Clinical Nutrition Management

© 2013, 2011, 2008, 2007, 2006, 2005, 2003, 2002, 2000, 1997, 1994, 1993, 1991, 1988 by Morrison, Inc (a sector of Compass Group, Inc.).

No part of this book may be reproduced, stored in any retrieval system, or transmitted in any form or by any means, including electronic, mechanical, photocopying, recording or otherwise, without written permission from Morrison, Inc.



MANUAL OF CLINICAL NUTRITION MANAGEMENT

Table of Contents

I. Normal Nutrition and Modified Diets

A.	Normal Nutrition	
	Statement on Nutritional Adequacy	A-1
	Estimated Energy Requirement (EER): Male and Females Under 30 Years of Age	A-2
	Estimated Energy Requirement (EER): Men And Women 30 Years of Age	A-2
	Estimated Calorie Requirements (Kcal): Each Gender and Age Group at Three Levels of Physical	l –
	Activity	A-3
	Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Macronutrients	A-4
	Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Vitamins	A-5
	Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Elements	A-6
	Dietary Reference Intakes (DRIs): Estimated Average Requirements	A-7
	Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels. Vitamins	A-8
	Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Elements	
	Food Fortification and Dietary Supplements	A-10
	Regular Diet – Adult	A-11
	High-Protein, High-Calorie Diet	A-13
	Nutrition Management During Pregnancy and Lactation	A-14
	Nutrition and The Older Adult	A-22
	Mechanical Soft (Dental Soft) Diet	A-28
	Pureed Diet	A-30
	Nutrition Management of Fluid Intake and Hydration	A-32
	Vegetarian Diets	A-36
	Kosher Guidelines	A-40
R	Transitional Diots	
р.	Clear Liquid Diet	R_1
	Full Liquid Diet	D-1
	Full Liquid Blenderized Diet	D-J R_A
	Nutrition Management of Dysphagia	D-+ R_6
	Nutrition Management of Dyspinagia	D 1E
	Nutrition Management in Pariatric Surgery	D-13
	Specialized Nutrition Support Therapy	D-1/
	Enteral Nutrition Support Therapy for Adulta	D-33
	Enteral Nutrition Support Therapy for Adulta	D-33
C	Madification of Could had wate and Eat	D-30
L .	Modification of Carbonyarate and Fat	
	Medical Nutrition Therapy for Diabetes Mellitus	C-1
	Medical Nutrition Therapy for Gestational Diabetes	C-15
	Dietary Management With the Exchange System	C-21
	Sugar in Moderation Diet	C-35
	Calorie-Controlled Diet for Weight Management.	C-36
	Medical Nutrition Therapy for Disorders of Lipid Metabolism	C-40
	Fat-Controlled Diet	C-55
	Medium-Chain Triglycerides (Mct)	C-57
D.	Modification of Fiber	
	Fiber-Restricted Diets	D-1
	High-Fiber Diet	D-4
	Dietary Fiber Content of Foods	D-11
	Gastrointestinal Soft Diet	D-14

	E. Pediatric Diets	
	Nutrition Management of the Full-Term Infant	E-1
	Infant Formula Comparison Chart	E-4
	Nutrition Management of the Toddler and Preschool Child	E-7
	Nutrition Management of the School-Aged Child	E-9
	Nutrition Management of the Adolescent	E-12
	Ketogenic Diet	E-14
	F. Modification of Minerals	
	Sodium-Controlled Diet	F-1
	No Added Salt Diet (4,000-Mg Sodium Diet)	
	Food Guide — 3.000-Mg Sodium Diet	
	2.000 Mg And 1.500 Mg Sodium Restricted Diet Patterns	
	Food Guide — 1.000-Mg Sodium Diet	
	Nutrition Management of Potassium Intake	
	Potassium Content of Common Foods	F-11
	Nutrition Management of Phosphorus Intake	
	Phosphorous Content of Common Foods	F-13
	Nutrition Management of Calcium Intake	F-14
	Calcium Content of Common Foods	F-15
	G. Modification of Protein	
	Protein-Controlled Diet for Acute and Refractory Henatic Encenhalonathy	G-1
	Protein-Based Exchanges	G-5
	Medical Nutrition Therapy for Chronic Kidney Disease	G-7
	Meal Patterns Using Healthy Food Guide	G-27
	Simplified Renal Diet	
	H Diets for Sensitivity/Miscellaneous Intolerances	
	Cluten-Free Diet	H-1
	Food Guide – Gluten-Free Diet	H-6
	Tvramine-Restricted Diet	H-10
	Lactose-Controlled Diet	Н-12
	Nutrition Management of Food Hypersensitivities	Н-16
II.	NUTRITION ASSESSMENT/INTERVENTION	
	Body Weight Evaluation and Indicators of Nutrition-Related Problems	II-1
	Stature Determination	II-5
	Body Mass Index (BMI)	II-6
	Determining Ideal Body Weight (IBW) Based on Height to Weight: The Hamwi Method	II-7
	Standard Body Weight (SBW) Determination Based On NHANES II	11-8
	Determination of Frame Size	
	Estimation of Ideal Body Weight and Body Mass Index for Amputees	
	Estimation of Energy Expenditures	
	Estimation of Protein Requirements	II-17
	Laboratory Indices of Nutritional Status	
	Classification of Some Anemias	
	Diagnostic Criteria for Diabetes Mellitus	
	Major Nutrients: Functional Deficiencies	
	Figsical Signs of Nutritional Deficiencies	
	Herb and Medication Interactions	
III	. CLINICAL NUTRITION MANAGEMENT	
	Introduction	III-1
	Anticoagulant Therapy	III-3
	Burns	III-6
	Cancer	III-10
	Chronic Obstructive Pulmonary Disease	III-16
	Corticosteroid Therapy	III-20
	Monitoring in Diabetes Mellitus	III-21

	Diabetes Mellitus: Considerations for Exercise	III-23
	Diabetes Mellitus: Considerations for Acute Illness	
	Diabetes Mellitus: Gastrointestinal Complications	III-27
	Diabetes Mellitus: Oral Glucose-Lowering Medications and Insulin	III-29
	Diabetes Mellitus: Fat Replacers and Nutritive/Nonnutritive Sweeteners	
	Dysphagia	III-35
	Relationship of Dysphagia to the Normal Swallow	III-37
	Enteral Nutrition: Management of Complications	
	Gastroesophageal Reflux Disease (GERD)	
	Heart Failure	
	HIV Infection and AIDS	III-46
	Hypertension	
	Hypertriglyceridemia	III-70
	Hypoglycemia	
	Inborn Errors of Metabolism	
	Iron Deficiency Anemia	
	Nephrotic Syndrome	III-79
	Obesity and Weight Management	
	Pancreatitis	
	Parenteral Nutrition (PN): Metabolic Complications	
	Calculating Total Parenteral Nutrition	
	Peptic Ulcer	III-103
	Pneumonia	III-104
	Pressure Ulcers	III-106
	Management of Acute Kidney Injury and Chronic Kidney Disease (Stage V)	III-110
	Nutrition Care Outcomes and Interventions In CKD (Stage V) Renal Replacem	ent Therapy III-116
	Wilson's Disease	III-119
IV. A	APPENDIX	
	Caffeine and Theobromine Content of Selected Foods and Beverages	IV-1
	Metric/English Conversions of Weight and Measures	IV-2
	Milligram/Milliequivalent Conversions	IV-2
	Salicylate Content of Selected Foods	IV-3

INDEX

I. NORMAL NUTRITION AND MODIFIED DIETS A. Normal Nutrition

Statement on Nutritional Adequacy	A-1
Estimated Energy Requirement (EER):Male and Females Under 30 Years of Age	A-2
Estimated Energy Requirement (EER): Men and Women 30 Years Of Age	A-2
Estimated Calorie Requirements (Kcal): Each Gender and Age Group at Three Levels of Physical Activity	A-3
Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Macronutries	ntsA-4
Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Vitamins	A-5
Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals, Elements	A-6
Dietary Reference Intakes (DRIs): Estimated Average Requirements	A-7
Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Vitamins	A-8
Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Elements	A-9
Food Fortification and Dietary Supplements	A-10
Regular Diet – Adult Food Guide For Americans (1800-2000 Calorie Pattern)	A-11 A-12
High-Protein, High-Calorie Diet	A-13
Nutrition Management During Pregnancy and Lactation Daily Food Group Guidelines Table A-1: Guidelines For Weight Gain After The First Trimester Of Pregnancy	A-14 A-14 A-14
Nutrition And The Older Adult Table A-2: Contributors To Unintended Weight Loss and Malnutrition in Older Adults	A-22 A-25
Mechanical Soft (Dental Soft) Diet Food Guide – Mechanical Soft (Dental Soft) Diet	 A-28 A-29
Pureed Diet Food Guide – Pureed Diet	 A-30 A-30
Nutrition Management Of Fluid Intake And Hydration Fluid Content Of The Regular Diet - Sample	A-32 A-33
Vegetarian Diets	A-36
Kosher Guidelines Food Guide – Kosher Diet	A-40 A-41

STATEMENT ON NUTRITIONAL ADEQUACY

The Dietary Reference Intakes (DRIs) of the Food and Nutrition Board of the Institute of Medicine, National Academy of Sciences, are used as the standard for determining the nutritional adequacy of the regular and modified diets outlined in this manual. DRIs reference values that are quantity estimates of nutrient intakes to be used for planning and assessing diets for healthy people. The DRIs consist of four reference intakes:

- Recommended Daily Allowances (RDA), a reference to be used as a goal for the individual.
- Tolerable Upper Intake Level (UL), the intake level given to assist in advising individuals of what intake levels may result in adverse effects if habitually exceeded.
- Estimated Average Requirement (EAR), the intake level which data indicates that the needs for 50% of individuals consuming this intake will not be met.
- Adequate Intake (AI), a recommended intake value for a group or groups of healthy people based on fewer data and substantially more judgment than used in establishing an EAR and subsequently the RDA.

An AI is given when the RDA cannot be set. Both of these reference intakes are to be used as goals in planning and assessing diets for healthy individuals (1,2). The DRIs do not cover special needs for nutrients due to various disease conditions. DRIs are reference values appropriate for both assessing population intakes and planning diets for healthy people (1,2).

When referring to energy, use Estimated Energy Intake (EER). EER is the average dietary energy intake that is predicted to maintain energy balance in a healthy adult of a defined age, gender, weight, height and level of physical activity, consistent with good health. For children, pregnant and lactating women, the EER includes the needs associated with deposition of tissues or the secretion of milk at rates consistent with good health (3).

The sample menus throughout this manual have been planned to provide the recommended DRIs for men, 31 to 50 years of age, unless indicated otherwise, and have been analyzed by a nutrient analysis software program. For specific values, refer to the following tables of recommended DRIs from the Food and Nutrition Board of the National Academy of Sciences. However, it is acknowledged that nutrient requirements vary widely. The dietitian can establish an adequate intake on an individual basis.

Nutrient analysis of the menus is available from Webtrition and reflects available nutrient information. Webtrition pulls nutrient information from either the USDA Standard Reference database (which includes 36 of the 41 RDA/DRI nutrients) or the manufactures information (manufactures are required only to provide 13 of the 41 RDA/DRI nutrients). Because of this, nutritional analysis data may be incomplete for some foods and/or some nutrients that are listed in the DRI. The Menu Nutrient Analysis Report in Webtrition uses a (+) to indicate a partial nutritional value and a (-) to indicate no nutritional value available.

The DRIs are provided in a series of reports (3-7). Full texts of reports are available at <u>www.nap.edu</u>.

References

- 1. Yates AA, Schlicker SA, Suitor CW. Dietary Reference Intakes: The new basis for recommendations for calcium and related nutrients, B vitamins, and choline. *J Am Diet Assoc.* 1998;98:699-706.
- 2. Trumbo P, Yates A, Schlicker S, Poos M. Dietary Reference Intakes: Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. J Am Diet Assoc. 2001;101(3):294-301.
- 3. Institute of Medicine's Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. (Macronutrients). Washington, DC: National Academy of Sciences, 2005: 107-180.
- 4. Institute of Medicine. *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride*. Food and Nutrition Board, Washington, DC: National Academy Press;1997.
- 5. Institute of Medicine. *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline.* Food and Nutrition Board, Washington, DC: National Academy Press;1998.
- 6. Institute of Medicine. *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Cartotenoids*. Food and Nutrition Board, Washington, DC: National Academy Press;2000.
- 7. Institute of Medicine. *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Molybdenum, Nickel, Silicon, Vandium and Zinc.* Food and Nutrition Board. Washington, DC: National Academy Press; 2001.

ESTIMATED ENERGY REQUIREMENT (EER) FOR MALE AND FEMALES UNDER 30 YEARS OF AGE

		Body Mass	Median Reference	Reference	
Age Se		Index (kg/m²) ^a	Height ^b cm(in)	Weight ^a kg (lb)	Kcal/day
2-6 mo	М		62(24)	6(13)	570
	F		62(24)	6(13)	520
7-12 mo	М		71(28)	9(20)	743
	F		71(28)	9(20)	676
1-3 y	М		86(34)	12(27)	1046
	F		86(34)	12(27)	992
4-8 y	М		115(45)	20(44)	1,742
	F		115(45)	20(44)	1,642
9-13 y	М	17.2	144(57)	36(79)	2,279
	F	17.4	144(57)	37(81)	2,071
14-18 y	М	20.5	174(68)	61(134)	3,152
	F	20.4	163(64)	54(119)	2,368
19-30 y	М	22.5	177(70)	70(154)	3,607°
-	F	21.5	163(64)	57(126)	2,403°

^aTaken from new data on male and female median body mass index and height-for-age data from the Centers for Disease Control and Prevention National Center for Health Statistics Growth Charts (Kuczmarski, et al., 2000).

^bCalculated from CDC/NCHS Growth Charts (Kuczmarski et al., 2000); median body mass index and median height for ages 4 through 19 years.

^cSubtract 10 kcal/day for males and 7 kcal/day for females for each year of age above 19 years.

Adapted from: Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington, DC: National Academies Press, 2002.

ESTIMATED ENERGY REQUIREMENT (EER) FOR MEN AND WOMEN 30 YEARS OF AGE^a

Height	PAL ^b	Weight for BMI	Weight for BMI	EER, Men	(kcal/day)º	EER, Women (kcal/day) ^c			
(m[in])		of 18.5 kg/m² (kg [lb])	of 24.99 kg/m² (kg [lb])	BMI of 18.5 kg/m ²	BMI of 24.99 kg/m ²	BMI of 18.5 kg/m ²	BMI of 24.99 kg/m ²		
1.50	Sedentary	41.6 (92)	56.2 (124)	1,848	2,080	1,625	1,762		
(59)	Low active			2,009	2,267	1,803	1,956		
	Active			2,215	2,506	2,025	2,198		
	Very Active			2,554	2,898	2,291	2,489		
1.65	Sedentary	50.4 (111)	68.0 (150)	2,068	2,349	1,816	1,982		
(65)	Low active			2,254	2,566	2,016	2,202		
	Active			2,490	2,842	2,267	2,477		
	Very Active			2,880	3,296	2,567	2,807		
1.80	Sedentary	59.9 (132)	81.0 (178)	2,301	2,635	2,015	2,221		
(71)	Low active			2,513	2,884	2,239	2.459		
	Active			2,782	3,200	2,519	2,769		
	Very Active			3,225	3,720	2,855	3,141		

^aFor each year below 30, add 7 kcal/day for women and 10 kcal/day for men. For each year above 30, subtract 7 kcal/day for women and 10kcal/day for men.

^bPhysical activity level.

^cDerive from the following regression equations based on doubly labeled water data:

Adult man: EER=661.8-9.53xAge (y)xPAx(15.91xWt [kg]+539.6xHt[m]

Adult woman EER=354.1 - 6.91xAge(y)xPAx(9.36xWt [kg] + 726xHt [m])

Where PA refers to coefficient for Physical Activity Levels (PAL)

PAL=total energy expenditure + basal energy expenditure.

PA=1.0 if PAL \geq 1.0 < 1.4 (sedentary).

PA=1.12 if PAL ≥ 1.4<1.6 (low active).

PA=1.27 if PAL > 1.6<1.9 (active).

PA=1.45 if $PAL \ge 1.9 < 2.5$ (very active).

Source: Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (2002). This report may be accessed via www.nap.edu.

Copyright 2002 by the National Academy of Sciences. All rights reserved.

ESTIMATED CALORIE REQUIREMENTS (IN KILOCALORIES) FOR EACH GENDER AND AGE GROUP AT THREE LEVELS OF PHYSICAL ACTIVITY (1) a

Estimated amounts of calories needed to maintain energy balance for various gender and age groups at three different levels of physical activity. The estimates are rounded to the nearest 200 calories and were determined using the Institute of Medicine equation.

			Activity Level ^b	
Gender	Age (years)	Sedentary ^b	Moderately Active	Active
Child	2-3	1,000—1,200 °	1,000-1,400 ^c	1,000-1,400°
Female ^d	4-8	1,200-1,400	1,400-1,600	1,400-1,800
	9-13	1,400-1,600	1,600-2,000	1,800-2,200
	14-18	1,800	2,000	2,400
	19-30	1,800-2,000	2,000-2,200	2,400
	31-50	1,800	2,000	2,200
	51+	1,600	1,800	2,000-2,200
Male	4-8	1,200-1,400	1,400-1,600	1,600-2,000
	9-13	1,600-2,000	1,800-2,200	2,000-2,600
	14-18	2,000-2,400	2,400-2,800	2,800-3,200
	19-30	2,400-2,600	2,600-2,800	3,000
	31-50	2,200-2,400	2,400-2,600	2,800-3,000
	51+	2,000-2,200	2,200-2,400	2,400-2,800

^aBased on Estimated Energy Requirements (EER) equations, using reference heights (average) and reference weights (healthy) for each age/gender group. For children and adolescents, reference height and weight vary. For adults, the reference man is 5 feet 10 inches tall and weighs 154 pounds. The reference woman is 5 feet 4 inches tall and weighs 126 pounds. EER equations are from the Institute of Medicine. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. Washington (DC): The National Academies Press; 2002.

^bSedentary means a lifestyle that includes only the light physical activity associated with typical day-to-day life. Moderately active means a lifestyle that includes physical activity equivalent to walking about 1.5 to 3 miles per day at 3 to 4 miles per hour, in addition to the light physical activity associated with typical day-to-day life. Active means a lifestyle that includes physical activity equivalent to walking more than 3 miles per day at 3 to 4 miles per hour, in addition to the light physical activity associated with typical day-to-day life.

^cThe calorie ranges shown are to accommodate needs of different ages within the group. For children and adolescents, more calories are needed at older ages. For adults, fewer calories are needed at older ages.

dEstimates for females do not include women who are pregnant or breastfeeding.

Reference

Dietary Guidelines for Americans 2010. Available at:

http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf Accessed Jan 31, 2011.

Food and Nutrition Board, Institute of Medicine, National Academies													
	Total		Total		Linoleic	α-Linolenic							
Life Stage Group	Water ^a	Carbohydrate	Fiber	Fat	Acid	Acid	Protein ^b						
0	(L/d)	(g/d)	(g/d)	(g/d)	(g/d)	(g/d)	(g/d)						
Infants													
0–6 mo	0.7*	60*	ND	31*	4.4*	0.5*	9.1*						
7–12 mo	0.8*	95*	ND	30*	4.6*	0.5*	11.0 ^c						
Children													
1-3 у	1.3*	130	19*	ND	7*	0.7*	13						
4-8 y	1.7*	130	25*	ND	10*	0.9*	19						
Males													
9–13 y	2.4*	130	31*	ND	12*	1.2*	34						
14–18 y	3.3*	130	38*	ND	16*	1.6*	52						
19–30 y	3.7*	130	38*	ND	17*	1.6*	56						
31–50 y	3.7*	130	38*	ND	17*	1.6*	56						
51–70 y	3.7*	130	30*	ND	14*	1.6*	56						
> 70 y	3.7*	130	30*	ND	14*	1.6*	56						
Females													
9–13 y	2.1*	130	26*	ND	10*	1.0*	34						
14–18 y	2.3*	130	26*	ND	11*	1.1*	46						
19–30 y	2.7*	130	25*	ND	12*	1.1*	46						
31–50 y	2.7*	130	25*	ND	12*	1.1*	46						
51–70 y	2.7*	130	21*	ND	11*	1.1*	46						
> 70 y	2.7*	130	21*	ND	11*	1.1*	46						
Pregnancy													
14–18 y	3.0*	175	28*	ND	13*	1.4*	71						
19–30 y	3.0*	175	28*	ND	13*	1.4*	71						
31–50 y	3.0*	175	28*	ND	13*	1.4*	71						
Lactation													
14–18 y	3.8*	210	29*	ND	13*	1.3*	71						
19–30 y	3.8*	210	29*	ND	13*	1.3*	71						
31–50 y	3.8*	210	29*	ND	13*	1.3*	71						

DIETARY REFERENCE INTAKES (DRIS): RECOMMENDED INTAKES FOR INDIVIDUALS, MACRONUTRIENTS

NOTE: This table presents Recommended Dietary Allowances (RDAs) in **bold** type and Adequate Intakes (AIs) in ordinary type followed by an asterisk (*). 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 percent) individuals in a group. For healthy infants fed human milk, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover the 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 individuals covered by this intake.

^a Total water includes all water contained in food, beverages, and drinking water.

^b Based on 0.8 g/kg body weight for the reference body weight.

^c Change from 13.5 in prepublication copy due to calculation error.

Dietary Reference Intakes (DRIs): Additional Macronutrient Recommendations Food and Nutrition Board. Institute of Medicine. National Academies

Macronutrient	Recommendation
Dietary cholesterol	As low as possible while consuming a nutritionally adequate diet
Trans fatty acids	As low as possible while consuming a nutritionally adequate diet
Saturated fatty acids	As low as possible while consuming a nutritionally adequate diet
Added sugars	Limit to no more than 25% of total energy

SOURCE: Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (2002).

Food and Nutrition Board, Institute of Medicine, National Academies														
Life Stage	Vit A	Vit C	Vit D	Vit E	Vit K	Thiamin	Riboflavin	Niacin	Vit B ₆	Folate	Vit B12	Pantothenic	Biotin	Choline ^g
Group	$(\mu g/d)^a$	(mg/d)	(µg/d) ^{b,c}	(mg/d) ^d	(µg/d)	(mg/d)	(mg/d)	(mg/d) ^e	(mg/d)	(µg/d) ^f	(µg/d)	Acid (mg/d)	(µg/d)	(mg/d)
Infants														
0-6 mo	400*	40*	15*	4*	2.0*	0.2*	0.3*	2*	0.1*	65*	0.4*	1.7*	5*	125*
7–12 mo	500*	50*	15*	5*	2.5*	0.3*	0.4*	4*	0.3*	80*	0.5*	1.8*	6*	150*
Children														
1-3 y	300	15	15*	6	30*	0.5	0.5	6	0.5	150	0.9	2*	8*	200*
4-8 y	400	25	15*	7	55*	0.6	0.6	8	0.6	200	1.2	3*	12*	250*
Males														
9–13 v	600	45	15*	11	60*	0.9	0.9	12	1.0	300	1.8	4*	20*	375*
14–18 y	900	75	15*	15	75*	1.2	1.3	16	1.3	400	2.4	5*	25*	550*
19–30 y	900	90	15*	15	120*	1.2	1.3	16	1.3	400	2.4	5*	30*	550*
31–50 y	900	90	15*	15	120*	1.2	1.3	16	1.3	400	2.4	5*	30*	550*
51–70 y	900	90	15*	15	120*	1.2	1.3	16	1.7	400	2.4^{i}	5*	30*	550*
> 70 y	900	90	20*	15	120*	1.2	1.3	16	1.7	400	2.4^{i}	5*	30*	550*
Females														
9–13 y	600	45	15*	11	60*	0.9	0.9	12	1.0	300	1.8	4*	20*	375*
14–18 y	700	65	15*	15	75*	1.0	1.0	14	1.2	400 ^{<i>i</i>}	2.4	5*	25*	400*
19-30 y	700	75	15*	15	90*	1.1	1.1	14	1.3	400 ^{<i>i</i>}	2.4	5*	30*	425*
31–50 y	700	75	15*	15	90*	1.1	1.1	14	1.3	400 ^{<i>i</i>}	2.4	5*	30*	425*
51-70 y	700	75	15*	15	90*	1.1	1.1	14	1.5	400	2.4^{h}	5*	30*	425*
> 70 y	700	75	20*	15	90*	1.1	1.1	14	1.5	400	2.4^{h}	5*	30*	425*
Pregnancy														
14-18 y	750	80	15*	15	75*	1.4	1.4	18	1.9	600 ^j	2.6	6*	30*	450*
19-30 y	770	85	15*	15	90*	1.4	1.4	18	1.9	600 ^j	2.6	6*	30*	450*
31-50 y	770	85	15*	15	90*	1.4	1.4	18	1.9	600 ^j	2.6	6*	30*	450*
Lactation														
14-18 y	1,200	115	15*	19	75*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*
19–30 y	1,300	120	15*	19	90*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*
31-50	1,300	120	15*	19	90*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*

Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Vitamins

NOTE: This table (taken from the DRI reports, see www.nap.edu) presents Recommended Dietary Allowances (RDAs) in **bold type** and Adequate Intakes (AIs) in ordinary type followed by an asterisk (*). An RDA is the average daily dietary intake level; sufficient to meet the nutrient requirements of nearly all (97 to 98 percent) healthy individuals in a group. It is calculated from an Estimated Average Requirement (EAR). If sufficient scientific evidence is not available to establish an EAR, and thus calculate an RDA, an AI is developed. 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 healthy individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

a As retinol activity equivalents (RAEs). 1 RAE = 1 mg retinol, 12 mg b-carotene, 24 mg a-carotene, or 24 mg b-cryptoxanthin. The RAE for dietary provitamin A carotenoids is twofold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A is the same as RE.

b As cholecalciferol. 1 μ g cholecalciferol = 40 IU vitamin D.

c In the absence of adequate exposure to sunlight.

d As a-tocopherol. a-Tocopherol includes RRR-a-tocopherol, the only form of a-tocopherol that occurs naturally in foods, and the 2R-stereoisomeric forms of a-tocopherol (RRR-, RSR-, RRS-, and RSS-a-tocopherol) that occur in fortified foods and supplements. It does not include the 2S-stereoisomeric forms of a-tocopherol (SRR-, SSR-, SRS-, and SSS-a-tocopherol), also found in fortified foods and supplements.

e As niacin equivalents (NE). 1 mg of niacin = 60 mg of tryptophan; 0–6 months = preformed niacin (not NE).

f As dietary folate equivalents (DFE). 1 DFE = 1 µg food folate = 0.6 µg of folic acid from fortified food or as a supplement consumed with food = 0.5 µg of a supplement taken on an empty stomach.

g Although Als 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.

h Because 10 to 30 percent of older people may malabsorb food-bound B12, it is advisable for those older than 50 years to meet their RDA mainly by consuming foods fortified with B12 or a supplement containing B12.

i 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 µg from supplements or fortified foods in addition to intake of food folate from a varied diet.

j It is assumed that women will continue consuming 400 µg from supplements or fortified food 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.

SOURCES: Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride (1997); Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid,Biotin, and Choline (1998); Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000); Dietary Reference Intakes for Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001); Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2005); and Dietary Reference Intakes for Calcium and Vitamin D (2011). These reports may be accessed via www.nap.edu.

Life Stage	Calcium	Chromium	Copper	Fluoride	Iodine	Iron	Magnesium	Manganese	Molybdenum	Phosphorus	Selenium	Zinc	Potassium	Sodium	Chloride
Group	(mg/d)	(µg/d)	$(\mu g/d)$	(mg/d)	(µg/d)	(mg/d)	(mg/d)	(mg/d)	(µg/d)	(mg/d)	(µg/d)	(mg/d)	(g/d)	(g/d)	(g/d)
Infants			(10)										(0,)		
0-6 mo	200*	0.2*	200*	0.01*	110*	0 27*	30*	0.003*	2*	100*	15*	2*	0.4*	0 12*	0 18*
7-12 mo	260*	5.5*	220*	0.5*	130*	11	75*	0.6*	3*	275*	20*	3	0.7*	0.37*	0.57*
Children	200	010		0.0	100		70	0.0	Ū	270	20	Ū	017	0107	0.07
1 2	700*	11*	240	0.7*	00	-	00	1 0*	17	460	20	2	2.0*	1.0*	1 5*
1-3 y	1 0 0 0*	11	340	1*	90	10	80	1.2	17	400	20	3	3.0*	1.0*	1.5
4-8 y	1,000*	15"	440	1.	90	10	130	1.5	22	500	30	Э	3.8	1.2	1.9*
Males															
9–13 y	1,300*	25*	700	2*	120	8	240	1.9*	34	1,250	40	8	4.5*	1.5*	2.3*
14–18 y	1,300*	35*	890	3*	150	11	410	2.2*	43	1,250	55	11	4.7*	1.5*	2.3*
19-30 y	1,000*	35*	900	4*	150	8	400	2.3*	45	700	55	11	4.7*	1.5*	2.3*
31–50 y	1,000*	35*	900	4*	150	8	420	2.3*	45	700	55	11	4.7*	1.5*	2.3*
51–70 y	1,000*	30*	900	4*	150	8	420	2.3*	45	700	55	11	4.7*	1.3*	2.0*
> 70 y	1,200*	30*	900	4*	150	8	420	2.3*	45	700	55	11	4.7*	1.2*	1.8*
Females															
9–13 y	1,300*	21*	700	2*	120	8	240	1.6*	34	1,250	40	8	4.5*	1.5*	2.3*
14-18 y	1,300*	24*	890	3*	150	15	360	1.6*	43	1,250	55	9	4.7*	1.5*	2.3*
19-30 y	1,000*	25*	900	3*	150	18	310	1.8*	45	700	55	8	4.7*	1.5*	2.3*
31–50 y	1,000*	25*	900	3*	150	18	320	1.8*	45	700	55	8	4.7*	1.5*	2.3*
51-70 y	1,200*	20*	900	3*	150	8	320	1.8*	45	700	55	8	4.7*	1.3*	2.0*
> 70 y	1,200*	20*	900	3*	150	8	320	1.8*	45	700	55	8	4.7*	1.2*	1.8*
Pregnancy															
14–18 v	1.300*	29*	1.000	3*	220	27	400	2.0*	50	1.250	60	12	4.7*	1.5*	2.3*
19–30 v	1,000*	30*	1,000	3*	220	27	350	2.0*	50	700	60	11	4.7*	1.5*	2.3*
31–50 y	1,000*	30*	1,000	3*	220	27	360	2.0*	50	700	60	11	4.7*	1.5*	2.3*
Lactation															
14-18 v	1,300*	44*	1,300	3*	290	10	360	2.6*	50	1,250	70	13	5.1*	1.5*	2.3*
19–30 v	1,000*	45*	1,300	3*	290	9	310	2.6*	50	700	70	12	5.1*	1.5*	2.3*
31–50 y	1,000*	45*	1,300	3*	290	9	320	2.6*	50	700	70	12	5.1*	1.5*	2.3*

Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Elements Food and Nutrition Board, Institute of Medicine, National Academies

NOTE: This table (taken from the DRI reports, see www.nap.edu) presents Recommended Dietary Allowances (RDAs) in **bold type** and Adequate Intakes (AIs) in ordinary type followed by an asterisk (*). An RDA is the average daily dietary intake level; sufficient to meet the nutrient requirements of nearly all (97 to 98 percent) healthy individuals in a group. It is calculated from an Estimated Average Requirement (EAR). If sufficient scientific evidence is not available to establish an EAR, and thus calculate an RDA, an AI is developed. 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 healthy individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

SOURCES: Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride (1997); Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B₆, Folate, Vitamin B₁₂, Pantothenic Acid,Biotin, and Choline (1998); Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000); Dietary Reference Intakes for Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001); Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2005); and Dietary Reference Intakes for Calcium and Vitamin D (2011). These reports may be accessed via www.nap.edu.

Dietary Reference Intakes (DRIs): Estimated Average Requirements

					L	oou all	u Nuti		Uaru, I	IIstitut		eureme,	natio	паі ле	auenn	5					
Life Stage Group	Cal- cium (mg/d)	CHO (g/d)	Protein (g/kg/d)	Vit A (mg/d)a	Vit C (mg/d)	Vit D (µg/d)	Vit E (mg/d) b	Thia- min (mg/d)	Ribo- flavin (mg/d)	Niacin (mg/d)c	Vit B6 (mg/d)	Folate (mg/d)d	Vit B12 (mg/d)	Cop- per (mg/d)	Iodine (mg/d)	Iron (mg/d)	Magnes- ium (mg/d)	Molyb- denum (mg/d)	Phos- phorus (mg/d)	Sele- nium (mg/d)	Zinc (mg/d)
Infants																					
0 to 6 mo																					
6-12 mo			1.0													6.9					2.5
Children																					
1–3 y	500	100	0.87	210	13	10	5	0.4	0.4	5	0.4	120	0.7	260	65	3.0	65	13	380	17	2.5
4-8 y	800	100	0.76	275	22	10	6	0.5	0.5	6	0.5	160	1.0	340	65	4.1	110	17	405	23	4.0
Males																					
9–13 y	1,100	100	0.76	445	39	10	9	0.7	0.8	9	0.8	250	1.5	540	73	5.9	200	26	1,055	35	7.0
14–18 y	1,100	100	0.73	630	63	10	12	1.0	1.1	12	1.1	330	2.0	685	95	7.7	340	33	1,055	45	8.5
19–30 y	800	100	0.66	625	75	10	12	1.0	1.1	12	1.1	320	2.0	700	95	6	330	34	580	45	9.4
31–50 y	800	100	0.66	625	75	10	12	1.0	1.1	12	1.1	320	2.0	700	95	6	350	34	580	45	9.4
51-70 y	800	100	0.66	625	75	10	12	1.0	1.1	12	1.4	320	2.0	700	95	6	350	34	580	45	9.4
> 70 y	1,000	100	0.66	625	75	10	12	1.0	1.1	12	1.4	320	2.0	700	95	6	350	34	580	45	9.4
Females	1 100	100	0.54	100		10	0	0.5		0		050	4 -	= 10	50		200	0.6	4.055	05	= 0
9–13 y	1,100	100	0.76	420	39	10	9	0.7	0.8	9	0.8	250	1.5	540	73	5.7	200	26	1,055	35	7.0
14-18 y	1,100	100	0.71	485	56	10	12	0.9	0.9	11	1.0	330	2.0	685	95	7.9	300	33	1,055	45	/.3
19-30 y	800	100	0.66	500	60	10	12	0.9	0.9	11	1.1	320	2.0	700	95	8.1	255	34	580	45	6.8
31-50 y	1 000	100	0.66	500	60	10	12	0.9	0.9	11	1.1	320	2.0	700	95	8.1 F	265	34	580	45	6.8
51-70 y	1,000	100	0.66	500	60	10	12	0.9	0.9	11	1.5	220	2.0	700	95	5	205	24	500	45	0.0 6 9
Pregnancy	1,000	100	0.00	300	00	10	12	0.9	0.9	11	1.5	320	2.0	700	93	5	203	34	300	45	0.0
14_{-18} v	1 000	135	0.88	530	66	10	12	12	12	14	16	520	22	785	160	23	335	40	1.055	49	10.5
19–30 v	800	135	0.88	550	70	10	12	1.2	1.2	14	1.0	520	2.2	800	160	22	290	40	580	49	9.5
31–50 y	800	135	0.88	550	70	10	12	1.2	1.2	14	1.0	520	2.2	800	160	22	300	40	580	49	9.5
Lactation	000	100	0.000	000		10		112			110	020		000	100		000	10	000		510
14–18 v	1,000	160	1.05	885	96	10	16	1.2	1.3	13	1.7	450	2.4	985	209	7	300	35	1.055	59	10.9
19–30 v	800	160	1.05	900	100	10	16	1.2	1.3	13	1.7	450	2.4	1.00	209	6.5	255	36	580	59	10.4
														0							
31-50 у	800	160	1.05	900	100	10	16	1.2	1.3	13	1.7	450	2.4	1,00 0	209	6.5	265	36	580	59	10.4

NOTE: An Estimated Average Requirements (EAR), is the average daily nutrient intake level estimated to meet the requirements of half of the healthy individuals in a group. EARs have not been established for vitamin K, pantothenic acid, biotin, choline, chromium, fluoride, manganese, or other nutrients not yet evaluated via the DRI process.

a As retinol activity equivalents (RAEs). 1 RAE = 1 μg retinol, 12 μg b-carotene, 24 μg a-carotene, or 24 μg β-cryptoxanthin. The RAE for dietary provitamin A carotenoids is two-fold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A is the same as RE.

b As α-tocopherol. α-Tocopherol includes RRR-α-tocopherol, the only form of α-tocopherol that occurs naturally in foods, and the 2R-stereoisomeric forms of α-tocopherol (RRR-, RSR-, RRS-, and RSS-α-tocopherol) that occur in fortified foods and supplements. It does not include the 2S-stereoisomeric forms of α-tocopherol (SRR-, SSR-, SRS-, and SSS-α-tocopherol), also found in fortified foods and supplements.
c As niacin equivalents (NE). 1 mg of niacin = 60 mg of tryptophan.

dAs dietary folate equivalents (DFE). 1 DFE = 1 μg food folate = 0.6 μg of folic acid from fortified food or as a supplement consumed with food = 0.5 μg of a supplement taken on an empty stomach.

SOURCES: Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride (1997); Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid,Biotin, and Choline (1998); Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000); Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001); Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (2002/2005); and Dietary Reference Intakes for Calcium and Vitamin D (2011). These reports may be accessed via www.nap.edu.

Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Vitamins ۵c

Food and Nutrition Board, Institute of Medicine, National Ac	cade	emi	ie
--	------	-----	----

Life Stage	Vitamin A	Vitamin C	Vitamin D	Vitamin E	Vitamin K	Thiamin	Ribo-	Niacin	Vitamin B ₆	Folate	Vitamin B12	Pantothenic	Biotin	Choline	Carote-
Group	(µg/d)a	(mg/d)	(mg/d)	(mg/d) <i>b,c</i>			flavin	(mg/d) <i>c</i>	(mg/d)	(mg/d) c		Acid		(g/d)	noids d
Infants															
0-6 mo	600	NDe	25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
7-12 mo	600	ND	37.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Children															
1-3 y	600	400	62.5	200	ND	ND	ND	10	30	300	ND	ND	ND	1.0	ND
4-8 y	900	650	75	300	ND	ND	ND	15	40	400	ND	ND	ND	1.0	ND
Males															
9-13 y	1,700	1,200	100	600	ND	ND	ND	20	60	600	ND	ND	ND	2.0	ND
14-18 y	2,800	1,800	100	800	ND	ND	ND	30	80	800	ND	ND	ND	3.0	ND
19-30 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
31-50 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
51-70 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
> 70 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
Females															
9-13 y	1,700	1,200	100	600	ND	ND	ND	20	60	600	ND	ND	ND	2.0	ND
14-18 y	2,800	1,800	100	800	ND	ND	ND	30	80	800	ND	ND	ND	3.0	ND
19-30 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
31-50 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
51-70 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
> 70 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
Pregnancy															
14-18 y	2,800	1,800	100	800	ND	ND	ND	30	80	800	ND	ND	ND	3.0	ND
19-30 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
31-50 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
Lactation															
14-18 y	2,800	1,800	100	800	ND	ND	ND	30	80	800	ND	ND	ND	3.0	ND
19-30 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND
31-50 y	3,000	2,000	100	1,000	ND	ND	ND	35	100	1,000	ND	ND	ND	3.5	ND

NOTE: A Tolerable Upper Intake Level (UL) is the highest level of daily nutrient intake that is likely to pose no risk of adverse effects to almost all individuals in the general population. Unless otherwise specified, the UL represents total intake from food, water, and supplements. Due to lack of suitable data, ULs could not be established for vitamin K, thiamin, riboflavin, vitamin B12, pantothenic acid, biotin, carotenoids. In the absence of ULs, extra caution may be warranted in consuming levels above recommended intakes. Members of the general population should be advised not to routinely exceed the UL. The UL is not meant to apply to individuals who are treated with the nutrient under medical supervision or to individuals with predisposing conditions that modify their sensitivity to the nutrient.

^{*a*} As preformed vitamin A only.

b As α-tocopherol; applies to any form of supplemental α-tocopherol.

d The ULs for vitamin E, niacin, and folate apply to synthetic forms obtained from supplements, fortified foods, or a combination of the two.

*d*b-Carotene supplements are advised only to serve as a provitamin A source for individuals at risk of vitamin A deficiency.

eND = Not determinable due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food only to prevent high levels of intake.

SOURCES: Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride (1997); Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B₆, Folate, Vitamin B₁₂,

Pantothenic Acid, Biotin, and Choline (1998); Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000); and Dietary Reference Intakes for Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001); and Dietary Reference Intakes for Calcium and Vitamin D (2011). These reports may be accessed via www.nap.edu.

