, maltodextrin	57 / 50	1060 / 1060	475	• Low carbohydrate content • 20% fat as MCT oil • Vanilla, chocolate, strawberry available
Reabilan (Nestle Clinical Nutrition)	1.0	32 / 12.5	Enzymatically hydrolyzed casein and whey	41 / 35	MCT oil, soybean oil, canola oil, soy lecithin	132 / 52.5	Maltodextrin, cornstarch	31 / 32	500 / 500	350	• Hydrolyzed protein formula • 50% fat as MCT oil • Unflavored • High protein version available

Renalcal (Nestle Clinical Nutrition)	2.0	34 — 7	Essential L-amino acids, select non-essential amino acids, whey protein concentrate	82 — 35	MCT oil, canola oil, corn oil, soy lecithin	290 — 58	Maltodextrin, modified cornstarch	– — –	– — –	600	• Low protein formula • Designed for acute or chronic renal failure • 9 g Arg/L • 70% fat as MCT oil • Unflavored; flavor packets available
Replete (Nestle Clinical Nutrition)	1.0	63 — 25	Calcium potassium caseinates	34 — 30	Canola oil, MCT oil, soy lecithin	113 — 45	Maltodextrin, corn syrup solids	38 — 39	1000 — 1000	300–350	• High protein formula • 25% fat as MCT oil • Fiber-containing version available
Respalor (Mead Johnson Nutritionals)	1.52	76 — 20	Sodium and calcium caseinates	71 — 41	Canola oil, MCT oil	148 — 39	Corn syrup, sucrose	55 — 38	710 — 710	580	• Moderate carbohydrate content • 30% fat as MCT oil
Resource Diabetic (Novartis Nutrition)	1.06	60 — 24	Sodium and calcium caseinates, soy protein isolates	44 — 40	High-oleic sunflower oil, soybean oil, soy lecithin	94 — 36	Hydrolyzed cornstarch, fructose, partially hydrolyzed guar gum	42 — 29	930 — 930	450	• Low carbohydrate formula • 1.3 g fiber/L • Vanilla, chocolate, strawberry available
Resource Just for Kids (Novartis Nutrition)	1.0	30 — 12	Sodium and calcium caseinates, whey protein concentrate	50 — 44	High-oleic sunflower oil, soybean oil, MCT oil	110 — 44	Hydrolyzed cornstarch, sucrose (fructose – chocolate only)	17 — 33	1140 — 800	390–440	• Designed for children aged 1–10 • 20% fat as MCT oil • Vanilla, chocolate, strawberry available • Fiber-containing version available

Table 16–10. continued

		Protein		*Fat*		*Carbohydrate*					
Formula (Manufacturer)	*Kcal/cc*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*g* / *% kcal*	*Source*	*mEq* / *Na/K*	*mg* / *Ca/P*	*mOsm/Kg Water*	*Comments**
SandoSource Peptide (Novartis Nutrition)	1.0	50 / 20	Casein hydrolysate, free amino acids, sodium caseinates	17 / 15	MCT oil, soybean oils, hydroxylated lecithin	160 / 65	Hydrolyzed cornstarch	52 / 41	570 / 570	490	• Hydrolyzed protein, low fat formula • 54% fat as MCT oil • Unflavored
Subdue (Mead Johnson Nutritionals)	1.0	50 / 20	Hydrolyzed whey protein concentrate	36 / 30	MCT oil, hydrolyzed whey protein concentrate, canola oil	127 / 50	Sucrose (flavored only), maltodextrin, modified cornstarch	47 / 40	850 / 850	330–525	• Hydrolyzed protein formula • 50% fat as MCT oil • Unflavored, orange-vanilla, chocolate-almond available
Suplena (Ross Products)	2.0	30 / 6	Sodium and calcium caseinates	96 / 43	High-oleic safflower oil, soy oil, soy lecithin	255 / 51	Maltodextrin, sucrose, cornstarch	34 / 29	1430 / 730	600	• Low protein formula designed for acute and chronic renal failure
Tolerex (Novartis Nutrition)	1.0	21 / 8	Free amino acids	1.5 / 1.0	Safflower oil	230 / 91	Maltodextrin	20 / 30	560 / 560	550	• Free amino acid, high carbohydrate, low fat formula • Flavor packets available
Traumacal (Mead Johnson Nutritionals)	1.5	82 / 22	Sodium and calcium caseinates	68 / 40	Soybean oil, MCT oil	142 / 38	Corn syrup, sucrose	51 / 36	750 / 750	560	• High protein formula • 30% fat as MCT oil

TwoCal HN (Ross Products)	2.0	84 17	Sodium and calcium caseinates	89 40	Corn oil, MCT oil, soy lecithin	216 43	Maltodextrin, sucrose	64 63	1055 1055	690	• 20% fat as MCT oil • Vanilla, butter pecan available
Ultracal (Mead Johnson Nutritionals)	1.06	44 17	Sodium and calcium caseinate	45 37	Canola oil, MCT oil	123 46	Maltodextrin, oat fiber, soy fiber	40 41	850 850	310	• 40% fat as MCT oil • 14.4 g fiber/L
Vital HN (Ross Products)	1.0	42 17	Partially hydrolyzed whey, meat, soy	11 9	Safflower oil, MCT oil	185 74	Maltodextrin, sucrose	25 36	667 667	500	• Hydrolyzed protein • High carbohydrate, low fat formula • 45% fat as MCT oil
Vivonex TEN (Novartis Nutrition)	1.0	38 15	Free amino acids	3 3	Safflower oil	210 82	Maltodextrin, modified cornstarch	20 20	500 500	630	• Free amino acids • High carbohydrate, low fat formula • Unflavored; flavor packets available • High protein version available
Vivonex Pediatric (Novartis Nutrition)	0.8	24 12	Free amino acids	24 25	MCT oil, soybean oil	130 63	Maltodextrin, modified starch	17 31	970 800	360	• Free amino acid formula designed for children aged 1–10 • 68% fat as MCT oil • Unflavored; flavor packets available

FOS = Fructo-oligosaccharides; BCAAs = branched chain amino acids; Arg = arginine; Gln = glutamine; MCT = medium chain triglycerides; RTF = ready to feed.

*Most products are available in vanilla only, unless otherwise indicated.

Table 16–11. Milk-Based Oral Supplements

Product	*Manufacturer*	*Form*	*Serving Size*	*Kcal/ serving*	*Protein (g/serving)*	*Carbohydrate (g/serving)*	*Fat (g/serving)*
Boost High Protein Powder*	Mead Johnson Nutritionals	Powder	8 oz	340	21	48	9
Carnation Instant Breakfast*	Nestle Clinical Nutrition	Powder	8 oz	280	12	39	8
Carnation Instant Breakfast No Sugar Added*	Nestle Clinical Nutrition	Powder	8 oz	220	12	24	8
Forta Shake*	Ross Products	Powder	8 oz	285	17	35	8
Health-Shake	Novartis Nutrition	Liquid	6 oz	280	9	48	6
Health-Shake Aspartame Sweetened	Novartis Nutrition	Liquid	6 oz	290	12	40 (contains 3 g soluble fiber)	9
Meritene*	Novartis Nutrition	Powder	8 oz	280	18	31	9
Ovaltine*	Himmel Nutrition	Powder	8 oz	230	10	30	8
Resource Standard	Novartis Nutrition	Liquid	8 oz	250	9	40	6
Resource Plus	Novartis Nutrition	Liquid	8 oz	360	13	52	11
Scandi-Shake*	Scandi-Pharm	Powder	9 oz	600	12	70	29
Scandi-Shake Sugar Free*	Scandi-Pharm	Powder	9 oz	600	15	67	29

* Prepared with 8 oz whole milk.

Concentration and Modular Components

Formulas may be modified to enhance their caloric density by concentration of the formula base and/or by the addition of carbohydrate, protein, or fat modular components. Children with increased calorie requirements and/or volume restriction may benefit from a more concentrated formula than is typically used. Table 16–12 lists the suggested calorie distribution of feedings that should be considered when formulating recipes containing modular components. Concentration of a base infant formula, by adding less water or more concentrate, is generally recommended up to 24 to 26 calories per ounce. When higher energy densities are required, use of modular components should be considered for further caloric enhancement. Nutrient densities of commonly used modulars are listed in Table 16–13. The selection of modular type depends on the clinical situation. Table 16–14 lists situations in which particular types of macronutrient modulation may be indicated. Excessively concentrated formula may have a high renal solute load, which will require attention directed to fluid adequacy (Table 16–15).

The osmolality of the final formula feeding must also be considered. Isotonic formulations have an osmolality of approximately 300 mOsm per liter, similar to blood. Electrolyte content and small molecule macronutrients affect the osmolality of a formula to a greater degree than do intact proteins and complex carbohydrates.[3] Hyperosmolar formulas may not be well tolerated by some infants and

Table 16–12. Recommended Calorie Distribution

	Carbohydrate	*Protein*	*Fat*
Infant ≤ 2 years of age	35–55%	10–20%	35–60%
> 2 years of age	55–60%	10–20%	< 30%

Reproduced with permission from Kleinman.[5,6]

Table 16–13. Approximate Nutrient Density of Formula Enhancement Components

*Standard Infant Formula**	
Liquid concentrate	40 kcal/oz
Powdered concentrate	40 kcal/tbsp
Ready-to-use liquid	20 kcal/oz
Carbohydrate Modulars	
Polycose powder†	23 kcal/tbsp
Moducal*	30 kcal/tbsp
Protein Modulars	
Promod†	17 kcal/tbsp, 3 g protein/tbsp
Casec*	17 kcal/tbsp, 4 g protein/tbsp
Fat Modulars	
Corn, canola, or vegetable oil‡	8.3 kcal/mL
MCT oil*	7.7 kcal/mL
Microlipid*	4.5 kcal/mL

*Mead Johnson Nutritionals, Evansville (IN).
†Ross Products Division, Abbott Laboratories, Columbus (OH).
‡Pennington JAT. Bowes & Church's food values of portions commonly used. 16th ed. Philadelphia: JB Lippincott Co.; 1994. p. 127–8.

Table 16–14. Indications for Calorie Enhancement by Commonly Used Modulars

Carbohydrate	*Long Chain Fats*	*Medium Chain Triglycerides*
Congenital heart disease	Bronchopulmonary dysplasia	Chylothorax
Delayed gastric emptying	Carbohydrate malabsorption	Fat malabsorption
Failure to thrive	Diarrhea	Lymphangiectasia
Gastroesophageal reflux	Failure to thrive	Prematurity
Glycogen storage disease	Hypermetabolic states	Thoracic duct trauma
Hypermetabolic states		

Reproduced with permission from Davis A, Baker S. The use of modular nutrients in pediatrics. JPEN 1996;20:228–36.

Table 16–15. Potential Renal Solute Load of Infant Formulas (PRSL)

Formula	*Protein (g/liter)*	*PRSL (mOsm/liter)*
Human milk	10.0	36
Milk-based formula	15.0	49
Soy-based formula	18.0	57
Evaporated milk formula	27.6	102
Cow's milk (whole)	32.9	120

Reproduced with permission from Fomon SJ, Ziegler EE. Renal solute load and potential renal solute load in infancy. J Pediatr 1999;134:11–4.

children.[4] Rates of infusion and concentration should not be advanced simultaneously.

Recommendations for Concentration of Infant Formulas

Caloric density should be advanced by two to four calories per ounce every 12 to 24 hours. To calculate a recipe, determine the volume of total formula needed, multiply it by the calories per volume desired from each component, and divide by the caloric density of the component. Water is the final ingredient added to achieve the total volume desired in each recipe using a concentrated base. This step avoids dilution of the nutrient-containing components due to fluid displacement. See Table 16–16 for formula recipe calculations.

Children receiving enhanced infant formulas must be closely monitored for signs of intolerance as well as adequacy of fluid and nutrient intake. Nutrition assessment, growth, and laboratory monitoring should occur periodically, particularly with changes in clinical status.[4]

Initiation and Advancement of Feedings

Enteral feedings should be given in a manner appropriate to the child's condition and quality of life. Supplemental

Table 16–16. Formula Recipes

Standard Infant Formula (24 Calories per Ounce)	
Using powdered concentrate:	3/4 cup powdered concentrate Water to make a total volume of 20 oz
Using liquid concentrate:	1 can liquid concentrate 9 oz. water to make a total volume of 22 oz
Using ready-to-use liquid:	3 tbsp + 1 tsp powdered concentrate 32 oz ready-to-use liquid infant formula
*Sample Recipe Progression to 30 Calories per Ounce Standard Infant Formula**	
26 calories per ounce: (24 by concentration, 2 by Polycose)	3/4 cup powdered concentrate 1 tbsp + 2 tsp Polycose powder Water to make a total volume of 20 oz
28 calories per ounce: (above + 2 by corn oil)	Above recipe, but before adding water add 5 mL corn oil
30 calories per ounce: (26 by concentration, 2 by Polycose, 2 by corn oil)	Above recipe, but before adding water add additional 1 tbsp powdered concentrate

*Using powdered concentrate.

tube feedings can be given at night to allow oral intake during daytime hours; total enteral nutrition may be provided as a combination of bolus/intermittent and/or continuous drip feedings. Bolus feedings allow gravity to dictate the speed of infusion while intermittent feedings usually require a pump to deliver formula at a constant rate of infusion for one to several daily periods.[7] Continuous feedings are given over 8 to 24 hour periods at a slower rate. Table 16–17 outlines the advantages and disadvantages of each feeding method.

Feedings can be initiated using either of the above methods of administration. Progression of feedings is dictated by tolerance to the previous step in advancement. The final goal is a predetermined formula volume based on the child's nutritional requirements. Adjustments to the goal volume

Table 16–17. Administration of Enteral Feeding

	Advantages	*Disadvantages*
Bolus/ Intermittent	Can mimic or supplement meals May not require a pump Freedom of movement between feedings	Increased risk of aspiration Not recommended for children with conditions associated with poor volume tolerance (eg, gastroesophageal reflux, delayed gastric emptying)
Continuous	Preferred method for small bowel feedings Slow infusion may improve tolerance Can be given nocturnally to avoid disruption of daytime schedule and oral intake	Requires pump Child is attached to equipment for duration of feeding Overnight feedings may result in morning fullness

Reproduced with permission from Warman[2] and Davis.[7]

may be required when a child "outgrows" the calories provided by the formula volume or with changes in clinical condition. Recommendations for initiating tube feedings and attaining the desired goals are outlined in Table 16–18.

Monitoring and Evaluation of Tube Feeding

In the early stages of tube feeding, monitoring is focused on assessing the patient's tolerance to the feeding plan. Once a feeding regimen has been established, monitoring involves ensuring that the goals of nutritional therapy are being met and that tube feeding support is still required. Table 16–19 outlines parameters that should be monitored during tube feeding in the hospital and outpatient settings. Stool characteristics will differ among formulas and with breastmilk; Table 16–20 offers comparison guidelines. Finally, Table 16–21 lists some common complications in tube feeding and their solution.

Table 16–18. Guidelines for Initiation and Advancement of Continuous and Intermittent Tube Feedings

Age	*Initial Infusion*	*Advances*	*Goal*
Continuous Feeds			
Preterm	1–2 mL/kg/hr	10–20 mL/kg/d	120–175 mL/kg/d
0–12 months	1–2 mL/kg/hr	1–2 mL/kg q 2–8 hr	6 mL/kg/hr
1–6 years	1 mL/kg/hr	1 mL/kg q 2–8 hr	4–5 mL/kg/hr
> 7 years	25 mL/hr	25 mL q 2–8 hr	100–150 mL/hr
Bolus/Intermittent Feeds			
Preterm (> 1200 g)	2–4 mL/kg/feed	2–4 mL/feed	120–175 mL/kg/d
0–12 months	10–15 mL/kg q 2–3 hr	10–30 mL/feed	20–30 mL/kg q 4–5 hr
1–6 years	5–10 mL/kg q 2–3 hr	30–45 mL/feed	15–20 mL/kg q 4–5 hr
> 7 years	90–120 mL q 3–4 hr	60–90 mL/feed	330–480 mL q 4–5 hr

Reproduced with permission from Davis A. Transitional and combination feeds. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994. p. 146.

Table 16–19. Monitoring of Tube Feedings

	Monitoring	
Parameter	*Hospitalization*	*Outpatient*
Mechanical		
Tube position	Initially, then every 8 hours	Daily
Nose care	Every 8 hours	Every 8 hours
Gastrostomy/jejunostomy site care	PRN	PRN
Gastrointestinal		
Gastric residuals	Initially every 2–3 hours, until 48 hours then every 8 hours thereafter	PRN
Stool		
Frequency/consistency	Each feed	Each feed
Hemetest	Daily until 48 hours with negative results, PRN thereafter	PRN
Reducing substances	Daily during advancement, PRN thereafter	PRN
pH*	Daily until 48 hours with pH > 6.0, PRN thereafter	PRN
Metabolic		
Fluid intake and output	Daily	Daily
Urine specific gravity	Every void during advancement, every 8 hours thereafter	PRN

Table 16–19. continued

Parameter	*Monitoring*	
	Hospitalization	*Outpatient*
Serum		
Electrolytes	Daily until stable	Monthly if stable
Glucose	Daily until stable	Monthly if stable, more frequently if diabetes
Alk phos, trig, chol, Hgb, Hct, MCV, Fe, TIBC, retic count, Ca, Mg, P, BUN/Cr	Initially, then weekly	Every 1–3 months
Visceral proteins	Initially	Every 2–4 weeks until normalized
Vitamins, trace elements	PRN	PRN
Growth		
Calories, protein, vitamins, minerals	Initially, daily, then weekly thereafter	Monthly
Weight	Daily	Monthly
Height	Initially, then weekly	Monthly
Head circumference	Initially, then weekly	Monthly
Triceps skinfold	Initially, then 2–4 weeks	Every 1–3 months
Midarm muscle circumference	Initially, then 2–4 weeks	Every 1–3 months

PRN = as required.

*Not valid with enteral antibiotics.

Adapted from Davis A. Indications and techniques for enteral feeds. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994.

Transitional Feedings

Enteral to Cyclic Enteral Nutrition

Once children are able to tolerate full volume feedings at a continuous rate over 24 hours, cycling feedings should be considered to allow time off. See Table 16–22 for steps in cycling enteral feedings and Table 16–23 for an example of transitional feeding to combined night-time and bolus feedings.

Enteral to Oral Nutrition

The transition to oral feedings is a process rather than a single event.[9] It may prove to be long and challenging, especially in the child who has been deprived of oral stimulation during critical stages in development and has not acquired appropriate feeding skills. Common problems encountered in the transition to oral feeding include gagging, retching, and vomiting when orally stimulated,[10,11] and an absence of the hunger-satiety cycle. To minimize feeding disorders, oral feedings should be initiated as soon as medically possible. Early and continuous oral stimulation, such as non-nutritive sucking (sucking on a nonfeeding nipple or finger) and touch-pressure in and

Table 16–20. Stool Characteristics

Source	*Stool Characteristics*
Breast milk	Pasty, yellow, soft
Modified skim milk	Formed, greenish brown, very little free water
Whey, casein	Small volume, pasty yellow, some free water (similar to breastmilk stool)
Soy protein isolate	Soft, yellowish brown
Sodium caseinate	Formed, greenish brown, little free water
Casein hydrolysate	Green, some mucus, small volume

Reproduced with permission from Warman KY. Enteral nutrition: support of the pediatric patient. In: Hendricks KM, Walker WA, editors. Manual of pediatric nutrition. 2nd ed. Toronto: B.C. Decker, Inc.; 1990.

Table 16–21. Complications Encountered in Tube Feeding

Complication and Possible Causes	*Intervention*
Gastrointestinal	
Constipation	
Low fiber intake	Use fiber-containing formula or add fiber module. Watch for clogging of the tube if fiber used
Diarrhea	
Formula delivered too rapidly	Decrease delivery rate
Hypertonic formula	Dilute formula to isotonicity and gradually increase concentration as tolerated. Alter carbohydrate and electrolyte content
Medications that change the gut flora or have cathartic effects	If possible, change the type of medication or time it is given. Note sorbitol content in drugs. Provide antidiarrheal agents
Bacterial contamination of formula	Use aseptic preparation techniques and limit the feeding time to 8 hours. Do not add new formula to bag containing old formula
Cold formula	Allow formula to reach room temperature before feeding
Substrate intolerance	Avoid formula with intolerant substrate
Mucosal atrophy and malnutrition	Use isotonic or diluted formula. Start at low rate and advance rate slowly
Malabsorption	Use elemental or semielemental formula, MCT oil
Excessive fluid intake	Limit to maintenance fluid
Hypoalbuminemia	Alter the protein and fat content. Dipeptide-based, low-fat formula may be beneficial
Lack of fiber	Use fiber-containing formula or add fiber module

Residuals	
Hypomotility caused by medications or hyperosmolar formula	A *single* gastric residual >1.5–2 times the hourly rate should not require cessation of tube feeding. Check residuals every 4–8 hrs. Inadequate gastric emptying if > 200 cc in NG feeding or 100 cc in gastrostomy tubes.
Medications and other fluids being added to stomach	Consider prokinetic agent, patient positioning (place in right lateral decubitus position), transpyloric feeds, continuous infusion, isotonic formula.
Vomiting, nausea, bloating	
Ileus/obstruction	Stop feedings. May require parenteral nutrition if ileus is prolonged
Improper tube placement	Check tube placement
Infusion rate too rapid	Reduce rate and increase gradually as tolerated
Delayed gastric emptying	Consider prokinetic agent, patient positioning, transpyloric feeds, continuous infusion, isotonic formula
Hyperosmolar formula	Dilute formula to isotonicity then increase concentration gradually
Hypertonic medications	Change timing or type of medication. Check for sorbitol content in drugs
High fat formula	Change to lower fat formula
Unpleasant odor of formula	Use flavor packets. Use different formula
Formula too hot or too cold	Use formula at room temperature
Patient positioning	Elevate head of bed 30 to 45 degrees
Swallowing excess air	Stop feeding pump once feeding is complete
Inadequate fluid intake	Monitor fluid balance closely. Increase fluid intake by changing formula concentration or rate or via fluid boluses. Increase physical activity
Inactivity	Avoid prolonged bedrest
Obstruction	Stop feedings
Fecal impaction	Enemas or stool softeners, digital disimpaction, increase fiber and/or fluid intake

Table 16–21. continued

Complication and Possible Causes	*Intervention*
Mechanical	
Aspiration	
Gastric hypomotility	Infuse formula past pylorus. Consider continuous infusion. Elevate head of bed 30 to 45 degrees
GER	
Neurologic damage	
Clogging of tube	
Improper or infrequent irrigation of tube	Flush tubing with water every 8 hours and after all medicines
Administration of medications via feeding tube	Crush medications well or use liquid form. Assess drug/formula interactions. Avoid mixing formulas with liquid medications with a pH < 5.0
Formula too viscous for diameter of feeding tube	Mix powdered formula thoroughly or use liquid form. Add 1 tsp of meat tenderizer to 1/4 cup of water and infuse into tube. Let it stand for 1/2 hour, then aspirate. Can also use pancreatic enzymes, cranberry juice, water, and carbonated drinks to unclog tubes. Pass guided wire under fluoroscopic guidance
Improper size or placement of tube	Change tube size. Check placement
Nasopharyngeal discomfort, nasal or esophageal erosions.	Wet the mucous membranes or use a smaller tube. H_2 blockers may help with erosions
Pressure on the nares or esophagus.	Alternate nares weekly

Gastrointestinal perforation Secondary to malpositioning of tube, excessive manipulation, use of guide wires.	Can be avoided by radiologic confirmation of tube placement, minimizing manipulation, fluoroscopic guidance of nasoenteric tube placement, choice of proper size tube
Metabolic	
Azotemia High protein intake, renal immaturity or dysfunction, liver disease, metabolic dysfunction	Decrease protein content
Congestive heart failure	Reduce fluid and/or sodium content. Provide diuretics
Dehydration	
Inadequate fluid intake	Increase fluid intake
Hyperosmolar or high protein formula	Decrease formula concentration. Change to isotonic or lower protein formula
Essential fatty acid deficiency	Change formula, add 5 mL of safflower oil, add modular fat.
Hyperglycemia	
Diabetes, insulin deficiency, severe malnutrition, trauma or sepsis, excessive carbohydrate intake.	Monitor blood sugar. Initiate or adjust insulin. Reduce carbohydrate content
Hyperkalemia	Monitor laboratory values
High-potassium formula or IV potassium, renal insufficiency, acidosis	Change formula. Stop or decrease IV potassium. Give Kayexalate, insulin, glucose

Table 16–21. continued

Complication and Possible Causes	*Intervention*
Hypokalemia	IV/PO potassium. Evaluate potassium intake from formula for adequacy
Protein-calorie malnutrition, refeeding syndrome, diarrhea, insulin administration	
Hyponatremia	
Overhydration	Restrict water. Evaluate sodium intake from formula for adequacy
Sodium depletion	Add sodium chloride
Hyperphosphatemia	Change formula. Use phosphate binder or calcium supplement
Hypophosphatemia	IV/PO phosphorus. Evaluate phosphorus intake from formula for adequacy
Severe malnutrition (refeeding syndrome), insulin administration	
Liver function, abnormal	May need to stop or change formula
Overhydration	
Infusion rate too rapid	Decrease rate
High sodium intake	Decrease sodium content
Severe protein-calorie malnutrition	Monitor input and output
Weight	Decrease concentration or amount of formula; evaluate electrolyte status
Rapid or excessive gain: excessive calories and/or fluid	
Slow or no weight gain: inadequate caloric intake	Evaluate macro- and micronutrient intake. Evaluate input and output

Psychomotor development	
Child kept on tube feeding for long period of time and missed important developmental steps for learning feeding skills	Nonnutritive sucking, taking very small amounts of food from a spoon, or taking liquid from a cup to desensitize the oral area, develop an association between oral activity and satiety and learn eating and feeding skills. Consult an occupational therapist or speech pathologist

MCT = medium chain triglycerides; NG = nasogastric; GER = gastroesophageal reflux.
Adapted from Warman[2] and Davis.[8]

Table 16–22. Recommendations for Transitioning to Cyclic Enteral Feedings

Attain goal feeding volume over 24 hours

Stop feedings for approximately 2 hours then increase rate by 1–2 mL/kg or 25 mL/hr (see Table 16–18) every 4–12 hours, as tolerated, with a corresponding decrease in the number of hours of feedings per day. The total volume of feedings per 24 hours should be constant

When planning supplemental night tube feedings, consider running feeds only during planned feeding hours. Increase rate over these hours to the total volume desired

For transitioning from continuous to bolus feedings:

- consider combination bolus and nighttime continuous feedings for exclusively tube-fed patients
- cycle to desired rate of overnight feed, then decrease number of hours and provide remaining volume as bolus feedings; start bolus volume at 1–2 times the hourly rate. Large volume bolus feedings may not be well tolerated in patients with delayed gastric emptying; many patients will require a pump to allow volume of bolus feed to run over 1/2 to 1 hour

Table 16–23. Example of Transitional Feeding to Combination Night-time and Bolus Feedings*

	Feeding Regimen	*Schedule*
Day 0	80 mL/hr	Continuous
Day 1	100 mL/hr for 19 hours	4 pm to 11 am
Day 2	115 mL/hr for 16 hours + 150 mL bolus	5 pm to 9 am, bolus at 1 pm
Day 3	115 mL/hr for 12 hours + 250 mL bolus × 2	7 pm to 7 am, bolus at 11 am and 3 pm
Day 4	115 mL/hr for 10 hours + 250 mL bolus × 3	8 pm to 6 am, bolus at 9:30 am, 1 pm, and 4:30 pm

*For a 9-year-old, 30 kg child on continuous intact protein nasogastric feedings of 30 kcal/oz formula at 80 mL/hr.

around the mouth area may prevent future oral sensitivities and delayed feeding skills (Table 16–24).

Home Tube Feeding

Home tube feeding can offer psychologic benefits for the child and family and be markedly less expensive than hospitalization. If the child is medically stable and can be discharged from the hospital, is tolerating tube feedings, and is expected to require them for longer than 1 week, home tube feeding should be arranged. It is important that caretakers and the patient (if old enough) be willing and able to administer tube feedings at home, and that the former are capable of it. Caretakers should be taught how to prepare, administer, and monitor the feedings, and be able to demonstrate their skills.

It should also be confirmed that caretakers have the necessary resources for administering tube feeds, such as running water, refrigeration, and storage space (Table 16–25). An ability to cover the cost of the formula and tube feeding equipment should be established prior to discharge. Outpatient medical and nutrition follow-up should be in

Table 16–24. Guidelines for Transition from Enteral to Oral Feedings

Assessment

1. *Assess the child's nutritional and medical status*
   Is the child at or near ideal body weight? The process of weaning may produce weight loss or a plateau in weight. The child should also be medically stable before undergoing feeding changes.
2. *Assess the child's oral motor and swallowing skills*
   If the child has a history of swallowing difficulties, have the child seen by a speech and language pathologist for a swallowing evaluation. Assess for aspiration of oral feeds. Determine appropriate feeding position, placement of food bolus in mouth, food/liquid size and consistency.
3. *Assess the caretaker's readiness to begin transition*
   Caretakers will require education on process and reassurance that transition may be gradual.

Transition to oral feeds

1. *Gradual transition to bolus feeding*
   Increase the rate of feedings and lengthen the time between feedings.
2. *Adjust bolus feeding to oral feeding schedule*
   Three meals and 2–3 snacks per day.
3. *Offer oral feeds first then bolus feed and/or nighttime feeds to make up caloric deficit*
4. *Reduce tube feeding gradually*
   Tube feeding can be reduced by 25% initially to promote hunger. Continue to gradually reduce by increments of 25%, based on improvements in oral intake, feeding problems, and growth. Once the child is consuming > 75% from oral feeds, tube feeding can be stopped.
5. *Other considerations*
   Consult a behavioral psychologist and/or speech therapist if feeding difficulties.
   Use behavior modification techniques, eg, positive reinforcement and oral stimulation exercises. Offer variety in food texture, color, taste, temperature, and smell, without overwhelming the child. Do not force feed. Allow the child to play with different foods; tube feed during family mealtime to create an association between feeding and hunger.

Adapted from Davis,[7] Blackman and Nelson,[10] and Tuchman.[12]

place. Nutrition follow-up will involve continued assessment of the patient's nutritional needs and adjustments to feeding regimen as needed. Support between clinic visits can be provided by a visiting nurse or social worker. Support groups in the area may also be available for the family.

Parenteral to Enteral Nutrition

Adequacy of fluid and nutrient intake must be closely monitored in the transition from parenteral (PN) to enteral nutrition (EN). Young infants are at greatest risk due to their proportionately higher nutrient needs. Most infants and children can successfully make the transition from PN to intact formula EN once gut function has resumed and enteral access is obtained. Patients with chronic gastrointestinal disease may require slower transitions, special

Table 16–25. Required Instructions for Home Tube Feeding

Preparation
How to use sanitary techniques, eg, proper handwashing, cleaning equipment before starting, avoiding contamination of formula
How to place and check the feeding tube, if using a nasogastric tube
How to prepare enteral feeding bags, program the feeding pump, and troubleshoot alarms
Monitoring
How to distinguish signs and symptoms of intolerance, eg, vomiting, abdominal distention, diarrhea. Excessive coughing, breathing difficulties, or skin color changes may indicate improper tube placement. The tube should be removed and replaced by either the caregiver if a g-tube, or hospital personnel in the case of j-tubes.
Support
Names and important phone numbers to be provided: physician, nutrition support nurse, dietitian, and 24-hour hotline from infusion company for technical support.

formulas, and closer monitoring. Table 16–26 lists the steps in transitioning children from PN to EN and Table 16–27 gives an example of transitional feeding.

Table 16–26. Recommendations for Transitioning from Parenteral to Enteral Nutrition

1. Match PN caloric density with desired EN caloric density when feasible.
2. Once initial EN rate is tolerated, decrease PN rate milliliter for milliliter with further increases in EN (see initiation and advancement guidelines, Table 16–18).
3. For fluid restricted patients: continue lipid infusion until goal EN rate is tolerated, then concentrate formula as required to meet caloric requirements prior to discontinuing lipids. Calorie requirements of enterally fed patients are generally 10% higher than parenterally fed patients.

Table 16–27. Example of Transitional Feeding*

	Parenteral Nutrition	*20% lipids*	*Enteral Nutrition*	*Total kcal*
Day 0	15 mL/hr	2 mL/hr	–	88 kcal/kg/d
Step 1	11 mL/hr	2 mL/hr	4 mL/hr	90 kcal/kg/d
Step 2	7 mL/hr	2 mL/hr	8 mL/hr	91 kcal/kg/d
Step 3	3 mL/hr	2 mL/hr	12 mL/hr	93 kcal/kg/d
Step 4 PN	Discontinue	2 ml/hr	16 mL/hr	98 kcal/kg/d
Step 5	– to 24 kcal/oz	1 mL/hr	Increase	98 kcal/kg/d
Step 6 lipids	–	Discontinue	18 mL/hr	96 kcal/kg/d

*For a 3.6 kg infant on PN with 15% dextrose and 2.5% amino acids; EN = 20 cal/oz breastmilk; fluid limited to 120 mL/kg/day.
Adapted from Davis A. Transitional and combination feeds. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994.

References

1. American Academy of Pediatrics Committee on Nutrition. Enteral nutrition. In: Kleinman RE, editor. Pediatric nutrition handbook. 4th ed. Elk Grove (IL): American Academy of Pediatrics; 1998.
2. Warman KY. Enteral nutrition: support of the pediatric patient. In: Hendricks KM, Walker WA, editors. Manual of pediatric nutrition. 2nd ed. Toronto: B.C. Decker, Inc; 1990.
3. Alpers DH, Stenson WF, Bier DM. Enteral nutrition therapy. In: Manual of nutritional therapeutics. 3rd ed. Boston: Little, Brown and Company; 1995.
4. Davis A, Baker S. The use of modular nutrients in pediatrics. JPEN 1996;20:228–36.
5. American Academy of Pediatrics Committee on Nutrition. Formula feeding of term infants. In: Kleinman RE, editor. Pediatric nutrition handbook. 4th ed. Elk Grove (IL): American Academy of Pediatrics; 1998.
6. American Academy of Pediatrics Committee on Nutrition. Hyperlipidemia. In: Kleinman RE, editor. Pediatric nutrition handbook. 4th ed. Elk Grove (IL): American Academy of Pediatrics; 1998.
7. Davis A. Transitional and combination feeds. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994.
8. Davis A. Indications and techniques for enteral feeds. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994.
9. Glass RP, Lucas B. Making the transition from tube feedings to oral feeding. Nutr Focus 1990;5:1–8.
10. Blackman JA, Nelson CLA. Reinstituting oral feedings in children fed by gastrostomy tube. Clin Pediatr 1985;24: 434–8.
11. Blackman JA, Nelson CLA. Rapid introduction of oral feedings to tube-fed patients. Dev Behav Pediatr 1987;8:63–6.
12. Tuchman DN. Oropharyngeal and esophageal complications of enteral tube feeding. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall; 1994.

Additional Resources

Books and Journals

1. Rombeau JL, Caldwell MD. Clinical nutrition—enteral and tube feeding. 2nd ed. Philadelphia: W.B. Saunders Company; 1990.
2. Smith BC, Pederson AL. Nutrition focus—tube feeding update. Nutr Focus 1990;5:1–6.
3. Hyams JS, Treem WR, Etienne NL, et al. Effect of infant formula on stool characteristics of young infants. Pediatrics 1995;95:50–4.
4. American Society for Parenteral and Enteral Nutrition. Standards of practice for home nutrition support. Nutr Clin Pract 1999;14:151–62.
5. Shikora SA, Ogawa AM. Enteral nutrition and the critically ill. Postgrad Med J 1996;72:395–402.
6. Lord LM. Enteral access devices. Nurs Clin North Am 1997;32:685–704.
7. Clevenger FW, Rodriguez DJ. Decision-making for enteral feeding administration: the why behind where and how. Nutr Clin Pract 1995;10:104–13.
8. Marks JM, Ponsky JL. Access routes for enteral nutrition. Gastroenterologist 1995;3:130–40.
9. Mobarhan S, DeMeo M. Diarrhea induced by enteral feeding. Nutr Rev 1995;53:67–70.
10. Holden CE, MacDonald A, Ward M, et al. Psychological preparation for nasogastric feeding in children. Br J Nurs 1997;6:376–81, 384–5.

Internet Resources

Children's Nutrition Research Center: www.bcm.tmc.edu/cnrc

Kennedy Krieger Institute: www.kennedykrieger.org

Kluge Children's Rehabilitation Center Encouragement Feeding Program: http://hsc.virginia.edu/cmc/kcrc/rehab_programs/rehab_programs.html

17

PARENTERAL NUTRITION

Sharon B. Collier, MEd, RD,
Denise S. Richardson, BSc, RN,
Kathleen M. Gura, PharmD, BCNSP,
and Christopher Duggan, MD, MPH

The advent of total parenteral nutrition (TPN or alternatively, PN) in the late 1960s was a landmark in the history of nutrition, and its development has profoundly affected the management of patients with gastrointestinal failure.[1] The application of PN to patients with nongastrointestinal disease such as malignancies, to critical care, and to other conditions has been quickly adopted although explicit evidence for its efficacy in many clinical scenarios is lacking. Parenteral nutrition represents the most aggressive and expensive method of providing nutritional support, and careful consideration should be given before it is prescribed. The literature suggests that a multidisciplinary PN consult team is a cost-effective method for patient selection, assessment, and monitoring.[2,3]

Indications for Parenteral Nutrition

Although it is axiomatic that enteral nutrition is the preferred route of nutrition support, there are several medical conditions for which enteral nutrition is not feasible and for which PN is thus usually indicated (Table 17–1).

Many of the conditions noted in Table 17–1 may be of indeterminate length, so the assessment of when PN should be started is very much a clinical decision. The benefits of PN need to be weighed against the multiple risks of the therapy (see below) as well as the risks of pro-

Table 17–1. Conditions Commonly Requiring Parenteral Nutrition

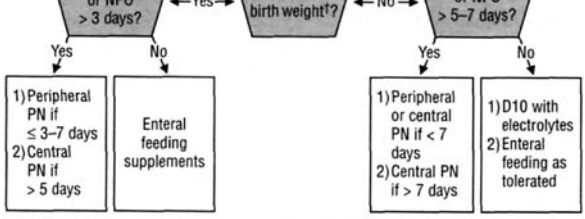
Conditions	*Examples/Comments*
Surgical gastrointestinal disorders	Gastroschisis, omphalocele, tracheoesophageal fistula, intestinal atresias, meconium ileus, peritonitis, malrotation and volvulus, Hirschsprung's disease, diaphragmatic hernia, and prolonged postoperative ileus
Short-bowel syndrome	
Pancreatitis	
Congenital heart disease	If blood supply to mesentery is compromised or dependent on patent ductus arteriosus
Acute alimentary disease	Pseudomembranous colitis, necrotizing enterocolitis, severe inflammatory bowel disease (including fistulas due to Crohn's disease), chronic or secretory diarrhea
Chronic idiopathic intestinal pseudo-obstruction syndrome	
Prematurity	
Gastrointestinal fistulas	
Bone marrow transplantation	
Hypermetabolic states	Burns, multiple trauma

viding nutrition in an alternative fashion or not at all. Because of increased metabolic requirements and decreased fuel storage in the forms of fat and protein, pediatric patients are more susceptible to the effects of starvation than are adults.[4,5] An estimate of 3 to 5 days is often used as the length of time for which the provision of 10 percent dextrose is a reasonable alternative for nutri-

tion support (Figure 17–1). If the period of minimal-to-no enteral nutrition is anticipated to be longer than 5 days, most pediatric patients would benefit from PN. In cases of severe malnutrition, low birth weight, hypermetabolism, or select other conditions, the provision of PN for less than 5 days may be justified.

If the period of minimal-to-no enteral nutrition is anticipated to be longer than 7 days, full PN with central venous access is usually indicated. Peripheral access and the infusion of a more dilute solution may be adequate for periods of less than 7 days although the limitation of solution osmolarity can make it difficult to meet the patient's full energy needs.

The ideal location for a central venous catheter tip is at the junction of the right atrium and superior vena cava. Venous flow rate is maximal in this large-diameter vessel, an important consideration when one is infusing a hypertonic solution such as PN. The practice at Children's Hospital in Boston is to document the tip location and entry site of every catheter used for PN administration

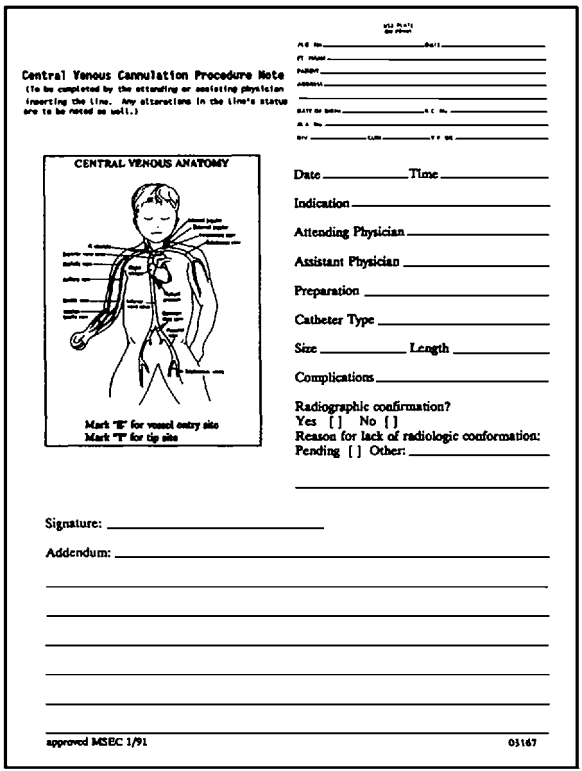


* < 5th percentile weight for age or weight for height.
† < 2500 g.

Figure 17–1. Decision tree used for the selection of method of nutrition support. GI = gastrointestinal; NPO = nothing by mouth; PN = parenteral nutrition.

(Figure 17–2). Radiographic confirmation of appropriate line-tip location is required, since malpositioned catheters can have serious and even fatal side effects.

Due to risks of phlebitis and sclerosis, the maximum osmolarity of PN for peripheral vein administration is 900 mOsm/L. This corresponds to a solution of 10 percent



Central Venous Cannulation Procedure Note
(To be completed by the attending or assisting physician inserting the line. Any alterations in the line's status are to be noted as well.)

CENTRAL VENOUS ANATOMY

Mark "E" for vessel entry site
Mark "T" for tip site

Date _____ Time _____

Indication _____

Attending Physician _____

Assistant Physician _____

Preparation _____

Catheter Type _____

Size _____ Length _____

Complications _____

Radiographic confirmation?
Yes [ ] No [ ]
Reason for lack of radiologic conformation:
Pending [ ] Other: _____

Signature: _____

Addendum: _____

approved MSEC 1/91 05167

Figure 17–2. Central line documentation used at Children's Hospital, Boston.

dextrose and 2 percent amino acid with standard amounts of electrolytes and minerals. Table 17–2 lists the osmolarity and estimated caloric density of several common PN solutions. Note that both 10 percent and 20 percent intravenous fat solutions provide an isotonic source of calories that can be given through a peripheral or central vein.

Fluids and Electrolytes

Parenteral fluid requirements in children are estimated according to the Holliday-Segar method shown in Table 17–3. Fluid requirements can be higher when there are increased losses, including insensible losses (fever or tachypnea) or sensible losses (diarrhea, vomiting, nasogastric output, ostomy losses, etc). Therefore, careful and routine clinical monitoring of hydration status is essential in the patient receiving PN.

If a patient's fluid requirement is being met but his or her energy needs are not, it is generally recommended to increase the volume of PN administered rather than to increase the concentration of nutrients in the PN. This minimizes the need to infuse hypertonic solutions, which are more damaging to the intima of blood vessels. This approach of course assumes that the patient's cardiovascular and renal systems can tolerate the increased volume of parenteral fluids.

Basal electrolyte requirements are shown in Table 17–4. Actual clinical needs may vary if exogenous losses are high (eg, diarrheal disease, diuretic use) or renal function is altered. Table 17–5 lists the estimated electrolyte losses in various gastrointestinal losses; precise quantification is possible by sending a specimen to the chemistry lab for measurement of electrolyte concentration.

Electrolyte disturbances may be corrected by increasing or decreasing the concentration of these components in the volume of PN infused in the daily prescription.

Table 17–2. Osmolarity and Energy Density of Select Parenteral Nutrition Solutions

Solution	*Osmolarity (mOsm/L)*	*Energy Density (kcal/mL)*
5% dextrose	300	0.17
10% dextrose	600	0.34
20% dextrose	1200	0.68
10% dextrose + 2% amino acid	900	0.42
20% + 2% amino acid	1500	0.76
25% + 3% amino acid	1800	0.97
30% + 3% amino acid	2200	1.14
10% lipids	276	1.1
20% lipids	258	2.0

However, acute changes in serum electrolytes should not be treated with abrupt changes in PN infusion rate. Frequent changes in PN infusion rate will adversely affect macronutrient metabolism (eg, causing swings in blood glucose) as well as alter the delivery of any medications added to PN.

Table 17–3. Daily Parenteral Fluid Requirements According to the Holliday-Segar Method

Body Weight	*Maintenance Parenteral Fluid Requirements*
0–10 kg	100 mL / kg
10–20 kg	1000 mL + 50 mL / kg over 10 kg
> 20 kg	1500 mL + 20 mL / kg over 20 kg

For example, in an 18 kg child:

1000 mL for the first 10 kg = 1000 mL
50 mL × 8 kg = 400 mL
Total maintenance fluid = 1400 mL per 24 hours

Table 17–4. Basal Electrolyte Requirements

Element	*Daily Amount*
Sodium	2–4 mEq/kg
Potassium	2–3 mEq/kg
Calcium	0.5–2.5 mEq/kg
Magnesium	0.25–0.5 mEq/kg
Phosphorus	1–2 mM/kg
Chloride	2–3 mEq/kg

Macronutrients in Parenteral Nutrition

The three macronutrients in most parenteral nutrition solutions are carbohydrate (as dextrose monohydrate), protein (as crystalline amino acids), and fat (as soybean and/or safflower emulsions) (Table 17–6).

Dextrose is the major source of calories for parenteral solutions. Initial rates of dextrose infusion should be approximately 5 mg/kg/min, and incremental increases should occur by daily increases of 2 to 5 mg/kg/min.* This usually corresponds to increments of 5 to 10 percent dextrose per day. In practice, infusion rates in neonates rarely should exceed 12 to 15 mg/kg/min. Excessive carbohydrate intake is associated not with an increased oxidation of this nutrient but conversion to fat. Hepatic steatosis, hyerglycemia, and osmotic diuresis can ensue. Although there are data published which suggest that 5 mg/kg/min is the maximum amount of glucose that critically ill children can oxidize,[6] total energy needs at this level of carbohydrate intake will often not be met.

Monitoring tolerance to infused dextrose includes frequent measurement of blood glucose, urine dipsticks, and

*Dextrose (mg/kg/min) = rate (mL/hr) × % dextrose × 0.166/weight (kg); eg, 10 mL/hr × 10% × 0.166/3kg = 5.5 mg/kg/min.

Table 17–5. Estimated Gastrointestinal Losses of Electrolytes

Fluid	*Na (mEq/L)*	*K (mEq/L)*	*Cl (mEq/L)*
Gastric	20–80	5–20	100–150
Pancreatic	120–140	5–15	40–80
Small bowel	100–140	5–15	90–130
Bile	120–140	5–15	80–120
Ileostomy	45–135	5–15	20–115
Diarrhea	10–140	10–80	10–110

Adapted from Hom X. Fluids and electrolytes. In: Barone M, editor. The Harriet Lane Handbook. 14th ed. St. Louis: Mosby-Year Book, Inc.; 1996. p. 233.

hydration status. In situations of catabolic stress, infection, or corticosteroid use, hyperglycemia is common, even in the absence of excessive carbohydrate loads. Either reducing the rate of dextrose infusion or the institution of low-dose insulin is recommended (0.01 units/kg/h, titrated as needed). Insulin is one of the few anabolic hormones in common use in patients receiving

Table 17–6. Macronutrients in Parenteral Nutrition

Macronutrient	*Energy Density*	*Examples*
Dextrose	3.4 kcal/g	10% = 10 g/dL × 3.4 = 34 kcal/dL = 0.34 kcal/mL
Amino acids	4.0 kcal/g	2% = 2 g/dL × 4.0 = 8 kcal/dL = 0.08 kcal/mL
Fats	9.0 kcal/g	10% = 10 g/dL × 9.0 = 90 kcal/dL → 1.1 kcal/mL*
		20% = 20 g/dL × 9.0 = 180 kcal/dL → 2.0 kcal/mL*

*Additional calories are provided by phospholipid emulsifiers and glycerol.

PN, and its combination with PN has been associated with better accretion of lean body mass.[7]

Crystalline amino acids are the protein source used in parenteral solutions. Initial rates of protein administration are 0.5 g/kg/d in the preterm neonate weighing less than 1.0 kg and 1.0 g/kg/d in all others. Daily advances in protein intake are made by 1.0 g/kg/d (or 0.5 g/kg/d in preterm infants). Intolerance to parenteral protein intake is marked by elevated blood urea nitrogen and rarely, elevated ammonia level. Parenteral protein requirements are listed in Table 17–7.

Amino acid solutions designed for infants have markedly different amino acid compositions than those designed for older children and adults, due to the different requirements in infants. Pediatric solutions contain more cysteine, taurine, glutamic acid, and aspartic acid than those for adults, as well as lower concentrations of methionine, glycine, and phenylalanine (Table 17–8).

Ideally, the protein provided in PN is used not as a primary fuel source (as are carbohydrates and fats), but as substrate for enzyme synthesis and lean body accretion. Therefore, some centers do not customarily include protein intake in their calculation of energy provided by the PN. Instead, energy intake is often expressed as "nonprotein" energy.

Table 17–7. Estimated Parenteral Protein Requirements

Patient	*Protein Requirement (g/kg/d)*
Extremely low birth weight	Up to 3.5
Very low birth weight	Up to 3.0
Full-term infants	2.5
Ages 2–13 years	1.5–2.0
Adolescents	1.0–1.5

The ratio of protein to nonprotein calories provided in PN is also a useful measure of macronutrient balance. When expressed as the ratio of nonprotein energy (kcal) to nitrogen (g), metabolism is generally optimal when this ratio is between 150:1 and 250:1. Burn patients and others with very high protein requirements may be optimally fed with a ratio of 100:1. The ratio is calculated as follows:

Carbohydrate calories + fat calories : protein intake (g)/6.25

Intravenous lipids are necessary for the provision of essential fatty acids and provide a concentrated, isotonic source of calories. Infusion rates begin at 1 g/kg/d and are advanced by increments of 1 g/kg/d. Lipid intake should generally not exceed 3 g/kg/d or 50 percent of energy intake. Fat emulsions should be used with caution in neonates with hyperbilirubinemia since free fatty acids can displace bilirubin from albumin, increasing the risk of kernicterus. A molar ratio of free fatty acids to serum albumin < 6 has been recommended as safe.[8]

Tolerance to intravenous fats is monitored by serum triglyceride levels, preferably drawn while lipids have not been infused for 4 hours. Although some cases of hypertriglyceridemia are attributable solely to excessive amounts of intravenous lipids, many patients receiving PN will have other reasons to have high blood triglycerides, including acute phase stress response, sepsis, or hepatic dysfunction. Multiple medications are also associated with hypertriglyceridemia (Table 17–9). When hypertriglyceridemia is noted, a reduction in infused lipids (either by reduced hours or by infusing 1 to 3 days per week) is usually indicated. A "fat overload" syndrome has been described with excessive administration of lipids, characterized by focal seizures, fever, hepatosplenomegaly, and thrombocytopenia. To prevent essential fatty acid deficiency, 3 to 5 percent of total energy needs should be met by the provision of intravenous fat.

Table 17–8. Brand-Specific Composition of Common Pediatric Parenteral Amino Acid Solutions

	Solutions Designed for Infants		*Standard Solutions Suitable for Ages 1 Year and Above*				
Product (Manufacturer)	*Aminosyn PF (Abbott)*	*TrophAmine (B. Braun/McGaw)*	*Aminosyn (Abbott)*	*Aminosyn II (Abbott)*	*FreAmine III (B. Braun/McGaw)*	*Novamine (Baxter)*	*Travasol (Baxter)*
Nitrogen mg per 100 mL of 1% solution	152	155	157	153	153	158	165
Amino acids (essential) mg per 100 mL of 1% solution							
Isoleucine	76	82	72	66	69	50	60
Leucine	120	140	94	100	91	69	73
Lysine	68	82	72	105	73	79	58
Methionine	18	34	40	17	53	50	40
Phenylalanine	43	48	44	30	56	69	56
Threonine	51	42	52	40	40	50	42
Tryptophan	18	20	16	20	15	17	18
Valine	67	78	80	50	66	64	58

Amino acids (nonessential) mg per 100 mL of 1% solution							
Alanine	70	54	128	99	71	145	207
Arginine	123	120	98	102	95	98	115
Histidine	31	48	30	30	28	60	48
Proline	81	68	86	72	112	60	68
Serine	50	38	42	53	59	39	50
Taurine	7	2.5	—	—	—	—	—
Tyrosine	4	4.4	4.4	27	—	2.6	4
Glycine	39	36	128	50	140	69	103
Glutamic Acid	62	50	—	74	—	50	—
Aspartic Acid	53	32	—	70	—	29	—
Cysteine	—	< 1.6	—	—	< 2.4	—	—
N-ac-L-tyrosine	—	24	—	—	—	—	—

Micronutrients in Parenteral Nutrition

The importance of vitamins in patients receiving PN has recently been underscored in the United States by a widespread shortage of parenteral multivitamins and by reports of symptomatic thiamine deficiency.[9] Fatal vitamin deficiencies have been reported in patients receiving PN without vitamins in as short a time as a few weeks. Table 17–10 lists the current parenteral vitamin products available in the United States. The pediatric version of multivitamins (M.V.I. Pediatric) is notable for its inclusion of vitamin K, a greater amount of vitamin D, and a lower amount of the B vitamins, as compared to formulations designed for adults. The MVC product contains no Vitamin B_{12}, biotin, or folate.

There is currently no parenteral vitamin preparation designed especially for premature infants, and there is some controversy concerning vitamin requirements for these patients. Recommendations for pediatric parenteral vitamin doses for full-term and preterm infants are shown in Table 17–11.

Trace elements commonly added to PN solutions include zinc, copper, manganese, and chromium. Table 17–12 lists recommendations for trace elements in PN. Due

Table 17–9. Medications Associated with Hypertriglyceridemia

Amiodarone	Interferons
β-Blockers	Isotretinoin
Cholestyramine	Itraconazole
Cyclosporine	L-Asparaginase
Estrogen therapy/oral contraceptives	Protease inhibitors
Fluconazole	Risperidone
Glucocorticoids	Thiazide diuretics

to biliary excretion of copper and manganese, these should be omitted in patients with cholestasis. Selenium, chromium, and molybdenum should be held in cases of renal dysfunction. Addition of selenium and carnitine may be necessary after 30 days of PN and no or minimal enteral intake.

The use of parenteral iron to treat iron deficiency anemia has been controversial due to the discomfort and possibility of sterile abscesses with intramuscular use, the risk of anaphylaxis and hypotension, and the possible effect of encouraging microorganism growth and sepsis.

Table 17–10. Comparison of Parenteral Multivitamin Preparations

Vitamin	*MVI Pediatric*	*MVI-12 (Adult)*	*Cernevit*	*Multi-12*	*MVC*
Manufacturer*	Astra	Astra	Baxter/Clintec	Sabex	APP
Unit dose	5 mL	10 mL	5 mL	10 mL	1 mL
Vitamin					
A (IU)	2300	3300	3500	3300	2000
D (IU)	400	200	220	200	200
E (IU)	7	10	11.2	10	1
K (μg)	200	0	0	0	0
Ascorbic acid (mg)	80	100	125	100	100
Thiamine (mg)	1.2	3	3.52	3	10
Riboflavin (mg)	1.4	3.6	4.14	3.6	2
Niacin (mg)	17	40	46	40	20
Pantothenate (mg)	5	15	17.25	15	5
Pyridoxine (mg)	1	4	4.54	4	3
B_{12} (μg)	1	5	6	5	0
Biotin (μg)	20	60	69	60	0
Folate (μg)	140	400	414	400	0

* For ordering information: Astra 1-800-225-4803; Baxter/Clintec 1-888-229-0001; American Pharmaceutical Partners, Inc. (APP) 1-800-386-1300.

Nonetheless, judicious use of iron dextran is warranted in those patients with iron deficiency (as noted by biochemical assessment of iron status) and for whom the enteral route is contraindicated. The total amount of iron (Fe)

Table 17–11. Recommended Intake Levels for Intravenous Multiple Vitamins

		*Preterm Infants (dose per kg)**	
Vitamin	*Term Infants (dose per day)†*	*Current Suggestion‡*	*Best New Estimate§*
A (IU)	2300	920	1643
D (IU)	400	160	160
E (mg)	7	2.8	2.8
K (μg)	200	80	80
Ascorbic acid (mg)	80	32	25
Thiamine (mg)	1.2	0.48	0.35
Riboflavin (mg)	1.4	0.56	0.15
Niacin (mg)	17	6.8	6.8
Pantothenate (mg)	5	2.0	2.0
Pyridoxine (mg)	1.0	0.4	0.18
B_{12} (μg)	1.0	0.4	0.3
Biotin (μg)	20	8.0	6.0
Folate (μg)	140	56	56

*Maximum dose not to exceed term-infant dose.
†These are all met by MVI Pediatric.
‡These are met by 2 mL/kg/d of the MVI Pediatric product (maximuim 5 mL/d).
§Based on data suggesting a reduced need for water-soluble vitamins and increased need for vitamin A in preterm infants.
Reprinted with permission from Greene HL, Hambidge KM, Schanler R, Tsang RC. Guidelines for the use of vitamins, trace elements, calcium, magnesium, and phosphorus in infants and children receiving total parenteral nutrition: report of the Subcommittee on Pediatric Parenteral Nutrient Requirements from the Committee on Clinical Practice Issues of the American Society for Clinical Nutrition. Am J Clin Nutr 1988;48(5):1324–42.

needed to normalize the hemoglobin level can be estimated according to the following formula:

$$\text{Fe (mg)} = \text{weight (kg)} \times 4.5 \times (13.5 - \text{patient's Hgb [g/dL]})$$

An initial test dose of 0.5 mL (25 mg Fe) (0.25 mL/12.5 mg Fe in infants) should be given on the first day at a rate of < 1 mL/hour to monitor for an anaphylactic response. Epinephrine should be readily available. If tolerated well, daily doses of 25 to 100 mg may be given IV for the few days needed to replenish stores. Long-term, maintenance doses at 1 mg/day may be indicated in some patients and may be added to the PN.

Calcium and Phosphorus

Neonates and infants have high requirements for calcium and phosphorus to maintain adequate bone mineralization. An optimal ratio of calcium-to-phosphorus intake is 1.7:1 (by weight) or 1.3:1 (by molar ratio). In situations of fluid restriction, however, high concentrations of calcium and phosphorus may cause precipitation within the solution. These calcium-phosphorus complexes may cause phlebitis and life-threatening emboli. Calcium gluconate is the preferred salt for use in PN solutions since it dissociates less than chloride salts and thereby remains in solution more readily. Other factors favoring the formation of calcium-phosphorus precipitates include low amino acid content, low dextrose content, high temperature, and high pH. Since solution pH is primarily determined by amino acid concentration, increasing amino acid intake, the use of more acidic amino acid solutions, and/or the addition of L-cysteine are common strategies used to prevent precipitation. Typically, 40 mg of L-cysteine is added per gram of protein. It is also recommended that if a PN solution contains 1 percent or less amino acids, only calcium or phosphorus (not both) be added. Consultation with the phar-

macy is encouraged when solutions of high calcium and phosphorus concentration are desired, and published nomograms are useful for specific compatibility information. A conservative estimate may be obtained by adding the sum of calcium and phosphorus concentrations (mmol/L); if the sum of these numbers exceeds 40, the risk of precipitation is high. It should be noted that these recommendations do not apply to total nutrient admixtures (so-called 3-in-1 solutions) since calcium and phosphorus solubility is lower in these mixtures. It is therefore recommended that neonates not receive 3-in-1 solutions.

Table 17–12. Suggested Intake of Trace Nutrients*

Element	*Weight < 2 kg*	*Weight > 2 kg*	*Comments*
Zinc	3 mg	1 mg	Increase dose with increased intestinal losses
Manganese	50 µg	60 µg	Decrease dose with cholestatic liver disease
Copper	200 µg	200 µg	Decrease dose with cholestatic liver disease
Chromium	1.7 µg	2 µg	Increase dose with intestinal losses, and decrease with renal dysfunction
Iron†	1 mg/d (see text)		Monitor for anaphylaxis with initial infusion
Selenium†	1–3 µg/kg/d max dose 30–40 µg/d		Reduce dose with renal disease (may have increased requirements with increased intestinal losses)
Carnitine†	8–16 mg/kg/d		Patients with primary carnitine deficiency will require higher doses

*Concentration per liter PN.
†May be added after 30 days of NPO status and/or minimal enteral intake.

Medication and Parenteral Nutrition

Patients requiring PN are often on multiple medications, and questions frequently arise concerning whether these medications can be coadministrated with PN. Because of the risks of precipitation and/or infection, coadministration of medications and PN should be avoided whenever possible. Furthermore, medications should not be added to the PN bag itself except by the pharmacy. Medications should be "Y-ed in" with the intravenous set up proximal to a filter. Whenever the PN prescription changes, coadministered medications and potential compatibility problems should be reviewed.

Tables 17–13 and 17–14 list medications generally considered safe for coadministration with PN and lipids, respectively. Tables 17–15 and 17–16 list those medications *incompatible* with PN and lipids. In all cases, consultation with the pharmacy is recommended.

Cycling Parenteral Nutrition Infusions

The provision of one day's worth of PN over fewer than 24 hours has been termed "cycling." Advantages of cycling include allowing the patient to be disconnected from intravenous tubing and pumps, avoiding chronic hyperinsulinemia, and (perhaps) an improved visceral protein status.[10,11] Cyclic PN may also help reduce the chances of developing PN-associated liver disease. The suitable candidate for cycling PN are those patients for whom long-term (> 1 month) of PN is anticipated and whose endocrine, renal, and cardiac functions can tolerate shifts in glucose and fluid delivery. Two to three days of metabolic stability on the solution providing the desired amount of fluid and energy are also required before cycling can begin.