Dietary Referen	nce	Intakes	(DI	RIs):	: Tolei	rable	Upper	Intake	Lev	vels, Elements	5
	1	NT	D	1 т		C 1 4 1		1.4	1		

					F	ood and	Nutriti	on Board	i, institute	e or mean	cine, Nat	lonal Ac	ademies	5					
Life Stage	Arsenic ^a	Boron	Calcium	Chromium	Copper	Fluoride	Iodine	Iron	Magne- sium ^b	Manga- nese	Molyb- denum	Nickel	Phos- phorus	Selenium	Silicon ^c	Vana- dium ^d	Zinc	Sodium	Chloride
Group		(mg/d)	(mg/d)		(µg/d)	(mg/d)	(µg/d)	(mg/d)	(mg/d)	(mg/d)	(µg/d)	(mg/d)	(g/d)	(µg/d)		(mg/d) <i>d</i>	(mg/d)	(g/d)	(g/d)
Infants																			
0-6 mo	NDe	ND	1,000	ND	ND	0.7	ND	40	ND	ND	ND	ND	ND	45	ND	ND	4	ND	ND
7-12 mo	ND	ND	1,500	ND	ND	0.9	ND	40	ND	ND	ND	ND	ND	60	ND	ND	5	ND	ND
Children																			
1-3 y	ND	3	2,500	ND	1,000	1.3	200	40	65	2	300	0.2	3	90	ND	ND	7	1.5	2.3
4-8 y	ND	6	2,500	ND	3,000	2.2	300	40	110	3	600	0.3	3	150	ND	ND	12	1.9	2.9
Males																			
9-13 y	ND	11	3,000	ND	5,000	10	600	40	350	6	1,100	0.6	4	280	ND	ND	23	2.2	3.4
14-18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	4	400	ND	ND	34	2.3	3.6
19-30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
31-50 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
51-70 y	ND	20	2,000	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
>70 y	ND	20	2,000	ND	10,000	10	1,100	45	350	11	2,000	1.0	3	400	ND	1.8	40	2.3	3.6
Females																			
9-13 y	ND	11	3,000	ND	5,000	10	600	40	350	6	1,100	0.6	4	280	ND	ND	23	2.2	3.4
14-18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	4	400	ND	ND	34	2.3	3.6
19-30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
31-50 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
51-70 y	ND	20	2,000	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
>70 y	ND	20	2,000	ND	10,000	10	1,100	45	350	11	2,000	1.0	3	400	ND	1.8	40	2.3	3.6
Pregnancy																			
14–18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	3.5	400	ND	ND	34	2.3	3.6
19-30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	3.5	400	ND	ND	40	2.3	3.6
31-50 y	ND	20	2.500	ND	10,000	10	1,100	45	350	11	2,000	1.0	3.5	400	ND	ND	40	2.3	3.6
Lactation																			
14-18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	4	400	ND	ND	34	2.3	3.6
19-30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	ND	40	2.3	3.6
31-50 v	ND	20	2.500	ND	10.000	10	1.100	45	350	11	2.000	1.0	4	400	ND	ND	40	2.3	3.6

NOTE: A Tolerable Upper Intake Level (UL) is the highest level of daily nutrient intake that is likely to pose no risk of adverse effects to almost all individuals in the general population. Unless otherwise specified, the UL represents total intake from food, water, and supplements. Due to lack of suitable data, ULs could not be established for vitamin K, thiamin, riboflavin, vitamin B12, pantothenic acid, biotin, carotenoids. In the absence of ULs, extra caution may be warranted in consuming levels above recommended intakes. Members of the general population should be advised not to routinely exceed the UL. The UL is not meant to apply to individuals who are treated with the nutrient under medical supervision or to individuals with predisposing conditions that modify their sensitivity to the nutrient.

a Although the UL was not determined for arsenic, there is no justification for adding arsenic to food or supplements.

b The ULs for magnesium represent intake from a pharmacological agent only and do not include intake from food and water.

c Although silicon has not been shown to cause adverse effects in humans, there is no justification for adding silicon to supplements.

d Although vanadium in food has not been shown to cause adverse effects in humans, there is no justification for adding vanadium to food and vanadium supplements should be used with caution. The UL is based on adverse effects in laboratory animals and this data could be used to set a UL for adults but not children and adolescents.

e ND = Not determinable due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food only to prevent high levels of intakes.

SOURCES: Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride (1997); Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline (1998); Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000); Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001); Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2005); and Dietary Reference Intakes for Calcium and Vitamin D (2011). These reports may be accessed via http://www.nap.edu/.

Manual of Clinical Nutrition Management

A-9

FOOD FORTIFICATION AND DIETARY SUPPLEMENTS POSITION OF THE ACADEMY OF NUTRITION AND DIETETICS*

"It is the position of the American Dietetic Association (ADA)* that the best nutritional strategy for promoting optimal health and reducing the risk of chronic disease is to wisely choose a wide variety of foods. Additional vitamins and minerals from fortified foods and/or supplements can help some people meet their nutritional needs as specified by science-based nutrition standards such as the Dietary Reference Intakes (DRIs) (1,2)."

Recommendations regarding supplementation and the therapeutic use of vitamins and minerals for treating specific conditions may be found in the corresponding sections of this manual. The latest recommendations from the Food and Nutrition Board for the first time include recommendations that supplements or fortified foods be used to obtain desirable amounts of some nutrients, eg, folic acid and calcium, in certain population groups.

Under the Dietary Supplement Health and Education Act of 1994, manufacturers must adhere to restrictions regarding the types of claims that are allowed on product labels. Statements regarding the efficacy of specific products in the treatment or prevention of particular conditions are prohibited. A claim statement is allowed if the "statement claims a benefit related to a classical nutrient deficiency disease and discloses the prevalence of such disease in the United States, describes the role of a nutrient or dietary ingredient intended to affect the structure or function in humans, characterizes the documented mechanism by which a nutrient or dietary ingredient acts to maintain such structure or function, or describes general well-being from consumption of a nutrient or dietary ingredient (1)."

The manufacturer must specify that the claims are truthful and not misleading. The following statement must also accompany any claims, "This statement has not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease (1)". In addition, all supplements must have the identity and strength of contents listed on the label, and meet appropriate specifications for quality, purity and composition (3).

*The American Dietetic Association (ADA) is now known as The Academy of Nutrition and Dietetics (AND).

References

- 1. Position of the American Dietetic Association: Nutrient Supplementation. J Am Diet Assoc. 2009; 109:2073-2085.
- 2. Position of the American Dietetic Association: Functional foods. J Am Diet Assoc. 2009;109: 735-746.
- 3. Dietary Supplement Health and Education Act of 1994. Public Law (S.784)(1994)(codified at 42 USC 287C-11).

REGULAR DIET – ADULT

Description

The diet includes a wide variety of foods to meet nutritional requirements and individual preferences of healthy adults. It is used to promote health and reduce the risks of developing major, chronic, or nutrition-related disease.

Indications

The diet is served when specific dietary modifications are not required.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy. The diet uses the 1800 - 2,000 kilocalorie level as the standard reference level for adults. Specific calorie levels may need to be adjusted based on age, gender and physical activity.

How to Order the Diet

Order as "Regular Diet," indicating any special instructions.

Planning the Diet

The *Dietary Guidelines for Americans* and portion sizes use the USDA Food Guide and the DASH (Dietary Approaches to Stopping Hypertension) Eating Plan as the basis for planning the menu (1). The *Dietary Guidelines* are intended for all Americans, healthy and those at increased risk of chronic disease. However, modifications may be required while treating patients who are ill, as the main goal is to encourage food intake, which frequently requires "comfort foods," such as soup, sandwiches, and other foods the patient is accustomed to. With that consideration, the number of servings of foods from each food group may differ from the recommendations. However, the meal will still be planned to meet the DRIs whenever possible.

Dietary Guidelines for Americans encompasses two overarching concepts (1):

- Maintain calorie balance over time to achieve and sustain a healthy weight
- Focus on consuming nutrient-dense foods and beverages within basic food groups while controlling calorie and sodium intake

Recommended healthy eating pattern:

- Daily sodium intake to less than 2,300 mg and further reduce intake to 1,500 mg among person who are 51 and older and any age who are African American or have hypertension diabetes, or chronic kidney disease. At the same time, consume foods with more potassium, dietary fiber, calcium and vitamin D.
- Increase daily intake of fruits and vegetables, whole grains, and nonfat or low-fat milk and milk products.
- Consume less than 10 percent of calories from saturated fatty acids by replacing with monounsaturated and polyunsaturated fatty acids. Oils should replace solid fats when possible.
- Keep *trans* fat as low as possible.
- Reduce the intake of calories from solid fats and added sugars.
- Limit consumption of foods that contain refined grains, especially refined grain foods that contain solid fats, added sugars, and sodium.
- If you drink alcoholic beverages, do so in moderation, for only adults of legal age.
- Keep food safe to eat.

Food Group	Recommended Daily	Serving Size
Fruits	3 – 4 servings Consume citrus fruits, melons, berries.	Medium-size orange, apple, or banana
	and other fruits regularly	¹ / ₂ cup of chopped, cooked, or canned fruit (no sugar added) ¹ / ₂ cup of 100% fruit juice
Vegetables	5 servings	
	Dark-green leafy vegetables: 3 Orange vegetables: 2 cups/week Legumes: 3 cups/week Starchy vegetables: 3 cups/week Other vegetable: 6 ½ cups/week	1 cup of raw leafy vegetables: spinach, lettuce ½ cup of other vegetables, cooked or chopped raw ½ cup of vegetable juice
Grains	6 servings	
	Whole-grain products: 3 daily	1 slice of bread
	Other grains: 3 daily	2 large or 4 small crackers
		½ cup cooked cereal, rice, or pasta
		1 cup ready-to-eat cereal
		1 small roll or muffin ½ English muffin, bagel, hamburger bun, or large roll
Meat, Poultry,	5-5 ½ ounces day	, 2 2
Drv Beans.	Choose fish, dry beans, peas, poultry	1 ounce of cooked fish, poultry, or lean meat
Eggs. and Nuts	without skin, and lean meat	¹ / ₄ cup cooked dry beans or tofu
88-7	· · · · · · · · · · · · · · · · · · ·	1 egg
		1 Tbsp peanut butter
		½ ounce nuts or seeds
Milk, Yogurt, and	3 servings	
Cheese	Choose skim milk and nonfat yogurt	1 cup of milk or yogurt
	Choose part-skim and lowfat cheeses	1 ½ ounces of natural cheese
		(Mozzarella, Swiss, Cheddar)
		2 ounces of processed cheese (American)
Oils	5 tsp daily	
	Oils and soft margarines include vegetables oils and soft vegetable oil table spreads that are low in saturated fat and are trans-free	

FOOD GUIDE FOR AMERICANS (1800-2000 calorie pattern) (1)

SAMPLE MENU

Breakfast	Noon	Evening				
Orange Juice	Rotisserie Baked Chicken	Braised Beef and Noodles				
Oatmeal	Rice Pilaf	Seasoned Green Beans				
Scrambled Egg	Steamed Broccoli with Carrots	Sliced Tomato Salad				
Biscuit	Whole-wheat Roll	French Dressing				
Margarine	Margarine	Peach halves				
Jelly	Fruit Cup	Dinner Roll				
Lowfat Milk	Lowfat Milk	Margarine				
Coffee	Iced Tea	Lowfat Milk				

References

Dietary Guidelines for Americans 2010. Available at: http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf. Accessed Jan 31, 2011.

HIGH-PROTEIN, HIGH-CALORIE DIET

Description

Additional foods and supplements are added to meals or between meals to increase protein and energy intake.

Indications

A high-protein, high-calorie diet is served when protein and energy requirements are increased by stress, protein loss (protein losing enteropathy, nephrotic syndrome), and catabolism. This diet may be indicated in patients with:

- protein-energy malnutrition
- failure to thrive
- cancer
- burns
- cystic fibrosis
- human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS)
- chronic gastrointestinal diseases

This diet may also be indicated in preparation for surgery. An increase in energy is required to promote the efficient utilization of proteins for anabolism.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "High-Protein, High-Calorie Diet." The dietitian determines a target level of protein and energy to meet individual needs based on guidelines as stated in Section II: Estimation of Protein Requirements.

Planning the Diet

The diet is planned as a Regular Diet with addition of between-meal supplements that increase energy intake by at least 500 kcal and protein intake by 25 g for adults. Examples of high-protein, high-energy supplements are milk shakes, eggnogs, puddings, custards, and commercial supplements.

For children, the diet generally should provide 120% to 150% of the Dietary Reference Intakes (DRIs) for energy and protein. The actual amounts of energy and protein provided will depend on the child's or adolescent's age, height, weight, medical status, and nutrition goals.

NUTRITION MANAGEMENT DURING PREGNANCY AND LACTATION

Description

Diets for pregnant or lactating women include additional servings of food from the Regular Diet to meet the increased requirement for nutrients during pregnancy and lactation.

Nutritional Adequacy

The food patterns will meet the Dietary Reference Intakes (DRIs) for pregnancy and lactation, as outlined in Section IA: Statement on Nutritional Adequacy, except for the iron requirements in the second and third trimesters of pregnancy. Factors that may increase nutritional requirements above the estimated demands of pregnancy include: poor nutritional status; young maternal age; multiple pregnancy; closely spaced births; breast-feeding during pregnancy; continued high level of physical activity; certain disease states; and the use of cigarettes, alcohol, and legal or illegal drugs. Dietary intake of iron, folate, zinc, protein, and calcium should be carefully assessed for adequacy (1). Supplementation is justified when evidence suggests that the inadequate intake of specific nutrients can increase the risk of an adverse effect on the mother, fetus, or pregnancy outcome. Vegetarians who exclude all animal products need 2 mg of vitamin B₁₂ daily (1). Also see Section IA: Vegetarian Diets.

How to Order the Diet

Order as "Regular Diet – Pregnancy" or "Regular Diet – Lactation." Any special instructions should be indicated in the diet order.

Planning the Diet Daily Food Group Guidelines (2)

	<u>No. of S</u>	Servings
Food Group	Pregnant Women	Lactating Women
Grains, Breads, and Cereals	nine	six to eleven
Fruits	three	two to four
Vegetables	four	three to five
Low-fat Meat, Poultry, Fish, and Eggs	two or more (6 oz)	two or more (7-8 oz)
Low-fat Milk, Yogurt, Cheese	three or four	four or five
Fats, Oils, and Sweets	As needed to	provide energy

Specific Nutrient Requirements During Pregnancy

Weight gain: The National Academy of Sciences' Food and Nutrition Board has stated that the optimal weight gain during pregnancy depends on the mother's weight at the beginning of pregnancy (1). The target range for weight gain is associated with a full-term, healthy baby, weighing an average of 3.1 to 3.6 kg (6.8 to 7.9 lb) (3). The optimum weight gain for a woman of normal prepregnancy weight for her height (body mass index (BMI), 19.8 to 26 kg/m²) who is carrying a single fetus is 25 to 35 lbs; however, there are individual differences based on maternal anthropometry and ethnic decent (3). The pattern of weight gain is more significant than the absolute weight gain. The desired pattern of weight gain is approximately 3 to 8 lb in the first trimester and about 1 lb/week during the last two trimesters.

The BMI, defined as weight divided by the height squared (kg/m^2) (2), is a better indicator of maternal nutritional status than is weight alone. Recommendations for weight gain during pregnancy should be individualized according to the prepregnancy BMI (1,3). (See Section II: Body Mass Index.) To identify the BMI categories and appropriate weight gain, use Table A-1 (1).

Table A-1: Guidelines for Weight Gain After the First Trimester of Pregnancy (1,3,4)

BMI (kg/m ²)	Recommended Weight Gain	Intervention Suggested	Overall Weight Gain
<18.5	1 lb/wk	<2 lb/mo	28-40 lb
(underweight)			
18.5-24.9	1 lb/wk	<2 lb/mo, >6.5 lb/mo	25-35 lb
(normal weight)			
>25-29.9	0.6 lb/wk	>3.5 lb/wk, <1 lb/wk	15-25 lb
(overweight)			
>30.0	Individualized	<l lb="" wk,="">2.5 lb/wk</l>	11-20 lb
(obese)			

Table A-1: Guidelines for Weight Gain After the First Trimester of Pregnancy (1,3)	3,4)
--	------

BMI (kg/m ²)	Recommended Weight Gain	Intervention Suggested	Overall Weight Gain
Twin pregnancy	Individualized	Individualized	
Normal BMI			37-54 lb
Overweight			31-50 lb
Obese			25-42 lb
Triplet pregnancy	Individualized	Individualized	45-55 lb (4)

The updated Institue of Medicine (IOM) guidelines are based on the World Health Organization (WHO) BMI categories and formulated as a range of weight gain for each category of prepregnancy BMI (1). The recommended weight gain ranges for short women and for racial or ethnic groups are the same as those for the whole population (1). In addition, teenagers who are pregnant should use the adult BMI categories to determine their weight gain range until more research is done to determine whether special categories are needed for them (1). Women who are pregnant with twins are given provisional guidelines (1). Those in the normal weight range should aim to gain 37 to 54 lbs; overweight, 31 to 50 lbs; and obese, 25 to 42 lbs (1). For women pregnant with triplets, a weight gain of 45-55 lbs has been suggested (4). The newer guidelines include a specific and relatively narrow range of recommended gain for obese women (1). For many women, this will mean gaining less weight, which may be particularly challenging for women who are overweight or obese at conception (1).

Energy: The total energy needs during pregnancy range between 2,200 and 2,900 kcal/day for most women (3,5). However, the mother's age, prepregnancy BMI, rate of weight gain, and physiologic appetite must be considered when determining individual needs (3). Based on a review of evidence, an average additional intake of approximately 340 to 452 kcal/day is suggested in the second and third trimesters (6). For normal weight and overweight women in developed countries, the additional energy need may actually be less than 300 kcal/day, especially in sedentary women (3). Appropriate weight gain and appetite are better indicators of energy sufficiency than the amount of energy consumed (3). It has been suggested that an additional 500 kcal/day for a twin pregnancy_is added to the calculated needs. There is no absolute recommendation in the literature for the amount of additional energy needed for a multiple pregnancy. The indication is to add 500 kcal/day in the first trimester as soon as the multiple pregnancy is diagnosed, because these pregnancies usually do not go to term and the goal is to maximize the early weight gain (4). Pregnant women should consume a variety of foods according to the *Dietary Guidelines* to meet nutrient needs and gain the recommended amounts of weight (3). MyPyramid guidelines include *MyPyramid for Moms*, which contains food plans for pregnant women (3).

Protein: The 2002 DRIs list the recommended daily allowances for protein for all age groups during pregnancy and lactation to be 1.1 g/kg per day or an additional 25 g/day in addition to the 0.8 g/kg per day for a nonpregnant state (6). On average, this recommendation equates to approximately 71 g, but for women with greater energy needs, the protein needs may need to be adjusted. For a twin pregnancy, an additional 50 g/day of protein above the recommended daily allowance of 0.8 g/kg per day for a nonpregnant state is suggested during the second and third trimesters (6). Protein utilization depends on energy intake. Therefore, adequate energy intake is important so that protein may be spared.

Vitamins and minerals: A multivitamin and mineral supplement is recommended in several circumstances (1,3). Pregnant women who smoke or abuse alcohol or drugs should take a multivitamin and mineral supplement (3). For women infected with the human immunodeficiency virus, especially women who receive antiretroviral treatment, a supplement containing B-complex, vitamin E, and vitamin C may slow the progression of disease and reduce complications (3). A multivitamin and mineral supplement is also recommended for women with iron deficiency anemia or poor-quality diets and women who consume animal products rarely or not at all (3). B₁₂ supplementation is recommended for persons who follow a vegetarian diet pattern, including the lacto-ovo vegetarian diet pattern (3). Women carrying two or more fetuses are also advised to consume a multivitamin and mineral supplement (3). Additional nutrients that may need to be supplemented include folic acid, iron, zinc, copper, calcium, and vitamin D. The Food and Nutrition Board recommends 400 μ g/day of synthetic folic acid from fortified foods, supplements, or both for women who are trying to become pregnant and 600 μ g/day for women who are pregnant (7).

Iron: To meet the DRI of 27 mg/day of ferrous iron during pregnancy, a low-dose supplement is recommended at the first prenatal visit (1,3,6). An iron supplement containing 150 mg of ferrous sulfate, 300

mg of ferrous gluconate, or 100 mg of ferrous fumarate can fulfill this additional need. Iron deficiency anemia is the most common anemia during pregnancy. If the maternal iron stores are low, 60 to 120 mg of iron may be recommended (4), in addition to a multivitamin supplement containing 15 mg of zinc and 2 mg of copper, since iron may interfere with the absorption of zinc and copper (3). If the laboratory values indicate macrocytic anemia, vitamin B_{12} and folate levels should be assessed.

Zinc and copper: Iron can interfere with the absorption of other minerals. Therefore, women who take daily supplements with more than 30 mg of iron should add 15 mg of zinc and 2 mg of copper (3). These amounts of zinc and copper are routinely found in prenatal vitamins.

Folate: The DRI for folate for women 19 to 50 years of age is 600 μ g/day (7.8). This level of folate should be consumed through synthetic folic acid from fortified foods or supplements or both, in addition to the intake of folate from a varied diet (3,6). Compared to naturally occurring folate found in foods, the folic acid contained in fortified foods and supplements is almost twice as well absorbed, so that 1 μ g from these sources is equivalent to $1.7 \mu g$ of dietary folate (3). Women who take folic acid at the time of conception are less likely to give birth to a child with neural tube defects (9-12). To ensure that blood vitamin levels are adequate at the time of neural tube closure, supplementation should begin at least 1 month before conception (3). Women who take multivitamins containing folic acid 1 to 2 months before conception have a reduced risk of having a child with orofacial clefts (13). Research also indicates that abnormal folate metabolism may play a role in Down syndrome and other birth defects (3). Women who have delivered an infant with neural tube defects may need to consume more than the recommended amount of dietary folate equivalents (3). Until more evidence is available, it is recommended that women older than 19 years of age not exceed the tolerable upper limit of 1,000 μ g/day of folate from foods, fortified foods, and supplements (3). Although extensive public education about the importance of folic acid has occurred in the past decade, the percentage of women who take folic acid remains low at approximately 33% (3). Dietitians should provide nutrition education and counseling as to the importance of folic acid consumption, especially for women who are nonwhite, Hispanic, low-income, or young or who lack a high school education (3).

Calcium: Due to the increased efficiency of calcium absorption during pregnancy, calcium requirements for pregnant women are similar to the requirements for women who are not pregnant. A daily intake of 1,000 mg is recommended for pregnant and lactating women (13) older than 19 years (<19 years old, 1,300 mg/day) (13). Women who avoid dairy products and rely on calcium-fortified orange juice or other fortified foods may have lower intakes of vitamin D and magnesium than milk consumers, therefore their diets should be evaluated for the adequacy of these nutrients (3).

Sodium: Sodium is required during pregnancy for the expanding maternal tissue and fluid compartments and to provide fetal needs. Routine sodium restriction is not recommended (6).

Vitamin A: High doses of vitamin A during pregnancy have caused birth defects of the head, heart, brain, and spinal cord. The Food and Drug Administration (FDA) and the Institute of Medicine recommend that vitamin A intake be limited to the DRI of 5,000 IU during pregnancy (14,15). In addition, pregnant women should limit their intake of liver and fortified cereals. The FDA recommends that women of childbearing age choose fortified foods that contain vitamin A in the form of beta carotene rather than preformed vitamin A. A high intake of fruits and vegetables rich in beta carotene and other carotenoids is not a concern (15).

Fluids: Adequate fluid intake is extremely important. The recommended daily fluid intake for pregnant women is 8 to 10 cups or 35 to 40 mL/kg of pregravid weight (3).

Fiber: Ingestion of fiber is important to speed digestion and prevent constipation and hemorrhoids. The 2002 DRI for adequate intake of total fiber is 28 g/day for all age groups during pregnancy (3,6).

Other Substances

Alcohol: The consumption of alcohol during pregnancy may result in fetal alcohol syndrome. Even light to moderate drinking may cause neurologic abnormalities not detectable at birth. Since a safe level of alcohol consumption has not been determined, pregnant women should abstain from alcohol (3).

Caffeine: Caffeine is rapidly absorbed and crosses the placenta freely. After ingestion of 200 of mg caffeine, intervillous blood flow in the placenta is reduced by 25% (16). High levels of caffeine intake are associated with delayed conception, spontaneous miscarriage, and low birth weight, but not with birth defects (3,16). The position of the Academy of Nutrition and Dietetics is that pregnant women should avoid caffeine intakes greater than 300 mg/day (3). Some studies have found no adverse effects as a result of moderate caffeine

consumption, while other studies have found an increase in stillbirths, spontaneous abortions, and fetal malformations in pregnant women who consumed high levels of caffeine (>300 to 500 mg/day) (16,17). A more recent prospective study found that fetal growth restriction occurred at caffeine intakes greater than 100 mg/day (18). Until further evidence provides guidelines for setting a specific limit on caffeine intake, women should be educated on the risks associated with caffeine consumption and the potential need to limit caffeine based on predisposing risk factors (3,19).

Olestra: Studies of the fat substitute olestra conclude that pregnant or breast-feeding women should not consume products containing olestra. Olestra causes gastrointestinal distress and diarrhea, which may lead to the loss of the fat-soluble vitamins A, D, E, and K (20).

Nonnutritive Sweeteners: The FDA has approved seven nonnutritive sweeteners for general use: aspartame, acesulfame-K, luo han guo extract, neotame, saccharin, sucralose, and stevia. All FDA-approved nutritive and nonnutritive sweeteners approved for use by the general public, includes pregnant and lactating women. The FDA and expert communities have concluded that these sweeteners are safe, based on studies of the effects of these sweeteners on the fetus and the reproductive abilities of females and males (21). Thus, consumption of acesulfame-K, aspartame, saccharin, sucralose, stevia, luo han guo, and neotame within the acceptable daily intakes is safe during pregnancy (21). Research continues to indicate that aspartame is safe during pregnancy, although women with phenylketonuria should exercise caution with this sweetener because they need to closely monitor their intake of phenylalanine (3,21). There is limited evidence that saccharin can pass through the placenta and that it remains in fetal tissues; therefore, women should moderate their intake of this sweetener (3). In a study in 2010, an association between intakes of nonnutritive sweetened carbonated and noncarbonated soft drinks and preterm birth was found among Danish women (21). Women who consumed one or more nonnutritive sweetened soft drink per day were significantly more likely to deliver preterm (21). The association was stronger for carbonated beverages with aspartame and acesulfame-K compared to noncarbonated beverages. This finding has not been confirmed in other studies to date (21).

Herbal and alternative therapies: Very few randomized clinical trials have examined the safety and efficacy of alternative therapies during pregnancy (3). Several herbal and botanical supplements are harmful if used during pregnancy (3). The American Academy of Pediatrics recommends that pregnant women limit their consumption of herbal teas. Women who opt to consume herbal teas should limit their intake to two 8-oz servings per day and choose herbal teas in filtered tea bags (3).

Fish: Due to the high levels of mercury in certain types of fish and mercury's adverse effects on the fetus, the US Department of Health and Human Services and the US Environmental Protection Agency suggest limiting the type and amount of fish consumed during pregnancy (3). Pregnant women should avoid consuming shark, swordfish, king mackerel, or tilefish. Twelve ounces or less per week of fish and shellfish lower in mercury, such as shrimp, canned light tuna, salmon, pollock, and catfish, is safe (3). Consumption of albacore ("white") tuna should be limited to 6 oz/week, because this type of tuna contains more mercury than canned light tuna (3). If no information regarding fish caught from local water sources is available, pregnant women should limit their consumption of these fish to 6 oz/week and not consume any other fish during that week (3).

Foodborne Illness During Pregnancy

Pregnant women and their fetuses are at higher risk of developing foodborne illness (3). Pathogens such as *Listeria monocytogenes, Salmonella*, and *Toxoplasma gondii* cause foodborne illness (3). Proper food storage and preparation techniques should be reviewed to ensure safety (3). Unpasteurized foods and raw or undercooked meat, poultry, or fish should be avoided to reduce the risk of exposure to pathogens (3). Careful sanitation methods should be used, and pets should not be handled before or during food preparation (3).

Risk Factors During Pregnancy (1,3)

Women should be evaluated for factors that may put them at risk for adverse maternal and/or fetal outcomes while they are pregnant. If any of the following risks are identified, appropriate medical and nutritional monitoring should be provided throughout the pregnancy.

Risk factors at the onset of pregnancy:

- Adolescence: younger than 15 years old at time of conception or less than 3 years since the onset of menses
- Older than 35 years of age
- Three or more pregnancies within 2 years
- History of poor obstetric or fetal performance
- Low income

- Unusual dietary practices
- Smoking
- Excessive alcohol intake
- Recreational drug use^a
- Chronic systemic disease
- Obesity
- Prepregnancy BMI <18.5 kg/m² or >29.9 kg/m²
- Multiple gestation

^aRecreational drugs or over-the-counter medications or dietary supplements that have adverse effects (eg, laxatives, antacids, or herbal remedies containing teratogens)

Risk factors during pregnancy (1,3):

- Hemoglobin level <11 g/dL (first and third trimesters), <10.5 g/dL (second trimester); or hematocrit <33% (first and third trimesters), <32% (second trimester)
- Inadequate weight gain: <1 lb/month for very overweight women
 - <2 lb/month for normal or slightly overweight women
 - <4 to 8 lb/month for women with multiple gestation and underweight women
- Excessive weight gain (>6.6 lb/month after first trimester), possibly associated with fluid retention
- Ferritin level $<20 \ \mu g/dL$ (22)
- Serum folate level <3 mg/dL
- Serum albumin level <2.5 g/dL
- Total serum protein level <5.5 g/dL
- Vitamin B₁₂ level <80 pg/mL

Nausea and Vomiting of Pregnancy

Nausea and vomiting are the most common symptoms experienced in early pregnancy, with nausea affecting 70% to 80% of women (23-25). Dry, salty foods are traditionally recommended for resolving nausea or vomiting; however, these foods do not always relieve symptoms (24). Foods with the following characteristics are well tolerated: cold, warm, sour, creamy, crunchy, soft, wet, salty, and chocolaty (25). Increased olfactory senses often are a leading cause of nausea during early pregnancy; thus, strong odors and sensitive unpleasant odors should be avoided (24,25). Individualization in meal planning is necessary. Other management techniques include the following recommendations (25):

- Eat small, frequent meals and snacks.
- Eat low-fat protein foods and easily digested carbohydrate foods.
- Eat dry crackers before rising in the morning.
- Avoid spicy foods and gas-forming fruits and vegetables.
- Drink fluids between meals (milk is often not well tolerated).
- Avoid drinks that contain caffeine or alcohol.

Hyperemesis gravidarum: Hyperemesis gravidarum is a condition characterized by severe, persistent nausea and vomiting that causes dehydration, fluid and electrolyte abnormalities, acid-base disturbances, ketonuria, and weight loss (ie, a 5% decrease from pregravid weight) (26). Hyperemesis gravidarum occurs in approximately 2% to 5% of pregnant women (25). Nausea and vomiting of pregnancy and hyperemesis gravidarum begin in the first trimester, usually between weeks 6 and 12, and symptoms often peak between weeks 15 and 17. Symptoms often begin to decrease by week 20 (25). The pathogenesis of hyperemesis gravidarum is not well understood. Nausea and vomiting of pregnancy and hyperemesis gravidarum are thought to be related to increased secretion of human chorionic gonadotrophin and increased estrogen levels (25). Other potential causes that have been implicated but not proven include thyroid changes, such as hyperthyroidism, and bacterial infections, such as an underlying *Helicobacter pylori* infection (25,26). Complications of hyperemesis gravidarum include dehydration, hyponatremia, inadequate weight gain, and Mallory-Weiss tears (26). Another complication, Wernicke's encephalopathy, is a result of insufficient thiamin levels that are related to vomiting or the result of glucose administration without the addition of thiamin (25).

Treatment of hyperemesis gravidarum depends on the risk level of the patient and the severity of symptoms, such as dehydration and the inability to meet nutrition needs orally. Intensive nutrition counseling and individualized meal planning is the first line of treatment (24,25). If nutrition and behavior modification does not alleviate symptoms, medications, such as metoclopramide (Reglan) and ranitidine (Zantac), or antiemetic drugs, such as prochlorperazine (Compazine) and ondansetron (Zofran), are often prescribed (26). Patients with severe symptoms may require hydration with intravenous fluids, electrolyte replacement, or vitamin replacement with vitamin B_6 (pyridoxine) and vitamin B_1 (thiamin) (25,26). If patients do not achieve the DRIs for thiamin (1.4 mg/day) and pyridoxine (1.9 mg/day) during pregnancy, dietary supplementation should be provided (25). A small percentage of patients with hyperemesis gravidarum may require nasogastric, gastrostomy, or jejunostomy feedings or total parenteral nutrition to ensure adequate nutrition support. Only 2% to 5% of women with hyperemesis gravidarumrequire total parenteral nutrition (25). Nearly all of the literature regarding nutrition support during pregnancy is anecdotal, consisting of case studies. Treatment and intervention strategies are based on experience and patient needs. If nutrition support is indicated, treatment should be consistent with standards outlined for nonpregnant adults or in managing coexisting medical conditions or risks (eg, refeeding syndrome). Refer to Section IB: Specialized Nutrition Support and to Specific Nutrient Requirements During Pregnancy earlier in this section.

Obesity

Obesity in pregnancy not only increases risks for pregnant women during gestation, but also increases risks for the future health of the child (27). Obesity during pregnancy has been associated with gestational diabetes, gestational hypertension, pre-eclampsia, birth defects, Cesarean delivery, fetal macrosomia, perinatal deaths, postpartum anemia, and childhood obesity (27,28). More women are beginning pregnancy with high BMI's, and more are gaining weight in excess of the 1990 Institute of Medicine (IOM) recommendations for gestational weight gain (27). Overweight and obese women are more likely to maintain excess weight with each successive pregnancy. Those who gain more are more likely to retain it and continue at a higher weight throughout their lifetime, as compared to women who gain less weight during pregnancy (27, 28). Weight gain during pregnancy has also been shown to have implications for the child's future risk of being overweight (1, 27,28). It is the position of The Academy of Nutrition and Dietetics and the American Society for Nutrition that all overweight and obese women of reproductive age should receive counseling prior to pregnancy, during pregnancy, and in the interconceptional period on the roles of diet and physical activity in reproductive health (27). During pregnancy overweight and obese clients should target IOM gestational weight gain targets, be advised not to lose weight during pregnancy, and counseled about healthful eating habits (27). In addition encouragement should be given to breastfeed and be made aware of the benefits for both the mom's and her child's health (27).

Gestational Hypertension (3)

Gestational hypertension is defined as systolic blood pressure of 140 mm Hg or greater or diastolic blood pressure of 90 mm Hg or greater with onset after 20 weeks' gestation (29). About 25% of women with gestational hypertension will develop preeclampsia, which is characterized by proteinuria (>300 mg in a 24-hour urine sample). Preeclampsia occurs more often in primigravid women and in women older than 35 years with chronic hypertension or renal disease.

Gestational hypertension is associated with marked changes in renal function that may lead to excessive extracellular fluid retention. Preeclampsia accompanied by grand mal seizures is a condition called eclampsia (3,29). Preeclampsia usually occurs after the 20th week of conception. Preeclampsia is more common in women with chronic hypertension and renal disease, adolescents, underweight women with inadequate weight gain, women who are older than 35 years, obese women, women with a history of preeclampsia, and women who are carrying multiple fetuses (3).

No specific nutrition therapy has been proven to be effective in preventing or delaying preeclampsia and improving pregnancy outcomes (3,29). Adequate calcium, protein, energy, and potassium may be necessary. A meta-analysis of 17 randomized controlled trials concluded that calcium supplements (1 to 2 g/day) reduced blood pressure and the risk of preeclampsia but had no significant effect on reducing maternal and infant morbidity and mortality (30). Studies of other nutrients, such as vitamins C and E, have yielded inconclusive results. The efficacy of dietary modifications, including sodium restriction, magnesium supplements, zinc supplements, and consumption of fatty fish oils, has not been proven (3,31). Diuretics should be avoided unless strict medical supervision is provided.

Specific Nutrient Requirements During Lactation

It is the position of the Academy of Nutrition and Dietetics that exclusive breastfeeding provides optimal nutrition and health protection for the first 6 months of life and breastfeeding with complementary foods from 6 months until at least 12 months of age is the ideal feeding pattern for infants (32). Breastfeeding is associated with a reduced risk of otitis media, gastroenteritis, respiratory illness, sudden infant death syndrome, necrotizing enterocolitis, obesity, and hypertension. Breastfeeding is also associated with improved maternal outcomes, including a reduced risk of breast and ovarian cancer, type 2 diabetes, and postpartum depression (32).

Energy: The average energy costs of lactation are 500 kcal/day (6) in the first 6 months and 400 kcal/day in the second 6 months (6). Excessive restriction of energy (<1,800 kcal/day) may cause decreased milk production.

Fluids: Daily intake of adequate fluid is encouraged. Current evidence does not support that increasing or decreasing fluid intake by 25 to 50 percent impacts breast milk production (Grade II)* (32).

Alcohol: A lactating woman should avoid alcohol consumption, unless it is permitted by her physician.

Caffeine: Lactating women should limit their daily consumption of caffeine to two 5-oz cups of coffee (<200 mg) (16).

Fiber: The 2002 DRI for adequate intake of total fiber is 29 g/day for all age groups during lactation (6).

Fish: The same guidelines provided for pregnancy should be applied while breastfeeding. Refer to Other Substances in the section above.

Omega-3 Fatty Acids: Consistent results from randomized control trials have shown that omega-3-fatty acid supplementation (fish oil, cod, liver oil, or docosahexaenoic acid [DHA]- rich oil) taken by pregnant women or breastfeeding mothers can increase omega-3-fatty acid levels in both breast milk and infants' plasma phospholipids (Grade II) (32). There is a dose-response relationship between doses of DHA supplementation and breast milk DHA levels, but the saturation remains unclear (32). These positive changes in breast milk omega-3-fatty acid compositions, however, do not always show a positive affect on children's visual acuity and cognitive development at long term follow-up (Grade I) (32).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- Institute of Medicine of the National Academies. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington, DC: The National Academies Press; 2009. Available at <u>http://www.iom.edu/~/media/Files/Report%20Files/2009/Weight-Gain-During-Pregnancy-Reexamining-the-Guidelines/Report%20Brief%20-%20Weight%20Gain%20During%20Pregnancy.pdf.</u> Accessed January 23, 2013. .
- 2. *Food Guide Pyramid: A Guide to Daily Food Choices.* Washington, DC: US Dept of Agriculture, Human Nutrition Information Service. Home and Garden Bulletin No. 252.
- 3. Position of the American Dietetic Association: nutrition and lifestyle for a healthy pregnancy outcome. *J Am Diet Assoc.* 2008;108:553-561.
- 4. Brown JE, Carlson M. Nutrition and multifetal pregnancy. *J Am Diet Assoc.* 2000;100:343-348.
- 5. Oken E, Taveras EM, Kleinman KP, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. *Am J Obstet Gynecol.* 2007;196:322.e1-322.e8.
- 6. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids.* National Academy of Sciences; 2002:265-334. Preprint available at: http://www.nap.edu.books/0309085373/html/index.html. Accessed September 16, 2002.
- 7. Yates AA, Schlicker SA, Suitor CW. Dietary Reference Intakes: the new basis for recommendations for calcium and related nutrients, B vitamins, and choline. *J Am Diet Assoc.* 1998;98:699-706.
- 8. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline*. Washington, DC: National Academy Press; 1998.
- 9. MRC Research Group. Prevention of neural tube defects: results of the Medical Research Council vitamin study. *Lancet.* 1991;338:131-137.
- 10. Department of Health and Human Services, Public Health Service. Recommendations for the use of folic acid to reduce the number of cases of spina bifida and other neural tube defects. *MMWR Morb Mortal Wkly Rep.* 1992;41(RR-14):1-7.
- 11. American College of Obstetricians and Gynecologists. Folic acid for the prevention of recurrent neural tube defects. Washington, DC: American College of Obstetricians and Gynecologists; 1993 (Level III). ACOG Committee Opinion 120.
- 12. American College of Obstetricians and Gynecologists. *Nutrition and Women*. Washington, DC: American College of Obstetricians and Gynecologists; 1996. Technical Bulletin No. 229.
- 13. Institute of Medicine, Food and Nutrition Board. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and

Fluoride. Washington, DC: National Academy Press; 1997.

- 14. Food and Drug Administration. Vitamin A and birth defects [press release]. October 6, 1995.
- 15. Institute of Medicine. *Nutrition During Pregnancy*. Washington, DC: National Academy Press; 1990:19.
- 16. Higdon JV, Frei B. Coffee and health: a review of recent human research. Crit Rev Food Sci Nutr. 2006; 46:101-123.
- 17. Klebanoff MA, Levine RJ, DerSimonian R, Clemens JD, Wilkins DG. Maternal serum paraxanthine, a caffeine metabolite, and the risk of spontaneous abortion. *N Engl J Med.* 1999;341:1639-1644.
- 18. CARE Study Group. Maternal caffeine intake during pregnancy and risk of fetal growth restriction: a large prospective observational study. *BMJ*. 2008;337:a2332.
- 19. Suitor CW. Nutrition for woman in their childbearing years: a review of the literature and summary of expert recommendations. *Nutr Clin Care.* 1999;2:11-45.
- 20. Middleton SJ, Dwyer J, Peters JC. An indirect means of assessing potential nutritional effects of dietary olestra in healthy subgroups of the general population. *J Nutr.* 1997;127(suppl 8):17105-17185.
- 21. Position of the Academy of Nutrition and Dietetics: use of nutritive and nonnutritive sweeteners. *J Acad Nutr Diet*. 2012;112:739-758.
- 22. Institute of Medicine. Iron Deficiency Anemia: Recommended Guidelines for the Prevention, Detection, and Management Among US Children and Women of Childbearing Age. Washington, DC: National Academy Press; 1993.
- 23. Jewell D, Young D. Interventions for nausea and vomiting in early pregnancy. Cochrane Database Syst Rev. 2003;4:CD000145.
- 24. American College of Obstetricians and Gynecologists. *Nausea and Vomiting of Pregnancy*. Washington, DC: American College of Obstetricians and Gynecologists; 2004. Practice Bulletin No. 52.
- 25. Hyperemesis gravidarum. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; 2004. Available at: www.nutritioncaremanual.org. Accessed January 25, 2009.
- 26. Ismail SK, Kenny L. Review on hyperemesis gravidarum. Best Pract Res Clin Gastroenterol. 2007;21:755-769.
- 27. Position of the American Dietetic Association and American Society for Nutrition: Obesity, Reproduction, and Pregnancy Outcomes. J Am Diet Assoc. 2009;109:918-927.
- Viswanathan M, Siega-Riz AM, Moos MK, Deierlein A, Mumford S, Knaack J, Thieda P, Lux LJ, Lohr KN. *Outcomes of Maternal Weight Gain, Evidence Report/Technology Assessment No. 168* (Prepared by RTI International-University of North Carolina Evidence-based Practice Center under contract No. 290-02-0016.) Rockville, MD: Agency for Healthcare Research and Quality; 2008. AHRQ Pub No. 08-E-09.
- 29. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 33. Diagnosis and management of preeclampsia and eclampsia. *Obstet Gynecol.* 2002;99:159-167.
- 30. Hofmeyr GJ, Atallah AN, Duley L. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. *Cochrane Database Syst Rev.* 2006;3:CD001059.
- 31. Makrides M, Duley L, Olsen SF. Marine oil, and other prostaglandin precursor, supplementation for pregnancy uncomplicated by pre-eclampsia or intrauterine growth restriction. *Cochrane Database Syst Rev.* 2006;3:CD003402.
- 32. Position of the American Dietetic Association: Promoting and supporting breastfeeding. JAm Diet Assoc. 2009;109:1926-1942.

NUTRITION AND THE OLDER ADULT

Health, physiologic, and functional changes associated with the process of aging can influence nutritional needs and nutrient intake (1). The practice of nutrition for older adults is no longer limited to those who are frail, malnourished, and ill (1). The promotion of healthy lifestyles, early detection of diseases, immunizations, and injury prevention has proven to be effective in promoting the health and longevity of older adults (1). One in every eight people in America is an older adult, defined by the Older Americans Act (OAA) as an individual who is age 60 years and older (2). By 2030, predictions indicate that the older-thanage- 65 years population will increase to approximately 72.1 million, or 19.3% of the population (3,4). Older adults 85 years of age is the fastest growing segment of this population group (1,4,5).

Older adults display wide individual variations in aging processes and thus in nutritional needs and concerns (1). Maximizing and maintaining functional status and quality of life becomes the priority healthcare objective in this population group (1). The nutritional care goal is to provide health promotion and nutrition education to achieve this objective, as decreased metabolic needs and activity levels, chronic disease management and illness, economic challenges, loss of social support systems, and other variables impact food and nutritional intake (1,4,5).

Each older adult should be viewed as a unique individual. Diet quality and quantity play major roles in preventing, delaying onset, and managing chronic diseases associated with aging (5,6). About 87% of older adults have diabetes, hypertension, dyslipidemia, or combination of these chronic diseases (5,6). Provision of medical nutrition therapy requires the regular assessment of each older individuals nutritional status and care plan. To optimize overall health and quality of life outcomes, the least restrictive regimen possible should be tailored to each person's medical condition, needs, desires, and rights (1,4).

Meal Planning Considerations for the Older Adult

When planning the diet for older adults, the Dietary Reference Intakes (DRIs) and *Dietary Guidelines for Americans* (1,7-9) provide population-specific guidelines. The DRIs divide the adult population older than 50 years into two life-stage groups: 51 through 70 years and older than 70 years (7-9). Overall nutrient requirements are similar between these age groups with the exception of the vitamin D requirement, which increases with age. To ensure adequate consumption of vitamin B₁₂ and vitamin D, the *Dietary Guidelines for Americans* recommends consuming vitamin B₁₂ in its crystalline form, eg, fortified foods or supplements, and consuming extra vitamin D from vitamin D–fortified foods and/or supplements (9).

Food intake typically declines with age, even in healthy older adults (4). Decreases in taste, olfaction, and changes in levels of hormones that control satiety and food intake can diminish appetite and lead to lower energy and overall nutrient intake (4). Food is an essential component of quality of life; an unpalatable or restrictive diet can lead to poor food and fluid intake, resulting in undernutrition and related negative health effects (4). When planning nutrient restrictions or therapeutic diets health care practitioners must assess risk versus benefit to ensure overall adequate nutrition intake (4).

Energy and Nutrient Considerations

Total and resting energy requirements decrease progressively with age because of decreases in the basal metabolic rate and in a large part decreases in physical activity level (1). The DRIs suggest that the daily energy intake should be reduced by 10 kcal for men and 7 kcal for women for each year of age above 19 years. For a 51-year-old man, this would equate to a 320 kcal reduction from the baseline DRI (10). (Refer to Section IA: Estimated Energy Requirement for Men and Women.) Meeting the nutritional needs of the older adult is challenging because although energy needs decrease, the requirements for protein, vitamins, and minerals remain the same or increase. The average daily energy intake for persons older than 51 years of age is 2,400 kcal for men and 2,000 kcal for women (10). Nutrients consistently found to be deficient in diets of older adults include antioxidants, calcium, zinc, iron, potassium, vitamin D, E, and K (1). In addition nutrients for which the digestion, absorption, or metabolism declines with age, such as vitamin B-12 and other B vitamins are also found to be deficient in this population group (1). A large proportion of adults age \geq 51 years do not consume sufficient amounts of many nutrients from food (11). When dietary selection is limited, nutrient supplementation with low-dose multivitamin and mineral supplements can be helpful for older adults to meet recommended intake levels (1).

Energy requirements: The Academy of Nutrition and Dietetics has reviewed studies to determine the energy needs of adults older than 65 years. The energy needs of healthy adults older than 65 years, as measured by indirect calorimetry, were reported to be 18 to 22 kcal/kg per day for women and 20 to 24

kcal/kg per day for men (Grade II)* (12). The daily energy needs of older adults who are acutely or chronically ill or underweight (body mass index (BMI) <20 kg/m²) were reported to be 18 to 22 kcal/kg for women and 20 to 23 kcal/kg for men (Grade II) (12). The resting metabolic rate increases with the degree of malnutrition and underweight status and may be as high as 27 to 28 kcal/kg per day in older adults with a BMI less than 20 kg/m² (Grade II) (12). Emerging research supports a relationship between an increased number of medications and decreased energy needs, however further research is needed in this area (12). Also, further research is required to determine differences in energy needs based on race and ethnicity (12).

Emerging data from the Academy of Nutrition and Dietetics evidence library suggest that a registered dietitian should prescribe a daily energy intake of 25 to 35 kcal/kg for healthy older women and 30 to 40 kcal/kg for healthy older men for weight maintenance. Research indicates that physical activity levels ranging from 1.25 to 1.75 can be applied to the resting metabolic rate (determined via indirect calorimetry) to yield the mean total daily energy estimates for healthy older adults (Grade II) (12).

When estimating energy needs for underweight older adults, the registered dietitian should prescribe a daily energy intake of 25 to 30 kcal/kg for weight maintenance or a greater energy intake for weight gain. Research indicates that physical activity levels ranging from 1.25 to 1.5 can be applied to the resting metabolic rate (determined via indirect calorimetry) to determine the mean total daily energy estimates in older adults who are chronically or acutely ill or underweight (Grade III) (12).

Protein requirements: The 2002 DRIs recommend that the RDA for protein should be a minimum of 0.8 g/kg per day for adults of all ages (10). However, an intake of protein moderately greater than this amount may be beneficial to enhance muscle protein anabolism and reduce progressive loss of muscle mass (13). Some experts suggest that a protein intake of 1.0 to 1.6 g/kg daily is safe and adequate to meet the needs of healthy older adults (1,14,15). Experts now recommend that older adults aim to consume between 25 and 30 g of high-quality protein at each meal to achieve this higher protein goal (16). This strategy along with regular resistance exercise may help prevent protein undernutrition contributing to sarcopenia in older adults (1). Including high-quality protein source at each meal is also advocated in the US Department of Agriculture's (USDA's) MyPlate food guidance system (1,17).

B vitamins: Metabolic and physical changes that affect the status of vitamin B_6 , B_{12} , and folic acid may alter behavior and general health, whereas adequate intake of these nutrients prevents some decline in cognitive function associated with aging (1,18). An estimated 6% to 15% of older adults have vitamin B-12 deficiency and approximately 20% are estimated to have marginal status (1,19). Documented complications of B-12 deficiency include macrocytic anemia, neurologic complications affecting sensory and motor function, osteopenia, and increased vascular risk (1,18). It has been suggested that persons older than 50 years should consume foods fortified with vitamin B_{12} or take a supplement containing the crystalline form of vitamin B_{12} , as 10% to 30% of older adults have protein-bound vitamin B_{12} malabsorption (9). Dietary intake of folic acid intake should be individually assessed in the diets of older adults as folic acid intake above the tolerable upper intake may mask the diagnosis of a vitamin B-12 deficiency (1). Folic acid fortification of cereal grain products and ready-to-eat cereals now provides a significant source of folic acid in the diets of older adults, especially if supplements containing folic acid are also consumed (1).

Antioxidants: Dietary antioxidant intake is associated with lower prevalence of degenerative diseases and maintenance of physiologic function in older adults (1). Cataracts and age-related macular degeneration (AMD) are common causes of blindness in older adults. Higher intakes of phytochemicals may help to prevent or delay the development of cataracts and AMD (1,20). A systematic Cochrane review of the results of the Age-Related Eye Disease Study found a beneficial effect of beta-carotene, vitamin C, vitamin E, lutein, zeaxanthin, zinc, and copper supplementation on delaying progression of advanced AMD (1). Because other studies have not confirmed similar results, the Academy of Nutrition and Dietetics evidence reports states further research is needed given the risks of over supplementation (Grade II) (1,21). Antioxidants have also been investigated in pathogenesis of cognitive impairment and Alzheimer's disease by protecting against damage to the brain resulting from oxidative stress (1,22). The majority of studies to date have been inclusive. The Academy advises against antioxidant supplementation for older adults with diagnosis of cognitive impairment or Alzheimer's disease because it has not been shown to have a beneficial effect and some formulations have side effects and contraindications (Grade II) (1 21).

Vitamin D and Calcium: Among their numerous benefits, adequate vitamin D and calcium are best known for their crucial role in the prevention and delay of progression of osteoporosis (1). Vitamin D levels may be reduced in the elderly even with adequate exposure to sunlight. This deficiency may be exacerbated by homebound status, use of sun block, poor dietary intake, decreased capabilities to synthesize cholecalciferol in the skin, and a decreased number of gastrointestinal receptors (1,12,23). For adults aged 51 to 70 years, the DRI for vitamin D is 600 IU (15 μ g) and increases to 800 IU (20 μ g) for older individuals. The recommended intake of calcium for adults older than 50 years is 1,200 mg/day (24). Because of inadequacy of intake and absorption issues, older adults and those with dark skin are in need of oversight for adequacy of these nutrients in order to maintain bone integrity and maintain serum 25 - hydroxy - vitamin D [25(OH)D] levels at 80 nmol/L (23). Other nutrients, including protein, vitamins A and K, magnesium, and phytoestrogens, are also involved in maintaining bone health and should be evaluated for adequate intake (1). The Surgeon General's report on bone health and osteoporosis recommendations include consuming recommended amounts of calcium and vitamin D, maintaining a healthful body weight, and being physically active, along with minimizing the risk of falls (25).

Sodium: The 2010 *Dietary Guidelines* for adults \geq 51 years of age are recommended to reduce sodium in their diets to 1,500 mg daily in an effort to lower their risk of high blood pressure and associated chronic diseases such as heart disease, stroke, and kidney disease (1,9). This recommendation is also supported by The Academy of Nutrition and Dietetics and the Food and Nutrition Board of the Institute of Medicine (1). Older adults should be guided to consume a wide variety of fresh foods including fruits and vegetables, use of low sodium season alternatives and less processed foods in accordance with the DASH meal planning guidelines (1).

Nutrition Assessment Considerations for Older Adults

Weight is a vital sign that should be routinely evaluated in the older adult population. Evidence-based nutrition practice guidelines recommend a baseline weight measurement, regardless of setting, upon initial visit, admission, or readmission, followed by weekly weight measurements for older adults (12). There is strong evidence in support of an association between unintended weight loss and increased mortality (Grade II) (12). The registered dietitian should use clinical judgment in interpreting nutrition assessment data to diagnose unintended weight loss and underweight (BMI <20 kg/m² or current weight compared with usual or desired body weight) or unintended weight 5% or more in 30 days (Grade II) (12). Studies also show an association between reduced appetite and poor protein and energy intake, resulting in weight loss and poor nutritional status (Grade I) (12). Medical nutrition therapy that includes a thorough nutrition assessment of biochemical data, medical tests and procedures, client history (see Table A-2 below) and food and nutrition-related history is needed to effectively determine the nutrition diagnosis and plan for nutrition interventions (1,12).

On the other side of the spectrum, there is increasing prevalence of sarcopenic obesity in the older adult population (1). Sarcopenic obesity, the coexistence of age-related loss of skeletal mass and strength and excess body fat, puts older adults at special risk for adverse outcomes including cardiovascular disease and functional impairment (1). Excess energy intake, physical inactivity, low grade inflammation, insulin resistance, and hormonal changes associated with aging have all been implicated in the etiology (1). Sarcopenic obesity presents treatment challenges requiring the clinician to weigh the risks associated with weight maintenance versus treatment to promote weight loss to optimize health, cardiovascular risk, and functional status (1). Older adults presenting with risk factors for sarcopenic obesity should have a comprehensive nutritional assessment considering existing comorbidities, weight history, and potential adverse health effects of excess body weight (1).

Dehydration, a form of malnutrition, is a major problem for the elderly, especially persons aged <85 years and institutionalized older adults (1). Fluid intake needs are the same for the young and the old, but the elderly are prone to inadequate fluid intake. Frequently, diseases will reduce the ability to recognize thirst, create an inability to express thirst, or decrease access to fluids (1,26). Even healthy elderly persons have reduced thirst in response to fluid deprivation. Fear of incontinence and difficulty making trips to the toilet, due to arthritic pain or other immobility, may also interfere with adequate fluid consumption (1). An important part of the nutrition assessment in older adults is an assessment of hydration status based on physical signs and symptoms including dry tongue, longitudinal tongue furrows, dry mucous membranes of the nose and mouth, eyes that appear recessed in their sockets, upper body muscle weakness, speech difficulty, and confusion (Grade III) (26). The elderly should be encouraged to ingest about 2 liters of fluid per day or 30 mL/kg of body weight.

The use of quantitative methods to assess the intake of food, fluids, nutrients, and energy over several days is recommended and supported by research (Grade II) (12). A multiday intake analysis based on the percentage of food eaten and individual plate waste studies is suggested over single interviews (Grade II) (12).

Contributors to Poor Nutritional Status in Older Adults

Studies indicate older adults, particularly those 85 years and older who are institutionalized are at increased risk of malnutrition, declining nutritional status, and adverse health effects (1,4,12). These unfavorable nutrition outcomes are associated with the female gender, cognitive decline, loss of appetite, swallowing problems, low activity level, eating dependency, recent hospitalization, and admission to healthcare communities (Grade I) (12). A variety of factors may contribute to poor nutritional status as individuals age (1,12). The factors listed in Table A-2 should be considered when evaluating nutritional status and developing a care plan to prevent, delay, or correct nutritional problems. Although a cure is not possible for some conditions, ameliorative or palliative nutritional interventions are often indicated and can improve the older individual's quality of life (1).

Table A-2: Contributors to Unintended Weight Loss (12) and Malnutrition in Older Adults (1,12)

Nutritional	Psychological
Alcohol and addictive substances	Bereavement
Decreased appetite (Grade I) (1,12)	Change in body habits
Drug-nutrient interactions (prescription/over-the-counter	Confusion
drugs)	Depression (Grade II) (12)
Inappropriate food intake	Fear
Increased nutrient requirements	Withdrawal
Overly restrictive dietary prescriptions (1,4,5)	

Physical	Social
Acute or chronic disease	Fixed income or poverty
Prevalence of infection (Grade I) (12)	Ignorance
Changes in body composition	Isolation
Changes in organ system structure/function	Limited food procurement, preparation, or
Changes in sensory perception	storage capabilities
Dependence or disability	Reliance on economic assistance programs
Cognitive impairment (eg, dementia disorders) (Grade I)(12)	
Decreases in activities of daily living (Grade I)(12)	
Infirmity or immobility	
Poor dentition or poorly fitting dentures	
Swallowing problems	

Nutrition Interventions to Improve Outcomes in Older Adults (1,4,12)

- Minimize dietary restrictions to encourage greater food intake. Liberalized diets and individualized meal planning are associated with increased food and beverage intake (Grade I) (1, 4,12). Maintaining the desire to eat and the enjoyment of food minimizes the risks of weight loss and undernutrition, especially in elders in long-term care (1,12). For these people, a more liberalized nutrition intervention, rather than a therapeutic diet, may be warranted to maintain quality of life (1,4). Research has not demonstrated benefits of restricting sodium, cholesterol, fat, and carbohydrate in older adults (Grade I) (12). Refer to Section IC: Medical Nutrition Therapy for Diabetes Mellitus for specific recommendations for older adults with diabetes mellitus.
- Involve older adults in planning menus and establishing meal patterns (4). The involvement of long-term care residents in menu planning and flexibility of the meal pattern and composition may result in improved intake of food and fluid (Grade I) (12).
- Encourage dining with others and creative dining programs. Research indicates that older adults who eat in a socially stimulating common dining area have improved food intake and nutritional status (4,12).
- Older adults are more prone to chewing and swallowing problems. Many older adults experience bone loss around their teeth, resulting in tooth loss and reduced bone mass. Some older adults have poorly fitting dentures that cause chewing problems or mouth pain. Dry mouth can cause problems, especially if

the food is hard to chew or dry. As we age, the amount of saliva we produce diminishes, affecting our ability to soften food and swallow it appropriately. Modification of food and fluid consistency can help with chewing and swallowing problems (27). Collaboration with a speech-language pathologist and other healthcare professionals can ensure that older adults with dysphagia receive appropriate and individualized modified-texture diets. Older adults consuming modified-texture diets report an increased need for assistance with eating, dissatisfaction with foods, and decreased enjoyment of eating, resulting in reduced food intake and weight loss (Grade I) (12).

- Provide medical food supplements for older adults who are undernourished or at risk for malnutrition. Studies support medical food supplementation as a method to provide energy and nutrient intake, promote weight gain, and maintain or improve nutritional status or prevent undernutrition (Grade I) (12).
- Consider enteral nutrition for older adults who are undernourished or at risk of undernutrition, especially in patients with dysphagia (12). Studies support enteral nutrition as a method to provide energy and nutrient intake, promote weight gain, and maintain or improve nutritional status or prevent undernutrition (12).
- Encourage adequate intake of high-fiber foods. The 2002 DRI for adequate intake of total fiber for adults older than 50 years is 14 g of fiber per 1,000 kcal or 30 g/day for men and 21 g/day for women (28). Include foods that can be easily chewed and not cause gastrointestinal discomfort. Frail older adults and those with poor appetite and anorexia need to be evaluated carefully so that a high-fiber diet does not lead to excess satiety leading to decreased food consumption and limiting nutrient intake (1). When making recommendations regarding fiber content in the diet of an older adult, fluid intake must be appropriately assessed and guidelines for fluid intake should accompany those for dietary fiber (1).
- Ensure that all older adults who need assistance to eat receive it by collaborating with other healthcare professionals. A review of research by the Academy of Nutrition and Dietetics (formerly American Dietetic Association) has found a positive association between eating dependency and poor nutritional status, especially in older adults with dysphagia who receive modified-texture diets (12). Also, the evidence indicates an association between poor nutritional status, frailty, underweight, and/or weight loss with cognitive impairment and a decrease in the activities of daily living, including the decreased ability to eat independently (12).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Position of the Academy of Nutrition and Dietetics: Food and nutrition for older adults: promoting health and wellness. *J Acad Nutr Diet*. 2012; 112:1255-1277.
- 2. Older Americans Act. US Department of Health and Human Services Administration on Aging website. http://www.aoa.gov/AoARoot/AoA Programs/OAA/index.aspx. Accessed December 1, 2010.
- 3. Profile of older Americans. US Department of Health and Human Services, Administration on Aging web site. http://www.aoa.gov/aoaroot/aging statistics/Profile/index.aspx. Accessed May 5, 2010.
- 4. Position of the American Dietetic Association: Individualized nutrition approaches for older adults in health care communities. *J Am Diet Assoc.* 2010;110:1549-1553.
- 5. Position of the American Dietetic Association, American Society for Nutrition, and Society for Nutrition Education: Food and Nutrition programs for community-residing older adults. *J Am Diet Assoc.* 2010; 110:463-472.
- 6. Institute of Medicine Committee on Nutrition Services for Medicare Beneficiaries. *The Role of Nutrition in Maintaining Health in the Nation's Elderly: Evaluating Coverage of Nutrition Services for the Medicare Population.* Washington, DC: National Academies Press; 2000.
- 7. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride.* Washington, DC: National Academy Press; 1997.
- 8. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B*₆, Folate, Vitamin B₁₂, Pantothenic Acid, Biotin, and Choline. Washington, DC: National Academy Press; 1998.
- 9. *Dietary Guidelines for Americans 2010.* Available at:
- http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf. Accessed Jan 31, 2011.
- Institute of Medicine, Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. National Academy of Sciences, 2002: 265-334; preprint at: www.nap.edu.books/0309085373/html/index.html. Accessed September 16, 2002.
- 11. Sebastian R, Cleveland L, Goldman J, Moshfegh A. Older adults who use vitamin/mineral supplements differ from nonusers in nutrient intake adequacy and dietary attitudes. *J Am Diet Assoc.* 2007;107(8):1322-1332.
- 12. Unintended Weight Loss in Older Adults Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2009. Available at: <u>www.andevidencelibrary.com</u>. Accessed October 24, 2012.
- 13. Paddon-Jones D, Short KR, Campbell WW, et al. The role of dietary protein in sarcopenia and aging. *Am J Clin Nutr*. 2008;87(suppl): 1562s-1566s.
- 14. American Dietetic Association Standards of Practice and Standards of Professional Performance for registered dietitians (generalists, specialty, advanced) in: sports dietetics. *J Am Diet Assoc.* 2009;109(3):544-552. E30.
- 15. Houston DK, Nicklas BJ, Ding J, et al. Dietary protein intake is associated with lean body mass in older community-dwelling adults:

The Health, Aging, and Body Composition (Health ABC) Study. Am J Clin Nutr. 2008;87(1):150-155.

- 16. Paddon-Jones D, Rasmussen B. Dietary protein recommendations and the prevention of sarcopenia. *Curr Opin Clin Nutr Metabl Care*. 2009;109(9): 1582-1586.
- 17. US Department of Agriculture. ChooseMyPlate.Gov. <u>http://www.choosemyplate.gov/</u>. Accessed October 24, 2012.
- 18. Green R. Is it the time for vitamin B-12 fortification? What are the questions? Am J Clin Nutr. 2009;89(2):712s-716s.
- 19. Allen L. How common is vitamin B-12 deficiency? *Am J Clin Nutr*. 2009;89(2):712s-716s.
- 20. Rhone M, Basu A. Phytochemicals and age-related eye diseases. Nutr Rev. 2008;66(8):465-472.
- 21. Food and nutrition for older adults promoting health and wellness evidence analysis project. Academy of Nutrition and Dietetics Evidence Analysis Library website. <u>http://www.andevidencelibrary.com/topic.cfm?cat=3987</u>. Accessed October 25, 2012.
- 22. Devore EE, Kang JH, Stampfer MJ, Grodstein F. Total antioxidant capacity of diet in relation to cognitive function. *Am J Clin Nutr.* 2010;92:1157-1164.
- 23. Trumbo P, Schlicker S, Yates AA, Poos M; Food and Nutrition Board of the Institute of Medicine, The National Academies Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. *J Am Diet Assoc.* 2003;102:1621-1630.
- 24. Dietary Reference Intakes for Calcium and Vitamin D. Institute of Medicine of the national Academies. Available at: www.iom.edu/vitamind. Accessed October 25, 2012.
- 25. Bone Health and Osteoporosis. A Report of the Surgeon General. Rockville, MD: US Department of Health and Human Services Office of the Surgeon General; 2004.
- 26. *Hydration Evidence Analysis Project*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2007. Available at: <u>www.andevidencelibrary.com</u>. Accessed November 13, 2012.
- 27. Position of the American Dietetic Association: oral health and nutrition. J Am Diet Assoc. 2007;107:1418-1428.
- 28. Position of the American Dietetic Association: health implications of dietary fiber. JAm Diet Assoc. 2008;108:1716-1731.

Bibliography:

- Position of the American Dietetic Association: liberalization of the diet prescription improves quality of life for older adults in long-term care. *J Am Diet Assoc.* 2005;105:1955-1965.
- Dwyer J. Screening Older Americans' Nutritional Health: Current Practices and Future Possibilities. Washington, DC: Nutrition Screening Initiative; 1991.
- White J. Risk factors associated with poor nutritional status in older Americans. In: Nutrition Screening 1: Toward a Common View. Washington, DC: Nutrition Screening Initiative; 1991.

MECHANICAL SOFT (DENTAL SOFT) DIET

Description

The diet is a modification of the Regular Diet for the edentulous resident who has difficulty chewing or swallowing, or for the resident who has undergone temporomandibular joint (TMJ) surgery. For the greatest variety of foods, all foods that are easily masticated are included in the diet.

Indications

The Mechanical Soft Diet is indicated for the resident who has difficulty chewing or swallowing.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "Mechanical Soft Diet" or "Dental Soft Diet."

"Non-chewing Diet" or "TMJ Diet" needs to be indicated to identify this variation of the Mechanical Soft Diet.

Planning the Diet

The menu selection and the individual resident's tolerances should be considered when planning a Mechanical Soft Diet.

Breakfast	Noon	Evening
Orange Juice	Honey Glazed Chicken, Chopped	Braised Beef (chopped) and Noodles
Cream of Wheat	Buttered Potatoes	Seasoned Green Beans
Scrambled Egg	Soft Cooked Broccoli	Peach Slices
Biscuit	Orange Mousse	Dinner Roll
Margarine	Dinner Roll	Margarine
Jelly	Margarine	Soft Cookie
Milk	Frosted Banana Cake	Iced Tea
Coffee	Milk	Sugar
Sugar	Теа	
Creamer	Sugar	

SAMPLE MENU

FOOD GROUP	RECOMMENDED	AVOID
Beverages	All	None
Develages	7.111	None
Broads and Crackors	Soft broads rolls	Broads with puts thick crusts
bleaus allu Clackels	Solit Di eaus, Tolis	Dieaus with huts, thick clusis
	Plain crackers softened in soup or beverage	Dry bread, toast, or tough bread
	Pancakes, plain muffins	Breads with raisins if not tolerated
	Biscuits	Hard crackers
Cereals and Grains	Cooked cereals	Cereals with raisins or nuts
Corouis una Grunis	Dry cereals	Granola-type cereals
	Dagta needlag rice	Coorres on dry concela such as
	Pasta, nooules, nice	Coarse of ury cerears, such as
	Moist bread dressing	shredded wheat or All Bran
Vegetables and	Tender soft-cooked vegetables	Raw or cooked vegetables with
Potatoes	Vegetable juices	tough skins or seeds; fried or raw
	6 ,	vegetables: cooked corn
		(egeubles) coonca corn
Fruits and Juices	Fruit juices	Fruit with tough skin if not tolerated
· · · · · · · · · · · · · · · · · · ·	Rine hanana melon neeled neaches nears	$(e\sigma raw apple dried fruit)$
	Cooked or frozon fruit: applosauce	Fresh or raw strawborries
	Cooked of frozen fruit, appresauce	Flesh of law strawberries
	Stewed prunes; other tender stewed dried	
	iruit	
	Canned peaches, pears, apricots, pineapple,	
	fruit cocktail, citrus sections	
Meat. Meat Substitutes.	Tender meat fish or poultry	Tough fibrous meats (e.g. sausage
Fntrees	Soft cheese	casings)
Life ees	Channed or ground mosts noultry	Fried ages
	Chopped of ground meats, pould y	Vegyet with mute or econut
	Solt casseroles	rogurt with nuts of coconut
	Meat, fish, or egg salads	
	Hard cooked or scrambled eggs	
	Smooth peanut butter; liverwurst	
	Yogurt without nuts or coconut	
Fata	All august the sec to avoid	Estavith secres difficult to show
Fats	All except those to avoid	Fats with coarse, difficult-to-chew,
		or chunky additives
Souns	Most soups	Soups with tough meats or
boups	1.100 boups	vegetables
		vegetables
Desserts	Cake, tender cookies	Desserts containing nuts, coarse
	Ice cream, sherbet, gelatin, custard, pudding,	dried fruit, or tough fruit
	frozen vogurt	Deserts baked to a hard consistency
	Die cream custard numphin coft fruit	2 coor to balled to a hard consistency
	Flavorad vogurt	
	riavoreu yogurt	
Sugar and Sweets	Soft candy	Candy containing tough fruits or
	Jelly, smooth jams	nuts, hard candy
	jenj, enie eti junio	nate, nur a canay

FOOD GUIDE – MECHANICAL SOFT (DENTAL SOFT) DIET

Diet following temporomandibular joint surgery: Foods such as breads, crackers, and cookies should be broken into small pieces before eating to avoid biting down or widely opening the mouth. Foods that may not be tolerated include: toast, unground meat, snack chips, foods containing coconut, and corn.
PUREED DIET

Description

The diet is soft in texture and mechanically nonirritating. Foods prepared on the Pureed Diet follow the standards of the Morrison Healthcare Classic Puree program/Morrison Senior Dining Simply Puree program. Select foods are allowed in their natural state provided they do not require additional mastication (i.e. cottage cheese, scrambled eggs, etc.).

Indications

The Pureed Diet is used for patients who have problems chewing and swallowing and patients who have esophageal inflammation or varices.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "Pureed Diet."

Planning the Diet

FOOD GUIDE – PUREED DIET		
FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages and Milk	All smooth, as desired	Beverages with seeds, lumps, or pulp
Cereals and Grains	 All farina-type cooked cereals; oatmeal Pregelled or slurried through the entire thickness: doughnuts, pancakes, waffles, French toast, and bread Pasta, rice, and dressing that are pureed to smooth consistency Regular soft bread if resident's swallowing ability permits 	Coarse cooked cereal; dry cereals; cereals with seeds or nuts All other breads Crackers
Vegetables and Potatoes/Soups	Pureed or strained vegetables without chunks or seeds; mashed white potatoes All smooth cream soups or broth- type soups with pureed or strained ingredients	Regular cooked or raw vegetables Potato skins and chips Fried or french-fried potatoes or vegetables Regular soups with rice, corn, peas, or large chunks of meat and vegetables
Fruits and Juices	Applesauce, pureed fruits, well- mashed bananas, fruit juices	Regular canned, fresh, or frozen fruits; fruit juice with pulp
Meats, Meat Substitutes, Entrees	Pureed or strained meats, poultry, or fish Soufflés that are smooth and homogenous Cottage cheese Scrambled egg Cheese sauce	Regular or chopped meats or casseroles Cheese slices or cubes Hard cooked egg Peanut butter Sandwiches Pizza

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Desserts	Custard, pudding, ice cream, sherbet, gelatin, fruit whips Cakes, cobblers, and pies pureed to a smooth and moist consistency Soft cookies and plain cakes, such as vanilla wafers or sugar cookies, prepared in a slurry Smooth custard and pudding; plain or custard-style yogurt	Regular cake, pie, cookies Bread and rice pudding Fruited yogurt
Fats	Butter, margarine, smooth gravy, cream sauces, mayonnaise, salad dressings, cream cheese, sour cream, whipped toppings	Fats with coarse or chunky additives
Miscellaneous	Sugar, jelly, honey, syrup Ketchup, mustard, smooth sauces	Jams and preserves Coarsely ground pepper and spices

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Classic Puree Chicken	Classic Puree Beef and
Cream of Wheat	Mashed Potatoes with Gravy	Noodles
Scrambled Egg	Classic Puree Carrots	Classic Puree Green Beans
Biscuit with Slurry	Classic Puree Rosy Pears	Tomato Juice
Milk	Pudding	Classic Puree Peaches
Coffee	Теа	Milk
Sugar	Sugar	
Creamer		

NUTRITION MANAGEMENT OF FLUID INTAKE AND HYDRATION

Description

Adequate fluid intake is necessary to maintain optimum hydration or to correct a state of dehydration or overhydration. The amount of fluid required to maintain the optimum hydration level varies with the medical condition of the patient. The vast majority of healthy people adequately meet their daily hydration needs (1). When assessing total water intake, all food and beverage sources providing water should be included in estimating daily fluid intake (1). The Dietary Reference Intake for water and daily fluid requirements of older adults is based on water consumed in the diet from a variety of sources including foods and beverages such as milk, tea, coffee, juice and water (1). Recommendations for daily water intake for all age groups can be found in Dietary Reference Intakes (DRIs): Recommended Intakes For Individuals, Macronutrients on page A-4 (1). The Dietary Reference Intake (DRI) for women is approximately 2.7 liters (91 ounces daily) and 3.7 liters (125 ounces daily) for men (1).

Indications

In the healthy individual, normal sensations of thirst promote the consumption of adequate fluid and the maintenance of optimum hydration (1). However, some patients may not recognize thirst, may not be able to communicate thirst, or may not freely consume liquids. Risk factors for dehydration include any of the following:

- unconscious; semiconscious and confused state
- severe depression
- tranquilizer or sedative use
- enteral feeding
- must be fed or require assistance with feeding
- diarrhea
- poor appetite
- immobility
- diuretic use
- frequent laxative use
- perspiration (in hot weather where air conditioning is unavailable)
- dysphagia/swallowing difficulties

- increased respiratory rate
- salivation decreased by medications or radiation
- therapy
- fever
- fistulous drainage
- high output ileostomy
- vomiting
- severe burns
- polyuria (e.g., glycosuria, ketonuria)
- high renal solute load (e.g., High-Protein Diet)
- denuded body surface
- hyperpnea

While consumption of beverages containing caffeine and alcohol have been shown in some studies to have diuretic effects, available information indicates that this may be transient in nature, and that such beverages can contribute to total water intake and thus can be used in meeting recommendations for dietary intake of total fluid (1). Evidence indicates that consuming up to six mg of caffeine per kilogram of body weight per day does not impact the hydration status of healthy adults, above that of a placebo or non-caffeine-containing beverage (Grade 1)*(2).

Nutritional Adequacy

See statement pertaining to diet order.

How to Order the Diet

The patient's usual diet can be amended as follows: ______ diet, _____ ml/day, or _____ diet, restrict fluids to ______ ml/day. Order should include amount of fluid to be given by Food and Nutrition Services with meals and snacks and amount of fluid to be given by nursing (i.e., with medications or between meals).

Planning the Diet

When the dietitian calculates the intake of fluids, foods that are liquid at room temperature should be counted by millileters. Such foods include water, carbonated beverages, coffee and tea, gelatin, milk, water ices and popsicles, soups, supplements, eggnog, ice cream and sherbet, and milk shakes.

Fluid is usually ordered in the form of cubic centimeters (ml) (1 mL = 1 cc). This can be converted to cups or ounces as follows:

30 ml = 1 fl oz 120 ml = 4 fl oz or ½ cup 180 ml = 6 fl oz or ¾ cup 240 ml = 8 fl oz or 1 cup 960 ml = 32 fl oz or 1 qt

(Container Size and Menus May Vary)		
Breakfast		
Juice (4 oz)	120 ml	
Milk (8 oz)	240 ml	
Coffee (6 oz)	180 ml	
Water (8 oz)	240 ml	
Noon		
Soup (6 oz)	180 ml	
Tea (8 oz)	240 ml	
Water (8 oz)	240 ml	
Evening		
Milk (8 oz)	240 ml	
Tea (8 oz)	240 ml	
Water (8 oz) 240 ml		
TOTAL 2160 ml		

Treatment and Prevention of Fluid Deficit

An appropriate assessment is made by the physician to determine if water depletion alone (dehydration) or the more common sodium/water (volume) depletion is present. Treatment is accomplished by increasing oral intake of fluid and electrolytes as needed. Patients with more severe cases and those who are unable to take fluids by mouth are treated by appropriate intravenous fluid replacement. (Note: Internal sequestering, also known as third spacing, may create a deficit of water in some compartments, although total body water is unaltered. Replacement water requirements may be greatly increased in peritonitis, pancreatitis, enteritis, ileus, or portal vein thrombosis.)

An evaluation of fluid requirements should be made on an individual basis. Urinary specific gravity (Usg) and urinary osmolality (Uosm) are good indicators of hydration or dehydration in young, healthy and active adult males and females (Grade II) (2). Urine color (Ucol) correlates well with urinary specific gravity and urinary osmolality and can be used as an indicator of hydration status (Grade II) (2). In addition, body mass loss of over 3% is another good indicator of dehydration (Grade II) (2). In some cases, a precise intake and output record may be necessary to determine and meet fluid requirements. There are several methods to determine fluid requirements (2). Currently, no evidence exists comparing which methods are best to use when estimating fluid needs in adults (Grade V) (2). The methods most frequently cited in the literature and also selected for review by The Academy of Nutrition and Dietetics evidence-analysis library include Holliday-Segar Method, RDA Method, and Fluid Balance Method (Grade V) (2). These along with other methods are described below:

Guidelines for calculating fluid needs based on age (applies to critical care patients) :

1.	Pediatrics (3)	
	<u>Weight (kg)ª</u>	<u>Fluid Requirement (ml/kg/day)</u>
	First 10 kg	100
	11 – 20 kg	1000 + 50 ml for each kg above 10 kg
	>20 kg	1500 + 20 ml for each kg >20 kg
	^a This method referred to as Holliday-Segar	
	Method, original citation: Holliday MA,	
	Seger WE. The maintenance need for water	
	in parenteral fluid therapy. Pediatrics.	
	1957;19:823-832.	

2.	Adults (4,5)	
	<u>Weight (kg)</u>	<u>Fluid Requirement (ml/kg/day)</u>
	First 10 kg of body weight	100
	Second 10 kg of body weight	50
	Each additional kilogram	20 mL/kg(<50 years of age) 15 mL/kg(>50 years of age)

*In obese patients, actual weight for height is used

Other Suggested Methods:

- RDA Method: 1 milliliter (mL) per kilocalorie (Kcal) (Grade V) (2)
- Fluid Balance Method: Urine output + 500 milliliter (mL) per day (Grade V)(2)
- Adults between 18 55 years: 35 ml/kg per day (4,5)
- Adults between 55-75: 30 ml/kg per day (5)
- Adults age > 75 years: 25 ml/kg per day (5)
- Fluid restriction: < 25 ml/kg per day (renal and cardiac disease, fluid overload status) (5)
- Patients receiving mechanical ventilation or other source of humidified oxygen can absorb up to an additional 1000 ml of fluid daily, whereas unhumidified oxygen therapy can result in a net loss of fluid (6).
- Patients treated on air-fluidized beds set at higher temperatures are at greater risk of dehydration due to an increase in insensible water loss associated with the warmer bed temperatures. Patients who require air-fluidized beds set at a higher temperature will need additional fluids, estimated to be approximately 10 to 15 mL/kg (7.8). For beds set at temperatures (86°F), fluid loss is similar to that on a conventional bed (480 ml/m2/24 h). However, when the bed temperature is high (94°F), fluid loss may increase up to 80% (938 ml/m2/24 h) in a 70-kg person (7). (Bed temperatures are adjustable and usually set between 88° and 93°F.)

Assessment of Fluid Status

The clinical assessment of total body water (TBW) is generally inaccurate (Grade II) (2). A body mass loss of over 3% is a good indicator of dehydration (Grade II) (2). More than 10% of TBW may be lost before evidence of hypovolemia appears. The thirst mechanism is activated when the decrease in TBW reaches approximately 2%. Serial assessment of body weight is probably the most reliable parameter, especially because water makes up such a large proportion of total body weight (2). Along with serial assessment, the following physical alterations can be assessed to help determine hydration status (9).

Volume deficit

- Decreased moisture in the oral cavity
- Decreased skin and tongue turgor (elasticity); skin may remain slightly elevated after being pinched
- Flattened neck and peripheral veins in supine position
- Decreased urinary output (<30 ml/h without renal failure)
- Acute weight loss (>1 lb /day)

Volume excess

- Clinical apparent edema is usually not present until 12 15 L of fluid has accumulated
- 1 L fluid = 1 kg weight
- Pitting edema, especially in dependent parts of the body (e.g., feet, ankles, and sacrum)
- Distended peripheral and neck veins
- Symptoms of heart failure or pulmonary edema
- Central venous pressure >11 cm H₂0

Laboratory values used to evaluate fluid status include urine specific gravity, urine osmolality, serum electrolytes; serum osmolality; hematocrit; blood, urea nitrogen (BUN); and urine specific gravity. Serum sodium is the best indicator of intracellular fluid disorders. The hematocrit reflects the proportion of blood plasma to red blood cells. Fluid loss causes hemoconcentration and serum osmolality; fluid gain causes hemodilution and decreases serum osmolality. A rise in BUN level frequently reflects a fluid deficit state and a fluid deficit causes urine to be concentrated (specific gravity >1.030); a fluid excess dilutes urine (specific gravity <1.010) (6).