The hourly goal rate for cycled PN is directly related to (1) the time of cycled PN infusion, and (2) the previous

Table 17–13. Medications Compatible with Parenteral Nutrition Solutions

Albumin*
Aldesleukin
Amikacin‖
Aminophylline†
Atracurium
Atropine
Aztreonam
Bumetanide
Cefepime
Cefotaxime
Cefoxitin
Ceftazidime
Ceftriaxone
Cefuroxime
Chloramphenicol
Chlorpromazine
Cimetidine
Clindamycin
Dexamethasone
Digoxin
Diphenhydramine
Dobutamine‖
Doxycycline
Enalaprilat
Epinephrine§
Erythromycin
Famotidine
Fentanyl
Fluconazole
Gentamicin‖
Glycopyrrolate
Granisetron
Heparin
Hydralazine
Hydrocortisone
Hydromorphone
Insulin (U-100 regular)
Iron dextran
Isoproterenol
Leucovorin
Levocarnitine
Lorazepam
Magnesium sulfate
Meperidine
Mesna
Methylprednisolone‡
Mezlocillin
Miconazole
Morphine
Nafcillin
Norepinephrine
Ondansetron
Oxacillin
Pancuronium
Penicillin G+ (aqueous)
Phenobarbital
Phytonadione
Piperacillin
Piperacillin/tazobactam
Promethazine
Pyridoxine
Ranitidine
Tacrolimus
Ticarcillin
Ticarcillin/clavulanic acid
Tobramycin‖
Tolazoline
Vancomycin‖
Vecuronium
Zidovudine

*Will clog filter if albumin concentration > 25 g/L.
†Do not exceed 3 mg/mL for piggyback administration.
‡Contains phosphate buffers which may precipitate in solutions high in calcium or phosphorus.
§Incompatible with iron containing PN solutions.
‖Incompatible with heparin-containing PN solutions.
Adapted from the 1999 IV Drug Administration Guidelines, Children's Hospital, Boston.

hourly rate (when 24 hours of PN were given). For example, PN administered at 40 mL/h for 24 hours provides 960 mL. To provide this over 20 hours, (960 mL/20 h = c 48 mL/h) would presumably be the new rate. However, in order to prevent hyper- or hypoglycemia, ramping up at the begin-

Table 17–14. Medications Compatible with Lipids

Aldesleukin	Diphenhydramine	Isoproterenol
Cefotaxime	Dobutamine	Lidocaine
Cefoxitin	Dopamine	Norepinephrine
Ceftazidime	Erythromycin	Oxacillin
Ceftriaxone	Famotidine	Penicillin
Chloramphenicol	Gentamicin	Ranitidine
Cimetidine	Hydrocortisone	Ticarcillin
Clindamycin	Hydromorphone	Tobramycin
Cyclosporine	Insulin, regular	Vancomycin
Digoxin		

Table 17–15. Medications Incompatible with Parenteral Nutrition Solutions

Acetazolamide	Cytarabine	Metoclopramide
Acyclovir	Diazepam	Metronidazole
Amphotericin	Doxorubicin	Midazolam
Amphotericin B lipid complex	Filgrastim	Nitroglycerin
Ampicillin	Foscarnet	Nitroprusside
Ampicillin/sulbactam	furosemide	Octreotide
Calcium salts	Ganciclovir	Phenytoin
Cefazolin	Imipenem	Promethazine
Ciprofloxacin	Indomethacin	Trimethoprim/sulfamethoxazole
Cis-platinum	Mannitol	Tromethamine
Cyclosporine	Methotrexate	

ning of the infusion and down at the end is recommended; 3 to 5 percent of the total PN volume is used as an adjustment factor for these ramping periods. The rates of PN administration are usually written as 1/2 of the hourly goal rate for 30 minutes while beginning the PN infusion and 1/2 of the hourly rate for 30 minutes, then 1/4 the hourly rate for another 30 minutes while coming off PN. Figure 17–3 illustrates this concept.

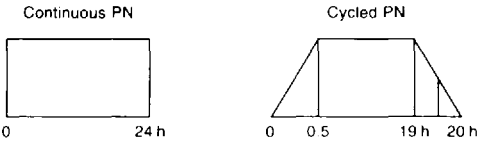
Monitoring and Potential Complications

Monitoring patients' clinical and biochemical responses to the initiation and continuation of PN is a vital part of patient management. In the pediatric patient, an especially important parameter with which to assess the effectiveness of PN is weight gain. In neonates and young children, weight gain and height gain along standard reference curves should be the goal for nutritional therapy, including PN. Serial measurements of head circumference and arm anthropometrics are also useful to monitor. (See Chapter 2, Anthropometric Evaluation.)

Table 17–16. Medications Incompatible with Lipids

Acetazolamide	Cyclosporine	Magnesium salts
Acyclovir	Diazepam	Metronidazole
Amikacin	Doxorubicin	Midazolam
Aminophylline	Filgrastim	Morphine
Amphotericin	Foscarnet	Nitroglycerin
Amphotericin B lipid complex	Furosemide	Nitroprusside
	Ganciclovir	Phenytoin
Ampicillin	Heparin	Trimethoprim/sulfamethoxazole
Ampicillin/sulbactam	Imipenem	
Calcium salts	Indomethacin	Tromethamine
Ciprofloxacin	Iron dextran	

Biochemical monitoring helps insure tolerance to the individual components of PN and helps avoid the myriad metabolic complications of this therapy. Recommended monitoring parameters for inpatients are listed in Table 17–17. Table 17–18 lists multiple metabolic conditions commonly seen among patients receiving PN, as well as suggested therapeutic steps. Finally, Table 17–19 lists common technical or catheter-related complications and recommended approaches for their prevention and treatment.



40 mL/h × 24h = 960 mL
For 20h infusion: 960 mL × 0.97* = 931 mL

20 h on, 4 h off
931 mL ÷ 18.5 h = 50 mL/h
plus ramp up = 25 mL/h × 30 min =
plus ramp off = 25 mL/h × 30 min
+ 12 mL/h × 30 min

Total = 931 + 12.5 + 12.5 + 6 = 962 mL

For 16h infusion: 960 mL × 0.95* = 912 mL

16 h on, 8 h off
912 mL ÷ 13.5 h = 68 mL/h
plus ramp up = 34 mL/h × 30 min =
plus ramp off = 34 mL/h × 30 min
+ 17 mL/h × 30 min

Total = 912 + 17 + 17 + 8 = 954 mL

* Adjustment factor.

Figure 17–3. Cycling parenteral nutrition.

Table 17–17. Suggested Monitoring Schedule for Inpatients Receiving Parenteral Nutrition

Parameter	*Daily*	*Weekly**	*Periodically**
Weight	X		
Fluid balance	X		
Vital signs	X		
Urine sugar/acetone	X		
Catheter site/function	X		
Laboratory test			
Sodium		X	
Potassium		X	
Chloride		X	
CO_2		X	
Glucose		X	
BUN		X	
Creatinine		X	
Triglycerides		X	
Calcium		X	
Magnesium		X	
Phosphorus		X	
Prealbumin		X	
Albumin		X	
Total protein		X	
ALT		X	
Alkaline phosphatase		X	
Bilirubin (total and direct)		X	
Selenium			X
Copper			X
Zinc			X
Iron			X

*More often as necessitated by clinical course.
CO_2 = bicarbonate; BUN = blood urea nitrogen; ALT = alanine aminotransferase.

Table 17–18. Common Metabolic Conditions Seen in Patients Receiving Parenteral Nutrition

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Macronutrient Substrate Complications				
Hyperglycemia	• Diabetes mellitus • Excessive dextrose infusion • Metabolic stress/sepsis • Corticosteroids • Peritoneal dialysis or CAVH-D • Obesity • Chromium deficiency	• Elevated blood glucose (> 200 mg/dL) • Glucosuria > 2%	• Limit initial dextrose infusion to approximately 10–15% • Limit increments in dextrose to 5% per day • Monitor serum glucose • Monitor urine glucose	• Decrease dextrose intake • Add regular insulin to PN or give IV insulin (starting dose 0.01 unit/kg/h)
Hyperglycemic, hyperosmolar, nonketotic dehydration/coma	• Sustained, uncontrolled hyperglycemia	• Very high blood glucose levels • Elevated serum osmolarity • Osmotic diuresis • Metabolic acidosis • Lethargy and confusion • Coma	• Goal: ≤ 200 mg/dL • Monitor: – blood and urine, glucose closely – serum osmolarity – fluid status	• Immediate discontinuation of PN • IV hydration, insulin • Correction of metabolic acidosis

Table 17–18. continued

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Hypoglycemia	• Sudden discontinuation of PN • Exogenous insulin administration • Sepsis	• Blood glucose < 50 mg/dL • Diaphoresis • Lethargy or palpitations • Agitation/irritability • Faintness • Confusion • Coma	• Avoid abrupt cessation of PN • Check blood glucose 1 hour after PN discontinued in cycled patients	• IV dextrose
Hypercapnia (elevated pCO2)	• Excessive dextrose or total caloric intake in patients with chronic lung disease	• Increased pCO_2 • Respiratory distress	• Avoid excessive caloric or dextrose infusion • Obtain indirect calorimetry measurement; adjust PN regimen to meet needs	• Decrease total caloric intake and/or increase calories as fat
Azotemia	• Dehydration • Renal insufficiency • Excessive amino acid infusion • Lean tissue catabolism	• Elevated BUN • Lethargy • Coma	• Adequate hydration prior to PN initiation • Avoid excessive amino acid infusion • Provide adequate	• Free water administration • Decrease amino acid infusion

	• Immature liver • Liver disease • Inborn errors of protein metabolism		nutrition to minimize lean tissue catabolism • Monitor BUN and NH_3	
Abnormal amino acid profile	• Inborn error of metabolism • Liver disease • Composition of PN solution	• Serum amino acid profile out of normal range	• Monitor serum amino acid levels • Avoid excess protein intake in liver disease	• Consider use of special amino acid solution
Hypertriglyceridemia	• Excessive lipid infusion • Decreased clearance (stress/sepsis, liver failure) • Sustained hyperglycemia • Congenital hyperlipidemia • Excessive caloric intake, especially glucose • Medications (see Table 17–9)	• Lipemia • Serum TG > 200 mg/dL	• Avoid excessive lipid infusion • Monitor serum triglycerides weekly • Infuse lipids over 18–20 hours	• Decrease lipid infusion • If sustained, provide only enough lipid to prevent EFAD (0.5–1.0 g/kg/d)

Table 17–18. continued

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Fluid and Electrolyte Disturbances				
Fluid overload	• Excessive fluid administration • Renal dysfunction, congestive heart failure, liver disease, trauma	• Rapid weight gain • Fluid intake > output • Increased blood pressure • Decreased serum sodium and hematocrit • Edema	• Avoid excessive fluid administration • Close monitoring of: – weight – intake/output – physical examination – electrolytes	• Concentrate PN solution • Fluid restriction • Sodium restriction and/or diuretics, if appropriate
Dehydration	• Inadequate fluid intake • Excessive diuresis • Increased GI losses • Fever	• Decreased urine output • Orthostasis • Increased serum sodium, BUN, hematocrit • Poor skin turgor • Thirst • Rapid weight loss	• Provide adequate fluid • Replace insensible and GI losses • Monitor fluid status	• Fluid replacement with separate IV from PN
Hypokalemia	• Inadequate potassium supplementation during anabolism/refeeding	• Metabolic alkalosis • Cardiac arrhythmias • Muscle weakness	• Adequate potassium in PN • Measure and replace	• Increase potassium in PN if mildly to moderately depleted

	• Increased GI losses (vomiting, diarrhea, ostomy) • Medications (eg, furosemide, amphotericin B, cisplatin, etc)	• Ileus	losses • Monitor serum levels daily until stable; biweekly thereafter	• Additional IV supplementation if severely depleted
Hyperkalemia	• Renal insufficiency • Excessive potassium administration • Medications (eg, spironolactone) • Catabolism	• Weakness • Paresthesias • Hyporeflexia • Cardiac arrhythmias	• Avoid excessive potassium administration • Monitor serum levels daily until stable; biweekly thereafter • Monitor serum potassium daily in patients with renal insufficiency; restrict as appropriate	• Decrease potassium in PN
Hyponatremia	• Fluid overload • SIADH • Excessive losses (urinary, GI, or transdermal)	• Irritability • Confusion • Lethargy • Seizures	• Adequate sodium in PN • Avoid excessive fluid administration • Monitor serum sodium daily until stable; biweekly thereafter	• Fluid restriction • Increase sodium in PN if sodium depleted • Replace with separate IV if increased losses

Table 17–18. continued

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Hypernatremia	• Dehydration • Excessive sodium administration • Osmotic diuresis secondary to hyperglycemia • Pituitary tumors	• Thirst • Restlessness • Muscle tremor and rigidity • Hyperactive reflexes • Coma • Convulsions	• Provide adequate fluid • Avoid excessive sodium administration • Monitor intake/output, urine sodium, osmolarity	• Fluid replacement if dehydrated • Decrease sodium in PN if appropriate
Metabolic acidosis	• Increased intestinal losses of bicarbonate (diarrhea, fistulas) • Renal bicarbonate losses • Ketoacidosis (diabetes, starvation) • Lactic acidosis (shock, cardiac arrest) • Chronic renal failure or renal tubular acidosis • Excessive chloride in PN (rare)	• Headache • Nausea/vomiting • Diarrhea • Convulsions	• Measure and replace intestinal losses • Avoid excessive chloride in PN	• Increase acetate and decrease chloride in PN
Metabolic alkalosis	• Gastric acid losses (increased NG output)	• Nausea/vomiting • Diarrhea	• Measure and replace NG output	• Treat underlying cause

	• Excess base administration • Aggressive diuretic therapy • Citrate toxicity due to large volume of blood products	• Sensory changes • Tremors • Convulsions		• Increase chloride and decrease acetate in PN • If severe, may need IV hydrochloric acid
Mineral Imbalances				
Hypocalcemia	• Blood products (citrate chelates with calcium) • Hypoalbuminemia • Hypomagnesemia • Hyperphosphatemia • Hypoparathyroidism • Malabsorption • Inadequate calcium in PN	• Muscular/abdominal cramping • Irritability • Confusion • Tetany • Seizures • Prolonged QT interval	• Adequate calcium in PN • Monitor serum calcium biweekly: check ionized calcium if total calcium decreased • Monitor PTH and vitamin D levels	• Correct magnesium deficiency • Increase calcium in PN if ionized calcium low
Hypercalcemia	• Neoplasm • Renal insufficiency • Excessive vitamin D administration • Bone resorption caused by prolonged immobilization/stress	• Confusion • Lethargy • Dehydration • Muscle weakness • Abdominal pain • Nausea and vomiting • Constipation • Arrhythmias • Extra skeletal calcification	• Monitor serum levels daily until stable; biweekly thereafter • Restrict as appropriate	• Decrease calcium in PN • Hydrate with isotonic saline • May need to remove vitamin D from PN

Table 17–18. continued

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Hypomagnesemia	• Increased GI losses (vomiting, diarrhea, fistula) • Increased urinary losses secondary to drugs (eg, cisplatin, cyclosporine, amphotericin B, aminoglycosides) • Inadequate magnesium supplementation during anabolism/refeeding	• Weakness • Muscle tremors • Ataxia • Tetany • Paresthesias • Dizziness • Disorientation/ irritability • Seizures • Cardiac arrhythmias	• Adequate magnesium in PN • Monitor serum levels daily until stable; biweekly thereafter	• Increase magnesium in PN if mildly to moderately depleted • Additional IV supplementation if severely depleted
Hyper-magnesemia	• Renal insufficiency • Excessive magnesium administration	• Nausea/vomiting • Lethargy/weakness • Cardiac arrhythmias • Hypotension • Respiratory depression	• Monitor serum levels daily until stable; biweekly thereafter • Monitor serum levels daily in patients with renal insufficiency; restrict as appropriate • Avoid excessive magnesium administration	• Decrease magnesium in PN

Hypo-phosphatemia	• Inadequate phosphorus supplementation during anabolism/refeeding • Exogenous insulin administration • Chronic use of phosphate-binding antacids • Alcoholism • Diabetic ketoacidosis	• Serum level < 2 mg/dL • Paresthesias • Confusion • Altered speech • Lethargy • Respiratory failure • Decreased red blood cell function • Coma	• Supplement in PN above standard amounts in patients at risk (diabetes, alcoholism, protein-energy malnutrition) • Monitor serum levels daily until stable; biweekly thereafter	• Increase phosphorous in PN if mildly to moderately depleted • Additional IV supplementation if severely depleted
Hyperphos-phatemia	• Renal insufficiency • Excessive phosphorus administration • PTH deficiency	• Prolonged elevations may lead to tissue calcification	• Monitor serum levels daily until stable; biweekly thereafter • Monitor serum levels daily in patients with renal insufficiency/ restrict as appropriate • Avoid excessive phosphorus administration	• Decrease phosphorus in PN

Table 17–18. continued

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Other				
Refeeding syndrome (see Chapter 15, Nutritional Assessment in Sick or Hospitalized Children)	• Rapid or excessive dextrose infusion (especially in malnourished patients)	• Hyperglycemia • Hypophosphatemia • Hypokalemia • Hypomagnesemia • Edema • Pulmonary edema/CHF	• Identification of patients at risk (chronically - malnourished, nutritionally depleted patients) • Replete serum electrolyte deficiencies prior to PN initiation • Limit initial caloric intake to basal requirements • Supplement phosphorus in PN • Advance PN cautiously • Monitor serum glucose, electrolytes, phosphorus, and magnesium daily until stable; biweekly thereafter	• Decrease infusion rate • Replete serum electrolyte, phosphorus, magnesium deficiencies; monitor closely • Limit fluid in presence of edema
Essential fatty acid deficiency (EFAD)	• Prolonged insufficient lipid infusion	• Dry, scaly skin • Hair loss • Thrombocytopenia	• Provide at least 0.5–1.0 g fat/kg/d (can be given 2–3	• Daily lipid infusion • Cutaneous application of

		• Triene:tetraene ratio > 0.4	times per week)	linoleic acid-rich oils if IV lipid contraindicated
Hepatic dysfunction	• Multiple	• Elevated LFTs and direct bilirubin	• Avoid excess energy intake • Avoid excess protein intake • Rule out infectious, metabolic or anatomic causes of cholestasis	• Reduce or eliminate Cu and Mn • Reduce energy and protein to meet requirements • Cycle PN
Trace mineral deficiencies	• Inadequate supplementation during long-term PN • Excessive losses via GI tract (diarrhea, fistula output)	• Varies depending on specific deficiency	• Adequate supplementation	• Supplement deficient nutrient
Trace Nutrient Deficiencies (see Table 17–12)				
Iron (Fe)	• Long-term NPO status without Fe supplementation • Increased blood loss	• Decreased ferritin • Decreased transferrin saturation • Decreased hemoglobin • Tachypnea/tachycardia • Poor weight gain • Poor feeding	• Monitor serum Fe levels	• Maintenance dose 1 mg/d in PN • Do not give Fe if being transfused • Watch for anaphylaxis with initial infusion

Table 17–18. continued

Complication	*Possible Causes*	*Clinical Findings*	*Prevention / Monitoring*	*Treatment*
Zinc (Zn)	• Increased GI losses • Acrodermatitis enteropathica	• Growth failure • Perineal and perioral lesions • Impaired wound healing	• Increase Zn if chronic GI losses • Monitor serum Zn • Monitor growth • Wound healing	• Increase Zn in PN if increased GI losses
Selenium (Se)	• Increased GI losses • Inadequate supplementation • Long-term NPO status without supplementation	• Cardiomyopathy • Muscle weakness • Reduced glutathione peroxidase activity • Hypopigmentation of hair and nails • Hemolytic anemia	• Provide Se supplementation with long-term NPO status • Monitor serum Se levels • Monitor serum or RBC glutathione peroxidase levels	• Supplement as necessary
Carnitine	• Long-term NPO status without supplementation	• Liver dysfunction • Steatosis • Progressive myopathy	• Monitor serum carnitine levels	• Supplement for long-term NPO if low serum levels

		• Growth failure • Hypertriglyceridemia • Hypoglycemia		
Metabolic bone disease	• Etiology unclear/ possibly multifactorial • Possible etiologies include: – altered vitamin D metabolism – aluminum toxicity – protein induced calcium loss – drug therapy (eg, diuretics) – inactivity – inadequate Ca or PO_4 or vitamin D	• Demineralization • Hypercalciuria • Pathologic fractures • Back pain • Bone pain	• Moderate nutrient provision • Monitor minerals and other PN solutions for presence of aluminum	• Adequate Ca, PO_4, vitamin D • Weight bearing exercise • Change diuretic therapy, if feasible

BUN = blood urea nitrogen; CAVH-D = continuous atrial-venous hemofiltration dialysis; CHF = congestive heart failure; EFAD = essential fatty acid deficiency; GI = gastrointestinal; IV = intravenous; LFT = liver function tests; NG = nasogastric; NH_3 = ammonia; NPO = nil per os; pCO_2 = partial pressure of carbon dioxide; PTH = parathyroid hormone; RBC = red blood cell; SIADH = syndrome of inappropriate antidiuretic hormone; TG = triglyceride; PN = parenteral nutrition.

Table 17–19. Common Mechanical and Central Line-Related Complications in PN Patients

Complication	*Clinical Signs and Symptoms*	*Management*	*Preventive Measure*
Obstruction in the infusion system	• PN does not flow to gravity • Occlusion alarm sounds	• Check that all clamps in the system are open • Check the IV tubing and catheter for kinks If the catheter has clotted, one may attempt to dislodge the clot by careful flushing with 1 mL of normal saline directly attached to catheter hub, using sterile technique. If unsuccesssful, a fibrinolytic agent may be necessary.	• Use a pump that detects obstruction immediately • Careful taping of catheter and dressing to prevent kinking of tubing
Dislodgment of the catheter with subcutaneous collection of PN solution	• Swelling at the insertion site of the catheter	• Removal of the catheter	• Careful handling of catheter • Securing line with tape and/or safety pin to clothing
Venous thrombosis	• Venous distention or edema of the part of the body drained by that vein	• Instillation of a fibrinolytic agent may declot a small thrombus • Attempt to aspirate blood to verify if catheter is patent and intact • Evaluate need for removal	• Use a pump that detects obstructions
Breakage of silastic catheter	• Leakage of PN fluid or blood	• Clamp catheter immediately • Obtain proper size repair kit	• Careful handling of the catheter

		• Consult surgical service to repair the catheter	• Clamping only where indicated on CVC • Avoid forceful flushing
Accidential uncoupling of joints in the infusion system	• Leakage of PN solution or spontaneous blood return • May cause hypoglycemic reaction or infection	• Clamp off system • Clean joints with alcohol before reconnecting	• Use of luer-lock connectors
Air embolus	• Sudden onset of respiratory distress • Cyanosis	• Immediately clamp catheter • Place patient in Trendelenburg's position with right side up	• Use a pump that detects air in the system • Clamping catheter when system is opened
Transient arrhythmias	• Irregular heart beat usually occurring during insertion	• May require repositioning of catheter	• Careful monitoring of heart beat immediately after insertion
Skin sloughings due to infiltration of PN solution	• Swelling at peripheral IV site with discoloration of surrounding skin	• Immediately remove IV • Cover skin slough with sterile dressing and warm soaks • Consider use of hyaluronidase 15 u/mL inject 0.5 mL x 5 doses SQ around site or apply nitroglycerin ointment 2%, 4 mm per kg q 8h	• Hourly observation of IV site for infiltration • Changing peripheral IV sites as needed

CVC = central venous catheter; IV = intravenous; PN = parenteral nutrition; SQ = subcutaneous.

Nursing Care in Parenteral Nutrition Administration

Meticulous nursing care is essential to successful PN administration to avoid serious infectious and metabolic complications. Several important nursing procedures are listed in Table 17–20.

Management of Catheter Occlusions

Obstruction of the intravenous infusion system is one of the most common complications of PN use. Initial management consists of insuring that there are no clamps or kinks. Using sterile technique, a 3-to-10 mL flush with normal saline should then be attempted.

Persistent obstructions are often treated with thrombolytic agents. Venography can help in differentiating among malpositioned catheters, a fibrin sheath at the central venous catheter (CVC) tip, or mural thrombi. Complete occlusions, however, cannot be evaluated by contrast studies. Depending on the nature of an intracatheter precipitate, Table 17–21 lists commonly used agents.

Most CVCs require 1.0 mL of the thrombolytic agent chosen, although inplantable ports require 2 mL. The agent is allowed to dwell within the lumen of the CVC for 30 to 60 minutes before being aspirated with a 10-mL syringe. A normal saline flush should then be attempted before the infusion is resumed.

Occasionally a prolonged infusion of a thrombolytic agent is required. Large thrombi often cannot be treated successfully and are generally an indication for catheter removal.

Weaning from Parenteral Nutrition

The transition from PN to oral intake (PO) can be highly variable. Factors affecting how quickly a patient can be

Table 17–20. Nursing Care in Parenteral Nutrition Administration

Action	*Rationale*
I. PN Administration	
1. Change PN administration sets every 96 hours (every 24 hours if lipid is used), using strict aseptic technique. Solution is to be changed every 24 hours.	Administration sets, solutions, or filters may be a source of contamination.
a) All connections in the PN system must be luer-locked or secured with adhesive tape.	a) Luer-lock connectors reduce the incidence of accidental uncoupling of joints.
b) Prep every connector and entry site with alcohol.	b) Alcohol will remove any traces of solution that might accumulate on connectors during setup.
c) Stopcocks are not to be used; avoid the use of added extension tubing or T-connectors.	c) Extra tubing may be a source of contamination.
d) Keep a smooth-edged clamp at the bedside.	d) If the PN system should become disconnected or break, immediate clamping of the catheter above the break will prevent air embolism or blood loss.
2. A volumetric infusion pump must be used.	A volumetric pump will accurately deliver desired volume as well as signal air and/or obstruction in the system.
3. Cover solution with UV-resistant light-sensitive bag.	Decrease potential risk of nutrient oxidation.

Table 17–20. continued

Action	*Rationale*
II. Prevention of Infection	
1. A single-lumen CVC placed for nutrition is not intended to serve any other purpose (ie, to measure central venous pressures, administer blood products, "piggyback" medications, or obtain blood samples).	Frequent manipulation of the PN system increases the risk of infectious and mechanical complications.
1. The following precautions must be taken before entering the line:	
a) All entry points are to be scrubbed with alcohol.	a) Alcohol is a disinfectant.
b) No medications may be added to the PN solution outside the pharmacy.	b) Medication may precipitate in PN solution. The addition of a medication is also a potential source of contamination.
c) If blood must be withdrawn from any catheter, it must be done by a physician or nurse specifically trained in this procedure.	c) If done improperly, blood withdrawal may cause clotting or infectious complications.
1. Cover the CVC exit site with a sterile dressing, and change at least every 7 days or as needed.	

III. Lipid Administration

1. Lipid may be administered with PN solution, provided:
   a) A separate pump is used.
      a) Use of a pump ensures accurate infusion and prevents backflow of PN into the lipid circuit.
   b) A 1.2-micron filter is used in the system, closest to the patient.
2. Lipid volumes < 60 mL are administered through a syringe pump.
   Lipid must be administered at a steady rate to prevent fat overload.
   a) Change tubing every 24 hours. Solution is to be changed every 12 hours.
      a) Administration sets, solutions, or filters may be a source of contamination.
   b) Coordinate lipid administration setup change with the change in solution to minimize manipulation of the line.
      b) This decreases the risk of infection.

IV. Cyclic PN

1. A luer-lock injection cap is placed on the CVC as follows:
   A luer-lock cap closes off the infusion system and prevents disconnection of the cap. It also provides a site for heparin instillation.
   a) Clamp the CVC.
   b) Disconnect IV tubing from CVC with sterile gauze pads.
   c) Scrub connection site with alcohol.
   d) Attach injection cap.

Table 17–20. continued

Action	*Rationale*
1. Inject 2.0 mL heparin (10 units/mL) into catheter via cap.	Heparin will prevent blood from clotting within the catheter when CVC not in use.
a) Scrub the rubber tip of the cap with alcohol prior to injection.	a) Alcohol will clean the cap to reduce the risk of infection.
b) Inject heparin solution. Clamp the CVC as the final 0.5 mL is administered.	b) This will prevent backflow at the CVC tip.

TPN = total parenteral nutrition; CVC = central venous catheter.

Table 17–21. Treatment of CVC Occlusion

Precipitate Suspected	*Clinical Scenario*	*Thrombolytic Agent*
Particulate (eg, Ca-P) with drugs that are soluble in acidic solution	Ca and/or P; use of etoposide or aminoglycosides	0.1 N hydrochloric acid
Particulate with drugs that are soluble in basic solutions	Phenytoin, imipenem, oxacillin, ticarcillin	Sodium bicarbonate 1 mEq/mL
Waxy	PN/IL	Ethanol 70% in water
None (ie, thrombus suspected)	No precipitate suspected	Alteplase 1 mg/mL

PN = parenteral nutrition; CVC = central venous catheter; IL = intralipid; CA = calcium; P = phosphorus; N = normal (solution).

weaned from PN include age, tolerance to PO advancement, length of time NPO, psychologic factors, and previous medical interventions (eg, prolonged intubation or nasogastric tube placement). For example, a well-nourished, school-aged child who is taking at least 50 percent of calories by mouth and has tolerated advancement from clear to full liquids may wean off PN rapidly. The volume of PN could be decreased by half, lipids could be discontinued, and there could be subsequent discontinuation of PN in 1 to 2 days. Younger patients, patients with questionable tolerance to enteral feedings, and those with a history of feeding difficulties may take weeks to months to wean off PN. One method of weaning in this situation may be to decrease the hours of infusion during the day to encourage increased oral intake. Another method is to decrease the hourly rate of infusion. Attention to detail, such as monitoring blood glucose and fluid status, is important during the weaning process.

Home Parenteral Nutrition

Home parenteral nutrition (HPN) may be an option for children who require long-term PN as, for example, those with short-bowel syndrome. Medical, social, psychologic, and financial factors must all be considered in the decision to use HPN. Home parenteral nutrition is generally delivered on a cyclic schedule, allowing the patient to perform normal daily functions while off PN. The duration of infusion time may vary from 8 to 20 hours, depending on the age, nutritional requirements, medications, and enteral intake of the patient.

Education of the primary caretakers while the patient is in the hospital and then at home is vital to the success of home nutritional therapy and to the reduction of the myriad possible complications. Monitoring of HPN is similar to in-hospital monitoring (see Table 17–17) but is usually less frequent for stable outpatients. It is generally recommended that the following parameters be monitored or reviewed every 4 to 8 weeks: (1) height, weight, head circumference (if applicable), and triceps skinfold measurements; (2) enteral intake (any record should be reviewed); (3) any catheter care issues; and (4) biochemical profiles (see Table 17–17). Laboratory values that need less frequent monitoring include trace elements (zinc, copper, manganese, selenium, iron, and total iron binding capacity) every 6 months and bone density every 6 to 12 months. Monitoring the biochemical and trace-element parameters helps determine necessary changes in PN solution composition. Assessing growth and enteral intake helps determine necessary changes in the amount and composition of the solution.

The length of therapy for HPN depends on the underlying medical reason for initiating nutrition support and can be as short as 1 month or as long as several years. Because of the high rate of infectious and noninfectious complications of prolonged PN in pediatric patients (most notably

chronic liver disease), every effort must be made to wean patients from prolonged PN dependence. Tolerance to enteral nutrition in this setting must be managed through a transition that is carefully monitored by all caretakers.

References

1. Wilmore D, Dudrick S. Growth and development of an infant receiving all nutrients exclusively by vein. JAMA 1968;203: 860–3.
2. O'Brien D, Hodges R, Day A, et al. Recommendations for nutrition support team promote cost containment. J Parenteral Enteral Nutr 1986;10:300–2.
3. Maurer J, Weinbaum F, Turner J, et al. Reducing the inappropriate use of parenteral nutrition in an acute care teaching hospital. J Parenteral Enteral Nutr 1996;20:272–4.
4. Cunningham J. Body composition and nutrition support in pediatrics: what to defend and how soon to begin. Nutr Clin Pract 1995;10:177–82.
5. ASPEN Board of Directors. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. J Parenteral Enteral Nutr 1993;17:1SA–52SA.
6. Sheridan R, Yu Y-M, Prelack K, et al. Maximal parenteral glucose oxidation in hypermetabolic young children: a stable isotope study. J Parenteral Enteral Nutr 1998;22:212–6.
7. Pearlstone DB, Wolf RF, Berman RS, et al. Effect of systemic insulin on protein kinetics in postoperative cancer patients. Ann Surg Oncol 1994;1(4):321–32.
8. Kerner JA Jr, Cassani C, Hurwitz R, Berde CB. Monitoring intravenous fat emulsions in neonates with the fatty acid/serum albumin molar ratio. J Parenteral Enteral Nutr 1981; 5(6):517–8.
9. Centers for Disease Control (US). Lactic acidosis traced to thiamine deficiency related to nationwide shortage of multivitamins for total parenteral nutrition—United States, 1997. MMWR (Morb Mortal Wkly Rep) 1997;46(23).
10. Morimoto T, Tsujinaka T, Ogawa A, et al. Effects of cyclic and continuous parenteral nutrition on albumin gene transcription in rat liver. Am J Clin Nutr 1997;65(4):994–9.
11. Matuchansky C, Messing B, Jeejeebhoy KN, et al. Cyclical parenteral nutrition. Lancet 1992;340(8819):588–92.

18

ACQUIRED IMMUNODEFICIENCY SYNDROME

Roseann Cutroni, MS, RD

Definition and Epidemiology of Pediatric Human Immunodeficiency Virus

Acquired immunodeficiency syndrome (AIDS) is caused by the human immunodeficiency virus (HIV),[1] a type of retrovirus. The latter enters the cell and after replication induces cell dysfunction or death. Cells of the immune system are the most commonly affected. Individuals infected by HIV may exhibit a range of symptoms, from being asymptomatic to very ill. The term "AIDS" refers to those individuals who display specific clinical symptoms as a result of HIV infection.[2] The primary route of transmission of HIV in children is perinatal. Since 1982, 7902 cases of AIDS in children under the age of 13 years have been reported to the Centers for Disease Control. Fortunately, the prognosis of children with AIDS in the United States is improving: in 1991, there were 390 reported deaths in an estimated population of 2125 children with AIDS (18%); in 1996, there were 440 deaths in 3450 children reported living with AIDS (12%).[3]

Complications in children with AIDS include growth failure, weight loss, feeding problems, and multiple nutrient deficiencies. It is often difficult to distinguish whether these are caused by the underlying illness or are, at least in part, a consequence of drug therapies. Antiretroviral

therapy for HIV infection includes three major drug categories: nucleoside reverse transcriptase inhibitors, protease inhibitors, and non-nucleoside reverse transcriptase inhibitors. Studies in adults show that protease inhibitors are associated with increases in abdominal fat deposition and elevated serum triglyceride levels.[4] In addition, numerous other medications are often used for the prophylaxis or treatment of secondary infections. These intensive drug therapies may induce side effects such as nausea, vomiting, and diarrhea that can have an impact on the child's nutritional state. Table 18–1 lists common causes of malnutrition seen in pediatric AIDS.

The immunosuppressive aspects of protein energy malnutrition are well known,[5] and it is likely that HIV infection and malnutrition are additive in their effects.

Table 18–1. Etiology of Malnutrition in Pediatric AIDS

Decreased nutrient intake
Altered taste
Difficulty chewing/swallowing
Oral ulcerations
Medication side effects
Anorexia/depression
Encephalopathy
Malabsorption
Diarrhea/enteropathy
Steatorrhea
Bacterial overgrowth
Opportunistic gastrointestinal infections
Pancreatic insufficiency
Hepatobiliary disease
Increased requirements
Fever
Infection
Catch-up growth
Metabolic abnormalities

Maintaining nutritional status of the HIV-infected child is therefore crucial for optimal immune function.

Nutritional Assessment

Nutritional assessment of the HIV-infected child should be initiated at diagnosis and repeated at least every 6 months with more frequent evaluations in the event of interrupted growth or the onset of clinical symptoms. Nutritional assessment guidelines are outlined in Table 18–2. Human

Table 18–2. Nutritional Assessment of the HIV-Infected Child

Diet history
Feeding history
Dietary intake and analysis
Availability of nutritious food
Safe food handling practices
Medical history
Transmission route
Duration of HIV infection
Drug therapies
Current symptoms
Nausea/vomiting
Diarrhea
Steatorrhea
Lactose intolerance
HIV-associated complications
Physical data
Height
Weight
Head circumference
Arm muscle circumference
Laboratory
Albumin, prealbumin, and transferrin
Selected micronutrient levels*
Lipoprotein and triglyceride levels

*Deficiencies of zinc, selenium, iron, folate, and vitamins A, E, B_6, B_{12}, and C have been reported.

immunodeficiency virus infection can have a significant impact on body composition (with preferential loss of lean body mass), even in the absence of weight loss.[6] Anthropometric measures such as midarm muscle area (see Chapter 1) that quantify lean body mass are therefore of special importance in these patients. Bioelectric impedance equations have also been developed for HIV-infected children.[7]

Nutritional Management

Nutritional management goals are listed in Table 18–3. Energy, protein, and micronutrient requirements for sustaining lean body mass and supporting normal growth and development in the AIDS setting are not well defined. Infectious diseases characteristically increase energy requirements, and HIV infection itself may increase basal metabolic rate.[8] Since weight loss or gain is the ultimate measure of energy needs, calorie requirements should be calculated according to the general guidelines in Chapter 5 with allowance made for energy needs of opportunistic infections or malabsorption.[9] Micronutrient deficiencies may be prevented by providing vitamin/mineral supplements at doses equal to one to two times the Recommended Dietary Allowance.[9-11] Strategies for nutritional management of the symptomatic HIV-infected child are summarized in Table 18–4.

Table 18–3. Goals of Nutritional Management in Pediatric HIV

Preserve lean body mass
Promote normal growth and development
Provide adequate levels of all nutrients
Minimize symptoms of malabsorption

Adapted from Task Force on Nutrition Support in AIDS. Guidelines for nutritional support in AIDS. Nutrition 1989;5:39–46.

Table 18–4. Nutritional Management in Pediatric AIDS

Problem	*Intervention*
Anorexia	Increase nutrient density of foods
	Small frequent feedings
	Nutritional supplements
	Appetite stimulants
	Vitamin/mineral supplements
	Tube feedings
	Parenteral feedings
Oral/esophageal lesions	Soft, nonirritating foods served cold or room temperature
	Topical medications prior to feeding
	Good oral hygiene
Early satiety	Small frequent feedings
	Gastrointestinal motility-enhancing agents
Diarrhea/malabsorption	Small frequent feedings
	Identify and manage lactose intolerance
	Evaluate and remedy food safety issues
	Protein hydrolysate formulas utilizing medium chain triglycerides
	Slow continuous drip tube feeding
	Parenteral feedings
Steatorrhea	Pancreatic enzyme replacements
Infection/pneumonia	Increase calories and protein

References

1. Falloon J, Eddy J, Pizzo P. Human immunodeficiency virus infection in children. J Pediatr 1989;114:1–30.
2. Centers for Disease Control. 1994 revised classification system for human immunodeficiency virus infection in children less than 13 years of age. MMWR CDC Surveill Summ 1994 Sept 30;43:1–9.
3. Centers for Disease Control. HIV/AIDS Surveillance Report. MMWR CDC Surveill Summ 1997 June;9:1–30.
4. Miller KD, Jones E, Yanovski JA, et al. Visceral abdominal fat-accumulation associated with use of indinavir. Lancet 1998;351:871–5.
5. Chandra RK. Mucosal immune responses in malnutrition. Ann N Y Acad Sci 1983;409:345–52.
6. Miller TL, Evans S, Orav EJ, et al. Growth and body composition in children with human immunodeficiency virus-1 infection. Am J Clin Nutr 1993;57:588–92.
7. Arpadi SM, Wang J, Cuff PA, et al. Application of bioimpedance analysis for estimating body composition in prepubertal children infected with human immunodeficiency virus type 1. J Pediatr 1996;129:755–7.
8. Melchior JC, Raguin G, Boulier A, et al. Resting energy expenditure in human immunodeficiency virus-infected patients: comparison between patients with and without secondary infections. Am J Clin Nutr 1993;57:614–9.
9. Coodley GO, Loveless MO, Merrill TM. The HIV wasting syndrome: a review. J Acquir Immune Defic Syndr Hum Retrovirol 1994;7:681–94.
10. Heller LS, Shattuck D. Nutrition support for children with HIV/AIDS. J Am Diet Assoc 1997;97:473–4.
11. Galvin T. Micronutrients: implications in human immunodeficiency virus disease. Top Clin Nutr 1992;7:63–73.

19

BURNS, TRAUMA, AND CRITICAL CARE

Alice O'Leary, MD,
and Christopher Duggan, MD, MPH

Trauma is the leading cause of death in children older than 1 year in industrialized countries, with approximately 22,000 deaths annually in the United States. Motor vehicle accidents account for the highest percentage of trauma deaths.[1] Two million people in the United States suffer burn trauma each year with 100,000 of these hospitalized. Children under 15 years of age comprise 30 to 40% of patients hospitalized with burn injuries.[2] Nutritional support in the trauma and/or burn patient is based on the premise that sparing the mobilization of body reserves is advantageous to recovery.[3] The provision of proper substrates to meet metabolic requirements should curtail catabolism, promote wound healing, and protect from infection.[4]

Metabolism

The body's response to trauma was first described by Cuthbertson as consisting of two phases, an initial ebb phase and a subsequent flow phase.[5] The ebb phase is associated with a decrease in metabolic rate and is equivalent in modern terminology to shock. The duration of the ebb phase reflects the magnitude of the injury and the time needed to restore circulating volume. The flow phase, which was further subdivided by Moore into two components, refers to the period of hypermetabolism (catabolism) followed by restoration (anabolism). This cascade of

physiologic responses to serious injury is modulated by neural, endocrine, and humoral mediators.

Ebb Phase: Fluid Requirements

Management during the ebb phase is centered on maintaining adequate cardiac output to prevent compromise to organ and tissue circulation. Fluid resuscitation is emphasized and little nutrition is provided until the patient has been stabilized. Most patients are stabilized within 48 to 72 hours postinjury, after which time fluid and nutritional requirements can be delivered either enterally or parenterally.

Formulas for calculating the fluid and electrolyte needs of pediatric burn victims during the resuscitation period vary in the total volume, rate of infusion, and composition of the solutions recommended (Table 19–1). The volume of fluid required by the pediatric burn patient depends on the percentage of the body surface area (BSA) burned and the depth of the tissue damage.[6] Some authors recommend the Modified Brooke formula (3 mL/kg/% BSA burned) for burns involving 25 to 35% of BSA, and the Parkland formula (4 mL/kg/% BSA burned) for burns involving more than 35% of BSA.[6] Others note that these formulas may not provide adequate fluid for basal needs,[7] although the Galveston formula explicitly includes maintenance needs. The use of body surface area instead of weight to estimate the volume required is particularly important in children as rates of heat exchange and insensible water losses relative to size and weight are considerably greater in children than in adults (Figure 19–1).[8] Whichever formula is chosen, it should be used only to *estimate* fluid needs and should not supplant close clinical and laboratory evaluation in assessing the adequacy of fluid replacement.

Isotonic electrolyte solutions such as lactated Ringer's have been fairly well established as the initial resuscitation fluid. Infants <12 months of age require less sodium because

Table 19–1. Fluid Resuscitation Formulas for Pediatric Burn Patients

	Modified Brooke	*Parkland*	*Galveston*
Day 1			
Colloid	None	None	After first 8 h postinjury 12.5 g of human albumin per liter of crystalloid
Crystalloid	LR 3 mL/kg/% of BSA burned	LR 4 mL/kg/% of BSA burned	LR 5000 mL/m^2 for burned area plus 2000 mL/m^2 BSA for maintenance
5% dextrose	None	None	None
Calculation of volume	Use total burn area for all burns involving > 25–35% of BSA	Use total burn area for all burns involving 35% of BSA	Use total burn area for all burns involving > 20% of BSA
Rate	1/2 total in first 8 h 1/4 total in next 8 h 1/4 total in next 8 h	1/2 total in first 8 h 1/4 total in next 8 h 1/4 total in next 8 h	1/2 total in first 8 h 1/4 total in next 8 h 1/4 total in next 8 h
Urine	1 mL/kg/h	1 mL/kg/h	1 mL/kg/h
Day 2			
Colloid	As needed to maintain Alb> 2	As needed to maintain Alb> 2	As needed
Crystalloid	None	None	None
5% dextrose	D5 1/2 NS as needed to maintain urine output	D5 1/2 NS as needed to maintain urine output	D5 1/3 NS with 10–20 meq/L Kphos 3750 mL/m^2 for burned surface area plus 1500 mL/m^2 for BSA maintenance

Calculation of volume	Generally 50–75% of first 24 h	Generally 50–75% of first 24 h	75% of first 24 h
Rate	Constant	Constant	Constant
Urine	1 mL/kg/h	1 mL/kg/h	1 mL/kg/h

BSA = body surface area; LR = lactated Ringer's; NS = normal saline; Alb = albumin (g/dL).

of immature renal function. Solutions containing 70 to 80 meq/L of sodium should be considered for this age group.[2]

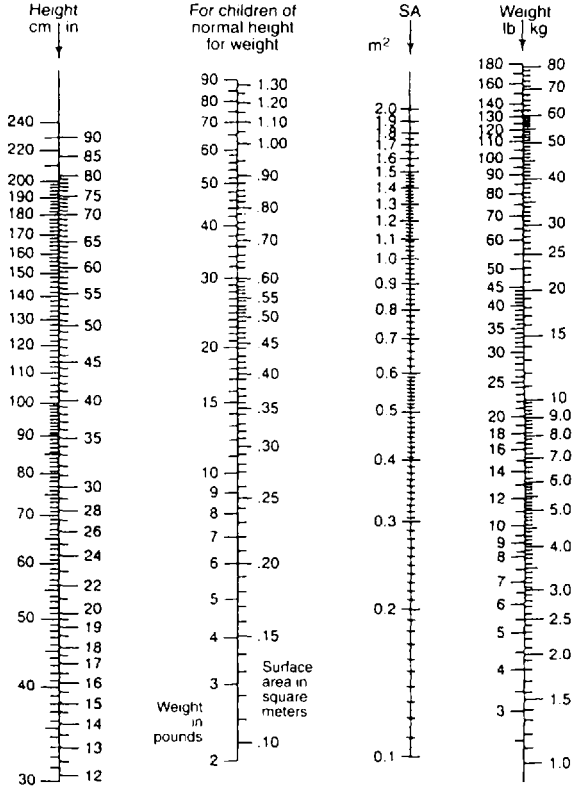


Figure 19–1 Body surface area nomogram. Reproduced with permission from Barone M. The Harriet Lane handbook. St Louis: Mosby-Year Book; 1996. Nomogram adapted by West CD, from data of Boyd E. Mosteller's equation used with permission from Mosteller RD. Simplified calculation of body surface area. N Engl J Med 1987;317: 1098.

Controversy still exists over when colloid solutions should be initiated. Studies have shown that most protein loss occurs within the first 6 to 8 hours after burn injury, indicating that colloid treatment is most effective if given within 8 to 24 hours after burn injury.[2] Albumin requirements for burned patients for the first 24 hours can be grossly estimated as 75 g + 12 g per square meter of body surface burned.[8] This can be given as 12.5 g human albumin per liter of crystalloid to achieve desired serum albumin levels.[9] Maintaining serum albumin levels in the normal range will improve intravascular oncotic pressure and reduce edema.

During the initial 24 hours following a burn, half of the total 24-hour fluid volume should be administered over the first 8 hours and the remainder over the next 16 hours. Fluid requirements in patients with burn injury decrease by 25 to 50% after the first 24 hours and remain constant for as long as the wound remains open.[2] Fluid should therefore be adjusted accordingly and infused at a constant rate to maintain adequate cardiac and urine output.

Flow Phase: Nutritional Requirements

The flow phase in the burn patient is similar in many respects to that of other pediatric ICU patients. Once the patient has been stabilized, the clinician must confront the challenge of meeting nutritional goals under adverse conditions. There are many potential hurdles to providing adequate nutrition, including limited intravenous access, fluid restriction, and hypermetabolism resulting in glucose and lipid intolerance. Precise caloric requirements are difficult to determine in the ICU and little data exist concerning disease-specific needs. Individual subjects can respond to similar injury states with widely diverse measured energy expenditure values.[10] In addition, medication, mechanical support, environmental temperature, fever, pain, and anxiety can all influence metabolic

demands. See Chapter 15 for methods of estimating and measuring energy needs in hospitalized pediatric patients.

In children with burns > 30% BSA, energy requirements are best calculated using the Galveston formula (Table 19–2).[11] Smaller burns (< 30% BSA) do not produce significant hypermetabolic responses and generally do not require specialized nutritional support.[12]

The need for nutritional support in critically ill patients depends on a complex relationship between intake, disease, and substrate stores. It is important to avoid both underfeeding and overfeeding. Malnutrition is associated with several complications, including depressed immune function and increased susceptibility to infection, poor wound healing secondary to insufficient protein intake, loss of muscle mass resulting in alteration of respiratory function and, when severe, multiorgan dysfunction.[13] The deleterious effects of overfeeding include hepatic dysfunction, respiratory compromise, and an increased risk of morbidity and mortality.[14] Regardless of the method used to determine caloric requirements, by achieving positive nitrogen balance and maintaining body weight within 5% of preadmission levels, nutritional goals may be met and complications of under- and overfeeding avoided.[2]

Table 19–2. Galveston Formula for Daily Caloric Determination in Pediatric Burn Patients

Infants (< 1 yr)	2100 kcal/m^2 BSA plus 1000 kcal/m^2 of burn surface area
Older children (1–12 yr)	1800 kcal/m^2 BSA plus 1300 kcal/m^2 burn surface area
Adolescents (> 12 yr)	1500 kcal/m^2 BSA plus 1500 kcal/m^2 burn surface area

BSA = body surface area.

Adapted from Herndon D, Rutan R, Rutan T. Management of the pediatric patient with burns. J Burn Care Rehabil 1993;14:4.

The type and amount of energy substrate provided are very important, especially during the period of hypermetabolism. Adequate amounts of carbohydrates, protein, and lipids should be given to minimize protein catabolism and promote tissue repair and growth. The individual macronutrients and the more important micronutrients in pediatric critical illness are discussed below. Table 19–3 reviews the effect of critical illness on energy and macronutrient needs.

Carbohydrates. Limited glucose availability triggers mobilization of fat stores and nitrogen wasting. Delivery of exogenous carbohydrate is required to prevent protein from being used for gluconeogenesis in meeting energy demands. Forty to sixty percent of energy requirements should come from carbohydrates. Glucose infusions of 5 to 7 mg/kg/min are usually well tolerated since glucose oxidation occurs at approximately 5 mg/kg/min. This rate of oxidation may be exceeded in hypermetabolic states.[4] Glucose infusion should be closely monitored since hyperglycemia and glucose intolerance are prevalent in the catabolic patient. Hyperglycemia is best treated by either decreasing the glucose infusion rate or adding exogenous insulin. Excess carbohydrate supplementation may increase CO_2 production and cause hyperosmolarity and osmotic diuresis.[15]

Protein. The goal of supplemental protein is to provide substrate for cellular protein synthesis and maintenance of lean body mass. Due to an increase in both catabolism and anabolism, protein needs may increase as much as 300% in a metabolically stressed patients.[4] Protein losses occur through the skin and feces but primarily through the urine. Urinary nitrogen excretion measurements are helpful in estimating protein requirements (see Chapter 4, Laboratory Assessment of Nutritional Status).

Lipids. Lipid supplementation is calorically dense, provides essential fatty acids, and promotes protein sparing. Preventing fatty acid deficiency in children requires

Table 19–3. Energy and Macronutrients in the Pediatric Intensive Care Unit Patient

	Energy	*Carbohydrate*	*Protein*	*Fat*
Intake goal	1.1–2.0 X BMR, depending on clinical states	50% of energy needs 5–7 mg/kg/min	15–20% of energy needs 1–1.5 X USRDA	40% of energy needs
Effect of deficiency	Protein energy malnutrition	Nitrogen wasting, mobilization of fat stores	Negative nitrogen balance, hypoalbuminemia, edema	Essential fatty acid deficiency
Effect of over supply	Hepatic steatosis, respiratory compromise, increased CO_2 production, obesity	Hepatic steatosis, hyperosmolarity, osmotic diuresis, increased CO_2 production	Prerenal azotemia, uremia	Elevated triglyceride and FFA levels, hepatic and RE system lipid deposition
Effect of critical illness	Variable; may be increased (eg, burns, sepsis) or decreased	Variable; early-on hyperglycemia secondary to stress response, glucose intolerance	Increased catabolism, espcially within the first 24–48 h after injury, resulting in breakdown in lean body mass	Altered lipid metabolism—possible impaired lipid oxidation, elevated FFA and triglyceride levels

RE = reticuloendothelial; FFA = free fatty acids; BMR = basal metabolic rate; USRDA = United States Recommended Dietary Allowance.

that 3 to 5% of energy intake be fat. Lipids commonly account for up to 40% of total calories in parenteral nutrition. Above a minimal carbohydrate load, lipids supplied as the primary energy source are at least as, if not more, nitrogen-sparing than glucose.[16,17] The oxidation of lipids during catabolism may be impaired due to decreased lipoprotein lipase activity.[18] As with carbohydrate infusions in the metabolically stressed patient, lipid infusions should be closely monitored because of the associated increased levels of triglycerides and free fatty acids.[18]

Micronutrients. Vitamin catalysts and trace element cofactors must be present to drive the metabolic machinery and achieve the desired anabolic effect.[19] The electrolytes calcium, magnesium, and phosphorus are used in increased amounts during tissue anabolism, and growing children have additional needs for calcium and phosphorus to support skeletal growth.[19] The water-soluble vitamins (B, C, folate) are not stored in appreciable amounts and may become rapidly depleted. Monitoring blood levels of these micronutrients may be required if intake is limited by critical illness.

Immunonutrition. Recent research has focused on the effects certain individual nutrients have on the immune system. Arginine, glutamine, ribonucleic acid, and omega-3 fatty acids have all been shown to influence one or more components of the immune system. Critically ill adults given early enteral immunonutrition, including arginine, purine nucleotides, and omega-3 fatty acids had a reduction in morbidity of their critical illness.[20] Further research in this area needs to be performed to substantiate these findings, especially in children.

Enteral Nutrition

Enteral nutrition (EN) is the preferred route of feeding in the ICU patient whenever possible. Unfortunately, oral

and gastric feeds are often poorly tolerated in critically ill patients because of diminished gastric motility related to underlying disease and to the use of sedatives and neuromuscular blocking agents.[21] Transpyloric enteral feeding is a good alternative to gastric feeding, with a proven low incidence of pulmonary infection and hepatic dysfunction.[21] The main complications of EN in the ICU patient are gastrointestinal (abdominal distention, diarrhea, excessive gastric residuals), electrolyte disturbances, pulmonary aspiration and infection, and technical complications related to feeding tube placement (see Chapter 16, Enteral Nutrition).[21]

Parenteral Nutrition

Parenteral nutrition should be considered if a child's medical condition, either for safety reasons or due to a poorly functioning gastrointestinal tract, precludes the use enteral nutrition (see Chapter 17, Parenteral Nutrition).

Conclusion

Nutritional support in pediatric trauma and burn patients plays a direct role in their recovery and final outcome. Appropriate delivery of fluids and nutrients must be achieved with careful and frequent monitoring of clinical and laboratory parameters.

References

1. Pollack E. Pediatric abdominal surgical emergencies. Pediatr Ann 1996;25:448–57.
2. Herndon D, Rutan R, Rutan T. Management of the pediatric patient with burns. J Burn Care Rehabil 1993;14:3–8.
3. Cunningham J. Body composition and nutrition support in pediatrics: what to defend and how soon to begin. Nutr Clin Pract 1995;10:177–82.
4. Schears G, Deutschman C. Common nutritional issues in pediatric and adult critical care medicine. Crit Care Clin 1997;13:669–90.

5. Deutschman C. Nutrition and metabolism in the critically ill child. In: Rodgers MC, editor. Textbook of pediatric intensive care. Vol. II. Baltimore: Williams and Wilkins; 1992. p. 1109–31.
6. O'Neill J. Fluid resuscitation in the burned child—a reappraisal. J Pediatr Surg 1982;17:604–7.
7. Merrell S, Saffle J, Sullivan J, et al. Fluid resuscitation in thermally injured children. Am J Surg 1986;152:664–9.
8. Carvajal H. A physiologic approach to fluid therapy in severely burned children. Surg Gynecol Obstet 1980;150: 379–84.
9. Herrin J, Antoon A. Pediatric critical care. In: Nelson W, editor. Textbook of pediatrics. Philadelphia: W.B. Saunders Company; 1986. p. 273.
10. Chwals W. The metabolic response to surgery in neonates. Curr Opin Pediatr 1994;6:334–40.
11. Schiller W. Burn management in children. Pediatr Ann 1996;25:434–8.
12. Cunningham J, Hegerty M, Meara P, Burke J. Measured and predicted calorie requirements of adults during recovery from severe trauma. Am J Clin Nutr 1989;49:404–8.
13. Coss-Bu J, Jefferson L, Walding D, et al. Resting energy expenditure in children in a pediatric intensive care unit: comparison of Harris-Benedict and Talbot predictions with indirect calorimetry values. Am J Clin Nutr 1998;67:74–80.
14. Vo NM, Waycaster M, Acuff RV, et al. Effects of postoperative carbohydrate overfeeding. Am Surg 1987;53:632–5.
15. Askanazi J, Rosenbaum SH, Hyman AL, et al. Respiratory changes induced by the large glucose loads of total parenteral nutrition. JAMA 1980;243:1444–7.
16. Bark S, Holm I, et al. Nitrogen-sparing effect of fat emulsion compared with glucose in the postoperative period. Acta Chir Scand 1976;142:423–7.
17. Bresson JL, Bader B, Rocchiccioli F, et al. Protein-metabolism kinetics and energy-substrate utilization in infants fed parenteral solutions with different glucose-fat ratios. Am J Clin Nutr 1991;54:370–6.
18. Robin AP, Askanazi J, Greenwood MRC, et al. Lipoprotein lipase activity in surgical patients: influence of trauma and infection. Surgery 1981;90:401–8.

19. Wesley J, Coran A. Nutritional support in pediatric trauma. In: Coran A, Harris B, editors. Pediatric Trauma: Proceedings of the Third National Conference. Philadelphia: J.B. Lippincott Company; 1990. p. 58–72.
20. Atkinson S, Sieffert E, Bihari D. A prospective, randomized, double-blind, controlled clinical trial of enteral immunonutrition in the critically ill. Crit Care Med 1998;26:1164–72.
21. Panadero E, Lopez-Herce J, Caro L, et al. Transpyloric enteral feeding in critically ill children. J Pediatr Gastroenterol Nutr 1998;26:43–8.

20

CARDIAC DISEASE

Deanne K. Kelleher, RD

The term congenital heart disease (CHD) refers to a heterogeneous group of malformations of the heart and/or central vessels present at birth.[1] The prevalence of all types of CHD is reported to be 4 to 6 cases per 1000 live births. Table 20–1 classifies CHD lesions based on the presence or absence of cyanosis; such classification is particularly helpful in determining a patient's expected growth patterns.

Nutrition Risk Factors in Congenital Heart Disease

The most profound nutrition issue in patients with CHD is growth failure. Malnutrition among these patients is thought to be multifactorial; common etiologies are outlined in Table 20–2.[2–5]

Table 20–1. Common Congenital Heart Disease Lesions

Acyanotic	*Cyanotic*
Atrial septal defect (ASD)	Transposition of the great arteries (TGA)
Ventricular septal defect (VSD)	Tetralogy of Fallot (TOF)
Patent ductus arteriosus (PDA)	Tricuspid atresia
Common A-V canal (CAVC)	Truncus arteriosis
Pulmonary stenosis	Total anomalous pulmonary vein return (TAPVR)
Coarctation of the aorta	Pulmonary atresia
	Ebstein's anomaly
	Hypoplastic left heart syndrome (HLHS)

Growth patterns in children with CHD are greatly affected by the type of lesion. Children with cyanotic lesions show retarded growth in weight and length while those with acyanotic lesions have their weight affected more than their length.[3,4] Awareness of the patient's potential growth pattern is important in assessing the child's growth and also in coun-

Table 20–2. Factors Contributing to Growth Failure in Patients with Congenital Heart Disease

Etiologies	*Comments*
Increased energy requirements	Tachypnea and tachycardia can significantly increase metabolic demands
Increased basal metabolic rate	
Increased total energy expenditure	
Increased demand of cardiac/respiratory muscle	
Infections	
Prematurity	
Decreased energy intake	
Anorexia	Common among chronically intubated infants
Dysphagia	
Gastroesophageal reflux	
Increased nutrient losses	
Gastrointestinal malabsorption	
Hyperosmolar formulas	
Anoxia and venous congestion of bowel/liver	Especially with right heart failure
Protein-losing enteropathy	Common after Fontan procedure
Renal electrolyte losses	With diuretic use
Insufficient utilization of nutrients	
Acidosis	
Hypoxia	
Increased pulmonary pressures	
Congestive heart failure	
Decrease in cardiac output and renal blood flow	
Stress response	
Decrease in gastric capacity	Leading to decreased volume of feeds

selling families. Delay in skeletal maturation varies in relation to the severity of the hypoxemia associated with cyanotic defects and is commonly observed in infants with cyanotic CHD.

Corrective or palliative surgery is available for many of these lesions. Correction of the hemodynamic abnormality typically results in the acceleration of growth rate with return to normal parameters. Children requiring staged repairs (ie, those with hypoplastic left heart syndrome [HLHS]) will often continue to be at risk for growth failure in the intermediate time between surgeries.

Patients who undergo the Fontan procedure are at risk for developing protein-losing enteropathy (PLE), the loss of protein and other nutrients from the gastrointestinal tract. Common etiologies include mucosal inflammation, enteric infection, and, in the cardiac patient, interrupted venous or lymphatic flow. In addition to albumin, these patients may lose transferrin, ceruloplasmin, fibrinogen, lipoproteins, alpha$_1$-antitrypsin, fat, minerals, calcium, and iron. Protein-losing enteropathy can present with edema, ascites, hypoproteinemia and/or lymphopenia. Elevated stool alpha-1-antitrypsin can confirm the diagnosis. Management includes providing a diet high in protein and low in long chain fats. Supplementing the diet with a medium chain triglyceride (MCT) containing formula and/or oil can be used with varying results. Medium chain triglyceride oil is used because of the presence of malabsorption and the mechanism of absorption directly via the portal vein. Patients may require additional calcium and fat soluble vitamins when malabsorption is present.[6]

Special Aspects of Nutritional Assessment

Many factors need to be examined for a thorough assessment of patients with CHD. Table 20–3 outlines the uniqueness of this assessment. Since many patients with

Table 20–3. Nutritional Assessment in Congenital Heart Disease

History	Type of lesion (cyanotic vs. acyanotic)	Ability to feed by mouth
	Age of diagnosis	Length of feeding
	Current medications (see Table 20–4 for common medications used)	Diaphoresis during feedings
Physical examination	Clubbing	Cyanosis/pallor of skin
	Fluid status/edema	Respiratory rate (oral feedings may be poorly tolerated with severe tachypnea)
	Oxygen saturation	Ability to coordinate suck, swallow, and breath
Laboratory	Serum electrolytes, Ca, Mg, P, albumin	Urine Na, K, Ca with diuretic use
	Calcium/ionized calcium in DiGeorge syndrome	

cardiac disease are on multiple medications, a medication history should also be elicited (Table 20–4).

Special Aspects of Nutritional Management

As evidenced by the frequent finding of growth failure, children with cardiac disease require additional calories beyond the Recommended Dietary Allowance (RDA) to establish growth. Energy needs vary throughout this population. For children having corrective surgeries, increased energy needs are usually present only before surgery whereas those undergoing palliative surgeries will have prolonged increased requirements. Energy needs may be roughly estimated by adding the RDA for age to 30 to 60 kcal/kg/d,[7] with energy needs titrated to growth patterns.[3]

Table 20–4. Common Medications Used in Congenital Heart Disease

Medication	*Drug-Nutrient Interaction*
Furosemide	Anorexia, nausea, decreased serum K, Na, Cl
Captopril	Decreased serum Zn; increased serum K
Digoxin	Nausea, feeding intolerance, diarrhea, decrease in serum K
Chlorothiazide	Anorexia, decreased K, Zn, Mg, riboflavin
Propranolol	Hypoglycemia

Determination of energy needs should be multifactorial and take into consideration the relative growth failure, CHD lesion, clinical condition, and presence of malabsorption. Many children are unable to tolerate the volume required to consume adequate calories at a standard dilution of formula, necessitating use of a hypercaloric formula.

There are certain cases when initial enteral feedings should be avoided with the CHD population (Table 20–5).

Table 20–5. Contraindications to Enteral Feedings in Cardiac Patients

Hemodynamic instability with low cardiac output, requiring increasing doses of vasoactive drugs
A PDA-dependent lesion with compromised mesenteric perfusion from ongoing left-sided or right-sided outflow obstruction (ie, interrupted aortic arch, HLHS, some coarctations of the aorta, some single ventricle physiology lesions)
Low systemic output for large left-to-right shunt without obstruction
Recent (< 24 hours) cardiac arrest requiring significant resuscitation
Endotracheal intubation or extubation within 4 hours
Functional or mechanical bowel obstruction
Active upper GI bleeding
Junctional ectopic tachycardia

Parenteral nutrition may be indicated, depending on the anticipated length of time these factors will be in play.

Infancy is a critical time in the development of feeding skills. Many infants with CHD suffer interruptions in development of their feeding skills due to underlying disease, surgery, and/or prolonged intubation. These may combine to limit their ability to consume adequate volumes of breastmilk and/or formula. Increased energy needs and decreased ability to take adequate volumes of oral feedings often necessitate the use of hypercaloric breastmilk/formula (see Chapter 16) and nasograstric, nasojejunal, or percutaneous gastrostomy tubes. Use of short-term home nasogastric tube feedings or gastrostomy tube feedings may be required to ensure adequate macro/micronutrient intakes in children with an inability to exclusively orally feed.

The ultimate goal for this patient population, like others, is achievement of normal growth and development. Since these patients often take lower total volumes, micronutrient intakes need to be evaluated and monitored closely and supplemented as needed. Individual mineral supplements may be needed in addition to multivitamins. Close attention should be paid to potassium, chloride, and magnesium as depletion of these can lead to growth retardation. Successful growth can be achieved by monitoring intake and attainment of volume of macro/micronutrients required to meet the patient's continued needs.

References

1. Gillette PC. The cardiovascular system. In: Behrman RE, Kliegaman RM, editors. Nelson essentials of pediatrics. 2nd ed. Philadelphia: W.B. Saunders Company; 1994.
2. Forchielli ML, McColl R, Walker WA, Lo CL. Children with congenital heart disease: a nutrition challenge. Nutr Rev 1994;52(10):348–53.

3. Gaedeke Norris MK, Hill CS. Nutritional issues in infants and children with congenital heart disease. Crit Care Nursing Clin North Am 1994;6(1):153–63.
4. Schwarz SM, Gewitz MH, See CC, et al. Enteral nutrition in infants with congenital heart disease and growth failure. Pediatrics 1990;86(3):368–72.
5. Hansen SR, Dorup I. Energy and nutrient intakes in congenital heart disease. Acta Paediatr 1993;82:166–72.
6. Durie PR. Protein-losing enteropathy. In: Lebenthal E, editor. Textbook of gastroenterology and nutrition in infancy. 2nd ed. New York: Raven Press, Ltd; 1989. p. 1275–80.
7. Kreiger I. Growth failure and congenital heart disease: energy and nitrogen balance in infants. Am J Dis Child 1970; 120:497.

Additional Resources

Internet Resources

www.americanheart.org
www.childrensheart.org
www.csun.edu/~hcmth011/heart/
www.pediheart.org
www.tch.harvard.edu/cardiovascular/index.html
www.tchin.org

21

CYSTIC FIBROSIS

Kattia M. Corrales, RD

Cystic fibrosis (CF) is an autosomal recessively inherited genetic disorder. It is caused by mutations in the gene that encodes for the CF transmembrane conductance regulator protein (CFTR). The CFTR protein is responsible for chloride ion exchange; a defect in this protein results in the production of abnormally thick mucus throughout the body. This mucus can clog tubules and airways (eg, bronchioles and pancreatic ducts) as well as function as a medium for bacterial growth.

Major clinical manifestations of CF include:

- Malnutrition
- Chronic pulmonary infections resulting in progressive lung failure
- Exocrine pancreatic insufficiency
- Meconium ileus
- Cholestatic liver disease
- CF-related diabetes mellitus
- Distal intestinal obstruction syndrome (DIOS), also known as meconium ileus equivalent

Epidemiology

Cystic fibrosis is the most common genetic disorder among Caucasians. It occurs in approximately 1 in 2500 live births among Caucasians, 1 in every 17,000 births among African-Americans, and is rare in Asian populations. There are approximately 30,000 people in the United States with cystic fibrosis.[1]

Nutritional Assessment

Malnutrition is a common clinical manifestation in CF, with about 20% of children in the 1998 national CF patient registry below the fifth percentile for height or weight for age.[2] Several factors are involved in the development of malnutrition in the patient with CF (Table 21–1). Improved nutritional status may slow the progression of pulmonary disease and improve long-term survival.[3,4] The nutritional assessment of the patient with CF involves a thorough review of medical history, nutrient intake, medications, laboratory values, and psychosocial factors (Table 21–2).

Nutritional Management

Nutritional counseling and education should occur at the time of diagnosis and regularly thereafter. Infants and children under the age of 2 years presenting with growth failure should be evaluated weekly or every other week until normal weight gain is achieved, then every 2 to 3 months. Patients should be seen at least once a year by a registered dietitian, who can make accurate anthropo-

Table 21–1. Nutritional Risk Factors in Cystic Fibrosis

Increased resting energy expenditure
Chronic cough
Pulmonary infections
Poor and deteriorating lung function
Possibly a genotype-dependent, energy-requiring cellular defect
Increased nutrient losses
Pancreatic insufficiency
Reduced bile acid and bile salt pool
Cough-emesis cycle
Poorly controlled blood sugars in CF-related diabetes mellitus
Poor energy intake
Fatigue
Anorexia
Esophagitis from gastroesophageal reflux
Depression

metric measurements (including arm anthropometrics), analyze dietary intake, help evaluate adequacy of pancreatic enzyme therapy, and make dietary recommendations.

Table 21–2. Special Aspects of Nutritional Assessment in Cystic Fibrosis

- Medical History
  - *Pulmonary*
    - Number of pulmonary exacerbations
    - Change in pulmonary function tests
  - *Gastrointestinal*
    - History of gastrointestinal disease, meconium ileus, DIOS, intussuception, or gastrointestinal surgery
    - Symptoms of malabsorption, eg, gas and bloating, frequent, bulky, loose stools, floating, fatty, foul-smelling, frothy stools
    - Abdominal pain, vomiting, gastroesophageal reflux
  - *Endocrine*
    - Polyuria, polydipsia, steroid use, history of abnormal blood sugars
  - *Liver disease*
    - History of biliary cirrhosis, ascites, or esophageal varices
- Anthropometrics
  - Weight, height, head circumference, midarm circumference, and triceps skinfold thickness, measured every 3 to 6 months
- Diet History
  - Total calorie and protein intake
  - Percent of total calories from fat and/or grams of fat per meal
  - Food allergies or intolerances
  - Appetite changes with illness
  - Use of nutritional supplements or tube feeding
  - Types and amounts of vitamin supplements
  - Use of complementary/alternative medicines
- Medications
  - *Enzymes*
    - Timing and method of pancreatic supplementation
    - Number of enzymes with meals, snacks and/or tube feeding
    - Units of lipase/kg of body weight per day, or per gram of fat
  - *Other medications*
    - Antibiotics, acid blockers (H_2 antagonists), steroids, alternative medicines

Table 21–2 continued

Biochemical
Electrolytes
Albumin and prealbumin
Vitamin A
Vitamin E
PT and PTT
Blood glucose, hemoglobin A_{1c}
Vitamin D
Iron studies
Zinc
Psychosocial
Socioeconomic status, medical insurance, employment, history of depression, anxiety

DIOS = distal intestinal obstruction syndrome; PT = prothrombin time; PTT = partial thromboplastin time.

Guidelines for nutritional management in CF are detailed in Table 21–3. Table 21–4 offers a stepwise approach for nutritional intervention in CF.

Energy Expenditure

Increased resting energy expenditure (REE) in the range of 104 to 130% of predicted values has been demonstrated in patients with CF.[9,10] In presymptomatic CF, energy expenditure may be closer to normal; with poorer lung function, however, greater levels of energy expenditure are seen.[9,11] On the other hand, elevated REE may not necessarily imply an increase in total energy expenditure (TEE) since individuals may adjust their spontaneous activity to compensate for increases in REE or the extent of their pulmonary disease. Energy needs for physical activity should therefore always be evaluated in CF patients. The level of hypermetabolism during an acute pulmonary exacerbation may also be dependent on the extent of lung disease, with mild to moderate lung disease (forced expiratory volume in one second [FEV_1] > 60%)

Table 21–3. Special Aspects of Nutritional Management in Cystic Fibrosis

Diet	High calorie diet, no fat restriction
Calories	120–150% RDA (even up to 200%)
Protein	RDA for age
Fat	40% total calories
Essential fatty acids	3–5% of total calories
Sodium	Increased needs at times of sweating and during hot weather 0–6 mo: 2 mmol/kg/d in form of NaCl (1 mL = 1mmol) 7–12 mo: 1 mmol/kg/d in form of NaCl 1–5 yr: 10 mmol/d (2 × 300 mg NaCl tablets) 6–10 yr: 20 mmol/d (2 × 600 mg NaCl tablets) 11 yr+: 30–40 mmol/d (3–4 × 600 mg NaCl tablets)
Vitamins and minerals	
Vitamin A	0–12 mo: 1,500 IU/d 1–2 yr: 1,500–3,000 IU/d 2–8 yr: 5,000 IU/d > 8 yr: 5,000–10,000 IU/d
Vitamin D	400–1000 IU/d
Vitamin E	0–6 mo: 25 IU/d 6–12 mo: 50 IU/d 1–4 yr: 100 IU/d 4–10 yr: 100–200 IU/d > 10 yr: 200–400 IU/d
Vitamin K	0–12 mo: 2.5 mg/wk 2.5 mg 2 times/wk if on antibiotics > 1 yr: 5 mg 2 times/wk
Water soluble	RDA × 2
Zinc	RDA unless deficient
Iron	RDA unless deficient
Calcium	RDA for age

Table 21–3. continued

Pancreatic enzymes	Recommended starting dosages of pancreatic enzymes
Infants	1,000–2,000 U lipase per 120 cc (4oz) formula or 500–1,000 U lipase/g dietary fat
Children ≤ 4 yr:	1,000 U lipase/kg/meal 500 U lipase/kg/snack
Children > 4 yr:	500 U lipase/kg/meal; 200 U lipase/kg/snack or 500–4,000 U lipase/g dietary fat (children and adults)
Suggested maximum dosages	2,500 U lipase/kg/meal 10,000 U lipase/kg/d

Adapted from Ramsey et al,[5] Green et al,[6] MacDonald,[7] Anthony et al.[8]

less likely to be associated with an increased REE than is the case with more severe lung disease.[12,13]

Fat

In an effort to control abdominal pain and other symptoms of steatorrhea, patients with CF were previously prescribed a low fat diet. Later, the implementation of a high fat diet with adjustments in exogenous pancreatic enzymes to control malabsorption was associated with better growth and survival among CF patients.[4] Current recommendations are to provide 35 to 40% of calories in the form of long chain fats (LCF). Medium chain triglycerides (MCT) have been used to supplement caloric intake in fat malabsorption since they can be absorbed in the absence of pancreatic lipase and bile salts. Medium chain triglycerides are not a source of essential fatty acids, however, and are expensive and unpalatable.

Protein

Protein loss is not as significant as fat loss in CF, especially if steatorrhea is well controlled. The recommended pro-

Table 21–4. Categories for Nutritional Management of Patients with Cystic Fibrosis

Category	*Target Group*	*Goals*
Routine management	All CF patients	Nutritional education, dietary counseling, pancreatic-enzyme replacement (for patients with pancreatic insufficiency [PI]), vitamin supplementation (for patients with PI)
Anticipatory guidance	CF patients at risk of developing energy imbalance (ie, severe PI, frequent pulmonary infections, and periods of rapid growth) but maintaining a weight/height index ≥ 90% of ideal weight	Further education to prepare for increased energy needs; increased monitoring of dietary intake; increased caloric density in diet as needed; behavioral assessment and counseling
Supportive intervention	Patients with decreased weight velocity and/or a weight/height index 85–90% of ideal weight	All of the above plus oral supplements as needed
Rehabilitative care	Patients with a weight-height index consistently < 85% of ideal weight	All of the above plus enteral supplementation via nasogastric tube or enterostomy as indicated
Resuscitative and palliative care	Patients with a weight-height index < 75% of ideal weight or progressive nutritional failure	All of the above plus continuous enteral feeds or parenteral nutrition

Adapted from Ramsey BW, Farrell PM, Pencharz P, and the Consensus Committee. Nutritional assesment and management in cystic fibrosis: a consensus report. Am J Clin Nutr 1992;55:108–16.

tein intake is the Recommended Dietary Allowance (RDA) for age. Higher protein intakes may reduce renal function already compromised by aminoglycoside antibiotic use.

Carbohydrate

As life expectancy has increased in patients with CF, so has the incidence of glucose intolerance. In a 5-year prospective study on glucose tolerance in CF, prevalence of diabetes increased from 11 to 24% during the study, with an annual age-dependent incidence of 4 to 9%.[14] The mean age for diagnosis of CF-related diabetes (CFRD) is 21 years of age.[14] Diabetes in CF is often asymptomatic and therefore often underdiagnosed. It may, however, present similarly to type I or type II diabetes (polydipsia, polyuria, weight loss, fatigue) but without ketoacidosis or hyperinsulinemia. The oral glucose tolerance test is the most reliable method of screening for CFRD although casual blood glucose and 2-hour postprandial glucose checks can also be used. Hemoglobin A_{1c} and fasting plasma glucose levels were not found to be reliable screening tools for CFRD as these may be normal even in the presence of glucose intolerance.[14] Treatment for CFRD includes insulin or oral hypoglycemic agents. Patients should continue on a high calorie diet. They should be advised to consume consistent amounts of carbohydrate-rich foods at meals and snacks or be taught carbohydrate counting (see Chapter 23). Insulin or oral hypoglycemic agents are adjusted based on intake.

Vitamins and minerals

Supplementation of the fat soluble vitamins is required for all patients with CF and pancreatic insufficiency (see Table 21–3). If serum levels indicate deficiency, compliance should be reviewed before initiating additional supplementation. The patient's financial ability to obtain vit-

Table 21–5. Assessment and Treatment of Fat Soluble Vitamin Deficiencies

Vitamin	*Assessment*		*Therapy if Deficiency*	*Considerations*
A	Normal:	> 20 μg/dL	Infants and children:	Serum level is not a good indicator of liver stores.
	Marginal stores:	10–19	Initially 100,000 units IM once then oral vitamin A	Low in chronic infection, liver disease, or during an acute phase response. Check retinol binding protein (RBP) circulation in plasma. Assess toxicity by using molar ratio of retinol to RBP (see text).
	Deficient:	< 10	< 1 year: 100,000 units every 4–6 months	
			1–8 years: 200,000 units every 4–6 months	
			Children > 8 years and adults: oral: 100,000 units/d for 3 d then 50,000 units/d for 14 d	
D	25-OHD: Normal: 9–75 ng/mL		Ergocalciferol (vitamin D_2):	Low in dietary deficiency, decreased absorption, UV light deficiency, prematurity, liver disease, and with certain drugs (anticonvulsants). Higher in summer.
			Children with malabsorption: 10,000–25,000 IU PO/d until normal	
			Childern with normal absorption: 1,000–5,000 U PO × 6–12 weeks	Watch for hypercalcemia and hypercalciuria and other signs of toxicity.
			Larger single IM doses may be given. Supplement with 400 IU/d thereafter	

E	Deficiency if: Serum level < 5 mg/L Vitamin E:total lipid ratio* < 0.6–0.8 mg/g in adults Vitamin E:chol + TG < 1.59 μmol/mmol† Erythrocyte hemolysis > 10%	100–400 IU/d or 1 mg/kg of water-miscible form plus usual vitamin E supplementation	Carried exclusively on plasma lipoproteins thus vitamin E:total lipid ratio or vitamin E:chol + TG is a better indicator of stores than serum levels Do not give with medications that interfere with vitamin E absorption (vitamin A, cholestyramine, and antacids)
K	Prothrombin time (PT)	Infants and children: 1–2 mg single IM dose Adults: 5–10 mg single IM dose	Deficiency in malabsorption, long-term antibiotic therapy

*Total lipids = cholesterol + triglycerides (TG) + phospholipids.

† Conversions: chol (mg/dl) × 0.0259 = chol (mmol/L); TG (mg/dl) × 0.0113 = TG (mmol/L); vitamin E (mg/L) × 2.32 = vitamin E (μmol/L).

Adapted from Thurnham et al[16] and Alpers et al.[17]

amins should also be assessed. Many insurance policies do not cover the expenses of vitamins and patients are forced to pay for these themselves.

Table 21–5 offers guidelines for assessing and treating vitamin deficiencies in CF. Since vitamin A toxicity is more likely to occur with increasing plasma levels of retinyl esters, laboratory measurements of these esters are the most direct way to assess overdosage of retinol supplements. Excess free retinol may also be diagnosed by measuring the molar ratio of retinol to retinol binding protein (RBP):

retinol (ug/dL) × 0.0349 = μmol/L
RBP (mg/dL) × 0.476 = μmol/L

This molar ratio should be between 0.8 and 1.0. Ratios > 1.0 suggest increased levels of free retinol and possible toxicity.

Water-soluble vitamin requirements can be met through diet and through supplementation with one or two daily multivitamins. Salt supplementation is required during hot weather or during periods of increased sweat (see Table 21–3). Given the relative low amounts of sodium in breastmilk, formulas, and infant foods, infants should generally receive about 1/8 to 1/4 tsp of salt per day. Patients with CF have an increased risk of developing osteoporosis in adulthood. Poor nutrition, malabsorption of calcium and vitamin D, prolonged use of corticosteroids, and increased concentrations of osteoclast-activating factors can lead to poor bone mineral density in CF.[15] Calcium needs should be met either through the diet or via supplementation. Vitamin D requirements are often met through a multivitamin supplement.

Pancreatic Enzymes

Administration. An estimated 85% of all CF patients are pancreatic insufficient. Oral pancreatic enzymes are used

to help normalize absorption and digestion (Table 21–6). Most enzyme products contain enteric-coated microencapsulated enzymes. The enteric coating prevents inactivation of the enzymes in the acidic environment of the stomach. Once in the higher pH of the upper small intestine, the enteric coating breaks down and the enzymes are released. Bicarbonate production may be poor in CF and lead to an abnormally acidic pH in the duodenum, reducing enzyme effectiveness. Acid blockers may be prescribed to reduce stomach acid production and acidity in the upper small intestine. Enzymes should be given with each feed and preferably within 30 minutes of starting the meal. The dosage may also be divided and given before and halfway through the meal, especially if meal time lasts longer than 30 minutes. If the child cannot swallow pills, enzymes should be opened and given in an acidic food (most fruits or vegetables except peas). They should not be given in alkaline foods (eg, milk), crushed, or allowed to sit in food since this can deactivate the enzymes. Foods that do not require enzymes are listed in Table 21–7.

Dosing. Guidelines for initiating enzymes are detailed in Table 21–3. Enzyme dosages are usually prescribed in units of lipase per kilogram of body weight per meal or snack. The dosage is titrated based on symptoms of steatorrhea and/or coefficient of absorption (see below). Poor growth and vitamin deficiencies may also indicate inappropriate enzyme therapy. Several factors can contribute to poor response to enzyme therapy and should be considered before adjusting enzyme dosage (Table 21–8). Enzyme dosage can also be titrated based on units of lipase per gram of fat consumed (eg, starting at 1,000 units of lipase per gram of fat). Higher lipase-containing enzymes should be considered if more than three pills are given with meals.

Fibrosing colonopathy is an inflammatory condition of the large intestine of unclear etiology; it has, however, been associated with the ingestion of high dosages of pancreatic enzymes. In one study,[18] the median daily dosage of patients with fibrosing colonopathy was 50,000 units of lipase/kg/d but dosages as low as 4,900 U/kg/d were also

Table 21–6. Types of Pancreatic Enzymes

Enzyme (Manufacturer)	*Lipase USP Units*	*Protease USP Units*	*Amylase USP Units*
Cotazym-S (Organon)	5,000	20,000	20,000
Zymase (Organon)	12,000	24,000	24,000
Creon 5 (Solvay)	5,000	18,750	16,600
Creon 10 (Solvay)	10,000	37,500	33,200
Creon 20 (Solvay)	20,000	75,000	66,400
Pancrease (McNeil)	4,500	25,000	20,000
Pancrease MT 4 (McNeil)	4,000	12,000	12,000
Pancrease MT 10 (McNeil)	10,000	30,000	30,000
Pancrease MT 16 (McNeil)	16,000	48,000	48,000
Pancrease MT 20 (McNeil)	20,000	44,000	56,000
Ultrase (Scandipharm)	4,500	25,000	20,000
Ultrase MT 12 (Scandipharm)	12,000	39,000	39,000
Ultrase MT 18 (Scandipharm)	18,000	58,500	58,500
Ultrase MT 20 (Scandipharm)	20,000	65,000	65,000
Pancrecarb MS-4* (Digestive Care)	4,000	25,000	25,000
Pancrecarb MS-8* (Digestive Care)	8,000	45,000	40,000
Nonenteric			
Viokase tablets	8,000	30,000	30,000
Viokase powder (0.7g or 1/4 tsp)	16,800	70,000	70,000

*Contains bicarbonate. Buffer capacity of 1.5 mEq/L.
Adapted from educational materials, Clinical Nutrition Service, Children's Hospital, Boston.

Table 21–7. Foods Not Requiring Pancreatic Enzymes

All fruits

Frozen desserts made without fat or protein
eg, Popsicle, Italian ice, sorbet, Jell-O

Candy (except chocolate)
eg, gummies, jelly beans, hard candy, mints, marshmallows, gum, fruit roll-ups

Beverages without protein or fat
eg, carbonated beverages, juices, fruit punch, or lemonade

Table 21–8. Factors Contributing to a Poor Response to Pancreatic Enzyme Therapy

Enzyme factors
- Outdated prescription
- Enzymes not stored in cool place

Dietary factors
- Excessive juice intake
- Parental perception that enzymes are not needed with milk or snacks
- "Grazing" eating behavior
- High-fat fast foods or snacks

Poor adherence to the prescribed enzyme therapy
- Willful refusal of toddler
- Chaotic household, multiple mealgivers
- Anger, or desire to be "normal"
- Teenagers' desire to be thin

Acid intestinal environment
- Poor dissolution of enteric coating
- Microcapsule contents released all at once

Concurrent gastrointestinal disorder
- Lactose malabsorption, enteric bacterial infection, bacterial overgrowth of the small intestine, hepatobiliary disease, cholestasis, celiac disease, short bowel syndrome, Crohn's disease, colitis

Adapted from Borowitz DS, Grand RJ, Durie PR, and the Consensus Committee. Use of pancreatic enzyme supplements for patients with cystic fibrosis in the context of fibrosing colonopathy. J Pediatr 1995;127:681–4.

associated with this condition. Maximum dosages of 2,500 units of lipase/kg/meal and 10,000 units of lipase/kg/d are now recommended although it is recognized that some CF patients will require higher dosages to adequately treat steatorrhea.

Assessing Absorption

Stool energy loss can be significant in CF. Malabsorption can be in the range of 5 to 20% of gross energy intake, even in the presence of pancreatic enzyme replacement (compared to < 5% among healthy children). The 72-hour fecal fat test is considered the "gold standard" for assessing fat malabsorption and is conducted as follows:

1. Collect stools for 72 hours. Freeze stool if possible, otherwise refrigerate.
2. Collect concomitant 3-day food record. Calculate average fat intake (in grams). Goal intake is 2 to 3 g fat/kg/d.
3. Calculate coefficient of fat absorption (COA):

$$\frac{\text{grams of fat consumed} - \text{grams of fat excreted}}{\text{grams of fat consumed}} \times 100 = \text{COA}$$

4. Normal COA: premature infants: 60–75%; newborns: 80–85%; 10 months–3 years: 85–95%; > 3 years: 95%
5. Considerations: notify the lab if the patient is using MCT. Discontinue mineral oil before starting the test.

Formulas and Enteral Feeding

The choice of infant formula to use will depend on the child's nutritional and medical status. Human breastmilk with appropriate enzyme replacement therapy is optimal for infants with CF.[20] Otherwise, milk or soy-based formulas can be used. Infants who undergo gastrointestinal surgery may require temporary use of a semielemental or

elemental formula if intolerance to conventional formula develops. Elemental formulas, however, are neither necessary nor recommended for routine nutritional care of the infant with CF.[21] All formulas, including semielemental, require pancreatic enzymes. The amount of pancreatic enzymes to administer will vary with the fat type and content in the formula. In the older patient, a variety of oral supplements are available (see Chapter 16, Enteral Nutrition). As with infants, nonelemental formulas with enzyme replacement are absorbed as well as are predigested formulas.[21]

The decision to initiate tube feeding should be based on the patient's nutritional status (see Table 21–4), their ability to meet nutrient needs by mouth, and their willingness to initiate or accept more aggressive nutritional support. The concept of tube feeding should be introduced to patients and families early in treatment, even when the child may not require tube feeding, and be presented as a realistic option for meeting the child's nutritional needs. Refer to Chapter 16 for guidelines on formula selection and administration. Pancreatic enzymes are required for all formulas containing long chain fat. Fewer enzymes are needed with elemental or semielemental formulas. No consensus exists on enzyme administration with tube feeding. Methods include providing two-thirds of a typical meal's dose at the beginning of the feed and one-third at the end of the feed or dosing based on the grams of fat in the formula (see Table 21–3), via either pancreatic enzymes by mouth or via viokase powder through the tube.

References

1. FitzSimmons SC. The changing epidemiology of cystic fibrosis. J Pediatr 1993;22:1–9.
2. Cystic Fibrosis Foundation. Patient registry 1998. Annual data report. Bethesda (MD). 1999 September.

3. Zemel BS, Kawchak DA, Cnaan A, et al. Prospective evaluation of resting energy expenditure, nutritional status, pulmonary function and genotype in children with cystic fibrosis. Pediatr Res 1996;40:578–86.
4. Corey M, McLaughlin FJ, Williams M, Levison H. A comparison of survival, growth and pulmonary function in patients with cystic fibrosis in Boston and Toronto. J Clin Epidemiol 1988;41:583–91.
5. Ramsey BW, Farrell PM, Pencharz P, and the Consensus Committee. Nutritional assessment and management in cystic fibrosis: a consensus report. Am J Clin Nutr 1992;55:108–16.
6. Green MR, Buchanan E, Weaver LT. Nutritional management of the infant with cystic fibrosis. Arch Dis Child 1995;72: 452–6.
7. MacDonald A. Nutritional management of cystic fibrosis. Arch Dis Child 1996;74:81–7.
8. Anthony H, Collins CE, Davidson G, et al. Pancreatic enzyme replacement therapy in cystic fibrosis: Australian guidelines. J Pediatr 1999;35:125–9.
9. Fried M, Durie P, Tsiu L, et al. The cystic fibrosis gene and resting energy expenditure. J Pediatr 1991;119:913–6.
10. Girardet JP, Tounian P, Sardet A, et al. Resting energy expenditure in infants with cystic fibrosis. J Pediatr Gastroenterol Nutr 1994;18:214–9.
11. Bronstein MN, Davies PS, Hambidge KM, Accurso FJ. Normal energy expenditure in the infant with presymptomatic cystic fibrosis. J Pediatr 1995;126:28–33.
12. Stallings VA, Fung EB, Hofley PM, Scanlin TF. Acute pulmonary exacerbation is not associated with increased energy expenditure in children with cystic fibrosis. J Pediatr 1998; 132:493–9.
13. Naon H, Hack S, Shelton MT, et al. Resting energy expenditure: evolution during antibiotic treatment for pulmonary exacerbation in cystic fibrosis. Chest 1993;103:1819–25.
14. Lanng S, Hansen A, Thorsteinsson B, et al. Glucose intolerance in patients with cystic fibrosis: a five year prospective study. BMJ 1995;311:655–9.

15. Aris RM, Renner JB, Winders AD, et al. Increased rate of fractures and severe kyphosis: sequelae of living into adulthood with cystic fibrosis. Ann Intern Med 1998;128:186–93.
16. Thurnham DI, Davies JA, Crump BJ, et al. The use of different lipids to express serum tocopherol lipid ratios for the measurement of vitamin E status. Ann Clin Biochem 1986; 23:515–20.
17. Taketomo CK, Hodding JH, Kraus DM, editors. Pediatric dosage handbook. 5th ed. Cleveland: Lexi-Comp Inc.; 1998.
18. FitzSimmons SC, Burkhart GA, Borowitz D, et al. High dose pancreatic-enzyme supplements and fibrosing colonpathy in children with cystic fibrosis. N Engl J Med 1995;336:1283–9.
19. Borowitz DS, Grand RJ, Dure PR, and the Consensus Committee. Use of pancreatic enzyme supplements for patients with cystic fibrosis in the context of fibrosing colonopathy. J Pediatr 1995;127:681–4.
20. Holliday KE, Allen JR, Waters DL, et al. Growth of human milk-fed and formula-fed infants with cystic fibrosis. J Pediatr 1991;118:77–9.
21. Ellis L, Kalnins D, Corey M, et al. Do infants with cystic fibrosis need a protein hydrolysate formula? A prospective, randomized comparative study. J Pediatr 1998;132:270–6.
22. Erskine JM, Lingard CD, Sontag MK, Accurso FJ. Enteral nutrition for patients with cystic fibrosis: comparison of a semi-elemental and non-elemental formula. J Pediatr 1998; 132:265–9.

Additional Resources

Books

Cystic Fibrosis Foundation. Managing cystic fibrosis related diabetes (CFRD): an instruction guide for patients and families. Cystic Fibrosis Foundation; 1999.

Internet Resources

Cystic Fibrosis Foundation web site:
www.cff.org

22

CEREBRAL PALSY AND DEVELOPMENTAL DISABILITIES

Heidi Puelzl Quinn, MS, RD

Developmental disabilities (DD) is a term used to describe a collection of disorders that cause an impairment in normal development and body function.[1] There are a wide range of disabilities, with varying degrees of impact on growth and nutritional status. It is estimated that the incidence of developmental disability in the pediatric population is approximately 3%.[2] Approximately 90% of children with developmental disabilities have nutritional concerns.[2] Some of these are outlined in Table 22–1.

Oral-motor and feeding difficulties are common in children with developmental disabilities. It is generally helpful to have an interdisciplinary feeding evaluation performed to establish an appropriate feeding plan that optimizes diet intake as well as feeding skill development. Ideally, the team should consist of the following clinicians: nutritionist, speech therapist, occupational therapist, physical therapist, behavioral management specialist, developmental pediatrician, nurse. Some children with DD may require support with supplemental tube feedings to meet their fluid and nutrient needs for adequate growth and good health. Coordination of tube feeding and oral feeding to maintain oral motor skills while insuring good growth and health is recommended, provided there are no contraindications to oral feeding, such as aspiration. Regular reassessment of the feeding plan is essential as the child grows and develops.

Table 22–1. Nutritional Risk Factors for Children with Developmental Disabilities

Altered growth
- Obesity (Prader-Willi, Laurence-Moon-Biedl, Carpenter's, and Down syndromes)
- Failure to thrive (Rett syndrome, cerebral palsy [CP])
- Short stature (Down, Hurler's, Russell-Silver, and Cornelia de Lange's syndromes)

Gastrointestinal symptoms
- Diarrhea
- Constipation
- Vomiting/gastroesophageal reflux

Oral-motor difficulties
- Discoordination of suck/swallow
- Structural abnormalities (cleft lip/palate; dentition)
- Poor oral containment (food/fluid loss)
- Tone abnormalities (hypo/hypertonic)
- Altered oral sensory response (hypo/hyper-responsive)
- Delayed oral motor skill development
- Aspiration

Altered nutrient needs/nutrient deficiencies
- Drug-nutrient interactions (anticonvulsants, diuretics, laxatives, tranquilizers)
- Restricted intake (metabolic disease, food allergies, food texture aversion)
- Inadequate intake (poor appetite, poor oral motor control, malabsorption)
- Increased calorie requirement (athetoid CP, spasticity)
- Inadequate fluid intake

Positioning for feeding
- Adaptive seating devices

Behavior
- Oral aversion
- Pica
- Rumination
- Hyperactivity
- Distractibility
- Perseverative behaviors
- Binge eating/overeating

Feeding skill development
- Self-feeder vs. dependent feeder
- Adaptive feeding equipment

Adapted from Hendricks K, Walker WA. Manual of pediatric nutrition. 2nd ed. Toronto: B.C. Decker, Inc.; 1990. p. 211–215.

Special Aspects of Nutritional Assessment and Management

History

There should be a complete review of birth, medical, and feeding history to determine the potential effects of long-term hospitalization, surgery, and medical procedures (ie, intubation, supplemental tube feedings) on overall development as well as on oral feeding and feeding skill development. Early medical/feeding history can provide information regarding development of feeding problems such as oral aversion.

Growth Assessment

Obtaining accurate weight, length/height, and head circumference measurements, and plotting serial points over time, provide critical information on growth adequacy. Growth should be plotted on National Center for Health Statistics (NCHS) growth charts or specialized syndrome-specific growth charts if available. Specialized growth charts are currently available for various diagnoses, including Down syndrome, Turner's syndrome, Prader-Willi syndrome, myelomeningocele, sickle cell disease, and achondroplasia (see Appendix E).[3] Accurate measurement of linear growth in children with DD may be compromised by the presence of contractures, scoliosis, kyphosis, or an inability to stand. Alternative methods of linear measurement include crown-rump length or sitting height, arm span, tibial length or segmented body length.[4,5] Use of these methods may also be compromised by contractures and/or scoliosis.

It is not uncommon for children with DD to be small for their age, with growth parameters below the 5th percentile on standard growth charts.[1] Assessment of weight for length/height is especially important in this population as this indicates individual proportionality, which is a more

appropriate way to evaluate adequacy of growth in children with DD. It is also important to note that alterations in head circumference (micro/macrocephaly) can skew the weight-for-age and weight-for-length parameters. Alterations in body composition with regard to muscle mass and body fat stores also impact the growth assessment. The use of midarm circumference and skin fold measurements are helpful in assessing these parameters.[4]

Alterations in activity level will also have an impact on weight goals. For nonambulatory individuals, weight-for-length of 10 to 25th percentile is generally an acceptable goal. In nonmobile individuals, additional weight is often accumulated as increased fat stores rather than muscle mass. Excessive weight can compromise care in terms of cardiorespiratory health as well as ease of transfers (bed, bath, wheelchair) and progression with gross motor skills. Given these considerations, a visual clinical assessment, in conjunction with growth history, is essential when assessing adequacy of growth in children with developmental disabilities.

Nutrient Requirements

Energy. Caloric requirements may be assessed in several ways: (1) calories per centimeter of body height/length (Table 22–2); (2) catch-up growth equations using height age instead of weight age; or (3) standard equation using Basal Energy Expenditure (BEE) × activity and injury factors.[1] It is important to note, however, that these methods are merely *guidelines* and that individual calorie requirements should be assessed based on changes in weight over time and/or measurement of basal metabolic rate, if possible. Therefore, regular weight monitoring is an essential component in managing children with DD. Caloric requirements may be as low as 5 kcal/cm of height in children with severe central nervous system impairment.[5]

Table 22–2. Guidelines for Estimating Caloric Requirements in Children with Developmental Disabilities

Condition	*Caloric Recommendation*
Ambulatory, ages 5–12 years	13.9 kcal/cm height
Nonambulatory, ages 5–12 years	11.1 kcal/cm height
Cerebral palsy with severely restricted activity	10 kcal/cm height
Cerebral palsy with mild to moderate activity	15 kcal/cm height
Athetoid cerebral palsy, adolescence	Up to 6,000 kcal/d
Down syndrome, boys ages 5–12 years	16.1 kcal/cm height
Down syndrome, girls ages 5–12 years	14.3 kcal/cm height
Myelomeningocele	Approximately 50% of RDA for age after infancy. May need as little as 7 kcal/cm height
Prader-Willi syndrome	10–11 kcal/cm height for weight maintenance; 8–9 kcal/cm height for weight loss

Adapted from: Frick MS. Developmental disability. In: Perberton CN, Moxness KE, German MJ, et al, editors. Mayo Clinic diet manual. 6th ed. Toronto: B.C. Decker; 1988. p. 320; and from Frick MS. Other nutritional considerations. In: Nelson JK, Moxness KE, Jensen MD, Gastineau CF, editors. Mayo Clinic diet manual. 7th ed. St. Louis: Mosby; 1994. p. 457.

Catch-up growth equations using height age:

1. $\text{kcal/kg} = \dfrac{\text{IBW for height} \times \text{RDA kcal/kg height age}}{\text{actual weight}}$

2. $\text{g protein/kg} = \dfrac{\text{IBW for height} \times \text{RDA g protein/kg height age}}{\text{actual weight}}$

IBW = ideal body weight; RDA = Recommended Dietary Allowance.

Protein. Protein requirements are estimated using RDA for chronologic age or height age if growth parameters are significantly below chronologic age.[1]

Vitamins/Minerals. The most common nutrient deficiencies seen in children with DD are vitamins A, C, D, and folate, as well as iron and calcium.[4]

Fluid. Fluid requirements may be higher in some children with DD due to constipation, increased fluid losses (drooling, excessive sweating), and/or increased requirements. Standard guidelines for fluid based on body weight should be followed, with adjustment for special considerations as noted above (see Table 17–3, Fluid Requirements).

Drug-Nutrient Interactions

Some children with DD are on multiple medications, which can interfere with nutrient absorption, appetite, elimination patterns, and level of alertness for feeding. For example, children with seizures who are on multiple anticonvulsant medications should be monitored for adequate vitamin D and folic acid intake as requirements for these nutrients are increased with some seizure medications (eg, phenytoin [Dilantin]). Also, some medications can contribute to constipation, which often inhibits appetite. Due to the potential for inadequate diet intake, drug-nutrient interactions, and possibly decreased mobility, laboratory values reflecting iron, protein, vitamin D, calcium, and phosphorus status should be monitored on a regular basis (see Appendix B, Drug-Nutrient Interactions).

Oral-Motor and Feeding Skill Development

Children with DD are at increased risk for feeding difficulties due to alterations in motor and neurodevelopmental status. Nutritional management often involves dietary modifications such as enhanced calorie intake (Table 22–3), enhanced fiber intake, and texture modification (ie, pureed diets, thick-

ened liquids [Table 22–4]) to meet oral-motor skill level as well as nutrient needs. Natural thickeners are preferred over commercial cornstarch-based thickeners as they provide additional nutrients as well as calories and in some cases contribute to fluid intake. Calorie level can be adjusted based on the choice of thickener. In addition, cornstarch-based thickeners can contribute to constipation, which is a common problem for children with DD.

Ideally, the nutritionist works in conjunction with a speech therapist or occupational therapist to develop a feeding plan. A videofluoroscopic swallow study (also called a "modified barium swallow") may be indicated to assess the efficiency and safety of the swallowing mechanism. This study is performed jointly by a radiologist and a speech language pathologist or occupational therapist with specialization in oral-motor feeding difficulties. The child must willingly consume fluid/foods of several textures in small amounts; this study cannot be performed on a child who will not or cannot consume food or fluid by mouth. Table 22–5 lists several "red flags" of feeding difficulties that indicate further assessment of swallowing function is warranted. Table 22–6 lists the common clinical indicators for performing a swallow study.

Table 22–3. Calorie Enhancers

Butter/ margarine	100 kcal/tbsp	Peanut butter	80 kcal/tbsp
Oil	126 kcal/tbsp	Nonfat dry milk powder	13 kcal/tbsp
Mayonnaise	100 kcal/tbsp	Parmesan cheese	25 kcal/tbsp
Heavy cream	50 kcal/tbsp	American cheese	100 kcal/oz
Light cream	29 kcal/tbsp	Karo syrup	60 kcal/tbsp
Wheat germ	25 kcal/tbsp	Molasses	54 kcal/tbsp
Avocado	375 kcal each	Polycose	23 kcal/tbsp

Table 22–4. Natural Thickeners

Pureed, blenderized, or babyfood vegetables/fruits (5–11 kcal/tbsp) (avoid banana if constipated)
Infant cereal (15 kcal/tbsp) (avoid rice if constipated)
Yogurt (8–16 kcal/tbsp)
Pudding (20 kcal/tbsp)
Soft tofu (10 kcal/tbsp)
Potato flakes (11 kcal/tbsp)
Wheat germ (25 kcal/tbsp)
Graham cracker crumbs (25 kcal/tbsp)
Bread crumbs (22 kcal/tbsp)

Adapted from Feucht S. Guidelines for the use of thickeners in foods and liquids. Nutrition Focus for Children with Special Health Care Needs 1995;10(6):2.

Table 22–5. "Red Flags" of Feeding Difficulties

Coughing, choking, gagging and/or sputtering during or after feeding
Change in vocal or respiratory quality during or after feeding (ie, gurgly, increased congestion)
Nasopharyngeal reflux (food/fluid coming out of the nose)
Increased fatigue associated with feeding
Decrease in oxygen saturation levels during feeding
Food/liquid suctioned from tracheostomy
Difficulty gaining weight
Food refusal
Frequent coughing during tube feeding

Adapted from Arden Hill MS. Presentation for Swallowing Disorders Program, Children's Hospital. Boston. 1998.

Table 22–6. Clinical Indicators for Swallow Study

Coughing, choking, or gagging with feedings
Chronic pulmonary difficulties (ie, recurrent respiratory infections, pneumonia, asthma)
Recurrent episodes of fever of unknown origin

Adapted from Arden Hill MS. *Presentation for Swallowing* Disorders Program, Children's Hospital, Boston. 1998.

Down syndrome, autism, and cerebral palsy are three common forms of developmental disability with distinct nutrition and feeding concerns.

Down Syndrome

Down syndrome is the most common chromosomal anomaly associated with mental retardation.[7] The chromosomal anomaly involves an extra chromosome 21 (trisomy 21). The incidence is reported to be 1 case per 800 to 1000 live births, with increasing incidence with increased maternal age.[8] Approximately 40% of children with Down syndrome are born with congenital heart defects, and 15% are born with gastrointestinal malformations.[8] Children with Down syndrome are also at risk for other medical complications that can affect their nutritional status as well as their overall development.

Special Aspects of Nutritional Assessment in Down Syndrome

History

The patient's medical and feeding history should be obtained to identify issues which may have an impact on growth and feeding (Tables 22–7, 22–8, and 22–9).

Growth

Down syndrome growth charts should be used to plot growth (see Appendix E). Weight for length/height should

Table 22–7. Medical Diagnoses Associated with Down Syndrome

Cardiac anomalies
Intestinal malformations (eg, duodenal atresia, Hirschsprung's disease)
Increased incidence of infections (ear, respiratory)
Endocrine (diabetes, hypothyroidism)
Orthopedic (atlantoaxial instability, hip dislocation)
Dental (delayed/missing dentition)
Increased risk of leukemia
Hearing loss

Table 22–8. Nutritional Risk Factors Associated with Down Syndrome

Poor weight gain (cardiac anomalies, recurrent infections, hypothyroidism)
Obesity
Constipation (hypotonia, hypothyroid, fluid loss)
Delayed oral motor skill development
Delayed feeding skill development
Selective intake
Reduced activity (hypotonia, orthopedic concerns)
Behavior difficulties

Table 22–9. Common Oral-Motor Feeding Difficulties Associated with Down Syndrome

Weak lip seal on nipple (fluid loss)
Tongue protrusion/thrust
Delayed chewing (secondary to delayed dentition and/or prolonged tongue thrust)
Difficulty with texture transition
Difficulty with thin liquids (increased fluid loss and coughing/sputtering)

be plotted with NCHS growth charts as this parameter is not available on the Down syndrome growth chart.

Nutrient Requirements

Energy. Given the short stature inherent to Down syndrome, it has been determined that caloric requirements for children with Down syndrome aged 5 to 12 years should be based on body height rather than body weight to avoid overestimating[8] (see Table 22–2). It is important to note that obesity is a significant nutritional risk factor for children with Down syndrome, with approximately 25% being affected.[7] Prevention should therefore be the focus by promoting healthy eating habits early in life and avoiding use of food as a reward for good behavior. Regular physical activity such as swimming or dancing should also be encouraged.

Protein. Protein requirements for children with Down syndrome should be assessed using the Recommended Dietary Allowance (RDA) based on sex and age.

Vitamins/Minerals. There is much controversy surrounding variations in vitamin and mineral requirements for children with Down syndrome. Studies to date have not shown any increased requirements due to Down syndrome itself. One study, however, showed that 80% of the children in the study had problems related to food intake or feeding, including excessive calorie intake and low intakes of iron, calcium, vitamin C, and fluid.[7] If diet intake is limited due to selective food intake, a multivitamin with iron may be indicated. Supplementation with additional nutrients beyond a standard multivitamin is not indicated at this time. Children who may be receiving supplementation at levels well above the RDA should be monitored to insure intake does not reach toxic levels.

Fluid. Extra fluid may be indicated for children with Down syndrome who have constipation.

Cerebral Palsy

Cerebral palsy (CP) comprises a group of chronic, nonprogressive disorders of the nervous system that produce abnormalities of posture, muscle tone, and motor coordination. It is classified according to the specific abnormality in muscle tone (hypertonia, hypotonia) and extrapyramidal signs (choreoathetosis, ataxia, and dystonia). There is an estimated incidence of 2 cases per 1000 live births.[1] Due to their motor involvement, children with CP may have many of the feeding problems listed in Table 22–1, including poor growth and oral-motor feeding difficulties due to poor oral-motor control. In addition, medications commonly used to help treat spasticity, seizures, constipation, and/or gastroesophageal reflux can impact nutrient intake and feeding skills as well as behavioral state (lethargy, distraction, drowsiness) at mealtime. Regular monitoring of growth, diet intake, and oral-motor feeding skills by a multidisciplinary team is essential to maximize growth, intake, oral-motor skills, and feeding skills. Feeding evaluations should also include assessment for adaptive seating and adaptive feeding utensils to facilitate intake.

Autism

Autism is a developmental disorder characterized by a severe impairment of language, cognitive skills, and social development.[10] Ritualistic and obsessive/compulsive behavior is frequently seen.[1] Approximately 70% of children with autism have some level of mental retardation.[11] The etiology of autism is unclear but the disorder is believed to have a neurobiologic basis with multiple possible causes, including structural abnormalities of the brain, viruses, genetic disorders, chromosomal abnormalities (fragile X syndrome), metabolic disorders (PKU), and specific seizure disorder (infantile spasms).[10] The incidence of autism has been reported as 1 in every 500–1,000 people.[12]

Table 22–10. Common Feeding Concerns for Children with Pervasive Developmental Disorder/Autism

Difficulty with texture transition
Heightened sensory responses
Restricted intake due to color/texture/temperature of foods
Decreased selection of foods over time
Difficulty accepting new foods
Difficulty with administration of multivitamin/mineral supplement
Difficulty with changes in mealtime environment

Adapted from Puelzl Quinn H, Levine K. Nutrition concerns for children with pervasive developmental disorder/autism. Nutrition Focus for Children with Special Health Needs 1995;10(5):3.

The primary nutrition and feeding concern in children with autism is selective intake, often due to altered sensory responses that affect how food tastes, smells, and feels inside their mouth (Tables 22–10 and 22–11). For some children, intake may be limited to as few as two or three foods or beverages. Foods may be refused due to color, temperature, texture, smell, or slight variations in taste; the accepted foods are frequently brand-specific. Supplementation with a multivitamin with minerals in a form the child will accept

Table 22–11. Helpful Mealtime Strategies for Children with Pervasive Developmental Disorder/Autism

Consistent mealtime environment
Calm, comfortable environment
Some children focus better on eating with accompaniment of music or video
Some children do better eating with others at the table, some do better eating alone

Adapted from Puelzl Quinn H, Levine K. Nutrition concerns for children with pervasive developmental disorder/autism. Nutrition Focus for Children with Special Health Needs 1995;10(5):3.

may be difficult, possibly requiring multiple trials with various forms (liquid, powder, tablet). It is important to note that an accepted food or beverage may subsequently be refused if alterations in the taste, smell, or texture are detected when the supplement is added. Despite selective intake, adequate growth is generally seen, with the exception of late infancy/early toddlerhood when there is sometimes poor growth as a result of the transition from baby foods to table foods. Specific energy requirements for children with autism have not been established but the RDA for age is generally used, with modifications as needed based on activity level. Some children with autism are quite sedentary while others are constantly active, often with self-stimulatory behavior (eg, spinning, hand flapping, rocking).

Due to the special feeding challenges presented by the child with autism, the nutrition and feeding assessment should be addressed by a team that includes a nutritionist, an occupational therapist or speech therapist with training in oral-motor sensory therapy and/or sensory integration, and a psychologist/psychiatrist.

Perhaps because there is no cure for autism, parents are often drawn to investigate alternative therapies. Current alternate therapies include high-dose vitamin B_6 and magnesium supplementation, dimethylglycine (DMG), a gluten-free/casein-free diet, and a yeast-free diet.[11] Further research regarding the efficacy of these therapies is required.

References

1. Selvaggi-Fadden K, Puelzl Quinn H, Kastner T. Developmental disabilities. In: Rickert VI, editor. Adolescent nutrition—assessment and management. New York: Chapman and Hall; 1996. 299–322.
2. Hendricks K, Walker WA. Manual of pediatric nutrition. 2nd ed. Toronto: B.C. Decker, Inc.; 1990. p. 211–5.
3. Walberg-Ekvall S. Nutritional assessment and early intervention. In: Walberg-Ekvall S, editor. Pediatric nutrition in

chronic diseases and developmental disorders—prevention, assessment, and treatment. New York: Oxford University Press; 1993. p. 41–76.

4. Pipes PL, Pritkin Glass R. Developmental disabilities and other special health care needs. In: Pipes PL, Trahms CM, editors. Nutrition in infancy and childhood. 5th ed. St. Louis: Mosby; 1993. p. 344–73.
5. Bandini LG, Puelzl Quinn H, Morelli J, Fukagawa N. Estimation of energy requirements in persons with severe central nervous system impairment. J Pediatr 1995;126(5): 828–32.
6. Feucht S. Guidelines for the use of thickeners in food and liquids. Nutrition Focus for Children with Special Health Care Needs 1995;10(6):1–6.
7. Patterson B, Walberg-Ekvall S. Down syndrome. In: Walberg-Ekvall S, editor. Pediatric nutrition in chronic diseases and developmental disorders—prevention, assessment, and treatment. New York: Oxford University Press; 1993. p. 149–56.
8. Culley WJ, Middleton TO. Calorie requirements of mentally retarded children with and without motor dysfunction. J Pediatrics 1969;75:380–4.
9. Gersh E, Riley J. The daily care of your baby. In: Stray-Gundersen K. Babies with Down syndrome–a new parents guide. Bethesda (MD): Woodbine House; 1995.
10. Patterson B, Walberg-Ekvall S, Dickerson Mayes S. Autism. In: Walberg-Ekvall S, editor. Pediatric nutrition in chronic diseases and developmental disabilities—prevention, assessment, and treatment. New York: Oxford University Press; 1993. p. 131–6.
11. Puelzl Quinn H, Levine K. Nutrition concerns for children with pervasive developmental disorder/autism. Nutrition Focus for Children with Special Health Needs 1995;10(5):1–7.
12. Filipek PA et al. The screening and diagnosis of autism spectrum disorder. J Autism Dev Disord 1999;29(6):439–84.
13. Bandini L, Patterson B, Walberg-Ekvall S. Cerebral palsy. In: Walberg-Ekvall S, editor. Pediatric nutrition in chronic diseases and developmental disorders—prevention, assessment, and treatment. New York: Oxford University Press; 1993. p. 93–8.

Resources

Newsletter

Nutrition Focus for Children with Special Health Needs
University of Seattle, Seattle, WA
Sharon Feucht, Editor
206-685-1297

Support Groups

March of Dimes Foundation
1275 Mamaroneck Ave.
White Plains, NY 10605
914-428-7100

National Down Syndrome Congress
1800 Dempster Road
Park Ridge, IL 60068-1146
800-232-NDSC

Autism Society of America
7910 Woodmont Ave., Suite 650
Bethesda, MD 20814
301-657-0881

United Cerebral Palsy Association
Seven Penn Plaza, Suite 804
New York, NY 10001
800-USA-1UCP

Textbook

Prefeeding Skills
Suzanne Evans Morris and Marsha Dunn Klein
Therapy Skill Builders
Tuscon, AZ
602-323-7500

Instruction Manual

Feeding and Nutrition for the Child with Special Needs
Marsha Dunn Klein and Tracy Delaney
Therapy Skill Builders
Tuscon, AZ
602-323-7500

23

DIABETES MELLITUS

Roberta D. Laredo, RD, CDE

Diabetes mellitus is a chronic disease resulting from absolute or relative insulin deficiency that occurs in both children and adults. Approximately one of every 600 children in the United States has diabetes, making it one of the most common chronic childhood illnesses. Diabetes occurs when insulin, normally produced by the beta cells of the pancreas, is either absent, insufficient, or not used properly by the target tissues. Glucose builds up in the blood stream when insulin is unavailable to allow it to enter the cells. Long-term elevated blood glucose levels can lead to the chronic complications of diabetes, including retinopathy, nephropathy, neuropathy, and macrovascular disease.

Type 1 diabetes is an autoimmune disease in which the beta cells of the pancreas eventually produce little or no insulin. Type 1 diabetes generally occurs in children and young adults. The body's immune system attacks and destroys the beta cells of the pancreas. Initial therapy involves medical management to correct the hyperglycemia, glycosuria, and ketonuria responsible for symptoms, including polydipsia, polyuria, dehydration, and weight loss. Management of type 1 diabetes requires insulin replacement via daily insulin injections and consistent timing and composition of meals and snacks.

Type 2 diabetes is a disease that results from the body's inability to produce enough insulin, and/or properly respond to it. Type 2 diabetes most commonly occurs in

people > 40 years of age, but can occur in overweight adolescents as well. The increasing number of obese children and adults has resulted in a corresponding increase in the prevalence of type 2 diabetes. This form of diabetes can go undiagnosed for long periods of time as the classic symptoms are less dramatic than in type 1 diabetes. The insulin resistance associated with obesity and characteristic of type 2 diabetes can often be controlled by weight loss, improved nutrition, and exercise. Sometimes oral medications and/or insulin are needed to control hyperglycemia.

Importance of Blood Glucose Control

The Diabetes Control and Complications Trial (DCCT) was a landmark 9-year multicenter trial designed to determine whether blood glucose control is related to the risk of developing the complications of diabetes. It involved 1,441 adults and teenagers with type 1 diabetes who were randomized into two therapy groups: (a) conventional therapy consisting of one to two daily insulin injections, quarterly visits with physician and diabetes nurse educator, and nutrition education as requested by the participant; and (b) intensive therapy involving three or more daily insulin injections, monthly visits with the DCCT team, and ongoing nutrition education to adjust the insulin dose to the planned food and exercise regimen. The intensively treated group had an average HbA_{1c} of 7.2% compared to 9.0% for the conventionally treated group; the nondiabetic reference range for the HbA_{1c} was 4.0 to 6.0%. The study also demonstrated definitively that improving blood glucose control slows or prevents the development of the long-term complications of type 1 diabetes—the risk of developing retinopathy was reduced by 76%, neuropathy by 60%, and evidence of renal disease was reduced 40 to 50%.[1]

Acute and Chronic Complications

The major acute complications of diabetes occuring in children are hypoglycemia, hyperglycemia, and diabetic ketoacidosis. Hypoglycemia is caused by too little food, delayed or missed meals and snacks, increased exercise, excessive insulin, or alcohol intake without food. Hyperglycemia is caused by increased food intake, inadequate insulin dose, or a decrease in usual exercise. Diabetic ketoacidosis results from an absolute lack of insulin and the build-up of ketoacids in the blood.

The chronic complications of diabetes are microvascular disease (neuropathy, nephropathy, retinopathy), macrovascular disease (ischemic heart disease, cerebrovascular disease, peripheral vascular disease), and poor growth and development. Many of the chronic complications can be prevented or delayed with optimal blood glucose control, management of dyslipidemia and hypertension, proper weight management, and smoking cessation.

Table 23–1 provides approximations of the onset, peak, and duration of the current insulin preparations available. The actual action time of insulin will vary between patients and is affected by a number of factors, including the size of the dose, site and depth of injection, and exercise.

Table 23–1. Common Insulin Preparations

	Humalog Short Acting	*Regular Short Acting*	*NPH Intermediate Acting*	*Lente Intermediate Acting*	*Ultralente Long Acting*
Onset	< 15 min	0.5–1 h	1–3 h	1–3 h	4–6 h
Peak	30–90 min	2–3 h	4–12 h	6–12 h	8–20 h
Duration	2–4 h	6–8 h	18–24 h	18–24 h	24–28 h

Reproduced with permission from A balancing act. The Children's Hospital, Boston guide to caring for a child with diabetes, 1999.

General Nutrition Therapy Goals

The American Diabetes Association has established the following goals for all people with diabetes:

- Maintain blood glucose levels as near-normal as possible by balancing food intake, insulin, and exercise
- Achieve optimal blood lipid levels
- Provide appropriate calories for normal growth and development
- Prevent/delay acute and chronic complications
- Improve health through optimal nutrition[2,3]

Nutrition Therapy Goals for Type 1 Diabetes

Daily management is based on the integration of insulin injections with eating and exercise habits. Consistency in the timing and composition of meals and snacks is the key to minimizing fluctuations of the blood glucose level. Long-term goals are to maintain normal growth and development, quality of life, and prevent/delay the chronic complications of diabetes.

Nutrition Therapy Goals for Type 2 Diabetes

The management goal is to achieve and maintain optimal blood glucose and lipid control by making nutrition and lifestyle changes. Recommendations are to space meals and snacks throughout the day, make healthy food choices, moderate total fat, saturated fat, and calorie intake, and increase physical activity. Insulin sensitivity improves with even a modest amount of weight loss. It is recommended that a moderate calorie restriction, 250 to 500 calories less than average daily intake, be implemented.[2,3] The long-term goal is to attain and maintain the healthiest weight possible.

Tables 23–2 and 23–3 summarize the nutritional assessment and therapy of pediatric patients with diabetes.

Table 23–2. Special Aspects of Nutritional Assessment in Diabetes

History	Duration of disease, nutrition history, pattern of growth and weight gain, activity pattern, psychosocial/economic issues, smoking, medical history (celiac disease, nephropathy, hyperlipidemia, eating disorder, high blood pressure, asthma, attention deficit disorder, hypothyroidism, and other autoimmune diseases), insulin regimen, oral glucose-lowering medications, blood glucose monitoring schedule
Physical examination	Current height/weight
Laboratory	HbA_{1c}, lipid profile, urine ketones/protein, microalbuminuria, blood pressure, fasting/nonfasting blood glucose

Adapted from The American Dietetic Association. Type 1 diabetes mellitus in children and adolescents medical nutrition therapy protocol. Medical nutrition therapy across the continuum of care. 1999. [In press].

Sweeteners

Historically, sucrose has been restricted in the diets of people with diabetes, based on the belief that sucrose is more rapidly digested and absorbed than starches. Research comparing the glycemic effect of high-sucrose and low-sucrose meals, however, has shown a consistently similar blood glucose effect for the two test meals. It is the total amount of carbohydrate eaten, rather than the type of carbohydrate, that has the greatest effect on blood glucose control. It is now accepted that sucrose can be substituted gram for gram for other carbohydrates within the context of a healthy meal plan.[4] Nutritive sweeteners include fructose, honey, corn syrup, molasses, fruit juice or fruit juice concentrates, dextrose, maltose, mannitol, sorbitol, xylitol, and hydrogenated starch hydrolysates. Research has shown no significant advantage or disadvan-

Table 23–3. Special Aspects of Nutritional Management in Diabetes

Calories	Based on nutritional assessment and requirements for growth
Carbohydrate	Based on nutritional assessment (generally 50 to 60% of total calories)
Protein	10 to 20% of total calories
Fat	Up to 30% of total calories, with saturated fat < 10% of calories
Fiber	20 to 35 g/d (same as general population) or 5 g/d + age (years) in children

Adapted from American Diabetes Association. Nutrition principles and recommendations (Position Statement). Diabetes Care 1994; 17:519–22.

tage over sucrose. The sugar alcohols (mannitol, sorbitol, and xylitol) should be limited due to their laxative effect if consumed in large amounts.[5]

Non-nutritive sweeteners such as aspartame, acesulfame K, saccharin, and sucralose can all be safely used by people with diabetes.[5] The usual daily intake of these sweeteners by children with diabetes is far less than the Acceptable Daily Intake (ADI) set by the Food and Drug Administration. The ADI is the level of non-nutritive sweetener that can be eaten for a lifetime without an effect on health. For example, the ADI for aspartame is 50 mg/kg/d, which equals six 12-ounce cans of diet soda every day for a 23-kg child.[6]

Alcohol

For adults with diabetes, alcohol consumption should be limited to no more than two drinks no more than two to three times per week. One drink is equal to 12 oz of beer, 5 oz of wine, or 1 1/2 oz of distilled alcohol.[2,3] The use of alcohol in the adolescent population must be acknowl-

edged. Adolescents with diabetes should follow the guidelines established for adults if they choose to drink alcohol. Alcohol should only be consumed with food. Alcohol should never be substituted for food in the meal plan, and extra insulin should not be taken when drinking alcohol due to the hypoglycemic effect of alcohol.

Meal Plan

Each child with diabetes should receive an individualized meal plan with the appropriate calorie level to promote normal growth and development. The meal plan is based on the exchange system created by the American Diabetes Association and the American Dietetic Association (Table 23-4). The exchange lists group specific servings of foods together because they have a similar amount of carbohydrate, protein, fat, and calories. One food can be substituted, or exchanged, for another within each exchange list. The meal plan sets consistent times and food composition for each meal and snack. Most children need to eat three meals and two to three snacks per day, spaced 2½ to 3 hours apart. While daytime snacks can contain exclusively carbohydrate, the bedtime snack should include protein and fat to minimize the risk of nocturnal hypoglycemia. The meal plan also encourages healthy, well-balanced meals and snacks by setting the portions of fruits, vegetables, and dairy products as well as starches, protein, and fat each child should consume daily. The meal plan should be reviewed every 3 to 6 months[7] to adjust for changes in growth and development, school routines, seasonal sports, and childcare arrangements.