Aging increases the risk for dehydration based on the physical and psychological changes. The elderly oftenManual of Clinical Nutrition ManagementA-34Copyright © 2013 Compass Group, Inc.
All rights reserved.

lack the ability to recognize thirst, have aged kidneys that may have a decreased ability to concentrate urine, fear urinary incontinence and thus do not drink sufficient fluids, have acute or chronic illnesses that alter fluid and electrolyte balance (10). For a discussion of calculation of free water in tube feeding, refer to Section IB: Enteral Nutrition.

Fluid Restriction

In heart failure, ascites, end-stage renal disease, and other disorders, patients retain fluid. A fluid restriction is often useful in the management of these conditions. Refer to Section III: Clinical Nutrition Management (Heart Failure and Medical Nutrition Therapy for Chronic Kidney Disease).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Institute of Medicine. *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate*. Washington, D. C: National Academy Press; 2004.
- 2. *Hydration Evidence-Analysis Project.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2007. Available at: http://www.andevidencelibrary.com. Accessed November 13, 2012.
- 3. Pediatric Nutrition Care Manual. Updated Annually. Chicago: II: The Academy of Nutrition and Dietetics; May, 2012.
- 4. Chidester JC, Spangler AA. Fluid intake in the institutionalized elderly. J Am Diet Assoc. 1997;97:23-28.
- Malone A, Seres D, Lord L. Complications of Enteral Nutrition (see Fluid Requirements p. 230). In: Mueller CM (ed). *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, MD: The American Society for Parenteral and Enteral Nutrition; 2012.
- 6. Dolan JT. Fluid and Electrolyte Physiology and pathophysiology. In: Dolan JT, ed. *Critical Care Nursing*. Philadelphia, Pa: FA Davis Co.; 1991:434.
- 7. Ayello EA, Thomas Dr, Litchford MA. Nutrition aspects of wound healing. *Home Healthc Nurse*. 1999;17:719-729.
- 8. Breslow RA. Nutrition and air-fluidized beds: a literature review. *Adv Wound Care*. 1994;3:57-60.
- 9. Lysen L. *Quick Reference to Clinical Dietetics*. Gaithersburg, Md: Aspen Publishers; 1997.
- 10. Position of the Academy of Nutrition and Dietetics: Food and nutrition for older adults: promoting health and wellness. *J Acad Nutr Diet*. 2012; 112:1255-1277.

VEGETARIAN DIETS

Description

A vegetarian is a person who does not eat meat, including fowl or seafood, or products containing these foods (1). A wide spectrum of dietary practices is considered vegetarian (1). A vegetarian whose diet consists of foods of plant origin only is a *total vegetarian* or *vegan*. However, many vegetarians also consume eggs (*ovovegetarian*), dairy products (*lactovegetarian*), or both eggs and dairy products (*lacto-ovovegetarian*). The two most common definitions for vegetarian diets in the research are vegan diets, which are devoid of all flesh foods, and vegetarian diets, which are devoid of all flesh foods but do include eggs, dairy products, or both (Grade II)* (1). According to the Academy of Nutrition and Dietetics Evidence Analysis Library, these broad categories mask important variations within vegetarian diets. Thus, the absolute categorization of vegetarian dietary practices is difficult and may result in unclear relationships between vegetarian diets and other health factors (Grade II) (1).

Indications

Vegetarian diets are adopted for a variety of health, ecological, economical, philosophical, and ethical reasons (1). Vegetarian diets offer a number of health advantages, including lower blood cholesterol levels, lower blood pressure levels, and lower risks of hypertension, heart disease, and type 2 diabetes (1). Vegetarians tend to have a lower body mass index and lower overall cancer rates (1). Vegetarian diets tend to be lower in saturated fat and cholesterol and have higher levels of dietary fiber, magnesium, potassium, folate, antioxidants (eg, vitamins C and E), carotenoids, flavonoids, and other phytochemicals (1). These nutritional differences may explain some of the health advantages of a varied, balanced vegetarian diet (1). Many epidemiologic studies suggest a positive relationship between vegetarian lifestyles and reduced risks of several chronic degenerative diseases, such as ischemic heart disease (Grade I)* (1), coronary artery disease, hypertension, type 2 diabetes, obesity, renal disease, and some cancers (1).

Nutritional Adequacy

Vegan, lactovegetarian, ovovegetarian and lacto-ovovegetarian diets are healthful and nutritionally adequate when appropriately planned for all stages of the life cycle, including pregnancy and lactation (1). Appropriately planned vegan, lactovegetarian, ovovegetarian and lacto-ovovegetarian diets will meet the nutrient needs of infants, children, and adolescents to support and promote normal growth and development (1-3).

A vegetarian diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy. Vegans and some other vegetarians may have lower intakes of vitamins B_{12} and D, calcium, zinc, long-chain n-3 fatty acids, and occasionally iron (1). The greater production and access to fortified and enriched foods is making it easier for vegetarians to improve their intake of these key nutrients. All vegetarians should have a reliable source of vitamin B_{12} and vitamin D. If sun exposure is limited, vitamin D supplements or fortified foods should be emphasized (1-6).

Results of evidence-based analysis suggest that vegetarian diets can be nutritionally adequate in pregnancy and can lead to a positive birth outcome (7). The nutrient and energy needs of pregnant and lactating vegetarian women do not differ from those needs of nonvegetarian women with the exception of higher iron recommendations for vegetarians (1). In addition to iron, key nutrients to assess in pregnancy include vitamin B₁₂, vitamin D, and folate, whereas key nutrients in lactation include vitamin B₁₂, vitamin D, calcium, and zinc (1). Breast-fed infants whose mothers do not have an adequate intake of vitamin B₁₂ should receive a vitamin B₁₂ supplement (1,4). In addition, the zinc intake of breast-fed infants should be carefully assessed; zinc supplements or zinc-fortified foods should be used when complementary foods are introduced if the diet is low in zinc or mainly consists of foods with low zinc bioavailability (1). Because of the variability of dietary practices among vegetarians, the individual assessment of dietary intakes is necessary (1).

How to Order the Diet

Order as "Regular Diet – Vegetarian." The patient's particular dietary constraints will be considered.

Planning the Diet

A vegetarian diet can be made nutritionally adequate by careful planning and giving consideration to the following guidelines (1):

- Choose a variety of foods, including fruits, vegetables, whole grains, legumes, nuts, seeds, tofu or other soy products, and, if desired, dairy products and eggs.
- Choose whole or unrefined grain products whenever possible, instead of refined products.

- Minimize intake of foods that are highly sweetened, high in sodium, or high in fat, especially saturated fat and *trans* fatty acids.
- If animal foods such as dairy products and eggs are used, choose lower-fat dairy products and use both eggs and dairy products in moderation.
- Use a regular source of vitamin B₁₂, and, if sunlight exposure is limited, provide a source of vitamin D.

In addition to these guidelines, the DRIs are a valuable resource for meal planning (1).

Protein: The body's need for essential amino acids can be met by consumption of animal or plant sources of protein. Although plant foods contain less of the essential amino acids than do equivalent quantities of animal foods, a plant-based diet can provide adequate amounts of amino acids when energy needs are met and a varied diet is consumed on a daily basis (1). Research indicates that an assortment of plant foods eaten over the course of a day can provide all essential amino acids and ensure adequate nitrogen retention and use in healthy adults; thus, complementary proteins do not need to be consumed at the same meal (1,8). A mixture of different proteins from unrefined grains, legumes, seeds, nuts, and vegetables will complement each other in their amino acid profiles to meet nutritional needs. Estimates of protein requirements may vary based on dietary choices selected, particularly for vegans. Isolated soy protein can meet protein needs as effectively as animal protein, whereas wheat protein eaten alone may be 50% less usable than animal protein (1). Therefore, protein needs might be somewhat higher than the Recommended Daily Allowance in those vegetarians whose dietary protein sources are mainly those that are less well digested, such as some cereals and legumes (1.9). The consumption of lysine, an essential amino acid, should be evaluated in persons who consume a vegan diet or who acquire a large percentage of protein from cereal sources. Cereals tend to be low in lysine. Dietary adjustments, such as the use of more beans and soy products in place of other protein sources that are lower in lysine or an increase in protein from all sources, can ensure an adequate intake of lysine (1,10).

Vitamin B₁₂: Unfortified plant foods do not contain significant amounts of active vitamin B₁₂. Although the requirement for vitamin B₁₂ is relatively small, vegetarians must include a reliable source of vitamin B₁₂ in their diets to reduce their risk of developing a deficiency. Lacto-ovovegetarians can obtain adequate vitamin B₁₂ from the regular consumption of dairy foods, eggs, fortified foods, or supplements (1). Vegans should supplement their diets with vitamin B₁₂ by selecting fortified foods, such as fortified soy or rice beverages, breakfast cereals, meat analogs, or Red Star Vegetarian Support Formula nutritional yeast; otherwise, a daily vitamin B₁₂ supplement is needed to ensure an adequate intake of the active form of the nutrient (1). Older adults who are vegetarian should consume fortified foods or supplements to increase their vitamin B₁₂ intake, because the absorption of vitamin B₁₂ often becomes less efficient in older adults due to atrophic gastritis (1). Also, breast-fed vegan infants should receive a source of vitamin B₁₂ if the mother's diet is not supplemented (1,4,10). If vitamin B₁₂ supplement of 5 to 10 mcg or a weekly B₁₂ supplement of 2,000 mcg (11). Vitamin B₁₂ status is best determined by measuring serum levels of homocysteine, methylmalonic acid, or holotranscobalamin II (1,12). Folacin-rich vegetarian diets may mask the hematological symptoms of vitamin B₁₂ deficiency; therefore, a deficiency may go undetected until the manifestation of neurological signs and symptoms (1,13).

Calcium: Calcium is present in many plant foods and fortified foods. The calcium intake of lactovegetarians is comparable to or higher than that of nonvegetarians (1). However, the calcium intake of vegans is generally lower than that of lactovegetarians and nonvegetarians and is often below the recommended level (1). In one study, the risk of bone fracture was similar for lacto-ovovegetarians and meat eaters, whereas vegans had a 30% higher risk of fracture possibly due to their considerably lower mean calcium intake (1,14). A diet that provides foods with relatively high ratios of sulfur-containing amino acid proteins, such as eggs, meat, fish, poultry, dairy products, nuts, and many grains, may increase calcium loss from the bones (1). Excessive sodium intake may also promote calcium loss from the bones (1). Studies show that the ratio of dietary calcium to protein is more predictive of bone health than calcium intake alone (1). Typically, this ratio is high in lacto-ovovegetarian diets and favors bone health. However, vegan diets have calcium-to-protein ratios that are similar to or lower than those of nonvegetarian diets (1,15). Lower oxalate greens, such as bok choy, broccoli, Chinese cabbage, collards, and kale, and fruit juices fortified with calcium citrate malate are good sources of highly bioavailable calcium (50% to 60% and 40% to 50%, respectively), while calcium-set tofu, and cow's milk have good bioavailability of calcium (30% to 35%), and sesame seeds, almonds, and dried beans have a lower bioavailability (23% to 27%) (1,15). Oxalates in some foods, such as spinach and Swiss chard, greatly reduce calcium absorption, making these vegetables a poorer source of usable calcium (1). Foods rich in phytate may also inhibit calcium absorption (1). If vegans do not meet calcium requirements from food, fortified foods and dietary supplements are recommended (1).

Vegetarian Diet

Vitamin D: Vitamin D status depends on sunlight exposure and intake of vitamin D-fortified foods or supplements (1). If sun exposure and intake of fortified foods are insufficient to meet nutritional_needs, vitamin D supplements are recommended (1). Vitamin D₃ (cholecalciferol) is of animal origin and may not be used by vegans. Vitamin D₂ (ergocalciferol) is produced from ergosterol from yeast and is a form that may be more frequently used by vegans. There is disparity in the research as to whether the bioavailability of vitamin D₂ is less than that of vitamin D₃ (1,16). The need for additional requirements when vitamin D₂ sources are primarily used is not currently suggested by the evidence (1,16). Both vitamin D₂ and vitamin D₃ are used in supplements and to fortify foods (1). Because cutaneous vitamin D production decreases with aging process, dietary or supplemental sources of vitamin D are important when assessing the diets of older adults (1,17).

Energy: Because vegan diets tend to be high in bulk, it can be challenging for vegans, especially infants, children, and adolescents, to meet their energy needs. Frequent meals and snacks and the use of some refined foods (such as fortified breakfast cereals, breads, and pasta) and foods higher in unsaturated fats can help vegan children meet their energy and nutrient needs (1).

Iron: The non-heme iron found in plant foods is more sensitive than heme iron to both inhibitors and enhancers of iron absorption (1). The inhibitors of iron absorption include phytate, calcium, and polyphenols in teas (including some herb teas), coffee, and cocoa (1). Fiber only slightly inhibits iron absorption (1,18). Some food preparation techniques, such as soaking and sprouting beans, grains, and seeds and the leavening of bread, can diminish phytate levels and thereby enhance iron absorption (1). Western vegetarians have a relatively high intake of iron from plant foods, such as dark-green leafy vegetables, iron-fortified cereals, and whole grains. Although vegetarian diets are higher in total iron than nonvegetarian diets, iron stores are lower because iron from plant foods is not absorbed as well as iron from animal sources (1). Because of the lower bioavailability of iron from a vegetarian diet, the recommended iron intakes for vegetarians are 1.8 times those of nonvegetarians (1,19). However, the frequency of anemia is not higher in the vegetarian population than in the nonvegetarian population (1). There is evidence of long-term adaptation to low iron intakes that involves both increased absorption and decreased losses (1,20,21). In addition, vitamin C and other organic acids in fruits and vegetables consumed by vegetarians can substantially enhance iron absorption and reduce the inhibitory effects of phytates, leading to improved iron status (1).

Zinc: Because phytate binds zinc, and animal protein is believed to enhance zinc absorption, total zinc bioavailability appears to be lower in vegetarian diets (1,22). Vegetarians, especially those who consume phytate-rich unrefined grains and legumes, should strive to meet or exceed the DRIs for zinc due to the low bioavailability of zinc from plant sources and the high phytate content of a vegetarian diet (1,19). In addition, breast-fed infants should have their diets evaluated for zinc intake. Zinc-fortified foods or supplements should be used when complementary foods are introduced, if the diet is low in zinc or mainly consists of foods with low zinc availability (1). Due to difficulty in evaluating zinc deficiency, it is not possible to determine the possible effect of lower zinc absorption from vegetarian diets (22). Zinc sources include soy products, legumes, grains, cheese, and nuts. Food preparation techniques, such as the soaking and sprouting of beans, grains, and seeds, as well as the leavening of bread, can reduce the binding of zinc by phytic acid and increase zinc bioavailability (1,23). Organic acids, such as citric acid, can also enhance zinc absorption to some extent (1,23).

Omega-3 fatty acids: Diets that do not include fish, eggs, or generous amounts of algae are generally low in docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), which are long-chain n-3 fatty acids important for cardiovascular health as well as eye and brain development (1). Vegetarians, particularly vegans, have lower blood levels of DHA and EPA than nonvegetarians (1,24). Diets that do not include fish, eggs, or sea vegetables generally lack direct sources of DHA and EPA (1). The bioconversion of alpha-linolenic acid (ALA), a plant-based n-3 fatty acid, to EPA is generally less than 10% in humans, and the conversion of ALA to DHA is substantially less (1,25). The DRIs for ALA may not be sufficient for vegetarians who consume little if any DHA and EPA (1). Vegetarians may need more ALA for the conversion to DHA and EPA. Therefore, vegetarian diets should include high-quality sources of ALA, such as walnuts, flaxseed, flaxseed oil, canola oil, and soy. Vegetarians with increased n-3 fatty acids (eg, cod liver oil, mackerel, salmon, crab, shrimp, and oyster), DHA-fortified foods, eggs from hens fed DHA-rich microalgae, or a DHA-rich microalgae supplement (1,26). DHA supplements derived from microalgae are well absorbed and positively affect blood levels of DHA and 6EPA through reconversion (1,27). Soy milk and breakfast bars fortified with DHA are now available in the marketplace (1).

^{*}The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Position of the American Dietetic Association and Dietitians of Canada: vegetarian diets. J Am Diet Assoc. 2009;109:1266-1282.
- 2. Messina V, Mangels R, Messina M. *The Dietitian's Guide to Vegetarian Diets: Issues and Applications.* 2nd edition. Sudbury, Mass: Jones and Bartlett Publishers; 2004.
- 3. Messina V, Mangela AR. Considerations in planning vegan diets: children. JAm Diet Assoc. 2001;101:661-669.
- 4. Mangela AR, Messina V. Considerations in planning vegan diets: infants. J Am Diet Assoc. 2001;101:670-677.
- 5. Subcommittee on Nutritional Status and Weight Gain During Pregnancy, Committee on Nutritional Status During Pregnancy and Lactation, Food and Nutrition Board, Institute of Medicine, National Academy of Sciences. *Nutrition During Pregnancy*. Washington, DC: National Academy Press; 1992.
- 6. Subcommittee on Nutrition During Lactation, Committee on Nutritional Status During Pregnancy and Lactation, Food and Nutrition Board, Institute of Medicine, National Academy of Sciences. *Nutrition During Lactation*. Washington, DC: National Academy Press; 1991.
- 7. Vegetarian Nutrition in Pregnancy. Vegetarian Nutrition Evidence-Analysis Project. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics. Available at: www.andevidencelibrary.com/topic.cfm?cat=3125. Accessed September 10, 2010.
- 8. Young VR, Pellett PL. Plant proteins in relation to human protein and amino acid nutrition. *Am J Clin Nutr.* 1994;59(suppl): 1203S-1212S.
- 9. Young VR, Fajardo L, Murray E, Rand WM, Scrimshaw NS. Protein requirements of man: comparative nitrogen balance response within the submaintenance-to-maintenance range of intakes of wheat and beef proteins. *J Nutr.* 1975;105:534-542.
- 10. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids.* Washington, DC: National Academy Press; 2002.
- 11. Messina V, Melina V, Mangels AR. A new food guide for North American vegetarians. J Am Diet Assoc. 2003;103:771-775.
- 12. Herrmann W, Schorr H, Purschwitz K, Rassoul F, Richter V. Total homocystei*ne, vitamin B*₁₂, and total antioxidant status in vegetarians. *Clin Chem.* 2001;47:1094-1101.
- 13. Herrmann W, Geisel J. Vegetarian lifestyle and monitoring of vitamin B₁₂ status. *Clin Chim Acta*. 2002;326:47-59.
- 14. Appleby P, Roddam A, Allen N, κey T. Comparative fracture risk in vegetarians and nonvegetarians in EPIC-Oxford. *Eur J Clin Nutr.* 2007;61:1400-1406.
- 15. Weaver C, Proulx W, Heaney R. Choices for achieving adequate dietary calcium with a vegetarian diet. *Am J Clin Nutr*. 1999;70(suppl): 543S-548S.
- 16. Holick MF, Biancuzzo RM, Chen TC, Klein EK, Young A, Bibuld D, Reitz R, Salameh W, Ameri A, Tannenbaum AD. Vitamin D_2 is as effective as vitamin D_3 in maintaining circulating concentrations of 25-hydroxyvitamin D. J Clin Endrocrinol Metab. 2008;93:677-681.
- 17. Holick MR. Vitamin D deficiency. *N Engl J Med.* 2007;357:266-281.
- Coudray C, Bellanger J, Castiglia-Delavaud C, Remesy C, Vermorel M, Rayssignuier Y. Effect of soluble or partly soluble dietary fibres supplementation on absorption and balance of calcium, magnesium, iron, and zinc in healthy young men. *Eur J Clin Nutr.* 1997;51:375-380.
- 19. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.* Washington, DC: National Academies Press; 2001.
- 20. Hunt JR, Roughead ZK. Adaptation of iron absorption in men consuming diets with high or low iron bioavailability. *Am J Clin Nutr.* 2000;71:94-102.
- 21. Hunt JR, Roughead ZK. Nonheme-iron absorption, fecal ferritin excretion, and blood indexes of iron status in women consuming controlled lactoovovegetarian diets for 8 wk. *Am J Clin Nutr.* 1999;69:944-952.
- 22. Hunt JR. Bioavailability of iron, zinc, and other trace minerals from vegetarian diets. Am J Clin Nutr. 2003;78(suppl): 633S-639S.
- 23. Lonnerdal B. Dietary factors influencing zinc absorption. J Nutr. 2000;130(suppl):1378S-1383S.
- 24. Rossell MS, Lloyd-Wright Zechariah, Appleby ON, Sanders TA, Allen NE, Key TJ. Long-chain n-3 polyunsaturated fatty acids in plasma in British meat-eating, vegetarian, and vegan men. *Am J Clin Nutr.* 2005;82:327-334.
- 25. Williams CM, Burdge G. Long-chain n-3 PUFA: plant v. marine sources. Proc Nutr Soc. 2006;65:42-50.
- 26. Conquer JA, Holub BJ. Supplementation with an algae source of docosahexaenoic acid increases (n-3) fatty acid status and alters selected risk factors for heart disease in vegetarian subjects. *J Nutr.* 1996;126:3032-3039.
- 27. Geppert J, Kraft V, Demmelmair H, Koletzko B. Docosahexaenoic acid supplementation in vegetarians effectively increases omega-3 index: a randomized trial. *Lipids*. 2005;40:807-814.

KOSHER GUIDELINES

Description

Kosher is a Hebrew word that means "fit" or "wholesome." Kosher dietary laws define foods and combinations of foods that are allowed or forbidden. The collective term for the Jewish laws and customs relating to the types of foods permitted for consumption and their preparation is *kashruth*. The observance of kosher dietary laws varies according to the traditions of the individual and interpretations of the dietary laws.

In a nonkosher food service facility, observance of dietary laws usually involves service of commercially prepared kosher dinners on disposable plastic ware for the patient following a strict kosher diet. For patients not following a strict kosher diet or if the patient so wishes, the foods usually prepared by the Food and Nutrition Services Department can be served, as long as milk and milk products are separated from meat and meat products and certain forbidden foods are excluded (see the following food guide).

The strict observance of the kashruth by the kosher food service requires separate sets of equipment, dishes, and silverware, as well as kosher food suppliers for many items. Dairy foods are stored and prepared separately from meat and meat products.

Indications

Kosher diets may be ordered for individuals of the Jewish faith if they so desire.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "Kosher Diet." Any additional dietary modifications that may be warranted should be stated in the diet prescription (eg, "Kosher Diet, Sugar in Moderation").

Guidelines for Food Selection:

- 1. Kosher meats and poultry may come only from animals that have cloven hooves, chew their cud, and are slaughtered according to the humane and specific guidelines prescribed by the Jewish dietary laws. In addition, kosher meats undergo a process called koshering, in which blood is extracted by soaking in salt or broiling on a regular grill. (Pan grilling is not acceptable.)
- 2. Foods are classified as dairy, meat, or pareve. Meals are classified either as dairy or meat. Meat and meat products are not to be combined with any dairy products in recipe, food preparation, or service. Pareve foods may be served at dairy or meat meals.
- 3. The strict observance of the Kashruth requires separate sets of equipment, dishes, and silverware for dairy or meat meals. In a kosher kitchen, dairy foods are stored and prepared separately from meat and meat products.
- 4. In a nonkosher food service facility, observance of dietary laws usually involves service of commercially prepared kosher dinners on disposable plastic ware for the patient following a strict kosher diet. For patients not following a strict kosher diet or if the patient so wishes, the usual foods prepared by the Food & Nutrition Services Department can be served, as long as milk and milk products are separated from meat and meat products and certain forbidden foods are excluded (see the following list).
- 5. Processed foods: No product should be considered kosher unless so certified by a reliable rabbinic authority whose name of insignia appears on the sealed package. The insignia, U which is the copyrighted symbol of the Union of Orthodox Jewish Congregations of America, indicates that the product is certified as to its kosher nature. Packages marked with other symbols may be suitable for certain but not all kosher diets. It is important that a kosher food package remains sealed when presented to the user. The package should be opened only under these circumstances: by the user, in the user's presence, or by someone authorized by the religious authorities to open the food package.
- 6. Nonkosher foods may be used if considered essential in the treatment of an ill person. However, a rabbi should be consulted before waiving dietary restrictions.

	FOOD GUIDE – KOSHER DIET				
	FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED		
Dairy	Milk Products	All foods containing milk or white sauces Note: Foods containing milk derivatives such as sodium caseinate and lactose are considered dairy			
Meat	Meat	Only meat from an animal that chews its cud and has split hooves	Pork and pork products		
		Beef: chuck, brisket, plate, shank, rib up to and including 12th rib Broiled liver	Beef: loin, rump, flank, shank, hindquarter		
		Veal/lamb: shoulder, rack, shank, breast	Veal: loin, leg, flank, shank, Lamb: loin, leg, hind quarter		
	Fowl	Most domesticated fowl are by tradition considered to be kosher: chicken, turkey, domestic duck	Wild fowl that is hunted		
Pareve	Breads, Cereals, and Grains	All except listed in Foods Excluded column	Bread made with lard or animal shortening. Note: Breads and cereals containing any dairy products are classified as dairy		
	Eggs	Eggs from domestic fowl	Eggs containing blood spots		
	Fish and Seafood	Fish having <i>both</i> fins and scales: halibut, flounder, cod, tuna, haddock, pollack, turbot, salmon, trout, whitefish, herring, etc.	Catfish, eel, marlin, sailfish, shark, sturgeon, swordfish, lumpfish, scallops, and shellfish such as lobster, shrimp, crab and oysters		
	Vegetables and Fruit	 All, prepared with pareve certification and allowed ingredients; fresh do not require Kashruth certification. Baked beans, catsup, chick peas, chow mein noodles, dehydrated or canned soup and bases, prepared sauces, tomato juice, tomato products, frozen fruits and vegetables in sauce, grape juice, blended fruit juice drinks and punches, must have Kashruth certification 			
	Fats	Pure vegetable oil Margarine made with vegetable shortening and without milk	Lard or animal shortening Margarine with added milk Butter		
	Sweets	Imitation sour cream or whipped topping with pareve certification Sugar, jam, jelly (grape jelly only if has Kashruth certification), syrup Candy without milk			
	Beverages	Coffee, tea, carbonated beverages Alcoholic beverages Nondairy creamer with pareve certification Those made with milk or milk products are considered to be a part of the dairy group			
Other	Desserts	Desserts made without milk or animal products are considered to be pareve certified	Desserts made with lard or animal shortening Monoglycerides and diglycerides and emulsifiers that may be from animal fats		

I. NORMAL NUTRITION AND MODIFIED DIETS B. Transitional Diets

Clear Liquid Diet	B-1
Food Guide – Clear Liquid Diet	B-1
Full Liquid Diet	B-3
- Food Guide – Full Liquid Diet	B-3
Full Liquid Blenderized Diet	B-4
Food Guide – Full Liquid Blenderized Diet	B-5
Nutrition Management Of Dysphagia	B-6
NDD Level 1: Dysphagia Pureed	B-9
NDD Level 2: Dysphagia Mechanically Altered	B-11
NDD Level 3: Dysphagia Advanced	B-13
Dumping Syndrome Diet	B-15
Nutrition Management In Bariatric Surgery	B-17
Table B-1: Routine Nutrient Supplementation After Bariatric Surgery	B-24
Table B-2: Foods to Choose on a Pureed/Soft Diet Following Bariatric Surgery	B-26
Table B-3: Guide to Choosing Regular Foods After Bariatric Surgery	B-29
Specialized Nutrition Support Therapy	B-33
Enteral Nutrition Support Therapy For Adults	B-35
Parenteral Nutrition Support For Adults	B-50
Table B-4: FDA Requirements for Parenteral Multivitamin Products	B-55
Table B-5: Daily Parenteral Trace Element Supplementation for Adults (Dose Per	Day) B-55
Table B-6: Daily Electrolyte Requirements	B-56
Table B-7: Monitoring Hospitalized Patients Receiving CPN	B-57

CLEAR LIQUID DIET

Description

The Clear Liquid Diet is designed to provide fluids mainly in the form of sugar and water to prevent overstimulating extensive digestive processes, minimize colonic residue, relieve thirst, and provide oral feedings that promote the return to the normal ingestion of food (1). The diet as served will yield 700 to 1,000 kcal when energy-containing clear liquids are served between meals.

Indications

The Clear Liquid Diet is indicated for the following:

- short-term use when an acute illness or surgery causes an intolerance for foods (eg, abdominal distention, nausea, vomiting, and diarrhea)
- to temporarily restrict undigested material in the gastrointestinal tract or reintroduce foods following a period with no oral intake when poor tolerance to food , aspiration, or an anastomotic leak is anticipated
- to prepare the bowel for surgery or a gastrointestinal procedure

Nutritional Adequacy and Nutrition Intervention

The Clear Liquid Diet is inadequate in all food nutrients and provides only fluids, energy, and some vitamin C. Long-term use of the Clear Liquid Diet may contribute to hospital malnutrition (1). Current preparation methods for bowel surgery or bowel procedures have decreased the time required for bowel preparation to 1 to 2 days (1). Knowledge regarding the time required for gastric emptying has increased; thus, usually only one preoperative meal as clear liquid is required before surgery (1). The American Society of Anesthesiologists' Task Force on Preoperative Fasting recommends abstaining from clear liquids for 2 or more hours before procedures requiring general anesthesia and abstaining from the intake of light meals or nonhuman milk at least 6 hours before elective surgery requiring general anesthesia (2). The resumption of bowel sounds is no longer a prerequisite to resume a regular diet after surgery (1). Improved anesthesia and evidence have led to the postoperative transition to a regular diet based on individual tolerance (1). The Clear Liquid Diet provides approximately 200 g/day of carbohydrate in equally divided amounts. Liquids are not sugar-free, even for persons with diabetes mellitus, because all patients require carbohydrates and energy to meet nutritional needs (3,4). Diabetes medications may need to be adjusted to achieve and maintain metabolic control (3,4).

How to Order the Diet

Order as "Clear Liquid Diet." Variations of this standard diet should be specifically ordered; specify the exclusion of certain foods or specify a diet limited to certain foods.

A diet order specifying the number of meals or days of liquids or the diet progressions, as tolerated, will ensure that this nutritionally inadequate diet is advanced or evaluated.

FOOD CHIDE CLEAD LIQUID DIET

FOOD GUIDE – CLEAR LIQUID DIE I		
FOODS ALLOWED	FOODS EXCLUDED	
Carbonated beverages, regular and decaffeinated;	All other foods or fluids except water	
Clear flavored gelatin, fruit ices, Popsicles		
Cranberry, apple, and grape juices		
Lightly seasoned clear broth or consommé (fat-free)		
Sugar, honey, syrup		

SAMPLE MENU (600 kcal)

Breakfast	Noon	Evening
Cranberry Juice	Beef Broth	Chicken Broth
Flavored Gelatin	Grape Juice	Apple Juice
Coffee or Tea	Flavored Gelatin	Water or Ice Chips
Sugar	Coffee or Tea	Coffee or Tea
	Sugar	Sugar

CLEAR LIQUIDS BETWEEN MEALS AS DESIRED

References

- 1. Clear liquid diet. In: *The Academy of Nutrition and Dietetics Nutrition Care Manual; Updated annually*. Available at: www.nutritioncaremanual.org. Accessed December 1, 2008.
- 2. American Society of Anesthesiologist Task Force on Preoperative Fasting. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures. *Anesthesiology*. 1999;90:896-905.
- 3. American Diabetes Association. Diabetes nutrition recommendations for health care institutions. *Diabetes Care*. 2004; 27: 55S-57S.
- 4. Clement S, Braithwaite SS, Magee MF, Ahmann A, Smith EP, Schafer RG, Hirsch IB. Management of diabetes and hyperglycemia in hospitals. *Diabetes Care.* 2004;27:553-591.

FULL LIQUID DIET

Description

The Full Liquid Diet consists of foods that are liquid at body temperature, including gels and frozen liquids. The diet provides nourishment that is easy to consume and digest with very little stimulation to the gastrointestinal tract.

Indications

The Full Liquid Diet may be indicated following oral surgery or plastic surgery of the face or neck area in the presence of chewing or swallowing dysfunction for acutely ill patients.

The Full Liquid Diet has been traditionally used as a postoperative transitional diet. The diet is intended for short-term use only; therefore, attempts are not usually made to increase the variety of foods offered to provide for the total adequacy of nutrients.

Contraindications

Due to the liberal use of milk and foods made with milk, the diet is high in lactose. A temporary lactose intolerance may occur in some patients following surgery. Symptoms of lactose intolerance upon ingestion of a Full Liquid Diet may result, and the diet should be modified for the patient. See Section IH: Lactose-Controlled Diet.

Nutritional Adequacy

The diet as served meets the Dietary Reference Intakes (DRIs) for ascorbic acid, vitamin D, vitamin B_{12} , calcium, phosphorus, and riboflavin. It may not meet the protein and caloric requirements of the individual. The diet as served will provide approximately 1200 kcal and 40 g of protein. When between-meal nourishment is added, the intake is increased to 1500 to 1800 kcal and 65 g of protein. Protein and caloric intake can be increased through the use of additional full liquid foods at meals and between meals. The diet can be nutritionally adequate when supplements are offered and consumed in sufficient amounts.

How to Order the Diet

Order as "Full Liquid Diet." Variations in this standard diet should be specifically ordered (eg, the exclusion of certain foods or a diet limited to certain foods). A diet order specifying the duration of the diet or the diet progression, as tolerated, will ensure that this nutritionally inadequate diet is advanced or evaluated.

FOOD GUIDE - FULL LIQUID DIET

FOODS ALLOWED	FOODS EXCLUDED
Carbonated beverages, regular and decaffeinated coffee and tea, soft drinks, cocoa	All solid foods
Cooked refined cereal, farina, cream of rice, or strained cereal	
Custard, plain gelatin, ice cream, sherbet, pudding, yogurt, all without nuts;	
fruit or preserves	
Eggnog [*] , milk shake, and other milk drinks	
Butter, margarine, cream	
Fruit and vegetable juices (including one serving of citrus fruit juice daily)	
Broth, bouillon, consommé, strained cream soup	
Honey, sugar, syrup	
*Made from pasteurized eggs only or commercial product.	

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Strained Cream of Chicken Soup	Strained Cream of Celery Soup
Cream of Wheat	Grape Juice	Apple Juice
Milk	Vanilla Ice Cream	Custard
Coffee or Tea	Milk	Milk
Sugar	Coffee or Tea	Coffee or Tea
	Sugar	Sugar

FULL LIQUIDS BETWEEN MEALS AS DESIRED

FULL LIQUID BLENDERIZED DIET

Description

The Full Liquid Blenderized diet consists of a variety of liquids, as well as semisolid foods that have been thinned to a consistency that can be consumed through a straw, fed by syringe, or sipped from a cup. The diet also includes foods that, if eaten by spoon, will turn to a liquid consistency in the mouth. The method of feeding will determine the desired viscosity of the liquid.

Indications

The objective of the diet is to provide oral nourishment in a form that requires no mastication. This diet is indicated for the following:

- patients following oral surgery or plastic surgery of the face or neck area in the presence of chewing or swallowing dysfunction (eg, a wired jaw or intermaxillary fixation surgery)
- acutely ill patients with oral esophageal disorders, neuromuscular disabilities, advanced carcinoma of the oral cavity, facial or neck trauma
- patients who have received radiation therapy and find eating difficult

Nutritional Adequacy

The Full Liquid Blenderized Diet can meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy, if the proper amount and variety of food is consumed by the patient. Because some patients experience palatability problems or may have difficulty consuming an adequate volume of liquids, liquid supplements may be necessary to meet their nutrient and fluid needs.

How to Order the Diet

Order as "Full Liquid Blenderized Diet" or "Wired Jaw." These terms will distinguish this diet from the Full Liquid Diet, which is lower in energy and nutrients. The dietitian determines the amount and type of food or supplements to be served, based on patient acceptance, nutrient needs, and change in condition.

FOOD GUIDE - FULL LIQUID BLENDERIZED DIET

NOTE: The foods listed below vary greatly in caloric and nutrient density. It may be necessary to encourage certain foods, depending on the nutritional goals for the individual.

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages and Milk	Milk, eggnog, milk shake, milk drinks All beverages: coffee, tea, etc. Yogurt without seeds or fruit (may need to be thinned for straw or syringe feeding)	All other
Bread and Cereals	Cereal gruels of farina, cream of rice, grits (Cereal gruels are equal parts whole milk with cereal) Strained oatmeal or cream of wheat Note: iron-fortified cereals are recommended	All other
Vegetables	Mashed white potato, thinned with soup or broth Vegetable juices Vegetable purees thinned with soups	All other
Fruits	Fruit juices Pureed fruits thinned with fruit juice Juices may need to be strained to remove excess pulp for straw or syringe feeding Note: Citrus juices may not be well tolerated by all surgical patients	All other
Meats, Poultry, Fish, Cheese, and Eggs	Pureed meats and poultry (baby strained), thinned with broth	All other
Fats	Margarine, butter Nondairy creamers, half-and-half	
Soups	Broth, strained cream soups	All other
Desserts	Ice cream, sherbet, plain gelatin, custards, puddings, fruit ices Popsicle (may need to be liquefied or melted for straw or syringe feeding).	All other
Sugar and Sweets	Sugar, syrup Hard candy if tolerated	All other
Miscellaneous	Salt, pepper, herbs Lemon juice, other condiments and seasonings as tolerated	All other

NUTRITION MANAGEMENT OF DYSPHAGIA

Description

Dysphagia is not a disease, but a disruption in swallowing function. Although dysphagia can occur at any age, it is particularly prevalent in older adults (1). Dysphagia may result from neurological disorders, degenerative diseases, cancers, or post intubation trauma (1). The nutrition management of dysphagia includes modifying the consistency and texture of foods and liquids according to the patient's tolerance, which is determined by a comprehensive medical, swallowing, and nutrition evaluation by the healthcare team including the physician, speech-language pathologist, and registered dietitian. Therapeutic goals for nutrition are customized and often include diet modifications and swallowing retraining. An individualized meal plan will generally include modifications in the texture and consistency of foods (eg, pureed or textured-modified foods and thickened liquids) that optimize the quality of nutritional intake while reducing the risk of aspiration or choking.

Indications

Dysphagia is an impairment in one or all stages of swallowing, resulting in the reduced ability to obtain adequate nutrition by mouth and a possible reduction of safety during oral feeding (1,2). Patients with dysphagia have difficulty moving food from the front to the back of the mouth, channeling the food into the esophagus, or both processes. Dysphagia may be caused by weak or uncoordinated muscles of the mouth and/or throat, motor and sensory defects impeding chewing or swallowing, or both conditions. If dysphagia is suspected, a swallowing evaluation should be performed by a qualified healthcare provider (1). This evaluation may include a bedside evaluation, indirect or fiberoptic laryngoscopy, fiberoptic endoscopic evaluation of swallowing, and a videofluoroscopic swallow study (VFSS), which is also known as a modified barium swallow study. The VFSS is a definitive test in diagnosing the type of dysphagia (1). Fiberoptic endoscopic evaluation of swallowing was recently shown to be as reliable as VFSS when using the Penetration-Aspiration Scale (1). However, the VFSS remains the preferred diagnostic tool for dysphagia because it determines any structural and functional problems that may occur with varied food and liquid consistencies and rules out inappropriate diet consistencies.

The nutrition care plan for patients with dysphagia is developed based on (3):

- results of VFSS
- recommendations from the speech-language pathologist
- nutrient requirements of the patient
- food preferences of the patient
- other medical, psychological, or social factors affecting the patient's eating

Nutritional Adequacy

Dysphagia diets can be planned to meet the Dietary Reference Intakes as outlined in Section IA: Statement on Nutritional Adequacy. Enteral feedings may be necessary to supplement oral intake until a sufficient quantity of food can be consumed. If enteral nutrition for neurological dysphagia is anticipated to last for longer than 4 weeks, a percutaneous endoscopic gastrostomy (PEG) tube is preferable to a nasogastric tube (4). PEG tubes are associated with fewer treatment failures and improved nutritional status as compared to nasogastric tubes, and they allow the patient to receive adequate nutrition while oral intake is stabilized (4). In one study, more than half of the patients who received a PEG tube due to poor tolerance of thickened food were eventually able to resume oral feedings (1,5). If a patient can tolerate oral liquids, the medical food supplements should be in compliance with the consistency prescribed for the patient.

A record of food intake, including fluid intake and enteral feedings, is necessary at all stages of dysphagia therapy. When oral intake approaches the patient's energy and protein requirements, the patient should begin to be weaned from the enteral feedings. A review of seven studies of modified-texture diets in older adults with dysphagia found that people who consume these diets report an increased need for assistance with eating, dissatisfaction with foods, and decreased enjoyment in eating, resulting in decreased food intake and weight loss (Grade I)* (4). Recognition of the social and psychological burden of dysphagia, creation of an individualized treatment approach, and provision of eating assistance may contribute to increased food intake and weight maintenance or weight gain in older adults (Grade I) (4).

How to Order the Diet

The dietitian and the speech-language pathologist should work collaboratively and use the results from both the medical evaluation and a swallowing study to choose appropriate foods and beverages for the individual patient (1). The National Dysphagia Task Force found that the Dysphagia Outcome and Severity Scale provided the best scale to determine the level at which the National Dysphagia Diet (NDD) should be recommended (1,6).

Prior to the initial oral feeding, a diet order specifying that the patient can eat must be obtained from the physician. The dietitian and the speech-language pathologist must coordinate efforts to determine the appropriate consistency of foods and liquids for the patient both before feedings begin and with subsequent feedings. The consistency of foods and liquids should be altered as the patient progresses.

The severity of dysphagia determines the level of the diet required. The food plan is divided into multiple levels of solid food and liquid consistency to maximize the dysphagic patient's nutritional intake (2). Diet orders should include the National Dysphagia Diet levels and the desired liquid consistency (eg, thin, nectar-like, honey-like, or spoon-thick) (6). With each progression of the diet, both the level of the diet and the liquid consistency need to be specified in the nutrition prescription (6). The three NDD levels are (6):

- NDD Level 1: Dysphagia Pureed: Foods are thick and smooth and have a moist pudding-like consistency without pulp or small food particles. They cling together, are easy to swallow, and require a minimum amount of manipulation in the mouth. Sticky foods or foods that require a bolus formation or controlled manipulation of the mouth (eg, melted cheese and peanut butter) are omitted. The diet provides no coarse textures (eg, fibrous foods) to prevent irritation. Food and fluid intake should be monitored.
- NDD Level 2: Dysphagia Mechanically Altered: Foods are moist, soft, and simple to chew, and they easily form a cohesive bolus. The diet provides a transition from pureed foods to easy-to-chew foods. Moistened ground meats (pieces should not exceed ¼-inch cube), vegetables cooked to a soft mashable texture, soft-cooked or canned fruits, and bananas are included. Some mixed textures are expected to be tolerated. More frequent feedings may be beneficial. Food and fluid intake should be monitored.
- **NDD Level 3: Dysphagia Advanced:** Foods are moist, soft, in bite-size pieces, and nearly regular in texture. Hard, sticky, and crunchy foods are excluded.

Planning the Diet

General considerations: Dietary considerations vary with each patient. The importance of individual food consistencies cannot be overemphasized. For example, dysphagic patients with an obstruction may be able to safely consume liquids, while other dysphagic patients may aspirate liquids and require thickened liquids with a puree consistency. A recent study showed that carbonated liquids are a dietary option for patients who experience penetration/aspiration into their airways (7); thickened liquids are also safe for these patients (1). If a patient cannot tolerate thin liquids, foods that become thin liquid at room temperature (70°F) or body temperature (98°F), such as gelatin, ice cream, and sherbet, should also be avoided.

The following guidelines should be considered when planning the diet for a dysphagic patient: One of the most important considerations of food texture is cohesiveness, or the ability to stay together. Patients can often chew through foods but are unable to press the food into a bolus unless it is naturally or artificially cohesive. For patients who cannot swallow a smooth pureed food, a higher-texture food (more viscous) is desired to rehabilitate muscles. The larger surface area provides stimulation to the nerve and muscle groups that assist the swallowing process.

- Do not combine textures, such as dry cereal with milk or chunky vegetable soup in broth, in the same bolus. Do not use fluids to wash down the bolus. It may be appropriate to alternate liquid and solids. Present foods and fluids separately, checking for complete swallows after each mouthful.
- Use smooth gravies on all ground meat.
- Rice and cottage cheese are difficult for some dysphagic patients to swallow. Use rice in casseroles with a soup base, and include only pureed small-curd cottage cheese in the diet. Milk does not cause mucous formation. However, milk can aggravate thickening of mucus in some people, which can reduce their ability to manage secretions. Blended yogurt or lactose-free supplements may be used if milk is not tolerated.

Nutrition Management of Dysphagia

Liquids: Patients who have dysphagia frequently have difficulty drinking thin liquids, which are not easily channeled to the back of the mouth. Fluid intake is often limited in patients with dysphagia, leading to an increased risk of dehydration (1). Adequate hydration can be ensured by the introduction of thickened liquids followed by the progression to thinner liquids as swallowing proficiency is gained. While there is some fluid content in many foods, especially pureed foods, the use of thickened liquids may be necessary to assure adequate hydration."

The effects of thickening powders in achieving targeted levels of thickness are generally inconsistent due to variations in the temperature, pH, and protein content of foods and beverages (2). There are currently no nationally recognized standards for thickened liquids (2). The majority of commercially prepared powder thickeners require 1 tbsp of thickening product per 4 fl oz to achieve a nectar-like thickness, 1½ tbsp per 4 fl oz for a honey-like thickness, and 2 tbsp per 4 fl oz for spoon-thick thickness. Registered dietitians need to be aware that there is wide variation in the viscosity of commercially prepared thickeners added to thin liquids also results in wide variations in viscosity (2).

Viscosity Borders and Ranges for Thickened Liquids

The NDD Task Force has suggested the following viscosity borders and ranges (All measurements were performed at 25 °C with a shear rate of 50 s⁻¹.) (2,6):

- thin liquid: 1-50 cP*
- nectar-like liquid: 51-350 cP
- honey-like liquid: 351-1750 cP
- spoon-thick liquid: >1750 cP
 *cP = centipoise, a measurement of liquid thickness

Thin liquids include all unthickened beverages and supplements such as clear juices (fruit or vegetable), water, coffee, tea, soda, milk, eggnog, juice from canned fruit, fruit with thin-liquid properties (watermelon, grapefruit, and orange sections), broth, ice cream, sherbet, malts, most nutritional supplements (at room temperature), frozen yogurt, gelatin, and other foods that will liquefy in the mouth within a few seconds. Nectar-like liquids include nectars, vegetable juices, chocolate milk, buttermilk, thin milkshakes, cream soups, and other beverages properly thickened (1.6). Medical food supplements providing 1.5 to 2.0 kcal/mL are usually considered nectar-like when chilled. Honey-like liquids are thickened to a honey consistency (1.6). Spoon-thick liquids, which include pudding, custard, and hot cereal, are thickened to pudding consistency and need to be eaten with a spoon (1.6).

Factors that affect thickened liquid viscosity include (1,8):

- temperature
- continuous hydration of the thickening agent in prethickened beverages
- ability of instant food thickener to maintain thickness"
- inconsistency across product lines within manufacturers or between competitors

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Dysphagia. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: nutritioncaremanual.org. Accessed September 20, 2010.
- 2. Niedert KC, ed. Nutrition Care of the Older Adult: A Handbook for Dietetics Professionals Working Throughout the Continuum of Care. 2nd ed. Chicago, Ill: American Dietetic Association; 2004:211.
- 3. Walker G, ed. *Pocket Resource for Nutrition Assessment*. Chicago, Ill: American Dietetic Association; 2005:173-181.
- 4. Unintended Weight Loss in Older Adults Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2006. Available at: www.andevidencelibrary.com. Accessed July 31, 2010.
- 5. Wilkinson TJ, Thomas K, MacGregor S, Tillard G, Wyles C, Sainsbury R. Tolerance of early diet textures as indictors of recovery from dysphagia after stroke. *Dysphagia*. 2002;17:227-232.
- 6. National Dysphagia Diet Task Force. National Dysphagia Diet: Standardization for Optimal Care. Chicago, Ill: American Dietetic Association; 2002.
- 7. Mahan K, Escott-Stump S. Medical nutrition therapy for neurologic disorders. In: *Krause's Food & Nutrition Therapy*. 12th ed. St. Louis, Mo: Saunders; 2008:1074-1077.
- 8. Adeleye B, Rachal C. Comparison of the rheological properties of ready-to-serve and powdered instant food-thickened beverages at different temperatures for dysphagic patients. *J Am Diet Assoc.* 2007;107:1176-1182.

FOOD GUIDE – DYSPHAGIA DIETS

NDD Level 1: Dysphagia Pureed

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages and Milk	Smooth beverages without lumps, chunks, or pulp. Beverages may need to be thickened to the appropriate consistency.	Milk, coffee, tea, sodas, nutritional supplements (<i>may be given if</i> <i>thin liquids are allowed</i>)
Cereals and Grains Cereals should have a pudding-like consistency and may have enough milk to moisten.	 Farina-type cooked cereals; cooked cereal should have a pudding-like texture Pregelled or slurried through the entire thickness: doughnuts, pancakes, waffles, French toast, bread Pasta, rice, and dressing that are pureed to a smooth consistency 	Coarse cooked or dry cereals Cereals with seeds or nuts All other breads Crackers Regular rice Oatmeal Muffins
Vegetables, Potatoes, and Soups	Pureed or strained vegetables without chunks or seeds; mashed white potatoes Smooth cream soups or broth-type soups with pureed and strained ingredients	Regular cooked or raw vegetables Potato skins and chips Fried or french-fried potatoes or vegetables Regular soups with rice, corn, peas, or large chunks of meat and vegetables
Fruits and Juices	Applesauce, pureed fruits, well- mashed bananas Fruit juices without pulp or seeds (may be given if thin liquids are allowed)	Regular canned, fresh, or frozen fruits Fruit juice with pulp or seeds (may be given if thin liquids are allowed)
Meats, Meat Substitutes, and Entrees	Pureed or strained meats, poultry, or fish Pureed scrambled eggs Soufflés that are smooth and homogenous	Regular, chopped, or ground meats or casseroles Cottage cheese Cheese slices or cubes Scrambled or hard-cooked eggs Peanut butter Sandwiches Pizza
Desserts	Smooth custard and pudding Plain or custard-style yogurt Desserts pureed to a smooth and moist consistency	Ice cream, sherbet, frozen yogurt, other ices <i>(may be given if thin liquids are allowed)</i> Regular cake, pie, cookies Bread and rice pudding Fruited yogurt
Fats	Butter, margarine, smooth gravy, cream sauces, mayonnaise, salad dressings, cream cheese, sour cream, whipped toppings	All fats with coarse or chunky additives
Miscellaneous	Sugar, jelly, honey, syrup Ketchup, mustard, smooth sauces	Jams and preserves Coarsely ground pepper and spices

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Classic Puree Chicken	Classic Puree Beef
Cream of Wheat	Mashed Potatoes with Gravy	Classic Puree Pasta
Pureed Scrambled Egg	Classic Puree Carrots	Classic Puree Green Beans
Biscuit with Slurry	Classic Puree Rosy Pears	Tomato Juice
Margarine	Margarine	Margarine
Jelly	Pudding	Classic Puree Peaches
Milk	Теа	Milk
Coffee	Sugar	
Sugar, Creamer		

Beverages thickened to appropriate viscosity, per diet order

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages and Milk	Beverages with minimal amounts of texture or pulp (any texture should be suspended in the liquid and should not precipitate out)	Milk, coffee, tea, sodas, nutritional supplements (may be given if thin liquids are allowed)
Cereals and Grains Cereals may have ¼ cup milk or just enough milk to moisten if thin liquids are restricted. The moisture should be well blended.	Soft pancakes, well moistened with syrup Cooked cereal with little texture, including oatmeal; slightly moistened cold cereals with little structure, such as corn flakes, Rice Krispies, Wheaties Unprocessed wheat bran stirred into cereals for bulk Pregelled or slurried breads that are gelled through the entire thickness Well-cooked pasta in sauce	Coarse cooked or whole grain dry cereals; cereals with seeds, nuts, or dry fruits All other breads Crackers Rice
Vegetables, Potatoes, and Soups Vegetables should be < ½ inch and easily mashed with a fork.	Soft-cooked or mashed vegetables, including cooked vegetables without hull or stringy fibers Well-cooked, moistened, boiled, baked, or mashed potatoes Well-cooked shredded hash brown potatoes that are not crisp Soups with easy-to-chew or easy- to-swallow meats or vegetables	Cooked peas or corn; raw vegetables Potato skins and chips Fried or french-fried potatoes or vegetables Broccoli, cabbage, brussels sprouts, asparagus, or other fibrous, non-tender, or rubbery cooked vegetables Soups with rice, corn, peas, or large chunks of meat and vegetables
Fruits and Juices	Soft drained canned or cooked fruits without seeds or skin; fresh soft/ripe banana, jelled cranberry sauce Fruit juices with small amounts of pulp (If thin liquids are restricted, fruit juices should be thickened to appropriate viscosity.)	Fruit cocktail, grapes, cherries, or apricots with skin; fresh, canned, or cooked pineapple; fresh fruits except ripe banana; dried fruits; frozen fruits Watermelon without seeds (may be given if thin liquids are allowed)
Meats, Meat Substitutes, and Entrees Meat pieces should not exceed ¼- inch cube and should be tender.	 Moist ground meat; casseroles (without rice); melted cheese in casseroles Protein salads, such as tuna or egg, without large chunks, celery, or onion Cottage cheese; smooth quiche without large chunks Scrambled eggs, soufflés Well-cooked, slightly mashed, moist legumes such as baked beans 	Dry or tough meats (such as bacon, sausage, hot dogs, bratwurst) Dry casseroles or casseroles with rice or large chunks Cheese slices or cubes Hard-cooked egg Peanut butter Sandwiches Pizza

NDD Level 2: Dysphagia Mechanically Altered

NDD Level 2: Dysphagia Mechanically Altered (Cont.)

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Desserts	Custard, pudding Soft fruit pies with bottom crust only Crisps and cobblers without seeds, coconut, or nuts and with soft breading or crumb mixture Soft, moist cakes with icing or slurried cakes Pregelled cookies or soft, moist cookies that have been dunked in milk, coffee, or other liquid Soft, smooth chocolate bars that are easily chewed	Ice cream, sherbet, frozen yogurt, other ices (may be given if thin liquids are allowed) Dry cookies or cake Bread and rice pudding Anything with nuts, seeds, pineapple, or dried fruit Chocolates with nuts and fruits
Fats	Butter, margarine, gravy, cream sauces, mayonnaise, salad dressings, cream cheese, sour cream, whipped toppings	All fats with coarse or chunky additives
Miscellaneous	Jams and preserves without seeds; jelly Sauces and salsas with small tender chunks (< ½ inch)	Seeds, coconut, nuts Sticky or hard foods

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Honey Glazed Chicken, Ground	Braised Beef with Gravy, Ground
Cream of Wheat	Buttered Potatoes	Soft-Cooked Noodles with Gravy
Scrambled Egg	Soft-Cooked Carrots	Soft-Cooked Green Beans
Biscuit with Slurry	Margarine	Peach Slices, Drained
Margarine	Frosted Banana Cake	Margarine
Jelly	Milk	Pudding
Milk	Теа	Iced Tea
Coffee	Sugar	Sugar
Sugar	-	-
Creamer		
Beverages thickened to appropriate viscosity, per diet order		

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages and Milk	All beverages	None
Cereals and Grains All items must be well moistened. Moisten cereals with ¼ cup milk or enough milk to moisten if thin liquids are restricted.	Cooked cereals Well-moistened dry cereals Pasta, noodles, rice Moist bread dressing Well-moistened soft breads, rolls, pancakes, plain muffins, biscuits (<i>Use margarine, butter,</i> <i>jelly, or syrup to moisten.</i>)	Cereals or breads with raisins or nuts Granola-type, coarse, or dry cereals such as shredded wheat or All Bran Thick-crust breads (such as French bread or baguettes) Crackers
Vegetables, Potatoes, and Soups	Tender soft-cooked vegetables Vegetable juices Most soups Shredded lettuce Fried, mashed, or baked potatoes without skin	Raw or cooked vegetables with tough skins or seeds; fried or raw vegetables; cooked corn Tough, crisp-fried potatoes Soups with tough meats or vegetables; clam or corn chowder
Fruits and Juices	Ripe banana, melon, peeled peaches, pears Cooked or frozen fruit Canned peaches, pears, apricots Fruit juices Soft berries with small seeds such as strawberries	Fruit cocktail, grapes, cherries, or apricots with skin; fresh fruit except ripe banana and those listed as allowed; dried fruits Watermelon without seeds (may be given if thin liquids are allowed)
Meats, Meat Substitutes, and Entrees All meats must be well moistened. Add extra gravy or sauces as needed.	Tender meat, fish, or poultry Soft cheese Chopped or ground meats, poultry Soft casseroles Meat, fish, or egg salads Eggs (prepared any way) Smooth peanut butter; liverwurst Yogurt without nuts or coconut	Dry or tough meats (such as bacon, sausage, hot dogs, bratwurst) Chunky peanut butter
Fats	All except those to avoid	Fats with coarse, difficult-to-chew, or chunky additives
Desserts	Cake, tender cookies Custard, pudding Ice cream, sherbet, frozen yogurt, other ices (may be given if thin liquids are allowed) Pies: cream, custard, pumpkin, soft fruit with bottom crust only	Desserts containing nuts, coarse dried fruit, or tough fruit Desserts baked to a hard consistency
Miscellaneous	Soft candy Jelly, smooth jams All sauces	Candy containing tough fruits, coconut, or nuts; hard candy Chewy caramel or taffy-type candies

NDD Level 3: Dysphagia Advanced

Breakfast	Noon	Evening
Orange Juice	Honey Glazed Chicken, Ground	Braised Beef with Gravy, Ground
Cream of Wheat	Buttered Potatoes	Noodles with Gravy
Scrambled Egg	Soft-Cooked Carrots	Soft-Cooked Green Beans
Biscuit	Dinner Roll	Peach Slices
Margarine	Margarine	Dinner Roll
Jelly	Frosted Banana Cake	Margarine
Milk	Milk	Soft Cookie
Coffee	Теа	Iced Tea
Sugar	Sugar	Sugar
Creamer		
Beverages thickened to appropriate viscosity, per diet order		

SAMPLE MENU

DUMPING SYNDROME DIET

Description

The diet is modified to prevent the rapid introduction of a hyperosmolar solution into the proximal jejunum ("dumping"). Several nutrition strategies may be employed, including altered macronutrient composition, size and timing of meals and avoidance of certain food constituents. The diet limits beverages and liquids at meals, limits the intake of simple carbohydrates and sugar, and is high in protein and moderate in fat. Fiber gradually integrated into the meal plan may also be beneficial in delaying gastric emptying (1).

Indications

The dumping syndrome is a complication that may result from:

- the reduced storage capacity of the stomach following gastrectomy
- any procedure that interferes with the pyloric sphincter or compromises the reservoir function of the stomach or alters secretion of GI hormones

The "dumping syndrome" occurs in response to the presence of undigested food in the jejunum. When this occurs, plasma fluids shift into the intestine area to equalize osmotic pressure, causing a drop in blood volume. Symptoms vary among individuals and may consist of the following: abdominal bloating, nausea, cramps, diarrhea, weakness, diaphoresis and tachycardia. In most cases, symptoms appear within 30 minutes after a meal (1). Some postgastrectomy patients experience "late dumping syndrome" characterized by hypoglycemia 1 to 3 hours after a meal. Late dumping syndrome results from rapid absorption of simple sugars in the small bowel, which triggers an exaggerated release of insulin resulting in reactive hypoglycemia (1). Patients with late dumping syndrome commonly complain of sweating, dizziness, tachycardia, irritability, hunger and syncopal symptoms (1). Dumping syndrome symptoms are more prevalent in the immediate post-operative period and frequently resolve overtime (2). Dumping syndrome unresponsive to diet manipulation may require use of gut-slowing medication (2).

Contraindications

If patient has malabsorption of fat, do not increase fat intake with the dumping syndrome diet.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy. The adequacy of the diet depends on the extent of the surgery and the individual's food tolerance. After gastric surgery some patients experience malabsorption, which may be specific for macro- or micronutrients. Vitamin and mineral supplementation may be necessary, depending on the extent of surgery and whether the dumping syndrome symptoms persist (1).

How to Order the Diet

Order as "Dumping Syndrome Diet" or "Postgastrectomy Diet." One or more features of the diet may be individually ordered, eg, Sugar in Moderation Diet, 120 cc fluid ½ to 1 hour before or after meals, 5 to 6 small meals, Lactose-Controlled Diet, Low-Fiber Diet, or other strategies listed under Planning the Diet.

Planning the Diet (1,3,4)

- 1. Simple carbohydrate (lactose, sucrose, and dextrose) consumption is kept to a minimum to prevent the formation of a hypertonic solution and the subsequent osmotic symptoms, as well as to prevent late hypoglycemia. (See Section IC: Sugar in Moderation Diet.) Complex carbohydrates and gradual increase of high fiber may be included (3).
- 2. Taking liquids with meals is thought to hasten gastrointestinal transit. Drink liquids 30 to 60 minutes either before or after meals (3). Consume adequate amounts of liquid throughout the day in small amounts at a time (1,3,4). Carbonated beverages and milk are included based on individual tolerance.
- 3. Smaller, more frequent feedings (5 to 6 per day) are recommended to accommodate the reduced storage capacity of the stomach and to provide adequate nourishment.
- 4. Lactose, especially in milk or ice cream, may be poorly tolerated due to rapid transit time. Cheese and yogurt are often better tolerated. A Lactose-Controlled Diet may be beneficial if symptoms are related to a primary or secondary lactose deficiency. (See Section IH: Lactose-Controlled Diet.)
- 5. Include high proteins food sources with each meal. Increase fats as necessary to meet energy requirements. A higher protein intake and increased fat intake also delays gastric emptying.
- 6. Encouraging to eat slowly, chewing all foods thoroughly as well as sitting upright while eating may lessen symptoms (3).
- 7. If adequate caloric intake cannot be provided due to steatorrhea, use medium chain triglyceride products.

(See Section IC: Medium Chain Triglycerides.)

8. Fiber gradually integrated into the meal plan may also be beneficial in delaying gastric emptying (1,3) Choose high-fiber foods when possible including whole wheat bread, fruits, vegetables, and beans (pinto, black , brown or kidney) (3).

References

- 1. Gastrointestinal Disease. In: Mueller CM (ed). *The A.S.P.E.N. Adult Nutrition Support Core Curriculum* 2nd ed. Silver Spring Md: American Society of Enteral and Parenteral Nutrition. 2012 (page 429).
- 2. Harju E. Metabolic problems after gastric surgery. Int Surg. 1990; 75:27-35.
- 3. Radigan A. Post-gastrectomy: managing the nutrition fall-out. *Pract Gastroenterol.* 2004;XXVIII(6): 63.
- 4. Gastrointestinal Disease. In: *Nutrition Care Manual*. The Academy of Nutrition and Dietetics. Updated annually. Available at: www.nutritioncaremanual.org. Accessed January 16, 2013.

NUTRITION MANAGEMENT IN BARIATRIC SURGERY

Discussion

Severe obesity (also referred to as morbid obesity) is a chronic condition that is difficult to treat with traditional weight loss methods (1-4). Surgery to promote weight loss by restricting food intake or interrupting the normal digestive process is recognized by the medical community as an effective medical option for severely obese people when other weight management approaches have proved unsuccessful (1-4). Bariatric surgery promotes weight loss by introducing anatomical alterations that reduce the size of the gastric reservoir or cause malabsorption (1-3). The role of nutrition is paramount in bariatric surgery. Patients who undergo bariatric surgery require intensive nutrition intervention, routine nutrition evaluation, and close nutrition monitoring to produce optimal nutrition care and weight loss outcomes (1.4). Another critical element is the patients themselves who must be committed to make permanent and sustainable lifestyle changes while undergoing medical monitoring for life.

The American Association of Clinical Endocrinologists, the Obesity Society, and the American Society for Metabolic and Bariatric Surgery evaluated presurgical and postsurgical management strategies for obesity treatment and outlined consensus and evidence-based practice guidelines (1). Bariatric surgery remains an emerging field. The long-term health implications and nutritional consequences associated with rapid and substantial weight loss continue to require study to determine the best treatment and management options (1,4).

Indications for Bariatric Surgery

The following groups of patients meet the criteria for surgical intervention for weight loss if they (and their parents, in the case of pediatric or adolescent patients) are motivated and able to comply with a lifelong program (1-3):

- adults with a body mass index (BMI) greater than or equal to 40 kg/m² with no comorbid conditions or health risks and acceptable operative risk
- adults with a BMI of 35 kg/m² or greater with comorbid conditions or health risks such as cardiopulmonary problems, type 2 diabetes mellitus, or physical limitations
- adults whose weight is 100 lb greater than their ideal body weight and who have made multiple unsuccessful attempts to lose weight with non-surgical methods
- children and adolescents who are above the 95th percentile of weight for age and have a severe comorbidity (1) (These bariatric procedures should be performed in specialized centers.)

The most common types of bariatric surgery are laparoscopic adjustable gastric banding and Roux-en-Y gastric bypass (1). Although risks and benefits are associated with both approaches, laparoscopic bariatric procedures are preferred to open procedures if sufficient surgical expertise exists (1). The best choice for any bariatric procedure should be based on the expertise of the surgeon or institution, patient preferences, and patient risk stratification (1). Only the laparoscopic adjustable gastric band and Roux-en-Y gastric bypass should be offered to children and adolescents (1). Restrictive procedures, such as the laparoscopic adjustable gastric band, may be combined with modified gastric bypass procedures, such as the Roux-en-Y gastric bypass, to further limit the absorption of energy or nutrients. An investigational procedure known as a firststage sleeve gastrectomy may be performed in high-risk patients to induce initial weight loss (25 to 45 kg), with the possibility of then performing a second-stage Roux-en-Y gastric bypass or a more advanced gastric bypass surgery (1). Although anatomical changes are made during both types of surgeries, the patient who does not strictly adhere to the volume restrictions and types of food allowed can negatively affect the outcomes of the procedure (1). Consuming too much food can stretch the surgical pouch (pouch dilation), and eating high-energy, low-nutrient quality foods can compromise the amount and rate of weight loss, thus defeating the primary goal of the surgery. As in other treatments of obesity, successful results depend mainly on long-term motivation, adherence, and behavior modification strategies employed by the patient (1-3).

Types of Bariatric Surgery

A variety of procedures are included under the umbrella term of bariatric surgery. These operations are categorized as either restrictive or malabsorptive (1-3). The following discussion addresses both categories of operations and the associated nutrition implications and problems. Nutrition interventions, including progression of meal planning, should be customized based on the type of procedure (1-3). A detailed overview of the bariatric diet, nutrition interventions, and meal planning approaches is provided later in this section.

Restrictive operations: Restrictive operations limit the amount of food that the stomach can hold by closing off or removing parts of the stomach. These operations also delay the emptying of the stomach (gastric pouch). Regurgitation is a common side effect of these procedures. The types of restrictive surgical procedures include (1):

- Laparoscopic adjustable gastric banding: A band made of special material is placed around the stomach near its upper end, creating a small pouch and a narrow passage into the larger remainder of the stomach. Necessary adjustments to the band are made during follow-up procedures (1) Laparoscopic adjustable gastric banding has vielded greater success in achieving weight loss outcomes than verticalbanded gastroplasty or adjustable silastic gastric banding (4).
- Vertical-banded gastroplasty: Both a band and staples are used to create a small stomach pouch similar to the pouch formed in laparoscopic adjustable gastric banding.
- Adjustable silastic gastric banding: Approved in 2001 for use in the United States, this operation is functionally similar to vertical-banded gastroplasty. This procedure makes a small pouch out of the upper stomach. The pouch is stapled off from the rest of the stomach except for a small opening, which is then reinforced with a ring made of Silastic, a soft and rubbery but strong material.
- **Sleeve gastrectomy or vertical sleeve gastrectomy:** This is a newer procedure in which the stomach capacity is restricted by stapling and dividing it vertically and removing more than 85% of the stomach. This part of the procedure is not reversible. The stomach that remains is shaped like a very slim banana and has a capacity of 1 to 5 oz (30 to 150 cc).

In addition to these procedures, another procedure performed less often is the silastic ring gastroplasty (1). Laparoscopic adjustable gastric banding has largely replaced vertical banded gastroplasty (1). Restrictive operations are the surgeries most commonly prescribed by bariatric surgeons (1).

Restrictive operations do not interfere with the normal digestive process (1). Rather, these procedures restrict food intake through the creation of a small pouch at the top of the stomach where the food enters from the esophagus. The pouch initially holds about 1 to 2 oz of food, but it expands to a 4- to 8-oz capacity over a 6- to 9-month period. The pouch's lower outlet has a diameter of approximately ¹/₄ inch. The small outlet delays the emptying of food from the pouch and causes a feeling of fullness. Although restrictive operations lead to weight loss in almost all patients, some patients do regain weight (1). Six to 8 weeks after a restrictive operation, the patient usually can eat ½ to 1 cup of food without discomfort or nausea. Food has to be well chewed and consumed slowly. Most patients are unable to eat a large amount of food at one time, but some individuals return to eating modest amounts of food without feeling hungry. A common risk of restrictive operations is vomiting (1.4). This occurs when insufficiently chewed food particles overly stretch the small stomach. Documented risks of vertical-banded gastroplasty include erosion of the band, breakdown of the staple line, and, in a small number of cases, leakage of stomach contents into the abdomen. This leakage requires an emergency operation. In less than 1% of cases, infection or death due to complications may occur (3). Because of the low-energy content and small volume of food consumed following a restrictive procedure, long-term multivitamin and mineral supplementation is required to meet the dietary reference intakes (DRIs) for most nutrients (1).

Malabsorptive operations: Malabsorptive operations are also referred to as gastric bypass operations. In these procedures, a surgeon makes a direct connection from the stomach to a lower segment of the small intestine, bypassing the gastric fundus, body, antrum, duodenum, and a variable length of proximal jejunum. Because gastric bypass operations cause both malabsorption and restricted food intake, these operations produce more weight loss than restrictive operations. Patients who have bypass operations generally lose two thirds of their excess weight within 2 years (2,3). The risks of pouch stretching, band erosion, breakdown of staple lines, and leakage of stomach contents into the abdomen are about the same as the risks in verticalbanded gastroplasty (3). Types of malabsorptive or gastric bypass operations include (1):

- **Roux-en-Y gastric bypass**: This operation is the most common gastric bypass procedure (1). First, to restrict food intake, a small stomach pouch is created by stapling or vertical banding. Next, a Y-shaped section of the small intestine is attached to the pouch to allow food to bypass the duodenum (the first segment of the small intestine) and the first portion of the jejunum (the second segment of the small intestine). This step of the procedure causes reduced absorption of energy and critical nutrients (1).
- Biliopancreatic diversion and biliopancreatic diversion with duodenal switch: In the biliopancreatic diversion operation, portions of the stomach are removed. The small pouch that remains Manual of Clinical Nutrition Management

is connected directly to the final segment of the small intestine, thus completely bypassing both the duodenum and jejunum. In the biliopancreatic diversion with duodenal switch, the distal stomach, duodenum, and entire jejunum are bypassed, leaving only a 50-cm distal ileum common channel for nutrients to mix with pancreatic and biliary secretions. This procedure has a substantial increase in the risk of nutritional and metabolic complications (1,3). Although this procedure successfully promotes weight loss, it is not commonly used because of the high risk of nutritional deficiencies, especially with the biliopancreatic diversion with duodenal switch (1).

Because gastric bypass operations cause food to bypass the duodenum, where most iron and calcium are absorbed, the risks of nutritional deficiencies are higher in these procedures (1,3,5,6). Iron deficiency is common secondary to the lack of contact of food iron with gastric acid and the consequently reduced conversion of iron from the relatively insoluble ferrous form to the more absorbable ferric form (1). Vitamin B_{12} deficiency may result from the lack of contact between food and the gastric intrinsic factors, decreases in acid and pepsin digestion of protein-bound cobalamins from foods, and the incomplete release of vitamin B_{12} from R binders (1). Vitamin D and calcium absorption may also be reduced since the duodenum and proximal jejunum, which are the preferential sites of absorption, are bypassed by this procedure (1). Anemia may result from malabsorption of vitamin B_{12} and iron in menstruating women, and decreased absorption of calcium may lead to the development of osteoporosis and metabolic bone disease (1,3,5,6). Shikora (7) studied the nutritional consequences of gastric bypass surgery and found deficiencies in vitamin B_{12} (26% to 70% of patients), folate (33% of patients), vitamin A (10% of patients), potassium (56% of patients), and magnesium (34% of patients). Eatients are required to take nutritional supplements that usually prevent these deficiencies. Lifelong supplements of multivitamins, vitamin B_{12} , iron, and calcium are mandatory following this procedure (1,5).

Gastric bypass operations often cause dumping syndrome (1). Dumping syndrome occurs when stomach contents move too rapidly through to the remaining small intestine (1). Symptoms include tachycardia, weakness, sweating, and abdominal pain that usually occur immediately after eating. Diarrhea may also occur, especially if the patient eats concentrated sweets. Patients with dumping syndrome will need to lie down until the symptoms pass. Refer to Section IB: Dumping Syndrome Diet for appropriate medical nutrition therapy intervention and treatment.

Rationale

The Bariatric Diet meal plan is for severely obese patients specifically being treated for weight management. The primary outcome of the diet approach is to compliment the surgical procedure by promoting substantial weight loss through reductions in food volume and energy intake. This diet is not intended for use with other types of gastric surgery, such as gastrectomy, that are used as the primary treatment for other conditions or diseases such as cancer of the gastrointestinal tract, peptic ulcer disease, or trauma. Refer to Section IB: Dumping Syndrome Diet or other transitional diets as needed.

Nutritional Adequacy

During the first 6 weeks after surgery, energy, protein, vitamin, and mineral needs are difficult to meet. However, the combination of diet and multivitamin and mineral supplementation can be planned to meet the DRIs as outlined in Section IA: Statement on Nutritional Adequacy. The adequacy of the diet will depend on the type and extent of surgery and on the postoperative progression of food based on the individual's tolerance. Up to 3 months after surgery, deficiencies in proteins, vitamins, and minerals may occur (1). Due to the small volume of food in the Bariatric Diet, vitamin and mineral supplementation is necessary to meet specific vitamin and mineral needs (1). Two to 3 days after surgery, patients should begin a multivitamin and mineral supplement regimen to meet 100% of the DRIs, including iron, B-complex vitamins (B₁₂, folate), and 1,200 to 2,000 mg of calcium (1). Additional supplementation is often required for patients who have had Roux-en-Y gastric bypass or biliopancreatic diversion with duodenal switch (1). The vitamin and mineral regimen should be consumed daily and considered necessary for lifelong maintenance of nutritional health (1,5). Chewable forms of supplements may be better tolerated in the initial stages after surgery (1-5). Refer to the discussion of vitamins and minerals later in this section under "Medical Nutrition Therapy and Nutrition Intervention After Bariatric Surgery" and "Strategies for Vitamin and Mineral Supplementation Following Bariatric Surgery".

How to Order the Diet

Order as "Bariatric Diet." One or more features of the diet may be individually ordered based on the postoperative stage, for example, Bariatric Clear Liquids, Bariatric Full Liquids, Bariatric Pureed, or Bariatric

Soft. Other modifications may be needed to promote individual tolerance and weight loss, such as Lactose-Controlled Diet, Sugar in Moderation Diet, Low-Fiber Diet, Low-Fat Diet, or other strategies discussed in Planning the Diet.

Planning the Diet

Limited scientific evidence is available to support specific guidelines and strategies for nutrition intervention following bariatric surgery. The existing guidelines are based on emerging evidence from bariatric centers and institutions that specifically provide bariatric surgical procedures and long-term obesity management (1). The guidelines are based on scientific evidence and identified nutrition intervention strategies that are effective in managing postsurgical bariatric surgery patients. "Bariatric Diet" is a general term for the overall diet approach applied to all patients following a bariatric surgery. Meal planning should be customized based on the type of procedure and the individual patient's needs. In addition, the clinician can refer to the summary by Mechanick et al for specific meal progression plans for laparoscopic adjustable gastric banding, Roux-en-Y gastric bypass, and biliopancreatic diversion gastric bypass (1). The Bariatric Diet meal plan incorporates the following nutritional guidelines (1).

Energy requirements: Total energy requirements are based on the postoperative stage, progression of meal plan, and volume of food tolerated. Ensuring nutrient quality is the primary goal when designing the meal plan.

- Protein should provide at least 25% of the total energy intake (or 60 to 120 g/day of protein or 1.5 g/kg of ideal body weight) to minimize lean muscle loss during rapid weight loss (1). Modular protein supplements may be required during the first 6 postoperative months until solid food intake is sufficient to meet the protein requirements (1).
- Fats should provide 25% to 30% of total energy. Small amounts of dietary fat, along with prescribed medication, can help maintain gallbladder emptying and reduce the risk of gallstone formation (5).
- Carbohydrates should provide approximately 50% of the daily energy intake. The intake of concentrated sugars should be limited for gastric surgery patients. Concentrated sweets or sugary foods should be avoided after Roux-en-Y gastric bypass to minimize the symptoms of dumping syndrome or after any bariatric procedure to reduce the energy intake (1). Foods that provide low-quality nutrients with high amounts of energy and fat may compromise the primary goal of promoting weight loss.
- After a diet of full liquids or semisolid food begins (usually 2 to 3 days postoperatively), initiate a chewable multivitamin and mineral supplement regimen (one or two supplements per day) that provides 100% of the DRIs for age and sex. Additional specialized supplementation may be required for iron, calcium (1,200 to 2,000 mg/day), vitamin B₁₂, folate, or other vitamins and minerals as indicated by laboratory assessment and the ability to consume food sources (1). Patients who have extensive malabsorption will have even higher supplementation requirements for vitamin D, calcium, and vitamin B₁₂ and possibly thiamin, copper, and vitamin A (1,5).
- Initially, the meal plan should provide multiple small meals each day, with the focus of chewing food thoroughly without drinking beverages at the same time; beverages should be consumed more than 30 minutes before or after solid foods (1,5).

Volume and consistency: The volume and consistency of foods provided depend on the postoperative stage and individual tolerance. Initially, the stomach can hold only 1 to 2 oz (2 to 4 tbsp). Over time, the stomach pouch will stretch until it can hold 4 to 8 oz (½ to 1 cup).

Fiber: High-fiber foods may not be initially tolerated. Fiber should be gradually introduced based on the patient's progress toward the consumption of solid foods and a regular diet (usually more than 6 to 8 weeks after surgery). Patients should adhere to a balanced meal plan that includes more than five daily servings of fruits and vegetables for optimal fiber consumption, colonic function, and phytochemical consumption (1). Anecdotal evidence suggests that bulky foods, such as bran, popcorn, raw vegetables, and dried beans, should be avoided or limited based on individual tolerance or until the diet progresses and tolerance of these foods is verified.

Fluid: Adequate consumption of fluids is essential to prevent dehydration. A minimum of 1.5 L or 6 cups (48 oz) should be consumed each day (1). When the patient is able to consume pureed or solid foods, fluids should be consumed at least 30 minutes before or after meals to prevent nausea and vomiting (1). Fluids should consist of water or controlled-energy (low-sugar, low-fat, or diet) beverages. Caffeine-containing beverages

and carbonated beverages may need to be limited based on individual tolerance.

Outcomes of Bariatric Surgery

Weight loss usually reaches a maximum between 18 and 24 months after bariatric surgery (1). More than 90% of patients experience substantial (21% to 25%) weight loss, and between 50% and 80% of patients maintain the weight loss for more than 5 years. In contrast, the 5-year efficacy of other weight loss approaches is approximately 5% (2). Mean percent excess weight loss at 5 years ranges from 48% to 74% after Roux-en-Y gastric bypass, from 50% to 60% after vertical-banded gastroplasty, and 50% following laparoscopic adjustable gastric banding (1). Bariatric surgery improves several comorbid conditions, such as glucose intolerance, diabetes mellitus, sleep apnea, hypertension, and serum lipid abnormalities (1). A 10-year follow-up study demonstrated that the excess weight loss of bariatric surgery patients remained within 14% to 25% of the baseline weight loss (8). Adams et al demonstrated that after 7.1 years, the adjusted long-term mortality decreased by 40% in patients managed with bariatric surgery (9).

The immediate operative mortality rates for the laparoscopic adjustable gastric banding procedure and the Roux-en-Y gastric bypass are relatively low (8-10). Morbidity in the early postoperative period following Roux-en-Y gastric bypass (eg, wound infections, dehiscence, leaks from staple breakdown, stomal stenosis, marginal ulcers, pulmonary problems, and deep thrombophlebitis) may be as high as 10% or more (1). However, the aggregate risk of the most serious complications of gastrointestinal leakage and deep venous thrombosis is less than 1% (1.8-10).

Medical Nutrition Therapy Approaches in Bariatric Surgery

The risks of complications and nutritional deficiencies increase as the extensiveness of the bypass operation increases (1). Patients with extensive bypasses of the normal digestive process require not only close monitoring but also the lifelong use of special foods and vitamin and mineral supplementation (1,5). Before any type of bariatric surgery, patients should have a comprehensive multidisciplinary screening, nutrition assessment, and nutrition education to address the long-term plans for postoperative nutrition care and weight loss strategies (1). Each individual should clearly understand the proposed operation. Bariatric surgery is a serious undertaking. Patients, physicians, psychologists, and dietitians should together carefully consider the benefits and risks during the nutrition assessment and evaluation period (1,2,4).

Benefits:

- Immediately after surgery, most patients lose weight rapidly, and they continue to lose weight for 18 to 24 months (1). Although most patients then start to regain some of their lost weight, few patients regain it all (1-3).
- Surgery improves most obesity-related conditions. Blood glucose levels return to normal after surgery in 65% of obese patients aged 45 to 71 years old with type 2 diabetes mellitus (6). The American Diabetes Association recognizes bariatric surgery as a viable option for patients with type 2 diabetes mellitus and a BMI of at least 35 kg/m² who have poor control of symptoms. Patients also usually experience lower blood pressure and lower serum cholesterol levels postoperatively (1).