Carbohydrate Counting

Carbohydrate counting is another meal planning method in which the grams of carbohydrate, or carbohydrate servings, eaten at each meal and snack are counted. Carbohydrate is the main nutrient in starches, fruits, milk, yogurt, and other

Table 23–4. The American Diabetes Association/ American Dietetic Association Exchange Lists for Meal Planning

Groups/Lists	*Carbohydrate (grams)*	*Protein (grams)*	*Fat (grams)*	*Calories*
Carbohydrate group				
Starch	15	3	1 or less	80
Fruit	15	–	–	60
Milk				
Skim	12	8	0–3	90
Low fat	12	8	5	120
Whole	12	8	8	150
Other carbohydrates	15	varies	varies	varies
Vegetables	5	2	–	25
Meat and meat substitute group				
Very lean	–	7	3	55
Medium fat	–	7	5	75
High fat	–	7	8	100
Fat group	–	–	5	45

Reproduced with permission from the American Diabetes Association/American Dietetic Association. Exchange lists for meal planning; 1995.

foods containing sugar and is the nutrient that has the greatest effect on blood glucose levels. About 90% of carbohydrate converts to glucose within 1 to 2 hours of eating. Using the exchange food lists, each starch, fruit, or milk exchange is counted as 15 grams of carbohydrate, that is, one carbohydrate serving. The grams of carbohydrate, or carbohydrate servings, can be calculated based on the number of starch, fruit, and milk exchanges allowed for each meal and snack. In addition, food labels list the amount of carbohydrate in grams for other foods not found on the exchange lists. Carbohydrate counting allows added flexibility in food choices and works well for the way children like to eat. For example, a breakfast of two starch exchanges

(30 g), one fruit exchange (15 g), and one milk exchange (12 g) is approximately equivalent to 60 g of carbohydrate, that is four carbohydrate servings.

Special Considerations

"No concentrated sweets" diets are not recommended as a meal planning option due to the similar effect of all carbohydrate-containing foods on blood glucose level.

Infection, illness, and surgery all make glucose control difficult due to multiple factors, including increased counter-regulatory hormones, anorexia, and altered meals and snacks. In general, sick children should be given their usual insulin dose. Insulin should never be skipped, and extra insulin is often required. Blood glucose levels should be checked every 3 to 4 hours and urine ketones should be checked if the blood glucose level is over 240 mg/dL. If the child is able to eat, he or she should be given 4 to 6 oz of sugar-free fluid each hour in addition to regular meals. If the child is not able to eat his usual meals and snacks, sugar-free drinks should be alternated with sugar-containing drinks. The carbohydrate grams or servings allotted in the meal plan should be replaced with sugar-containing sodas, popsicles, juices, and gelatin.

Exercise, whether gym class, a soccer game, or a bike ride, lowers the blood glucose level both during and for up to 6 to 24 hours after the exercise. This delayed effect of exercise is called the "lag effect." To evaluate the effect of exercise, the blood glucose level should be checked before and after exercise. Exercise is not recommended if the blood glucose level is > 250 mg/dL with ketones in the urine or > 300 mg/dL without ketones in the urine. The safest time to exercise is after a meal or snack, when the blood glucose level is slightly higher. Basic snack guidelines are to add one starch or fruit exchange (15 g carbohydrate) for every 30 to 60 minutes of exercise.

Table 23–5. Blood Glucose Goals for Children with Diabetes

Children < 5 years of age: 100–200 mg/dL
Children 5–11 years of age: 80–180 mg/dL
Children 12–18 years of age: 70–150 mg/dL

Reproduced with permission from A balancing act. Children's Hospital, Boston guide to caring for a child with diabetes. 1999.

Table 23–5 provides age-dependent blood glucose targets for pediatric patients with diabetes.

Stages of Life

Infants and Toddlers

Since young children are not consistent in their eating habits and cannot recognize symptoms of hypoglycemia, strict blood glucose control is not usually attainable. Generally, higher blood glucose goals are accepted (see Table 23–5), and the main goal is to avoid hypoglycemia. Infants with diabetes may certainly continue to breastfeed. Toddlers are more independent in their eating habits. Their appetites are decreasing, and they are often more selective in their food choices. Toddlers should be allowed to eat in a calm, relaxed manner and should never be force-fed. Meal plans encouraging consistent meals and snacks should be taught at this age but the variability in a toddler's eating habits must be acknowledged and accepted. Parents are ultimately responsible for providing appropriate meals and snacks; the child will decide how much and what to eat. Insulin can be given after meals for young children who are especially unpredictable in their eating habits, with the dose based on the amount of food the child actually eats.

Preschool and School Age

More structured meal and snack times should be established at this time. Limit snacks to regularly scheduled times as much as possible. School, sports, and the physical education schedule should be reviewed. Most school lunches fit into a child's meal plan. School lunch menus should be reviewed for the child's preferences and for acceptability within the meal plan. The child with diabetes should be encouraged to help with menu planning, buying groceries, preparing meals, and choosing snacks. Information should be provided on how to make the best food choices at parties, sleepovers, and restaurants.

Adolescents

Diabetes management is often most challenging at this time as the teenager is becoming more independent in managing his or her diabetes care. There are more meals away from home, with less parental supervision. Appetite and growth parameters should be monitored to guide the teenager toward making appropriate food choices. Practical information should be provided on accommodating fast food, managing restaurant eating, and adjusting the meal plan for school sports, activities, jobs, and other times away from home.

References

1. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329:977–86.
2. American Diabetes Association. Nutrition principles and recommendations (Position Statement). Diabetes Care 1994; 17:519–22.
3. Franz M, Coulston A, Horton C, et al. Nutrition principles for the management of diabetes and other complications [technical review]. Diabetes Care 1994;490–518.

4. Holler HJ, Pastors JG, editors. Diabetes medical nutrition therapy. Chicago: The American Dietetic Association/ American Diabetes Association; 1997.
5. The American Dietetic Association. Appropriate use of nutritive and nonnutritive sweeteners (Position Statement). J Am Diet Assoc 1993;93:816–21.
6. Geil PB. Complex and simple carbohydrates in diabetes therapy. In: Powers MA, editor. Handbook of diabetes medical nutrition therapy. 2nd ed. Gaithersburg (MD): Aspen Publishers, Inc; 1996. p. 308–9.
7. Connell JE, Thomas-Doberson D. Nutritional management of children and adolescents with insulin-dependent diabetes mellitus: a review by the Diabetes Care and Education dietetic practice group. J Am Diet Assoc 1991;91:1556–64.

Additional Resources

Books, Journals, Guidelines

1. Diabetes Care and Education Dietetic Practice Group of The American Dietetic Association. Type 1 nutrition practice guidelines. Chicago: The American Dietetic Association; 1996.
2. Holzmeister LA. Update on nutrition therapy for children with IDDM. Top Clin Nutr 1997;12(4):26–36.
3. Diabetes Care and Education Dietetic Practice Group of The American Dietetic Association. Selected aspects of diabetes nutrition-prevention and management in infants and children. On the Cutting Edge. 1993; summer 14(4).
4. American Diabetes Association. Clinical practice recommendations 2000. Diabetes Care 2000;23 Suppl 1:S1–S116.
5. Holzmeister LA. Medical nutrition therapy for children and adolescents with type 1 diabetes mellitus. Diabetes Spectrum 1997;10(4):268–74.
6. Satter E. Child of mine, eating with love and good sense. Palo Alto (CA): Bull Publishing; 1987.
7. American Diabetes Association. Single Topic Diabetes and Nutrition Resources: Children with diabetes (birth to 5 years); Children with diabetes (6–11 years); Teens, food and making choices. American Diabetes Association; 1995.

Professional Organizations

American Diabetes Association
1160 Duke St., Alexandria, VA 22314
1-800-232-3472
Website: http://www.diabetes.org

The American Dietetic Association
216 West Jackson Blvd. Chicago, IL 60611-3901
1-800-366-1655
Website: http://www.eatright.org

The Juvenile Diabetes Foundation
120 Wall Street, New York, NY 10005-4001
1-800-JDF-CURE
Website: http://www.jdf.org

American Association of Diabetes Educators
444 North Michigan Ave., Suite 1200
Chicago, IL 60611-3901
1-800-338-3633
Website: http://www.aadenet.org/

International Diabetic Athletes Association
1647 West Bethany Home Road, Phoenix, AZ 85015
1-800-898-IDAA
Website: http://www.diabetes-exercise.org

Internet Resources

Children with Diabetes
Website: http://www.childrenwithdiabetes.com/
Ask Noah about: Diabetes
Website: http://www.noah.cuny.edu/diabetes/diabetes.html

24

EATING DISORDERS

Susan E. Frates, MS, RD, and Heidi Schauster, MS, RD

Eating disorders are characterized by a disturbed relationship between nutritional intake and body image, often leading to subsequent medical problems. While eating disorders are found predominantly in the adolescent and young adult populations, they are increasingly being recognized in children and preadolescents. Eating disorders are the third most common chronic illness in adolescents following obesity and asthma.[1] Anorexia nervosa is estimated to occur in < 3% of adolescent women and bulimia nervosa in 1 to 4%.[2] Undiagnosed disordered eating appears to afflict many school-aged Americans. In 1995, over one-third of Boston high school students reported that they were trying to lose weight. Six to seven percent of these students reported having vomited or taken laxatives "in the last 30 days" to avoid absorbing calories.[3] Males are also currently emerging as a population at risk for disordered eating. The age of onset of eating disorders appears to be decreasing.

The Diagnostic and Statistical Manual of the American Psychiatric Association (DSM IV) details three official classifications of eating disorders: anorexia nervosa (both restrictive and binge/purge types), bulimia nervosa, and eating disorder NOS (not otherwise specified);[4] these are summarized in Tables 24–1, 24–2, and 24–3.

Medical complications often lead to inpatient hospitalizations. The sequelae of physiologic complications detailed in Table 24–4 may afflict major body systems and include cardiac instability, electrolyte imbalance, endocrine

dysfunction, and skeletal system weakness.[5] In serious cases, sudden death from cardiac arrest and refeeding syndrome have been reported. Refeeding syndrome refers to severe extracellular hypophosphatemia as the body moves from using catabolized muscle and fat to carbohydrate with refeeding. Ultimately this may result in decreased ATP, which can lead to cardiac and respiratory failure.[6]

Criteria for inpatient hospitalization, outlined in Table 24–5, include medical instability, severe malnutrition, acute food refusal, and psychiatric emergency.[7] The National Center for Health Statistics (NCHS) growth charts are the common tool for determining ideal body weight ranges in children and adolescents (typically the 10th to 50th percentile weight/height/age). The most important evidence of a weight problem, however, is a sudden crossing of a percentile that does not correspond to linear growth.

Successful treatment for eating-disordered patients involves a treatment team consisting of a medical doctor, nutritionist (registered dietitian), therapist (individual and family), and in some cases a psychiatrist or psychopharmacologist.[8] Inpatient medical treatment includes a progression of caloric intake, with medical monitoring and restriction of physical activity. Meal plans usually start with a base calorie level of 1,500 for females and 1,750 for males. This may increase by 250 calories daily. Although a consistent protocol is important, care must be individualized to respond to the unique situations and needs of each patient. Table 24–6 outlines a sample of the Anorexia Nervosa Eating Disorder Protocol used at Children's Hospital, Boston.

Recommendations for treatment of eating disorders include a rapid diagnosis of problematic eating behaviors

and assembly of a collaborative, multidisciplinary treatment team. Outpatient management of eating disorders typically takes a more gradual, integrated approach to weight management and normalization of eating habits. Efforts to educate children on the development of healthful eating habits, sound body image, and self-esteem are recommended as a means of prevention.

Table 24–1. DSM IV Diagnostic Criteria for Anorexia Nervosa (307.1)

Refusal to maintain body weight at or above a minimally normal weight for age and height	
Weight loss to < 85% expected weight for height	
Failure to make expected weight gain during a period of growth, leading to body weight < 85% of that expected	
Intense fear of gaining weight or becoming fat, even though underweight	
Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self evaluation, or denial of the seriousness of the current low body weight	
Amenorrhea in postmenarchal women	
The absence of at least three consecutive menstrual cycles	
Also, if menstrual periods occur only after administration of hormones such as estrogen	
Specify Type:	
Restricting type:	no regular use of binge eating or purging behavior (self-induced vomiting or the misuse of laxatives, diuretics, or enemas)
Binge eating/ purging type: laxatives,	regular use of binge eating or purging behavior (self-induced vomiting or the misuse of diuretics, or enemas)

Adapted from the American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington (DC): American Psychiatric Association; 1994.

Table 24–2. DSM IV Diagnostic Criteria for Bulimia Nervosa (307.51)

Recurrent episodes of binge eating, characterized by:

1. Eating, in a discrete period of time (ie, within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances; and
2. A sense of lack of control over eating during the episode (ie, a feeling that one cannot stop eating or control what or how much one is eating)

Recurrent inappropriate compensatory behavior in order to prevent weight gain (ie, self-induced vomiting, misuse of laxatives, diuretics, enemas, or other medications; fasting; excessive exercise)

The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months

Self-evaluation is unduly influenced by body shape and weight

The disturbance does not occur exclusively during episodes of anorexia nervosa

Specify Type:

Purging type:	Regular use of self-induced vomiting or the misuse of laxatives, diuretics, or enemas
Nonpurging type:	Use of other inappropriate compensatory behaviors, such as fasting or excessive exercise. No regular use of self-induced vomiting or misuse of laxatives, diuretics, or enemas

Adapted from the American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington (DC): American Psychiatric Association; 1994.

Table 24–3. DSM IV Diagnostic Criteria for Eating Disorder Not Otherwise Specified (307.50)

This is a category for disorders of eating that do not meet the criteria for any specific eating disorder. Examples include:

- All of the criteria for anorexia nervosa but the individual has regular menses
- All of the criteria for anorexia nervosa except that, despite substantial weight loss, the individuals' weight is in the normal range
- All of the criteria for bulimia nervosa are met, except binges occur at a frequency of less than twice a week or for a duration of less than 3 months
- An individual of normal body weight who regularly engages in inappropriate compensatory behavior after eating small amounts of food (ie, self-induced vomiting after consuming two cookies)
- An individual who repeatedly chews and spits out, but does not swallow, large amounts of food
- Binge eating disorder: recurrent episodes of binge eating in the absence of regular use of inappropriate compensatory behaviors characteristic of bulimia nervosa

Adapted from the American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington (DC): American Psychiatric Association; 1994.

Table 24–4. Medical Complications of Eating Disorders

Cardiovascular	*Dermatologic*
Bradycardia	Acrocyanosis
Orthostatic hypotension	Yellow dry skin (hypercarotenemia)
Electrocardiographic abnormalities	Brittle hair and nails
	Lanugo
Ipecac cardiomyopathy*	Hair loss
Congestive heart failure	Russel sign (calluses over knuckles)*
	Pitting edema
Electrolyte and Fluid Imbalance	
	Endocrine
Hypokalemia	Growth retardation and short stature
Hyponatremia	Delayed puberty
Hypochloremic alkalosis	Amenorrhea
Elevated BUN	Low T3 syndrome
Inability to concentrate urine	Hypercortisolism
Ketonuria	
Gastrointestinal	*Hematologic*
Parotid hypertrophy*	Bone marrow suppression
Constipation	Low sedimentation rate
Delayed gastric emptying	Impaired cell-mediated immunity
Esophagitis*	
Mallory-Weiss tears*	
Neurologic	*Skeletal*
Myopathy	Osteopenia
Peripheral neuropathy	Fractures
Cortical atrophy	Cavities (dental and enamel erosion)*

BUN = blood urea nitrogen.

*Applies specifically to persons utilizing self-induced vomiting behaviors.

Adapted by permission of Elsevier Science from Fischer M, Golden N, Katzman D. Eating disorders in adolescents: a background paper. J Adolesc Health 1995;3(16):420–37. Copyright 1995 by The Society for Adolescent Medicine.

Table 24–5. Criteria for Hospitalization for Eating Disorders

Unstable vital signs
- Orthostasis
- Severe bradycardia
- Severe hypothermia
- Severe hypotension
- Cardiac dysrhythmia

Severe malnutrition
- Loss of > 25% ideal body weight
- Weight < 75% ideal body weight
- Arrested growth and development

Dehydration

Electrolyte abnormality

Refeeding syndrome

Acute food refusal

Uncontrollable binging and purging

Acute psychiatric emergencies
- Suicidality/suicidal ideation
- Acute psychosis

Comorbid diagnosis disrupting treatment of eating disorder
- Severe depression
- Obsessive compulsive disorder
- Severe family dysfunction

Failure of outpatient therapy

Adapted by permission of Elsevier Science from Fischer M, Golden N, Katzman D. Eating disorders in adolescents: a background paper. J Adolesc Health 1995;3(16):420–37. Copyright 1995 by The Society for Adolescent Medicine.

Table 24–6. Sample Inpatient Medical Protocol

Anorexia Nervosa Protocol, Children's Hospital, Boston

Goals:

To stabilize heart rate, blood pressure, electrolytes, and body temperature via improving nutritional status

Medical monitoring:

Vital signs taken every 4 hours
Minimal vital signs: HR > 50 BP > 90/50 temperature > 97°F
Heart monitor is used if HR is low at night
If vital signs are below criteria, strict bed rest and restricted/supervised use of commode
If vital signs are WNL, supervised room rest, may walk to activity room and bathroom
Weight taken every morning
Urine specific gravity every morning

Nutrition therapy:

Start with 1,500 calorie meal plan for females
Start with 1,750 calorie meal plan for males
(Lower base calorie levels may be established in more compromised patients)
Meal plans typically increase 250 calories per day until calorie level is met for weight gain goals
Nutrition consultation with RD within 24 hours of admission to create individual meal plans
Vegetarian and religious dietary guidelines are respected
Fat-free, lite, and diet products are not allowed
Food from home is not allowed
Patients may select food preferences using exchange system for meal planning

Fluids:

Maintenance of fluid needs provided daily
Minimum of 8 oz calorie-containing fluid per meal is provided

Supplementation:

If meal is not completed within 30 minutes, supplement equivalent is offered, equaling the entire caloric content of meal
If patient is unable to drink the supplements within 10 minutes, nasogastric tube is placed and supplement is provided enterally

Standard multivitamin with minerals daily
Phosphorus: 500 mg Neutraphos bid. Dose adjusted after follow-up phosphorus labs

Weight gain expectations:
Baseline weight established on first morning after admission, after adequate hydration is met
Patient is weighed every morning, in johnny, after urine void (to check specific gravity)
0.2 kg weight gain is expected every day of hospitalization
If expected weight gain is not met, additional supplement is provided as follows:
- additional 250 calories on day 1
- additional 500 calories for day 2
- additional 750 calories for day 3, etc.

Exercise:
Not permitted during hospitalization

HR = heart rate; BP = blood pressure; WNL = within normal limits; RD = registered dietician.

References

1. The Society for Adolescent Medicine. Eating disorders in adolescence: a position paper of the society for adolescent medicine. J Adolesc Health 1995;3(16):476–80.
2. Fischer M, Golden N, Katzman D. Eating disorders in adolescents: a background paper. J Adolesc Health 1995;3(16): 420–37.
3. Massachusetts Department of Public Health. Youth Behavior Risk Survey data file for Boston Public Schools, 1993–1995.
4. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Washington (DC): American Psychiatric Association; 1994.
5. Mitchel JE, Pomeroy C, Adson DE. Managing medical complications. In: Garner D, Garfinkel P. Handbook of treatment for eating disorders. 2nd ed. New York: The Gilford Press; 1997.
6. Solomon S, Kirby D. The refeeding syndrome: a review. J Parenteral Enteral Nutr 1990;14(1):90–7.

7. Rome E, Vazquez IM, Emans SJ. Nutritional problems in adolescence. In: Walker WA, Watkins J. Nutrition in pediatrics. Hamilton (ON): B.C. Decker, Inc.; 1997.
8. Reiff D, Reiff K. Eating disorders: nutrition therapy in the recovery process. Mercer Island (WA): Life Enterprises; 1997.

Additional Resources

Eating Disorder Organizations

AABA: American Anorexia/Bulimia Association
1265 West 46th St. #1108
New York, New York 10036
212-575-6200
http://members.aol.com/Amanda

IAEDP: International Association of Eating Disorders Professionals
123 NW 13th Street, #206
Boca Raton, FL 33432-1618
800-800-8126
www.iaedp.com

MEDA: Massachusetts Eating Disorders Association, Inc.
92 Pearl Street
Newton, MA 02158
617-558-1818
www.medainc.org

25

FOOD ALLERGIES

Laurie A. Higgins, RD

Food allergy can be defined as an "immunologic reaction resulting from the ingestion of a food or a food additive" as opposed to the more general term "food intolerance," which includes any abnormal response to a food or food additive.[1] It has been estimated that 6 to 8% of all children are affected by food allergies.[2,3] Allergies may occur after a small amount of the allergen is ingested and are unrelated to any physiologic effect of the food, food additive, or cross-contaminant.[2] The most common allergies encountered during infancy and childhood are to cow's milk protein (CMP), soy protein, fish, eggs, and cereals. Other foods children may be allergic to include berries, nuts, peanuts, and chocolate.[4]

There are four types of hypersensitivity reactions that may occur alone or in combination to cause allergic responses (Table 25–1). Clinical symptoms may be gastrointestinal, respiratory, dermatologic, or systemic in nature (Table 25–2).[5] The "gold standard" for the diagnosis of food allergies is a double-blind, placebo-controlled food challenge. The cessation of symptoms after removal of the offending food(s) and their reappearance after its reintroduction is also a common test.[6] In addition, several clinical and laboratory tests exist to help in the diagnosis of food allergies (Table 25–3).

Formula-fed infants have a higher incidence of allergic symptoms than do breastfed infants, likely due to their earlier exposure to cow's milk protein. Since foreign antigens can be expressed in breastmilk, however, breastfed infants

Table 25–1. Four Types of Food Sensitivity Reactions

Type I:	IgE, immediate (anaphylactic) hypersensitivity
Type II:	Antibody-dependent cytotoxic hypersensitivity
Type III:	IgG, immune complex-mediated hypersensitivity
Type IV:	Cell-mediated hypersensitivity, T cells

may still suffer from food allergies.[7] Vomiting, diarrhea, and occult or frank blood in the stool due to food allergy is often referred to as allergic colitis.[2] The rectum and colon

Table 25–2. Food Allergy Symptoms

Gastrointestinal
- Abdominal pain, bloating
- Diarrhea, malabsorption, failure to thrive
- Gastrointestinal bleeding
- Nausea
- Vomiting
- Constipation

Skin
- Eczema/atopic dermatitis
- Urticaria, angioedema, lip swelling
- Itching, rash

Respiratory
- Asthma
- Chronic cough
- Rhinitis/rhinorrhea
- Wheezing

Systemic/general
- Anaphylaxis

Other
- Headache
- Behavioral changes

Adapted from Stern M. Allergic enteropathy. In: Walker WA, Durie PR, Hamilton JR, et al. Pediatric gastrointestinal disease. 2nd ed. St. Louis: Mosby; 1996. p. 677–92.

Table 25–3. Common Laboratory Tests for Food Allergies

Laboratory Test	*Procedure*	*Comments*
Skin test (prick test)	A small amount of the allergen is introduced to the skin; a wheal greater than 3 mm is usually interpreted as positive	Confirms sensitivity to the antigen but does not confirm diagnosis; positive test in children younger than the age of 1 year is likely to be significant A positive skin test to some foods may persist when clinical symptoms are no longer present
Radioactive immunosorbent test (RAST)	IgE antibodies to specific foods	IgE-mediated forms of food allergy only; may be substituted for skin test when there is a suspicion for anaphylaxis; poor correlation with oral challenge
Serum IgE	Blood test	Wide range of normal values; nonspecific and insensitive
Small bowel biopsy	Endoscopy	Nonspecific histopathology with patchy distribution

are inflamed due to immune-mediated responses to ingested proteins. Cow's milk protein is most often responsible for allergic colitis. Changing the infant's formula or removing the suspected antigens from the mother's diet will usually result in a decrease in bleeding within 72 hours; complete resolution, however, can take up to 4 to 6 weeks.[2] Bottle-fed infants with allergic colitis should be treated with formula containing an extensively hydrolyzed protein since 10 to 50% of infants with cow's milk protein allergy will also have an allergy to soy protein.[5,7,9] A small number of infants who may not respond to the extensively hydrolyzed formula could benefit from an amino acid-based formula.[9] If the infant is breastfed, the mother should eliminate all milk and soy proteins from her diet. If the symptoms of colitis resolve, soy protein can often be reintroduced slowly, with the child followed for signs of intolerance. In some cases, further maternal diet restrictions may be necessary. Other foods that have been associated with allergic colitis are wheat, eggs, corn, fish, seafood, and nuts.[10] Care must be taken to avoid overrestriction of the maternal diet since nursing mothers require 300 to 500 more calories and 15 to 20 g more protein per day than usual.

Many pediatric patients will outgrow their food allergies by the age of 3 to 5 years, with the exception of allergies to peanuts, fish, shellfish, and nuts.[1] Reintroduction of foods can be done in either an open or a blinded format. The type of challenge should be determined by the physician and depends on the age of the child and the symptoms of the allergic presentation. In an open challenge, the patient is given a small amount of the food protein and followed for tolerance. In a double blind, placebo-controlled food challenge, the patient and caretaker are unaware of when the patient is receiving the suspected antigen. The challenge is usually done in a controlled

environment so that the child can be observed closely and treated if adverse reactions occur. Intravenous access should be secured before the challenge if severe symptoms are possible. The suspected antigen is in the form of a flour or the food itself ground into a powder. The placebo should consist of a powder of similar appearance and be mixed with a neutral food. The patient is given either the suspected antigen or placebo in an alternating and random order. The patient is observed and given a serving every 20 to 30 minutes, with the amount of food increasing until a total of 8 to 10 g dry or 60 to 100 g of wet food protein is ingested.[11] If the patient does not react, then the double-blind challenge is followed by an open challenge to ensure that the allergy is no longer present.

Single food allergies, with the exception of milk, do not usually propose a nutritional risk for most children. Certain combinations of two or more food allergies, however, can make it difficult for the patient to consume a diet adequate in all macro- and micronutrients. This can be a particular concern in pediatrics since food variety can sometimes be limiting. Evaluation by a registered dietitian can provide the appropriate information, education, and suggestions for nutrient supplementation when a patient is placed on a restricted diet due to food allergy.

Examples of Restricted Diets*

Corn-Free Diet

The corn-free diet is a modification of the normal diet, with the following ingredients eliminated: corn, cornstarch, corn syrup, corn oil, corn sweeteners, maize, and popcorn. The following ingredients may also contain corn

*The following diets have been reproduced with the written permission of Children's Hospital, Boston (MA).

and should also be eliminated: hydrolyzed plant protein (HPP), hydrolyzed vegetable protein (HVP), starch (usually is cornstarch but can be wheat or other vegetables, which need not be eliminated).

Egg-Free Diet

The egg-free diet is a modification of the normal diet, with the following ingredients eliminated: albumin (protein part of the egg), eggs, egg white, egg yolk, dried egg, egg powder, egg solids, some egg substitutes (which contained eggs), eggnog, globulin (could be egg protein), livetin, lysozyme (used in Europe), mayonnaise (made with egg whites and oil), meringue (made with egg whites and sugar), ovalbumin (principle protein in eggs), ovomucin, ovomucoid or ovovitellin (synonyms for egg protein), and Simplesse (fat substitute made from either egg or milk protein).

The following are also potential sources of egg protein:

- Egg white or albumin contains most of the protein but the yolk should also be avoided.
- Many baked products that have a yellow color or shiny glaze are made with eggs/or egg whites.
- Egg whites are often used as a clarifying agent in broths or soups. Always check with the chef when dining out.
- Measles, mumps, and rubella vaccine includes egg protein.
- Influenza vaccines are grown on egg embryos and could contain trace amounts of egg protein.
- Intravenous lipids use egg proteins as an emulsifier.

Tips for Egg-Free Cooking. There are a number of eggs substitutes that can be used in cooking. The following suggestions governing their use may be helpful:

- Use an egg substitute such as Jolly Joan, Golden Harvest, or Ener-G Foods egg replacer. Other brands such as Egg Beaters may have egg whites in them.

- Mashed bananas and apricot puree add flavor and act as both a binder and a thickener in place of egg in quick breads, cakes, cookies, or other sweets.
- Use 2 tbsp of pureed fruit for each egg in recipe. Also, 2 tbsp of pureed vegetables can replace an egg in soups, sauces, and other dishes.
- To bind or thicken fruit desserts, use 1 tsp of dry, unflavored gelatin mixed with 2 tbsp of liquid to replace one egg.
- Because baked goods without eggs crumble easily, use smaller pans. For example, make cupcakes instead of a cake, or muffins instead of bread. Xanthan gum is excellent for holding baked goods together. Use 1 tsp per recipe. To help leaven baked goods, add an extra 1/2 tsp egg-free baking powder for each egg called for in a recipe, with an additional egg substitute to bind or thicken.
- For thickening cream dishes and sauces, add extra flour, cornstarch, or xanthan gum.
- To enhance the flavor of egg-free cookies or cake, add extra ingredients such as raisins, nuts, coconuts, seeds, or spices.

In egg-free baked goods, the following egg substitutes may be used:*

- Tahini (ground sesame seeds): 2 tbsp to replace each egg
- Any nut butter: 2 tbsp to replace each egg
- Oat flour: 2 tbsp plus 1 tbsp water to replace each egg
- 1 tsp baking powder, 1 tbsp liquid, and 1 tbsp vinegar to replace each egg
- 1 tsp yeast dissolved in 1/4 cup warm water to replace each egg
- 1 1/2 tbsp water, 1 1/2 tbsp vegetable oil, and 1 tsp baking powder to replace each egg

*Adapted from Yoder ER. Allergy-free cooking. Addison-Wesley Publishing Co.; 1987.

Milk-Free Diet

The milk-free diet is a modification of a normal diet with the following ingredients eliminated: artificial butter flavor, butter, butter fat, buttermilk, casein (milk protein), caseinates (ammonium, calcium, magnesium, potassium, sodium), cheese, cottage cheese, curds, cream, custard, pudding, ghee (clarified butter), Half and Half, hydrolysates (casein, milk protein, protein, whey, whey protein), lactoglobulin, lactose, milk (derivative, protein, solids, malted, condensed, evaporated, dry, whole, low fat, nonfat, skim), nondairy creamer (check for casein), nougat, rennet (curdled milk), sour cream, sour cream solids, whey-milk protein (delactosed, demineralized, protein concentrate), and yogurt.

The following foods or ingredients may indicate the presence of milk or milk proteins: brown sugar flavoring, caramel flavoring, chocolate, high protein flour (protein source could be skim milk powder), margarine (may contain whey), natural flavoring, and Simplesse (could be made from eggs or milk protein).

The following are potential sources of milk or milk proteins:

- Parve or pareve are words that indicate that the product is milk and meat free under Jewish law. The Food Allergy Network "no longer recommends relying on parve-labeled products for milk-free diets," since small amounts of milk may still be present.[12]
- Product labels that include "K_D" or "U_D" indicate the presence of milk. The ingredient list does not always list the milk source. Some labels are now labeled "K_{DE}," indicating that the product is kosher but made on dairy equipment.
- Medication: certain vitamin and mineral supplements as well as some prescribed and over-the-counter drugs contain lactose as a filler.

- Delicatessen meats often contain whey/casein in the brines surrounding the meat in prepackaged products. Crosscontamination from other meats/cheese products can also occur on slicing.

Nutritional Adequacy. If the patient is taking a fortified milk substitute (eg, soy or rice milk), a supplement may be unnecessary. If a fortified milk substitute is not consumed, the diet may be deficient in calcium, phosphorus, and vitamin D. Supplementation with these nutrients is then recommended (see Chapter 5, Nutritional Requirements: Dietary Reference Intakes and Chapter 13, Vitamin and Mineral Supplements).

Peanut-Free Diet

The peanut is a legume, not a nut. Legumes are edible seeds enclosed in pods and include soybeans, lima beans, carob, and sweet clover. Ingredients to avoid for those with peanut allergy include cold pressed peanut oil, ground nuts, mixed nuts, peanuts, peanut butter, and peanut flour.

The following foods or ingredients may contain peanuts or peanut products: African, Chinese, and Thai dishes, baked goods (pastries, cookies, etc), candy, chili and spaghetti sauce (may use peanut butter as a thickening agent), chocolate candies, HPP, HVP, and marzipan (usually made from almonds but can often be a mixture of nuts).

The following are important considerations for those with peanut allergy:

- Peanut allergy is not usually outgrown.
- Peanut oil is usually not a problem provided it is free of peanut protein.
- Check all candy labels since they will often list peanuts on the label if made in the same facility as a candy containing peanuts. For example, Plain M & M's and Raisinetts both indicate on the label that they may contain peanuts but

peanuts are not necessarily included in the ingredients list.
- Avoid artificial nuts that may contain peanuts.
- Some ethnic restaurants often use peanuts in a variety of foods, making crosscontamination highly possible.
- Egg rolls are occasionally sealed with peanut butter.
- Soy butter is available as a peanut butter substitute.

Tree Nut-Free Diet

Most nuts are the seeds or dried fruits of trees. They grow all over the world in assorted shapes and sizes. Those with nut allergy should avoid foods with the following ingredients: almonds, Brazil nuts, cashews, filberts, hazelnuts, hickory nuts, macadamia, pecans, pine nuts (pignoli, pinon nuts, Indian nuts), pistachios, walnuts (black & Persian), Gianduja or Nutella (a creamy mixture of chocolate and chopped toasted nuts), marzipan/almond paste, nut butters (almond, cashews), nut oil, and nut paste.

It should also be noted that:

- Artificial nuts consist of a variety of nuts ground and reshaped into other nuts.
- Natural extracts such as almond extract and natural wintergreen extract (usually made with filbert/hazelnut) should be avoided.
- Imitation rather than natural flavoring should be used.
- Nuts are added to a variety of foods, cereal, crackers, wheatless cakes, ice cream, and baked goods.
- Nuts are used in many ethnic dishes.
- Coconut, nutmeg, and water chestnuts are not in the tree nut family.

Shellfish-Free Diet

Edible shellfish are usually divided into two categories, mollusks and crustaceans. Mollusks such as clams and mussels have two shells; the abalone, which has a shell

covering and a soft underpart, is also considered a mollusk. Crustaceans have segmented bodies covered with an armor-like section of thick and thin shells (eg, lobster). Those with shellfish allergy should avoid the following ingredients: abalone, clams (cherrystones, littleneck, pismo, quahog, surf clam, steamer, geoduck, razor, mud, and white), crab (Atlantic blue crab, soft-shell crab, stone crab), crawfish (crayfish, écrevisse), lobster (spiny or rock lobster), mussels, oysters (blue points, lynnhavens, chincoteagers), scallops (bay, sea, and calico), mollusk, shrimp (prawn, crevette), and cockle (periwinkle, sea urchin).

Soy-Free Diet

The soy-free diet is a modification of the normal diet with the elimination of soybeans and all foods containing byproducts of soybeans. Soybeans are a legume and are a staple of Asian diets. Those with soy allergy should also avoid the following ingredients: edamame (green vegetable soybeans), hydrolyzed soy protein, lecithin (extracted from soybean oil and used as an emulsifier), natto (made from fermented whole cooked soybeans), miso (a rich salty condiment used in Japanese cooking), soya, soy sauce (tamari, shoyu, teriyaki), soy fiber (okra, soy bran, soy isolate fiber), soy flour, soy grits, soy milk, soy nuts, soy sprouts, soy protein concentrate, soy protein isolates, soy oil, tempeh (Indonesian—a chunky, tender soybean cake), textured vegetable protein (TVP), tofu (soybean curds), and yuba (made by lifting and drying the thin layer formed on the surface of cooling soy milk).

The following foods or ingredients may contain soy protein: flavoring, HVP, HPP, natural flavoring, textured soy protein (TSP), textured soy flour (TSF), vegetable broth, vegetable gum, and vegetable starch.

Most people with soy allergies may safely eat soy lecithin and soy oil. Soy lecithin is a mixture of fatty substances, a byproduct of soybean processing. Lecithin is often used as a stabilizer, emulsifier, or an antioxidant.

Wheat-Free Diet

The wheat-free diet is a modification of the normal diet with the following ingredients eliminated: bread crumbs, bran, bulgur, cereal extract, cracker meal, enriched flour, farina, flour, gluten (protein in wheat), graham flour (can be a blend of flours containing wheat), matzo or matzo meal, high gluten flour, high protein flour, malt vital gluten, wheat bran, wheat grain, wheat gluten, wheat starch, and whole wheat flour.

The following foods or ingredients contain wheat proteins: gelatinized starch, HVP, modified food starch, natural flavoring, soy sauce, starch, vegetable gum, and vegetable starch.

It should also be noted that:

- One cup wheat flour can be substituted by 1/2 cup oat flour and 1/2 cup rice flour.
- Ethnic cookbooks contain many wheat-free recipes (eg, Hispanic/Latino and Asian cookbooks often use rice).
- Spaghetti squash and corn or rice pasta may substitute for regular pasta.
- Fresh, frozen, and canned vegetables are usually wheat-free whereas prepackaged vegetables in sauces often contain wheat as filler.
- Gluten-free means wheat-free.
- Triticale is a cross of wheat and rye.

References

1. Sampson HA, Metcalf DD. Food allergies. JAMA 1992; 268:2840–4.
2. Bock SA, Sampson HA. Food allergy in infancy. Pediatr Clin North Am 1994;41(5):1047–67.
3. Young E, Stoneham MD, Petruckevitch A, et al. A population study of food intolerance. Lancet 1994;343:1127–30.
4. Goldman AS, Kantak AG, HamPong AJ, Goldblum RM. Food hypersensitivities: historical perspectives, diagnosis and clinical presentations. In: Brostoff J, Challacombe SJ, editors. Food allergy and intolerance. London: Bailliere Tindall; 1987. p. 797–805.
5. Stern M. Allergic enteropathy. In: Walker WA, Durie PR, Hamilton JR, et al. Pediatric gastrointestinal disease. 2nd ed. St. Louis: Mosby; 1996. p. 677–92.
6. Patrick MK, Gall DG. Protein intolerance and immunocyte and enterocyte interaction. Pediatr Clin North Am 1988; 35(1):17–34.
7. Lake AM, Whitington PF, Hamilton SR. Dietary protein-induced colitis in breastfed infants. J Pediatr 1982;101: 906–10.
8. Odze RD, Wershil BK, Leichtner AM, Antonioli DA. Allergic colitis in infants. J Pediatr 1995;126:163–70.
9. Vanderhoof JA, Murray ND, Kaufman SS, et al. Intolerance to protein hydrolysate infant formulas: an under-recognized cause of gastrointestinal symptoms in infants. J Pediatr 1997;131(5):741–4.
10. Sampson HA. IgE-mediated food intolerances. J Allergy Clin Immunol 1988;81:495–504.
11. Watson WTA. Food allergy in children. Clin Rev Allergy Immunol 1995;13:347–59.
12. Regenstein JM. Are "pareve" products really milk-free? Food Allergy News 1998;7(6):1.

Additional Resources

National Support Groups

The Food Allergy Network
4744 Holly Avenue
Fairfax, VA 22030-5647
Fax 703-691-2713
Website http://www.foodallergy.org.

The Food Allergy Network (FAN) is a national nonprofit organization established to help families living with food allergies and increase public awareness about food allergies and anaphylaxis. The focus is on children but there are many adult members. All the resources are checked for medical accuracy by FAN's nine-member medical advisory board. There is a subscription fee.

Allergy and Asthma Network/Mothers of Asthmatics, Inc.
10400 Eaton Place Suite 107
Fairfax, VA 22030
703-691-3179 or 800-929-4040

The Allergy and Asthma Network/Mothers of Asthmatics, Inc. publishes a monthly newsletter with practical information for patients and families. Books, videos, and other educational materials available are also available.

Other Organizations

Nut Allergy

Vermont Nut Free Chocolates,
P.O. Box 67, Grand Isle, VT 05458,
1-888-4-NUT-FREE, phone/fax: 802-372-4654,
email: vtnutfree@aol.com/

Soy Allergy

Indiana Soybean Board. U.S. 1998 Soyfoods Directory.
Stevens and Associates, Inc.,
4816 North Pennsylvania Street, Indianapolis, IN 46205-1774.
Website: http://www.soyfoods.com/

Internet Resources

Milk Protein Allergy

http://www.non-diary.org/
http://www.tofutti.com/
http://www.whitewave.com/
http://www.vegetariantimes.com/
http://www.choclat.com/
http://www.navigator.tufts.edu/ (a rating guide to nutrition websites)
http://www.eatright.org/ (The American Dietetics Association)

Peanut Allergy

http://www.peanutallergy.com/

26

GASTROINTESTINAL DISEASES

Laurie A. Higgins, RD

Gastroesophageal Reflux

Gastroesophageal reflux (GER) is the effortless movement of gastric contents into the esophagus. While GER is considered a normal physiologic process rather than a disease, it can produce clinical symptoms ranging from mild heartburn to esophagitis, respiratory disease, and even apnea. Gastroesophageal reflux disease (GERD) refers to these symptoms. Pediatric patients with GERD may present with chest pain, dyspepsia, vomiting, burping, dysphagia, postprandial fullness, chronic hoarseness and cough, wheezing, and respiratory symptoms of unknown etiology. Gastroesophageal reflux disease is also a major cause of anorexia, resulting in malnutrition among pediatric patients with a variety of chronic illnesses. Multiple physiologic factors are generally thought to be responsible for GERD, including decreased lower esophageal sphincter (LES) tone, esophageal mucosal irritation from hydrochloric acid and pepsin, delayed esophageal peristalsis, and delayed gastric emptying.

Treatment for GERD may include lifestyle (Table 26–1), dietary (Table 26–2), and pharmacologic therapies.

Pharmacologic therapy should be used only if dietary and lifestyle changes do not alleviate the symptoms. Medications used to control GERD include antacids, H_2 receptor antagonists, proton pump inhibitors, and proki-

Table 26–1. Nonmedical Therapy for Infants with Gastroesophageal Reflux Disease

Positioning: elevate the head of the bed
Small frequent feeds
Thickening feeds: 1 tsp (25 cal/oz) up to 1 tbsp (32–35 cal/oz) rice cereal to each ounce of formula
Avoid feeding before nap or bedtime

netic agents. Surgery may be indicated in severe cases of GERD refractory to medical management.

Celiac Disease

Celiac disease is a lifelong disease of the small intestine characterized by an abnormal mucosa and associated with a permanent intolerance to gluten. Gluten is a protein that

Table 26–2. Dietary Therapy for Gastroesophageal Reflux Disease

Avoid foods high in:	*Comments*
Fat	Fat delays gastric emptying
Acidic and spicy foods	May worsen heartburn
Caffeinated and decaffeinated beverages	May lower LES pressure
Tea, carminatives (spearmint and peppermint), chocolate	May lower LES pressure
Small frequent meals (6 meals a day)	Avoid overdistention of stomach
Drink fluids between vs during meals	Avoid overdistention of stomach
Lifestyle changes:	
Avoid napping or activity immediately after mealtime	Prevents GER
Elevate head of bed 6 inches	Gravity promotes esophageal clearance

LES = lower esophageal sphincter; GER = gastroesophageal reflux.

makes up 50% of the total proteins in wheat, rye, and barley; gliadin is the water soluble protein fraction of prolamin. The prevalence of celiac disease in the United States is estimated at 1:2,000 to 3,000, with populations in western Ireland and Italy showing prevalences estimated at 1:300.[1] Population screening suggests that subclinical or preclinical celiac disease is not uncommon.

Clinical symptoms of celiac disease usually present in toddlers but can do so at any age. Exposure to dietary gluten is required for symptoms to occur, and celiac disease is more prevalent in countries where wheat is a staple food. Clinical symptoms vary widely (Table 26–3). Celiac disease has been associated with other autoimmune diseases such as Addison's disease, pernicious anemia, autoimmune thrombocytopenia, sarcoidosis, insulin-dependent diabetes mellitus, cystic fibrosis, and dermatitis herpetiformis.[1] There is also a higher prevalence of celiac disease in children with Down syndrome.[2]

The development and widespread use of serologic screening tests for celiac disease (ie, antiendomysial and antigliadin antibodies) has revolutionized its diagnosis. Small intestinal biopsy, however, is still considered the "gold standard" of diagnosis. The characteristic histologic features of the disease (flattened villi, crypt hyperplasia, and intraepithelial lymphocytes) should all normalize with dietary avoidance of gluten.

Following initial diagnosis and a period of gluten-free diet, a gluten challenge is often suggested to confirm the diagnosis. For this challenge, the author recommends surreptitiously adding wheat flour to the child's usual gluten-free foods since sudden liberalization of the diet may interfere with attempts to restrict it in the future. Providing a consistent amount of wheat protein (Table 26–4) for 2 to 3 months or until symptoms recur is recommended. If clinical symptoms return, serologic confirmation of the diagno-

Table 26–3. Clinical Symptoms of Celiac Disease

Abdominal distention	Hyperphagia
Abdominal pain	Irritability/fatigue
Anorexia	Malnutrition
Constipation	Rash or skin infections
Dental hypoplasia	Rectal prolapse
Dermatitis herpetiformis	Short stature
Diarrhea	Sleep disturbance
Foul smelling stools	Vomiting

sis may be adequate. If symptoms do not return, a second small intestinal biopsy may be warranted.[3]

The primary treatment for individuals with celiac disease is a gluten-free diet. The response to removal of gluten in the diet is rapid, with symptoms usually improving within a few weeks. Persistent symptoms may be due to secondary lactase deficiency; lactose may need to be temporarily removed from the diet, depending on the extent of mucosal damage to the intestine.[4]

The gluten-free diet should provide adequate nourishment while eliminating foods that contain gliadin. Oats do not contain gliadin but have avenin in their prolamin fraction. It is not well understood at this time whether avenin

Table 26–4. Wheat Protein Intake in a Normal Diet in Children of Different Ages[1]

Age (yr)	*Wheat Protein (g/d)*
1 year	Variable
1–3	5–10
3–6	7–12
6–9	10–15
9+	15–30

is as harmful as gliadin to the intestinal mucosa. The effects of gluten-free diets with and without oats were compared in adults with celiac disease and no adverse effects were seen.[5] The inclusion of oats in the pediatric gluten-free diet remains controversial.

Avoiding obvious dietary gluten is not always adequate for controlling symptoms, and all sources of gluten must be identified to ensure that the diet is gluten free. Gluten may be hidden in many food additives or preservatives, including textured vegetable protein (TVP), hydrolyzed vegetable protein (HVP), hydrolyzed plant protein (HPP), starch, malt or malt flavoring, vegetable gum, distilled white or grain vinegar, and medications.

Another challenge of maintaining a gluten-free diet is the constant changing of ingredients by food manufacturers. Food labels must be checked regularly (Tables 26–5 and 26–6). Many gluten-free foods can be found in supermarkets, health food stores, Asian markets, kosher markets (especially during Passover), or can be ordered from a variety of companies specializing in gluten-free foods.

The gluten-free diet shown below in Table 26–7 provides guidelines for the gluten-free diet recommended at Children's Hospital, Boston.

Constipation

Constipation is a common medical condition in pediatric patients, especially those between the ages of 1 and 5 years. Most cases of constipation are idiopathic in nature although structural and metabolic causes also occur.

Diets low in fiber and fluids are often a contributing factor in constipation. Dietary management is the first step in treatment and is often sufficient for relief of symptoms. The American Health Foundation (AHF) recommends a daily dietary fiber intake of "age (years) plus five" grams for all children older than 2 years.[6] When increasing fiber

Table 26–5. Additives and Ingredients to Avoid in a Gluten-Free Diet

Additives*	Alcohol*	Cereal products*
Barley	Bran*	Dextrins*
Coloring*	Bulgar	Durham wheat
Emulsifiers*	Distilled white vinegar	Groats
Hydrolyzed plant protein*	Farina	Hydrolyzed vegetable protein*
Malt flavoring*	Kasha	Modified food starch*
Matzo meal	Matzo farfel	Oat
Modified starch*	Mono- and diglycerides	Oat gum
Oat groats	Oatmeal	Rusks
Preservatives*	Rye	Starch*
Semolina	Stabilizers*	Vegetable gum*
Textured vegetable protein*	Triticale	Wheat
Wheat flour	Vegetable protein*	Wheat germ oil
Wheat starch	Wheat germ	Whole wheat flour
Wheat stabilizers	White enriched flour	

*Before adding to the diet, verify with manufacturers that ingredients do not include gluten.
Reproduced with permission from Gluten Intolerance Group, 15110 10th Ave. SW, Ste. A, Seattle WA 98166;1987.

content in the diet (Table 26–8), it is important to provide adequate fluids to ensure the fiber's effectiveness.

Not all fiber is equivalent in modifying stool size and consistency. Wheat bran is the most effective in increasing weight of the stool, followed by fruits, vegetables, oats, corn, soya, and pectin.[7] The bulking effect of the fiber is multifactorial, affecting colonic microflora, interaction with intestinal liminal contents, water retention, and other mechanical factors. Fiber should be introduced to the diet slowly and be adjusted based on symptoms. Medications

(eg, stool softeners, laxatives, and/or stimulants) may be required but their use should not obviate the need for a high fiber diet in patients with constipation.

Table 26–6. Additives and Ingredients Allowed in a Gluten-Free Diet

Adipic acid	Ascorbic acid	Butylated hydroxy-anisole (BHA)
Butylated hydroxy-toluene (BHT)	Beta carotene	Biotin
Calcium phosphate	Calcium chloride	Calcium pantothenate
Carboxymethyl-cellulose	Carrageenan	Citric acid
Corn sweetener	Corn syrup solids	Demineralized whey
Dextromaltose	Dextrose	Dioctyl sodium sulfosuccinate
Folic acid-folacin	Fructose	Fumaric acid
Gums: acacia, arabic, carob bean, cellulose, guar, locust bean, tragacanth, xanthan	Invert sugar	Lactic acid
Niacin	Lecithin	Magnesium hydroxide
Potassium citrate	Malic acid	Mannitol
Propylgallate	Microcrystalline cellulose	Monosodium glutamate (MSG)
Sodium acid pyrophosphate	Polyglycerol	Polysorbate 60 and 80
Sodium caseinate	Potassium iodide	Propylene glycol
Sodium nitrate	Pyridoxine hydrochloride	Monostearate
Sucrose	Sodium ascorbate	Riboflavin
Thiamine hydrochloride	Sodium citrate	Sodium benzoate
Vitamins and minerals	Sodium silaco aluminate	Sodium hexametaphosphate
	Sulfosuccinate	Sorbitol
	Tricalcium phosphate	Tartaric acid
	Vitamin A (palmitate)	Vanillin

(*Note:* this is not an exhaustive list.)

Reproduced with permission from Gluten Intolerance Group, 15110 10th Ave. SW, Ste. A, Seattle WA 98166;1987.

Table 26–7. Gluten-Free Diet

Type	*Recommended*	*Not Recommended*
Grains and flours	Almond; arrowroot starch, artichoke; corn starch, cornmeal; maize and waxy maize; legume flours (peas, beans, hung beans, lentils); potato starch, potato flour; rice bran; rice flours (plain, sweet, brown, white, and polished rice); sesame; sorghum; soy flour; sunflower; tapioca (cassava) starch	Low-gluten flours; all flours containing wheat, rye, barley, and oats; durham wheat, all-purpose flour, white enriched flour, wheat flour, wheat germ, whole wheat flour, wheat starch; wheat bran; oat bran; amaranth; buckwheat; buckwheat groats; bulgar; graham; kasha; kumat; matzo; matzo meal; millet; quinoa (kneen-wa); rusks; semolina; spelt; teff; triticale
Breads	Specially prepared breads using only the allowed flours (100% potato, corn, arrowroot, soybean); commercial gluten-free baking mixes	All breads, rolls, etc. made with wheat, oats, barley, and rye
Cereal	Hot or cold cereals made from corn, rice, or hominy*	All cereals containing wheat, oats, barley and rye, farina, bran (except rice), graham, wheat germ, kasha, bulgar, buckwheat, millet, triticale Malt (a flavoring usually derived from barley)
Noodles and pasta	Gluten-free corn pasta; special gluten-free low protein pastas; rice pasta or bean pastas	Pastas, noodles, spaghetti, and macaroni made from wheat or other gluten-containing grains
Crackers and snack foods	Pure cornmeal tortillas; rice wafer or crackers, rice cakes,* popcorn, crackers made with allowed flours (100% potato, corn, rice, arrowroot, soybean); potato chips	All crackers and snack foods containing wheat, wheat starch, rye, barley, oats, bran (except rice), graham, wheat germ, malt, kasha, bulgar, buckwheat, matzo, millet, durham wheat, sorghum, rusks, amaranth, triticale

Table 26–7. continued

Type	*Recommended*	*Not Recommended*
Milk	Fresh, dry, evaporated, or condensed milk; sour cream;† whipping cream;† yogurt†	Malted milk; commercially prepared milkshakes; some nondairy cream;* some commercial chocolate drinks*
Meat and meat alternatives	Fresh meat, fish, poultry, and eggs; fish in oil, water, or brine (check ingredients), luncheon meats, frankfurters, and prepared meat products packaged without food starch or gluten derivatives: peanut butter	Any meat or meat products containing wheat, rye, barley, oats, or gluten derivatives; some canned tuna or fish in vegetable broths;* some sausage, frankfurters, luncheon meats, and sandwich spreads;* canned soups, chilies, stews;* bread containing products such as Swiss steak, meatballs, pot pies, croquettes, etc; self-basting turkeys with hydrolyzed vegetable proteins (HVP) injected as part of the basting solution
Cheese	Aged cheese (100% cheddar, Swiss, Parmesan, etc); cottage cheese,† cream cheese; processed, low fat or fat free cheese*†	Cheese foods; cheese spreads or dips; imitation cheese products
Fruit and juices	Most fresh, frozen, dried, or canned fruit	Thickened or prepared fruits (as in pie fillings)*
Vegetables	Most plain, fresh, frozen, or canned vegetables; dried beans, peas, and lentils; tomato puree and paste; white and sweet potatoes; yams; hominy; rice	Vegetable sauces;* commercially prepared vegetables; most packaged rice, mixes

Fats	Most margarines,* butter, vegetable oil, lard, shortening, nuts, pure mayonnaise made with allowed vinegars*	Commercial salad dressings and dips, unless product ingredients are known to be gluten-free*
Sweets and desserts	Special commercial gluten-free cakes, cookies, and baking mixes; homemade puddings with cornstarch, rice, tapioca; some pudding mixes, gelatin desserts, custards, and ices;* sherbet and ice cream if they do not contain gluten-containing stabilizers;* most hard candy, honey, molasses, marshmallow, coconut, or chocolate;* most jelly and jams;* most nonbuttered syrups;* some candy*	Most commercially prepared cakes, cookies, and other baked goods; "instant" puddings and bread puddings; ice cream cones; frozen desserts containing gluten stabilizers
Miscellaneous beverages	Fruit juice; plain tea; plain brewed coffee; hot chocolate made with pure cocoa powder; carbonated drinks except most root beers; wine and brandy without dyes or preservatives; most rums; vodka distilled from potatoes	"Instant" drinks such as tea, coffee, cocoa, and fruit punch that are processed with additives, stabilizers, or emulsifiers; ground coffee with added grains;* some flavored coffees;* some herbal teas;* most root beers;* all beer and ale; all whiskies (including corn whiskey); bourbon; any liquor made from grain alcohol; vodka distilled from grain
Soups	Homemade broth and soups made with the allowed ingredients; special gluten-free commercial soups or broths	Most canned soups and soup mixes;* bouillon, bouillon cubes or powder

Table 26–7. continued

Type	*Recommended*	*Not Recommended*
Miscellaneous	Cider, rice, or wine vinegar; salt; black or red pepper; herbs; pure spices; monosodium glutamate (MSG) if made in USA; bicarbonate of soda; pure cocoa; most yeast; baking powder; cream of tartar; imitation flavoring	Distilled white vinegar; most white pepper; some curry powders;* some dry seasoning mixes;* some gravy extracts and meat sauces;* yeast flakes;* extracts;* natural flavoring containing alcohol;* ketchup, prepared mustard, and horseradish;* pickles unless cured in allowed vinegars or lemon juice

*Manufacturer should be contacted to confirm gluten status.
†May contain gluten-containing vegetable gums.

Table 26–8. Dietary Fiber Content of Foods

	Little (0 g)	*Low (1 g)*	*Moderate (2 g)*	*High (3 g)*	*Highest (> 4 g)*
Dairy	Milk Yogurt Pudding Ice cream Cheese				
Protein	Eggs Beef Chicken Pork Turkey Fish		*2 tbsp* Peanut butter	*1/2 cup* Garbanzo beans Lima beans	*1/2 cup* Lentils (5 g) Northern beans (4 g) Navy beans (5 g) Pork and beans (6 g) Kidney beans (6 g)
Fruit	Fruit juice Watermelon Cherries	*Fresh* Grapes *1/2 cup canned* Pears Pineapple Fruit cocktail Peaches	*Fresh* 1 peach 3 apricots 1/2 grapefruit *1/2 cup* Applesauce Blueberries Strawberries	*Fresh* 1 apple 1 orange 1 banana 3 dates *1/2 cup* Raspberries	*Fresh* 1 pear (5 g) 1/2 avocado (4 g) 3 plums (4 g) 3 prunes (4 g)

Table 26–8. continued

	Little (0 g)	*Low (1 g)*	*Moderate (2 g)*	*High (3 g)*	*Highest (> 4 g)*
Fruits (con't)				*1/4 cup* Raisins Dried peaches Apricots Apples	
Vegetables		*1/2 cup* Tomato juice Lettuce Spinach Celery Cauliflower Cucumber Green beans	*1/2 cup* Tomato Cabbage	*1/2 cup* Sweet potato Broccoli Carrots Peas Potato salad Corn	1 baked potato with skin (4 g)
	Little (< 0.5 g)	*Low (1 g)*	*Moderate (2 g)*	*High (3 g)*	*Highest (> 4 g)*
Breads and Cereals	***Bread*** *1 slice* French bread Italian bread Raisin bread White bread	***Bread*** *1 slice* Cracked wheat Pumpernickel Rye	***Bread*** *1 slice* 100% whole wheat	***Bread*** *1 slice* Granola	***Bread*** *1 slice* Flourless breads (5 g)

1 each	*1 each*		1 bran muffin	
Pancake	Tortilla			
Doughnut	Whole wheat			
1/2 bagel	pancake			
Cereals	***Cereals***	***Cereals***	***Cereals***	***Cereals***
1/2 cup	*1/2 cup*	*1/2 cup*	*1/2 cup*	*1/2 cup*
Corn Flakes	Oatmeal	Shredded wheat	Bran flakes	100% bran (9 g)
Frosted Flakes	Life	Granola	Raisin Bran	All Bran (9 g)
Lucky Charms	Nutrigrain	Crispy Wheats 'n	Grapenuts	Fiber 1 (12 g)
Cheerios	Wheaties	Raisins	Wheat germ	
	Total	Wheat Chex		*1/4 cup*
	Honey Nut			Unprocessed wheat
	Shredded Wheat			bran (7 g—2 g/tbsp)
Pasta 1/2 cup	***Pasta 1/2 cup***	***Pasta 1/2 cup***		***Pasta 1/2 cup***
Macaroni	Egg noodles	Brown rice		Whole wheat
	White rice			
Crackers	***Crackers***	***Crackers***	***Crackers***	***Crackers***
Goldfish	2 graham	3 Harvest Wheats	1 rye crisp	Metamucil wafers
Saltines	16 Wheat Thins	3 Triscuits		
Ritz	1 granola bar			

Table 26–8. continued

	Little (< 0.5 g)	*Low (1 g)*	*Moderate (2 g)*	*High (3 g)*	*Highest (> 4 g)*
Desserts	Chocolate chip cookies	Oatmeal cookies	Fig Newtons Peak Freans Bran Crunch (3 g)		
Miscellaneous	Beverages Fats Sweets	1 cup popcorn	*1/4 cup* Cashews Pecans	*1/4 cup* Almonds Peanuts Walnuts	*1/4 cup* Coconut

Adapted from Hendricks KM, Walker WA. Pediatric Nutrition. 2nd ed. Philadelphia: B.C. Decker, Inc.; 1990.

Acute Diarrhea

In the United States, acute diarrhea remains one of the most common reasons for outpatient physician visits, with more than 220,000 admissions for acute diarrhea annually.[8] Appropriate nutritional therapy for acute diarrhea consists of rehydration with a commercially available oral rehydration solution (Tables 26–9 and 26–10), replacement of excess fluid losses, and continued feeding. Compared to gradual reintroduction of food, continued feeding during diarrhea results in reduced duration of illness and better weight gain.[9] Although children with acute diarrhea commonly suffer anorexia, their regular diet should be offered as much as possible during illness (Table 26–11). High fat foods may malabsorbed after acute diarrhea as steatorrhea has been described for several days following improvement of symptoms. Drinks high in simple carbohydrates, such as juices and sodas, may also be poorly tolerated due to the osmotic loads of such nutrients. Most important, highly restricted diets should be avoided so that a cycle of weight loss and persistent diarrhea does not ensue.

Undiluted cow's milk and cow's milk-based formulas can safely be given to most children with acute diarrhea although a minority of patients will have clinically significant lactose intolerance (see Lactose Intolerance, below). Children with malnutrition or severe dehydration are more likely to have lactose intolerance[10] and would probably benefit from a diet reduced in lactose. Other children whose stool outputs increase significantly while ingesting lactose-containing foods may also be placed on a lactose-free diet. Soy polysaccharide added to infant formula increases stool consistency but does not decrease nutrient or water loss in the stool.[11]

Table 26–9. Commercially Available Oral Rehydration Solutions

Name (Manufacturer)	*Carbohydrate (g/L)*	*Na (mEq/L)*	*Base (mEq/L)*	*K (mEq/L)*	*Osmolality (mmol/L)*	*Comments*
ORS (Jaianas)	20	90	30	20	311	Solution recommended by the World Health Organization
Equalyte (Ross)	30	78	30	22	290	Fructo-oligosaccharides and dextrose are carbohydrate source
Rehydralyte (Ross)	25	75	30	20	310	
CeraLyte 70 (Cera Products)*	40	70	30	20	235	Whole cooked rice is carbohydrate source
KaoLectrolyte (Breckenride Pharmaceutical, Inc.)	21	50	30	20	232	1 packet makes 8 oz
Enfalyte (Mead Johnson)	30	50	34	25	200	Rice-syrup solids are carbohydrate source
Pedialyte (Ross)	25	45	30	20	250	
Pediatric Oral Maintenance Solution (NutraMax)	25	45	30	20	250	Marketed under several different generic titles

*CeraLyte is also available in CeraLyte 50 and CeraLyte 90.

Table 26–10. Oral Therapy for Acute Diarrhea[7]

Degree of Dehydration	*Rehydration Therapy*	*Replacement of Ongoing Stool and Vomit Losses*	*Diet*
None	None required	4–8 oz of ORS for each watery stool; increased dietary fluids to prevent dehydration	Regular diet for age, including breast milk or full-strength formulas for infants and complex carbohydrates, fresh fruits, vegetables, and lean meats for older children
Mild to moderate	60–80 mL/kg of ORS given over 4 hours with periodic re-evaluation	As above	As above
Severe	20 mL/kg bolus(es) of Ringer's lactate by IV route until perfusion and mental status improve, then 60–80 mL/kg of ORS, with periodic re-evaluation	As above	As above; N.B.: children with a history of severe dehydration may have a higher likelihood of lactose intolerance during recovery

ORS = oral rehydration solution (see Table 26–9).