Risks:

- Of patients who have weight-loss operations, 10% to 20% require follow-up operations to correct complications (2,3). Abdominal hernias are the most common complications that require follow-up surgery. Less common complications include breakdown of the staple line and stretched stomach outlets (3).
- Gallstones develop in more than one third of obese patients who have gastric surgery (2,11,12). Gallstones are clumps of cholesterol and other matter that form in the gallbladder. During rapid or substantial weight loss, the risk of developing gallstones is increased. Gallstones can be prevented by taking supplemental bile salts such as ursodiol for the first 6 months after surgery (1,2). In addition, consuming dietary fat (approximately 30% of total energy or 10 g of fat per meal) can help maintain gallbladder emptying and decrease the risk of gallstone formation (1,5).
- Nearly 30% of patients who have bariatric surgery develop nutritional deficiencies such as anemia, osteoporosis, and metabolic bone disease (1,3,6,7). These deficiencies can be avoided if adequate vitamin and mineral intakes are maintained through lifelong supplementation.
- Women of childbearing age should avoid pregnancy for at least 12 months perioperatively and until their weight stabilizes, because rapid weight loss and nutritional deficiencies can harm a developing fetus (1). Women who become pregnant after these surgical procedures need specific attention from the surgical care team. There are several reports in the literature of pregnancy outcomes following gastric bypass without evidence of fetal impairment (1,13).
Medical Nutrition Therapy and Nutrition Intervention After Bariatric Surgery

Guidelines are based on scientific evidence and emerging evidence from bariatric centers and institutions that specifically provide management and treatment for gastric bypass patients (1). The meal plan must be individualized based on the type of surgery performed, postoperative stage, and individual tolerance to volume and consistency of food. The dietitian should closely monitor patients for nutrition-related signs and symptoms that may indicate vitamin, mineral, or protein deficiencies; meal planning problems; and problems meeting weight loss goals (1,5). Nutrition diagnoses are common in patients following any gastric bypass surgery due to the malabsorptive process used to achieve weight loss.

Energy requirements: An objective estimation of the required total energy is difficult due to the rapid weight loss and rapid changes in the ratio of fat-free mass to fat mass. One study evaluated changes in the measured resting energy expenditure after Roux-en-Y gastric bypass for severe obesity. The measured resting energy expenditure was significantly less than the Harris-Benedict-predicted resting energy expenditure before the operation, but it increased to the predicted value by 6 weeks postoperatively and remained so during the 24-month evaluation period (14). The measured resting energy expenditure less than 15% the Harris-Benedict-predicted resting energy expenditure less than 15% the Harris-Benedict-predicted resting energy expenditure) increased significantly despite reductions in energy intake (14). Daily energy intake was approximately 2,603 kcal before surgery for all subjects and fell to an average of 815 kcal at 3 months, 969 kcal at 6 months, 1,095 kcal at 12 months, 1,259 kcal at 18 months, and 1,373 kcal at 24 months postoperatively (14). Total energy requirements of the Bariatric Diet will not meet predicted or measured energy requirements in most cases and will depend on the postoperative stage and volume of foods consumed by the patient. The goal of the diet is energy deficit to promote substantial weight loss. As energy intake increases, the rate of weight loss generally decreases or plateaus (1).

Protein requirements: Most programs recommend that at least 25% of the total energy intake should be from protein to minimize lean muscle loss during rapid weight loss, build new tissue after surgery, and maintain lean muscle tissue long term (1). During energy restriction, patients should consume 60 to 120 g/day of high-quality protein (1,15) or 1.5 g/kg of ideal body weight (5). Protein malnutrition is common in biliopancreatic diversion surgeries and increases to 17.8% if the pouch volume is less than 200 mL (5). Patients with biliopancreatic diversion or biliopancreatic diversion with duodenal switch procedures require the higher end of the protein range with a minimum consumption of 80 g/day (1). Patients who ingest too little protein (<40 g/day) or protein that is mostly low biological quality are at risk of developing ventricular arrhythmias (16). Consuming adequate sources of high-biological value protein at each meal and snack is suggested. One to 2 weeks after surgery, high-protein liquids that provide at least 15 g of protein per 8-oz serving with less than 20 g of total carbohydrate and less than 5 g of fat are suggested (5). Foods such as regular (not reduced sugar) Carnation Instant Breakfast[™], Ensure[™], Slim Fast[™], and Boost[™] may not meet these criteria and should be avoided (5). Once solid foods are tolerated (generally 4 to 6 weeks after surgery), foods that are low in fat and high in protein, such as lean red meat or pork, chicken, or turkey without the skin, fish, eggs, and cottage cheese, are good protein sources. Modular protein supplements may be required during the first 6 postoperative months until solid food intake is sufficient to meet the requirements, especially since meat and dairy products are some of the most frequently reported food intolerances after surgery (17). The protein digestibility corrected amino acid score for the evaluation of protein quality should be assessed for patients who are dependent on supplements for a large percentage of their protein intake. This score reflects the overall quality of a protein, because it represents the relative adequacy of the most limiting amino acid (18). The practitioner must review the amino acid composition of the patient's selected commercial protein-products to ensure that they include adequate amounts of all limiting amino acids (18). Patients should be carefully monitored and evaluated for symptoms of protein deficiency and protein-energy malnutrition. Hair loss is an indicator of inadequate protein intake and can be a side effect of gastric bypass procedures (1). (Refer to the discussion of Medical Complications and Nutrition Evaluation and Monitoring Following Bariatric Surgery for additional information.)

Fat: Fat should provide approximately 25% to 30% of total energy. Fat may be difficult to digest or tolerate after gastric bypass surgery, especially fried foods and snack foods. Steatorrhea is often a complication of the biliopancreatic diversion and biliopancreatic diversion with duodenal switch procedures. Too much fat can delay gastric emptying and cause reflux leading to heartburn. After bariatric surgery, all patients are at increased risk for gallstone formation due to the rapid weight loss (1). Small amounts of dietary fat consumed

at each meal (approximately 10 g per meal) can be helpful in maintaining gallbladder emptying and preventing gallstone formation (5,11,12). Gallstones can also be prevented with supplemental bile salts (eg, ursodiol orally administered 300 mg twice per day) taken for the first 6 months after surgery (1). Long-term, a prudent low-fat diet following the National Cholesterol Education Program Adult Treatment III guidelines should be advocated to sustain weight loss and reduce risk factors for other comorbid conditions (1).

Carbohydrate: Carbohydrates should provide approximately 45% to 50% of total energy. Carbohydrates help to prevent the loss of lean tissue (15). While carbohydrate intake does not have to be high, it is suggested that energy-restricted diets contain more than 100 g/day of carbohydrate to minimize ketosis (15). Hyperuricemia can also result from weight loss, particularly with the use of a low-carbohydrate diet. Ketone bodies, products of fat oxidation in the energy-restricted patient, compete with urate for tubular reabsorption in the kidney, resulting in increased uric acid levels and an increased risk of gout. Increasing the carbohydrate content of the diet will reduce the risk of increasing uric acid levels (19). Sugar intake should be limited; sugars are generally not tolerated and can cause symptoms associated with the dumping syndrome (1). In addition, sugary foods often provide low-quality nutrients and high amounts of energy and fat. Foods to avoid or limit include candy, cookies, ice cream, milkshakes, slushes, soda pop, sweetened juices or gelatin, and most desserts.

Fiber: Anecdotal evidence suggests that high-fiber foods are generally not tolerated and should be avoided until progression to regular foods has occurred (usually >6 to 8 weeks postoperatively). Bulky foods, such as bran, popcorn, raw vegetables, and dried beans, may need to be avoided until individual patient tolerance is verified. It is thought that the newly created surgical pouch does not have the capacity to hold many of these foods. In addition, gastric acid is reduced and may not be as readily available to help digest fibrous foods. However, more recent guidelines recommend that patients should be advised to adhere to a balanced meal plan that contains more than five servings of fruits and vegetables daily for optimal fiber consumption, colonic function, and phytochemical consumption (1).

Vitamins and minerals: Because the Bariatric Diet allows only a small amount of foods and limits the types and variety of foods, vitamin and mineral supplementation is necessary. Immediately after a full liquid or semisolid food diet begins (usually 2 or 3 days after surgery), a patient should begin supplementation with a chewable multivitamin and mineral supplement that provides 100% of the appropriate DRIs for the patient's age and sex (1). For gastric bypass procedures that cause malabsorption (eg, Roux-en-Y, biliopancreatic diversion, and biliopancreatic diversion with duodenal switch), additional supplementation may be required for key nutrients whose absorption is impacted, predominately iron, folate, vitamin B₁₂, and calcium (1), or other vitamins and minerals as indicated by routine laboratory assessment. The Roux-en-Y and biliopancreatic diversion procedures have a greater impact on nutrient absorption because of the anatomical alterations and the impact on gastric acidity (1,17,20). Because these procedures can lead to metabolic bone disease, routine diagnostic testing is recommended so that the appropriate intervention can be delivered (1). For patients who have Roux-en-Y or biliopancreatic diversion procedures, vitamin and mineral supplement regimens that contain higher doses of iron, calcium, vitamin B_{12} , and folate are often indicated (17,20). Intakes of 40 to 65 mg of elemental iron (1,17) and 800 to 1,000 μ g of folate per day have been recommended (17,21). An average daily dose of 350 μ g of sublingual vitamin B₁₂ maintains adequate stores (1). Although this dose is 175 times the DRI, a small percentage of patients will still become vitamin B_{12} deficient and require monthly intramuscular injections (1,17,20,21). Calcium supplements of 1,200 to 2,000 mg/day plus 400 to 800 IU/day of vitamin D should be provided to all patients following bariatric surgery (1,17). Calcium citrate with vitamin D is the preferred preparation because it is more soluble than calcium carbonate in the absence of gastric acid production (1,21,22). Calcium should be divided into doses of no more than 500 mg throughout the day (17). For patients with the biliopancreatic diversion procedure who have clinical steatorrhea, a high-dose calcium supplementation regimen (2,000 mg/day) and a monthly intramuscular vitamin D injection is recommended to reduce the risk of metabolic bone disease (17). Patients who have the biliopancreatic diversion procedure must also take supplements of fat-soluble vitamins A, D, E, and K, if clinically indicated (5). (Refer to Table B-1 (1).) In addition, the summary by Mechanick et al outlines recommended medical testing and routine evaluation and monitoring for nutritional deficiencies that may occur specific to malabsorptive bariatric surgical procedures such as Roux-en-Y gastric bypass, biliopancreatic diversion, or biliopancreatic diversion with duodenal switch (1). Signs and symptoms of nutritional deficiencies should be routinely evaluated and monitored postoperatively to determine if additional vitamin or mineral supplementation is necessary (1).

11	
Supplement	Dosage
Multivitamin	One to two daily
Calcium citrate with vitamin D	1,200-2,000 mg/day of calcium plus 400-800 IU/day of vitamin D
Folic acid	400 mcg in multivitamin
Elemental iron with vitamin D ^b	40-65 mg/day
Vitamin B ₁₂	>350 mcg/day orally
	or 1,000 mcg/month intramuscularly
	or 3,000 mcg every 6 months intramuscularly
	or 500 mcg/week intranasally

Table B-1: Routine	Nutrient Sunn	lementation After	Bariatric Surgerv ^a
I abic D I. Routine	nutiting Supp		

^aPatients with preoperative or postoperative biochemical deficiencies require additional supplementation (1). ^bFor menstruating women

Adapted from: Mechanick JI, Kushner RF, Sugerman HJ, Gonzalez-Campoy JM, Collazo-Clavell ML, Guven S, Spitz AF, Apovian CM, Livingston EH, Brolin R, Sarwer DB, Anderson WA, Dixon J. Executive summary of the recommendations of the American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic & Bariatric Surgery medical guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient. *Endocr Pract.* 2008;14:331.

Studies have found a significant correlation between eating habits (quality of food consumed) and laboratory values (1,15,20). In a study of patients who received a Roux-en-Y gastric bypass, the mean levels of serum iron saturation, vitamin B₁₂, and folic acid were significantly higher in patients who ate meat than in patients who did not eat meat (15). Iron status continued to decline 6 to 8 years after surgery, depending on eating behavior (15). Oral vitamin and mineral supplementation significantly improved the nutritional status of the study patients (15). Patients who have biliopancreatic diversion or biliopancreatic diversion with duodenal switch procedures should be evaluated frequently to assess benefits or improvements in nutritional outcomes following bariatric surgery (1).

Fluids: Adequate consumption of fluids is essential to prevent dehydration. A minimum of 6 cups (48 oz) should be consumed per day, with a goal of consuming 64 oz/day (1,5,20). Patients should initially consume 2 to 3 oz at a time and gradually increase their fluid intake to 3 to 4 oz at a time 8 weeks after surgery (1,5,20). Once pureed or solid foods are introduced into the diet, fluids should be consumed at least 30 minutes before meals and should consist of water or energy-controlled (low-sugar, low-fat, or diet) beverages. The patient should delay drinking beverages with food; rather, the patient should wait at least 30 minutes after meals to prevent increasing the transit time of food through the pouch, which may lead to nausea and vomiting (1). After 6 to 9 months, most patients can tolerate drinking fluids with meals. Intake of caffeine-containing beverages and carbonated beverages should be individualized according to the patient's tolerance.

Volume, consistency, and timing: The volume and consistency of foods depend on the postoperative stage and individual tolerance. Initially after gastric bypass surgery, the stomach can hold only 1 to 2 oz (2 to 4 tbsp). Over time, the stomach pouch will stretch until it can hold 4 to 8 oz (or about ½ to 1 cup). The Bariatric Diet progresses in stages from clear liquids (1 to 2 days) to full liquids and pureed foods (4 to 6 weeks) and then to soft and regular foods (6 to 8 weeks). The timing of progression varies among patients, so it is appropriate for individuals to adjust their own progress depending on how they feel. Four to six small meals per day may be better tolerated long term; however, coordinating the meals with fluid intake may be challenging and should be individualized (5). After the pouch matures (6 months), most food consistencies can be tolerated. (Refer to Table B-2.)

Food intolerances: Patient reports have indicated common postoperative intolerances to specific foods (23-25). Red meat, milk, and high-fiber foods are among the foods most commonly reported as not well tolerated. One study found meat intolerance in 51% of patients during postoperative months 0 to 12; 60.3% of patients at 13 to 24 months; 59.5% of patients at 25 to 72 months; and 55.1% of patients at 73 to 96 months (15). Soft breads are often not tolerated. However, crispy breads and crackers (eg, well-toasted breads, Melba toast, and low-fat crackers) are generally better tolerated. Milk intolerance may be caused by an intolerance to fat or a secondary lactose deficiency related to the surgical procedure (1,5,23-25). Fat-free milk in small amounts is suggested to improve tolerance. Individual meal planning should be accommodated to meet nutrient-specific needs if certain foods are not tolerated.

Postoperative Bariatric Diet Meal Planning

The following meal planning and behavior modification recommendations should be given to the patient after surgery (1). The meal plan must be individualized based on the type of procedure performed, postoperative stage, and individual tolerance to food volume and consistency (1,5,). The following stages and meal patterns are based on a review of bariatric surgery programs and published practice guidelines (1,5,23-25).

Stage 1 (postoperative days 1 and 2)

Days 1 to 2: Bariatric Clear Liquids

- Provide six to eight small feedings of clear liquid foods. Begin with sips of water, then add bouillon or clear broth, unsweetened juices, diet gelatin, and flat (no fizz) diet soda. In general, avoid carbonated beverages.
- Portion: Sip 2 to 3 oz (1 to 2 tbsp) at a time.
- Water: Sip 2 to 3 oz at a time throughout the day.
- The combined volume of six to eight small feedings and the water intake should be at least 48 to 64 oz/day (6 to 8 cups) to meet hydration needs.
- If the patient tolerates Bariatric Clear Liquids well, progress to Bariatric Full Liquids and then Bariatric Pureed/Soft Diet (see below).

Stage 2 (postoperative day 3 and discharge diet)

Days 3 to 4: Bariatric Full Liquids

- Provide six to eight small feedings per day. Begin with high-protein liquids such as diet instant breakfast (using fat-free milk), Glucerna[™], or specialized high-protein (low-fat, sugar-free, or <15 g of sugar per serving) drinks. Gradually increase intake to 60 to 80 g/day of protein (1,15). May also use nonfat milk with whey or soy protein powder (limit to 20 g of protein per serving), Lactaid milk or soy milk with soy protein powder, light yogurt (blended), or plain nonfat yogurt (1).
- Portion: Sip 2 to 3 oz (1 to 2 tbsp) at a time. May increase to 4 oz (6 to 8 tbsp) by week 2.
- Water: Sip 2 to 3 oz at a time throughout the day.
- The combined volume of the six to eight small feedings plus water intake should be at least 48 to 64 oz/day (6-8 cups) to meet hydration needs.
- Begin a chewable multivitamin and mineral supplement (usually two doses per day) to meet 100% of the DRIs for age and sex. Also begin chewable or liquid calcium citrate with vitamin D (1). (See Table B-1.)
- A full-liquid menu plan usually lasts 1 to 2 weeks postoperatively.

Stage 3 (weeks 2 to 6)

Weeks 2 to 5: Bariatric Pureed/Soft Diet

- Provide four to six small feedings of pureed or soft semisolid foods. Use high-quality protein foods such as scrambled eggs, Egg Beaters, low-fat cheese or cottage cheese, or blenderized lean meats such as tuna fish, chicken, or pork. Strained baby foods are a convenient option. Integrate a high-quality protein food with each meal or snack (1,5,23-25). (Refer to Table B-2.)
- Portion: 2 to 4 oz (4 to 8 tbsp) at a time of solid foods.
- Avoid rice, bread, and pasta until the patient is comfortably consuming 60 g of protein per day plus fruits and vegetables (1).
- Consume protein food first, vegetables and fruit second, and starch foods last to help ensure adequate protein consumption (5).
- Alternate fluid intake with food intake. Avoid consuming fluids with meals. Wait at least 30 minutes after consumption of solid foods or meals to drink fluids (1). The combination of water with controlled-energy beverages and milk should equal at least 6 cups/day (48 oz/day).
- Avoid alcohol, as it may lead to dehydration and does not provide necessary nutrients (5).
- Avoid chewing gum. If swallowed, gum can block the stomach opening (5).
- Avoid drinking from straws because the air swallowed can cause bloating and stretch the pouch (5).
- Behavior techniques need to be applied and reinforced (eg, eating small amounts, eating slowly, and chewing food completely before swallowing).
- At about 4 to 6 weeks, begin a gradual introduction of soft to regular consistency, low-fat, controlledenergy foods. The patient should keep a food record to document tolerance to foods to discuss with the dietitian during follow-up visits and to assess adherence to vitamin and mineral supplementation regimen.

Stage 4 (weeks 6 to 8)

Weeks 6 to 8: Bariatric Diet

- Provide four to six small feedings of regular-consistency food. Use high-quality protein foods such as scrambled eggs, Egg Beaters, low-fat cheese or cottage cheese, or lean meats such as tuna fish, chicken, or pork. (Refer to Table B-3 (1,5).)
- Portion: 4 to 6 oz (8 to 12 tbsp) at a time of solid foods.
- Alternate fluid intake with food intake. Consume fluids at least 30 minutes before or after solid foods. The combined volume of water, low-energy beverages, and milk should equal at least 6 cups/day, and at least half of the beverages should be a good source of protein (1).
- Avoid rice, bread, and pasta until the patient is comfortably consuming 60 g/day of protein plus fruits and vegetables (1).
- Consume protein food first, vegetables and fruit second, and starch foods last to help ensure adequate protein consumption (5).
- Patients should avoid alcohol, as it may lead to dehydration and does not provide necessary nutrients (5).
- Avoid chewing gum; if gum is swallowed, it can block the stomach opening (5).
- Behavior techniques need to be applied and reinforced (eg, eating small amounts, eating slowly, chewing food completely before swallowing, and eating from small plates).
- Avoid drinking from straws because the air swallowed can cause bloating and stretch the pouch (5).
- The patient should keep a food record to document food intake and eating behavior, including foods tolerated or not tolerated, to discuss with the dietitian during follow-up visits and to assess adherence to the multivitamin and mineral regimen.

Food	Choose	Avoid
Beverages	Crystal Light, decaffeinated coffee or tea, carbonation-free beverages, and high- quality nutrition drinks such as Glucerna [™] or no-sugar added diet instant breakfast_prepared with_fat-free milk	Fruit drinks, iced tea with sugar, alcohol, soda and other carbonated beverages with a high sugar content (>15 g of sugar per serving) (1)
Soup	Egg drop soup, reduced-fat cream soups, broth, bouillon, low-fat soups with added protein powder or strained meat	All others
Breads, cereals, rice, and pasta	Cooked, refined/strained cereals (with no added sugar) Soft diet: Add pasta, rice, toasted breads, Melba toast, and crackers. Choose items with <2 g fiber per serving.	All others
Vegetables	Pureed vegetables, vegetable juice <i>Soft diet:</i> Add soft cooked vegetables and mashed potatoes without skin.	All others Soft diet: vegetables with tough hulls or skins such as peas and corn
Fruits	Unsweetened applesauce, pureed banana or other fruit without seeds or hulls, unsweetened fruit juice <i>Soft diet:</i> Add soft unsweetened canned fruits or fresh soft fruits without skin or seeds.	All others

 Table B-2:
 Foods to Choose on a Pureed/Soft Diet Following Bariatric Surgery (5)

Food	Choose	Avoid
Meat, protein, and protein substitutes	Pureed fish, tuna, poultry, veal, pork, or beef; low-fat cottage cheese; low-fat or nonfat cheese; baby-food meats (all types); mashed or pureed tofu; pureed egg or egg substitute <i>Soft diet:</i> Add ground lean meat; fish or poultry; creamy or smooth peanut butter; casseroles made with ground meat and soft cooked vegetables; and chopped tofu.	Crunchy peanut butter, all others
Milk and milk products	 Fat-free or 1% milk; sugar-free or low-fat yogurt with live cultures (without fruit), soy milk (with no added sugar) or powdered milk; select lactose-free or soy milk if lactose intolerant. Soft diet: Add sugar-free or fat-free yogurt with fruit and low-fat, sugar-free frozen yogurt or ice cream. 	Chocolate milk, sweetened condensed milk, 2% or whole milk, ice cream
Others	Sugar-free gelatin or Popsicles; low-fat frozen yogurt, pudding, or custard	All others

Sample Menu: Stage 2: Bariatric Full Liquids (postoperative days 3 and
--

Time	Suggested Meal/Food
8 AM	¼ to ½ cup unsweetened fruit juice
9-9:30 ам	½ to ¾ cup fat-free milk with one package of diet instant breakfast
11 ам	¼ to ½ cup unsweetened fruit juice
12 рм	¹ / ₂ to ³ / ₄ cup strained low-fat cream soup
1-1:30 рм	½ to ¾ cup fat-free milk with one package of diet instant breakfast
2 рм	½ to 1 cup water
3-3:30 рм	$\frac{1}{4}$ to $\frac{1}{2}$ cup of fat-free, sugar-free yogurt (may try two or three saltine crackers or Melba toast)
5 рм	½ to 1 cup water
6-6:30 рм	¹ / ₂ to ³ / ₄ cup strained low-fat cream soup
7 РМ	$\frac{1}{2}$ to $\frac{3}{4}$ cup fat-free milk with one package of diet instant breakfast
8 PM	1/4 to 1/2 cup sugar-free, low-fat pudding (may try two or three saltine
	crackers or Melba toast)
9 рм	½ cup diet gelatin
Sample Menu: Stage 3: Bariatri	ic Pureed/Soft Diet (postoperative weeks 2 to 4)
Time	Suggested Meal/Food
8 AM	4 to 6 tbsp cooked refined cereal; add 2 tbsp fat-free milk to thin cereal.
9-9:30 ам	½ to 1 cup fat-free milk
11 ам	3 tbsp low-fat cottage cheese, 1 tbsp unsweetened applesauce
12 РМ	½ to 1 cup unsweetened fruit juice
1-1:30 рм	2 to 3 tbsp pureed ham, 2 tbsp mashed potatoes, 2 tbsp pureed broccoli
2 РМ	1 cup water
3-3:30 рм	$\frac{1}{2}$ to 1 cup of fat-free milk with one package of diet instant breakfast
5 pm	4 to 6 tbsp fat-free, sugar-free yogurt
6-6:30 рм	2 to 3 tbsp pureed chicken, 2 tbsp pureed carrots, 2 tbsp mashed potato with 1 tbsp fat-free gravy
7 РМ	½ to 1 cup fat-free milk
8 рм	Two slices of fat-free or low-fat cheese, 3 to 4 tbsp pureed or finely
	chopped canned pears (in natural juice)
9 рм	1 cup low-fat cream soup or lentil soup

Time	Suggested Meal
7:30 ам	½ cup orange juice
8:15 AM	Scrambled egg, one to two slices of toast, 2 tsp low-fat margarine
9:30 ам	1 cup fat-free milk with one package of diet instant breakfast
11 АМ	One slice of cheese melted over 2 tbsp cooked pasta, one small soft cooked broccoli flowerette
11:45 ам	½ to ¾ cup water
12:30 рм	3 tbsp flaked fish, 2 tbsp chopped cooked spinach, one pear half
1:30 рм	½ cup orange juice
2:30 рм	½ to ¾ cup fat-free, sugar-free yogurt
3 РМ	½ to ¾ cup water
4 PM	½ cup sugar-free, low-fat pudding
5:30 рм	1 cup fat-free milk
6:30 рм	4 tbsp diced chicken and rice casserole, 2 tbsp soft cooked green beans, one peach half (canned in natural juice)
8 pm	¹ / ₂ to ³ / ₄ cup water
8:30 рм	2 oz of tuna, 1 tsp low-fat or fat-free mayonnaise, four saltine crackers
9:30 рм	½ to ¾ cup water

Sample Menu: Stage 3: Bariatric Soft Diet (postoperative weeks 4 to 6)

building to the building bidding bidding the bidding of the bidding bi
--

Bumpie Menu Buge II Buriati	ie blee (postoperative weeks o a	
Breakfast	Lunch	Dinner
Banana—a quarter (medium)	Broiled Chicken Breast—2 oz	Haddock, Baked or Broiled—2 oz
Scrambled Egg—one	Carrots, Boiled—¼ cup	Green Beans—¼ cup
Toast, White—one half slice	Margarine—1 tsp	Dinner Roll—one half
Margarine—1 tsp	Pasta Salad—¼ cup	Margarine—1 tsp
Chewable Multiple Vitamin	Chewable Calcium Tablet	Chewable Multiple Vitamin
	(if prescribed)	

Morning Snack	Afternoon Snack	Evening Snack
Graham Crackers—two squares	Fruit Cocktail (water-packed)—	Cheese, American—1 oz
Pudding, Sugar-free (made with	½ cup	Saltine Crackers—two squares
fat-free milk)—½ cup		Mustard—1 tsp
		Chewable Calcium Tablet
		(if prescribed)

^aConsume fat-free milk between meals throughout the day. Drink approximately $\frac{1}{2}$ to $\frac{3}{4}$ cup at a time, for a daily total of 2 cups. Add protein powders to beverages to increase protein content as needed. Some patients may need protein supplementation to achieve a minimum of 80 g/day, especially those patients with a pouch volume less than 200 mL after biliopancreatic diversion or biliopancreatic diversion with duodenal switch (1).

Sam	ple	Menu:	Staae	4:	Reaular	Diet-	-Nutrient	Content	Breakdown
					- 0				

Energy	1056 kcal	Fat	42 g
Protein	71 g	Calcium	1065 mg
Carbohydrates	97 g	Iron	6 mg

Food Category	Foods Allowed	Foods That May Be Difficult to Tolerate	Foods to Limit for Best Weight Loss
Beverages	Water, tea, club soda, diet soft drinks, coffee, fat-free milk	Milk may not be tolerated except for fat-free milk in small amounts. Use low-fat or fat-free, sugar-free yogurt and cheeses for other sources of calcium.	High-energy drinks: whole milk, milk shakes, alcoholic beverages, sweetened fruit juices and drinks; avoid drinks with more than 15 g of sugar per serving.
Breads	Dry, coarse, or well- toasted bread; crispy crackers or baked tortilla chips	Soft breads that become gummy; breads with nuts, seeds, or dried fruit	Sweet breads, bagels, Danish pastries, and donuts
Cereals	All cooked and dried cereals without added sugar	Cereals containing dried fruit or nuts	Cereals with added sugar or providing more than 10- 15 g of sugar per serving
Potatoes, rice, pasta	Boiled, mashed, or baked potatoes (white or sweet potatoes); well- cooked pasta and rice	Potato skins, sweet potatoes, rice, noodles	None
Fruits	Fresh, canned, frozen, or cooked fruit; be cautious with apples, grapes, or other fruits with peel or skin.	Dried fruits; fruits with core, seeds, or skin; whole citrus fruits (eg, grapefruit, oranges); citrus fruits should be juiced.	Canned fruit in syrup
Vegetables	Fresh, canned, frozen, or cooked vegetables	Vegetables with tough skins or seeds	None
Meats	All if diced to ¼ inch (size of a pencil eraser) and well chewed	Tough meats or those with gristle; some patients do not tolerate red meat.	Bacon, sausage, lunch meats
Desserts	Frozen yogurt; low-fat gelatin, sherbet, fruit pies; low-fat, low-sugar pudding; sugar–free gelatin or Popsicles	Any dessert with nuts, dried fruit, seeds, or coconut; candy containing sugar, nuts, dried fruit, jams, or marmalade	All, except fresh fruit
Fats	All, in small amounts	Fried foods with a hard crusty coating	All
Miscellaneous		Soups with large pieces of meat; popcorn; nuts; chili and other highly spiced foods; avoid chewing gum.	Fried, salty snack foods; creamed soups unless made with nonfat milk

 Table B-3:
 Guide to Choosing Regular Foods After Bariatric Surgery (1,5)

Strategies for Vitamin and Mineral Supplementation Following Bariatric Surgery

Vitamin and mineral supplementation is necessary for maintenance of nutritional stores and should be a part of the patient's life-long dietary strategies following bariatric surgery. A multiple vitamin and mineral tablet usually given twice a day (one at breakfast and one at dinner) is recommended to meet the DRIs for most patients (5). Chewable forms (or liquid forms) of supplements are recommended for at least 2 to 3 months, after which the patient may switch to a form that they can swallow (5). Prenatal vitamins are good for individuals who need extra iron (5). Allow at least 2 hours between iron and calcium supplements to avoid interference and absorption (5). One option is to give the calcium supplement at lunch while providing the iron supplements at breakfast and dinner (5). If iron is needed, it is recommended to be consumed with vitamin C food sources (5). Signs and symptoms of nutritional deficiencies should be routinely evaluated and monitored postoperatively to determine if additional vitamin or mineral supplementation is necessary (1).

Strategies for Behavior Modification Following Bariatric Surgery

Behavior modification is a critical element for short-term and long-term success following bariatric surgery, as it directly affects for the Bariatric Diet. Behavior modification helps to improve tolerance during the initial postoperative stages. The continued application of the behavior modification techniques described below will also lead to improved long-term weight loss outcomes (1,8,23-25).

- 1. The patient should eat slowly, chewing foods completely before swallowing. The suggested average time to complete a meal is 20 to 30 minutes (8,23-25).
- 2. The patient should drink low-fat, low-sugar beverages including water between meals. The patient should avoid consuming fluids with meals and wait at least 30 minutes after meals to resume fluid intake (1).
- 3. The patient should consume protein foods first, vegetables and fruit second, and starch foods last to help ensure that adequate protein is consumed (5).
- 4. Portion control is critical. Foods should be cut, diced, and portioned to prevent overeating. The use of small serving plates may also be helpful with portion control (1).
- 5. The patient should become aware of satiety sensations and signs of pouch fullness. A feeling of pressure or nausea after consuming a food or beverage is a sign that the pouch may be full. The patient should avoid overeating or eating until fullness. There is a delay in response from when the pouch is full and when the brain signals fullness, so sticking with planned portions is important. Patients should be assured that hunger is common and normal postoperatively (1). The patient is encouraged to eat three to six servings of protein foods through the day to help satiety since hunger is common (especially within 1 week postoperatively) (1). Chronic overeating may cause pouch dilation, ineffective weight loss, and premature weight gain (1,23,24).
- 6. If vomiting occurs after eating, the patient should eat more slowly at the next meal. The patient should properly chew food, wait at least 30 minutes after eating before drinking fluids, and avoid overeating (1). Lying down after eating may be helpful (23-25). Prolonged or protracted vomiting or intolerance to food consumption should be immediately reported to the physician to prevent complications of malnutrition, dehydration, and thiamin deficiency (1).
- 7. Food intolerances are common. The patient should keep detailed food records to determine how to achieve a high-quality meal plan that integrates a variety of nutrient-dense foods.

Medical Complications and Nutrition Evaluation and Monitoring Following Bariatric Surgery

Close medical monitoring is critical during the acute postoperative stages of weight loss (1 to 16 weeks) to determine the adequacy of nutritional intake and the physiologic impact of rapid and substantial weight loss. Vital signs, such as blood pressure and heart rate, should be routinely monitored, and the patient's electrolyte levels, hydration status, and cardiac status should be monitored with laboratory assessments. Risks associated with rapid weight loss include the loss of potassium and body protein, which could lead to ventricular arrhythmias (16). However, losses of body protein are less in severely obese patients, which may provide protection from arrhythmias during rapid weight loss (15). Due to low-energy intake during the initial stages of the Bariatric Diet, urinary ketone levels are generally increased. Urinary ketones interfere with the renal clearance of uric acid, resulting in increased serum uric acid levels, which may lead to gout (19). Daily consumption of more than 100 g of carbohydrates may help minimize ketosis and uric acid levels (15). Higher serum cholesterol levels resulting from mobilization of adipose tissue may increase the risk of gallstone formation (1).

Patients who do not appropriately modify their behavior and patients who have anatomical complications may experience constant postprandial vomiting. Because of a complete lack of nutrition, these patients can develop complications such as protein-energy malnutrition and thiamin deficiency, which can lead to Wernicke-Korsakoff syndrome (1,17,22,26). A 3% to 5% incidence of hospitalization for treatment of proteinenergy malnutrition after biliopancreatic diversion procedures has been reported (17,26). If prolonged vomiting or projectile vomiting occurs, the patient should be clinically assessed for medical complications, such as thiamin deficiency, and treated for dehydration or thiamin deficiency, if indicated (1). Patients should be evaluated for enteral or parenteral nutrition support whenever necessary to prevent complications associated with malnutrition or nutritional deficiencies (1). Enteral feedings can be provided by using a smallcaliber nasogastric tube placed into the distal stomach or remaining small bowel. An isotonic elemental formula given slowly with a pump over a 24-hour period may promote greater tolerance (26). With any feeding regimen, the clinician should be alert for refeeding syndrome (17,19,26) by carefully monitoring serum levels of phosphorus, potassium, and magnesium (19). If parenteral nutrition is indicated, the initial 24-hour infusion should contain only 50% of the estimated energy needs and 50% of the estimated fluid volume. A hypocaloric feeding with adequate protein, such as 14 to 18 kcal/kg of ideal body weight and 1.5 to 2.0 g of protein per kg of ideal body weight, is often recommended in the literature (10,19). (Refer to Section B: Specialized Nutrition Support for additional information on refeeding syndrome.) Patients who experience prolonged vomiting may develop acute neurological deficits 1 to 3 months after restrictive gastric surgery (1,26). Symptoms of these neurological deficits include double vision, ataxia, nystagmus, bilateral facial weakness, acute polyneuropathy with paralysis, reduced deep tendon reflex, and mental confusion. Wernicke-Korsakoff syndrome related to thiamin deficiency has also been observed in this population. Patients who manifest neurological symptoms should be treated with 100 mg of thiamin that is intravenously or intramuscularly administered for 7 to 14 days, followed by oral administration of 100 mg/day of thiamin until the patient fully recovers or neurologic symptoms resolve (1).

Skeletal and mineral homeostasis, including nephrolithiasis, is common after Roux-en-Y gastric bypass, biliopancreatic diversion, and biliopancreatic diversion with duodenal switch (1). Laboratory tests to evaluate calcium and vitamin D metabolism and metabolic bone disease are recommended for all patients who have these procedures. Treatment with orally administered calcium, ergocalciferol (vitamin D₂), or cholecalciferol (vitamin D₃) is indicated in these patients to prevent or minimize secondary hyperparathyroidism without inducing hypercalcuria (1). The bone density of these patients should be evaluated to assess for the development of osteoporosis (1). Orally administered bisphosphonates for bariatric surgery patients with osteoporosis include: alendronate (70 mg/week), risedronate (35 mg/week or two tablets of 75 mg/month), and ibandronate (150 mg/month) (1). Oral phosphate supplementation may be provided for mild to moderate hypophosphatemia (1.5 to 2.5 mg/dL), which is usually due to vitamin D deficiency (1). The management of oxalosis and calcium oxalate stones includes avoidance of dehydration and adoption of a low-oxalate meal plan and oral calcium and potassium citrate therapy (1). Probiotics that contain *Oxalobacter formigenes* improve renal oxalate excretion and are a treatment option (1).

References:

- 1. Mechanick JI, Kushner RF, Sugerman HJ, Gonzalez-Campoy JM, Collazo-Clavell ML, Guven S, Spitz AF, Apovian CM, Livingston EH, Brolin R, Sarwer DB, Anderson WA, Dixon J. Executive summary of the recommendations of the American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic & Bariatric Surgery medical guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient. *Endocr Pract.* 2008;14:318-336.
- National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.* Bethesda, Md: National Institutes of Health; 1998. NIH Publication No. 98-4083. Available at: http://nhlbi.nih.gov/nhlbi/htm. Accessed February 19, 2009.
- 3. *Gastrointestinal Surgery for Severe Obesity.* Consensus Statement. NIH Consensus Development Conference, March 25-27, 1991. Bethesda, Md: US Public Health Service, National Institutes of Health, Office of Medical Applications of Research.
- 4. Position of the American Dietetic Association: weight management. *J Am Diet Assoc.* 2009;109:330-346.
- 5. Bariatric surgery. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed January 15, 2009.
- 6. Fujioka K. Follow-up of nutritional and metabolic problems after bariatric surgery. Diabetes Care. 2005;28:481-484.
- 7. Shikora A. The nutritional consequences of gastric restrictive surgery. Presented at: ASPEN 22nd Clinical Congress; January 20, 1998; Lake Buena Vista, Fla.
- 8. Sjostrom L, Narbo K, Sjostrom CD, Karason K, Larsson B, Wedel H, Lystig T, Sullivan M, Bouchard C, Carlsson B, Bengtsson C, Dahlgren S, Gummesson A, Jacobson P, Karlsson J, Lindross A-K, Lonroth H, Naslund I, Olbers T, Stenolf K, Torgerson J, Agren G. Effects of bariatric surgery on mortality in Swedish obese subjects. *N Eng J Med*. 2007;357:741-752.
- 9. Adams TD, Gress RD, Smith SC, Halverson RC, Simper SC, Rosamond WD, LaMonte MJ, Stroup AM, Hunt SC. Long-term mortality after gastric bypass surgery. *N Eng J Med.* 2007;357: 753-761.
- 10. Shah MV. Nutrition support in the complicated bariatric patient. Support Line. 2007; 29(6): 7-11.
- 11. Weinsier RL, Ullmann DO. Gallstone formation and weight loss. Obes Res. 1993;1:51-56.
- 12. Gebhard RL, Prigge WF, Ansel HJ, Schlasner L, Ketover SR, Sande D, Holtmeier K, Peterson FJ. The role of gallbladder emptying in gallstone formation during diet-induced rapid weight loss. *Hepatology*. 1996;24:544-548.

Nutrition Management in Bariatric Surgery

- 13. Wittgrove AC, Jester L, Wittgrove P, Clark GW. Pregnancy following gastric bypass for morbid obesity. *Obes Surg.* 1998;8:461-464.
- 14. Flancbaum L, Choban PS, Bradley LR, Burge JC. Changes in measured resting energy expenditure after Roux-en-Y gastric bypass for clinically severe obesity. *Surgery*. 1997;122: 943-949.
- 15. Nonas CA. A model for chronic care of obesity through dietary treatment. JAm Diet Assoc. 1998; 98(suppl 2):16S-22S.
- 16. VanItallie TB, Yang MU. Current concepts in nutrition: diets and weight loss. N Eng J Med. 1977; 297:1158-1161.
- 17. Kushner R. Managing the obese patient after bariatric surgery: a case report of severe malnutrition and review of the literature. *J Parenter Enteral Nutr.* 2000;24:126-132.
- Aills LA, Blankenship J, Buffington C. Bariatric nutrition: suggestions for the surgical weight loss patient. Surg Obes Relat Dis. 2008;4:S73-S108.
- 19. Pi-Sunyer FX. Short-term medical benefits and adverse effects of weight loss. Ann Intern Med. 1993;119:722-726.
- 20. Elliot K. Nutritional considerations after bariatric surgery. Crit Care Nurs Q. 2003;26:133-138.
- 21. Rhode BM, Arseneau P, Cooper BA, Katz M, Gilfix BM, MacLean LD. Vitamin B₁₂ deficiency after gastric surgery for obesity. *Am J Clin Nutr*. 1996;63:103-109.
- 22. O'Donnell K. Small but mighty: selected micronutrient issues in gastric bypass patients. Pract Gastroenterol. 2008; XXXII(5):37-48.
- Nutritional Guidelines Following Adjustable Gastric Banding (AGB) and Laparoscopic or Roux-en-Y Gastric Bypass. Johns Hopkins Center for Bariatric Surgery Bayview Medical Center. Available at: www.hopkinsbayview.org/bariatrics/docs/nutrition_band.pdf. Accessed January 20, 2009.
- 24. Pilcher J and Surgical Consultants of San Antonio, Tex. New Dimensions Weight Loss Center. *Gastric Bypass Diet*. Available at: http://www.sabariatric.com/_life_and_/diet_plan.htm. Accessed January 22, 2009.
- 25. Schirmer B. University of Virginia Gastric Bypass Program Guidelines. Charlottesville, Va: University of Virginia Health System; 2002.
- 26. Mason EE. Starvation injury after gastric reduction for obesity. World J Sug. 1998;22:1002-1007.

Bibliography

Aills L, Blankenship J, Buffington C, Furtado M, Parrott J, Allied Health Sciences Section Ad Hoc Nutrition Committee. Suggestions for the surgical weight loss patient [corrected proof, 21 May 2008]. Surg Obes Relat Dis. Available at: www.soard.org. Accessed July 30, 2008. Brolin RE. Bariatric surgery and long-term control of morbid obesity. JAMA. 2002;288:2793-2796.

SPECIALIZED NUTRITION SUPPORT THERAPY

Description

Specialized nutrition support therapy is the provision of nutrients orally, enterally, or parenterally with therapeutic intent (1). The preferred route for patients who cannot meet their nutritional needs through voluntary oral intake is enteral nutrition, the nonvolitional delivery of nutrients by tube into the gastrointestinal tract through a feeding tube, catheter, or stoma (1-4). Parenteral nutrition is the administration of nutrients intravenously (1). The modality of nutrition therapy selected should permit the delivery of required nutrients by the safest, most cost-effective route for the patient.

The goals of nutrition support therapy in both well-nourished and malnourished critically ill patients are to prevent the depletion of lean body mass, promote acute phase and whole body protein synthesis, and prevent physiologic deterioration (2). Traditionally, nutrition *support* in critically ill patients was regarded as adjunctive care designed to provide energy to support the patient during the stress response (4). Recently, these goals have become more focused on nutrition *therapy*, specifically attempting to attenuate the metabolic response to stress, prevent oxidative cellular injury, and favorably modulate the immune response (4).

The following section is a brief outline of nutritional management with these two modalities of nutrition support therapy. More detailed information related to specialized nutrition support therapy for critically ill patients can be found in the cited literature and evidence-based guidelines (2-4).

Indications (1-4)

Evidence-based guidelines for managing critically ill patients support early nutrition intervention. Enteral nutrition is recommended over parenteral nutrition in critically ill patients who are hemodynamically stable and have a functioning gastrointestinal tract. Enteral nutrition along with adequate fluid resuscitation should be initiated 24 to 48 hours after injury or admission to the intensive care unit (Grade I)* (2-4). In the critically ill patient, early enteral nutrition is associated with a reduction in infectious complications (Grade I) (2) and may reduce the length of hospitalization (2). Patients who receive enteral nutrition experience lower rates of septic morbidity and fewer infectious complications than patients who receive parenteral nutrition (Grade I) (2-5). In a large sample of patients with traumatic brain injury, early nutrition intervention was associated with improved medical outcomes and reduced mortality (6). It is advised that patients with traumatic brain injury should receive some form of nutrition support within 24 to 48 hours after injury to support their increased energy needs (5). In surgical patients and critically ill patients, enteral feedings should be provided without waiting for the resumption of flatus or bowel movements (5).

According to guidelines from the American Society for Parenteral and Enteral Nutrition and the Society of Critical Care Medicine, if enteral nutrition is not feasible or available for the first 7 days following the intensive care unit admission of a critically ill patient who was previously healthy with no evidence of protein-calorie malnutrition, no nutrition support therapy should be provided (4). Refer to Section II for guidelines to identify patients who are malnourished or may become malnourished. The use of parenteral nutrition should be reserved and initiated only after the first 7 days of hospitalization (4). However, if there is evidence of protein-calorie malnutrition on admission and enteral nutrition is not feasible, it is appropriate to initiate parenteral nutrition as soon as possible following adequate resuscitation (4). If a patient is expected to undergo major upper gastrointestinal surgery and enteral nutrition is not feasible, parenteral nutrition should be provided under the specific conditions described below (4):

- If the patient is malnourished, parenteral nutrition should be initiated 5 to 7 days preoperatively and be continued into the postoperative period (4).
- Should enteral nutrition not be feasible after surgery, the initiation of parenteral nutrition should be delayed for 5 to 7 days (4).
- Parenteral nutrition should be initiated only if the duration of therapy is anticipated to be at least 7 days.
- Parenteral nutrition therapy provided for a duration of less than 5 to 7 days would be expected to have no beneficial effect_and may result in an increased risk to the patient (4).

Contraindications

Specialized nutrition therapy is usually not indicated for: malnourished patients who are eating adequate amounts to meet their estimated nutrient requirements; well-nourished patients who are anticipated to resume adequate oral intake within 7 days; and patients whose prognosis does not warrant aggressive nutritional care

Specialized Nutrition Support Therapy

(1,4,5,7). The decision to provide nutrition therapy should be based on effective communication with the patient and family, realistic goals, and respect for patient autonomy (4).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. ASPEN Board of Directors and Standards Committee. American Society for Parenteral and Enteral Nutrition. Definition of terms, style, and conventions used in A.S.P.E.N. guidelines and standards. *Nutr Clin Pract.* 2005;20:281-285.
- 2. *Critical Illness Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- Bankhead R, Boullata J, Brantley S, Corkins M, Guenter P, Krenitsky J, Lyman B, Metheny NA, Mueller C, Robbins S, Wessel J; ASPEN Board of Directors. Enteral nutrition practice recommendations. J Parenter Enteral Nutr. 2009;33:122-167. Also available at: www.eatright.org ("Evidence-Based Practice" link). Accessed September 20, 2010.
- 4. McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; ASPEN Board of Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr. 2009;33:277-316.
- 5. Braunschweig CL, Levy P, Sheean PM, Wang X. Enteral compared with parenteral nutrition: a meta-analysis. *Am J Clin Nutr.* 2001;74:534-542.
- 6. Hartl R, Gerber LM, Ni Q, Ghajar J. Effect of early nutrition on deaths due to severe traumatic brain injury. *J Neurosurg.* 2008;109:50-56.
- 7. Position of the American Dietetic Association: ethical and legal issues in nutrition, hydration, and feeding. *J Am Diet Assoc.* 2008;108:873-882.

ENTERAL NUTRITION SUPPORT THERAPY FOR ADULTS

Definition

Enteral nutrition support therapy is the provision of nutrients to the gastrointestinal tract via a feeding tube, catheter, or stoma to maintain or replete the patient's nutritional reserves (1). Enteral nutrition is the preferred route for the provision of nutrition for patients who cannot meet their needs through voluntary oral intake (1). This section pertains to nutrition support via enteral tube feeding.

Nutrition Assessment

Indications (1-5)

Enteral nutrition support via tube feeding should be considered as a proactive therapeutic strategy for patients who are unable to ingest adequate amounts of nutrients orally and have an adequately functioning gastrointestinal tract. The advantages of enteral nutrition over parenteral nutrition include:

- a much lower cost (Grade II)* (3) and shorter length of hospital stay (5-8)
- the avoidance of complications associated with parenteral feedings (eg, infectious complications (Grade I) (3), pneumothorax, catheter embolism, and cholecystitis) (3,4,9,10) the support of the metabolic response to stress and a favorable modulation of the immune response in critically ill patients (5)
- the maintenance of gastrointestinal mucosal integrity and prevention of bacterial translocation (4,10)

Nutritional management of the stress response involves early enteral nutrition, appropriate macronutrient and micronutrient delivery, and glycemic control (5). Early enteral nutrition is well tolerated by intensive care unit (ICU) patients (3). Evidence-based guidelines for critically ill patients recommend initiating enteral nutrition 24 to 48 hours after injury or admission to the ICU if the patient is hemodynamically stable, has a functioning gastrointestinal tract, and is adequately fluid resuscitated (Grade I) (3). Early enteral tube feeding may prevent bacterial translocation, which is the passage of bacteria across the intestinal wall due to atrophy of intestinal villi (10). Maintaining gastrointestinal integrity by enteral feedings is theorized to prevent translocation, which leads to fewer infectious complications (5,10-12).

Guidelines from the American Society for Parenteral and Enteral Nutrition (ASPEN) and the Society of Critical Care Medicine (SCCM) state that traditional nutrition assessment tools (albumin, prealbumin, and anthropometry) are not validated in critical care patients (5). Before the initiation of feedings, assessments should include the evaluation of weight loss and previous nutrient intake prior to admission, level of disease severity, comorbid conditions, and function of the gastrointestinal tract.

Contraindications (1,3-5)

Enteral nutrition support should be avoided in patients who do not have an adequately functioning gastrointestinal tract *or* who are hemodynamically unstable. Specific contraindications include:

- intractable vomiting
- severe diarrhea
- high-output enterocutaneous fistula (greater than 500 mL/day) and distal to site of feeding tube tip placement
- conditions warranting total bowel rest, such as severe acute necrotizing pancreatitis (unless jejunal enteral feeding can be provided beyond the ligament of Treitz) (1,3)
- severe inflammatory bowel disease
- upper gastrointestinal hemorrhage (caused by esophageal varices, portal hypertension, or cirrhosis) (4)
- short-bowel syndrome (less than 100 cm of small bowel remaining)
- intestinal obstruction (depending on location)
- a prognosis that does not warrant aggressive nutrition support

The initiation of enteral feedings is not contraindicated by a lack of bowel sounds, flatus, or stool passage (4,5,10,11,13). Paralytic ileus is the temporary loss of contractile movements of the intestinal wall that results in an absence of bowel sounds or flatus. Ileus was once considered a contraindication to enteral feedings; however, it is now known that ileus has different effects on different areas of the intestine. For example, postoperative ileus appears to affect colonic and stomach function to a greater extent than small bowel function (5,13). The period of time that a patient's oral intake is prohibited due to diagnostic tests or procedures should be minimized. A delay in the resumption of feeding or oral intake may exacerbate the *Manual of Clinical Nutrition Management* **B-35** Copyright © 2013 Compass Group, Inc.

Enteral Nutrition Support Therapy for Adults

potential for ileus (5). The clinical condition of the patient is an important consideration in the decision to initiate enteral nutrition. A soft, nontender abdomen, adequate perfusion, and hemodynamic stability are indicators of the potential for the safe administration of enteral nutrition (3,4). For most patients, lower gastrointestinal bleeding does not affect the administration of enteral support (4,14).

Nutrition Intervention

Enteral feedings can be nutritionally adequate if an appropriate formula is selected with consideration of each patient's individual estimated requirements. Energy requirements may be calculated by predictive equations or measured by indirect calorimetry (5). Predictive equations should be used with caution, as they provide a less accurate measure of energy requirements than indirect calorimetry (5). In the obese patient, the predictive equations are even less accurate (5). (Refer to Section II: Estimation of Energy Requirements.) Tube feedings may be used as the sole source of nutrients or as a supplement to inadequate oral nutrition. Enteral nutrition should be initiated within 24 to 48 hours of injury or admission to the ICU, and the average intake delivered within the first week should be at least 60% of the total the estimated energy requirements, as determined by the nutrition assessment (Grade II) (3). Provision of enteral nutrition within this time frame and at this intake level is associated with fewer infectious complications (Grade II) (3). Guidelines for critically ill patients from ASPEN and the SCCM include similar recommendations (5). These guidelines recommend the provision of more than 50% to 65% of the estimated energy requirements during the first week of hospitalization to achieve the clinical benefits of enteral nutrition (4,5). The impact of a specific threshold of enteral nutrition delivery on mortality, hospital length of stay (LOS), and days on mechanical ventilation is unclear due to inconsistent results produced by existing studies (Grade II) (3).

Based on limited evidence available, permissive underfeeding rather than overfeeding obese critically ill patients may produce better medical outcomes. In obese, critically ill adults, the Registered Dietitian (RD) may consider prescribing hypocaloric, high protein enteral feedings (Grade III) (3). According to the Academy of Nutrition and Dietetics (AND) guidelines, very limited research in patients receiving enteral nutrition shows that the effect of hypocaloric, high protein feeding (< 20 kcal per kg adjusted body weight (ABW) and 2 g protein per kg ideal body weight (IBW) promoted shorter intensive care unit (ICU) stays, although total hospital length of stay (LOS) did not differ (3). In the group receiving hypocaloric, high protein feedings, nitrogen balance was not adversely affected (Grade III) (3). Guidelines from ASPEN and the SCCM recommends permissive underfeeding or hypocaloric feeding with enteral nutrition in the critically ill obese patient (5). For patients with a body mass index (BMI)_greater than 30 kg/m², the goal of the enteral nutrition regimen should not exceed 60% to 70% of target energy requirements or 11 to 14 kcal/kg actual body weight per day (or 22 to 25 kcal/kg ideal body weight per day) (5).

In addition to the delivery of energy, the adequacy of protein provision should be assessed on an ongoing basis (5). The use of additional modular protein supplements is a common practice, as standard enteral formulations tend to have a high ratio of nonprotein energy to nitrogen (5). The protein requirements of critically ill patients with a BMI less than 30 kg/m^2 are 1.2 to 2.0 g/kg actual body weight per day; these requirements may be higher in burn patients or multiple-trauma patients (5). Critically ill patients who are obese have higher protein requirements to maintain an adequate nitrogen balance and accommodate the needs for wound healing. The protein requirement for class I and II patients (BMI, 30 to 40 kg/m²) is greater than 2.0 g/kg ideal body weight per day, and the protein requirement for class III patients (BMI >40 kg/m²) is greater than 2.5 g/kg ideal body weight per day (5).

Antioxidant vitamins (including vitamin E and ascorbic acid) and trace minerals (including selenium, zinc, and copper) may improve patient outcome, especially in burns, trauma, and critical illness requiring mechanical ventilation (5). A combination of antioxidant vitamins and trace minerals (specifically selenium) should be provided to all critically ill patients receiving specialized nutrition therapy (5).

How to Order the Diet

The physician in collaboration with the dietitian determines the appropriate prescription for the enteral nutrition regimen, including the route and type of formula. A dietitian should facilitate the selection of the formula type and goal rate for tube feeding. Once the goal rate is reached, a nutrient intake study may be beneficial to verify that the total nutrient intake (oral plus enteral) is adequate.

The order specifies:

- product, either by name or as "Standard Tube Feeding," according to hospital protocol
- volume, rate, and timing, including the initial volume and rate, as well as the progression and goal volume and rate (At a standard dilution of 1.0 kcal/mL, the volume will be roughly equal to the number of kilocalories specified.)
- administration and monitoring, following either the facility's standard procedures or individualized orders, including the administration of extra water to flush the tube or meet fluid requirements

See Section III: Clinical Nutrition Management, Enteral Nutrition: Management of Complications

Routes of Access for Enteral Tube Feeding (4)

The type and route of feeding tube should depend on the patient's needs and the route that optimizes nutrient delivery (stomach or small bowel) for disease management. The smallest tube possible should be used for patient comfort (4), and correct placement of the feeding tube should be confirmed by X-ray prior to use (4). When the anticipated need for enteral nutrition exceeds 4 to 6 weeks, a more permanent enteral access device is indicated (4).

There are several types of feeding tube placements:

- **Orogastric:** The feeding tube is inserted through the mouth, with the tip resting in the stomach.
- **Nasogastric:** The feeding tube is inserted through the nose, with the tip resting in the stomach.
- **Nasoduodenal:** The feeding tube is inserted through the nose, with the tip resting in the duodenum.
- **Nasojejunal:** The feeding tube is inserted through the nose, with the tip resting in the jejunum.
- **Esophagostomy:** The feeding tube is inserted through a surgical opening in the neck and passed through the esophagus, with the tip resting in the stomach.
- **Gastrostomy:** The feeding tube is inserted through the abdominal wall into the stomach via percutaneous endoscopic guidance or surgical placement (surgical "open" gastrostomy).
- **Jejunostomy:** The feeding tube is inserted through the abdominal wall into the jejunum via percutaneous endoscopic guidance or surgical placement (surgical "open" jejunostomy).

Enteral Formula: Categories and Selection

Choosing the most appropriate tube-feeding formula is critical for achieving nutritional goals. Formulas should be selected based on digestibility/availability of nutrients, nutritional adequacy, viscosity, osmolality, ease of use, and cost (4). In addition, the nutritional status of the patient, including electrolyte balance, digestive and absorptive capacity, disease state, renal function, medical or drug therapy, and possible routes of enteral infusion should be considered (4). Enteral formulations are considered medical foods by the Food and Drug Administration (FDA); therefore, their labels can make "structure and function" claims without the approval of the FDA (4). Limited evidence is available regarding the efficacy and outcomes associated with the use of specialized enteral formulations (3,4).

Enteral formulas can be classified as standard (or polymeric), elemental, or semi-elemental. Standard formulas include synthetic formulas and blenderized formulas. Specialized enteral formulas include disease-specific formulas and nutrient-modified formulas. Additionally, individual modular components that can supplement the formula are available (15). Most enteral formulations provide adequate amounts of vitamins and minerals to meet the Reference Daily Intakes when provided in volumes of 1,000 mL to 1,500 mL daily (16). Enteral formulas contain a large amount of water; their water content generally ranges from 70% to 85% (15). It is important to be aware of patients with potential food allergies when prescribing an enteral formula. Enteral formulations may contain milk, soy, corn, or egg products, all of which are common allergens (15). Most enteral formulations are lactose free and gluten free (15).

Standard or polymeric formulas: Standard or polymeric formulas must be digested into dipeptides and tripeptides, free amino acids, and simple sugars in the small bowel. Polymeric formulas require adequate digestive and absorptive capability and are indicated in patients with normal or near-normal gastrointestinal function. There are two basic types of polymeric formulas, synthetic formulas and blenderized formulas. Synthetic formulas are the most commonly used formulas due to their safety and feasibility in an institutional setting (4).

Synthetic formulas are used for standard tube feedings. Their energy content ranges from 1.0 to 2.0 kcal/mL. The protein content provides 12% to 20% of total energy and consists of intact protein, generally

Enteral Nutrition Support Therapy for Adults

casein or soy protein isolate (15). Lactalbumin, whey, and egg albumin are also sources of intact proteins. Formulas that contain intact proteins require normal levels of pancreatic enzymes for digestion and absorption (15). Carbohydrate sources include corn syrup solids, hydrolyzed cornstarch, maltodextrin, sucrose, fructose, and sugar alcohols. Carbohydrates provide 40% to 90% of total energy (15). The fat content ranges from less than 10% to more than 50% of total energy. Common fat sources are corn oil and soybean oil; however, safflower, canola, and fish oils are also used in enteral formulas (15). The osmolality of synthetic formulas ranges from 270 to 700 mOsm/kg. Because a high incidence of lactase deficiency in illness is presumed, lactose is not present in most synthetic enteral formulas (15).

Elemental or semi-elemental formulas: These formulas consist of hydrolyzed macronutrients. Protein is present either as free amino acids (monomeric) or as bound amino acids in dipeptides or tripeptides (oligomeric). Carbohydrate sources consist of oligosaccharides, sucrose, or both. Most monomeric formulas are low in fat or contain a large percentage of medium-chain triglycerides (MCT) oil. These formulas are low residue, hyperosmolar, and usually lactose free. They are indicated for patients with compromised gastrointestinal function, such as patients who have acute pancreatitis (15) or persistent diarrhea (5). Formulas with predigested nutrients should not be used for patients with normal digestion and absorption, because they are unnecessary for these patients and cost more than standard intact (polymeric) nutrient formulas.

Modular components: These products are individually packaged components that may be combined in varying amounts to meet the patient's individual nutritional needs. Examples include protein powders, carbohydrate powders, MCT oil, fiber, and specific amino acids (eg, glutamine and arginine). Protein powders, which provide 7 to 15 g of protein per serving, are the most commonly used modular additives, as standard enteral formulations tend to have a high ratio of nonprotein energy to nitrogen (5,15). Modular components may also be added to premixed formulas to enhance the intake of one or more macronutrients. If modular components are added to premixed formulas, the preparation should follow the organization's Hazard Analysis and Critical Control Point Enteral Nutrition Plan (7).

Nutrient-Modified and Disease-Specific Formulas

Nutrient-modified and disease-specific formulas have been altered in one or more nutrients in an attempt to optimize nutrition support without exacerbating the metabolic disturbances associated with various diseases. Limited evidence is available regarding the efficacy and outcomes associated with the use of most disease-specific enteral formulations (4). Standard enteral formulas are appropriate for most critically ill patients (3). Disease-specific formulas are more expensive than standard enteral formula, and a dietitian should carefully evaluate their potential benefit for an individual patient before recommending them. If such formulas are used, the patient should be monitored and advanced to a standard formula as soon as possible (3).

Nutrient-modified formulas include:

- Formulas containing fiber: Fiber is added to enteral formulas for a variety of potential health benefits (17). Soluble fibers, such as guar gum, oat fiber, and pectin, are fermented to short-chain fatty acids, which are easily absorbed by the gastrointestinal mucosa. Short-chain fatty acids, the preferred fuel for colonocytes, help to increase mucosal growth and promote sodium and water absorption (17). It is important to consider the type of fiber supplemented in the enteral formula. Soy polysaccharide an insoluble fiber, and guar gum a soluble fiber are common types of fiber used in formulas. Soluble fiber reduces the incidence of diarrhea, but studies of formulations supplemented with insoluble fiber have not yielded the same results (5,15,18-20). ASPEN guidelines recommend avoiding the use of insoluble fiber formulas in critically ill patients (5). It has also been suggested that both soluble and insoluble fiber should be avoided in patients at high risk for bowel ischemia or severe dysmotility (5,15). Cases of bowel obstruction from the use of these formulations have been reported (14,21,22). Enteral formulas containing fiber generally provide 5 to 14 g/L, which is less than the recommended 20 to 35 g of fiber per day. When a fiber formula is used, adequate free-water needs should be maintained and tolerance closely monitored, especially for patients receiving larger volumes of fiber-containing formulas (eg, greater than 2 L per 24-hour period). Soluble fiber-containing formulas using guar gum are indicated in critically ill patients when there are no contraindications for their use and the goal is to maintain a normally functioning gastrointestinal tract and defecation pattern, or to manage diarrhea (Grade III) (3,5,23).
- **Formulas containing omega-3 fatty acids:** The type and amount of fat in enteral formulas may affect immune function (24-27). Fish oils, a rich source of omega-3 fatty acids, provide eicosapentaenoic acid. Unlike omega-6 fatty acids, which are found in more common fat sources (eg, corn oil and soybean oil)

and have a greater immunosuppressive effect, eicosapentaenoic acid metabolizes into less immunosuppressive prostaglandins and leukotrienes. A study has shown the beneficial effects of omega-3 fatty acids on the length of hospital stay and the infection rate following burn injury (26). In another study, fewer gastrointestinal and infectious complications occurred in patients who received a formula rich in fish oils when compared to patients who received a standard polymeric formula (27). Enteral supplementation with omega-3 fatty acids has beneficial effects in the treatment of acute respiratory distress syndrome (ARDS). In a study of 146 patients with ARDS, patients who received enteral formula supplemented with omega-3 fatty acids (eicosapentaenoic acid) and gamma-linolenic acid (an omega-6 fatty acid) had significant improvements in oxygenation, lower ventilation variables, fewer days of ventilator support, and shorter stays in the ICU when compared to controls (28). According to both AND and ASPEN guidelines, critically ill ARDS patients and those with severe acute lung injury may be placed on an enteral formulation characterized by an anti-inflammatory lipid profile (eg, omega-3 fish oils, borage oil) and antioxidants based on a comprehensive review of studies (Grade II) (3,5).

- Formulas containing arginine: Arginine is a conditionally essential amino acid during stress due to the greater utilization of the urea cycle. Arginine is important in immune function and wound healing and is commonly found in immune-enhancing and wound-healing enteral formulas. The immune-enhancing properties of arginine include effects on the production and maturation of T lymphocytes and natural killer cells (29,30). Arginine may be useful in treating inflammatory diseases and acquired immunodeficiency syndrome, and it enhances collagen formation in wound healing (31,32). Although studies have demonstrated positive outcomes of arginine supplementation, other studies have associated arginine-containing formulations with an increased risk of mortality as compared to standard enteral formulas in severely septic ICU patients (5). The mechanism proposed for this adverse effect was hemodynamic instability caused by the conversion of arginine to nitric oxide in patients with severe sepsis (6). More research is needed to determine the optimal dose, timing, and specific patient criteria prior to the use of arginine supplemented formulas (3,5). (See the discussion of immune-enhancing enteral formulations for suggested patient criteria.)
- Formulas containing glutamine: Classified as a nonessential amino acid, glutamine is the primary oxidative fuel for rapidly dividing cells, such as enterocytes and leukocytes. During stress or injury, the metabolic demand for glutamine can exceed the capacity of skeletal muscle to release it (33). A fall in glutamine concentration is associated with atrophy of the intestinal mucosa, impaired immune function, and decreased protein synthesis (29). Substantial research has been completed in the past decade on the effects of glutamine in enhancing small-intestine growth and repair from injury. Glutamine is not present in standard parenteral formulas, and it is present in only small amounts in enteral formulas (16). Formulas that contain intact proteins contain some bound glutamine. Although most research has involved parenteral glutamine, one study demonstrated that patients who received glutamine-rich enteral formula had significantly reduced hospital costs due to the prevention of secondary infections, when compared to patients who received isoenergetic control formula (34). However, other studies have found no effect with glutamine supplementation (3,35,36). The optimal amount and form of oral glutamine required to achieve beneficial results is still unknown (3). Dosage and safety studies have found that 20 to 40 g/day of glutamine supplementation is safe and well tolerated by adults (37). According to ASPEN and SCCM, the addition of enteral glutamine to an enteral nutrition regimen (not already containing supplemental glutamine) may be considered in burn, trauma, and ICU patients with multiple medical conditions or comorbidities (5) A review of studies found that additional glutamine is usually provided in two or three divided doses that yield 0.3 to 0.5 g/kg body weight per day." (5). The Academy of Nutrition and Dietetics does not support this recommendation due to limited evidence supporting enteral glutamine (Grade III) (3). Glutamine supplementation is contraindicated in patients with hyperammonemia, hepatic failure, or renal failure due to excess ammonia production (4). Enteral glutamine should not be added to an immune-modulating formulation already containing supplemental glutamine (5).
- **Formulas containing branched-chain amino acids (BCAA):** Formulas supplemented with BCAA have primarily been used for patients with hepatic failure in an attempt to improve the ratio of BCAA to aromatic amino acids and prevent or improve hepatic encephalopathy. The use of BCAA-supplemented enteral formulas has not been validated because studies have provided mixed results. A randomized study found that BCAA supplementation reduced hospital admissions and improved nutritional status in patients with advanced liver disease when compared to a standard formulation; however, there was no difference in encephalopathy scores between the two groups (38). The routine use of BCAA-enriched formulas is not recommended (15). Considering the limited evidence to support these formulations, the ASPEN guidelines for nutrition therapy in liver disease restrict the use of BCAA-enriched formulas to

Enteral Nutrition Support Therapy for Adults

patients with refractory (chronic) encephalopathy that is unresponsive to pharmacotherapy (1,2,39).

Disease-specific formulas include:

• Formulas for renal disease: Renal formulas are energy-dense formulas that contain whole proteins and have a modified electrolyte content (eg, sodium, potassium, phosphorus, and magnesium). These formulas may be indicated in renal patients whose serum electrolyte levels are difficult to manage or for whom renal dialysis is delayed (15). ASPEN recommendations suggest that ICU patients with acute renal failure receive standard enteral formulations and that standard ICU recommendations for protein and energy provision should be followed (5). If significant electrolyte abnormalities exist or develop, then a specialty formulation designed for renal failure (with an appropriate electrolyte profile) may be considered (5).

Most specialty renal formulas are energy dense, so that volume can be restricted if needed. The protein content ranges from less than 40 g to more than 70 g in 2,000 kcal. These formulas meet the Dietary Reference Intakes for most nutrients with the exception of select vitamins, minerals, and electrolytes that are normally restricted in renal insufficiency (eg. potassium, sodium, phosphorus, and magnesium). If dialysis is delayed, an energy-dense, reduced-protein formula is appropriate (40). However, long-term use of these formulas requires close monitoring of the patient's nutritional status (15). Patients receiving hemodialysis or continuous renal replacement therapy should receive increased protein, up to a maximum of 2.5 g/kg per day (5). Protein should not be restricted in patients with renal insufficiency as a means to avoid or delay the initiation of dialysis therapy (5,15,41). The nutrition goals of patients with renal failure should include adequate protein and energy intake, with modifications in fluid volume and electrolyte content that are individualized based on the patient's clinical condition. There is insufficient data to determine if renal formulas produce different outcomes than standardized formulas (5,42).

- **Formulas for hepatic disease:** Standard formulations should be used in ICU patients with acute and chronic liver disease (1,4,5,15). Nutrition regimens should avoid restricting protein in patients with liver failure (5). Special enteral formulas have been designed for patients with hepatic failure. These formulas are modified in fluid, protein, and mineral content and may not meet the Dietary Reference Intakes for various nutrients. These formulas contain increased levels of BCAA along with decreased levels of aromatic amino acids in an attempt to treat or prevent hepatic encephalopathy. The total protein content varies among formulas and is often low. Studies of these formulas are inconclusive (5,39). The BCAA-enhanced formulations should be reserved for the rare encephalopathic patient who is refractory to standard treatment with luminal-acting antibiotics and lactulose (5,39).
- Formulas for pulmonary disease and ARDS: Pulmonary disease formulas are low in carbohydrate and high in fat to decrease carbon dioxide (CO_2) production in patients with compromised pulmonary function. Formulas that contain omega-3 fatty acids may be beneficial in patients with early ARDS (1). The ASPEN guidelines, which are based on a comprehensive review of studies, suggest that critically ill ARDS patients receive enteral formulations that have an anti-inflammatory lipid profile (eg, omega-3 fish oils, borage oil) and contain antioxidants (5). The Academy of Nutrition and Dietetics provides similar guidelines which recommend the use of immune-modulating formulas with fish oil, borage and antioxidants to be considered for intensive care unit (ICU) patients with acute respiratory distress syndrome (ARDS) or acute lung injury (Grade II) (3). The evidence regarding the impact of higher fat and lower carbohydrate enteral formula composition on CO_2 production is still limited. Although a few studies have shown decreased CO_2 levels in hypercapnic patients who received these formulas (28,43), more data are needed. Talpers et al found that an excess of total energy was as deleterious to CO_2 production as carbohydrate intake (43). Thus, an excess of total energy should be avoided, and energy intake should be equal to or less than the energy needs of patients with pulmonary disease and CO_2 retention (1,15). The high lipid content of these formulas may cause delayed gastric emptying (44). These formulas tend to contain intact nutrients and are usually low in fluid and nutritionally complete. Fluidrestricted, energy-dense formulations (1.5 to 2.0 kcal/mL) should be considered for patients with acute respiratory failure to prevent fluid accumulation and pulmonary edema (5). If pulmonary formulas are used, the patient's ventilatory status and CO₂ production should be monitored, and overfeeding should be avoided (2.15).
- **Formulas for diabetes mellitus:** Patients with diabetes mellitus who follow a diet with increased intake of monounsaturated fatty acids along with lower carbohydrate intake may have improved blood glucose control (15). Therefore, enteral formulas with modified amounts and types of carbohydrate and fat have

been developed. These formulas contain less carbohydrate (34% to 40%), more modified fat (40% to 49%), and 10 to 15 g/L of fiber (15). Outcomes data regarding these formulas are limited in their context and their applicability to persons with diabetes mellitus (15). A prospective study that compared a diabetic formula to a standard fiber-containing formula in long-term care patients did not demonstrate any clinically significant benefit from the diabetic formula, with the exception of improved high-density lipoprotein cholesterol levels (45). A study of hospitalized diabetes patients concluded that diabetic formulations had no effect on glycemic control (46). According to The Academy of Nutrition and Dietetics Evidence Analysis Project for Diabetes, there is insufficient evidence to determine whether the nutrient composition of enteral formulations has an impact on medical costs, mortality rates, infectious complications, and length of hospital stay in patients with diabetes (Grade V) (46,47). Diabetic formulas may be appropriately used in patients with blood glucose levels that are difficult to control with traditional methods (15). The American Diabetes Association suggests that either a standard (50% carbohydrate) or a lower carbohydrate content (33% to 40%) formula be used for tube-fed patients (47). It is generally recommended that diabetic patients receive a standard formula with close monitoring of blood glucose levels and that insulin be used as needed for glycemic control. The total grams of CHO provided in the formulation has the greatest impact on blood glucose response. Monitoring the total grams of CHO remains a key strategy in achieving glycemic control and is more important than the source or type of CHO. (15,48).

Formulas containing immune modulating nutrients: Formulas have been designed with increased amounts of immune-enhancing nutrients (eg. arginine, glutamine, nucleotides, omega-3 fatty acids or fish oil) and antioxidant vitamins and minerals. Use of immune-modulating enteral formulas (an enteral formula that contains pharmacological doses of nutrients intended to impact the immune system) should be carefully evaluated for ICU patients depending on primary diagnosis (Grade II) (3). For ICU patients with acute respiratory distress syndrome (ARDS) or acute lung injury, the Registered Dietitian may consider using immune-modulating formulas with fish oil, borage and antioxidants (Grade II) (3). Use of immunemodulating enteral formulas is not associated with reductions in infectious complications, length of hospital stay, cost of medical care, days on mechanical ventilation, or mortality in moderately to less severely ill ICU patients (Grade II) (3). These formulas may be associated with increased mortality in severely ill ICU patients, although adequately powered trials evaluating this finding have not been conducted (3). ASPEN guidelines state that immune-modulating enteral formulations be used only for the appropriate patient population, which includes patients who undergo major elective gastrointestinal surgery, patients who have abdominal trauma, patients who have burns on more than 30% of their bodies, head and neck cancer patients, and critically ill patients on mechanical ventilation who are not severely septic; immune-modulating enteral formulations should be used with caution in patients who have severe sepsis (5). Patients in the ICU who do not meet these criteria should receive standard enteral formulations (5). These guidelines also suggest the provision of at least 50% to 65% of goal energy requirements from the immune-modulating formulations to receive optimal therapeutic benefits (5).

Water/Fluid Requirements

The National Research Council recommends 1 mL of fluid per 1 kcal of energy expenditure for adults with average energy expenditure who live under average environmental conditions (50). Medical conditions that may reduce fluid requirements include heart failure, acute respiratory failure, renal failure, ascites, syndrome of inappropriate antidiuretic hormone, and malignant hypertension. Fluid requirements may be increased for pregnant patients; patients with fever, burns, diarrhea, vomiting, or high-output fistulas or ostomies; and patients receiving ventilatory support (51). Patients with pressure ulcers and patients medically managed on air-fluidized beds also have additional fluid needs. (Refer to Section IA: Nutrition Management of Fluid Intake and Hydration.)

There are several methods to determine fluid requirements (50). There is no evidence that compares the effectiveness of these methods for estimating the fluid needs of adults (Grade V) (51). The methods include (51):

Method 1: Holliday-Segar Method^a

Body Weight (actual) $\leq 10 \text{ kg}$ between 10 kg and 20 kg $\geq 20 \text{ kg}$ **Water Requirement** 100 mL/kg 1,000 mL + 50 mL/kg for each kg > 10 kg 1,500 mL + 20 mL/kg for each kg > 20 kg

Method 2: Recommended Daily Allowances Method^b 1 mL per kilocalorie of energy expenditure

Method 3: Fluid Balance Method (Grade IV)^c

Urine output + 500 mL/day

^aHolliday MA, Segar WE. The maintenance need for water in parenteral fluid therapy. *Pediatrics*. 1957;19:823-832.

^bInstitute of Medicine. *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate.* Washington, DC: National Academy Press; 2004.

cHydration Evidence-Analysis Project. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2007. Available at: http://www.andevidencelibrary.com. Accessed November 13, 2012.

Approximate	ree Water ^a Conten	it of Nutritional Forn	nulas

Formula	mL H ₂ O/mL Formula	mL H ₂ 0/kcal
1.0 kcal/mL	0.84	0.84
1.0 kcal/mL with fiber	0.83	0.83
1.5 kcal/mL	0.78	0.52
2.0 kcal/mL	0.71	0.36

^aFree water delivered in tube feeding = milliliters of formula delivered \times milliliters of H₂O per milliliter of formula

Criteria for Formula Selection (15)

There are a variety of enteral nutrition products on the market, many of which have only subtle differences in composition. The following criteria should be considered when selecting a formula:

- **Energy density:** An energy density of 1 kcal/mL is considered standard. Additional free water is usually necessary to meet fluid requirements. Higher energy concentrations (1.5 to 2.0 kcal/mL) may be indicated when fluid must be restricted or when feeding volumes sufficient to meet energy requirements cannot be tolerated. Fluid-restricted, energy-dense formulations should be considered for patients with acute respiratory failure to prevent fluid accumulation and pulmonary edema (5).
- **Osmolality:** Products are available at isotonic osmolalities (300 mOsm/kg), moderate osmolalities (400 mOsm/kg), and high osmolalities (700 mOsm/kg). The main contributors to osmolality are sugars, free amino acids, and electrolytes. Lipids are isotonic and therefore do not contribute to osmolality. High-carbohydrate, amino acid-based, or peptide-based formulas have moderate to high osmolality. Formula osmolality has not been conclusively found to be a direct cause of diarrhea (15).
- **Protein:** Protein sources are intact proteins, peptides, or amino acids. Generally, protein contributes 9% to 24% of total energy. High-nitrogen formulas may not be well tolerated in patients with certain renal or hepatic disorders. High-nitrogen concentrations can result in a higher renal solute load and can predispose elderly patients to dehydration. One gram of nitrogen requires 40 to 60 mL of water for excretion.
- **Fat:** Fat sources are long-chain triglycerides (LCT) and MCT. The fat content usually ranges from 3% to 35% of energy for amino acid-based or peptide-based formulas and from 25% to 55% of energy for standard formulas. Fats do not contribute to osmolality. Inclusion of MCT may be beneficial for patients who experience fat malabsorption or maldigestion, since MCT do not require pancreatic lipase for absorption, and intraluminal hydrolysis is more rapid and complete than with LCT (15). The MCT do not supply essential fatty acids, and they may cause complications for cirrhotic patients who have a limited ability to oxidize MCT. The administration of MCT along with LCT increases the total intestinal absorption of both types of fats, as compared with the absorption of either fat administered alone.
- **Carbohydrate**: Carbohydrate is the most easily digested and absorbed nutrient. Enzyme digestion is very efficient, as surface digestion is not rate limiting (except with lactose). The transport process is the slowest part of carbohydrate metabolism. Carbohydrate sources are monosaccharides, oligosaccharides, and lactose. The carbohydrate content of formulas ranges from 35% to 90% of energy. Longer carbohydrate molecules exert less osmotic pressure, taste less sweet, and require more digestion than do shorter ones. Glucose polymers are better absorbed than free glucose and enhance absorption of calcium, zinc, and magnesium in the jejunum. Some specialty formulas include fiber, fructose, and/or fructo-oligosaccharides. Fructo-oligosaccharides occur naturally in a variety of fruits and vegetables and provide sweetening at a lower cost than sucrose. Fructo-oligosaccharides are poorly absorbed by the small intestine and fermented in the colon, where they promote the growth of healthy species of bacteria (15).
- **Lactose:** Lactose is present in milk-based formulas and some blenderized formulas. Most commercial formulas are lactose free. Due to the presumed high incidence of secondary lactase deficiency in illness, lactose is not present in most enteral formulas (15).

- **Residue:** Milk-based formulas and other formulas with intact nutrients are generally low residue. Blenderized and fiber-supplemented formulas leave a moderate to high residue.
- Fiber: See the previous discussion of formulas containing fiber in the nutrient-modified formulas list.
- **Sodium and potassium:** Select formula according to the patient's nutrition prescription and laboratory profile.
- **Renal solute load:** The main contributors to renal solute load are protein, sodium, potassium, and chloride. A high-renal solute load in sensitive patients can result in clinical dehydration.
- **Safety:** A ready-to-use closed bag or system is recommended, as it is more sterile and provides less risk for contamination than canned or powdered products (52). Formulas that are made in a blender in the facility are discouraged because they carry a greater risk of infection, require careful handling, tend to clog tubes, and need a high volume to meet nutrient needs. If formulas are mixed, follow the organization's Hazard Analysis and critical Control Point Enteral Nutrition Guidelines to ensure safety (52).
- **Viscosity:** Blenderized, high-fiber, and high-density formulas should not be administered through tubes with a diameter smaller than 10 French unless a pump is used. Formulas may flow through an 8-French diameter tube when a pump is used (15).
- Vitamin K: Patients who are receiving enteral nutrition support while on anticoagulant therapy should be monitored closely. Significant vitamin K intake from enteral formulas can antagonize the effect of the anticoagulant drug warfarin and result in treatment failure (53). Most enteral formulations contain modest amounts of vitamin K and provide daily vitamin K intake similar to the average dietary intake from foods (53). Consistent intake of an enteral formulation containing less than 100 mcg of vitamin K per 1,000 kcal is not expected to cause warfarin resistance (53). Refer to Section III: Anticoagulant Therapy for specific guidelines for enteral nutrition regimens in this group of patients.
- **Cost:** Amino acid-based formulas and peptide-based formulas are usually more expensive than synthetic formulas containing intact nutrients.

Enteral Feeding Administration (52,54)

Continuous feeding/delivery: Continuous feedings require that the enteral formula be administered at a controlled rate with a pump over a 24-hour period. The pump should deliver the controlled rate within 10% accuracy and be calibrated periodically to ensure accuracy (52). Continuous feedings are indicated for unstable critically ill patients, patients unable to tolerate high-volume feedings, patients with malabsorption, and patients at increased risk for aspiration (5). Feedings may be initiated at full strength in the stomach or at an isotonic strength in the small bowel at a rate of 10 to 30 mL/hour. Then, the rate may be gradually increased as tolerated in increments of 10 to 25 mL/hour every 4 to 8 hours to the goal rate. Strength and volume should not be increased simultaneously.