Table 26–11. Nutritional Management of Acute Diarrhea

Timing of feeding
As soon as rehydration has occurred
Components of feeding
Breastmilk ad libitum
Full-strength cow milk or cow milk formula (if monitored for signs of malabsorption)
Complex carbohydrates (rice, wheat, potatoes, bread, cereal)
Lean meats (eg, chicken)
Yogurt, fruits, vegetables
Avoid foods high in fats or simple sugars
Avoid highly restrictive diets

Adapted from Provisional Committee on Quality Improvement Subcommittee on Acute Gastroenteritis. Practice parameter: the management of acute gastroenteritis in young children. Pediatrics 1996;97:424–56.

Persistent Diarrhea

An episode of diarrhea lasting for more than 14 days is termed persistent, or chronic, diarrhea. In the world's developing countries, the cycle of persistent diarrhea, malabsorption, anorexia, and malnutrition is one of the leading causes of death in children under the age of 5 years. Nutritional and medical management of these patients centers on treating infection, correcting acidosis and dehydration, gradually reintroducing enteral nutrition, and correcting micronutrient deficiencies. Culturally acceptable and cost-effective formulas have been employed with great success.[12]

In industrialized countries, diarrhea and malnutrition should prompt an evaluation of the patient for malabsorption or systemic illness (Table 26–12). In cases of persistent diarrhea resulting from extensive gastrointestinal mucosal disease (eg, allergic disease, celiac disease, or

other flat gut lesions), nutritional management may be undertaken with a wide range of enteral formulas. These are generally lactose-free (to avoid lactose malabsorption), protein hydrolysate or peptide-based (to treat possible protein sensitivities), and isotonic (to prevent osmotic loads worsening diarrhea) (see Chapter 16, Enteral Nutrition). Parenteral nutrition is occasionally indicated but every effort should be made to use the enteral route first.[13]

Chronic Nonspecific Diarrhea

Chronic, nonspecific diarrhea (CNSD), often called toddlers' diarrhea, is characterized by two or more loose, odoriferous, voluminous stools per day, and generally occurs in infants aged 6 to 36 months. The diarrhea lasts longer than 4 weeks and is not associated with significant abdominal pain, fever, or growth failure. Its etiology is unknown although it may be initiated by an acute infection.

Nutritional management of CNSD (Table 26–13) centers on normalizing the diet as much as possible to avoid iatrogenic malnutrition. The prognosis of CNSD is excellent, with most children improving by the age of 4 years.

Table 26–12. Persistent Diarrhea in Industrialized Countries

Weight Loss Present	*Normal Nutritional Status*
Cystic fibrosis or other causes of pancreatic insufficiency	Infection (eg, giardia)
Hepatobiliary diseases	Lactose intolerance
Protein-losing enteropathy	Chronic nonspecific diarrhea
Crohn's disease or ulcerative colitis	Excessive juice intake
AIDS or other immunodeficiencies	
Celiac disease or allergic disorders	

Table 26–13. Dietary Manipulations of Chronic Nonspecific Diarrhea

Normalize diet, including milk and milk products
Limit fruit juice
Fat content of diet should be at least 35% of total calories
Avoid excessive fluid intake
Add fiber to diet
Increase fiber in diet
Psyllium bulk agents can be added if diarrhea is persistent 2–3 tbsp bid for 2 weeks[14]

Lactose Intolerance

Lactose intolerance refers to gastrointestinal symptoms resulting from an inability to digest lactose, the main carbohydrate in milk. Intolerance may include a variety of symptoms, including abdominal pain, bloating, diarrhea, and flatulence. Lactose malabsorption is attributed to a relative deficiency of the disaccharidase lactase, which can be primary (ie, a genetic deficiency) or secondary (ie, due to mucosal disease). Since most individuals produce less lactase after being weaned, one could actually describe primary lactase deficiency after the age of 2 years as the normal pattern, with others having abnormal persistence of lactase production. Lactose deficiency before the age of 6 months is highly unusual. In addition to the presence or absence of the lactase enzyme, other factors determining whether a person will have symptoms of lactose malabsorption include the amount of lactose in the diet, the mixture of lactose with other foods, gastric emptying rate, colonic scavenge of malabsorbed carbohydrate, race, ethnic origin, and age.

Lactose intolerance is treated by removing lactose from the diet (Table 26–14). In cases of primary lactose intoler-

ance, this must be a permanent dietary change. Lactose is a common ingredient in many foods, including breads, crackers, soups, cereals, cookies, granola bars, chocolate, candy, salad dressings, luncheon meats, and baked goods. Eliminating or reducing the lactose-containing ingredients will usually be adequate to alleviate symptoms. In cases of possible secondary lactose intolerance, it is recommended that all lactose be eliminated from the diet for a short period of time (2 to 6 weeks). If symptoms resolve, lactose may be slowly reintroduced into the diet as tolerated by the individual. The amount of lactose an individual can tolerate varies. Many children can tolerate small amounts of lactose without discomfort, especially in the form of yogurt and hard cheeses (as opposed to milk or ice cream). Since approximately 25 percent of adults in the United States and a higher percentage worldwide are lactose intolerant, there are a variety of lactose-free and low lactose food choices available (Table 26–15). Lactase-treated products, containing 70 to 100% less lactose than standard foods, are commercially available. Many adults who consider themselves lactose-intolerant can in fact tolerate moderate amounts of milk.[15]

Children maintained on a strict lactose-free diet may not meet their recommended calcium and vitamin D needs. Supplementation with one of the products listed in Table 26–16 is recommended.

Table 26–14. Lactose-Containing Ingredients

Cheese	Margarine
Cream	Milk
Curds	Nonfat dry milk powder
Dry milk solids	Skim milk powder
Ghee	Whey
Lactose	

Table 26–15. Low Lactose Foods High in Calcium

Food Item	*Mg of Calcium/Serving*
Milk/dairy products	
Lactose-free milk, 1 cup	300
Cheddar cheese, 1 oz	200–260
Sherbet, 1/2 cup	25
Protein	
Dried beans, 1 cup	90
Cod, 3 oz	136
Soybeans, 1/2 cup	130
Tofu, 1/2 cup	434
Vegetables	
Broccoli, 1/2 cup	89
Spinach, 1/2 cup	61
Fruits	
Orange juice (calcium-fortified) 8 oz	300–450
Prunes, 4	49
Miscellaneous	
Black strap molasses, 1 tbsp	137

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) refers to Crohn's disease or idiopathic ulcerative colitis. Approximately 25,000 new cases are diagnosed each year in the United States, with 20 to 30% of them being children.[16] Whereas ulcerative colitis involves the colon, Crohn's disease can involve the entire gastrointestinal tract, including the colon, small intestine, stomach, esophagus, and mouth. Both forms of IBD may present with bloody diarrhea but Crohn's disease of the small intestine is more likely to be preceded by weight loss due to malabsorption and anorexia. Growth failure and nutrient deficiencies are common complications in IBD occurring during childhood and adolescence. Factors contributing to the nutritional complications in

Table 26–16. Over-the-Counter Calcium Supplements

Product	*Type of Calcium*	*Manufacturer*	*Mg Calcium/ Tablet*
Citrate	Calcium citrate	Mission Pharmacal	200
Caltrate + Vitamin D	Calcium carbonate	Lederle Lab	600
Os-Cal 500 chewable	Calcium carbonate	Marion Lab	500
Tums	Calcium carbonate	Smith-Kline Beecham	200
Tums 500	Calcium carbonate	Smith-Kline Beecham	500
Rolaids	Calcium carbonate	Warner-Lambert	400

pediatrics include inadequate dietary intake, weight loss, malabsorption, increased nutrient requirements, and long-term corticosteroid use. Significant drops in height velocity have been documented in children with IBD in the months before diagnosis.[17]

Management of children with IBD often requires a combination of nutritional therapy, pharmacologic agents, surgical consultation, and psychologic intervention.[19] The main goals of nutritional therapy are to provide adequate calories, vitamins, and minerals and to correct specific nutrient deficiencies (Table 26–17). Controlled studies have shown that the use of elemental (hydrolyzed protein) diets are as effective as corticosteroids in inducing remission in Crohn's disease.[20] Because of bad taste, difficulty with compliance, and the expense of elemental diets, more recent studies have examined the role of polymeric (whole protein) enteral diets and have found that they can also be effective in treating Crohn's disease.[19]

Occasionally, a low fiber or lactose-restricted diet may be necessary when reintroducing food after a Crohn's flare, depending on the child's tolerance. Restrictions

Table 26–17. Nutritional Recommendations and Supplementation for the Pediatric Patient with Inflammatory Bowel Disease

Routine Supplementation	*Comments*
Energy and Protein	100–150% of USRDA for height age
Vitamins and Minerals	One age-appropriate multiple vitamin with minerals per day
Therapeutic Supplementation	
Calcium	500–1,300 mg/d; routine Ca+ supplementation is probably indicated with steroid use
Iron	2–4 mg elemental Fe/kg/d
Zinc	100–200% USRDA; stool losses may be high during acute exacerbations
Vitamin B_{12}	100 µg IM every 3 months if terminal ileum was lost due to surgery or disease
Folate	100–200% USRDA; sulfasalazine may interfere with metabolism

IM = intramuscular; USRDA = United States Recommended Dietary Allowance.

should be made on an individual basis, however. Minor food sensitivities and intolerances may occur during a flare but generally do not persist and are not sufficiently important to warrant elimination from the child's diet.[20]

Nutritional support and monitoring are essential in the pediatric patient with IBD to minimize malnutrition and growth failure. The use of specialized enteral formulas versus pharmaceutical therapy will continue to be widely debated. In many parts of the world, enteral diets are the treatment of choice for children with Crohn's disease and should be considered when medically appropriate.

References

1. Walker-Smith JA. Celiac disease. In: Walker WA, Durie PR, Hamilton JR, et al. Pediatric gastrointestinal disease. St. Louis: Mosby; 1996.
2. Amil DJ, Walker-Smith J. Down's syndrome and coeliac disease. J Pediatr Gastroenterol Nutr 1990;10:41–3.
3. Branski D, Troncone R. Celiac disease: a reappraisal. J Pediatr 1998;133:181–7.
4. Trier JS. Celiac sprue. N Engl J Med 1991;325(24):1709–19.
5. Kumar PJ, Farthing MG. Oats and celiac disease. N Engl J Med 1995;333(16):1075–6.
6. Williams CL, Bollella M, Wynder EL. A new recommendation for dietary fiber in childhood. Pediatrics 1995;96(5):985–8.
7. Cummings JH. Constipation, dietary fibre and the control of large bowel function. Postgrad Med J 1983;1:1206.
8. Duggan C, Santosham M, Glass RI. Centers for Disease Control and Prevention. The management of acute diarrhea in children: oral rehydration, maintenance, and nutritional therapy. MMWR 1992;41(No. RR-16):1–20.
9. Duggan C, Nurko S. Feeding the gut: the scientific basis for continued enteral nutrition during acute diarrhea. J Pediatr 1997;131:801–8.
10. Brown K, Peerson J, Fontaine O. Use of nonhuman milks in the dietary management of young children with acute diarrhea: a meta-analysis of clinical trials. Pediatrics 1994;93:17–27.
11. Brown K, Perez F, Peerson J, et al. Effect of dietary fiber (soy polysaccharide) on the severity, duration, and nutritional outcome of acute, watery diarrhea in children. Pediatrics 1993; 92:241–7.
12. International Working Group on Persistent Diarrhea. Evaluation of an algorithm for the treatment of persistent diarrhea: a multicentre study. Bull World Health Organ 1996; 74:479–89.
13. Orenstein SR. Enteral versus parenteral therapy for intractable diarrhea of infancy: a prospective, randomized trial. J Pediatr 1986;109(2):277–86.
14. Klish WJ. Chronic diarrhea. In: Walker WA, Durie PR, Hamilton JR, et al. Pediatric gastrointestinal disease. 2nd ed. St. Louis: Mosby; 1996.

15. Suarez FL, Savaiano DA, Levitt MD. A comparison of symptoms after the consumption of milk or lactose-hydrolyzed milk by people with self-reported severe lactose intolerance. N Eng J Med 1995;333:1–4.
16. Bousvaros A. Inflammatory bowel disease. Int Semin Paediatr Gastroenterol Nutr 1997 June;6(2):2–3.
17. Kanof ME, Lake AM, Bayless TM. Decreased height velocity in children and adolescents before diagnosis of Crohn's disease. Gastroenterology 1988(95);6:1523–7.
18. O'Morain C, Segal AW, Levi AJ. Elemental diet as primary treatment of acute Crohn's disease: a controlled trial. BMJ 1984;288:1859–62.
19. Riguard D, Cosnes J, Le Quintrec Y, et al. Controlled trial comparing two types of enteral nutrition in treatment of active Crohn's disease: elemental v polymeric diet. Gut 1991;32: 1492–7.
20. Pearson M, Teahon K, Levi AJ, Bjarnason I. Food intolerance and Crohn's disease. Gut 1993;34;783–7.

Additional Resources

Celiac Disease

Support Groups

American Celiac Society Dietary Support Coalition
Contact: Annette Bentley
58 Musano Court, West Orange, NJ 07052-4114
201-325-8837
E-mail: bentleac@umdnj.edu

Celiac Sprue Association
Contact: Leon Rottman
P.O. Box 31700, Omaha, NE 68131-0700
402-558-0600
President: Janet Rinehart, 713-783-7608
E-mail: 76131.2257@compuserve.com
Website: http://members.aol.com/celiacusa/celiac.htm

Celiac Disease Foundation
Contact: Elaine Monarch
13251 Ventura Blvd., Suite 3, Studio City CA, 91604-1838
818-990-2379
E-mail: Group: cdf@celiac.org

Gluten Intolerance Group of North America
PO Box 23053 Seattle, WA 98102
206-325-6980
E-mail: Cynthia R Kupper: c.kupper@juno.com

Lactose Intolerance

Resources

Zurkin J. The Newsletter for People
with Lactose Intolerance and Milk Allergy
Commercial Writing Service
P.O. Box 3074, Iowa City IA, 52244
319-351-1354.

Lactaid: 1-800-LACTAID
1-800-Why-Milk, information hotline

Inflammatory Bowel Disease

Support Groups

Canadian Foundation of Ileitis and Colitis
21 St. Clair Ave East, Suite 301
Toronto, ON M4T IL9
Canada
416-920-5035
Fax: 416-929-0364

Crohn's and Colitis Foundation of America
444 Park Ave. South
New York, NY 100616
212-685-3440
800-343-3637
Fax: 212-779-4098

27

GROWTH FAILURE

Kattia M. Corrales, RD, and Jan P. Hangen, MS, RD

Growth failure, also known as failure to thrive (FTT) or malnutrition, is a term used to describe children who demonstrate inadequate physical growth or are unable to maintain the expected rate of growth over time (Table 27–1). It is often identified in the first 3 years of a child's life. Growth failure is prevalent among urban and rural families living in poverty but can be seen across all socioeconomic strata.[1]

Growth is best assessed longitudinally, as a single point on the growth chart will not reflect growth fluctuations or a child's growth pattern. Birth weight percentile is often used to estimate the child's expected growth pattern. The maximum weight percentile achieved by a child between 4 and 8 weeks of age, however, is a better predictor of the percentile at 12 months of age than is birth weight percentile.[2] Infants normally double their birth weight by age 4 months

Table 27–1. Diagnostic Criteria for Growth Failure

Delayed Growth	*Decreased Growth Velocity*
Weight for age less than 5th percentile on the NCHS growth chart	Weight decreasing more than two major percentiles or two standard deviations over a 3 to 6 month period
Weight for height less than 5th percentile on the NCHS growth chart	

NCHS = National Center for Health Statistics.

and triple it by 12 months. Table 27–2 shows mean incremental weight and height data for healthy children in the United States. Serial data from studies of infants at the

Table 27–2. Mean Increments in Weight and Length*

	Weight (g/d)		*Length (mm/d)*	
Age (mo)	*Boys*	*Girls*	*Boys*	*Girls*
Up to 3	31	26	1.07	0.99
1–4	27	24	1.00	0.95
2–5	21	20	0.84	0.80
3–6	18	17	0.69	0.67
4–7	16	15	0.62	0.60
5–8	14	14	0.56	0.56
6–9	13	13	0.52	0.52
7–10	12	12	0.48	0.48
8–11	11	11	0.45	0.46
9–12	11	11	0.43	0.44
10–13	10	10	0.41	0.42
11–14	10	10	0.39	0.40
12–15	9	9	0.37	0.38
13–16	9	9	0.36	0.37
14–17	8	9	0.35	0.36
15–18	8	8	0.33	0.34
16–19	8	8	0.32	0.33
17–20	8	8	0.31	0.32
18–21	7	8	0.30	0.32
19–22	7	7	0.30	0.31
20–23	7	7	0.29	0.30
21–24	7	7	0.28	0.29

*From birth through 3 months, Iowa data; from 3 through 6 months, combined data; from 6 through 24 months, Fels data.
Reproduced with permission from Guo S, Roche AF, Fomon SJ, et al. Reference data on gains in weight and length during the first two years of life. J Pediatr 1991;119:355–62.

University of Iowa and from the Fels Longitudinal Study were used to develop these tables.

Growth failure should be differentiated from genetic short stature, constitutional growth delay, intrauterine growth retardation (IUGR), and normal shifting. Normal shifting of growth percentiles may occur during the first 2 years of life due to genetic adjustment.[3] In these cases, body weight tends to remain proportional to height and bone age equals chronologic age. In constitutional growth delay, deceleration of growth can occur in the first 2 years of life and later normalize.[4] Intrauterine growth retardation refers to a heterogeneous group of children who failed to grow in utero due to a variety of factors and whose postnatal growth patterns are diverse. Infants with asymmetric IUGR (weight affected more than height and/or head circumference) have a better potential for catch-up growth than those with symmetric IUGR (weight, height, and head circumference equally affected).[5] Breastfed infants show a greater growth velocity than formula-fed infants during the first 6 months of life but have lower rates of weight gain from 6 to 12 months of life.[6]

Etiology

Children with growth failure have not taken in, have not been offered, or have not retained adequate calories to meet their nutritional needs.[1] Growth failure is often classified as nonorganic or organic. Nonorganic growth failure is usually psychosocial in nature, including disordered maternal-infant bonding, disordered feeding techniques, poor feeding interactions, and failed breastfeeding (Table 27–3).

Organic risk factors for growth failure are generally thought to involve the inability to obtain or retain adequate calories, as in the case of oral-motor difficulties, malabsorption, or maldigestion. Organic risk factors may also be secondary to increased caloric requirements, as in

congenital heart disease, or to altered growth potential resulting from factors such as fetal alcohol syndrome.[7]

Some clinicians view the dichotomy between nonorganic and organic growth failure as misleading. While it is impor-

Table 27–3. Risk Factors in Growth Failure

Nonorganic growth failure
Disordered maternal-infant bonding
Incorrect formula preparation
Failed breastfeeding
Underfeeding
Excessive juice intake, especially if juice displaces foods more dense in calories and nutrients
Delayed introduction of solids
Intolerance of new foods
Coercive feeding
Distractions at mealtime
Psychosocial stressors, including:
Divorce, death, drug abuse, violence, neglect, food withholding, new home, new siblings, homelessness, inadequate medical care, unusual health and nutrition beliefs in the family (eg, fear of obesity, belief in exclusivity of breastfeeding for longer than recommended)
Organic growth failure
Acquired illness
IUGR
Congenital syndromes
Teratogenic exposures
Milk-protein allergies/intolerances
Celiac disease
HIV infection
Cystic fibrosis
Congenital heart disease
Gastroesophageal reflux
Metabolic, chromosomal, or anatomic abnormalities

IUGR = intrauterine growth retardation; HIV = human immunodeficiency virus.

tant to identify both physiologic and psychosocial factors in the diagnosis of growth failure, a distinct division between the two may not be beneficial in planning treatment. For example, children with medical conditions such as congenital heart disease not only have increased caloric requirements but may also develop secondary feeding problems that lead to psychosocial disorders in the feeding environment. Children with fetal alcohol syndrome may have both reduced potential for growth and a disordered social situation. In summary, it is important to consider all diagnostic considerations when planning the treatment protocol for the child with growth failure.

Another classification of feeding disturbances is based on both the stages of infant development and the concepts of separation and individuation. Within this context, three separate stages of feeding development are identified: homeostasis, attachment, and separation and individuation.[8] During the period of homeostasis, the infant learns to regulate sucking, swallowing, and the termination of feeding with signals of hunger and satiety. Failure to master these skills leads to problems with attachment, the next stage of development. In the attachment period, the infant develops interactional patterns with his/her caretakers, specifically appetite and pleasurable eating. Lack of pleasure or appetite in the feeding process may lead to dysfunctional behaviors such as vomiting or rumination. Feeding problems developing in the third phase may lead to an inability to distinguish between physiological and emotional needs with respect to feeding (Table 27–4).

Nutritional Management

Nutrition services should take place in the context of multidisciplinary care that addresses physiologic, nutritional, and social factors related to undernutrition. The treatment team should therefore include a physician and/or nurse

Table 27–4. Classification of Feeding Disorders in Infants and Children with Growth Failure

Disorder Type	*Age of Onset*	*Possible Causes*	*Features of Infant*	*Features of Caretaker*	*Treatment*
Homeostasis	0–2 months	Limited experience with oral feeds (eg, respiratory distress)	Excitable Irritable Passive	Anxious Depressed Over or understimulates infant	Pacifier during nasogastric feeds Occupational therapy re: suck and swallow
Attachment	2–6 months	Prolonged hospitalization or separation from mother Developmental delay	Sad Hypervigilant Arches or resists when picked up	Detached Depressed Holds infant loosely	Emotional nurturance Developmental stimulation Education of caretaker re: needs of infant
Individuation or separation	6 months to 3 years	Any condition that limits or restricts food intake (eg, diabetes, celiac disease)	Refuses food Defiant Plays with food	Frustrated Doesn't allow infant to self-feed	Regularly scheduled mealtimes Separate mealtimes from playtimes Encourage self-feeding

Reproduced with permission from Chatoor I, Dickson L, Shaefer S, Egan J. A developmental classification of feeding disorders associated with failure to thrive: diagnosis and treatment. In: Drotar D, editor. New directions in failure to thrive: implications for research and practice. New York: Plenum Press; 1985.

practitioner, a social worker and/or psychologist, and a nutritionist. Within this context, the nutritional management of undernutrition comprises three components:

1. Nutrition assessment (Table 27–5)
2. Provision of nutrients to meet catch-up growth requirements (Table 27–7 and 27–8)
3. Concrete and individualized nutritional instruction

The nutritional assessment of the child with growth failure involves evaluating the child's growth pattern, extent of malnutrition, and factors that may be affecting nutrient intake. In the case of severe undernutrition, a nutritionist should be consulted early so that consequences from refeeding syndrome can be avoided. For mild to moderate undernutrition, ad libitum oral feedings are appropriate, and caregivers should be advised to increase the caloric intake by increasing the caloric density of both liquids and solids (Table 27–8). The 24-hour recall can provide the basis for improving the diet.

Table 27–5. Special Aspects of Nutritional Assessment in Growth Failure

Review medical history
- Diagnosis
- Medications and vitamin supplementation
- Bone age
- Stool studies: O and P, reducing substances, malabsorption
- Sweat test to rule out cystic fibrosis
- Immunology
- Laboratory data: extensive laboratory work-up is rarely needed in growth failure unless indicated by history and/or physical examination
  - Hgb, Hct, iron studies if indicated; lead, zinc, albumin, or prealbumin

Assess nutrient intake
- Refer to Chapter 1, Nutritional Assessment: Dietary Evaluation

Table 27–5. continued

Assess growth
- Refer to Chapter 2, Nutritional Assessment: Anthropometrics and Growth

Estimate catch up growth needs
- See Table 27–6

Behavioral/feeding assessment
- Questions to ask:*
- Who eats with the child (family, siblings, and other children)? Are they role models for healthy eating patterns?
- What types of food and beverages are available? Note serving size, textures, child appeal, variety, and quantity.
- When are meals and snacks offered? Are they offered on a schedule? How long does it take the child to eat?
- Where are meals (at home, daycare, school, in kitchen, playground, on floor, in front of television)?
- Why does the child express hunger or satiety? Is there positive or negative feedback at meals? Does the child ask for seconds and/or request particular foods?
- How do others participate with the meal or feeding of the child? Is there force feeding, bribing, rewarding? Does the child communicate his/her wants and needs? How?

Feeding observation
- Parental behaviors
  - Anxious, inattentive to child, force feeding, bothered by food messes, does not allow child to participate in feeding, props bottle, ignores child's feeding signals
- Child behaviors
  - Cries, spits up, gags, vomits or ruminates, holds food in mouth, arches, plays with food or toys at mealtime, takes > 30 minutes to eat, refuses to stay seated

O and P = ova and parasites.

*Adapted from Tougas L. Nutritional assessment and management of the child with failure to thrive. Presentation for Pediatric Nutrition Conference; 1996; Children's Hospital, Boston.

Table 27–6. Estimating Catch-Up Growth Needs in Growth Failure*

Calorie needs (range):

$$\frac{\text{RDA for energy} \times \text{ideal weight for height (kg)}}{\text{Actual weight (kg)}} \quad \text{to} \quad \frac{\text{RDA for energy} \times \text{ideal weight for age (kg)}}{\text{Actual weight (kg)}}$$

Method

1. Determine the child's Recommended Daily Allowances (RDA) for age
2. Determine the child's ideal weight for height or ideal weight for age (50th percentile on the NCHS growth charts)
3. Multiply RDA for age by ideal weight
4. Divide by the child's actual weight

Protein needs:

$$\frac{\text{RDA protein for age} \times \text{ideal weight for height (kg)}}{\text{Actual weight}}$$

Method

Same as above

NCHS = National Center for Health Statistics.
*Catch-up growth: a period of accelerated growth; formulas estimate calorie requirements to reach ideal weight.
Alternate formula: 120 kcal/kg × ideal weight for actual height/actual weight (kg).

Table 27–7. Special Aspects of Nutritional Management in Growth Failure

1. Provision of energy and protein needs to meet the requirements for catch-up growth

   Infants

Formula:	Concentrate formula: using carbohydrate, protein, or fat modulars (see Chapter 16)
Solids:	Offer high calorie, high protein baby foods (eg, cheese, ground meats)
Juice:	Discourage juice

   Toddlers

Formula:	Increase the caloric density of liquids Concentrate whole milk using carbohydrate, protein, or fat modulars Offer nutritional supplements (eg, 30 kcal/oz formula—see Chapter 16)
Solids:	Offer high calorie, high protein foods (see Table 27–8)
Juice/soda:	Limit juice to a maximum of 4 oz/d Discourage soda

2. Feeding strategies
   a. Establish a regular schedule of meals and snacks, every $2\frac{1}{2}$ to 3 hours
   b. Be consistent with the daily meal and snack schedule
   c. Restrict food and beverages to meal and snack times only, offering water between feedings
   d. Set limits on the amount of time allowed for meals and snacks, usually 20–30 minutes
   e. Make foods and textures appropriate. For example, toddler portions are about $\frac{1}{3}$ to $\frac{1}{4}$ of an adult portion size of food
   f. Provide comfortable seating that gives support and try to confine meals to one general setting, eg, the kitchen table
   g. Reinforce good eating behavior with praise and positive reinforcement but do not concentrate too much on eating itself
   h. Have all foods ready before the child is seated
   i. Limit distractions (eg, toys, radio, television, video, etc)
   j. Be a good role model for eating behavior
   k. Serve a variety of foods and textures
   l. Include foods that the child likes and introduce new foods slowly

Table 27–7. continued

3. Additional interventions
   Identify food resources if needed, eg, WIC, Food Stamps, and food pantries
   Identify support systems to minimize parental stress if needed, eg, childcare, counseling
   Parenting classes, especially for younger parents
   Hospitalization
   Appetite stimulants
   NG feedings

WIC = Supplemental Program for Women, Infants and Children; NG = nasogastric.

Table 27–8. High Calorie, High Protein Diet

Dairy products

Powdered milk, half and half, evaporated milk: add to whole milk, yogurt, casseroles, milk shakes, bread, muffins, cookies, sauces, gravies, and cream soups. Can also use sweetened condensed milk in desserts or shakes (eg, 1 tbsp per 6–8 oz of liquid)

Yogurt: use in fruit, desserts, pancakes, waffles, muffins, and cereals

Cream or cottage cheese and/or sour cream: add to casseroles, potatoes, vegetables, rice, pasta, bread, and crackers (eg, 1–2 tbsp per cup)

Cheese: add to sandwiches, meat, potatoes, salads, vegetables, pasta, rice, and cream sauces

Butter and margarine: add to bread, grains, cereals (especially hot cereal), pancakes, waffles, casseroles, and vegetables (eg, 1–2 tsp per slice of bread or per $^1/_2$ cup cereal)

Pudding, cocoa, milk shakes, cream soup, custard, eggnog can be offered as snacks or desserts

Protein group

Cooked meats, fish, poultry or eggs: add to salads, casseroles, soups, vegetables, omelets, pasta, rice, and noodles

Eggs: add to French toast or pancake batter, custards, puddings, and cakes

Table 27–8. continued

Peanut butter and other nut butters: spread on breads and crackers, fruits and vegetables, or blend in ice cream or yogurt

Nuts: can be added to desserts, salads, ice cream, puddings, vegetables and fruits

Textured vegetable protein: can be used in casseroles, soups, pasta, rice, or noodles

Fruits and vegetables

Mashed fruit: add to milk, yogurt, shakes, ice cream, and pudding

Jell-O: can be made with juice instead of water

Honey or syrup: add to fruit in natural or sweetened juice

Dried fruits: in muffins, cookies, pancakes, waffles, cereals, or other grains

Vegetables: in sauces, soups, and casseroles. Can also be fried

Grains

Hot cereals: can be made with juice, milk, or fortified milk instead of water

High protein noodles and grains: can be used in casseroles and soups

Meat: can be breaded or floured before cooking

Whole grain desserts: eg, oatmeal, raisin bran, or peanut butter cookies

Granola: can be added to ice cream, yogurt, pudding, or other desserts

Caution is advised, however, as dietary recalls often under- or over-report actual intake. Foods with minimal nutritional value should be discouraged in favor of higher calorie meals and snacks. Juice and other sugary beverages have been implicated in growth failure and should be discouraged.[9] A multivitamin preparation including both iron and zinc should be incorporated into the nutritional management.[1,10]

References

1. Bithoney WG, Dubowitz H, Egan H. Failure to thrive/growth deficiency. Pediatr Rev 1992;13:453–9.
2. Edwards AGK, Halse PC, Parkin JM, Waterson AJR. Recognizing failure to thrive in early childhood. Arch Dis Child 1990;65:1263–5.
3. Smith DW, Truog W, Rogers FE, et al. Shifting linear growth during infancy: illustration of genetic factors in growth from fetal life through infancy. J Pediatr 1976;89:225–30.
4. Horner JM, Thorsson AV, Hintz RL. Growth deceleration patterns in children with constitutional short stature: an aid to diagnosis. Pediatrics 1978;62:529–34.
5. Albertson-Wickland K, Karlberg J. Natural growth in children born small for gestational age with and without catch-up growth. Acta Paediatr Suppl 1994;339:64–70.
6. Dewey KG, Heinig MJ, Nommsen LA, et al. Growth of breastfed and formula-fed infants from 0 to 18 months: the DARLING study. Pediatrics. 1992;89:1035–41.
7. Roseann D, Loeb L, Jura M. Differentiation of organic from nonorganic failure to thrive syndrome in infancy. Pediatrics 1980;66:689–92.
8. Chatoor I, Schaffer S, Dickson L, Egan J. Non-organic failure to thrive: a developmental perspective. Pediatr Ann 1984;13:829–43.
9. Dennison BA, Rockwell HL, Baker SL. Excess fruit juice consumption by preschool-aged children is associated with short stature and obesity. Pediatrics 1997;99:15–22.
10. Walravens PA, Hambidge KM, Koepfer DM. Zinc supplementation in infants with a nutritional pattern of failure to thrive: a double-blind, controlled study. Pediatrics 1989;83:532–8.

28

HEPATOBILIARY DISEASES

Nancy S. Spinozzi, RD

The liver plays a crucial role in maintaining nutritional homeostasis. In acute and chronic liver disease, the metabolism, absorption, and storage of carbohydrates, protein, fat, vitamins, and minerals are adversely affected, exacerbating malnutrition.[1]

Some of the most common chronic liver diseases in children include extrahepatic biliary atresia, intrahepatic cholestasis, and metabolic disorders (see Chapter 31 for discussion of common metabolic diseases). Given the persistent and progressive nature of some chronic liver diseases, early and aggressive nutritional intervention is essential. Acute hepatic injury, on the other hand, usually requires short-term, straightforward nutritional strategies.[2] Table 28–1 reviews the risk factors associated with both acute and chronic liver disease.

Table 28–2 highlights the specific components of nutritional assessment in this patient population. It should be noted that it is at times difficult to interpret serum levels of proteins and some micronutrients in the context of liver dysfunction. Traditional markers of protein-energy malnutrition (PEM) may be influenced by liver function itself, and not reflective of PEM.

Tables 28–3 and 28–4 list recommendations for vitamin and mineral supplementation for chronic liver disease and the common medications prescribed for patients with chronic liver disease, respectively.

Table 28–1. Nutritional Risk Factors in Acute and Chronic Liver Diseases[1]

Nutrient Affected	*Sequelae*	*Etiology*
Carbohydrate	Glucose intolerance[3] Fasting hypoglycemia/ anorexia Hyperglycemia	Severe, acute hepatic dysfunction Insulin resistance
Protein	Ascites/peripheral edema Refractory coagulopathy Hepatic encephalopathy	Decreased hepatic albumin synthesis Decreased plasma oncotic pressure Decreased synthesis of clotting factors Decreased aromatic amino acid metabolism[4]
Fat	Malabsorption Steatorrhea Essential fatty acid deficiency	Decreased synthesis, secretion, transport of bile salts Portal hypertension Decreased fat soluble vitamin absorption Hypercholesterolemia/ hypertriglyceridemia Impaired absorption
Vitamin A	Night blindness, degeneration of the retina, xerophthalmia, poor growth and hyperkeratosis	Impaired absorption
Vitamin D	Rickets, osteoporosis, osteomalacia, cranial bossing, epiphyseal enlargement, persistently open anterior fontanelle in infants	Impaired absorption Decreased 25-hydroxylation
Vitamin E	Peripheral neuropathy, ataxia	Impaired absorption

Table 28–1 continued

Vitamin K	Coagulopathy, hemorrhagic manifestations such as bruising	Impaired absorption
Minerals	Increased iron Decreased iron, zinc, copper, selenium, calcium	Multiple transfusions Impaired absorption

Aggressive nutritional management of children with chronic liver disease is essential to ensure optimal growth and development, to lesson the impact of the numerous complications of the underlying disease, and to contribute to the successful outcome of eventual transplantation. Table 28–5 reviews many of the issues related to nutritional management. Patients with liver disease have an abnormal

Table 28–2. Special Aspects of Nutritional Assessment

Diet history
Total calorie intake
Total protein intake and source
Total fat intake and source
Sodium intake
Medications with potential effects on nutritional status
Stooling pattern
Physical examination
Evidence of edema or ascites
Hepatosplenomegaly
Laboratory tests
Tests of hepatic synthesis: albumin, prothrombin time (PT), clotting factor levels
Serum ammonia
Vitamin A and retinol binding protein
Zinc
Vitamin D: 25-OH-D
Vitamin E:total lipid ratio

Table 28–3. Recommendations for Vitamin and Mineral Supplementation

Nutrient	*Drug*	*Dose*
Vitamin A[5]	Emulsified vitamin (Aquasol A)	5,000–25,000 IU/d
Vitamin D[6]	Vitamin D_3 (Calderol)	3–5 μg/kg/d
Vitamin E[7]	TPGS (Liqui E)	15–25 IU/kg/d
Vitamin K_1	Vitamin K_1 (Mephyton)	2.5–5 mg/d
Zinc[6]	Zinc sulfate	1 mg/kg/d
Calcium[6]	Elemental calcium	25–100 mg/kg/d
Phosphorus[6]	Elemental phosphorus	25–50 mg/kg/d

TPGS = d-a-tocopherol polyethylene glycol-1000 succinate

Table 28–4. Medications Commonly Used in Chronic Liver Disease

Drug	*Potential Side Effects Affecting Nutritional Status*	*Nutritional Therapy*
Cholestyramine	Fat soluble vitamin malabsorption Fat malabsorption	Monitor and supplement fat soluble vitamins Consider MCT oil
Phenobarbital	Sedation Altered vitamin D metabolism	Vitamin D supplementation
Neomycin	Mucosal toxicity Steatorrhea Carbohydrate malabsorption Vitamin B_{12} deficiency	Consider MCT oil Supplement with vitamin B_{12}
Lactulose	Osmotic diarrhea Hyponatremia Hypokalemia	Monitor and adjust diet Supplement electrolytes

MCT = medium chain triglycerides.
Adapted from Kleinman R, Warman KY. Nutrition in liver disease. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall, Inc.; 1994. p. 264.

serum amino acid profile, specifically, increased aromatic amino acids (AAA) and decreased branched chain amino acids (BCAA). The use of branched-chain amino acid supplementation for the nutritional support of patients with liver disease remains controversial, although both enteral and parenteral products enriched in BCAAs are available.

Table 28–5. Special Aspects of Nutritional Management

Daily calorie and protein requirements:[1,8–10]
- Energy—100–150% USRDA
- Restrict protein intake only in cases of hepatic encephalopathy

Use gut if possible; tube feedings if inadequate POs

Total parenteral nutrition (TPN)
- Use cautiously, as TPN itself can cause liver dysfunction (cholestasis in infants)[11,12]
- Standard amino acid solutions generally tolerated
- BCAA-use in patients with uncontrolled encephalopathy

Enteral product selection
- Infant formula:
  - Pregestimil (Mean Johnson)
  - Alimentum (Ross Products)
  - Portagen (Mean Johnson)
- Pediatric elemental products:
  - Peptamen Junior Diet (Nestle Clinical Nutrition)
  - Vivonex Pediatric (Sandoz Nutrition Co.)
- Adult enteral products:
  - Hepatic Aid II (McGaw)
  - NutriHep Diet (Nestle Clinical Nutrition)
  - Vital HN (Ross Products)
  - Vivonex TEN (Sandoz Nutrition Co.)
  - Peptamen (Nestle Clinical Nutrition)

Postliver transplantation
- Initiation of enteral feeds (with normal liver function):[13]
  - Diet appropriate for age with mild sodium restriction, 2° steroids
  - Tube feeding may be necessary to ensure optimal calories and protein

USRDA = United States Recommended Dietary Allowance

References

1. Molleston JP. Acute and chronic liver disease. In: Walker WA, Watkins JB, editors. Nutrition in pediatrics. 2nd ed. Hamilton (ON): B.C. Decker, Inc.; 1997. p. 565.
2. Hendricks KM. Liver disease in the child. Top Clin Nutr 1987;2:79–87.
3. Petrides AS, Strohmeyer G, DeFronzo RA. Insulin resistence in liver disease and portal hypertension. Prog Liver Dis 1992;10:311–5.
4. Marchesini G, Bianchi G, Zoli M, et al. Plasma amino acid response to protein ingestion in patients with liver cirrhosis. Gastroenterology 1983;85:283–9.
5. Kaufman SS, Murray ND, Wood P, et al. Nutritional support for the infant with extrahepatic biliary atresia. J Pediatr 1987;110:679–86.
6. Ramirez RO, Sokol RJ. Medical management of cholestasis. In: Suchy FJ, editor. Liver disease in children. St. Louis: Mosby; 1994. p. 356.
7. Sokol RJ, Heubi JE, Butler-Simon N, et al. Treatment of vitamin E deficiency during chronic childhood cholestasis with oral d-α-tocopheryl polyethylene glycol-1000 succinate. Gastroenterology 1987;93:975–81.
8. Pierro A, Koletzko B, Carnielli V, et al. Resting energy expenditure is increased in infants and children with extrahepatic biliary atresia. J Pediatr Surg 1989;24:534–8.
9. Kaufman SS, Scrivner DJ, Guest JE. Preoperative evaluation, preparation, and timing of orthotopic liver transplantation in the child. Semin Liver Dis 1989;9:176–9.
10. Sutton M. Nutritional support in pediatric liver transplantation. Diet Nutr Supp Newsletter 1989;11:1–3.
11. Quigley EMM, Marsh MN, Schaffer JL, Markin RS. Hepatobiliary complications of total parenteral nutrition. Gastroenterology 1993;104:286–301.
12. Kleinman R, Warman KY. Nutrition in liver disease. In: Baker SB, Baker RD, Davis A, editors. Pediatric enteral nutrition. New York: Chapman and Hall, Inc.; 1994. p. 264.
13. Pereira GR. Hyperalimentation-induced cholestasis. Am J Dis Child 1981;135(9):842-5.

29

HYPERLIPIDEMIA

Roseann Cutroni, MS, RD

Hyperlipidemia enhances lipid accumulation on arterial walls and is one of several risk factors associated with cardiovascular disease (CVD). Other risk factors include hypertension, cigarette smoking, family history, obesity, diabetes, and a sedentary lifestyle.

Detection and Screening

Although detection and treatment efforts have decreased the mortality of CVD, it continues to be the leading cause of death in the United States. Fifty-eight million Americans have some form of cardiovascular disease; the United States ranks sixteenth in age-adjusted CVD death rates among industrialized nations.[1]

Vascular changes predictive of CVD, such as fatty streaks of the intima, are evident in childhood. Children and adolescents with high cholesterol levels are more likely than the general population to have hyperlidemia as adults.[2] Identification and risk management should therefore begin in childhood.[3]

Mass screening versus selective screening of children remains controversial. In weighing the benefits of mass screening and comprehensive early identification against the potential for early labeling and attendant harmful psychological impact, the National Cholesterol Education Program (NCEP) recommends the following selective screening of children: begin screening after the age of 2 years in children whose parents or grandparents ≤ 55 years of age have had coronary atherosclerosis or suffered docu-

mented myocardial infarction, angina pectoris, peripheral vascular disease, or sudden cardiac death. Children and adolescents with one or more CVD risk factors and unavailable family history may be tested to identify need for specific dietary or medical intervention. Risk assessment parameters are described in Figure 29–1.

Assessment

Table 29–1 outlines assessment data pertinent to the evaluation of the pediatric patient with hyperlipidemia. Lipoproteins derived from diet or stored fat are synthesized

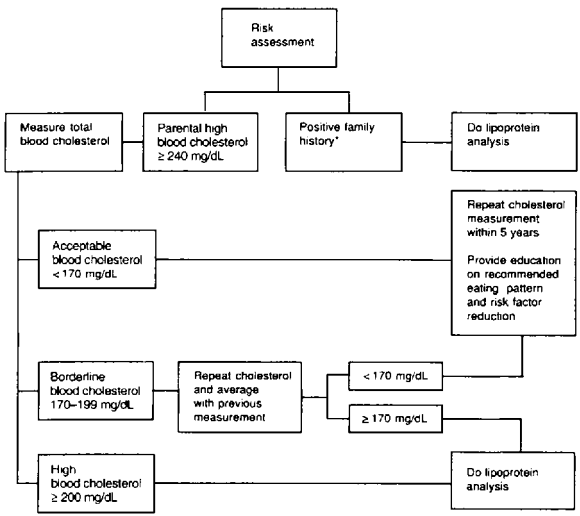


Figure 29–1 Risk assessment. *Defined as a history of premature (before age 55 years) cardiovascular disease in a parent or grandparent. With permission from National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. Dept. of Health and Human Services (US), National Institutes of Health; 1991 Sept. NIH Publication No.: 91-2732. p. 7.

Table 29–1. Nutritional Assessment of Pediatric Hyperlipidemia

- History
  - Family history
    - CVD
    - Diabetes
    - Lipid levels and response to diet, lifestyle changes, and medications
  - Child's history
    - Weight and growth
    - Diet history
    - Exercise patterns
    - Changes in lipid levels
    - Overall health history
    - Medications, supplements
- Physical
  - Height and weight
  - Blood pressure
  - Xanthomas
- Laboratory
  - Total cholesterol
    - Fasting lipid profile (see Figure 29–2 for indications)
    - Fasting triglyceride level (if there is a family history of high triglycerides or if the child is obese)

in the liver and contain cholesterol, triglycerides, and phospholipids. Table 29–2 lists the classification of the five primary hyperlipidemias. Types I and V are very rare inherited disorders that are managed through control of the amount and type of dietary fat consumed. Types II, III, and IV are related to CVD risk and are discussed here in more detail. The lipoproteins low density lipoprotein (LDL), high density lipoprotein (HDL), and very low density lipoprotein (VLDL) are distinguishable by the varying amounts of cholesterol, triglycerides, phospholipids, and protein they contain. Low density lipoprotein carries the most cholesterol with little protein while HDL is protein-rich with less cho-

Table 29–2. Classification of Hyperlipidemias

Type	*Cholesterol*	*Triglycerides*	*Plasma*
I. (increased chylomicrons)	Normal or slightly elevated	Very high (> 1000 mg/dL)	Supernatant cream layer present; infranate clear
IIa. (increased LDL)	Elevated	Normal	Plasma clear
IIb. (increased LDL and VLDL) mg/dL	Elevated (LDL cholesterol > 190 mg/dL)	Elevated (200–400 mg/dL)	Plasma slight to moderately turbid
III.* (increased IDL)	Elevated	Elevated	Plasma turbid (200–1000 mg/dL)
IV. (increased VLDL)	Normal or slightly elevated (LDL cholesterol > 190 mg/dL)	Elevated (400–1000 mg/dL)	Plasma turbid to opaque
V. (increased chylomicrons and VLDL)	Moderately elevated	Markedly elevated (> 1000 mg/dL)	Supernatant cream layer plus turbid plasma infranate

*Definitive diagnosis of type III requires lipoprotein ultracentrifugation and characterization of Apo E isoforms.
LDL = low density lipoprotein; IDL = intermediate density lipoprotein; VLDL = very low density lipoprotein.

lesterol: LDL is also the major transporter of cholesterol to the arterial walls. The NCEP classification of total cholesterol and LDL levels is outlined in Figure 29–2. High den-

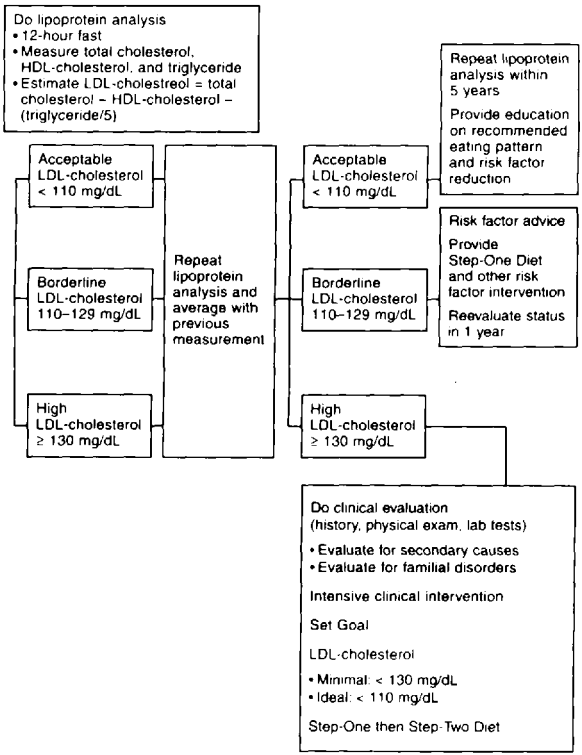


Figure 29–2 Classification, education, and follow-up based on low density lipoprotein-cholesterol. Reproduced with permission from National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. Dept. of Health and Human Services (US), National Institutes of Health; 1991 Sept. NIH Publication No.: 91-2732. p. 8.

Table 29–3. Serum Lipoproteins and Cardiovascular Risk

Lipoprotein	*Risk*
Low density lipoprotein (LDL)	High levels (> 130 mg/dL) increase risk
High density lipoprotein (HDL)	Low levels (< 45 mg/dL) increase risk
Triglycerides	High levels (> 150 mg/dL) with high LDL and low HDL are associated with increased risk

sity lipoprotein is believed to function as the reverse cholesterol transporter, deterring lipid accumulation in the arterial wall. The goal HDL serum level for children ages 5 to 19 is 50 mg/dL, with any level below 35 mg/dL considered to be too low.[4] Cardiovascular disease risk is determined by the ratio of total cholesterol to HDL. As HDL increases, risk decreases. The average ratio in children is 3.5. Table 29–3 outlines the role of lipoproteins and triglycerides specific to cardiovascular risk.

Elevated fasting triglycerides in a patient with lower than expected HDL levels may also be a risk factor for CVD.[5] Expected serum triglyceride levels are age-specific. Means range from 50 mg/dL to 60 mg/dL between the ages of 2 and 5 years and then increase to a mean of 75 mg/dL by the age of 20 years.[6]

Management

Management of pediatric hyperlipidemias begins with dietary and lifestyle changes. Appropriate diet and consistent physical activity control can prevent obesity, which has a beneficial effect on total cholesterol, HDL, and triglyceride levels; it is the most effective intervention.

Dietary management of hyperlipidemia is designed in two phases; the Step-One and Step-Two Diets are outlined

Table 29–4. Step-One and Step-Two Diets

	Recommended Intake	
Nutrient	*Step-One Diet*	*Step-Two Diet*
Total fat	Average of no more than 30% of total calories	Same
Saturated fatty acids	< 10% of calories	< 7% of calories
Polyunsaturated fatty acids	Up to 10% of total calories	Same
Monounsaturated fatty acids	Remaining fat calories	Same
Cholesterol	< 300 mg/d	< 200 mg/d
Carbohydrates	About 55% of total calories	Same
Protein	About 15–20% of total calories	Same
Calories	To promote normal growth and development and to reach or maintain desirable body weight	Same

Adapted from National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. Dept. of Health and Human Services (US), National Institutes of Health; 1991 Sept. NIH Publication No.: 91-2732.

in Table 29–4. The Step-One Diet reduces the intake of obvious saturated fat and cholesterol. Monosaturated fat (eg, canola and olive oil) is the dietary fat of choice.

Polyunsaturated fats (eg, corn, soybean oil) are also recommended for up to 10% of calories.[7] Trans-fatty acids (eg, hydrogenated oils) are associated with increased LDL levels and should be avoided.[8] Various dietary fats and their dietary sources are described in Table 29–5.

The effectiveness of dietary management is evaluated by assessing the improvement in lipid levels as defined in Figure 29–2. The goal for a child initially evaluated in the high lipid category is to achieve a borderline category. The

Table 29–5. Types and Effects of Dietary Fat

Fat Type and Effect	*Food Sources*
Saturated fat: increases LDL and HDL	Animal foods: meats, fish, poultry Fat-containing dairy products Palm and coconut oils Chocolate Stick margarines Cereals, crackers, chips Number of grams saturated fat is shown on food labels
Monosaturates: decrease LDL; no effect on HDL	Oils: olive, canola, rapeseed, peanut Ingredient list on food labels describes fat source
Polyunsaturates: decrease LDL and HDL	Vegetable oils: safflower, sunflower, soybean, corn, cottonseed oils Tub margarines, chips, crackers, cereals Ingredient list on food labels describes fat source
Trans fatty acids: May increase LDL May increase CVD risk	Hydrogenated oils: stick margarines, cookies, crackers, cereals, desserts Ingredient list on food labels indicates presence of hydrogenated oils

aim for a child in the borderline category is to achieve acceptable levels.

If the goals for the child are not achieved after 3 months on the Step-One Diet, therapy is progressed to the Step-Two Diet, which further restricts saturated fat and cholesterol intake. Medication is considered if after 1 year of dietary adjustment the LDL remains > 190 mg/dL at age > 10 years, or LDL is > 160 mg/dL with two other risk factors present. Medications are used as an adjunct to the Step-Two Diet.

Elevated triglycerides respond to sugar restriction and maintenance of healthy weight. Sweetened beverages such as sodas, juices, and sports drinks as well as candies and sweets are the major dietary sources of sugar that contribute to excessive weight gain. Limiting these sugar sources to 7 to 10% of calories, along with other weight loss strategies when warranted, will decrease serum triglycerides. See Chapter 32 for further details.

Diet manipulation in childhood must be tempered by attention to adequate nutrition and be tailored to each child's needs. While rigid diet control is counterproductive, institution of appropriate heart-healthy eating after the age of 2 years (generous servings of fruits and vegetables and less meat, eggs, fatty dairy, and oils) does not compromise growth and development.[9] Eating patterns should be constructed for the whole family to ensure easier compliance and optimal health benefits for everyone.

Emerging Data

Recent studies implicate other dietary factors in CVD (Table 29–6). The implications for both adults and children are as yet unclear but nutritional management of so-called adult-onset disease will probably soon begin in childhood.

Table 29–6. Emerging Data: Nutrients and Cardiovascular Disease

Nutrient	*Effects*	*Practical Pediatric Application*
Antioxidants (especially vitamin E)[10]	Antioxidants prevent plaque-forming effects of free radicals and may slow progression of plaques	Dose to establish effect is unclear. Eating generous amounts of fruits and vegetables will optimize natural antioxidant sources
Dietary fiber (especially soluble fiber)[11]	High fiber diets are lower in fat and cholesterol. Water-soluble fibers may enhance the excretion of bile	Increased food volume with decreased caloric density of high fiber diets may negatively impact growth in children with high caloric needs
Fish oils[12]	Fish oil may inhibit platelet aggregation. Fish eaters have a lower incidence of heart attack and overall mortality	Replacing meat with a fish meal twice a week decreases saturated fat and provides a source of fish oil
Homocysteine and folate[13]	Homocysteinuria is associated with accelerated atherosclerosis. Moderately elevated serum homocysteine is associated with greater CVD risk Folate and vitamin B_6 are necessary to metabolize homocysteine. High folate levels are associated with low homocysteine levels	A diet high in whole grains, legumes, and leafy greens provides low fat sources of folate

References

1. American Heart Association. Cardiovascular diseases, heart and stroke facts: 1995 statistical supplement. ICD/9: 390–459, 745–7. (Website: http://www.americanheart.org/Scientific/dHStats98/03cardio.html).
2. Berenson GS, Wattigney WA, Tracy RE, et al. Artherosclerosis of the aorta and coronary arteries and cardiovascular risk factors in persons aged 6 to 30 years and studied at necropsy (the Bogalusa Heart Study). Am J Cardiol 1992;70:851–8.
3. Nicklas TA, Farris RP, Smoak CG, et al. Dietary factors relate to cardiovascular risk factors in early life. Arteriosclerosis 1988;8:193–9.
4. National Heart, Lung, and Blood Institute. The Lipid Research Clinics Population Studies Data Book: Volume 1—the Prevalence Study. Bethesda (MD): Dept. of Health and Human Services (US), Public Health Service, National Institutes of Health; 1980 July. NIH Publication No.: 80-1527.
5. LaRosa JC. Triglycerides and coronary risk in women and the elderly. Arch Intern Med 1997;157:961–8.
6. National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. Dept. of Health and Human Services (US), National Institutes of Health; Sept. 1991. NIH Publication No.: 91-2732.
7. Mattson FH, Grundy SM. Comparison of effects of dietary saturated, monosaturated, and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. J Lipid Res 1985;26:194–202.
8. Judd JT, Clevidence BA, Muesing RA, et al. Dietary trans fatty acids: effects on plasma lipids and lipoproteins of healthy men and women. Am J Clin Nutr 1994;59:861–4.
9. Dixon LB, McKenzie J, Shannon BM, et al. The effect of changes in dietary fat on the food group and nutrient intake of 4- to 10-year-old children. Pediatrics 1997;100:863–72.
10. Stephens NG, Parsons A, Schofield PM, et al. Randomised controlled trial of vitamin E in patients with coronary disease: Cambridge Heart Antioxidant Study. Lancet 1996;347:781–6.

11. Rimm EB, Ascherio A, Giovannucci E, et al. Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. JAMA 1996;275:447–51.
12. Sellmayer A, Witzgall H, Lorenz RL, Weber PC. Effects of dietary fish oil on ventricular premature complexes. Am J Cardiol 1995;76:974–7.
13. Morrison HI, Schaubel D, Desmeules M, Wigle DT. Serum folate and risk of fatal coronary heart disease. JAMA 1996;275:1893–6.

30

THE KETOGENIC DIET

Marilyn Bernard, MS, RD

The ketogenic diet is an eating plan that allows the body to stay in a constant state of ketosis. It is used therapeutically to manage refractory seizures or to help reduce the side effects of antiepileptic medications. The diet consists of individually calculated amounts of foods to achieve a high-fat, low-carbohydrate, and lowered-protein diet.

Nearly a century ago, several investigators noticed that epileptic patients had fewer seizures while fasting or while on a "water diet."[1] The original ketogenic diet was developed in the 1920s to mimic the biochemical changes associated with starvation. The diet was an effective and widely used therapy for seizures until the 1950s, when antiepileptic medications became increasingly available. Recently, the diet has regained popularity as an effective alternative or adjunct to these medications. A variety of studies have shown significant reductions in seizure frequency with the ketogenic diet.[2,3] A recent study reported the efficacy rate of the diet at intervals spanning 1 year.[4] At 6 months, 51% of the children experienced more than a 50% decrease in seizure frequency, and this effect continued for the 1 year of the study.

Ketogenic Diet Therapy

Although several factors are considered necessary for its efficacy, the diet's exact mechanism of action has not yet been determined. It is known that a high level of ketosis must be reached. Traditionally, ketosis has been monitored by measuring urinary acetoacetate. Measurement of blood ketones (eg, β-hydroxybutyrate) may give a more

accurate picture of ketosis level although this is not feasible for routine outpatient care.

Strict adherence to the diet is essential since only a small variation in dietary intake can affect the maintenance of ketosis and thus seizure control. Food is usually divided into three meals per day. Intake between meals is restricted to those foods that provide little or no carbohydrates. These include sugar-free fruit-flavored drinks made with saccharin, sugar-free soda, water, or measured amounts of nuts or olives. The diet must be supplemented with a multivitamin and multimineral supplement as well as additional calcium. The carbohydrate content of all medications must be determined and calculated as part of the total carbohydrate content of the diet.[5]

Table 30–1 describes variations of the ketogenic diet. There are two broad categories of ketogenic diets, based on the predominant fat source, either medium chain triglyceride oil or long chain dietary fats (cream, butter, oil, and margarine).

Table 30–1. Ketogenic Diet Variations

	MCT Oil Diet	*Cream Diet (Traditional Diet)*
History	Introduced in 1971 by Huttenlocher[6]	Introduced in 1921 by Wilder[7]
Fat source	MCT oil (60% of kcal) Long chain fat (11% kcal)	Long chain fat (87–90% kcal)
kcal content	RDA for age	75–80% of either RDA or typical intake
Fluid restriction	Restricted only if necessary	Restricted
Initiation of diet	After 3 large urinary ketone readings	After 1 large urinary ketone reading

MCT = medium chain triglyceride; RDA = Recommended Dietary Allowance.

Diet initiation is best done in an inpatient setting, due to the real potential for hypoglycemia, dehydration, and acidosis. The child is fasted until urinary ketones, measured as acetoacetic acid on urinary dipsticks, are large (80 to 160 mg/dL). This usually occurs in 24 to 36 hours. Eggnog (composed of heavy cream, pasteurized egg, and artificial sweetener and flavoring) is given for the first 1 to 2 days to allow tolerance to develop to the high fat content of the diet. One-third of the estimated energy needs are given on the first day, and two-thirds on the second day. Once the full amount of calories are reached, either eggnog or real food may be served.

Meal plans are calculated to provide the determined amounts of carbohydrate, protein, and fat per meal according to their individual diet prescription (see instructions on calculating the diet prescription, below). This relationship of grams of fat to grams of protein plus carbohydrates is the ketogenic ratio. A ketogenic diet will typically have a 4:1 or 3:1 ratio. In a 4:1 ratio, there is four times as much fat as protein and carbohydrate. Computer programs are available for calculating meal plans. Table 30–2 illustrates a typical meal plan for a day on the ketogenic diet.

One of the attractions of the ketogenic diet is that its use may allow the reduction or discontinuation of antiepileptic medications, drugs that may have adverse side effects,

Table 30–2. Typical Day's Menu for an 8-Year-Old Boy

Diet: 1885 kcal 62.8 g Fat 8.5 g Protein 7 g Carbohydrate

Breakfast	*Lunch*	*Dinner*
57 g egg	25 g cheese	67 g hot dog
47 g orange	72 g broccoli	27 g grapes
51 g butter	35 g oil	35 g mayonnaise
55 g heavy cream	55 g heavy cream	50 g heavy cream

Nonetheless, the diet itself may result in a number of short-term and long-term complications for which all patients should be carefully monitored.[4,8,9] Table 30–3 reviews common possible side effects of ketogenic diet therapy and suggests steps for their prevention or treatment.

Sample Diet Calculation (Traditional Cream-Based Diet)

April is a 6-year-old girl. She weighs 21 kg (50 to 75 percentile weight for age) and is 110 cm tall (10 to 25 percentile height for age). Her weight for height is 90 to 95 percentile. She is on the following medications: Tegretol 700 mg/day and Celontin 600 mg/day.

1. **Energy needs.** Caloric needs for patients on the ketogenic diet are lower than the Recommended Daily Allowance (RDA). (Table 30–4). If the child is overweight, energy needs are based on ideal weight for height. For example:

   April is overweight. Her ideal weight would be 18.25 kg. Calorie amount = 18.25 kg × 65 kcal/kg = 1186 kcal/day.

2. **Ketogenic ratio.** Most children are started on a 4:1 ketogenic ratio. Very young (< 15 months) or overweight children may be started on a 3:1 or 3.5:1 ratio of fat to protein and carbohydrates. Use a 3:1 ratio for children > 12 years old. (eg, April is overweight; therefore, she will start with a 3:1 ratio.)
3. **Dietary Units.** Dietary units are the building blocks of the ketogenic diet. One dietary unit reflects the amount of calories in one block of the ratio, as follows:

Ratio	Fat	CHO+PRO	kcal/Dietary Unit
2:1	2 g × 9 kcal/g = 18	1 g × 4 kcal/g = 4	18 + 4 = 22
3:1	3 g × 9 kcal/g = 27	1 g × 4 kcal/g = 4	27 + 4 = 31
4:1	4 g × 9 kcal/g = 36	1 g × 4 kcal/g = 4	36 + 4 = 40
5:1	5 g × 9 kcal/g = 45	1 g × 4 kcal/g = 4	45 + 4 = 49

Table 30–3. Possible Complications of the Ketogenic Diet

Complication	*Therapy*
Short-term	
Dehydration	Increase fluid. If already at maximum fluid allotment, increase by 5–10%
Hypoglycemia	Assure that meals are fully completed. May need to decrease the ratio (eg, change from a 4:1 ratio to a 3:1 ratio)
Vomiting	Maintain the ketogenic diet as tolerated. Monitor fluid to prevent dehydration. May require dextrose-free IV fluid
Diarrhea	Monitor fluid intake to prevent dehydration. Increase fiber intake (either by increasing use of vegetables or with calculated amounts of bran fiber)
Food refusal	Adjust meals to decrease portion size. May use a lower ketogenic ratio temporarily to help make meals more appealing (a lowered ratio will increase the portion size of solids and decrease the amount of added fat)
Long-term	
Kidney stones	Increase fluid intake
Metabolic acidosis	Increase fluid intake. May need to decrease the ketogenic ratio
Hyperuricemia	Increase fluid intake
Lethargy	Assure that kcal level and ratio are appropriate
Refusal to eat	(See food refusal, above). May need to discontinue diet if prolonged
Malnutrition	Monitor weight weekly during the first month on the diet. Weight loss should not exceed 1 lb per week, and is not encouraged for patients younger than 2 years. Monitor serum albumin every 3–4 months; may need to increase the protein allotment or calories
Carnitine deficiency	Monitor blood carnitine profiles; may need supplementation

For example, April will have a 3:1 ratio, so each dietary unit will be made up of 31 kcal.

4. **Dietary unit quantity per day.** Divide the total daily calories by the number of calories per dietary unit. For example:

$$\frac{1186 \text{ kcal}}{31 \text{ kcal/dietary unit}} = 38 \text{ dietary units/day}$$

5. **Fat allowance.** Multiply the number of dietary units by the units of fat in the prescribed ketogenic ratio to determine the grams of fat permitted daily. For example:

$$38 \text{ units} \times 3 \text{ g fat/dietary unit} = 114 \text{ g fat/day}$$

6. **Protein-carbohydrate allowance.** Multiply the number of dietary units by the number of units of protein plus carbohydrate, in the prescribed ketogenic ratio. For example:

$$38 \text{ units} \times 1 \text{ g CHO+PRO/dietary unit} = 38 \text{ g CHO + PRO}$$

(where CHO = carbohydrate; PRO = protein)

7. **Protein Allowance.** Determine the protein allowance, using Table 30-5. Base the calculation on ideal weight

Table 30–4. Energy Intake Goals for the Ketogenic Diet

kcal	*Age (years)*	*Ketogenic kcal*
	< 1	80 kcal/kg
	1–1½	75 kcal/kg
	1½–3	70 kcal/kg
	4–6	65 kcal/kg
	7–8	60 kcal/kg
	9-10	< 55 kcal/kg
	11–14	≤ 37.5–40 kcal/kg (females)
	Adults	≤40–45 kcal/kg (males)

Table 30–5. Protein Intake Goals for the Ketogenic Diet

Age	*Allowance*
< 2 yr	RDA
2–12 yr	1 g/kg
> 12 yr	0.75–1 g/kg

RDA = Recommended Dietary Allowance.

if overweight or very underweight; otherwise, use actual weight. For example:

$$1 \text{ g/kg} \times 18.25 \text{ kg} = 18 \text{ g/day}$$

8. **Carbohydrate allowance.** Subtract the grams of protein from the grams of carbohydrate and protein. For example:

$$38 \text{ g CHO+PRO} - 18 \text{ g PRO} = 20 \text{ g CHO/day}$$

9. **Additional carbohydrates.** The carbohydrate content of all required medications must be determined. The extra carbohydrate must be factored into the diet (usually replacing a portion of the fruit or vegetable serving). Using Table 30–6, for example:

$$\text{CHO in meds} = 325 \text{ mg CHO}$$
$$\text{CHO allowance} - \text{CHO in meds} = 20 \text{ g} - 0.325 \text{ g} = 19.7 \text{ g CHO}$$

Table 30–6. Carbohydrate Content of Two Seizure Medicines

Medication	*Dosage*	*Dosage × CHO/Dose*	*Total CHO*
Tegretol	3.5 × 200 mg tab	3.5 × 50 mg CHO	175 mg
Celontin	2 × 300 mg cap	2 × 75 mg CHO	150 mg

CHO = carbohydrate.

10. **Composition of meals.** Divide each daily allotment by 3 to get 3 equal meals. For example:

$$\frac{\text{Fat 120 g/day}}{3} = 40 \text{ g/meal} \qquad \frac{\text{PRO 18 g/day}}{3} = 6 \text{ g/meal}$$

$$\frac{\text{CHO 19.7 g/day}}{3} = 6.6 \text{ g/meal}$$

11. **Fluid Allowance.** For example:

    1 cc per calorie of ketogenic diet = 1186 cc

    The cream is included in the fluid allotment. In hot seasons or climates, extra carbohydrate-free, calorie-free fluid in an amount equal to the total amount of cream in the diet may be added.

12. **Supplements.** The following are recommended:
    - Calcium (600 to 650 mg/day in sugar-free form)
    - Multivitamin (1 age-appropriate dose in sugar-free form)
    - See Chapter 13, Vitamin and Mineral Supplements for list of sugar-free supplements

References

1. Lennox, WG. Epilepsy and related disorders. Boston: Little, Brown and Company; 1960.
2. Schwartz RH, Eaton J, Bower BD, et al. Ketogenic diets in the treatment of epilepsy: short-term clinical effects. Child Neurol 1989;31:145–51.
3. Kinsman SL, Vining EP, Quaskey SA, et al. Efficacy of the ketogenic diet for intractable seizure disorders: review of 58 cases. Epilepsia 1992;33:1132–6.
4. Freeman JM, Vining EP, Dillas DJ, et al. The efficacy of the ketogenic diet—1998; a prospective evaluation of intervention in 150 children. Pediatrics 1998;102:1358–63.
5. Feldstein, TJ. Carbohydrate and alcohol content of 200 oral liquid medications for use in patients receiving ketogenic diets. Pediatrics 1996;97:506–11.

6. Huttenlocher PR, Wilbourn AJ, Signore JM. Medium chain triglycerides as therapy for intractable childhood epilepsy. Neurology 1971;21:1097.
7. Wilder RM. The effects of ketonuria on the course of epilepsy. Mayo Clin Proc 1921;2:307.
8. Ballaban-Gil K, Callahan C, O'Dell C, et al. Complications of the ketogenic diet. Epilepsia 1998;39:744–8.
9. Herzberg GF, Fivush BA, Kinsman SL, et al. Urolithiasis associated with the ketogenic diet. J Pediatr 1990;117:743–5.

Additional Resources

Books

Freeman J, Kelly M. The epilepsy diet treatment: an introduction to the ketogenic diet. New York: Demos Vermande Publication; 1996.

Brake D, Brake C. The ketogenic cookbook. Gilman (CT): Pennycorner Press; 1997.

Computer Program

The ketogenic diet computer program
Available from the Epilepsy Asociation of Maryland
300 East Joppa Road, Suite 1103, Towson (MD)
1-410-828-7700

Videocassette

Introduction to the ketogenic diet:
a treatment for pediatric epilepsy
The Charlie Foundation, 501 10th Street,
Santa Monica, CA 90402
1-800-FOR-KETO

Internet Resources

www.stanford.edu/group/ketodiet
www.scottishritechildren.org/services/ketogenic.shtml
www.members.aol.com/ketooption/index.htm
www.ketogenic.org

31

METABOLIC DISORDERS

Ann Munier, MS, RD,
and Frances Rohr, MS, RD

Inborn errors of metabolism are inherited disorders caused by a defect in enzymes required to metabolize protein, carbohydrate, or fat. The inheritance of most metabolic disorders is autosomal recessive and the incidence uncommon. Many of the disorders result in severe clinical manifestations that often appear soon after birth. Rapid diagnosis and treatment are essential to prevent neurologic damage, mental retardation, and possible death. Because these disorders are rare and require careful monitoring of metabolic stability, individuals afflicted with them are best served by clinics specializing in inherited metabolic disorders. Referral centers are listed in Appendix C.

The absence or reduced activity of a specific enzyme or cofactor in metabolic disorders results in a buildup of the substrate and deficiency of the product. Treatment is based on the specific metabolic defect and is designed to correct the primary metabolic imbalance by reducing available substrate through dietary restriction, supplementing the product of the blocked pathway, supplementing cofactors in vitamin-responsive defects, and/or using medications that facilitate excretion and detoxification of toxic metabolites.

Nutrition therapy is a key component in treating metabolic disorders. The overall goal of nutrition therapy is to correct the metabolic imbalance while providing adequate energy, protein, and nutrients for normal growth and development. Frequent monitoring of growth, laboratory

values, and nutrient intake is necessary to evaluate the adequacy of the diet. Small, frequent changes in the diet prescription are needed to ensure metabolic stability and optimal growth. Selected metabolic disorders are discussed briefly below, with specific guidelines presented in Table 31–1. Detailed protocols on the nutritional management of specific metabolic disorders are available.[1]

Disorders of Amino Acid Metabolism

Nutritional management of amino acid disorders involves reducing available substrate by restricting one or more essential amino acids to the minimum requirement and supplying the product of blocked reactions. Amino acid-restricted diets require the use of chemically defined formulas such as those listed in Table 31–2. These special formulas, which are age- and diagnosis-specific, provide a major portion of the daily intake of protein, calories, vitamins, and minerals. Formulas are generally composed of L-amino acids. Since these amino acids are absorbed and oxidized more rapidly than amino acids derived from digestion of whole protein, recommended protein and calorie intakes for children with metabolic disorders are often higher than the Recommended Dietary Allowance (RDA).

To supply only the minimum amount of restricted amino acids required for growth, intake of natural protein is very limited. For infants, the natural protein is generally supplied by breastmilk or standard infant formula. In older children, table foods with low to moderate protein content provide the natural protein. The recommended protein or amino acid restriction is based on the specific disorder as well as age, growth rate, and individual tolerance. Over-restriction of protein or specific amino acids is detrimental and can result in poor growth and, in severe cases, the classic symptoms of kwashiorkor (eg, hypoalbuminemia, edema, fatty liver, and dermatitis).

Table 31–1. Inherited Metabolic Disorders

Disorder	*Enzyme Affected*	*Biochemical Findings*	*Clinical Features*	*Nutritional Modification*	*Vitamin Therapy*
Amino Acid Disorders					
Phenylketonuria (PKU): Severe (classic), Moderate (atypical), Mild (hyperphenylalaninemia)	Phenylalanine hydroxylase	Increased blood phenylalanine severe: > 1200 µmol/L moderate: 360–1200 µmol/L mild: 120–360 µmol/L	If untreated, mental retardation, seizures, hyperactivity, and eczema; normal development with proper treatment	Phenylalanine restriction, tyrosine supplementation	None
Maternal phenylketonuria			Untreated PKU in the mother causes mental retardation, congenital heart disease, low birth weight, and microcephaly in offspring		
Hyperphenylalaninemia (pterin defect)	Dihydropteridine reductase; GTP cyclohydrolase	Mild to moderate hyperphenylalaninemia (see above)		± phenylalanine restriction, tyrosine supplementation	Tetrahydropterin (2 mg/kg/d) orally ± neurotransmitter supplements

Tyrosinemia type I	Fumarylaceto-acetate hydrolase	Increased blood phenylalanine, tyrosine, ± methionine, increased alpha-fetoprotein, urinary succinylacetone	Liver failure, renal tubular disease, FTT, vomiting, diarrhea, rickets, porphyric crises, hepatic carcinoma	Phenylalanine and tyrosine restriction, ± methionine restriction (diet used in conjunction with NTBC or until liver transplantation is possible)	None
Tyrosinemia type II	Tyrosine aminotrans-ferase	Increased blood phenylalanine and tyrosine	Mental retardation, photophobia, palmar keratosis	Phenylalanine and tyrosine restriction	None
Homocystinuria (pyridoxine non-responsive)	Cystathionine β-synthase	Homocystine in blood and urine, increased methionine and decreased cystine in blood	Dislocated lenses, marfanoid-like skeletal changes, intravascular thromboses, mental retardation, osteopenia	Methionine restriction; cystine, betaine, folate supplementation	Betaine 100 mg/kg/d orally
Homocystinuria (pyridoxine responsive)	Cystathionine β-synthase	Same as above	Same as above	None	Pyridoxine 25–100 mg/d orally

Table 31–1. *continued*

Disorder	*Enzyme Affected*	*Biochemical Findings*	*Clinical Features*	*Nutritional Modification*	*Vitamin Therapy*
Maple syrup urine disease	Branched chain ketoacid dehydrogenase complex	Elevated blood, urine, and CSF leucine, isoleucine, valine, alloisoleucine	Neonatal form: poor feeding, fluctuating tone, apnea, seizures, death, developmental delay Variant forms: milder ketoacidosis triggered by protein load or illness	Valine, isoleucine, and leucine restriction	Only in variant forms, where 100–300 mg/d oral thiamin may enhance residual enzyme activity
Glutaric acidemia type I	Glutaryl-CoA dehydrogenase	Elevated blood, urine, and CSF glutaric acid and 3-OH-glutaric acid, metabolic acidosis	Acute metabolic crisis (vomiting, acidosis and neurologic deterioration triggered by illness), macrocephaly, ataxia, choreoathetosis, developmental delay	Lysine and tryptophan restriction; carnitine supplementation	May have partial response to ribloflavin (100–300 mg/d) orally

Glutaric acidemia type II	Multiple acyl-CoA dehydrogenase	Elevated blood, urine, and CSF glutaric acid and 2-OH glutaric acid, metabolic acidosis, hyperammonemia, hypoglycemia (± ketones), impaired fatty acid oxidation	Malformations in most severe form, hypotonia, hepatomegaly, developmental delay	Mild protein and fat restriction; fasting avoidance, ± carnitine supplementation	Riboflavin 100–300 mg/d orally
Isovaleric acidemia	Isovaleryl-CoA dehydrogenase	Elevated blood, urine, and CSF isovaleric acid, metabolic acidosis, hyperammonemia, hypoglycemia	Poor feeding, vomiting, sweaty-feet body odor, seizures, coma, death if untreated	Leucine restriction, glycine and carnitine supplementation	None
Methylmalonic acidemia	Methylmalonyl-CoA mutase	Metabolic acidosis, ketonuria, hypoglycemia, hyperammonemia, hyperglycinemia	Lethargy, failure to thrive, vomiting, hepatomegaly, hypotonia, coma, death if untreated	Isoleucine, methionine, valine, threonine restriction; carnitine supplementation	None

Table 31–1. continued

Disorder	*Enzyme Affected*	*Biochemical Findings*	*Clinical Features*	*Nutritional Modification*	*Vitamin Therapy*
Methylmalonic acidemia	Cobalamin processing defect (hydroxy-cobalamin or adenosyl-cobalamin)	Metabolic acidosis, ketonuria, ± homocystine in urine and blood, ± folate deficiency	Lethargy, failure to thrive, vomiting, hepatomegaly, hypotonia, coma, and death if untreated	Carnitine supplementation	Hydroxycobalamin (1–2 mg daily to weekly intramuscularly)
Propionic acidemia	Propionyl-CoA carboxylase	Metabolic acidosis, ketonuria, hyperglycinemia, hypoglycemia, hyperammonemia	Poor feeding, vomiting, lethargy, hypotonia, seizures, coma, and death if untreated; develop-mental delay	Isoleucine, methionine, valine, threonine restriction; carnitine supplementation	None
Urea Cycle Disorders					
Carbamyl phosphate synthetase deficiency	Carbamyl phosphate synthetase	Hyperammonemia, respiratory alkalosis	Lethargy, vomiting, apnea, coma and death if untreated	Protein restriction; essential amino acids, arginine or citrulline supplementation, sodium phenylbutyrate	None

Ornithine transcarbamylase deficiency	Ornithine transcarbamylase	Hyperammonemia, respiratory alkalosis	Lethargy, vomiting, apnea, coma, and death if untreated; developmental delay	Protein restriction; None essential amino acids, arginine or citrulline supplementation, sodium phenylbutyrate
Citrullinemia	Argininosuccinic synthetase	Hyperammonemia, respiratory alkalosis	Lethargy, vomiting, apnea, coma, and death if untreated; developmental delay	Protein restriction; None essential amino acids, arginine supplementation, sodium phenylbutyrate
Argininosuccinic aciduria	Argininosuccinic lyase	Hyperammonemia, respiratory alkalosis	Lethargy, vomiting, apnea, coma, and death if untreated; developmental delay	Protein restriction; None essential amino acids, arginine supplementation
Argininemia	Arginase	± hyperammonemia	Spastic diplegia, mental retardation	Protein restriction; None essential amino acids, ± sodium phenylbutyrate
Hyperornithinemia-hyperammonemia-homocitrullinuria (HHH syndrome)	Defect in mitochondrial transport of ornithine	Hyperornithinemia, hyperammonemia, homocitrullinuria, hyperglutaminemia, hyperalaninemia	Ataxia, lethargy, vomiting, choreoathetosis, seizures, coma, developmental delay	Protein restriction; None arginine or citrulline supplementation

Table 31–1. continued

Disorder	*Enzyme Affected*	*Biochemical Findings*	*Clinical Features*	*Nutritional Modification*	*Vitamin Therapy*
Disorders of Carbohydrate Metabolism					
Galactosemia	Epimerase	Galactose in blood and urine	Hepatomegaly, jaundice, vomiting	Restrict galactose, ± calcium supplementation	None
Galactosemia	Galactokinase	Galactose in blood and urine	Cataracts	Restrict galactose, ± calcium supplementation	None
Galactosemia	Galactose-1-phosphate uridyl transferase	Galactose in blood and urine; renal Fanconi's syndrome	Cataracts, diarrhea, failure to thrive, hepatomegaly, jaundice, vomiting, *Escherichia coli* sepsis	Restrict galactose, ± calcium supplementation	None
Pyruvate dehydrogenase complex deficiency	Pyruvate dehydrogenase	Elevated blood pyruvate and lactate, elevated blood alanine	Hypotonia, failure to thrive, seizures, ± dysmorphism, developmental delay	Restrict carbohydrate, provide high fat diet (70% of energy)	Thiamin (50–100 mg/d orally)
Glycogen storage disease type I	Glucose-6-phosphatase	Hypoglycemia, elevated lactate, alanine,	Hepatomegaly, growth retardation	Avoid sucrose, lactose, and fructose; provide frequent	None

		triglycerides, and uric acid in blood		feedings and complex carbohydrates; uncooked cornstarch (after age 9 months)	
Glycogen storage disease type III	Amylo-1, 6-glucosidase	Hypoglycemia, hypertriglyceridemia, ketosis, low lactate and alanine in blood	Hepatomegaly, growth retardation	High protein diet (25% of kcal after infancy), moderate carbohydrate and fat intake; frequent feedings, ± uncooked cornstarch (after age 9 months)	None
Glycogen storage disease type IV	α-1,4-glucan 6-glucosyltransferase	± hypoglycemia	Hepatic cirrhosis, portal hypertension, growth retardation	Provide frequent feedings and complex carbohydrates; provide high protein except in cirrhosis; supplement with uncooked cornstarch (after age 9 months)	None
Glycogen storage disease type V	Muscle phosphorylase		Muscle weakness and cramping	Provide high protein diet; supplement with L-alanine	None

Table 31–1. continued

Disorder	*Enzyme Affected*	*Biochemical Findings*	*Clinical Features*	*Nutritional Modification*	*Vitamin Therapy*
Disorders of Fatty Acid Oxidation					
VLCAD deficiency LCHAD deficiency	Very long chain acyl-CoA dehydrogenase long chain hydroxyacyl-CoA dehydrogenase	Hypoketotic hypoglycemia, ± hyperammonemia	Cardiomyopathy, failure to thrive, hypotonia, hepatomegaly, lethargy, coma	Fasting avoidance; ± long chain fat restriction (15% of energy), MCT oil, ± carnitine and ± essential fatty acid supplementation	None
MCAD deficiency	Medium chain acyl-CoA dehydrogenase	Hypoketotic hypoglycemia, mild hyperammonemia, metabolic acidosis	Metabolic decompensation with fasting (lethargy, vomiting, coma), hepatomegaly	Fasting and MCT avoidance; ± long chain fat restriction (20–25% of energy); ± carnitine and ± essential fatty acid supplementation	None
SCAD deficiency SCHAD deficiency	Short chain acyl-CoA dehydrogenase short chain hydroxyacyl-CoA dehydrogenase	Ketotic hypoglycemia, hyperammonemia, metabolic acidosis	Poor feeding, vomiting, failure to thrive; ± developmental delay	Fasting avoidance; ± long chain fat restriction (20–25% of energy), ± carnitine and ± essential fatty acid supplementation	None

FTT = failure to thrive; NTBC = 2-(nitro-4-trifluoromethylbenzoyl)-1,3-cyclohexanedione; CoA = coenzyme A; MCT = medium chain triglycerides.

Table 31–2. Formulas for the Nutritional Support of Metabolic Disorders

		Intended for Use By					
Disorder	*Formula*	*Infant*	*Child*	*Adult*	*Protein (g/100 g)*	*Fat*	*Vitamins/Minerals*
Phenylketonuria	Periflex (SHS)		x	x	20		
	Phenex 1 (Ross)	x	< 4 yr		15		
	Phenex 2 (Ross)		x	x	30		
	PhenylAde (AN)		x	x	25		
	PhenylAde Amino Acid Blend (AN)		x	x	76	none	none*
	Phenyl Free-1 (Mead J)	x			16		
	Phenyl Free-2 (Mead J)		x	x	22		
	Phenyl Free-2 HP (Mead J)		x	x	40		
	Phlexy 10 powder (SHS)†		x	x	42	none	none*
	PKU 1 (Milupa)	x			50	none	no selenium
	PKU 2 (Milupa)		x		67	none	no selenium
	PKU 3 (Milupa)			x‡	68	none	no selenium
	XP Analog (SHS)	x			13		
	XP Maxamaid (SHS)		x		25	none	
	XP Maxamum (SHS)		> 8 yr	x‡	39	none	
Tyrosinemia	TYR1 (Milupa)	x			47	none	no selenium
	TYR2 (Milupa)		x	x	63	none	no selenium
	Tyrex 2 (Ross)		x	x	30		
	Tyromex 1 (Ross)	x	< 4 yr		15		
	Tyros-1 (Mead J)	x			17		

Table 31–2. continued

		Intended for Use By					
Disorder	*Formula*	*Infant*	*Child*	*Adult*	*Protein (g/100 g)*	*Fat*	*Vitamins/Minerals*
	Tyros-2 (Mead J)		x	x	22		
	XPHEN TYR Analog (SHS)	x			13		
	XPHEN TYR Maxamaid (SHS)		x	x	25	none	
Homocystinuria	HCY-1 (Mead J)	x			16		
	HCY-2 (Mead J)		x	x	22		
	Hominex 1 (Ross)	x	< 4 yr		15		
	Hominex 2 (Ross)		x	x	30		
	HOM 1 (Milupa)	x			52	none	no selenium
	HOM 2 (Milupa)		x	x	69	none	no selenium
	XMET Analog (SHS)	x			13		
	XMET Maxamaid (SHS)		x		25	none	
	XMET Maxamum (SHS)		> 8 yr	x	39	none	
Maple syrup urine disease	Ketonex 1 (Ross)	x	< 4 yr		15		
	Ketonex 2 (Ross)		x	x	30		
	MSUD-1 (Mead J)	x			16		
	MSUD-2 (Mead J)		x	x	22		
	MSUD1 (Milupa)	x			41	none	no selenium
	MSUD2 (Milupa)		x	x	54	none	no selenium
	MSUD Analog (SHS)	x			13		
	MSUD Maxamaid (SHS)		x		25	none	
	MSUD Maxamum (SHS)		> 8 yr	x	39	none	

Organic acidemia	OA-1 (Mead J)	x			16		
	OA-2 (Mead J)		x	x	22		
	OS1 (Milupa)	x			42	none	no selenium
	OS2 (Milupa)		x	x	56	none	no selenium
	Propimex 1 (Ross)	x	< 4 yr		15		
	Propimex 2 (Ross)		x	x	30		
	XMTVI Analog (SHS)	x			13		
	XMTVI Maxamaid (SHS)		x		25	none	
	XMTVI Maxamum (SHS)		> 8 yr	x	39	none	
Urea cycle disorders	Cyclinex 1 (Ross)	x	< 4 yr		7.5		
	Cyclinex 2 (Ross)		x	x	15		
	UCD-1 (Mead J)	x			6.5		
	UCD-2 (Mead J)		x	x	8		
	UCD1 (Milupa)	x			56	none	no selenium
	UCD2 (Milupa)		x	x	67	none	no selenium
Carbohydrate restriction	RCF (Ross)	x	x	x	4		
Protein restriction	PFD-1 (Mead J)	x			0		
	PFD-2 (Mead J)		x	x	0		
	ProPhree (Ross)	x	x	x	0		

*Vitamin packets sold separately.
†Also available in prepacked capsules and fruit-flavored bars.
‡Designed for use in pregnancy.
Mead J = Mead Johnson Nutritionals, Evansville, IN; Ross = Ross Products, Columbus, OH; SHS = SHS North America, Gaithersburg, MD; AN = Applied Nutrition Corp, Randolph, NJ; Milupa = Distributed in the United States by Mead Johnson.

Adequate energy intake from nonprotein sources is essential to provide for growth and minimize tissue catabolism that can lead to poorer metabolic control. Table foods such as fruits, vegetables, and limited grain products are supplemented with concentrated sweets and fats to provide adequate calories. Use of special low protein foods (pasta, breads, baked products) helps provide additional energy and variety in the diet without significantly increasing protein intake. Inadequate caloric intake may result from diet restrictions that severely limit food choices, unpleasant taste of medical foods containing L-amino acids, and poor appetite.

Recommended vitamin and mineral intakes follow the RDA guidelines. In low protein diets where chemically defined formulas provide the majority of protein intake, the variety of natural foods is very limited. Intake of vitamins and minerals needs to be monitored. Low plasma levels of ferritin, zinc, and retinol have been reported.[2,3] Pharmacologic doses of vitamins, which function as cofactors to enzymes, are useful in some metabolic disorders.

Phenylketonuria (PKU) is the most common amino acid disorder. In PKU there is a defect in the enzyme phenylalanine hydroxylase, which converts phenylalanine to tyrosine. The recommended diet is restricted in phenylalanine (substrate) and supplemented with tyrosine (product). Early treatment of PKU prevents severe mental retardation. This diet should be continued for life as learning difficulties and behavioral problems have been reported in children who have discontinued the diet or have poor dietary control.[4] Age-appropriate tasks that help children develop the knowledge and skills necessary to manage their diet successfully as an adult are outlined in Table 31–3.

Pregnant women with PKU who do not follow phenylalanine-restricted diets experience an increased incidence of low birth weight, microcephaly, cardiac anomalies, and

Table 31–3. Developmental Steps Toward Dietary Independence in Phenylketonuria

Toddlers (2–3 years)
Drinks formula out of a cup
Helps prepare formula (pours, stirs)
Names foods
Knows *yes* and *no* foods
Asks before eating foods he/she is uncertain of
Is aware of difference in diet from family and friends
Preschool (4–6 years)
Prepares formula (with assistance)
Knows phenylalanine intake is limited
Explains PKU diet in simple terms
Knows basic reasons for his/her clinic visits
Begins to deal with diet restrictions in social situations
School age (7–10 years)
Prepares formula (with supervision)
Reports foods eaten
Takes blood sample with assistance
Explains PKU and PKU diet
Knows what blood phenylalanine levels are safe and how to maintain them
Makes appropriate diet choices in social situations
Adolescence (11+ years)
Prepares formula independently
Keeps diet diary independently
Calculates daily phenylalanine intake
Prepares low-protein recipes
Takes blood samples independently
Knows the genetics of PKU
Copes with social pressures pertaining to PKU and the PKU diet
Understands the issue of maternal PKU

mental retardation in their offspring. These problems are believed to result from the effects of high maternal blood phenylalanine level on the developing fetus and are prevented by a strict phenylalanine-restricted diet implemented prior to conception.

Organic Acidemias

Organic acidemias are inherited enzyme deficiencies

affecting the catabolic pathways of amino acids. The disorders propionic acidemia and methylmalonic acidemia are caused by defects in the enzymes propionyl-CoA carboxylase and methylmalonyl-CoA mutase, respectively. Clinical symptoms of the neonatal form of organic acidemias include vomiting and dehydration, poor feeding, failure to thrive, hypotonia, metabolic ketoacidosis, and hyperammonemia. Immediate treatment is necessary to correct metabolic imbalances. Information on long-term outcomes is limited. Children show varying degrees of growth retardation and neurologic impairment.

Long-term nutritional management of organic acidemias involves restricting the offending essential amino acids to the minimum requirement. In propionic acidemia and methylmalonic acidemia, the amino acids isoleucine, methionine, threonine, and valine are restricted, usually through the use of a chemically defined formula supplemented with small amounts of natural protein from standard formulas and table foods. Adequate nonprotein energy is essential to prevent tissue catabolism. Carnitine supplementation is recommended. The intermittent use of antibiotics helps reduce gut bacteria loads, a major source of endogenous propionate production. Some patients with methylmalonic acidemia respond to pharmacologic doses of vitamin B_{12}. Dietary management may be complicated by poor appetite, necessitating nasogastric or gastrostomy tube feedings. Frequent monitoring of growth, laboratory values, and nutrient intake is important.

During illness, acute metabolic decompensation can occur from catabolism of amino acids stored as protein, resulting in metabolic acidosis, hyperammonemia, and ketonuria. Nutritional therapy consists of discontinuing protein, providing adequate calories in the form of glucose to suppress gluconeogenesis, and increasing fluid to prevent dehydration and assist in removal of abnormal metabolites in the urine. Intravenous dextrose or protein-

free formula are used for 24 to 48 hours to lower ammonia levels. Protein is gradually reintroduced. Inadequate energy intake or use of a protein-free diet for more than two days can lead to protein catabolism and rebound ketoacidosis and hyperammonemia. Parenteral nutrition can be used when enteral feedings are not tolerated, with protein supplied through specially formulated amino acid mixtures or with a standard solution providing 0.5 g protein/kg body weight.

Urea Cycle Disorders

Urea cycle disorders result from a defect in one of the enzymes involved in the conversion of ammonia to urea in the liver. Hyperammonemia is common to all the disorders. The enzyme defects include: (1) carbamyl phosphate synthetase (CPS) deficiency, (2) ornithine transcarbamylase (OTC) deficiency, (3) citrullinemia (argininosuccinic acid synthetase deficiency), (4) argininosuccinic aciduria (argininosuccinic acid lyase deficiency), and (5) argininemia (arginase deficiency). Infants present with hyperammonemia, poor feeding, vomiting, and hypotonia, which may progress to seizures, coma, and death. Rapid diagnosis and treatment is important. Long-term outcome and intellectual development in urea cycle disorders is variable.

The long-term goals of nutritional therapy are to reduce ammonia levels to normal by restricting protein intake, to provide sufficient nitrogen for optimal growth, and to provide adequate calories to prevent catabolism. Dietary protein is limited, with chemically defined formula mixtures of essential amino acids often used to provide approximately 50% of the protein. Low-protein products help provide additional calories and variety in the diet. Supplementation of L-arginine is required in all of the urea cycle defects except arginase deficiency. Sodium benzoate and sodium phenylacetate or sodium phenylbu-

tyrate are used to provide alternate pathways for waste nitrogen excretion. Growth, laboratory values, and nutrient intake must be monitored frequently. Dietary management may be complicated by poor appetite.

Catabolism during illness can lead to life-threatening hyperammonemia. Nutritional therapy during acute metabolic crisis consists of discontinuing protein and providing intravenous fluids and glucose to correct dehydration and provide energy. Intravenous dextrose or protein-free formula are used for 24 to 48 hours to decrease ammonia levels; protein is then gradually reintroduced. Inadequate energy intake or use of a protein-free diet for more than 2 days can lead to protein catabolism and rebound hyperammonemia.

Galactosemia

Galactosemia is an inherited disorder of galactose metabolism resulting from a defect in one of the enzymes required to convert galactose to glucose. The most common defect is in the galactose-1-phosphate uridyltransferase (GALT) enzyme. Symptoms of vomiting, diarrhea, failure to thrive, jaundice, hepatomegaly, cataracts, and *Escherichia coli* sepsis are usually seen within the first 2 weeks of life. Galactosemia is treated by restricting dietary galactose. Because lactose is hydrolyzed into galactose and glucose, both lactose and galactose must be eliminated from the diet. Galactosemia is therefore one of the few true contraindications to breastfeeding. Dietary restrictions should be followed for life.

Infants with galactosemia are fed soy-based formulas. Older children must avoid milk and milk products as well as incidental sources of lactose found in prepared foods and in medications. Labels on all processed foods and on medications should be checked to avoid ingredients such as whey, casein, nonfat dry milk, milk solids, lactose, lac-

toglobulin, lactalbumin, caseinate, and hydrolyzed protein. Organ meats and legumes should be avoided. Certain fruits and vegetables may also contain substantial amounts of galactose.[5] The availability of galactose from these foods is not known; whether they should be eliminated from the diet remains a matter of debate.

Early treatment with a galactose-restricted diet prevents neonatal sepsis, corrects liver disease, and causes regression of cataracts; dietary treatment, however, does not guarantee a normal long-term outcome. Even with good dietary control, many children have speech and visual-perception problems. Growth may be stunted and primary ovarian failure is seen in most females. Reduced calcium intake from elimination of dairy products leads to decreased bone density. Calcium supplements are usually required.

Glycogen Storage Disease

Glycogen storage diseases (GSD) are disorders in which glycogen cannot be metabolized to glucose because of an abnormality in the enzymes involved in glycogenolysis. The major sites of glycogen deposition are liver and muscle tissue. Clinical manifestations include hypoglycemia, hepatomegaly, poor growth, muscle weakness, cramping, and fatigue. The most common types of GSD that respond to nutritional therapy are GSD type I and GSD type III. The goal of dietary treatment is to prevent hypoglycemia.

Glycogen storage disease type I (glucose-6-phosphatase deficiency) results from a deficiency in the enzyme glucose-6-phosphatase, which is needed for the production of glucose from both glycogenolysis and gluconeogenesis. Biochemical abnormalities include hypoglycemia, hyperlipidemia, hyperuricemia, and lactic acidemia. Because endogenous glucose production is limited, nutritional therapy involves supplying a constant exogenous source of glucose to prevent hypoglycemia. The diet should be high in

complex carbohydrates, with an energy distribution of 60 to 70% carbohydrate, 10 to 15% protein, and the remainder as fat. Frequent daytime feedings are required. Since patients cannot metabolize fructose and galactose, the diet is limited in dairy products, fruits, and simple carbohydrates. Vitamin and mineral supplements are often necessary.

Continuous overnight nasogastric or gastrostomy feedings are used to prevent nocturnal hypoglycemia. Patients should eat immediately after the overnight feeding has been discontinued. An alternative approach uses oral doses of uncooked cornstarch (UCS) every 4 to 6 hours to provide a continuous source of glucose. The UCS doses are calculated using 1.75 to 2.5 g/kg body weight per dose. The UCS is mixed in cool water or a sugar-free beverage. Use of UCS is not recommended in infants under 9 months of age as pancreatic amylase activity may be insufficient.

Glycogen storage disease type III (debrancher enzyme deficiency) results from a deficiency of the enzyme amylo-1,6-glucosidase. Clinical manifestations are generally less severe than in GSD type I and include fasting ketosis, less significant hypoglycemia and hyperlipidemia, and the absence of lactic acidemia and hyperuricemia. These patients are able to synthesize glucose through gluconeogenesis.

Diets high in protein have been advocated to provide adequate substrate for gluconeogenesis, with energy distributions of 25% protein, 45% carbohydrate, and 30% fat. Frequent high protein, low carbohydrate feedings are provided during the day, with a high protein snack at night. Continuous overnight feedings may be necessary in infants and young children.

Fatty Acid Oxidation and Carnitine Transport Defects

Fatty acid oxidation defects are inborn errors of fatty acid metabolism usually impairing the production of ketones

as an energy source for the brain and other organs. Fatty acid oxidation defects often present following a period of fasting, febrile illness, or increased muscle activity. Features include encephalopathy, hypoketotic hypoglycemia, cardiomyopathy, episodic vomiting, liver dysfunction, and muscle weakness. Age of presentation varies but these defects often occur in infancy.

Defects have been identified in enzymes involved in the transport of long chain fatty acids by carnitine into the mitochondria as well as in the mitochondrial fatty acid beta-oxidation cycle. Transport defects include carnitine transporter defect (CTD), carnitine-acylcarnitine translocase deficiency, and carnitine palmitoyl transferase deficiency (CPT 1, CPT 2). Identified defects in mitochondrial fatty acid oxidation include very long chain acyl-CoA dehydrogenase (VLCAD) deficiency, long chain acyl-CoA dehydrogenase (LCAD) deficiency, medium chain acyl-CoA dehydrogenase (MCAD) deficiency, short chain acyl-CoA dehydrogenase (SCAD) deficiency, multiple acyl-CoA dehydrogenase deficiency (glutaric acidemia type II), long chain 3-hydroxyacyl CoA dehydrogenase (LCHAD) deficiency, short chain 3-hydroxyacyl-CoA dehydrogenase (SCHAD) deficiency, and medium chain 3-ketoacyl-CoA thiolase (MCKAT) deficiency. The most common of these disorders, MCAD deficiency, has been associated with sudden infant death syndrome.

The goal of treatment in the acute phase is to provide sufficient glucose to correct hypoglycemia and reduce the need to use ketones as a substrate for energy. Long-term management involves avoidance of prolonged fasting and increased intake of carbohydrate calories during periods of increased energy demand. Frequent meals and snacks that are high in carbohydrate are used during the day. Overnight fasting longer than 6 hours in infants or 8 to 12 hours in children should be avoided. Use of uncooked

cornstarch (1.5 to 2.0 g/kg body weight) to delay onset of fasting, or overnight feedings, may be helpful. Low fat diets may be prescribed. In defects of long chain fatty acid oxidation, diets which are restricted in long chain fat can be supplemented with medium chain triglycerides. To prevent essential fatty acid deficiency in low fat diets, linoleic acid and alpha-linolenic acid should provide 3% and 1% of total energy, respectively. Carnitine supplementation is indicated in carnitine transport defects but remains controversial in the management of other fatty acid oxidation defects.

Mitochondrial Disorders

Mitochondrial disorders are a group of diseases that affect mitochondrial energy metabolism. Mitochondrial disorders result in decreased energy production and impaired body functioning. Age of presentation varies. They can affect virtually any organ or tissue and are often multisystem in nature. They are progressive and usually result in significant disabilities. Mitochondrial disorders show a wide range of symptoms, including seizures, developmental delay, autonomic nervous system dysfunction (breathing problems, temperature instability, diarrhea, or pseudoobstruction), cardiomyopathy, hepatic and renal dysfunction, muscle weakness, gastrointestinal dysmotility, endocrine abnormalities such as diabetes, vision and hearing problems, and poor growth.

Treatment is mainly supportive and is based on individual symptoms. Muscle fatigue, developmental delay, gastroesophageal reflux, and poor oropharyngeal coordination all predispose to poor intake. Undernourished states may produce symptoms that suggest an accelerated deterioration in the status of the patient. Attention to adequate nutrition is essential to maintain optimal growth, development, and level of functioning in these patients.

Carnitine and vitamin supplementation have been used with varying degrees of success. Supplements may include carnitine (50 to 100 mg/kg body weight), coenzyme Q (4.3 mg/kg body weight), vitamin C (50 to 1000 mg), thiamin (100 mg), and riboflavin (100 mg).

References

1. Acosta P, Yannicelli S. The Ross Metabolic Formula System Nutrition Support Protocols. 3rd ed. Columbus (OH): Ross Products Division; 1997.
2. Acosta PB, Fernhoff PM, Warshaw HS, et al. Zinc status and growth of children undergoing treatment for phenylketonuria. J Inherit Metab Dis 1982;5:107–10.
3. Acosta PB. Nutrition studies in treated infants and children with phenylketonuria: vitamins, minerals, trace elements. Eur J Pediatr 1996;155 Suppl:136–9.
4. Azen C, Koch R, Friedman E, et al. Summary of findings from the United States Collaborative Study of children treated for phenylketonuria. Eur J Pediatr 1996;155 Suppl:29–32.
5. Gross KC, Acosta PB. Fruits and vegetables are a source of galactose: implications in planning the diets of patients with galactosemia. J Inherit Metab Dis 1991;14:253–8.

Additional Resources

Books

Scriver CR, Beaudet AL, Sly WS, editors. The metabolic and molecular basis of inherited disease. 7th ed. New York: McGraw-Hill; 1995.

Acosta P, Yanicelli S. The Ross Metabolic Formula System Nutrition Support Protocols. 3rd ed. Columbus (OH): Ross Products Division; 1997.

Medical Food Resources

Applied Nutrition 1-800-605-0410

Mead Johnson Nutritionals 1-800-457-3550

Ross Products
Metabolic Hot-Line 1-800-986-8755
To Order Formula 1-800-551-5838

SHS North America 1-800-365-7354

Low Protein Food Resources

Dietary Specialties/Menu Direct
865 Centennial Dr.
Piscataway, NJ 08854
1-888-MENU123

EnerG Foods, Inc.
PO Box 84487
Seattle, WA 98124
1-800-331-5222

Med-Diet Laboratories, Inc.
3050 Ranchview Ln.
Plymouth, MN 55447
1-800-633-5580

SHS North America
PO Box 117
Gaithersburg, MD 20878
1-888-LOPROGO

Internet Resources

National PKU News: www.pkunews.org
National Organization for Rare Diseases: www.rarediseases.org
United Mitochondrial Disease Foundation: www.umfd.org

32

OBESITY

Linda Gallagher Olsen, MEd, RD, and Jan P. Hangen, MS, RD

Obesity, the excessive accumulation of body fat, is a leading cause of morbidity and mortality in the United States and its incidence among youth is increasing. Although prevalence statistics vary depending on the assessment standard used, it is now estimated that between 18 to 28% of children and adolescents in the United States are obese.[1] Obesity among the pediatric population has increased by at least 50% since 1976.[2] As noted in Table 32–1, there are many health conditions associated with childhood obesity.

It is estimated that between 25 and 74% of obese children and adolescents will become obese adults.[3] The Harvard Growth Study found that mortality risks associated with adolescent onset of obesity were greater than the health risks associated with adult onset of obesity.[4]

Etiology

Endocrine and genetic disorders account for only a small percentage of childhood obesity. The role of heredity is significant, with an increased risk of obesity among children of obese parents, probably as a result of genetic and shared environmental factors. For children under the age of 10 years, parental obesity more than doubles the risk of becoming an obese adult.[5] Modifiable environmental components, such as food intake and physical activity, are also major contributors to obesity status.

Table 32–1. Health Conditions Associated with Childhood Obesity

System	*Condition*
Cardiovascular	Hypertension, hypercholesterolemia, hypertriglyceridemia, increased LDL and VLDL, decreased HDL
Endocrine	Hyperinsulinism/insulin resistance, non–insulin-dependent diabetes mellitus, acanthosis nigricans, early puberty and menarche, decreased testosterone, Cushing's syndrome, hypothyroidism
Gastrointestinal	Cholecystitis, steatohepatitis, gastroesophageal reflux, abdominal pain, gallstones
Pulmonary	Pickwickian syndrome, obstructive sleep apnea, primary alveolar hypoventilation
Musculoskeletal	Slipped capital femoral epiphysis, Blount's disease, osteoarthritis
Neurologic	Recurrent headaches, pseudotumor cerebri
Genetic Factors	Prader-Willi, Laurence-Moon-Biedl syndromes
Psychologic	Depression, poor self-image, peer rejection

LDL = low-density lipoproteins; VLDL = very low-density lipoproteins; HDL = high-density lipoproteins.
Adapted from Dietz WH, Robinson TN. Assessment and treatment of childhood obesity. Pediatr Rev 1993;14:341.

Measurement and Assessment

Assessment of an overweight child should include a thorough medical evaluation, review of serial growth points, dietary history with discussion of food frequency and family eating patterns, evaluation of psychosocial status, and an inquiry about physical activity.

Since direct measures of body fat mass are impractical and expensive, indirect measurements are typically used. Body mass index (BMI), calculation of percent ideal body

weight, and skinfold measurements are the most commonly used indirect assessment tools (Table 32–2).

Body mass index is defined as weight in kilograms divided by height in meters squared (kg/m^2). It is a clinically important measure of body fat since it controls to some degree for the influence of height, allowing comparison of obesity status across age groups. It does not, however, take into account lean body mass or pubertal status. Despite some shortcomings, BMI has become the preferred measure of obesity in clinical practice and research. Body mass index references have been established for ages 6 through 75 years (Tables 32–3 and 32–4). The 95th percentile has been suggested as the definition of obesity whereas children with BMI > 85th percentile for their age are generally considered at "risk" for developing obesity.

Another measure of obesity is a weight-for-height in excess of 120% of standard on the National Center for Health Statistics (NCHS) growth chart. Skinfold thick-

Table 32–2. Pediatric Obesity Assessment Methods and Reference Standards

Method	*Definition*
Body mass index	BMI > 95th percentile for age and sex
Triceps skinfold measurements	TSF > 95th percentile for age and sex
Relative weight	Mildly obese: 120–149% of IBW Moderately obese: 150–199% of IBW Severely obese: > 200% of IBW
Growth charts	Body weight increases of > 2 major percentile channels (NCHS growth chart)

TSF = triceps skinfold; IBW = ideal body weight; the 50th percentile of weight for children of the same height, age, and sex. Adapted from Brown DK. Childhood and adolescent weight management. In: Dalton S, editor. Overweight and weight management. New York: Aspen; 1997.

Table 32–3. 95th Percentile of Body Mass Index for Boys 5 to 17 Years of Age*

Age	Asian	African American	Hispanic	Caucasian	U.S. Weighted Mean	NHANES I†
5	17.1	18.1	19.4	18.1	18.3	–
6	17.8	18.8	20.2	18.9	19.0	18.0
7	18.8	19.9	21.2	19.9	20.0	19.2
8	20.2	21.3	22.7	21.4	21.5	20.3
9	21.7	22.9	24.4	23.0	23.1	21.5
10	23.2	24.4	25.9	24.5	24.6	22.6
11	24.3	25.5	27.1	25.6	25.7	23.7
12	25.1	26.3	27.9	26.4	26.5	24.9
13	25.6	26.9	28.5	27.0	27.1	25.9
14	26.3	27.6	29.2	27.6	27.8	26.9
15	27.2	28.5	30.1	28.5	28.7	27.8
16	28.2	29.6	31.2	29.6	29.8	28.5
17	28.6	29.9	31.6	30.0	30.1	29.3

Table 32–4. 95th Percentile of Body Mass Index for Girls 5 to 17 Years of Age*

Age	Asian	African American	Hispanic	Caucasian	U.S. Weighted Mean	NHANES I†
5	16.6	19.8	19.6	18.1	18.5	–
6	17.4	20.7	20.5	18.9	19.3	17.5
7	18.4	21.8	21.6	20.0	20.4	18.9
8	19.9	23.1	22.9	21.2	21.7	20.4
9	20.9	24.5	24.3	22.6	23.0	21.8
10	22.4	26.1	25.8	24.1	24.5	23.2
11	23.8	27.6	27.4	25.6	26.1	24.6
12	25.2	29.1	28.9	27.0	27.5	26.0
13	26.3	30.3	30.0	28.1	28.6	27.1
14	26.9	31.0	30.7	28.8	29.3	28.0

Table 32–4. continued

Age	Asian	African American	Hispanic	Caucasian	U.S. Weighted Mean	NHANES I†
15	27.2	31.3	31.0	29.1	29.6	28.5
16	27.5	31.6	31.4	29.4	29.9	29.1
17	28.8	33.0	32.8	30.8	31.3	29.7

NHANES = National Health and Nutrition Examination Survey.
*Adapted from Rosner B, Princeas R, Loggie J, Daniels S. Percentiles for body mass index in U.S. children 5 to 17 years of age. J Pediatr 1998;132:211–22.
†Adapted with permission from Must A, Dallal GE, Dietz WH. Reference data for obesity: 85th and 95th percentiles of body mass index (wt/ht^2) and triceps skinfold thickness. Am J Clin Nutr 1991;53:839–46. © American Society for Clinical Nutrition. (Note: this data was compiled from the NHANES I survey.)

ness, measured with calipers, provides an indication of subcutaneous body fat. Biceps and triceps skinfold measures are considered indicators of peripheral fat while subscapular and suprailiac measures tend to reflect central fat deposition. Excess central fat deposition is highly correlated with increased incidences of hypertension, hypertriglyceridemia, and glucose intolerance.

Caloric Requirements

Obese children have basal metabolic rates equal to or greater than their nonobese counterparts, due largely to the increased lean body mass needed to support the extra weight. The average caloric intake of children of various age groups based on median weight and height are shown in Chapter 5, Table 5–4. Because of variability in the timing and magnitude of the adolescent growth spurt, caloric recommendations are broad estimates of individual need. Current recommendations for energy intake may be excessive as total energy expenditure in younger children was found in one study to be about 25% lower than recom-

mended intake.[6] Ascertaining a detailed dietary history/recall will provide some insight into a child's actual intake, although children and adults have been found to underreport intake by as much as 40%.[7]

For many obese prepubertal children, a reasonable goal is to stabilize weight while linear growth continues to accelerate. When weight loss is desired, a reasonable goal is 1 to 2 pounds of weight loss/week. Reducing intake by 200 to 500 calories/day may achieve gradual weight loss in younger children while in older children and adolescents a 500 to 1000 calorie/day deficit may be required. This often translates to a 30 to 40% decrease in the usual caloric intake.[8]

Adjusted Body Weight

Among individuals of normal weight, basal metabolic rate can be calculated from standard equations or tables, using body weight, height, age, and gender. When applied to obese individuals, however, these equations are invalid since they assume a relatively fixed ratio of lean body mass (eg, metabolically active mass) to fat mass. Since this ratio is significantly altered in those individuals 125% heavier than ideal body weight, an "adjusted body weight" is preferred for calculating their basal metabolic rate,[9] as follows:

$$([ABW - IBW] \times 0.25) + IBW = \text{wt in kg for use in calculating BEE and protein requirement}$$

Where: ABW = actual body weight in kg; IBW = ideal body weight for height; 0.25 = 25% of body fat tissue is estimated to be metabolically active; BEE = basal energy expenditure.

It should be noted that this formula has been validated in adults but not in children.

Treatment

The goal of treatment for obesity is weight reduction and maintenance of a lower body weight to minimize health risks and improve quality of life. Treatment should be multidisciplinary and include counseling on diet, exercise, and behavior modification.

Nutrition education should focus on healthy food choices and include adequate protein, fat (≤ 30% of total kilocalories, with an emphasis on monounsaturated fatty acids), and enough carbohydrates for energy and to spare protein for growth and tissue repair. Fad diets should be discouraged owing to the risk of nutritional imbalances. There may be a variety of approaches used in nutrition education. For example, the United States Department of Agriculture (USDA) Food Guide Pyramid is often used as a tool to present an overall view of the suggested macronutrient composition of a diet. Approaches to nutrition education should be individualized to the child's treatment goals and lifestyle, however.

Emphasis should be placed on physical activity that is consistent, enjoyable, and in keeping with the patient's lifestyle. Exercise is helpful in maintaining weight loss and/or preventing further weight gain. Children who decrease the time they spend in sedentary activities exhibit greater decreases in body weight. Decreasing the amount of time children watch television, for example, may both limit excessive snacking and encourage increased physicial activity.

Behavior modification should include self-monitoring, family intervention and involvement, cognitive restructuring, and reinforcement. Table 32–5 summarizes these behavioral approaches and a variety of treatment methods for use in managing pediatric obesity. Many of these approaches include the multidisciplinary components of nutritional, behavioral, exercise, and medical intervention.

Table 32–5. Treatment of Childhood Obesity

Behavior Modification[12]

- Self-monitoring. The patient keeps a daily record of food consumed and physical activity expended
- Stimulus control. Internal and external cues and triggers associated with eating and overeating are considered
- Changing eating behavior. The patient learns about behaviors associated with eating and overeating so the behaviors may be modified
- Reinforcement. Healthy eating strategies are encouraged with reinforcers, rewards, and behavioral contracts
- Cognitive behavioral techniques. The patient develops alternative behaviors for eating and overeating to cope with high-risk situations

Nutritional Counseling[13]

- USDA Food Guide Pyramid/dietary guidelines approach[14]
  - Emphasizes low fat breads, cereals, and grains (6–11 servings/day)
  - Other dietary guidelines, including:
    - Eat a variety of foods that are low in calories and high in nutrients
    - Eat less fat and sugar-containing foods
    - Eat smaller portions and limit second helpings of foods high in fat and calories
    - Eat more vegetables and fruits
    - Eat pasta, rice, breads, and cereals without added fats and sugars
- Individually varied, nutritionally balanced approach
  - Macronutrient variation
    - Low fat diet
      - Less than 30% fat, with an emphasis on monounsaturated fats
    - Stoplight diet[15]
      - Foods are categorized as green (foods forming the basis of the diet), yellow (foods to be used cautiously), and red (foods to limit and/or decrease)
    - Low glycemic diet[16]
      - 40–50% carbohydrates, with an emphasis on low glycemic carbohydrates, 20–30% protein, and 30–40% fat, with an emphasis on monounsaturated fatty acids

Table 32–5. continued

Energy balance or deficit

Prescribed calorie level

Use of an exchange system (eg, Jenny Craig, Weight Watchers)

Very low calorie diets

Provides 400–800 calories/day in a nutritionally complete formula, often in the form of a shake

Very low calorie diets are not recommended for children due to the possibility of iatrogenic malnutrition

Protein-sparing modified fast[17]

A form of the very low calorie diet that also includes lean protein, particularly meat, fish, and poultry

The very low calorie diet and protein-sparing modified fast are both supplemented with vitamins and minerals, and all patients are supervised by medical professionals

Physical Activity and Exercise

Aerobic and anaerobic exercise

Minimum of 30 minutes three times/week, with special emphasis on enjoyment and family and/or peer participation

Lifestyle changes

Increased lifestyle physical activity (eg, walking, stairs)

Decreased sedentary activities (TV, computer)

Pharmacotherapy[18]

Appetite-suppressing agents combined with comprehensive behavioral therapy

No drugs are currently established to be safe and/or effective for the pediatric population

Surgery

Surgery (eg, gastric bypass, vertical banded gastroplasty, jejunoileal bypass) is rarely indicated in the pediatric population unless the patient is an adolescent with a significant comorbidity

School-Based Programs[19]

School lunch program

Macronutrient variation, with an emphasis on achieving ≤ 30% fat in diet

Portion control, with an emphasis on decreasing total calories

School-based obesity treatment

Table 32–5. continued

	Emphasizes health promotion aimed at reducing risk factors in the development of chronic diseases
Family-Based Programs[20]	
	Emphasizes separate treatment for parents and children, often in group settings
	Both nutrition and behavior modification are utilized

Unfortunately, the prognosis for the obese child is poor, with only 10 to 30% maintaining weight loss following weight reduction efforts.[10] Frequent follow-up, particularly for nutritional and behavioral counseling, is recommended to optimize successful weight loss.[11]

References

1. Roberts SB, Vinken AG. Energy and substrate regulation in obesity. In: Walker WA, Watkins JB, editors. Nutrition in pediatrics. 2nd ed. Hamilton (ON): B.C. Decker, Inc.; 1996. p. 716–23.
2. Schonfeld-Warden N, Warden C. Pediatric obesity. Pediatr Clin North Am 1997;44:339–61.
3. Dietz WH, Robinson TN. Assessment and treatment of childhood obesity. Pediatr Rev 1993;14:337–44.
4. Must A, Jacques PF, Dallal GE, et al. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 1992;327:1350–5.
5. Whitaker RC, Wright JA, Pepe MS, et al. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;337:869–73.
6. Goran MI, Figueroa R, McGloin A, et al. Obesity in children: recent advances in energy metabolism and body composition. Obes Res 1995;3:277–89.
7. Bandini L, Schoeller DA, Cyr HN, Dietz WH. Validity of reported energy intake in obese and nonobese adolescents. Am J Clin Nutr 1990;52:421–5.

8. Figueroa-Colon R, Franklin FA, Lee JY, et al. Feasibility of a clinic-based hypocaloric dietary intervention implemented in a school setting for obese children. Obes Res 1996;4:419–29.
9. American Dietetic Association. Manual of dietetics. Adjustment in body weight for obese patients. Chicago: Chicago Dietetic Association and South Suburban Dietetic Association; 1989. Appendix 48:622–3.
10. American Academy of Pediatrics. Committee on Nutrition. Nutritional aspects of obesity in infancy and childhood. Pediatrics 1981;68:880–3.
11. Epstein LH. Methodological issues and ten-year outcomes for obese children. Ann N Y Acad Sci 1993;699:237–49.
12. Epstein LH, Wing RR. Behavioral treatment of childhood obesity. Psychol Bull 1987;101:331–42.
13. Haddock CK, Shadish WR, Slesges RC, Stein RJ. Treatments for childhood and adolescent obesity. Ann Behav Med 1994; 16:235–44.
14. Nutrition and your health: dietary guidelines for Americans 4th ed. Washington (DC): Dept. of Agriculture (US), Dept. of HHS; 1995 Home and Garden Bulletin No.:232.
15. Epstein LH, Squires S. The stop-light diet for children. Boston: Little, Brown and Company; 1998.
16. Wolever TMS, Jenkins DJA, Jenkins AL, Josse RG. The glycemic index: methodology and clinical implications. Am J Clin Nutr 1991;54:846–54.
17. Suskind RM, Sothern, MS, Farris, RP, et al. Recent advances in the treatment of childhood obesity. Ann N Y Acad Sci 1993;699:181–99.
18. Dietz WH. Pharmacotherapy for childhood obesity? Maybe for some. Obes Res 1994;2:54–5.
19. Resnicow K. School-based obesity prevention: population versus high-risk intervention. Ann N Y Acad Sci 1993;699: 154–66.
20. Brownell DK, Kelman JH, Stunkard AJ. Treatment of obese children with and without their mothers: changes in weight and blood pressure. Pediatrics 1981;71:515–23.

33

ONCOLOGY AND BONE MARROW TRANSPLANTATION

Lori J. Bechard, MEd, RD, CNSD

Cancer in children comprises a group of diseases of abnormal cell growth. The most common type of pediatric cancer is acute lymphocytic leukemia (ALL),[1] accounting for 23% of cancer in children under 15 years of age, followed by central nervous system tumors, which account for 21% of pediatric cancers.[2] Due to advances in medical treatment, approximately 65% of children with cancer will survive disease-free for more than 5 years. Treatment for refractory or high-risk cancers may include bone marrow transplantation (BMT). This may also be indicated for treating a variety of nonmalignant diseases, such as aplastic anemia, severe combined immunodeficiency disease, and lysosomal storage diseases.[3] The success of BMT is related to the stage and type of disease as well as the conditioning regimen and transplant itself.

Nutrition intervention may be indicated to prevent or correct abnormalities in growth caused by active disease or cancer treatment. Although there are many reasons why the pediatric oncology patient may be malnourished, malnutrition need not be accepted as an unavoidable consequence of cancer and/or its therapy. Long-term survivors of childhood leukemia may also benefit from healthy diet and lifestyle education, given their tendency to become obese adults.[4]

Nutrition Risk Factors

Cancer treatment in pediatric patients includes chemotherapy, radiation, and surgery. This treatment may cause a degree of anorexia and poor tolerance to oral intake that is more severe than the effects of the cancer alone.[5] Since the type and stage of neoplasm largely determine the prescribed treatment, nutritional risk can be also characterized by disease (Table 33–1). Solid tumors and tumors of the gastrointestinal tract are more likely to present with protein-energy malnutrition than are cancers of blood-forming cells.

Pediatric cancers are often treated with a combination of agents. Side effects of cancer treatment combined with the effects of the malignancy itself cause complications that can interfere with optimal nutritional status. Cachexia is defined as a state of ill health, malnutrition, and wasting caused by malignancy. The multifactorial causes of cancer cachexia are presented in Figure 33–1.

Chemotherapeutic agents have a variety of side effects that have an impact on nutritional intake and metabolism.

Table 33–1. Relative Risk of Pediatric Cancer Subtypes

High Nutritional Risk	*Low Nutritional Risk*
Wilms' tumor—stage III and IV	Nonmetastatic solid tumors
Neuroblastoma—stage III and IV	Low-risk ALL
Rhabdomyosarcoma	Disease in remission
Ewing's sarcoma	
Acute nonlymphoblastic leukemia	
Multiple relapse leukemia	
Medulloblastoma	

Adapted from Yu CL. Nutrition and childhood malignancies. In: Suskind RM, Lewinter-Suskind L, editors. Textbook of pediatric nutrition. 2nd ed. New York: Raven Press, Ltd.; 1993.

Examples of commonly used chemotherapies and their notable toxicities are presented in Table 33–2.

The effects of other medicines used in treating a child's cancer may compound the effects of chemotherapy. Steroids such as prednisone or dexamethasone are often used in treating leukemia or graft-versus-host disease (GVHD) in the post-BMT patient. Cyclosporine and tacrolimus are also used as immunosuppressive agents for GVHD prophylaxis. See Appendix B for further discussion of drug-nutrient interactions.

Radiation and surgery are frequently components of treatment for pediatric cancer. Surgery often involves placement of central venous catheters for access, placement of enteral feeding devices, or resection of tumor.

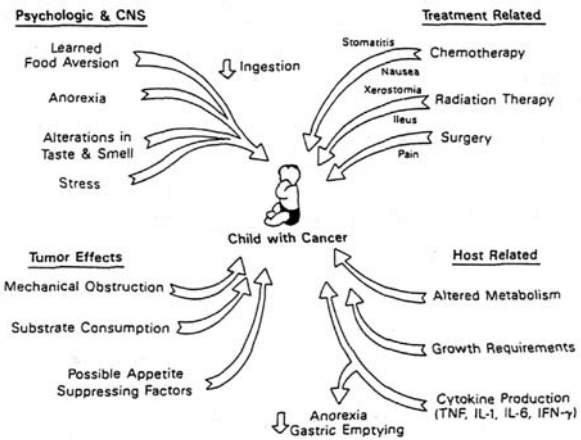


Figure 33–1. Etiology of cachexia in a child with cancer. TNF = tumor necrosis factor; IL = interleukin; IFN = interferon. Reproduced with permission from Alexander HR, Rickard KA, Godshall, B. Nutritional supportive care. In: Pizzo PA, Poplack DG, editors. Principles and practice of pediatric oncology. 3rd ed. Philadelphia: Lippincott-Raven Publishers; 1997.

Table 33–2. Side Effects of Common Chemotherapeutic Agents used in Pediatric Oncology

Agent	*Indication*	*Side Effects*
Bleomycin	Lymphoma, testicular, and other germ cell	Lung, skin, mucositis, hypersensitivity, altered taste/smell
Busulfan	CML, leukemias (BMT)	N&V, mucositis, neurotoxicity, pulmonary, hepatic (high dose)
Carboplatin	Brain tumors, germ cell, neuroblastoma, sarcomas	N&V, hepatic (mild)
Cisplatin	Germ cell, osteosarcoma, brain tumors, neuroblastoma	N&V, renal, neurotoxicity, ototoxicity
Cyclophosphamide	Lymphomas, leukemias, sarcomas, neuroblastoma	N&V, cystitis, water retention, cardiac (high dose), altered taste/smell
Cytarabine	Leukemia, lymphoma	N&V, mucositis, diarrhea, GI toxicity, flu-like syndrome, neurotoxicity, ocular, skin (high dose)
Dactinomycin	Wilms' tumor, sarcomas	N&V, mucositis (can be severe), hepatic, diarrhea
Daunomycin	Leukemia (ALL, ANL), lymphomas	Mucositis, N&V, diarrhea, cardiac (acute and chronic)
Doxorubicin	Leukemia (ALL, ANL), lymphomas, most solid tumors	Mucositis (can be severe), N&V, diarrhea, cardiomyopathy
Etoposide	Leukemias (ALL, ANL), lymphomas, neuroblastoma, sarcomas, brain tumors	N&V, mucositis, mild neurotoxicity, hypotension
Fluorouracil	Carcinomas, hepatic tumors	Mucositis, N&V, diarrhea,skin, neurotoxicity, ocular, cardiac

Table 33–2. continued

Agent	*Indication*	*Side Effects*
Idarubicin	Leukemia (ALL, ANL), lymphomas	Mucositis, N&V, diarrhea, cardiac (acute and chronic)
Ifosfamide	Sarcomas, germ cell	N&V, cystitis, neurotoxicity, renal, cardiac (high dose)
L-asparaginase	Leukemia (ALL), lymphomas	Coagulopathy, pancreatitis, hepatic, neurotoxicity, hyperglycemia, altered taste/smell
Lomustine, Carmustine	Brain tumors, lymphoma, Hodgkin's disease	N&V, renal, pulmonary
Melphalan	Rhabdomyosarcoma, sarcomas, neuroblastoma, leukemias (high dose)	N&V, mucositis, diarrhea (high dose)
Mercaptopurine	Leukemia (ALL, CML)	Hepatic, mucositis, altered taste/smell
Methotrexate	Leukemia, lymphoma, osteosarcoma	Mucositis (severity increased with radiation), diarrhea, rash, hepatic, renal, neurotoxicity (high dose)
Procarbazine	Hodgkin's disease, brain tumors	N&V, neurotoxicity, rash, mucositis, altered taste/smell
Thioguanine	Leukemia (ANL)	N&V, mucositis, hepatic
Vincristine	Leukemia (ALL), lymphomas, most solid tumors	Neurotoxicity, SIADH, hypotension, ileus/constipation

N&V = nausea and vomiting; ALL = acute lymphocytic leukemia; ANL = acute nonlymphocytic leukemia; CML = chronic myelogenous leukemia; BMT = bone marrow transplantation; SIADH = syndrome of inappropriate antidiuretic hormone secretion; GI = gastrointestinal.

Adapted from Balis FM, Holcenberg JS, Poplack DG. General principles of chemotherapy. In: Pizzo PA, Poplack DG, editors. Principles and practice of pediatric oncology. 3rd ed. Philadelphia: Lippincott-Raven Publishers; 1997.

Side effects of surgery are usually short-term unless a significant amount of the gastrointestinal tract is resected, causing short gut syndrome. Combined with the effects of chemotherapy, radiation can cause significant nutritional risk for many children undergoing aggressive treatment (Table 33–3).

Special Aspects of Nutritional Assessment

The nutritional assessment of a child with cancer involves a thorough dietary history and anthropometric evaluation as well as a review of the current stage of disease and

Table 33–3. Nutrition-Related Side Effects of Radiation Therapy

Head and neck
Nausea, anorexia
Mucositis, esophagitis
Decreased taste and smell
Damage to developing teeth
Decreased salivation—thick, viscous mucous
Decreased jaw mobility
Thoracic
Pharyngeal and esophageal inflammation and cell damage
Sore throat, dysphagia
Abdominal or pelvic
Nausea, vomiting, diarrhea
Ulceration
Colitis
Malabsorption
Fluid and electrolyte imbalance
Total body
Nausea, vomiting, diarrhea
Mucositis, esophagitis
Decreased taste and salivation
Anorexia

Adapted from Barale KV. Oncology and marrow transplantation. In: Queen PM, Lang CE, editors. Handbook of pediatric nutrition. Gaithersburg (MD): Aspen Publishers, Inc.: 1993.

treatment. Calorie counts may reflect suboptimal intake of varying degrees. Psychosocial and medical intervention play an important role in improving oral intake. Issues to consider when planning nutritional support include past, current, and future treatment plans with respect to chemotherapy, radiation, surgery, and bone marrow transplantation. Although many patients are well nourished and in remission when presenting for transplantation, the preparative conditioning for BMT causes significant toxicities.[3] Many patients receiving BMT will require parenteral nutrition (PN) support to avoid acute malnutrition; the efficacy of PN has been demonstrated in this setting.[6] Nutrition-related complications commonly found in pediatric cancer patients are presented in Table 33–4.

Assessment of physical and laboratory characteristics provides additional information when formulating the nutritional care plan. Weight, height, weight for height, and arm anthropometry should be measured at baseline and at follow-up points to assess for change in nutritional status. Fluid shifts resulting from medical interventions or organ failure may complicate the interpretation of weight changes and arm anthropometrics. Usual laboratory indices for assessing nutritional status are utilized, with notice given to the effects of treatment modalities. Prior to initiating treatment and up to 5 days after, patients are at risk for tumor lysis syndrome. Tumor lysis syndrome results from the destruction of tumor cells and the release of their contents into the circulation.[7] Table 33–5 presents nutritional planning options for dealing with this metabolic crisis.

Special Aspects of Nutritional Management

Two methods for estimating energy requirements of pediatric cancer patients are (1) the Recommended Dietary Allowances and (2) estimated basal metabolic rate plus a broad range of 20 to 100% for effects of stress and catab-

Table 33–4. Etiology of Nutrition Related Complications in Childhood Cancer Treatment

	Malignancy	*Chemotherapy*	*Radiation*	*Surgery*	*BMT*
Anorexia	X	X	X		X
Infection		X	X		X
Diarrhea	X	X	X		X
Nausea and vomiting		X	Depends on site		X
Malabsorption	X	X	X	With significant gut resection	X
Blood loss	X	X			X
Renal damage		X			X
Ileus or intestinal obstruction	X	X		X	
Dysgeusia and xerostomia	X	X	X		X

Adapted from Mauer AM, Burgess JB, Donaldson SS, et al. Special nutritional needs of children with malignancies: a review. J PEN 1990;14:315–24.

Table 33–5. Nutritional Care Planning and Tumor Lysis Syndrome

Laboratory	*Prevention and Management Strategies*
Hyperkalemia	Minimize potassium intake Diuretics; avoid if hypovolemic
Hyperuricemia	Allopurinol Use of acetate salts in parenteral nutrition solutions Maintain urine pH at 7.0–7.5
Increased blood urea nitrogen	Aggressive hydration with added bicarbonate
Hyperphosphatemia	Phosphate binders
Hypocalcemia	Calcium repletion
Increased serum creatinine	Dialysis if conservative measures are ineffective

Adapted from Kelly KM, Lange B. Oncologic emergencies. Pediatr Clin North Am 1997;44:809–27.

olism.[6,8] The energy requirements of pediatric oncology patients have not been extensively studied. Acutely ill patients may require fewer calories due to less activity. In a study by Szeluga and colleagues, bone marrow transplant patients required 45 to 65 kcal/kg/day and 30 to 50 kcal/kg/day in children and adults, respectively, to achieve nitrogen balance.[9] True resting energy expenditure can be measured with indirect calorimetry; adequacy of feeding is best judged by appropriateness of weight gain in the patient with normal fluid status.

Many children with cancer are able to adequately support themselves with oral intake. The method of provision of nutrition support in patients at highest nutritional risk remains controversial. While it is agreed that the least invasive and most physiologic means of nutritional support should be used, some centers have had positive experiences with enteral nutrition (EN), even in the bone mar-

row transplant setting.[10] Unique situations that complicate the choice of nutrition support for the child unable to meet requirements orally include mucositis, neutropenia, psychologic perception, and effects of medications on the gastrointestinal tract.

Oral Nutrition

Table 33–6 presents several suggestions that may be helpful to the patient with cancer and their family.

Although there is little evidence to support its benefit, a diet low in bacterial content is often recommended during periods of profound neutropenia, particularly in the bone marrow transplant setting. Sanitary food practices

Table 33–6. Strategies for Improving Oral Intake during Cancer Treatment

Loss of appetite
Small frequent feedings (6–8 meals or snacks per day)
Encourage nutrient-dense beverages between meals
Offer favourite nutritious foods during treatment-free periods to prevent learned food aversions
Nausea and vomiting
Feed 3–4 hours before therapy that typically causes nausea and vomiting
Offer small amounts of cool foods and encourage slow eating; avoid strong odors
Offer clear liquids between meals; using a straw in a covered cup may facilitate sipping
Mouth sores
Serve soft or pureed bland food or liquids
Add butter, gravy, sauce, or salad dressing to moisten foods
Avoid highly seasoned or hard, rough foods
Altered taste perception
Use stronger seasonings; avoid excessively sweet foods
Offer salty foods, eg, hot dogs, pizza, canned pasta
Try new flavors of foods

Adapted from Alexander et al,[5] and Yu.[11]

should be closely followed to minimize the risk of food-borne illness in immunocompromised states (Table 33–7).

Enteral Nutrition

Tube feedings to supplement oral intake in the child with cancer have been used successfully in many centers. Although nasogastric tube feeding remains controversial in the neutropenic patient, it is less expensive and associated with fewer life-threatening complications than is PN.[11] Even children who depend on PN for the majority of their nutritional needs may benefit from small amounts of EN due to its stimulatory effects on the gastrointestinal mucosa.

Parenteral Nutrition

The use of PN in the pediatric oncology patient is well accepted in the setting of a poorly functioning gastrointestinal tract. Parenteral nutrition in the BMT setting has been demonstrated to shorten the time to engraftment,[6] although further studies need to be done to evaluate its clinical impact. Glutamin-supplemented PN has been shown to

Table 33–7. Sanitary Food Practices for Immunocompromised Patients

Good handwashing before and after preparing and eating meals
Do not share food with others
Avoid foods from street vendors, salad bars, food bins in grocery stores
Wash raw foods well prior to eating
Cook meat until well done
Avoid raw eggs
Keep foods at < 40°F or > 140°F to minimize growth of bacteria
Clean all preparation items thoroughly before and after use to avoid crosscontamination
Keep refrigerated leftovers for no more than 3 days

reduce the incidence of clinical infection and length of hospital stay in adult BMT patients;[13] pediatric studies are underway. The risks and benefits of PN are outlined in Chapter 17. The combined effects of cancer treatment and PN on systemic infection and toxicities are unique to the oncology patient.

References

1. National Institutes of Health, National Cancer Institute. Young people with cancer—a handbook for parents. Bethesda (MD): National Cancer Institute; 1993.
2. Robison LL. General principles of the epidemiology of childhood cancer In: Pizzo PA, Poplack DG, editors. Principles and practice of pediatric oncology. 3rd ed. Philadelphia: Lippincott-Raven Publishers; 1997.
3. Pinkel D. Bone marrow transplantation in children. J Pediatr 1993;122:331–41.
4. Didi M, Didcock E, Davies HA, et al. High incidence of obesity in young adults after treatment of acute lymphoblastic leukemia in childhood. J Pediatr 1995;127:63–7.
5. Alexander HR, Rickard KA, Godshall B. Nutritional supportive care. In: Pizzo PA, Poplack DG, editors. Principles and practice of pediatric oncology. 3rd ed. Philadelphia: Lippincott-Raven Publishers; 1997.
6. Weisdorf S, Hofland C, Sharp HL, et al. Total parenteral nutrition in bone marrow transplantation: a clinical evaluation. J Pediatr Gastroenterol Nutr 1984;3:95–100.
7. Kelly KM, Lange B. Oncologic emergencies. Pediatr Clin North Am 1997;44:809–27.
8. Copeman MC. Use of total parenteral nutrition in children with cancer: a review and some recommendations. Pediatr Hematol Oncol 1994;11:463–70.
9. Szeluga DJ, Stuart RK, Brookmeyer R, et al. Energy requirements of parenterally fed bone marrow transplant recipients. JPEN 1985;9:139–43.
10. Papadopoulou A, Williams MD, Darbyshire PJ, et al. Nutritional support in children undergoing bone marrow transplantation. Clinical Nutrition 1998;17:57–63.

11. Yu CL. Nutrition and childhood malignancies. In: Suskind RM, Lewinter-Suskind L, editors. Textbook of pediatric nutrition. 2nd ed. New York: Raven Press; 1993.
12. Aquino VM, Smyrl CB, Hagg R, et al. Enteral nutritional support by gastrostomy tube in children with cancer. J Pediatr 1995;127:58–62.
13. Ziegler TR, Young LS, Benfell K, et al. Clinical and metabolic efficiency of glutamine-supplemented parenteral nutrition after bone marrow transplantation. Ann Intern Med 1992;116:821–28.

Internet Resources

Oncol.ink—University of Pennsylvania:
www.oncolink.upenn.edu/specialty/ped_onc
CancerNet—National Cancer Institute:
www.cancernet.nci.nih.gov
National Childhood Cancer Foundation:
www.nccf.org
American Cancer Society:
www.cancer.org
Marrow Transplant Nutrition Links:
www.students.washington.edu/kziemer/mtnutrlinks.html
Fred Hutchinson Cancer Research Center:
www.fhcrc.org

34

PREMATURITY

Jill Kostka Fulhan, MPH, RD

The length of human gestation is approximately 40 weeks, during which the fetus grows, organ systems mature, and nutrients are stored in preparation for the infant's transition to extrauterine life. When an infant is born prematurely, an abrupt cessation of nutrients from the mother to the fetus occurs, effectively halting all previous rates of growth and development as well as nutrient accretion. Increased nutritional demands and poor nutrient stores, combined with the many potential health risks faced by the premature infant (Tables 34–1 and 34–2), make the delivery of optimal nutrition crucial to the infant's further growth and development.

Nutrition Assessment

Nutrition assessment and therapy in premature infants must begin immediately after birth. Initial nutritional assessment includes accurate determination of gestational age and degree of prematurity as well as accurate measurement of birth weight, length, and head circumference (Table 34–3). Although a variety of intrauterine and postnatal growth curves are available,[1–5] no single reference set has been used exclusively. Most widely used are the growth curves of Lubchenco (Figure 34–1) and Babson (Figure 34–2) although the former data are derived from infants born at a high altitude. These curves remind clinicians that the "gold standard" for postnatal growth of premature infants is the expected rate of weight gain seen in utero. In practice, daily weight increments of 10 to 30 g with weekly length gains

Table 34–1. Nutritional Risk Factors in Prematurity

Increased nutritional demands
- Rapid growth phase
- Tissue development
- Stresses of medical/surgical course
- Prolonged illness
- Poor temperature control, cold stress
- Increased metabolic demand of SGA infants

Immature organ function
- Immature GI tract
- Utilization of nutrients may be decreased
- Glucose instability
- Renal immaturity

Poor nutrient stores
- Cessation of placental nourishment interrupts natural fetal accretion

Altered feeding patterns
- Suck/swallow/breathe coordination develops at 32–34 weeks GA
- Lactation consultant may enhance infant's transition to feeding at breast
- Infants with chronic lung disease, CHD, and NEC may require prolonged NPO status
- Occupational therapy may help infant transition to PO feeds

SGA = small for gestational age; GI = gastrointestinal; GA = gestational age; CHD = congenital heart disease; NEC = necrotizing enterocolitis; NPO = nil per os; PO = per os.

of 0.8 to 1.1 cm are often seen. Head circumference generally increases by 0.5 to 0.8 cm per week.

Once an infant has reached 40 weeks postconceptional age, his or her anthropometric data should be plotted on the National Center for Health Statistics (NCHS) curves, using "corrected age." An infant's corrected age (CA) is the chronologic age adjusted by the number of weeks of prematurity. For example, a premature infant who is born at 32 weeks gestational age (GA) is born 8 weeks early (40 weeks full term − 32 weeks GA = 8 weeks). At a chronological age of 12 weeks, this infant would have a

Table 34–2. Medical Risk Factors in Prematurity

Necrotizing enterocolitis (NEC)
- Over 90% of cases occur in premature infants
- Acquired GI disease of undetermined etiology, likely multifactorial
- Cause may be related to feeding rate, volume, or substrate provided
- Presents as mild feeding intolerance (NEC watch) to extreme necrotic bowel with perforation
- Treatment can include 3–21 days NPO, TPN, gradual re-introduction of feeds

Bronchopulmonary dysplasia (BPD)
- Chronic lung disease secondary to extended mechanical ventilation and/or O_2 support
- Potential for growth failure, increased metabolic demand
- May require increased calories (130–160 kcal/kg/d)
- May require fluid restriction, increased caloric concentration of feeding
- Steroids used in treatment may impede growth
- Increased risk of osteopenia (diuretics, steroids, losses of Ca and P)
- Increased WOB and/or decreased suck/swallow efficiency may increase caloric needs

Osteopenia of prematurity
- Decreased bone mass due to inadequate provision of mineral substrate
- Presents from mild demineralization to nontraumatic stress fractures
- May impair linear growth
- Risk increased with medications that cause mineral excretion, decreased absorption (diuretics, steroids)
- Incidence has decreased with availability of human milk fortifiers/preterm formulas

NPO = nil per os; TPN = total parenteral nutrition; WOB = work of breathing.

corrected age of 4 weeks (12 weeks of age – 8 weeks premature = 4 weeks CA). This infant's weight, length, and head circumference should therefore all be plotted at the 1 month position on the NCHS curves.

Table 34–3. Prematurity and Birth Weight Classifications

Maturity by gestational age (GA)	
Preterm:	< 38 weeks
Term:	38–42 weeks
Post-term:	> 42 weeks
Birth weight	
LBW:	< 2,500 g (low birth weight)
VLBW:	< 1,500 g (very low birth weight)
ELBW:	< 1,000 g (extremely low birth weight)
Birth weight for gestational age	
IUGR:	weight < 3rd percentile (intrauterine growth retardation)
SGA:	weight < 10th percentile (small for GA)
— asymmetric SGA:	weight only < 10th percentile—acute malnutrition or placental insufficiency
— symmetric SGA:	weight, length, head circumference < 10th percentile—prolonged malnutrition, genetic processes, or congenital anomalies
AGA:	weight 10th–90th percentile (appropriate for GA)
LGA:	weight > 90th percentile (large for GA)

Nutrition Therapy

The majority of premature infants will require a combination of parenteral and/or specialized enteral nutrition, the former providing recommended fluid and nutrient estimates (Table 34–4) until the latter is tolerated at sufficient volumes for growth and development (Table 34–5). A premature infant's ability to take "full feeds" is dependent on many factors, including efficient suck/swallow/breathe coordination for nipple feeds, maturity of the gastrointestinal tract, stomach capacity, respiratory status, presence of medical

complications, and gestational readiness. The nutrition regimen for the preterm infant must maximize nutrition and growth without compromising metabolic status.

Parenteral nutrition (PN) should begin in the first 24 to 48 hours of life if an infant is expected to be nil per os (NPO) for more than 3 to 5 days. Early PN is especially important for the very low birth weight (VLBW) infant since PN can reverse some of the metabolic effects of starvation. Parenteral nutrition is also recommended during periods of bowel rest (> 3 to 5 days) as with necrotizing enterocolitis (NEC) or after surgery. Pediatric amino acid

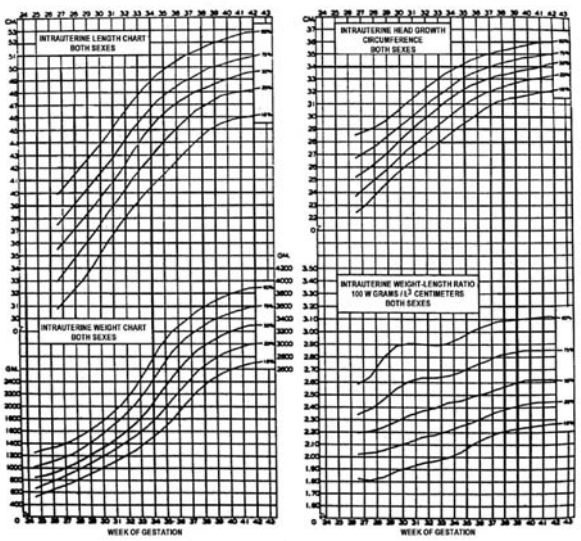


Figure 34–1. Lubchenco intrauterine growth curve. (Reproduced with permission from Lubchenco LO, Hansman C, Boyd E. Intrauterine growth in length and head circumference as estimated from live births at gestational ages from 26–42 weeks. Pediatrics 1966;37:403–8.)

solutions are preferred due to their inclusion of cysteine and taurine, amino acids thought to be essential for the premature infant. A 20 percent lipid solution is recommended as a high-density energy source as well as for essential fatty acids. Protein, fat, and carbohydrate should be advanced as tolerated and daily laboratory values monitored until estimated needs are met. Thereafter, weekly nutrition panels are used to evaluate the adequacy of the prescribed regimen. Each component must be calculated on the basis of recommended fluid volumes and individual needs (see Table 34–4) (see also Chapter 17, Parenteral Nutrition).

Enteral nutrition should generally begin in the first 48 to 72 hours of life. The gestational age of the infant will

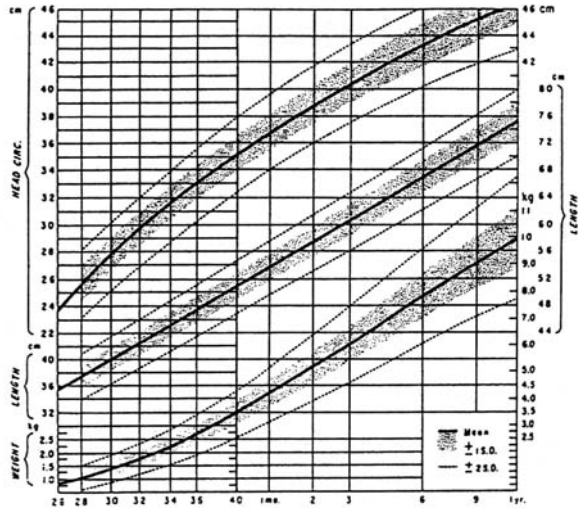


Figure 34–2. Babson growth curve. (Reproduced with permission from Babson SG, Benda GJ. Growth graphs for the clinical assessment of infants of varying gestational ages. J Pediatr 1976;89:814–20.)

Table 34–4. Parenteral Nutrition in Premature Infants

Fluid requirements

- Premature infants have greater ECF volumes
- Initial diuresis constitutes 10–15% birth weight, 20% for ELBW infants
- Initial fluid requirements: 80–140 cc/kg/d*
- ELBW infants may require up to 200 cc/kg/d
- Goal after fluid stabilization: 100–150 cc/kg/d
- Fluid restriction may be required for infants with PDA, BPD, CHF, renal failure, cerebral edema
- Insensible water loss increases with
  — increased skin permeability at birth
  — increased BSA to weight ratio
  — phototherapy
  — radiant warmer beds
  — respiratory distress syndrome
  — cold stress, increased activity
- Insensible water loss decreases with
  — heat shields
  — humidified incubators
- Fluid loss also results from
  — vomiting
  — diarrhea
  — ostomy output
  — chest tube drainage

Carbohydrate

- Initial glucose load: 4–6 mg/kg/min
- Adjust by 1–2 mg/kg/min as tolerated, advancing to meet nutritional need
- Limit to < 14 mg/kg/min to prevent overfeeding, fatty liver, increased CO_2 production
- Dextrose concentration: can maximize with 12.5% in a peripheral line

Protein

- Infants < 1000 g BW: begin at 0.5 g/kg/d and advance by 0.5 g/kg/d to goal
- Infants > 1000 g BW: begin at 1.0 g/kg/d and advance by 1.0 g/kg/d to goal

Table 34–4. continued

Fat

- Infants < 1,000 g BW: begin at 0.5 g/kg/d and advance by 0.5 g/kg/d to goal
- Infants > 1,000 g BW: begin at 1.0 g/kg/d and advance by 1.0 g/kg/d to goal
- May run lipid via central or peripheral access, over 20–24 hours
- Monitor with serum triglyceride, normal range < 150 mg/dL
- EFAD may occur in < 1 week without lipid source; provide minimum 0.5 g/kg/d to prevent EFAD
- May need to limit lipid with extreme hyperbilirubinemia, to prevent kernicterus

Energy needs

- Tsang*: 80–90 *nonprotein* kcal/kg/d with 3 g protein/kg/d
- Zlotkin†: > 70 nonprotein kcal/kg/d with 2.7–3.5 g protein/kg/d

Additives

Na 2–4 mEq/kg/d	Ca*: 60–90 mg/kg/d
K 2–4 mEq/kg/d	Phos*: 47–70 mg/kg/d
Cl 2–3 mEq/kg/d	Mg*: 4.3–7.2 mg/kg/d

MVI Pediatric:

< 1 kg: 30% of standard 5mL
1–3 kg: 65% of standard 5mL
> 3kg: 100% of standard 5mL

Biochemical parameters to monitor

- Daily (as initiating and advancing PN and lipids)
  - Na, K, Cl, CO_2, glucose, triglyceride
- Weekly (and prior to initiating PN and lipids)
  - add Ca, Mg, P, alk phos, BUN, Cr, triglyceride, total protein, albumin, bilirubin, AST, ALT, hematocrit

ALT = alanine transaminase; AST = aspartate transaminase; BPD = *bronchopulmonary dysplasia*; BSA = *body surface area*; BUN = blood urea nitrogen; BW = birth weight; CHF = congestive heart failure; ECF = extracellular fluid; EFAD = essential fatty acid deficiency; ELBW = extremely low birth weight; PDA = patent ductus arteriosus; PN = parenteral nutrition.

*Data from Tsang RC, Lucas A, Uauy R, et al., editors. Nutritional needs of the preterm infant: a scientific and practical guide. Baltimore (MD): Williams and Wilkins; 1992.

†Data from Zlotkin SH, Bryan MH, Anderson GH. Intravenous nitrogen and energy intakes required to duplicate in utero nitrogen accretion in prematurely born human infants. J Pediatr 1981;99:115–20.

Table 34–5. Enteral Nutrition: Feeding Advancement and Goals

Feeding initiation and advancement

- Initiate based on birth weight and advance accordingly, as tolerated*

Birth weight (g)	*Initial rate (cc/kg/d)*	*Rate increase (cc/kg/d)*
< 800	10	10–20
800–1,000	10–20	10–20
1,001–1,250	20	20–30
1,251–1,500	30	30
1,501–1,800	30–40	30–40
1,801–2,500	40	40–50
> 2,500	50	50

Warning signs of feeding intolerance

- Increase in gastric residuals to > twice previous hour's rate (continuous feed) or > $^1/_2$ the previous bolus
- Increase in abdominal distention/girth
- Vomiting
- Bilious residuals and/or vomiting
- Heme positive/frank blood in stools
- Reducing substances > 0.5%
- Change in bowel sounds (peristalsis)
- Increase in apnea/bradycardia with feeds

Energy goal

- 110–130 kcal/kg/d
- Some infants may have increased needs up to 150–160 kcal/kg (BPD, SGA)

Protein goal

- 3.0–4.0 g protein/kg/d

Caloric distribution

- PRO: 9–12% calories
- CHO: 40–45% calories
- Fat: 40–50% calories

Calcium

- 120–230 mg/kg/d†

Phosphorus

- 60–140 mg/kg/d†

Table 34–5. continued

Multivitamin
- Infants receiving breastmilk with HMF or premature formula do not usually require multivitamin supplementation
- Preterm infants receiving breastmilk exclusively, recommend multivitamin supplementation

Vitamin E
- Recommended dose: 6–12 IU/kg/d
- May require supplementation if receiving elemental iron greater than 4 mg/kg/d, to decrease risk of hemolytic anemia

Iron
- Preterm infants are born with low stores and are subject to many blood draws
- Recommend initiating iron supplement at 4–6 weeks of age
- Infant should be at full feeds (150 cc/kg/d at 24 kcal/oz) prior to start of supplementation
- See Table 34–6.

*Data from Sun Y, Awnetwant EL, Collier SB, et al. Nutrition. In: Cloherty JP, Stark AR, editors. Manual of neonatal care. 4th ed. Philadelphia: Lippincott-Raven Publishers; 1998. p. 101–34.
†Data from Tsang RC, Lucas A, Uauy R, et al., editors. Nutritional needs of the preterm infant: a scientific and practical guide. Baltimore: Williams and Wilkins; 1992. p. 135–55.

affect the decision to feed by mouth or tube since suck/swallow/breathe coordination does not develop until 32 to 34 weeks gestation (Table 34–7). Feeding initiation and advancement is often based on birth weight with close attention to feeding tolerance. Most premature infants begin with "trophic feeds," low-volume feedings of 10 to 20 cc/kg/d, to stimulate gut hormones, motility, and gastrointestinal maturation.

Due to the premature infant's increased nutritional requirements, close attention should also be paid to the choice of substrate provided to ensure that both macronutrient and micronutrient needs are met (Table 34–5). Breastmilk is the preferred feeding choice for nearly all infants, including premature infants (see Chapter 6, Breastfeeding).

Table 34–6. Iron Supplementation Guidelines in the Premature Infant*

	Birth Weight				
	< 1,000 g	*1,000–1,500 g*	*1,500–1,800 g*	*> 1,800 g*	*Notes*
Total dose	*4 mg/kg/d*	*3–4 mg/kg/d*	*2–3 mg/kg/d*	*2 mg/kg/d*	—
Formula					
Low iron	Supplement with elemental iron 4 mg/kg/d	Supplement with elemental iron 3–4 mg/kg/d	Supplement with elemental iron 2–3 mg/kg/d	Supplement with elemental iron 2 mg/kg/d	—
Iron fortified	Supplement with elemental iron 2 mg/kg/d	Additional elemental iron 1–2 mg/kg/d	Additional 1 mg/kg/d as needed	No additional supplementation	—
Human milk (HM) only	Elemental iron 4 mg/kg/d	Elemental iron 3–4 mg/kg/d	Elemental iron 2 mg/kg/d	Elemental iron 2 mg/kg/d	Infants under 1,800 g should be on 24 cal/oz HM (with human milk fortifier) before iron supplementation is begun
Combination (formula plus HM)					
Low iron	Supplement with elemental iron 4 mg/kg/d	Supplement with elemental iron 3–4 mg/kg/d	Supplement with elemental iron 2–3 mg/kg/d	Supplement with elemental iron 2 mg/kg/d	—

Table 34–6. continued

	Birth Weight				
	< 1,000 g	*1,000–1,500 g*	*1,500–1,800 g*	*> 1,800 g*	*Notes*
Total dose	*4 mg/kg/d*	*3–4 mg/kg/d*	*2–3 mg/kg/d*	*2 mg/kg/d*	—
Iron fortified	Calculate for total iron dose of 4 mg/kg/d	Calculate for total dose of 3–4 mg/kg/d	Additional 1 mg/kg/d as needed	No additional supplementation	—

BPD = bronchopulmonary dysplasia; CHO = carbohydrate; HMF = Enfamil Human Milk Modifier; PRO = protein; SGA = small for gestational age.

*Data from Sun Y, Awnetwant EL, Collier SB, et al. Nutrition. In: Cloherty JP, Stark AR, editors. Manual of neonatal care. 4th ed. Philadelphia: Lippincott-Raven Publishers; 1998. p. 101–34. Reproduced with permission.

Table 34–7. Enteral Feeding Methods

Indications for nipple feeding
- Minimum 32–34 weeks postconceptual age, though some infants may do well at the breast earlier
- Coordinated suck/swallow/breathe pattern is present
- Infant is free of apnea and bradycardia
- Respiratory rate < 60 breaths/min
- Infant may benefit from gradual transition from gavage to nipple feeding
- Consider partial gavage feeding if infant takes > 30 minutes/feed to prevent excess energy expenditure

Indications for NG/OG tube feeding
- Infant < 32 weeks GA, poor suck/swallow/breathe coordination
- Respiratory rate > 60 breaths/min
- No gag reflex evident
- Continuous
  - — may be better tolerated in smaller infants
  - — for infants with previous intolerance to bolus feeds
  - — requires less frequent tube change, less disruption to baby
  - — may require less energy expenditure than bolus
  - — may decrease risk of aspiration
  - — may prevent increase in respiratory rate (vs bolus)
- Bolus
  - — every 2–3 hours
  - — may improve gastric emptying
  - — allows hunger/satiety, can alternate with nipple feeds
  - — allows more mobility, parents can hold and provide care more easily

Indications for transpyloric feeding
- Consider for infants with intolerance to gastric feeding, GER, risk for aspiration, nasal CPAP
- Requires continuous feeding
- Placement of tube is more difficult

Indications for G-tube feeding
- For infants who will be unable to nipple feed for several months
- May prevent oral aversion associated with long-term nasogastric tube feeding

CPAP = continuous positive airway pressure ventilation;
GA = gestational age; GER = gastroesophageal reflux;
NG/OG = nasogastric/orogastric; G-tube = gastrostomy tube.

Table 34–8. Enteral Nutrition: Choice of Feeding Substrate

Breastmilk is preferred for the following advantages:

- Anti-infective factors
- Whey dominant protein
- Taurine and cysteine
- Bile salt–stimulated lipase and lipoprotein lipase aid in fat digestion and absorption
- Decreased renal solute load
- May enhance the mother/infant bond
- May protect against NEC
- May improve cognitive outcome

Breastmilk may require supplementation for premature infants

- Otherwise, volume required to meet protein, energy, Ca, P, Mg, and other vitamin and mineral needs is excessive
- Fat in breastmilk may adhere to the NG/OG tubing, which decreases available calories and EFAs

Fortification of breastmilk:

- Powder fortifier: Enfamil Human Milk Fortifier (HMF)
  - adds protein, carbohydrate, vitamins, and minerals
  - increase to 22 and 24 cal/oz
  - preferred when adequate amount of breastmilk is available
- Liquid fortifier: Similac Natural Care (SNC)
  - 24 cal/oz formula added in equal volumes to breastmilk
  - increase to 22 cal/oz
  - may use when breastmilk supply does not meet volume demand
- Tolerance
  - continue to monitor nutrition labs, especially Ca, P, and alkaline phosphatase
  - ELBW infants at increased risk for hypercalcemia

Breastmilk fortifiers are indicated for:

- Infants born at < 34 weeks GA and/or < 2,000 g
- Infants with increased needs who are fluid restricted
- Hospital use only
- Bottle or tube feeds until infant is:
  - taking sufficient volume at the breast
  - 40 weeks corrected age and/or ≥ 2.5 kg
  - ready for discharge

Premature formulas

- Alternative to fortified breastmilk when breastmilk is not available

Table 34–8. continued

- Preferred for its composition, increased calories, protein, Ca, P
- Available in 20 and 24 cal/oz, RTF (ready-to-feed)
- PRO: whey predominant
- Fat: 50% MCT oil, may improve fat absorption and weight gain
- CHO: 50% lactose, 50% glucose polymers

Premature discharge formulas
- When continued fortification is recommended for smaller, more premature infants
- Designed for home use up to 12 months of age
- RTF is 22 cal/oz
- Powder may be added to fortify breastmilk

Standard term, cow's-milk-based formulas
- Not recommended for premature infants; do not meet needs at volumes tolerated
- May be provided for AGA infants > 34 weeks GA and > 2.0 kg at birth
- May be provided for growing premature infant > 34 weeks CA and > 2.0–2.5 kg, who is ready for discharge home

Standard soy-based formulas
- Not recommended for premature infants
- Low bioavailability of Ca and P, adverse effects on bone
- May be indicated for lactose intolerance, galactosemia, secondary lactose intolerance
  - — would require vitamin/mineral supplementation
  - — may require caloric concentration

Therapeutic formulas
- Include protein hydrolysates, free amino-acid-based, or high MCT-containing formulas
- Not recommended long term due to suboptimal nutrient composition; may need to fortify if used

Modulars
- When increased caloric demands require further caloric supplementation
- MCT oil, corn oil, carbohydrate and/or protein supplements may be added to
  24 cal/oz fortified breastmilk/formula
- Attention must be paid to distribution of calories, osmolality, renal solute load

AGA = appropriate for gestational age; CHO = carbohydrate; EFA = essential fatty acids; ELBW = extremely low birth weight; MCT = medium chain triglycerides; NEC = necrotizing enterocolitis; NG/OG = nasogastric/orogastric; PRO = protein; CA = corrected age.

Table 34–9. Discharge Criteria

- HMF, SNC or premature formula has been discontinued
  - do not provide at home due to high vitamin and nutrient content, potential for toxicity
- Transition to all breastmilk or term formula
  - recommended once infant tolerates 180 cc/kg/d, weighs > 2.0 kg
- Transition to premature discharge formula if increased needs persist
- Provide parents with recipe/instructions for home feeding regimen as needed
- Multivitamin supplementation
  - breastfeeding: 1.0 cc/d
  - formula: 0.5 cc/d
  - continue until infant tolerates 750 cc/d or reaches 3.5 kg
- Iron
  - continue supplementation
  - may need to give throughout the first year, depending on feeding choice

HMF = Human Milk Fortifier (Enfamil); SNC = Similac Natural Care.

Fortification of breastmilk (or use of premature formulas if breastmilk is not available) is recommended for all infants born < 34 weeks gestational age or with a birth weight of < 2000 g. It may also be necessary for infants who are fluid restricted. Fortification may require one or more of a variety of additives to meet estimated needs and should be prescribed on an individual basis. Soy-based, term, or protein hydrolysate formulas are generally discouraged for routine use in the premature infant since they are not specially designed to meet the growing premature infant's needs.

Discharge Planning

Many premature infants will continue to be at nutritional risk on discharge home. Infants at special risk include those

who were VLBW and/or small for gestational age (SGA) at birth, who have a history of poor weight gain or poor feeding skills, who required long-term total parenteral nutrition (TPN), and who have had NEC, bronchopulmonary dysplasia (BPD), osteopenia/rickets, neurologic impairment, oral-motor impairment, developmental delay, retinopathy of prematurity, congestive heart failure/congenital heart disease (CHF/CHD) (requiring fluid restriction), or prolonged tube feeding.

Prior to discharge home, the infant should demonstrate adequate and consistent weight gain, free of parenteral nutrition. Ideally, the infant should take all feeds by mouth, but in some cases, nasogastric or G-tube feeding may be appropriate, based on medical condition and parental readiness. A minimum weight goal may also be required for discharge home. If the infant requires enhanced breast-milk or formula feeding at home, the family should be able to demonstrate how to prepare these accurately, based on a prescribed recipe. Premature infant formulas and certain human milk fortifiers are not recommended for home use. Transition to a safe and appropriate home feeding regimen is required (Table 34–8). Referral to a community lactation consultant, Special Supplemental Food Program for Women, Infants, and Children (SSFPfWIC), Early Intervention Program (EIP), and/or home-health nursing may also be beneficial for assistance with feeding skills and weight checks, and may help ensure further follow-up as needed.

References

1. Dancis J, O'Connell JR, Holt LE. A grid for recording the weight of premature infants. J Pediatr 1948;33:570–2.
2. Shaffer SG, Quimiro CL, Anderson JV, et al. Postnatal weight changes in low birth weight infants. Pediatrics 1987; 79:702–5.

3. Wright K, Dawson JP, Fallis D, et al. New postnatal grids for very low birth weight infants. Pediatrics 1993;91:922–6.
4. Lubchenco LO, Hansman C, Boyd E. Intrauterine growth in length and head circumference as estimated from live births at gestational ages from 26–42 weeks. Pediatrics 1966;37:403–8.
5. Babson SG, Berda GJ. Growth graphs for the clinical assessment of infants of varying gestational ages. J Pediatr 1976; 89:814–20.

Additional Reading

Groh-Wargo S, Thompson M, Cox JH. Nutritional care for the high-risk newborns. Chicago: Precept Press; 1994.
RE Kleinman, editor. Pediatric nutrition handbook, 4th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1998.

35

RENAL DISEASE

Nancy S. Spinozzi, RD

Chronic renal failure (CRF) occurs when renal function has deteriorated so that glomerular filtration rate (GFR) is reduced and progression to endstage renal disease (ESRD) is inevitable. Endstage renal disease usually denotes the point at which conservative management of the patient is no longer effective and renal replacement strategies (dialysis and transplantation) are necessary. The major cause of ESRD in children is chronic glomerulonephritis (primarily in older patients, 15 to 19 years of age) followed by cystic/hereditary/congenital diseases of the kidney (primarily in younger children, 0 to 4 years of age).[1] Table 35–1 lists the nutritional and metabolic consequences of CRF in children and provides guidelines for treatment.

Nutritional assessment of the child with CRF is complex and must be performed on a frequent, regular basis.[5] Since growth retardation is such a common occurrence in CRF, all aspects of a child's potential growth characteristics must be taken into account. Table 35–2 outlines the specific aspects of nutritional assessment in children with CRF.

Once the initial nutritional assessment is complete, recommendations must be made to ensure optimal nutrition within the limitations of kidney function. There are currently no data to show that a reduced protein intake (< the Recommended Dietary Allowance [RDA] for age) will delay the progression of endstage renal disease in pediatric patients. Protein and energy recommendations are based on the RDA for height age.

Table 35–1. Nutritional and Metabolic Consequences of Chronic Renal Failure

Problem	*Etiology*	*Treatment*
Protein/energy malnutrition	Anorexia/dysgeusia Caloric requirements > normal (RDA)	Protein/energy supplementation[2] (refer to Table 35–4) Tube feeding[3]
Acidosis	Tubular dysfunction (bicarbonate loss)	Oral base solutions: sodium bicarbonate, bicitra
Salt-wasting	1° (eg, obstructive uropathy, cystic diseases)	Sodium supplementation
Renal osteodystrophy[4]	Decreased PO_4 excretion leading to decreased serum Ca^{++} resulting in increased PTH secretion, ultimately leading to secondary hyperparathyroidism	Decrease PO_4 intake PO_4 binders (eg, Ca^{++} carbonate, Ca^{++} acetate)
	Decreased renal conversion of 25 hydroxycholecalciferol to 1,25-dihydroxycholecalciferol	Provide active form of vitamin D (eg, 1,25-dihydroxycholecalciferol, dihydrotachysterol
Hyperkalemia[5]	Decreased renal excretion	Decrease intake Sodium polystyrene sulfonate (Kayexalate) Correct acidosis
Hypermagnesemia	Decreased renal excretion	Decrease intake Avoid Mg-containing antacids or laxatives

Growth retardation	Growth hormone resistance	Growth hormone[6] (rhGH)
Hypertension/fluid retention[5]	Increased angiotensin II formation Volume-sensitive HTN	Decrease sodium intake (no added salt diet) ± fluid restriction Antihypertensives
Anemia	Decreased production of erythropoietin	Erythropoietin (rhEPO)[7] Iron supplementation[8]
Hypovitaminosis	Water soluble vitamins lost in dialysate Malnutrition	Supplement with H_2O soluble vitamins only (eg, Nephrocaps,* Nephrovites† —both examples of dialysis vitamins) or MVI with the addition of 0.5–1.0 mg folate[9]
Elevated homocysteine levels	Most likely due to antioxidant deficiency	Folate, B_6, and B_{12} supplementation
Hypervitaminosis A	Impaired retinol binding protein excretion	Avoid vitamin A supplementation
Renal oxalate stones	Reduced clearance	Avoid vitamin C supplements and oxalate-rich foods

PTH = parathyroid hormone; HTN = hypertension.

*Fleming Laboratories, Fenton, MO.

†R & D Laboratories, Inc., Marina Del Rey, CA.

Table 35–2. Special Aspects of Nutritional Assessment in Renal Disease

History
Diagnosis
Primary disease, if known
Current renal replacement modality (conservative, dialysis, transplant)
Diet recall
Calories
Protein
Electrolytes (Na^+, K^+, Mg)
Vitamins, minerals (Ca^{++}, PO_4)
Fluid intake
Medications
Physical
Height
Weight-fluid dependent
Head circumference
MAMC and TSF
Laboratory
BUN, Cr, Na^+, K^+, glucose, PO_4, Ca^{++}, Mg, albumin, CO_2, hematocrit, triglyceride, cholesterol

MAMC = mid-arm muscle circumference; TSF = triceps skinfold.

Peritoneal dialysis is the more common dialysis treatment for children with ESRD, and renal transplantation is more common in younger children than in older children and adults. Transplantation is highly encouraged for all suitable children, eventually providing them a more normal lifestyle.

Tables 35–3 and 35–4 review some of the critical issues related to providing adequate enteral nutrition in children with renal disease.

Parenteral Nutrition Considerations in Renal Disease

The volume of fluid available to provide adequate nutrition is a major consideration when prescribing parenteral

Table 35–3. General Considerations for Enteral Feedings in Renal Disease[10,11]

Fluid allowance/metabolic status, GFR
Formula feedings will likely need to be calorically dense since infant's complete nutrition source is fluid
Increase caloric density of formula/feeds through carbohydrate and fat modules rather than concentration (which will increase renal solute load)
Gastroesophageal reflux is a major problem for infants with CRF.[12] Tube feedings are often necessary to ensure adequate intake of nutrients. Continuous nighttime infusions are usually well tolerated
Monitor weight, BUN, electrolytes, albumin, lipids; adjust feeds and diet as necessary (at least monthly)

nutrition (PN). Fluid may be severely restricted due to oliguria/anuria resulting in the need for very hypertonic solutions. Energy intake via PN should be guided by the RDA for height age. Mixed amino acid solutions are well tolerated in renal patients, with the protein goal being the RDA for height age. The utility of specially formulated amino acid solutions for renal patients is controversial.

Table 35–4. Specific Nutritional Products for Renal Patients

Product	*Manufacturer*	*Comments*
Similac PM 60/40	Ross Products	Infant formula reduced in electrolytes and PO_4
Amin-aid	R & D Laboratories, Inc.	Tube feeding which contains no electrolytes
Nepro	Ross Products	For dialysis patients
Suplena	Ross Products	For predialysis patients
Renalcal Diet	Nestle Clinical Nutrition	Contains no electrolytes

Endstage renal disease patients should begin with a parenteral solution without added K^+, Mg^+, and PO_4 titrated according to serum levels. Because of intake of PO_4 and other cations, malnourished renal patients should be closely monitored for the refeeding syndrome (see Chapter 17). Since micronutrients (eg, vitamin A, selenium) are excreted primarily through the kidneys, long-term use of standard parenteral multivitamins may lead to toxicities. Parenteral supplementation with folate, vitamin C, and B complex vitamins is recommended instead.

Nutritional Considerations in the Postrenal Transplant Patient

When planning nutritional support after transplantation, it is important to monitor a patient's renal function.[13] It may be necessary to reinitiate dialysis treatments during the first several months. Continuation of pretransplant diet restrictions may, therefore, be indicated.

Many of the common transplant medications (immunosuppressive drugs such as prednisone and cyclosporine) can cause side-effects which will have an impact on dietary recommendations, including hyperkalemia, increased appetite, hypertension, glucose intolerance, and gastric irritation. Most children, however, will require only a no added salt diet after transplantation. Hypophosphatemia is a common finding post-transplant and usually requires PO_4 supplementation. Many children undergoing renal transplantation are unfamiliar with a healthy diet for age and should, along with their parents, be guided in appropriate food choices following transplantation.

References

1. Pediatric end-stage renal disease. In: United States Renal Data System. USRDS 1997 Annual Data Report. Bethesda (MD): National Institutes of Health, National Institutes of Diabetes and Digestive and Kidney Diseases; 1997. p. 113–28.
2. Spinozzi NS, Nelson P. Nutrition support in the newborn intensive care unit. J Ren Nutr 1996;6:188–97.
3. Brewer ED. Supplemental enteral tube feeding in infants undergoing dialysis—indications and outcome. Semin Dial 1994;7:429–34.
4. Salusky IB, Goodman WG. The management of renal osteodystrophy. Pediatr Nephrol 1996;10:651–3.
5. Stover J, Nelson P. Nutritional recommendations for infants, children and adolescents with ESRD. In: Gillit D, Stover J, Spinozzi NS, editors. A clinical guide to nutritional care in ESRD. Chicago (Il): American Dietetic Association; 1987. p. 71–94.
6. Fine RN, Kohout EC, Brown D, Perlman AJ. Growth after recombinant human growth hormone treatment in children with chronic renal failure: report of a multicenter randomized double-blind placebo-controlled study. J Pediatr 1994;124:374–82.
7. Eschbach MD, Egrie JC, Downing MR, et al. Correction of the anemia of end-stage renal disease with recombinant human erythropoietin. N Engl J Med 1987;310:73–8.
8. Van Wyck DB, Stivelman JC, Ruiz J. Iron status in patients receiving erythropoietin for dialysis-associated anemia. Kidney Int 1989;35:712–6.
9. Warady BA, Kriley M, Alon U, Hellerstein S. Vitamin status in infants receiving long-term peritoneal dialysis. Pediatr Nephrol 1994;8:354–6.
10. Harvey E, Secker D, Braj B, et al. The team approach to the management of children on chronic peritoneal dialysis. Adv Ren Replace Ther 1996;3:3–13.
11. Grupe WE, Harmon WE, Spinozzi NS. Protein and energy requirements in children receiving chronic hemodialysis. Kidney Int 1983;24:S6–S10.

12. Ruley EJ, Boch GH, Kerzner B, Abbott AW. Feeding disorders and gastroesophageal reflux in infants with chronic renal failure. Pediatr Nephrol 1989;3:424–9.
13. Gammarino M. Renal transplant diet: recommendations for the acute phase. Dial Transplant 1987;16:497.

36

SHORT BOWEL SYNDROME

Sherri L. Utter, MS, RD, CNSD, and Christopher Duggan, MD, MPH

Short bowel syndrome (SBS) is a disorder characterized by decreased gastrointestinal mucosal surface area and increased transit time. This can lead to malabsorption of macro- and micronutrients, electrolyte abnormalities, dehydration, and ultimately malnutrition. Table 36–1 lists common etiologies of SBS in children. The prognosis of SBS depends on several factors, including length and portion of bowel resected, presence or absence of the ileocecal valve, the adaptive and functional capacity of the remaining bowel, and the health of other organs assisting with digestion and absorption. Two other recently report-

Table 36–1. Common Causes of Short Bowel Syndrome in Infants and Children

Necrotizing enterocolitis (NEC)
Intestinal atresia
Gastroschisis
Midgut volvulus
Inflammatory bowel disease
Tumors
Radiation enteritis
Ischemic injury
Intestinal pseudo-obstruction
Total intestinal aganglionosis

ed prognostic factors are bacterial overgrowth[1] and proportion of nutrition given enterally by 12 weeks of age.[2]

Length of Small Bowel Resected

Normal small intestine length is approximately 217 ± 24 cm in infants 27 to 35 weeks gestational age and 304 ± 44 cm in infants ≥ 35 weeks (Figure 36–1). At term, mean length is reported to be 250 to 300 cm. Another 2 to 3 meters is added to its length during growth to adulthood. The large intestine is 30 to 40 cm at birth, growing to 1.5 to 2 meters in adult life.[3]

Loss of intestinal length can limit digestion by reducing exposure of nutrients to brushborder hydrolytic enzymes as well as pancreatic and biliary secretions. Many studies have examined the relationship between length of residual small intestine and success at being weaned from parenteral nutrition (PN). It appears that infants require approximately 10 to 30 cm of small intes-

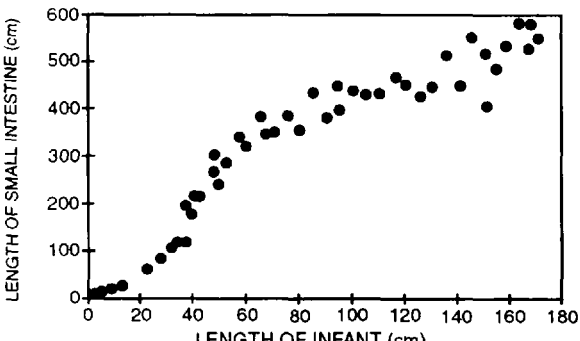


Figure 36–1. Small intestinal length from conception to maturity. *Adapted from* Weaver LT. Anatomy and embryology. In: Walker WA, editor. Pediatric gastrointestinal disease. Philadelphia: B.C. Decker, Inc.; 1991. p. 195–215.

tine, with an intact ileocecal valve, to avoid lifelong dependence on PN. If the ileocecal valve is not present, 30 to 50 cm of small intestine is generally required for successful weaning from PN.[4]

Portion of Small Bowel Resected

The location of resected bowel has an impact on nutrient loss in SBS. Duodenal resection may result in iron or folate malabsorption. Calcium absorption may also be impaired with proximal small bowel resection. The jejunum, with long, large villi, extensive absorptive surface area, highly concentrated digestive enzymes, and many transport carrier proteins, is the primary digestive and absorptive site for most nutrients. Loss of jejunum is also associated with reduction of cholecystokinin and secretin levels, which secondarily impairs pancreatic and biliary secretion.[5]

Loss of the terminal ileum results in malabsorption of bile acids. Steatorrhea and the formation of lithogenic bile may ensue. The terminal ileum is also the primary site for vitamin B_{12} absorption; therefore, resection can lead to B_{12} deficiency. The ileum also secretes hormonal substances that slow gastrointestinal motility in response to fat malabsorption.

Intestinal transit time decreases following small bowel resection. In the normal intestine, motility is rapid in the jejunum and slow in the distal ileum. Consequently, ileal resection reduces intestinal transit time more than duodenal resection does. Gastric emptying is also more rapid following ileal resection but can be normalized if the colon is retained. Colon resection reduces transit even further.[6]

Presence or Absence of the Ileocecal Valve

The ileocecal valve (ICV) serves to regulate the flow of enteric contents from the small bowel into the colon. The absence of an ICV shortens gastrointestinal transit and

increases fluid and nutrient losses. In addition, colonic bacteria may contaminate the small intestine, causing an inflammatory response that damages small bowel mucosa, resulting in an exacerbation of the malabsorptive state.[6] Bile salts and B_{12} may be deconjugated by the bacteria, further contributing to fat and vitamin B_{12} malabsorption.[5]

Adaptive and Functional Capacity of the Remaining Small Intestine

Following intestinal resection, the remaining intestine has an ability to compensate depending on the area of resection and other trophic stimuli. Intestinal adaptation refers to the gross anatomic and histologic changes that occur after significant intestinal resection (Table 36–2). These adaptive changes begin 12 to 24 hours after massive intestinal resection and will continue for more than 1 year.[5] Villi lengthen, the intestinal absorptive surface area increases, and absorptive function gradually improves.[7] Due to the opportunity for further growth of intestinal length, the younger infant is at an advantage for improvements in bowel function over time when compared to adults.

Enteral nutrition is an important stimulant of mucosal hyperplasia, and much research focuses on whether specific nutrients promote adaptation more than others.

Table 36–2. Adaptive Changes in the Small Bowel following Extensive Resection

Increased bowel circumference
Increased bowel wall thickness
Increased bowel length
Increased villus height
Increased crypt depth
Increased cell proliferation and migration to villus tip

Health of Other Organs Assisting with Absorption and Digestion

Cholestasis and liver dysfunction can occur in patients with SBS, thereby affecting the absorption and utilization of nutrients. The major cause of death in children with SBS is PN-associated liver disease. The relationship between PN use and cholestasis is likely multifactorial, with sepsis, mucosal atrophy, and bacterial overgrowth being risk factors. Every effort should therefore be made to reduce the risk of PN-associated cholestasis (Table 36–3).

Special Aspects of Nutritional Therapy in Short Bowel Syndrome

The goal of nutritional therapy in SBS is to maintain normal growth, promote intestinal adaptation, and avoid complications associated with intestinal resection and parenteral nutrition.

What to Feed

Fluid, Electrolytes, and Parenteral Nutrition. During the early postoperative phase, fluid and electrolyte balance is the goal of therapy. Large fluid losses are com-

Table 36–3. Reducing the Risk of Parenteral Nutrition Associated Cholestasis

Method	*Comments*
Avoid overfeeding	90–100 kcal/kg usual parenteral energy requirement
Cycle PN off at least 2–6 hours per day	Promotes cyclic release of GI hormones
Aggressively treat and prevent infections	Meticulous CVC care; treat bacterial overgrowth
Push enteral nutrition	The ultimate goal of therapy

GI = gastrointestinal; CVC = control venous catheter.

mon and tend to be high in sodium content. Parenteral solutions with at least 80 to 100 mEq/L of sodium are often required to maintain sodium balance.

Meticulous attention needs to be paid to the fluid and electrolyte status of SBS patients. This includes daily weights, careful measurement of urine, stool, and ostomy losses, and laboratory monitoring of electrolytes. Parenteral nutrition (PN) is indicated in managing SBS until small bowel growth and adaptation permit growth on enteral nutrition alone.

Day-to-day variation in fluid loss is common. It is therefore often advantageous to place the patient on a standard PN solution with fluid and electrolytes appropriate for age, size, and metabolic considerations, and subsequently replace abnormal losses with a separate solution based on measurement of actual fluid losses. For example, ostomy fluid can be measured for sodium content and replacement fluid prescribed accordingly. When losses have stabilized, the additional fluid and electrolytes can be added to the PN.

Enteral Feedings. Once the patient's fluid and electrolyte status has stabilized and postoperative ileus has resolved, a slow introduction of enteral feedings should be started. Mothers of newborns with SBS should be referred to a lactation consultant to encourage continued breastmilk production. The special immunologic and anti-infective properties of breastmilk are especially advantageous to the infant having undergone intestinal resection although the transition from parenteral to full enteral feedings still may take weeks to months. Breastmilk from mothers of premature infants with SBS may require protein and caloric fortification (see Chapter 6). Breastmilk contains growth factors, nucleotides, glutamine, and other amino acids that may play an important role in assisting intestinal adaptation.

The selection of an enteral formula if breastmilk is unavailable is somewhat controversial. Studies suggest that complex nutrients requiring more work for digestion

and absorption tend to stimulate adaptation more effectively.[7] On the other hand, the limited mucosal surface area can lead to lactose, protein, and long chain fatty acid malabsorption with the use of intact formulas. If malabsorption is severe, fluid, electrolyte, and metabolic balance can be difficult to achieve. Therefore, it is customary to use protein hydrolysate formulas that are lactose-free and include a portion of their fat source as medium chain triglycerides (MCTs).

Medium chain triglycerides are more water soluble than long chain triglycerides (LCTs) and are better absorbed in the presence of bile acid or pancreatic insufficiency. Medium chain triglyceride fats, however, have a slightly lower caloric density and exert a greater osmotic load in the small intestine; a mixture of LCT and MCT may be helpful. Although fat tends to be poorly absorbed in SBS, it is a dense calorie source. Considering the relatively greater adverse effect of carbohydrate on osmotic diarrhea, it is usually advantageous for patients with SBS to include at least moderate amounts of fat in their diets.[7]

Carbohydrates may be poorly tolerated as they are broken down by gastrointestinal bacteria into small, osmotically active organic acids that can exert a major osmotic load in the distal small intestine and colon. Glucose may be absorbed without hydrolysis, but its small molecular weight increases solution osmolality. Carbohydrate can be given as glucose polymers to decrease the osmotic load. Fiber supplementation may be helpful in the older child with SBS since some fermentation will occur, producing trophic short chain fatty acids (SCFAs), which are an important fuel for the colonocyte.

How to Feed

Continuous enteral feedings via a nasogastric or gastrostomy tube are advantageous in the patient with SBS as

they permit constant saturation of carrier transport proteins, thus taking full advantage of the absorptive surface area available. Older children and adults have a better capacity to regulate gastric emptying and therefore tolerate gastric bolus or oral bolus feedings better than infants. Enteral feedings are slowly advanced, first by concentration then by volume. Parenteral calories are decreased by rate or number of hours to maintain nutritional status, control fluid losses, and ensure intestinal adaptation. Intravenous lipids can be continued for provision of additional calories while enteral feedings are advanced. Small quantities of oral feedings should be introduced in infants two or three times a day to stimulate sucking and swallowing and minimize the effect of feeding aversion once enteral feedings are discontinued.

The rate of advancement of enteral feeds should be determined by multiple factors, including stool or ostomy output, gastric residuals, and signs of malabsorption. It is acceptable to have five to ten loose stools per day as long as reducing substances are < 0.5% and stool pH is > 5.5. Elevated reducing substances and/or low stool pH may indicate excessive carbohydrate malabsorption. If intolerance occurs soon after an increase in rate or concentration of the formula, a return should be made to the previously tolerated rate or concentration. Once tolerance is established, advancement can be attempted again. Frequent setbacks are not unusual. Enteral feedings may eventually be transitioned to oral/bolus feedings or oral/bolus and nocturnal feedings to allow more freedom from the feeding pump. Oral feedings should consist of small, frequent meals. Tables 36–4 and 36–5 outline guidelines for intiation and advancement of enteral feedings, respectively.

Excess fluid and electrolyte losses may continue to complicate the management of SBS patients on enteral feedings, particularly in patients with high output jejunos-

Table 36-4. Suggested Guidelines for Enteral Feeding Initiation in the Infant with Short Bowel Syndrome

Common contraindications to enteral feedings

- Paralytic or drug-induced ileus
- Grossly bloody stools or ostomy output and/or radiologic changes of intestinal ischemia
- Shock/poor perfusion due to cardiac or respiratory insufficiency
- Bilious and/or persistent vomiting (defined as more than three episodes of emesis in 12 h)
- Clinical suspicion of obstruction or ileus (severe abdominal distension, decreased ostomy or stool output, and/or radiologic changes of obstruction or ileus)

If no contraindications exist:

- Feeds start with breastmilk (full strength) or semielemental formula (20 cal/oz) at 10–20 ml/kg/d continuously x 24 h

Table 36-5. Suggested Guidelines for Enteral Feeding Advancement in the Infant with Short Bowel Syndrome

Feeding advancement principles

Quantify feeding intolerance primarily by stool or ostomy output and secondarily by reducing substances. Reducing substances should be measured daily

Tolerance assessed no more than twice per 24 h. No more than one advance per 24 h period

Ultimate goals: 130–200 mL/kg/d
100–140 kcal/kg/d

If ostomy/stool output precludes advancement at 20 cal/oz for 7 d, then increasing caloric density of the formula should be performed

Isocaloric reductions in PN support should be undertaken simultaneous with feeding advancement

Guidelines for feeding advancement

Stool output:

- If < 10 g/kg/d or < 10 stools/d, then advance rate by 10–20 mL/kg/d
- If 10–20 g/kg/d or 10-12 stools/d, then no change
- If > 20 g/kg/d or > 12 stools/d, then reduce rate or hold feeds*

Table 36-5. continued

Ostomy output:
- If < 2 g/kg/h, then advance rate by 10–20 mL/kg/d
- If 2–3 g/kg/h, then no change
- If > 3 g/kg/h, then reduce rate or hold feeds*

Stool-reducing substances:
- If < 1%, then advance feeds per stool or ostomy output
- If = 1%, then no change
- If > 1%, then reduce rate or hold feeds*

Signs of dehydration:
- If absent, then advance feeds per stool or ostomy output
- If present, then reduce rate or hold feeds*, provide additional rehydration fluid.

Gastric aspirates:
- < four times previous hour's infusion, then advance feeds
- > four times previous hour's infusion, then reduce rate or hold feeds*

NB: Oral feeds may be offered as follows:
1. Infant is developmentally able to feed by mouth (PO)
2. *One hour's* worth of continuous feeds may be offered PO QD-TID after 5 days of continuous feeds. During this time, tube feeds should be held
3. *More than 1 hour's* worth of continuous feeds may be offered PO once the infant has reached full volume of feeds by continuous route or at least 7 days have passed on the feeding advancement protocol

PN = parental nutrition.
*Feeds should generally be held for 8 h, then restarted at $^3/_4$ the previous rate.

tomies. Oral rehydration solutions with a sodium concentration of 75 to 90 mEq/L should be used to replace losses (see Chapter 26 for commercially available oral rehydration solutions).

Experimental Nutrients in Short Bowel Syndrome

The role of glutamine in gut adaptation in humans remains controversial with supportive data on both sides. Glutamine in combination with growth hormone has been evaluated as a therapy for adults with SBS.[8,9] Growth hormone

causes hypertrophy of the gastrointestinal (GI) tract and increases body weight, distal ileal weight, and mucosal weight in rats undergoing 75% resection of small bowel.[10] Studies of growth hormone use in humans have shown mixed results, although its use in young children is being actively researched.

Metabolic Complications of Short Bowel Syndrome

In patients with steatorrhea, long chain fatty acids (LCFA) combine with magnesium and calcium, contributing to a deficiency of these minerals. Calcium becomes unavailable for the formation of calcium oxalate, and bile salts in the colon are thought to increase mucosal permeability to oxalate. These two factors combine to increase enteric oxalate absorption, which in turn increases the risk of oxalate renal stones.[5]

There is an increased incidence of gallstones among patients with a jejunostomy and those with short bowel in continuity with the colon. It is assumed that precipitation of cholesterol occurs due to the low concentration of bile salts in bile as a consequence of ileal resection causing an interruption of the enterohepatic circulation.[11]

Gastrin secretion is increased, probably due to the loss of the normal feedback mechanism. This results in excess gastric acid, which alters luminal pH of the small bowel and adversely dilutes or inhibits pancreatic lipase or trypsin activity.[5] Hyperacid secretion impairs carbohydrate and protein digestion and absorption, micellar formation, and fat lipolysis, which causes malabsorption and diarrhea.[12] Acid blockers may be used to decrease gastric acid and improve absorption.

In SBS, overgrowth of bacteria in the small intestine results in deconjugation of bile acids and maldigestion. Bacterial overgrowth should be suspected whenever patients with SBS experience growth regression, require

additional calories, or lose weight.[7] An additional complication of bacterial overgrowth is a neurologic syndrome associated with D-lactic acidosis. Symptoms include headache, drowsiness, stupor, confusion, behavioral disturbance, ataxia, blurred vision, opthalamoplegia, and nystagmus. This should be suspected when there is an acidosis with an unexplained anion gap. Bacterial overgrowth can be treated with broad-spectrum antibiotics.

Once patients are off PN, vitamin replacement is usually necessary. Oral replacement may require several times the minimum daily requirements (see Chapter 5 for USRDAs).

Trace element requirements need to be monitored closely. Zinc and copper deficiencies are common in SBS patients, especially those with intestinal stomas.[5] Iron deficiency can result from loss of duodenal-jejunal absorptive area. Calcium supplementation may be required to minimize oxalate absorption.

Summary

Nutritional management of SBS is a multistage process that may take years. Aggressive use of enteral nutrition to stimulate intestinal adaptation, and recognition and treatment of possible complications, can significantly improve prognosis.

References

1. Kaufman SS, Loseke CA, Lupo JV, et al. Influence of bacterial overgrowth and intestinal inflammation on duration of parenteral nutrition in children with short bowel syndrome. J Pediatr 1997;131:356–61.
2. Sondheimer JM, Cadnapaphornchai M, Sontag M, Zerbe GO. Predicting the duration of dependence on parenteral nutrition after neonatal intestinal resection. J Pediatr 1998; 132(1):80–4.

3. Weaver LT. Anatomy and embryology. In: Walker WA, editor. Pediatric gastrointestinal disease. Philadelphia: B. C. Decker, Inc.; 1991. p. 195–215.
4. Kurkchubasche AG, Rowe MI, Smith SD. Adaptation in short-bowel syndrome: reassessing old limits. J Pediatr Surg 1993;28:1069–71.
5. Ziegler MM. Short bowel syndrome in infancy: etiology and management. Clin Perinatol 1986;13:163–73.
6. Vanderhoof JA. Short bowel syndrome. In: Walker WA, Watkins JB, editors. Nutrition in pediatrics. Hamilton (ON): B.C. Decker, Inc.; 1997. p. 609–18.
7. Vanderhoof JA. Short bowel syndrome: pathophysiology and management. Int Semin Pediatr Gastroenterol Nutr 1997; 6:3–9.
8. Byrne TA, Persinger RL, Young LS, et al. A new treatment for patients with short-bowel syndrome. Ann Surg 1995;222: 242–55.
9. Scolapio JS, Camilleri M, Fleming CR, et al. Effect of growth hormone, glutamine, and diet on adaptation in short-bowel syndrome: a randomized, controlled study. Gastroenterology 1997;113:1074–81.
10. Shulman DI, Hu CS, Duckett G, Lavallee-Grey M. Effects of short-term growth hormone therapy in rats undergoing 75% small intestinal resection. J Pediatr Gastroenterol Nutr 1992; 14(1):3–11.
11. Lennard-Jones JE. Review article: practical management of the short bowel. Aliment Pharmacol Ther 1994;8:563–77.
12. Lifschitz CH. Enteral feeding in short small bowel. In: Baker SB, Baker RD, Davis AD, editors. Pediatric enteral nutrition. New York: Chapman & Hall; 1994. p. 280–90.

Appendix A

Linda Gallagher Olsen, MEd, RD

Conversion Tables (Approximate)

Mass/Weight

1 ounce	=	28 grams
1/4 pound	=	0.11 kilograms
1/2 pound	=	0.23 kilograms
3/4 pound	=	0.34 kilograms
1 pound	=	0.45 kilograms
1 gram	=	0.036 ounces
1 kilogram	=	2.2 pounds

To convert ounces to grams, multiply by 28; grams to ounces, divide by 28. To convert pounds to kilograms, multiply by 0.45; kilograms to pounds, multiply by 2.2.

Length

1 inch	=	2.54 centimeters
1 foot	=	30.5 centimeters
1 yard	=	0.91 meters
1 mile	=	1.61 kilometers
1 millimeter	=	0.04 inches
1 centimeter	=	0.4 inches
1 meter	=	3.3 feet
1 meter	=	1.1 yard
1 kilometer	=	1093.6 yards

To convert inches to centimeters, multiply by 2.54; centimeters to inches, multiply by 0.4.

Area

1 square inch	=	6.5 square centimeters
1 square foot	=	0.0929 square meters
1 square yard	=	0.84 square meters
1 square centimeter	=	0.16 square inches
1 square meter	=	1.2 square yards

Liquid

1 teaspoon	=	5 milliliters
1 tablespoon	=	15 milliliters
1 ounce	=	30 milliliters
8 ounces	=	236 milliliters
32 ounces (1 quart)	=	946 milliliters

1 milliliter	=	0.03 fluid ounces
1 liter	=	1.06 quarts

To convert milliliters to ounces, divide by 30; ounces to milliliters, multiply by 30.

Temperature		
Water freezes	0°C	32°F
Room temperature	27°C	80.6°F
Body temperature	37°C	98.6°F
Water boils	100°C	212°F

To convert Fahrenheit to Celsius (centigrade), subtract 32, multiply by 5, divide by 9; Celsius (centigrade) to Fahrenheit, multiply by 9, divide by 5, and add 32.

Milliequivalent-Milligram Conversion Table

Mineral Element	*Chemical Symbol*	*Atomic Weight*	*Valence*
Calcium	Ca	40	2
Chlorine	Cl	35.4	1
Magnesium	Mg	24.3	2
Phosphorus	P	31	2
Potassium	K	39	1
Sodium	Na	23	1
Sulfur	S	32	2
Zinc	Zn	65.4	2
Sulfate	S04	96	2

$$\text{Milliequivalents} = \frac{\text{milligrams}}{\text{atomic weight}} \times \text{valence}$$

Example: convert 2,000 mg sodium to milliequivalents of sodium:

$$\frac{2{,}000}{23} \times 1 = 87 \text{ mEq sodium}$$

To change milliquivalents back to milligrams, multiply the milliequivalents by the atomic weight, and divide by the valence.

Example: convert 20 mEq sodium to milligrams of sodium:

$$\frac{20 \times 23}{1} = 460 \text{ mg sodium}$$

Nitrogen to Protein

$$\text{grams of nitrogen} = \frac{\text{grams of protein}}{6.25}$$

Appendix B

Kathleen M. Gura, PharmD, BCNSP

Drug-Nutrient Interactions

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Anticoagulants		
Coumarins (warfarin, Coumadin)	Vitamin K antagonist; concomittent use of warfarin with vitamin K may decrease anticoagulant effects; high doses of vitamins A, E, or C may alter prothrombin time; fried or boiled onions may ↑ drug effect by ↑ fibrinolytic activity	N/V/D; hemorrhage, anorexia
Antihistamine drugs		
Cyproheptadine (Periactin)	Appetite stimulant; increased weight gain and growth rate	Xerostomia; N/V/D, abdominal pain
Anti-infective drugs		
Antibiotics General	Decreased synthesis of vitamin K by gut microflora; some are folate and B_{12} antagonists	N/V/D
Aminoglycosides	Increased urinary excretion of potassium and magnesium	Decreased appetite, N/V
Cephalosporins	Possible nephrotoxicity with vitamin K deficiency	GI mucosa damage
Chloramphenicol (Chloromycetin)	Decreased protein synthesis; increased need for riboflavin, B_6, B_{12}	V/D, stomatitis, enterocolitis
Macrolides		Abdominal pain, cramping, N/V/D, stomatitis

Possible Effects on Laboratory Values	*Comments/Recommendations*
Increased bleeding time	Consistent intake of vitamin K essential; breast-fed infants may be more sensitive to warfarin due to low amounts of vitamin K in breast milk; herbal teas/tonka beans/melilot and woodruff contain natural coumarins and will ↑ warfarin effects
May interfere with response to diagnostic antigen skin tests; ↑ amylase; ↓ fasting glucose	Administer with food, milk, or water
Anemia	
↑ BUN, ↑ AST, ↑ ALT, ↑ LDH, ↑ bilirubin; ↓ calcium, ↓ magnesium, ↓ potassium, ↓ sodium	
Prolongation of PT; ↓ potassium	
Anemia	Take on an empty stomach
False (+) urinary catecholamines, 17-hydroxycorticosteroids, 17-ketosteroids	Avoid milk/acidic beverages 1 hour before of after a dose; administer with food to ↓ GI upset

Drug Nutrient Interactions continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Neomycin (Mycifradin)	Decreased absorption of fat, MCT, vitamins A,D,K, and B_{12}, sodium, glucose, lactose, sucrose, xylose	N/V/D; colitis; candidiasis; inactivation of bile salts; GI mucosal damage; decreased activity of disaccharidases; lipase inhibition
Penicillins	Increased urinary potassium excretion; may inactivate B_6; food ↓ drug absorption	Decreased appetite; diarrhea
Quinolones	Dairy foods decrease drug concentrations; may increase caffeine concentrations	N/V/D, GI bleeding, abdominal pain, pseudomembranous colitis
Sulfonamides	Decreased synthesis of folic acid, B vitamins, vitamin K; decreased iron absorption; increased urinary excretion of vitamin C; presence of food delays but doesn't ↓ absorption	Decreased appetite, N/V, stomatitis, pseudomembranous colitis, abdominal pain
Tetracyclines	Chelate divalent ions; decreased absorption of calcium, iron, magnesium, zinc, amino acids; increased urinary excretion of vitamin C; absorption of tetracycline hydrochloride ↓ by 50% when taken with milk/dairy products	N/V/D, anorexia, stomatitis, glossitis; antibiotic associated pseudomembranous colitis; esophagitis, oral candidiasis
Trimethoprim (Trimpex, TMP)	Decreased folate concentrations	N/V, epigastric distress

Possible Effects on Laboratory Values	*Comments/Recommendations*
Increased BUN, creatinine	
↓ potassium, false positive or negative urinary glucose determined using Clinitest, positive Coombs' (direct); false positive urinary serum proteins	Administer on an empty stomach (1 hour before or 2 hours after meals)
Anemia, ↑ ALT, ↑ AST, ↑ alk phos, ↑ BUN, ↑ creatinine	Administer 2 hours after meals, may take with food to ↓ GI upset
	Avoid large amounts of vitamin C or acidifying agents (cranberry juice) to prevent crystalluria
↑ BUN, ↑ alk phos, ↑ bilirubin, ↑ AST, ↑ ALT, false (–) urine glucose with Clinistix	Take on empty stomach 1 hour before/2 hours after dose; avoid milk/dairy products, polyvalent ions within 2–3 hours of dose; Doxycycline and minocycline may be given without regard to meals but best to avoid concurrent administration with milk/dairy products
Anemia, ↑ AST, ↑ ALT, ↑ alk phos, ↑ BUN, ↑ creatinine	Leucovorin may be given until normal hematopoiesis is restored

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Antifungals		
Amphotericin B (Fungizone)	Possible nephrotoxicity with increased urinary excretion of potassium and magnesium	Decreased appetite, N/V, steatorrhea, diarrhea with oral formulation
Fluconazole (Diflucan)	Food delays time of peak absorption but has no effect on total amount of drug absorbed	Mild–moderate GI upset (N/V/D), abdominal pain
Flucytosine (Ancobon)	Food ↓ rate but not extent of absorption; magnesium or aluminum salts delay rate of absorption	N/V/D; enterocolitis
Griseofulvin (Grisactin, Fulvicin)	High fat foods ↑ absorption rate	N/V/D; oral thrush
Itraconazole (Sporanox)	Food ↑ absorption of capsule formulation; hypochlorhydria may ↓ absorption	N/V/D; abdominal pain; anorexia
Ketoconazole (Nizoral)	Food ↑ rate and extent of absorption	N/V/D, abdominal discomfort, GI bleeding
Antimalarials		
Chloroquine phosphate (Aralen)		N/V/D, anorexia, stomatitis, weight loss
Hydroxychloroquine (Plaquenil)		N/V/D, anorexia
Primaquine phosphate		N/V, abdominal cramps
Pyrimethamine (Daraprim)	↓ serum folate concentrations	Anorexia, abdominal cramps, V/D, atrophic glossitis

Possible Effects on Laboratory Values	*Comments/Recommendations*
↓ potassium, magnesium ↑ BUN, creatinine	Monitor potassium, magnesium; supplementation usually necessary
↓ potassium, ↑ cholesterol, ↑ triglycerides, ↑ AST, ↑ ALT, ↑ alk phos	
↑ BUN, creatinine ↑ ALT, AST, CK, LDH, ↑ alk phos False ↑ in serum creatinine values if Ektachem analyzer used	May take with food
False (+) urinary VMA levels	Give with fatty meals to ↑ absorption as well as avoid GI upset
↓ potassium; ↑ ALT, ↑ AST, ↑ LDH, ↑ alk phos, ↑ Tg	Take capsules with food; take oral solution on an empty stomach
↑ AST, ↑ ALT, ↑ alk phos	Take with food to ↓ GI upset
	Take with food to ↓ GI upset; bitter taste may be masked by mixing with chocolate syrup
↑ ALT, ↑ AST, ↑ bilirubin, ↑ PT, anemia	Take with food to ↓ GI upset
Anemia	Take with food to ↓ GI upset, drug has bitter taste
Anemia	Take with meals to ↓ GI upset; leucovorin may be given until normal hematopoiesis is restored

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Sulfadoxine and Pyrimethamine (Fansidar)	↓ serum folate concentrations	Anorexia, gastritis, glossitis, V/D
Antitubercular agents		
Cycloserine (Seromycin)	B_6 antagonist; ↓ absorption of calcium, magnesium, vitamin B_{12}; decreased folate utilization and vitamin K synthesis	
Ethambutol (Myambutol)		N/V, abdominal pain, anorexia
Ethionamide (Trecator)	Increased pyridoxine requirements	N/V/D, abdominal pain, excessive salivation, metallic taste, anorexia, weight loss, stomatitis
Isoniazid (Laniazid)	Inactivates and increases excretion of B_6; blocks conversion of tyrosine to niacin; interacts with foods containing histamine to cause headache, redness, itching, chills, palpitations, and hypotension; isoniazid also has some monoamine oxidase inhibitor activity, and hypertensive crisis may result if taken with tyramine-containing foods; rate & extent of isoniazid absorption ↓ when given with food	Decreased appetite, N/V, epigastric distress, diarrhea seen with use of syrup
Paramino-salicylic acid (PAS, Paser)	Malabsorption of B_{12}, folate, calcium, iron, magnesium	Altered mucosal transport mechanisms (↑ peristalsis); GI upset

Possible Effects on Laboratory Values	*Comments/Recommendations*
↑ ALT, ↑ AST, anemia	Take with meals; leucovorin may be given until normal hematopoiesis is restored
Anemia, ↑ ALT, ↑ AST	Some neurotoxic effects may be prevented or lessened by pyridoxine supplementation
↑ uric acid levels, abnormal LFTs	Take with food to ↓ GI upset
↓ BS, ↑ ALT, ↑ AST, ↑ bilirubin	Take with food to ↓ GI upset Increase dietary intake of pyridoxine to prevent neurotoxic effects
Anemia, ↑ ALT, ↑ AST, ↑ bilirubin; false (+) urinary glucose with Clinitest	Avoid foods with histamine or tyramine; increase dietary intake of folate/niacin/pyridoxine; pyridoxine supplementations should be given to malnourished patients, patients on meat- or milk-deficient diets, and breast-fed infants; take on an empty stomach
↑ ALT, ↑ AST, ↓ potassium (rare); ↓ cholesterol	B_{12} supplementation for patients receiving PAS >1 month; administer in acidic foods or juices

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Rifabutin (Mycobutin)	High-fat meals ↓ rate but not extent of absorption	N/V/D, abdominal pain, dyspepsia, taste perversion, ageusia
Rifampin (Rifadin, Rimactane)	Food ↓ absorption and bioavailability	Heartburn, N/V/D, anorexia, abdominal cramps
Antiviral agents		
Acyclovir (Zovirax)		N/V
Didanosine (Videx)	May alter GI absorption of various nutrients due to prolonged GI transit time	N/V, constipation, xerostomia, dry throat, dysphagia
Famciclovir (Famvir)	Rate of absorption and/or conversion to penciclovir and peak concentration are ↓ with food; bioavailability not affected	N/V/D, constipation, anorexia, abdominal pain
Foscarnet (Foscavir)		N/V/D, weight loss, pancreatitis
Ganciclovir (Cytovene)	Food ↑ AUC; time to peak concentration is prolonged	N/V/D, pancreatitis
Indinavir (Crixivan)	↓ absorption when given with high amounts of protein or fatty foods; grapefruit juice ↓ AUC by 26%	N/V/D, abdominal pain

Possible Effects on Laboratory Values	*Comments/Recommendations*
↑ ALT, ↑ AST, anemia	May take with meals to ↓ GI upset
↑ ALT, ↑ AST, ↑ bilirubin, ↑ alk phos, anemia, ↑ BUN, ↑ serum uric acid, ↓ hemoglobin; rifampin interferes with microbiologic assays for serum folate and B_{12}	Take on an empty stomach; may take with food to ↓ GI upset
Anemia; ↑ AST, ↑ ALT, ↑ alk phos, ↑ BUN, ↑ creatinine	Keep well hydrated; suspension is banana-flavored; may administer with food Buffered powder for oral solution is inactivated in acidic juices/fluids
	May take with food to ↓ GI upset
↓ calcium, ↓ magnesium, ↓ potassium, alterations in phosphorus (↑↓), ↑ BUN, ↑ creatinine, ↓ Hct, ↓ Hb	
Anemia, ↑ ALT, ↑ AST, ↑ alk phos, ↑ BUN, ↑ creatinine	May take with food
↑ ALT, ↑ AST, ↑ alk phos, ↑ bilirubin, hyperglycemia (rare)	Ensure adequate hydration; take on empty stomach; if GI upset a problem, take with light meals or other liquids

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Lamivudine (Epivir, 3TC)	Food may ↓ rate of absorption and peak serum concentrations, but not does not significantly change the AUC	N/V/D, feeding problems, abdominal discomfort, pancreatitis, anorexia
Nelfinavir (Viracept)	Food ↑ absorption	N/V/D, abdominal pain, anorexia, dyspepsia epigastric pain, mouth ulceration, GI bleeding, pancreatitis
Ritonavir (Norvir)	Food ↑ absorption; may cause avitaminosis	N/V/D, taste perversion, abdominal pain, pancreatitis
Saquinavir (Fortovase, Invirase)	High-fat meals maximize bioavailability; grapefruit juice ↑ saquinavir levels	N/V/D, abdominal discomfort, stomatitis
Stavudine (Zerit, d4T)	Food ↓ peak serum concentrations by 45%, bioavailability not changed	N/V/D, abdominal pain, anorexia, pancreatitis
Zalcitabine (Hivid, ddC)	Food ↓ rate and extent of absorption, AUC ↓ by 14%	N/V/D, oral/esophageal ulcers, dysphagia, anorexia, abdominal pain, constipation, pancreatitis, weight loss, anemia
Zidovudine (Retrovir, AZT, ZDV)	Folate/B_{12} deficiency increases zidovudine-associated myelosuppression; rate of absorption and peak serum concentration may ↓ when taken with food	N/V/D, anorexia

Possible Effects on Laboratory Values	*Comments/Recommendations*
↑ ALT, ↑ AST, ↑ bilirubin, ↑ amylase	
↑ ALT, ↑ AST, ↑ alk phos, hyperlipidemia, hyperuricemia, hyperglycemia, anemia	Powder formulation contains 11.2 mg phenylalanine per gram powder; do not administer with acidic foods or juices (results in bitter taste)
↑ triglycerides, ↑ cholesterol, ↑ creatine phosphokinase, hyperglycemia (rare), ↑ ALT, ↑ AST, ↑ alk phos, alterations in potassium (↑ ↓)	Administer with food to ↑ absorption; liquid formulations taste unpleasant, reserve use for tube-fed patients or mix with chocolate milk or nutritional supplement
Hyperglycemia, ↑ creatine phosphokinase, ↑ ALT, ↑ AST, ↑ bilirubin, ↑ amylase, alterations in potassium and phosphorus (↑ ↓), ↑ calcium	Take within 2 hours of a full meal; high-calorie/high-fat meals ↑ AUC & C max more than low-calorie/low-fat meals
↑ ALT, ↑ AST	Take without regard to food
Hyperglycemia, ↓ calcium	Take on empty stomach
↑ AST, ↑ LDH, ↑ alk phos, anemia	May take with food; take capsules while in upright position to ↓ risk of esophageal ulceration; syrup is strawberry-flavored

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Miscellaneous anti-infective agents		
Clofazimine (Lamprene)	Food ↑ extent of absorption	N/V/D, abdominal pain, constipation, bowel obstruction, GI bleeding, dysgeusia
Furazolidone (Furoxone)	Large doses or prolonged therapy ↑ risk of hypertensive effects if taken with tyramine-containing foods	N/V/D
Methenamine (Hiprex, Mandelamine)	Foods/diets that alkalinize urine pH > 5.5 ↓ activity of methenamine; cranberry juice can be used to acidify urine and ↑ activity of methenamine	N/V/D, abdominal cramping, anorexia, stomatitis
Nalidixic acid (NegGram)		N/V/D, abdominal pain
Nitrofurantoin (Furadantin, Macrodantin)	Food ↑ total amount absorbed; cranberry juice or other urine acidifiers enhance drug action	N/V, anorexia, pancreatitis
Pentamidine (Pentam)		N/V, metallic taste, pancreatitis
Antihyperlipidemics		
Cholestyramine (Questran)	Decreased absorption of fat, MCT, fat-soluble vitamins, B_{12}, iron, folate, calcium, glucose, xylose, electrolytes	Constipation, nausea, anorexia, weight changes, abdominal distention

Possible Effects on Laboratory Values	*Comments/Recommendations*
Hyperglycemia	Administer with meals/milk to maximize absorption
Hypoglycemia; false (+) urine glucose results with Clinitest	
Albuminuria, ↑ AST, ↑ ALT, ↓ urine pH	
Anemia, false (+) urine glucose with Clinitest, false ↑ in urinary VMA	Suspension is raspberry flavored
Anemia	Administer with food or milk
Anemia, ↑ potassium, hypo/hyperglycemia, ↓ magnesium, ↓ calcium, ↑ BUN, ↑ creatinine	
↑ Tg, ↑ ALT, ↑ AST, ↑ phosphorus, ↑ chloride, ↑ alk phos; ↓ cholesterol, ↓ LDL, ↓ calcium, ↓ potassium, ↓ sodium	Administer before meals; to minimize binding, administer vitamins/minerals 1 hour before or 4–6 hours after cholestyramine

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Clofibrate	↓ absorption of carotene, B_{12}, iron, electrolytes, MCT, glucose, xylose	↓ activity of intestinal disaccharidases; N/D
Colestipol	↓ absorption of fat-soluble vitamins	
Antihypertensives		
Hydralazine (Apresoline)	Inactivates B_6	N/V/D, constipation, paralytic ileus, anorexia
Methyldopa (Aldomet)	Increased need for B_{12}, folate	N/D, colitis, liver disorders, xerostomia, "black" tongue
Anti-inflammatory agents		
Salicylates	Increased vitamin C requirements; possible iron deficiency; may ↓ serum folate levels	N/V/D, GI bleeding
Indomethacin (Indocin)	Decreased absorption of amino acids, glucose, xylose	N/V/D,constipation, dyspepsia, GI bleeding
Corticosteroids	↑ protein catabolism, ↓ glucose tolerance, ↑ sodium and water retention, ↓ absorption and ↑ excretion of potassium, zinc, vitamin C, calcium, and phosphorus; accelerated vitamin D metabolism; ↓ B_6 and folate requirements; possible growth suppression and impaired wound healing	Increased appetite, N/V

Possible Effects on Laboratory Values	*Comments/Recommendations*
↑ ALT, ↑ AST, ↑ CPK	
↓ Hgb, ↓ WBC	Administer with food
Anemia, ↑ BUN, ↑ alk phos, ↑ AST, ↑ ALT, ↑ bilirubin, ↑ sodium, ↑ potassium	Dietary requirements for B_{12} and folate may be increased
Proteinuria, increased bleeding time; interferes with Gerhardt's test, VMA determinators, 5-HIAA, xylose tolerance test	Administer with food
↑ potassium, anemia, ↓ vitamin C	Administer with food
↑ glucose, ↑ Tg, ↑ cholesterol, ↑ sodium; ↓ potassium, ↓ calcium, ↓ T4, ↓ uric acid, ↓ zinc	Administer with food/milk

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Antineoplastic drugs		
Cyclophosphamide (Cytoxan)		Anorexia, N/V, mucosal injury
Dactinomycin (Cosmegen)	Decreased absorption of calcium, iron, and fat	Anorexia; N/V
Fluorouracil (5-FU)	Increased need for B_1; malabsorption of glucose, xylose	Severe N/V/D, GI bleeding, anorexia, stomatitis, esophagitis
Methotrexate (MTX)	Folate antagonist, ↓ absorption of fat, B_{12}, lactose, carotene, cholesterol	GI mucosal injury, anorexia, N/V/D, stomatitis
Autonomic drugs		
Anticholinergics (general)	Decreased absorption of electrolytes, iron, increased absorption of monosaccharides	N/V, constipation
Cardiovascular drugs		
Cardiac glycosides	↓ absorption glucose, xylose; ↑ renal excretion of Ca, Mg, Zn, K	GI irritation, anorexia, diarrhea, constipation
Central nervous system drugs		
Anticonvulsants		
Phenobarbital Phenytoin Primidone	Vitamin D deficiency; osteomalacia, ↓ absorption of folate, B_{12}; ↑ vitamin K catabolism; high doses of pyridoxine may decrease effects of phenobarbital	N/V/D

Possible Effects on Laboratory Values	*Comments/Recommendations*
Anemia, ↑ uric acid	Maintain high fluid intake; take with food only if GI distress occurs
Anemia, ↓ albumin, ↑ alk phos, ↑ ALT, ↑ bilirubin, ↑ LDH	Increase dietary intake of thiamine; use of acidic solutions to dilute fluorouracil for oral use may result in precipitation of drug and ↓ absorption
Anemia, ↑ uric acid, ↑ ALT, ↑ bilirubin	Milk-rich foods may decrease absorption, folate may decrease drug response
↑ potassium with acute toxicity	Meals high in fiber or pectin ↓ oral absorption of digoxin. Maintain adequate amounts of potassium in diet to ↓ risk of hypokalemia and digoxin toxicity
↑ alk phos; ↓ calcium, ↓ magnesium, ↓ folate, ↓ B_{12}, ↓ vitamins K, B_6, C; megaloblastic anemia	*Tube feedings ↓ phenytoin* bioavailability; to ensure consistent absorption, administer at same time with regard to meals

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Psychotherapeutic agents		
Chlorpromazine	Interferes with riboflavin metabolism; ↓ B_{12} absorption	Constipation; increased appetite and weight, xerostomia
Imipramine Amitriptyline	May increase need for riboflavin	N/V/D, constipation, increased or decreased weight; altered taste
Lithium (Eskalith, Lithobid)	Decreased calcium uptake by bones; may inhibit magnesium-dependent enzymes; alters glucose tolerance	N/V/D, increased appetite, xerostomia
Cerebral stimulants		
Dextro-amphetamine (Dexedrine)	Acidic foods, juices, or vitamin C may decrease GI absorption	Appetite suppression and weight loss, growth suppression, N/V/D, xerostomia, metallic taste
Methylphenidate (Ritalin)	Food may increase oral absorption	Decreased appetite, depression of height and weight, nausea
Sedatives		
Barbiturates	Increased excretion of vitamin C; folate and vitamin D deficiency; ↓ absorption of B_1	N/V
Electrolytes and water balance drugs		
Diuretics		
Thiazides	↑ excretion of potassium, magnesium, zinc, riboflavin; glucose intolerance; calcium excretion	N/V/D, constipation, anorexia; pancreatitis

Possible Effects on Laboratory Values	*Comments/Recommendations*
↑ cholesterol, ↑ bilirubin, false positives for PKU, amylase, uroporphyrins, urobilinogen	Administer with food to ↓ GI upset; dilute oral concentrate solution in juice before administration (undiluted oral concentrate may precipitate in tube feeding)
Increased or decreased glucose	
↑ magnesium, ↑ glucose	Administer with food to ↓ GI upset; avoid changes in sodium content; ↓ in sodium can ↑ glucose; lithium toxicity
	Do not crush or allow patient to chew sustained-release capsules (ie, Spansules)
	Administer on an empty stomach; do not crush/chew sustained-release tablets
↓ B_{12}	Administer phenobarbital elixer with water, milk, or juice
↑ uric acid, ↑ calcium, ↓ potassium, ↓ magnesium, ↓ chloride, ↓ bicarbonate, ↓ phosphorus, ↑ ↓ glucose	Take with food

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Loop diuretics	↑ excretion of magnesium, calcium, potassium, zinc; ↓ carbohydrate tolerance	N/V/D, anorexia; oral solutions may cause diarrhea due to sorbitol content
Spironolactone (Aldactone)	Potassium sparing; ↑ calcium excretion	N/V/D, anorexia, gastritis, cramping, GI bleeding
Replacement solutions		
Potassium chloride	↓ absorption of B_{12}	N/V/D, abdominal pain, GI lesions
Gastrointestinal drugs		
Antacids		
Aluminum hydroxide	↑ calcium absorption; may cause hypophosphatemia. ↓ absorption of vitamins A,C; inactivates B_1	Constipation, anorexia
Magnesium hydroxide		Diarrhea
Antihyperammonemic agents		
Lactulose (Cephulac, Chronulac)		N/D; abdominal discomfort
Cathartics		
Bisacodyl (Dulcolax)	↓ absorption of glucose	N/V, abdominal cramps
Docusate sodium (Colace)	Alters intestinal absorption of water and electrolytes	Diarrhea, abdominal cramping, intestinal obstruction, throat irritation
Magnesium sulfate	↓ nutrient absorption	↑ intestinal transit time; N/V/D

Possible Effects on Laboratory Values	*Comments/Recommendations*
↓ calcium, ↓ magnesium, ↓ potassium, ↓ chloride, ↑ glucose, ↑ BUN, ↑ uric acid	Avoid use of salt substitutes, administer with food; do not mix with acidic solutions
↑ potassium, ↑ BUN, ↑ *creatinine*, ↑ *magnesium*, ↑ uric acid, ↓ sodium, ↓ chloride	
↑ potassium	Administer with food
↓ magnesium, ↓ phosphorus	
↑ magnesium	
↓ ammonia	Contraindicated in galactose-restricted diets; administer with juice/milk
↓ potassium, ↓ calcium	Administer on empty stomach, do not administer within 1 hour of ingesting milk or dairy products (causes GI irritation)
↑ glucose, ↓ potassium	Administer liquid (not syrup) with milk/juice to mask bitter taste
↑ magnesium	

Drug Nutrient Interactions. continued

	Nutritional Considerations	*Possible Gastrointestinal Side Effects*
Mineral oil	May ↓ absorption of fat-soluble vitamins; impairs calcium, carotene, and phosphorus absorption	Decreased weight; anorexia, N/V/D, abdominal cramps, anal itching
Antisecretory agents		
H_2 antagonists	↓ B_{12}, ↓ absorption of iron salts	Nausea, constipation
Proton pump inhibitors	↓ absorption of iron salts	N/V/D; abdominal pain, constipation, xerostomia, anorexia; dysgeusia, discoloration of feces
Sucralfate	↓ absorption of fat-soluble vitamins; aluminum salt may accumulate in renal failure	N/D, constipation, gastric discomfort, xerostomia
Hormones and synthetic substitutes		
Oral contraceptives	↓ absorption of water-soluble vitamins, ↓ magnesium, ↓ zinc; ↑ copper absorption	N/V, ↑ ↓ weight, bloating
Unclassified agents		
Colchicine	↓ absorption of B_{12}, vitamin A, folate, potassium, fat, sodium, nitrogen, lactose	Intestinal mucosal damage; N/V/D, constipation, GI bleeding, steatorrhea

Possible Effects on Laboratory Values	*Comments/Recommendations*
	Emulsified mineral oil more palatable than non-emulsified products; administer non-emulsified mineral oil on an empty stomach
↑ AST, ↑ ALT, ↑ creatinine	Administer with food. Limit xanthine-containing foods
hypoglycemia, ↑ AST, ↑ ALT	Capsule should be swallowed whole. Contact pharmacy for NJ or NG tube administration
↑ aluminum (in renal failure)	Take on empty stomach
Megaloblastic anemia, ↑ glucose, ↑ Tg, ↑ vitamin A, ↑ vitamin E, ↑ iron, ↑ copper, ↑ alk phos, ↑ bilirubin; ↓ folate, ↓ calcium, ↓ magnesium, ↓ B_6, ↓ B_{12}, ↓ zinc	
↑ alk phos, ↑ AST, ↓ B_{12}, ↓ vitamin A, ↓ cholesterol	May need low purine diet during an acute gouty attack

Appendix C

Frances Rohr, MS, RD

Pediatric Genetic/Metabolic Referral Centers

State	*Genetic/Metabolic Referral Centers*
Alabama	Univ. of Alabama, Sparks Clinic, Birmingham, AL
Alaska	Univ. of Washington, PKU Program, Child Development and Mental Retardation Center, Seattle, WA
Arizona	Univ. of Arizona, Dept. of Pediatrics, Section of Genetics, Phoenix, AZ
Arkansas	Univ. of Arkansas for Medical Sciences, Arkansas Genetics Program, Little Rock, AR
California	Children's Hospital, Oakland, Child Development Center, Oakland, CA
	UC San Francisco Medical Center, Pediatrics, San Francisco, CA
	Stanford University Medical Center, Dept. of Pediatrics, Palo Alto, CA
	UC Davis Medical Center, Medical/Pediatrics, Davis, CA
	Harbor/UCLA Medical Center, Div. of Medical Genetics, Torrance, CA
	Children's Hospital of Los Angeles, Medical Genetics, Los Angeles, CA
	Los Angeles/USC Medical Center, Los Angeles, CA
	UCLA School of Medicine, Dept. of Pediatrics/Genetics, Los Angeles, CA
Colorado	The Children's Hospital, Inherited Metabolic Diseases Clinic, Denver, CO
Connecticut	Univ. of Connecticut Health Center, Dept. of Pediatrics, Div. of Human Genetics, Hartford, CT
	Yale Univ. School of Medicine, Metabolic Clinic, New Haven, CT
Delaware	see Pennsylvania
District of Columbia	Howard Univ. & School of Medicine, Genetics Clinic, Washington, DC

	Georgetown University, Center for Genetic Counseling, Washington, DC
	Children's Hospital National Medical Center, Clinical Genetics, Washington, DC
Florida	University Hospital of Jacksonville, Div. of Genetics, Jacksonville, FL
	Univ. of Miami Medical Center, Mailman Center for Child Development, Miami, FL
Georgia	Emory Univ. School of Medicine, Div. of Medical Genetics, Atlanta, GA
	Medical College of Georgia, Div. of Medical Genetics, Augusta, GA
Hawaii	Medical Genetics Services, Chaplain Medical Center, Honolulu, HI
Idaho	The Oregon Health Sciences Univ., Metabolic Clinic, Portland, OR
Illinois	Children's Memorial Hospital, PKU Program, Chicago, IL
	Univ. of Illinois at Chicago, Dept. of Pediatrics, Chicago, IL
Indiana	James Whitcomb Riley Hospital for Children, Metabolism Clinic, Indianapolis, IN
Iowa	Univ. of Iowa, Child Development Center, Iowa City, IA
Kansas	Univ. of Kansas Medical Center, PKU Clinic, Kansas City, KS
Kentucky	Univ. of Kentucky Medical Center, Div. of Endocrine/Metabolism, Lexington, KY
	Univ. of Louisville School of Medicine, Inborn Errors of Metabolism, Louisville, KY
Louisiana	Tulane Univ. Medical Center, Human Genetics Program, New Orleans, LA
Maine	Maine Medical Center, Metabolism Program, Portland, ME
	Eastern Maine Medical Center, Genetics Clinic, Bangor, ME
Maryland	The Johns Hopkins Hospital, Pediatric Genetics Clinic, Baltimore, MD
	Univ. of Maryland Genetics Program, Univ. of Maryland Hospital, Baltimore, MD

Massachusetts	The Children's Hospital, Genetics/Metabolism Program, Boston, MA
	New England Medical Center, Amino Acid Disorders Clinic, Boston, MA
	Massachusetts General Hospital, Amino Acid Disorders Laboratory, Boston, MA
Michigan	University Hospital, Pediatric Metabolic Disease Center, Ann Arbor, MI
	Children's Hospital, Clinic for Genetic, Metabolic, & Developmental Disorders, Detroit, MI
Minnesota	Univ. of Minnesota, Pediatric Metabolism, Minneapolis, MN
	Mayo Clinic, Dept. of Medical Genetics, Rochester, MN
Mississippi	Univ. of Mississippi Medical Center, Dept. of Preventive Medicine, Jackson, MS
Missouri	Cardinal Glennon Memorial Hospital for Children, PKU Clinic, St. Louis, MO
	Washington Univ. School of Medicine, St. Louis Children's Hospital, St. Louis, MO
	Children's Mercy Hospital, Genetic Counseling Center, Kansas City, MO
Montana	See Oregon, Colorado, Utah
Nebraska	Univ. of Nebraska Medical Center, Metabolic Diseases Clinic, Omaha, NE
Nevada	Univ. of Nevada School of Medicine, Nevada Genetics Network, Las Vegas, NV
New Hampshire	See Massachusetts
New Jersey	University Medical School, Div. of Genetics, Newark, NJ
	Rutgers Medical School, University Medical Center, Cooper Hospital, Camden, NJ
	Children's Hospital of New Jersey, PKU Program, East Orange, NJ
New Mexico	Univ. of New Mexico School of Medicine, Metabolic Clinic, Albuquerque, NM
New York	Mt. Sinai Medical Center, Pediatric Metabolic Disease Center, New York, NY
	Albany Medical College, Inherited Metabolic Defects Diagnostic and Treatment Center, Albany, NY

	North Shore University Hospital, Pediatric Endrocrinology, Manhassett, NY
	Children's Hospital, Metabolic Clinic, Buffalo, NY
	Univ. of Rochester Medical Center, Univ. Affiliated Program for Developmental Disabilities, Rochester, NY
North Carolina	Univ. of North Carolina at Chapel Hill, Div. of Genetics and Metabolism, Chapel Hill, NC
	Duke University Medical Center, Div. of Pediatric Metabolism, Durham, NC
	Bowman Gray School of Medicine, Section of Medical Genetics, Winston-Salem, NC
North Dakota	Fargo Clinic Merit Care, Pediatric Endocrine and Metabolic Disease Clinic, Fargo, ND
Ohio	Children's Hospital Research Foundation, Div. of Inborn Errors of Metabolism, Cincinnati, OH
	Cleveland Clinic Foundation, Pediatrics and Adolescent Endocrinology, Cleveland, OH
	Case Western Reserve Univ., Genetics Center, Cleveland, OH
	Children's Medical Center, PKU Clinic, Dayton, OH
Oklahoma	Oklahoma Children's Memorial Hospital, Health Sciences Center, Genetic, Endocrine, and Metabolic Disease Section, Oklahoma City, OK
Oregon	Oregon Health Sciences Univ., Metabolic Clinic, Portland, OR
Pennsylvania	Children's Hospital, PKU Clinic, Pittsburgh, PA
	Milton S. Hershey Medical Center, PKU Treatment Center, Div. of Genetics, Hershey, PA
	Children's Hospital of Philadelphia, Metabolic Diseases, Philadelphia, PA
	St. Christopher's Hospital for Children, PKU Treatment Center, Philadelphia, PA
Rhode Island	Child Development Center, Inherited Metabolic Diseases Program, Providence, RI
South Carolina	Medical Univ. of South Carolina, Dept. of Genetics, Charleston, SC
	Univ. of South Carolina School of Medicine, Dept. of Pediatrics, Columbia, SC

South Dakota	Univ. of South Dakota School of Medicine, Dept. of Pediatrics, Sioux Falls, SD
Tennessee	Univ. of Tennessee, Memphis, Inborn Errors of Metabolism Clinic, Child Development Center, Memphis, TN
	Vanderbilt Univ. School of Medicine, Div. of Genetics, Nashville, TN
Texas	Univ. of Texas Southwest Medical Center, Metabolic Clinic, Dallas, TX
	Texas Children's Hospital, Genetic Metabolic Clinic, Houston, TX
	University of Texas Medical School, Dept. of Pediatrics, Metabolic Clinic, Houston, TX
	Univ. of Texas Medical School, Dept. of Pediatrics, Div. of Child Development, Galveston, TX
	Univ. of Texas Health Sciences Center, San Antonio Medical School, Dept. of Pediatrics, San Antonio, TX
Utah	Univ. of Utah School of Medicine, Dept. of Pediatrics, Salt Lake City, UT
Vermont	Vermont Health Department, Children With Special Health Needs, Child Development Clinic, Burlington, VT
Virginia	Medical College of Virginia, Dept. of Human Genetics, Richmond, VA
	University Hospital, Dept of Pediatrics, Div. of Medical Genetics, Charlottesville, VA
Washington	Univ. of Washington, PKU Program, Child Development and Mental Retardation Center, Seattle, WA
West Virginia	West Virginia Univ. Medical Center, Dept. of Pediatrics, West Virginia Genetics Center, Morgantown, WV
Wisconsin	Children's Hospital of Wisconsin, PKU Program, Milwaukee, WI
	Waisman Center on Mental Retardation and Human Development, Metabolic Clinic, Madison, WI
	Dept. of Pediatrics, Pediatric Endocrinology and Metabolism, Marshfield, WI
Wyoming	See Colorado

Appendix D

Linda Gallagher Olsen, MEd, RD

Major Manufacturers of Enteral Nutrition Products

Applied Nutrition
273 Franklin Road
Randolph, NJ 07869
1-800-605-0410
www.medicalfood.com

Mead Johnson Nutritionals
2400 West Floyd Expressway
Evansville, IN 47721
1-800-457-3550
www.meadjohnson.com

Nestle Clinical Nutrition
Three Parkway North
Suite 500, P.O. Box 760
Deerfield, IL 60015-0760
1-800-422-2752

Novartis Nutrition Corporation
5320 West 23rd Street
Minneapolis, MN 55416
1-800-999-9978
www.novartis.com

Ross Laboratories
625 Cleveland Ave.
Columbus, OH 43215
1-800-544-7495
www.ross.com

SHS North America
P.O. Box 117
Gaithersburg, MD 20884
1-800-365-7354
www.shsna.com

Appendix E

Heidi Quinn, MS, RD

Growth Charts for Specific Syndromes

Anthropometric assessment of the nutritional status of patients with genetic and other medical conditions can be difficult using the National Center for Health Statistics (NCHS) data. To help evaluate the growth patterns of these patients, special weight and height curves for several syndromes have been published. Below are weight and height curves for the following common syndromes (Down, Prader-Willi, Turner's) as well as contact information for obtaining other curves for less common syndromes (Table 1).

Table 1. List of Some Special Growth Charts*

Condition	*Reference(s)*
Achondroplasia	Horton WA, et al. J Pediatr 1978;93:435. Stature, growth velocity, head circumference, upper and lower segments
Brachmann-de Lange syndrome	Kline AD, et al. Am J Med Gent 1993;47: 1042. Length- and weight-for-age birth to 36 months, height- and weight-for-age 2 to 18 years, and head circumference-for-age birth to 18 years
Cerebral palsy (quadriplegia)	Krick J, et al. J Am Diet Assoc 1996;96:680. Stature and weight-for-age and weight-for-stature age birth to 10 years
Down syndrome	Cronk CE, et al. Pediatrics 1978;61:564 and Pediatrics 1988;81:102. Length-for-age and weight-for-age birth to 36 months; stature-for-age and weight-for-age 2 to 18 years
Marfan syndrome	Pyeritz RE. In: Emery AH, Rimoirn DL, editors. Principles and practice of medical genetics. New York: Churchill Livingstone; 1983, and Pyeritz RE. Papadatas CJ, Bartsocas CS, editors. In: Endocrine genetics and genetics

	of growth (Prog Clin Biol Res v200). Alan R. Liss, Inc.; 1985. Stature- and weight-for-age 2 to 18 years, 20 to 24 years, and > 24 years. Upper and lower segment ratios 2 to 20 years and adult
Myelomeningocele	Appendix 2. Ekvall S, editor. Pediatric nutrition in chronic disease and development disorders: prevention, assessment, and treatment. New York: Oxford University Press; 1993. (Preliminary charts) height- and weight-for-age 2 to 18 years
Noonan's syndrome	Witt DR, et al. Clin Genet 1985;30:150. Stature-for-age birth to 18
Prader-Willi syndrome	Holm VA. Appendix A. In: Greeway LR, Alexander PC, editors. Management of Prader-Willi syndrome. New York: Springer Verlag; 1998. p. 317. Height-for-age 3 to 25 years
	Butler MG, et al. Pediatrics 1991;88(4):853. Weight, height, sitting height, head circumference, triceps, and subscapular skinfold (plus other measure) for age 2 to 22 years
Sickle cell disease	Phebus CK, et al. J Pediatr 1984;105:28. Height- and weight-for-age birth to 18 years
	Tanner JM, et al. J Pediatr 1985;107;317–29. Height velocity (cm/yr) age $2^1/_2$ to 19 years
Silver-Russell syndrome	Tanner JM, et al. Pediatr Res 1975;9:611. Height- and height velocity-for-age 2 to 19 years (includes periods of treatment with human growth hormone)
Turner's syndrome	Lyon AJ, et al. Arch Dis Child 1985;60:932. Height-for-age birth to 18 years (girls)
Williams syndrome	Morris CA, et al. J Pediatr 1988;113:318. Stature-for-age birth to 24 months and birth to 18 years, weight-for-age birth to 18 years, and head circumference-for-age birth to 36 months and 2 to 18 years

*Unless otherwise specified, charts are available for both girls and boys.

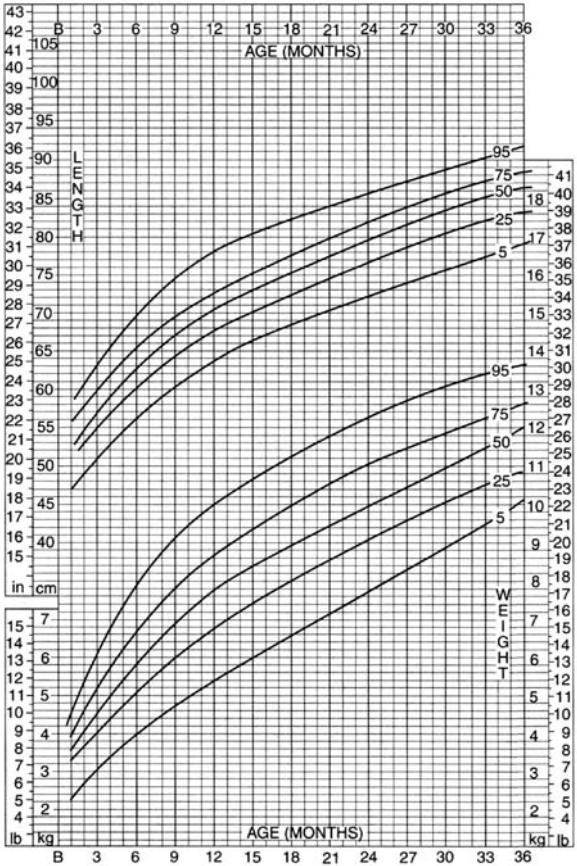


Figure 1. Physical growth of females with Down syndrome (1 to 36 months).

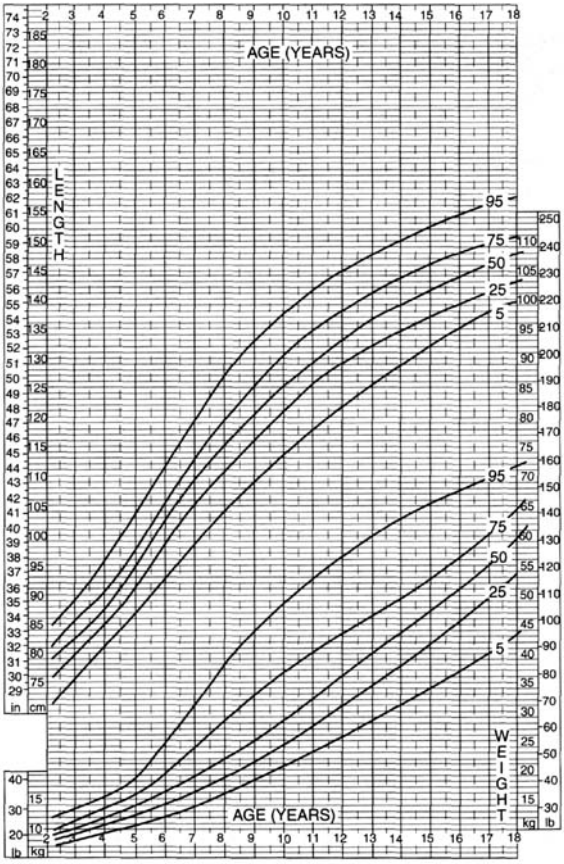


Figure 2. Physical growth of females with Down syndrome (2 to 18 years).

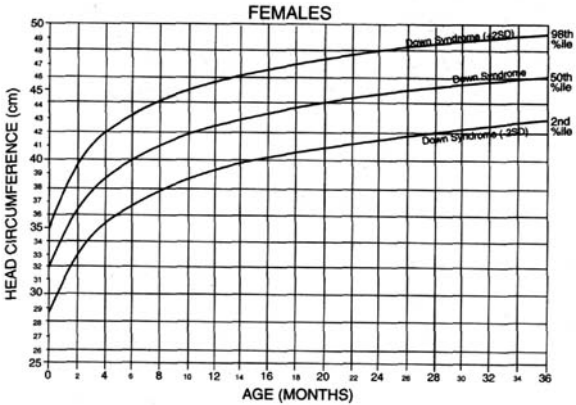


Figure 3. Head circumference of females with Down syndrome (0 to 36 months).

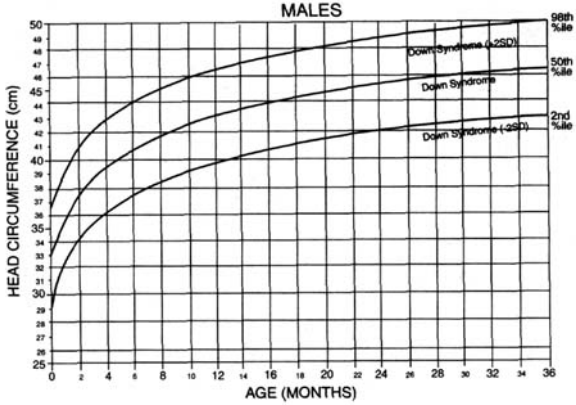


Figure 4. Head circumference of males with Down syndrome (0 to 36 months).

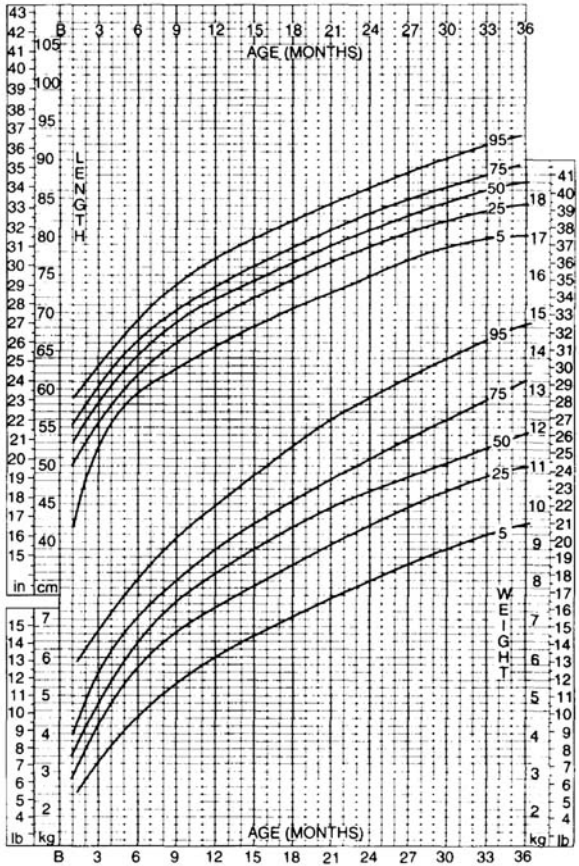


Figure 5. Physical growth of males with Down syndrome (1 to 36 months).

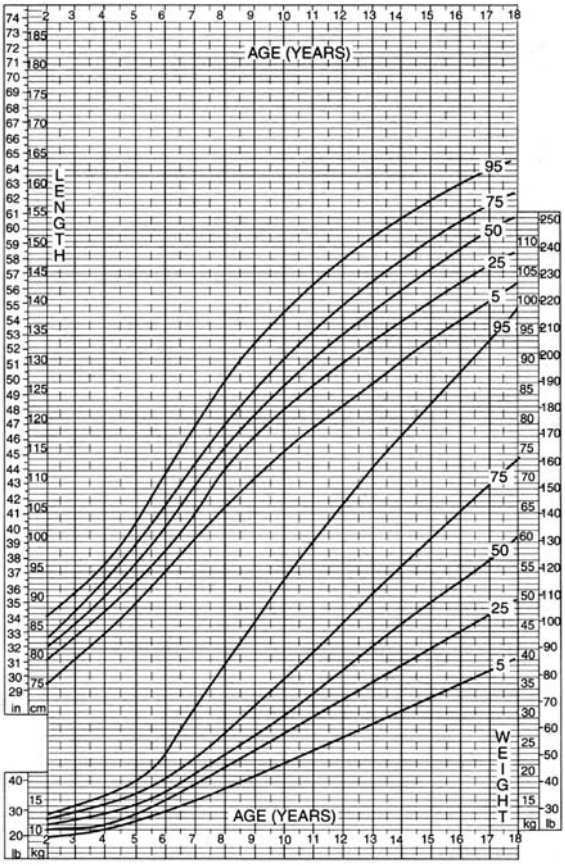


Figure 6. Physical growth of males with Down syndrome (2 to 18 years).

Figure 7. Standardized weight curves (95th, 50th, and 5th percentiles) for 42 Caucasian Prader-Willi syndrome males compared to normal controls.

Figure 8. Standardized height curves (95th, 50th, and 5th percentiles) for 42 Caucasian Prader-Willi syndrome males compared to normal controls.

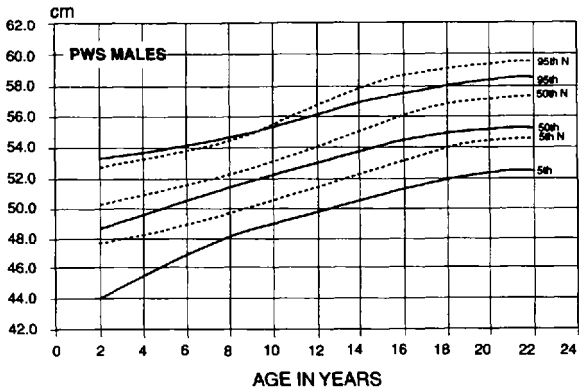


Figure 9. Standardized head circumference curves (95th, 50th, and 5th percentiles) for 42 Caucasian Prader-Willi syndrome males compared to normal controls.

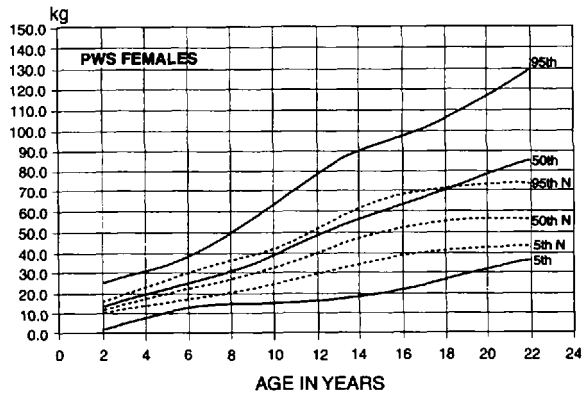


Figure 10. Standardized weight curves (95th, 50th, and 5th percentiles) for 29 Prader-Willi syndrome females compared to normal controls.

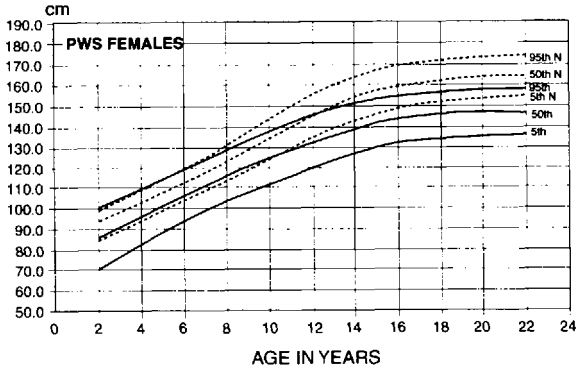


Figure 11. Standardized height curves (95th, 50th, and 5th percentiles) for 29 Prader-Willi syndrome females compared to normal controls.

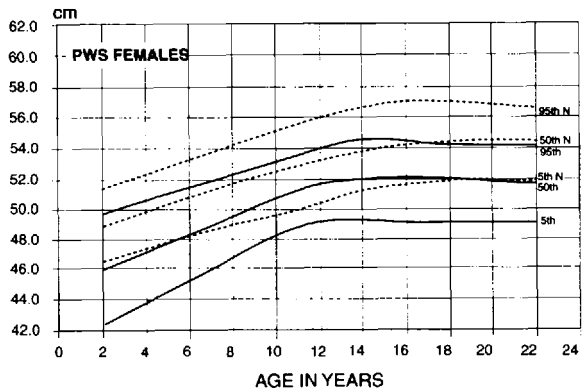


Figure 12. Standardized head circumference curves (95th, 50th, and 5th percentiles) for 29 Prader-Willi syndrome females compared to normal controls.

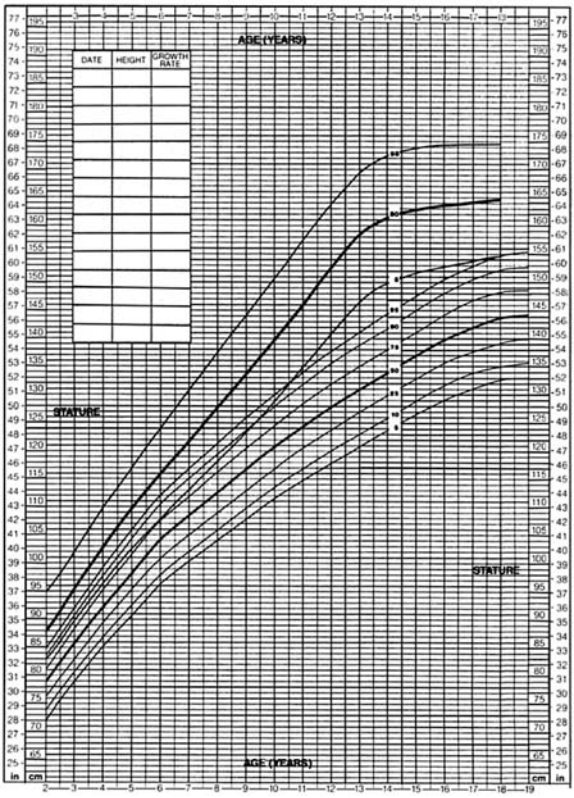


Figure 13. Turner girls 2 to 19 years physical growth. Normal girls (*top three lines*) percentiles derived from National Center for Health Statistics. Untreated Turner patients growth rates derived from Lyon AJ, Preece MA, Grant DB. Growth curve for girls with Turner syndrome. Arch Dis Child 1985;60:932–5.

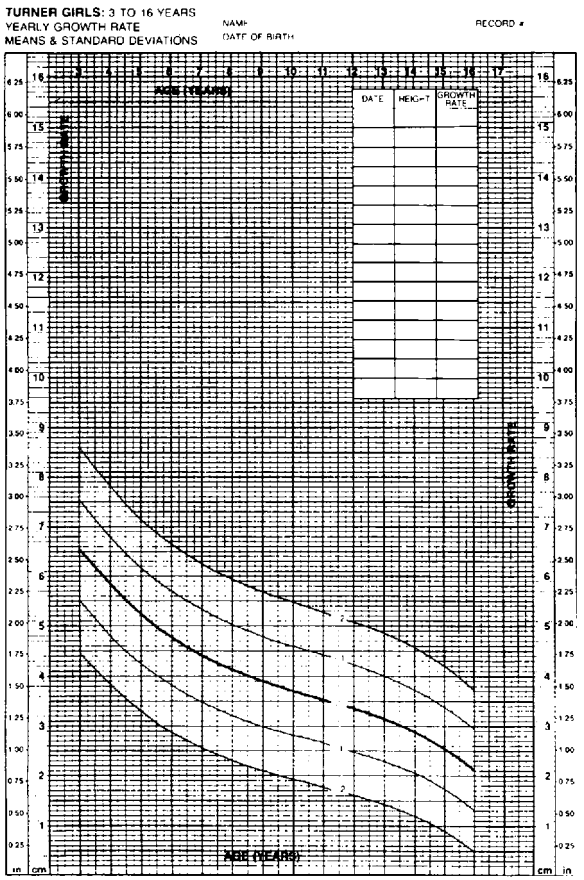


Figure 14. Turner girls 3 to 16 years yearly growth rate means and standard deviations. Turner growth rates derived from Ranke MB, et al. Turner syndrome: spontaneous growth in 150 cases and review of the literature. Eur J Pediatr 1983;141:81–8.

Index