Intermittent or cyclic feeding/delivery: Intermittent or cyclic feedings are administered over an 8- to 20hour period by using a pump to control the rate of delivery. This method of tube feeding is most beneficial for patients who are progressing from complete tube feeding support to oral feedings as discontinuation of feedings during the day may help to stimulate the appetite. Intermittent or cyclic feeding is also beneficial for ambulatory home-care patients who are unable to tolerate bolus feedings because it allows freedom from the pump and equipment. Since this method of delivery usually requires a higher infusion rate, monitoring for formula and delivery tolerance is necessary. Formula and delivery intolerance can be avoided by a gradual transitioning of the patient from continuous feeding to an intermittent feeding schedule.

Bolus formula delivery not requiring a pump: The syringe bolus-feeding method involves the delivery of 240 to 480 mL of formula via a feeding tube over a 20- to 30-minute period, three to six times a day, to meet estimated nutritional requirements. This method is usually restricted to gastric feedings and may be contraindicated in patients who have a high risk of aspiration, disorders of glucose metabolism, or fluid management issues.

Enteral Feeding Formula and Equipment Maintenance Guidelines (52) Formula:

- Bring formula to room temperature before feeding, but do not allow the formula to remain unrefrigerated for more than 12 hours (52).
- The hang time for formula in an open system should be less than 8 hours or as specified by the manufacturer. Formula from a closed system is provided in ready-to-hang, prefilled containers and may hang for 24 to 48 hours per manufacturer's guidelines (52). Discard any formula remaining in the container after the hang time has expired.
- Opened, unused formula should be kept refrigerated for no longer than the manufacturer's specifications (usually 24-48 hours) (52).

Enteral Nutrition Support Therapy for Adults

• Refer to the ASPEN Enteral Nutrition Practice Recommendations or organization-specific interdisciplinary enteral nutrition monitoring protocol and policy as needed (52).

Formula delivery guidelines:

- Irrigate the tube every 4 hours with 20 to 30 mL of warm sterile water or saline to ensure patency for continuous feeding (52). Also, irrigate the tube before and after each intermittent feeding or medication administration (2,52).
- To reduce bacterial contamination, flush water through the bag and tube every 8 hours before adding new formula when an open system is in place.
- Avoid putting food and beverages into the tube (eg, juice, milk, and soda).
- Flush tube with purified sterile water or saline before and immediately after the administration of medicines to avoid clogging the tube (52).
- To reduce the risk of contamination and infection, the feeding bag should be properly labeled, and tubing should be changed every 24 hours or as specified by the manufacturer (52).
- Refer to organization-specific interdisciplinary enteral nutrition monitoring protocol and policy as needed.

Patient Monitoring Guidelines

Refer to the Academy's *Critical Illness Evidence-Based Nutrition Practice Guideline*, the ASPEN Enteral Nutrition Practice Recommendations, the ASPEN Guidelines for Nutrition Support Therapy in the Adult Critically Ill Patient, and the Morrison Nutrition Practice Guideline – Enteral Nutrition (3,5,52,55,56). Also refer to organization-specific interdisciplinary enteral nutrition monitoring protocols as needed. Guidelines for patient monitoring and avoidance of complications associated with the delivery of enteral nutrition are described below.

Patients with nasoenteric tubes:

- Provide mouth and nose care every 8 hours to prevent parotitis and skin breakdown around the nose.
- Verify the placement of a nasoenteric tube prior to feeding initiation and every 4 to 8 hours thereafter, or as specified by the organization's protocol (52).

Avoidance of intestinal hypoxia and bowel necrosis (4,15,56):

- Assess bowel sounds every 8 hours.
- Feed into the small bowel (postpyloric position).
- Administer feeding in patients who are adequately fluid resuscitated and have a sustained mean arterial pressure of at least 70 mm Hg, and are at steady or decreasing dos of vasoactive agents.
- Use iso-osmolar, low residue formulations, initiated at 10 20 ml/hr and advance the feedings when tolerance is demonstrated.
- Assess the need to hold feedings if the patient experiences a sudden period of hypotension (mean arterial pressure < 60 70 mm Hg), if the dosages of pressor agents (eg, dobutamine, norepinephrine, and epinephrine) are increased, or if the need for ventilatory support is increased (3, 56).
- Assess the need to hold feedings if the patient develops increased nasogastric output, abdominal distention, or abdominal pain.

Avoidance of gastrointestinal intolerance and aspiration (3,5,52,57):

- Recommendations from the North American Summit on Aspiration in the Critically III state that feeding ideally should be into the small bowel with the tube tip at or below the ligament of Treitz when two or more risk factors for aspiration are present (4,5,57). These risk factors include: prior aspiration, decreased level of consciousness, neuromuscular disease, structural abnormalities of the aerodigestive tract, endotracheal intubation, vomiting, persistently high gastric residual volumes (GRVs), and the need for a supine position (57). ASPEN guidelines also suggest that patients at high risk for aspiration receive a continuous infusion to decrease intolerance to gastric feeding (5). The Academy of Nutrition and Dietetics recommends small bowel feeding tube placement for critically ill mechanically ventilated patients requiring enteral nutrition (Grade II) (3). Some large research studies with ventilator-associated pneumonia (VAP) as a primary outcome suggest that small bowel enteral nutrition vs. gastric enteral nutrition reduces VAP (Grade II) (3).
- Use a 50-mL or 60-mL syringe to check GRVs in nasogastric-fed or gastrostomy-fed patients before each intermittent or bolus feeding (52). Feeding tubes with a diameter smaller than 10 French may be unreliable in determining residuals (58,59). The GRV should be checked before bolus feedings,

intermittently during continuous pump feedings, or when there are signs of feeding intolerance (eg. abdominal distention or vomiting). Guidelines for GRV cut-off values vary among organizations (3,5). The Academy of Nutrition and Dietetics recommends against holding enteral nutrition when the GRV is less than 500 mL in the absence of signs of intolerance (e.g., abdominal distention, nausea, vomiting) in critically ill adult patients (3). Holding enteral nutrition when the GRV is less than 500 mL is associated with delivery of less enteral nutrition (Fair, Grade III) (3,52). The ASPEN guidelines recommend that GRVs in the range of 200 to 500 mL should raise concern and lead to the implementation of measures to reduce the risk of aspiration; however, feedings should not automatically be stopped if the GRV is less than 500 mL and there are no other signs of intolerance (5). These ASPEN guidelines are based on studies that demonstrated no greater risk for regurgitation, aspiration, or pneumonia when a 250-mL to 500-mL cutoff value for GRV was used (5). In any case, the clinician should consider the individual patient circumstance and adhere to organization-specific policies when determining cut-off values for GRV. If a patient has reoccurring elevated GRVs that exceed target cut-off goals, the underlying causes should be evaluated, and preventive interventions should be provided. For example, consider a small-bowel tube placement in patients who have more than 250 mL GRV or formula reflux in two consecutive measures (Grade II) (3). Small-bowel tube placement (postpyloric position) is associated with reduced GRV (Grade I) (3). Adequately powered studies have not been conducted to evaluate the impact of GRV on aspiration pneumonia (3). Patients with GRVs ranging from 200 to 500 mL may benefit from medications that stimulate gastric motility (eg, metoclopramide, erythromycin, or narcotic antagonists such as naloxone Patients with consistently high GRVs must be evaluated to exclude and alvimopan) (Grade II) (3,5). underlying medical problems (eg, ileus, bowel impaction, gastroparesis, or pancreatitis) that may cause feeding intolerance.

- Blue dye should not be added to enteral formulas to detect aspiration. The risk of using blue dye outweighs any perceived benefit. The presence of blue dye in tracheal secretions is not a sensitive indicator for aspirations (Grade III) (3,5,60).
- If not contraindicated, maintain the head of the patient's bed at a 30° 45° angle during feedings to reduce the risk of aspiration pneumonia (Grade II) and the reflux of gastric contents into the esophagus and pharynx (Grade I) (3,4). If bolus feeding, keep the head of the bed in this position for 30 to 60 minutes after feeding.
- If the patient has a history of gastroparesis or repeated high GRVs ranging from 200 to 500 mL, then consider the use of a promotility agent, if there are no contraindications (Grade II) (3). Promotility agents (eg, metoclopramide or others listed above (5)) have been associated with increased gastrointestinal transit, improved feeding tolerance, improved enteral nutrition delivery, and possibly a reduced risk of aspiration (Grade II) (3).
- Use of chlorhexidine mouthwash twice a day should be considered to reduce the risk of ventilatorassociated pneumonia (5). Two studies have demonstrated that the use of chlorhexidine mouthwashes twice daily reduced the incidence of respiratory infection and nosocomial pneumonia in patients undergoing heart surgery (5).

Diarrhea and Probiotics

• Diarrhea in the ICU patient receiving enteral nutrition should prompt an investigation to distinguish between infectious diarrhea, osmotic diarrhea, or malabsorption. Check for excessive intake of hyperosmolar medications and substances, such as sorbitol; the use of broad-spectrum antibiotics that can cause pseudomembranous colitis due to an overpopulation of *Clostridium difficile*; and other infectious etiologies. *Clostridium difficile* is prevalent and increasingly severe nosocomial infection that presently accounts for 15% to 25% of hospital cases of antibiotic-associated diarrhea (61). *S. Boulardii* has been effective as an adjunctive therapy for the treatment and reoccurrence of *C. difficile* (62, 63, 64). The dosage of 250 mg twice daily for prevention and 250 mg four times daily to prevent recurrent *C difficile* toxins as an adjunct to antibiotic treatment with metronidazole or vancomycin has been suggested (61). Most episodes of nosocomial diarrhea are mild and resolve independently (5). Soluble fiber (eg, guar gum) may be beneficial for fully resuscitated, hemodynamically stable, critically ill patients who receive enteral nutrition and develop diarrhea (5).

Metabolic/laboratory data monitoring guidelines: Serum protein levels are no longer considered to be reliable or valid as an indicator of a critical care patient's nutritional status or response to nutrition therapies (64). Evidence has shown that serum protein levels are markers of the stress response and disease severity, rather than indicators of nutritional status or protein requirements. For monitoring the adequacy of protein intake, the most useful (although still limited) laboratory indicator may be nitrogen balance, which reflects the adequacy of protein intake in matching catabolic demand (64). (Refer to Section II: Estimation of Protein

Requirements or Section III: Burns.)

In critically ill adult patients the promotion of glucose levels between 140 and 180 mg/dL is currently recommended by the Academy of Nutrition and Dietetics (Grade II) (3). Tight blood glucose control (80 to 110 mg per dL) is not associated with reduced hospital length of stay (LOS), infectious complications, cost of medical care, days on mechanical ventilation or mortality and increases risk of hypoglycemia as previously suggested (Grade II) (3). However, glucose level > 180 mg/dL is associated with increased mortality in the critically ill adult patients (Grade II) (3). In surgical (primarily cardiac) patients, tight control of blood glucose reduces the risk of some types of infectious complications. However, this effect has not been consistently demonstrated in other types of ICU patients (Grade II) (3). ASPEN guidelines recommend a target range of 100 to 150 mg/dL when providing nutrition support therapy to critically ill patients (5). In addition, specific recommendations are also provided for critically ill patients with diabetes. Dietitians should be involved in promoting the attainment of blood glucose control when providing nutrition support therapies and should refer to emerging evidence, practice guidelines, and facility-specific protocols for recommendations (3.5). Refer to Section C: Medical Nutrition Therapy for Diabetes Mellitus for specific glycemic goals in managing hospitalized patients with diabetes mellitus.

Suggested laboratory monitoring guidelines include:

- daily measurements of sodium, potassium, chloride, CO₂, blood urea nitrogen, creatinine, and glucose levels until stable, then biweekly to weekly measurements
- baseline measurements of prealbumin or albumin for patients, such as chronic kidney disease patients, to determine the morbidity, mortality, or severity of disease or inflammatory status (not reliable in critically ill patients)
- baseline measurements of liver function tests
- daily measurements of calcium, magnesium, and phosphorus until stable, then weekly measurements
- baseline complete blood cell count, then measured as needed
- a 24-hour urine analysis for urine urea nitrogen (or total urea nitrogen) once the goal rate of tube feeding is attained, then weekly measurements until stable

Other patient monitoring guidelines:

- Weigh the patient at least once per week.
- Maintain daily records of intake, output, and bowel movements.
- Monitor vital signs to determine whether a systemic inflammatory response is present (5). The systemic inflammatory response syndrome is nonspecific and common in critical care patients. Its presence on the second day after ICU admission prognosticates morbidity (organ failure) and mortality and therefore might be one criterion in identifying the patient at higher risk for complications and who might benefit from early initiation of enteral feeding (64). Signs of systemic inflammatory response syndrome include the following (65):
 - body temperature of >38.0°C or <36.0°C
 - heart rate of >90 beats/minute
 - breathing rate of >20 breaths/minute (often obscured by mechanical ventilation)
 - leukocyte count of >12,000 mm³ or <4,000 mm³, or a left shift (>10% bands)

Medications via enteral feeding tubes: Feeding tubes should be irrigated with at least 15 mL of warm purified or sterile water (or saline) before and immediately after the administration of medications (52). Since crushed medications can clog tubes, liquid medications should be used when possible. Many oral medicines formulated for slow release may be surrounded by an enteric coating and should not be crushed and administered through the feeding tube. Multiple types of medication should be administered separately (52). Temporary cessation of enteral feeding may be indicated for 1 hour before and 1 hour after the administration of phenytoin sodium (Dilantin), a commonly used anticonvulsant medication, because components of the enteral formula, such as calcium, decrease the bioavailability of this drug (15,66-68).

Transitional feedings, enteral to oral or supplemental parenteral nutrition: Depending on the swallowing function of the patient, oral intake should begin with liquids and advance to appropriate foods as tolerated. When oral intake reaches 500 kcal or more, the dosage of tube feedings may be proportionately tapered. Switching the patient from a continuous tube feeding to night tube feeding only or discontinuing tube feeding 1 to 2 hours before meals will often stimulate the appetite and speed transition to adequate oral intake. When oral intake consistently meets or exceeds 60% of the patient's energy requirements and 100%

of the fluid requirements, discontinuation of tube feedings should be considered (3,5). If a critically ill patient is unable to meet energy requirements (100% of the target energy goal) after 7 to 10 days by the enteral route alone, supplemental parenteral nutrition should be considered (5). The provision of supplemental parenteral nutrition prior to this 7- to 10-day period does not improve the patient's nutritional status and may be detrimental (5).

See Section III: Clinical Nutrition Management, Enteral Nutrition: Management of Complications

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. ASPEN Board of Directors and Standards Committee. American Society for Parenteral and Enteral Nutrition. Definition of terms, style, and conventions used in A.S.P.E.N. guidelines and standards. *Nutr Clin Pract.* 2005;20:281-285.
- Joint Standards Task Force of ASPEN and the American Dietetic Association Dietitians in Nutrition Support Practice Group. Russell M, Stieber M, Brantley S, Freeman AM, Lefton J, Malone AM, Roberts S, Skates J, Young LS. American Society for Parenteral and Enteral Nutrition and American Dietetic Association: standards of practice and standards of professional performance for registered dietitians (generalist, specialty, and advanced) in nutrition support. J Am Diet Assoc. 2007;107:1815-1822.
- 3. *Critical Illness Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- 4. Brantley SL and Mills B. Overview of enteral nutrition. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum* 2nd *ed.* Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:170-184.
- McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; ASPEN Board of Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr. 2009;33:277-316.
- 6. Neumayer LA, Smout RJ, Horn HG, Horn SD. Early sufficient feeding reduces length of stay and charges in surgical patients. *J Surg Res.* 2001;95:73-77.-
- 7. Farber MS, Moses J, Korn M. Reducing costs and patient morbidity in the enterally fed intensive care unit patients. J Parenter Enteral Nutr. 2005;29:S562-S569.
- 8. Kudsk DA, Minard G, Croce MA, Brown RO, Lowrey TS, Pritchard FF, Dickerson RN, Fabian TC. A randomized trial of isonitrogenous enteral diets after severe trauma: an immune-enhancing diet reduces septic complications. *Ann Surg.* 1996;224:531-543.
- 9. Magnotti LJ, Deitch EA. Burns, bacterial translocation, gut barrier function, and failure. *J Burn Care Rehabil.* 2005; 26:383-391.
- 10. Choi J, O'Connell TX. Safe and effective early postoperative feeding and hospital discharge after open colon resection. *Am Surg.* 1996;62:853-856.
- 11. Ugarte F. Re: Safe and effective early postoperative feeding and hospital discharge after open colon resection. *Am Surg.* 1997;63:565-566.
- 12. Magnotti LJ. Mechanism and significance of gut barrier function and failure. In: Rolandelli RH, Bankhead R, Boullata J (eds). *Clinical Nutrition Enteral and Tube Feeding*. 4th ed. Philadelphia, PA: WB Saunders; 2005;23-31.
- 13. McClave SA, Change WK. When to feed the patient with gastrointestinal bleeding. *Nutr Clin Pract.* 2005;20:544-550.
- 14. Cresci G, Lefton J, and Halasa Esper D. Enteral formulations. In: Mueller CM, ed. *The A.S.P.E.N. Nutrition Support Core Curriculum 2nd* ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:185-205.
- 15. Charney P, Russell MK. Enteral formulations: standard. In: Rolandelli RH, ed. *Clinical Nutrition: Enteral and Tube Feeding.* 4th ed. Philadelphia, Pa: Elsevier Saunders; 2005:216-223.
- 16. Malone A. Enteral formula selections: a review of selected product categories. Pract Gastroenterol. 2005;24:44-74.
- Spapen H, Diltoer M, Malderen CV, Opdenacker G, Suys E, Huyghens L. Soluble fiber reduces the incidence of diarrhea in septic patients receiving total enteral nutrition: a prospective, double blind, randomized, and controlled trial. *Clin Nutr.* 2001;20:301-305.
- 18. Rushdi TA, Pichard C, Khater YH. Control of diarrhea by fiber-enriched diet in ICU patients on enteral nutrition: a prospective randomized controlled trial. *Clin Nutr.* 2004;23:1344-1352.
- 19. Frankenfield DC, Beyer PL. Soy-polysaccharide fiber: effect on diarrhea in tube-fed, head-injured patients. *Am J Clin Nutr.* 1989;50:533-538.
- 20. McIvor AC, Meguid MM, Curtas S, Warren J, Kaplan DS. Intestinal obstruction from cecal bezoar; a complication of fiber-containing tube feedings. *Nutrition*. 1990;6:115-117.
- 21. McClave SA, Chang WK. Feeding the hypotensive patient: does enteral feeding precipitate or protect against ischemic bowel? *Nutr Clin Pract.* 2003;18:279-284.
- 22. McIvor AC, Meguid MM, Curtas S, Warren J, Kaplan DS. Intestinal obstruction from cecal bezoar: a complication of fiber-containing tubefeedings. *Nutrition*. 1990;6:115-117.
- 23. Gottschlich MM. Selection of optimal lipid sources in enteral and parenteral nutrition. Nutr Clin Pract. 1992;7:152-165.
- 24. Kinsella JE, Lokesh B. Dietary lipids, eicosanoids, and the immune system. *Crit Care Med.* 1990;18:S94-S113.
- 25. Gottschlich MM, Jenkins M, Warden GD, Baumer T, Havens P, Snook JT, Alexander JW. Differential effects of three enteral dietary regimens on selected outcome variables in burn patients. *J Parenter Enteral Nutr*. 1990;14:225-236.
- Kenler AS, Swails WS, Driscoll DE, DeMichele SJ, Daley B, Babineau TJ, Peterson MB, Bistrian BR. Early enteral feeding in postsurgical cancer patients: fish oil structured lipid-based polymeric formula versus a standard polymeric formula. *Ann Surg.* 1996;223:316-333.
- 27. Gadek JE, DeMichele SJ, Karlstad MD, Pacht ER, Donahoe M, Albertson TE, VanHoozen C, Wennberg AK, Nelson JL, Noursalehi M. Enteral Nutrition in ARDS Study Group. Effect of enteral feeding with eicosapentaenoic acid, gamma-linolenic acid, and antioxidants in patients with acute respiratory distress syndrome. *Crit Care Med.* 1999;27:1409-1420.
- 28. Singer P, Theilla M, Fisher H, Gibstein L, Grozovski E, Cohen J. Benefit of an enteral diet enriched with eicosapentaenoic acid and gamma-linolenic acid in ventilated patients with acute lung injury. *Crit Care Med.* 2006;34(4): 1033-1038.
- 29. Senkal M, Kemen M, Homann HH, Eickhoff U, Baier J, Zumtobel V. Modulation of postoperative immune response by enteral nutrition with a diet enriched with arginine, RNA, and omega-3 fatty acids in patients with upper gastrointestinal cancer. *Eur J Surg.*

Enteral Nutrition Support Therapy for Adults

1995;161:115-122.

- Jacobs DG, Jacobs DO, Kudsk KA, Moore FA, Oswanski MF, Pooole GV, Sacks G, Scherer LR 3rd, Sinclair KE. Practice management guidelines for nutrition support of the trauma patient. J Trauma. 2004;57:660-679.
- 31. Stehle P. Nutrition support in critical illness: amino acids. In: Cynober L, Moore FA, eds. *Nutrition in Critical Care*. Nestle Nutrition Workshop Series Clinical and Performance Program, Basel, Nestec, Ltd; Vevey/S. Karger AG. 2003;8:57-73.
- 32. Savy GK. Enteral glutamine supplementation: clinical review and practical guidelines. Nutr Clin Pract. 1997;12:259-262.
- 33. Jones C, Palmer TE, Griffiths RD. Randomized clinical outcome study of critically ill patients given glutamine-supplemented enteral nutrition. *Nutrition*. 1999;15:108-115.
- 34. Jensen GL, Miller RH, Talabiska DG, Fish J, Gianferante L. A double-blind, prospective, randomized study of glutamine-enriched compared with standard peptide-based feeding in critically ill patients. *Am J Clin Nutr*. 1996;64:615-621.
- 35. Coghlin TM, Wong RM, Negrin RS, Shizuru JA, Johnston LJ, Blume KG, Stockerl-Goldstein KF. Effect of oral glutamine supplementation during bone marrow transplantation. *J Parenter Enteral Nutr.* 2000;24:61-66.
- Ziegler TR, Benfell K, Smith RJ, Young LS, Brown E, Ferrari-Baliviero E, Lowe DK, Wilmore DW. Safety and metabolic effects of Lglutamine administration in humans. J Parenter Enteral Nutr. 1990;14:137S-146S.
- Marchesini G, Giampaolo B, Manuela M, Bianchi G, Merli M, Amodio P, Panella C, Loguercio C, Rossi Fanelli F, Abbiati R, Italian BCAA Study Group. Nutritional supplementation with branched-chain amino acids in advanced cirrhosis: a double blind, randomized trial. *Gastroenterology*. 2003; 124:1792-1801.
- Blei AT, Cordoba J, and the Practice Parameter Committee of the American College of Gastroenterology. Hepatic encephalopathy. *Am J Gastroenterol.* 2001;96:1968-1975.
- 39. Malone AM. The clinical benefits and efficacy of using specialized enteral feeding formulas. Support Line. 2002;24:3-11.
- 40. Russell MK, Charney P. Is there a role for specialized enteral nutrition in the intensive care unit? *Am J Kidney Dis.* 2005;46:387-405.
- 41. Stratton RJ, Bircher G, Fongue D, Stenvinkel P, deMutsert R, Engfer M, Elia M. Multinutrient oral supplements and tube feeding in maintenance dialysis: a systematic review and meta-analysis. *Am J Kidney Dis.* 2005;46:387-405.
- 42. Talpers SS, Romberger DJ, Bunce SB, Pingleton SK. Nutritionally associated increased carbon dioxide production: excess total calories vs high proportion of carbohydrate calories. *Chest.* 1992;102:551-555.
- 43. Akrabawi SS, Mobarhan S, Stoltz RR, Ferguson PW. Gastric emptying, pulmonary function, gas exchange, and respiratory quotient after feeding a moderate versus high fat enteral formula meal in chronic obstructive pulmonary disease patients. *Nutrition*. 1996;12:260-265.
- 44. Craig LD, Nicholson S, Silverstone FA, Kennedy RD. Use of a reduced-carbohydrate, modified-fat enteral formula for improving metabolic control and clinical outcomes in long-term care residents with type 2 diabetes: results of a pilot trial. *Nutrition*. 1998;14:529-534.
- 45. Leon-Sanz M, Garcia-Luna PP, Sanz-Paris A, et al for the SPAI-97-004 Study Cooperative Group. Glycemic and lipid control in hospitalized type 2 diabetic patients: evaluation of 2 enteral nutrition formulas (low carbohydrate-high monounsaturated fat vs. high carbohydrate). *J Parenter Enteral Nutr.* 2005;29:21-29.
- 46. Diabetes Mellitus (DM) Type 1 and Type 2 Evidence-Based Nutrition Practice Guideline for Adults. Academy of Nutrition and Dietetics; 2008. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- American Diabetes Association. Nutrition recommendations and interventions for diabetes. Diabetes Care. 2008;31(suppl1): 61S-78S.
 Charney P, Hertzler SR. Management of blood glucose and diabetes in the critically ill patient receiving enteral feeding. *Nutr Clin Pract.* 2004;19:129-136.
- 49. Food and Nutrition Board, National Academy of Sciences. *Recommended Dietary Allowances*. 10th ed. Washington, DC: National Academy Press; 1989.
- 50. Kleiner SM. Water: an essential but overlooked nutrient. *J Am Diet Assoc.* 1999;99:200-206.
- 51. *Hydration Evidence-Analysis Project.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2007. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- 52. Bankhead R, Boullata J, Brantley S, Corkins M, Guenter P, Krenitsky J, Lyman B, Metheny NA, Mueller C, Robbins S, Wessel J; ASPEN Board of Directors. Enteral nutrition practice recommendations. *J Parenter Enteral Nutr.* 2009;33:122-167.
- Rollins CJ. Drug-nutrient interactions. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:298-312.
- 54. Fang, JC, Bankhead RR, and Kinikini M. Enteral access devices. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:207-217.
- 55. Morrison Nutrition Practice Guideline Enteral Nutrition. In: Inman-Felton A, Smith KG. *Morrison Nutrition Practice Guidelines*. Atlanta, Ga: Morrison Management Specialists Inc; 2009. Available at: <u>www.morrisontoday.com/Documents/Nutrition/MHFS</u> <u>Nutrition</u>.
- 56. Kattelmann K, Hise M, Russell M, Charney P, Stokes M, Compher C. Preliminary evidence for a medical nutrition therapy protocol: enteral feedings for critically ill patients. *J Am Diet Assoc.* 2006; 106:1226-1241.
- 57. McClave SA, DeMeo MT, Delegge MH, DiSario JA, Heyland DK, Maloney JP, Metheny NA, Moore FA, Scolapio JS, Spain DA, Zaloga GD. North American Summit on Aspiration in the Critically Ill: consensus statement. *J Parenter Enteral Nutr.* 2002;26:S80-S85.
- 58. American Gastroenterological Association. American Gastroenterological Association technical review on tube feeding for enteral nutrition. *Gastroenterology*. 1995;108:1282-1301.
- 59. Russell M, Cromer M, Grant J. Complications of enteral nutrition therapy. In: Gottschlich MM, ed. *The Science and Practice of Nutrition Support: A Case-Based Core Curriculum*. Dubuque, Iowa: Kendall/Hunt Publishing Co; 2001.
- 60. Maloney JP, Ryan TA, Brasel KJ, Binion DG, Johnson DR, Halbower AC, Frankel EH, Nyffeler M, Moss M. Food dye use in enteral feedings: a review and a call for a moratorium. *Nutr Clin Pract.* 2002; 17:169-181.
- 61. Mullin GE. Probiotics and digestive health. *Nutr Clin Pract.* 2012;27:300-302.
- 62. McFarland LV. Meta-analysis of probiotics for the prevention of traveler's diarrhea. Travel Med Infect Dis. 2007;5:97-105.
- 63. Tung JM, Dolovich LR, Lee CH. Prevention of Clostridium difficile infection with Saccharomyces boulardii: a systematic review. Can J Gastroenterol. 2009;23:817-821.
- 64. Critical care. Nutrition care. In: *The Academy of Nutrition and Dietetics Nutrition Care Manual*. Updated annually. Available at: nutritioncaremanual.org. Accessed January 17, 2013.
- 65. Robertson CM, Coopersmith CM. The systemic inflammatory response syndrome. Microbes Infect. 2006;8:1382-1389.
- 66. Doak KK, Haas CE, Dunnigan KJ, Reiss RA, Reiser JR, Huntress J, Altavela JL. Bioavailability of phenytoin acid and phenytoin sodium with enteral feedings. *Pharmacotherapy*. 1998;18:636-645.

- 67. Faraji B, Yu PP. Serum phenytoin levels of patients on gastrostomy tube feeding. J Neurosci Nurs. 1998;30:55-59.
- 68. Gilbert S, Hatton J, Magnuson B. How to minimize interaction between phenytoin and enteral feedings: two approaches. *Nutr Clin Pract.* 1996;11:28-31.

Bibliography

- Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum.* 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012.
- *Critical Illness Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- Position of the American Dietetic Association: ethical and legal issues in nutrition, hydration, and feeding. J Am Diet Assoc. 2008;108:873-882.
- Joint Standards Task Force of ASPEN and the American Dietetic Association Dietitians in Nutrition Support Practice Group. Russell M, Stieber M, Brantley S, Freeman AM, Lefton J, Malone AM, Roberts S, Skates J, Young LS. American Society for Parenteral and Enteral Nutrition and American Dietetic Association: standards of practice and standards of professional performance for registered dietitians (generalist, specialty, and advanced) in nutrition support. *J Am Diet Assoc.* 2007;107:1815-1822.
- Bankhead R, Boullata J, Brantley S, Corkins M, Guenter P, Krenitsky J, Lyman B, Metheny NA, Mueller C, Robbins S, Wessel J; ASPEN Board of Directors. Enteral nutrition practice recommendations. *J Parenter Enteral Nutr.* 2009;33:122-167. Also available at: www.eatright.org ("Evidence-Based Practice" link). Accessed September 20, 2010.

PARENTERAL NUTRITION SUPPORT FOR ADULTS

Overview

Parenteral nutrition is the provision of nutrients intravenously. Since the 1960s, major advances have been seen in the technique, delivery, and formulation of parenteral nutrition (1). The use of guidelines for practice has improved nutritional markers and reduced the rates of complications of patients receiving parenteral nutrition (1,2). The two primary types of parenteral nutrition are central parenteral nutrition (CPN) and peripheral parenteral nutrition (PPN).

In CPN (or total parenteral nutrition), nutrients are provided through a large-diameter vein, usually the superior vena cava, by access of the subclavian or internal jugular vein. The CPN formulas are hyperosmolar (1,300 to 1,800 mOsm/L) and consist of dextrose (15% to 25%), amino acids, and electrolytes to fully meet the patient's nutritional needs. The nutrient and fluid composition of CPN can be adjusted to meet the individual needs of patients who require fluid restriction (3). When venous access for the delivery of nutrients is required for longer than 2 weeks, CPN is indicated because it can be maintained for prolonged periods (1).

In PPN, a peripheral vein provides venous access. This form of parenteral nutrition is similar to CPN except that lower formula concentrations must be used because the peripheral vein can only tolerate solutions that are less than 900 mOsm/L. Compared with CPN formulas, PPN formulas have lower concentrations of dextrose (5% to 10%) and amino acids (3%). Because higher concentrations cannot be infused into the peripheral vein, PPN requires larger fluid volumes to provide energy and protein doses comparable to the doses provided by CPN. The larger fluid volume poses a challenge for patients who require fluid restriction. The maximum volume of PPN that is usually tolerated is 3 L/day (125 mL/hour). Repletion of nutrient stores is not a goal of PPN, and it should not be used in severely malnourished patients (1). The use of PPN is indicated only for mildly to moderately malnourished patients who are unable to ingest adequate energy orally or enterally, or for patients in whom CPN is not feasible. Typically, PPN is used for short periods (5 days to 2 weeks) because of limited tolerance and the vulnerability of peripheral veins (eg, risk of peripheral venous thrombophlebitis) (1).

Nutrition Assessment

A meta-analysis of parenteral nutrition suggests that this route of nutrition support increases infection rates without measurable beneficial outcomes when compared to controls (4). Parenteral nutrition is costly and may result in serious complications and risks if the patient is not monitored closely (5,6). Steps must be taken to maximize the benefit and efficacy of parenteral nutrition while reducing the inherent risks of hyperglycemia, immune suppression, increased oxidative stress, and potential infectious morbidity (6). Patients who are candidates for parenteral nutrition must be carefully evaluated, and steps must be taken to provide the most effective dose, content, monitoring process, and supplemental additives (6).

Indications (1-3)

Guidelines for the implementation of parenteral nutrition have been developed by the American Society for Parenteral and Enteral Nutrition (ASPEN) (1,6). Parenteral nutrition is indicated for patients who are unable to receive adequate nutrients via the enteral route (eg, patients who have a nonfunctional or severely compromised gastrointestinal tract). The indications for parenteral nutrition include (1,3,6,7):

- malnutrition when enteral nutrition is not feasible (6)
- major surgical procedures where the preoperative assessment indicates that enteral nutrition is not feasible through the perioperative period, and the patient is malnourished (6)
- perioperative support of moderately to severely malnourished patients with gastrointestinal cancer
- after 7 days of hospitalization when enteral nutrition has not been feasible or has been insufficient to consistently meet the target energy goal (6)
- severe malabsorption caused by massive bowel resection (\geq 70% resected) or severe diarrhea
- intractable vomiting
- severe acute, necrotizing pancreatitis in patients who have a history of poor tolerance to enteral nutrition or for whom enteral feeding is not possible at or beyond the ligament of Treitz (6,8,9) (Parenteral nutrition should not be initiated until after the first 5 days of hospitalization.) (6)
- paralytic ileus requiring prolonged bowel rest
- complete intestinal obstruction
- enterocutaneous fistula with an output greater than 500 mL/day (A trial of enteral nutrition can be

considered when the output is less than 200 mL/day or when enteral access may be placed distal to the fistula.) $_{(1)}$

- acute exacerbations of inflammatory bowel disease (eg, Crohn disease) complicated by fistulas
- radiation therapy or allogeneic bone marrow transplantation
- hemodynamic instability with mean arterial pressure < 70 mm Hg
- increased doses of vasopressors
- increased need for mechanical ventilation
- worsening signs of GI intolerance

Contraindications (1,3,6,7)

Parenteral nutrition is not indicated for patients:

- who have a fully functional and accessible gastrointestinal tract
- for whom venous access cannot be obtained
- whose prognosis does not warrant aggressive nutrition support
- whose need for parenteral nutrition is expected to be less than 7 days (6)
- for whom the risks of parenteral nutrition exceed the potential benefits, such as in cases of severe hyperglycemia (>300 mg/dL), azotemia, encephalopathy, hyperosmolality (>350 mOsm/kg), and severe fluid and electrolyte disturbances (3)

Nutrition Intervention

Parenteral Feeding Formulations

The osmolarity of a parenteral formula depends on the energy substrate mixture, primarily the dextrose (5 mOsm/g), amino acid (10 mOsm/g), and electrolyte (1 mOsm/mEq) content (3). For example, the estimated osmolarity of a parenteral feeding formula that provides 150 g/L of dextrose, 50 g/L of amino acids, and 150 mEq/L of electrolyte additives is 1,400 mOsm/L (3). The maximum osmolarity tolerated by a peripheral vein is 900 mOsm/L (10,11). Formulas for peripheral vein administration usually require more fluid and a higher content of fat as an energy source than formulas for central access, as lipids are isotonic (3). Midline catheters can be used to improve peripheral vein tolerance to the nutrition infusion because these catheters can access larger veins where the blood flow may dilute the parenteral feeding formulations to a more tolerable concentration (3). Fluid-restricted, energy-dense formulations should be considered for patients with acute respiratory failure (6).

Nutrient Sources and Indications

Energy requirements: Mildly permissive underfeeding should be considered, at least initially, for all adult critically ill intensive care unit (ICU) patients receiving parenteral nutrition (6). The ultimate goal or dose of parenteral feeding should be 80% of the patient's energy requirements (6). This strategy avoids the potential for insulin resistance, greater infectious morbidity, prolonged duration of mechanical ventilation, and increased length of hospitalization that is associated with excessive energy intake (6). As the patient stabilizes, parenteral nutrition may be increased to meet energy requirements (6). For obese patients (body mass index (BMI) >30 kg/m²), the dose of parenteral nutrition with regard to protein and energy provision should follow the same recommendations given for enteral nutrition (6). Guidelines for critically ill adult patients from ASPEN and the Society of Critical Care Medicine recommend permissive underfeeding or hypocaloric feeding with enteral nutrition for obese patients (6). The goal of the enteral nutrition regimen for obese patients should not exceed 60% to 70% of target energy requirements or 11 to 14 kcal/kg actual body weight per day (or 22 to 25 kcal/kg ideal body weight per day) (6).

Carbohydrate sources: The most commonly used source of carbohydrate is dextrose. Dextrose in its monohydrate form provides 3.4 kcal/g. Commercial dextrose solutions are available in concentrations ranging from 2.5% to 70% (11). These solutions are acidic, with a pH ranging from 3.5 to 6.5 (11). Formulas with final dextrose concentrations greater than 10% are reserved for central venous access (11). Sugar alcohol glycerol is a less frequently used carbohydrate source, and it provides 4.3 kcal/g. Parenteral formulas containing sugar alcohol glycerol are protein sparing and induce a smaller insulin response as compared to dextrose-based solutions (12-14). More research is needed to determine the efficacy of the routine use of parenteral formulas that contain sugar alcohol glycerol.

Carbohydrate requirements: The minimum requirements for dextrose are 1 mg/kg per minute (approximately 100 g/day for a 70-kg person). The maximum amount of carbohydrate tolerated is approximately 5 to 7 mg/kg per minute (1,15). Hyperglycemia, which is caused by various factors including stress, is the most common complication of parenteral nutrition (11). When carbohydrate is provided in

Parenteral Nutrition Support for Adults

excess, hyperglycemia, hepatic steatosis, and increased CO₂ production can occur. Maintaining glucose in the range of 80 to 110 mg/dL has been associated with decreased morbidity and mortality in cardiac surgical intensive care patients (16-18). However, recent studies have suggested that moderate glucose control (140 to 180 mg/dL) reduces the risk of hypoglycemia and hypoglycemia-associated mortality as compared to stricter glucose control (80 to 110 mg/dL) (6). Considering this evidence, ASPEN guidelines recommend targeting a more moderate range of 100 to 150 mg/dL when providing nutrition support therapy to critically ill patients (6). The *Standards of Medical Care in Diabetes*, updated annually by the American Diabetes Association, recommends a target glucose level of 140 to 180 mg/dL in critically ill patients with existing diabetes mellitus (19). The Academy of Nutrition and Dietetics (AND) recommends glucose levels targeting 140 to 180 mg/dL for all critically ill patients (18). Dietitians should collaborate with the medical team to identify protocols for optimal glucose control based on clinical management guidelines (6).

For patients with hyperglycemia, the parenteral-nutrition dextrose should be started conservatively and gradually increased to the patient's individualized goal rate. It is suggested PN be initiated at half the patients estimated energy needs or 150 to 200 grams dextrose in the first 24 hours, or approximately 100 grams dextrose if patient is already hyperglycemic or requiring insulin therapy. (20). It is recommended that the goal CHO rate should not exceed 4 to 5 mg/kg per minute or 20 to 25 kcal/kg per day from CHO (20). Capillary blood glucose should be measured at least every 6 hours and more frequently in hyperglycemic patients (19). Regular insulin can be provided subcutaneously, intravenously via insulin infusion, or added directly to the parenteral solution (18.20). An insulin drip provides more consistent and safe glucose control (19). Blood glucose concentrations can be controlled by an initial insulin regimen of 0.05 to 0.1 U/g of dextrose in the parenteral solution or by 0.15 to 0.2 U/g of dextrose in hyperglycemic patients (20). When added to the solution, it is recommended that two thirds of the total amount of sliding-scale insulin required over 24 hours be added to the next day's parenteral formula (20). A proportional increase in fat may be necessary to increase the energy provided to patients whose blood glucose levels are difficult to control (20). Rarely, hyperglycemia is caused by a chromium deficiency. If insulin is ineffective or a chromium deficiency is confirmed, increasing the chromium contained in the parenteral formula may be appropriate (21). Refer to Section III: Metabolic Complications of Central Parenteral Nutrition and Section IC: Medical Nutrition Therapy for Diabetes Mellitus for specific glycemic goals in managing hospitalized patients with diabetes mellitus.

Patients at risk of developing refeeding syndrome should be monitored closely. Refeeding syndrome refers to metabolic and physiologic shifts of electrolytes and minerals (eg, phosphorus, magnesium, and potassium) caused by aggressive nutrition support (1,22). Carbohydrate delivery stimulates insulin secretion, which causes an intracellular shift of these electrolytes and minerals with the potential for severe hypophosphatemia, hypomagnesemia, and hypokalemia (19,22). Symptoms of refeeding syndrome include fatigue, lethargy, muscle weakness, edema, cardiac arrhythmia, and hemolysis (19,22). Patients who have marginal nutrient stores secondary to a disease or medical therapy are at the greatest risk for refeeding syndrome; these patients should initially receive 15 to 20 kcal/kg of formula, 100 – 200 grams CHO or no more than 2 mg/kg/min per day, and then the amount of parenteral formula should be slowly increased (20,22). (Refer to Section III: Metabolic Complications of Central Parenteral Nutrition.)

Protein sources: Crystalline amino acids, which provide 4 kcal/g, are the most common source of protein in parenteral formulas. Standard or balanced amino acid products are mixtures of essential and nonessential amino acids ranging in concentrations from 3% to 20%, although 8.5% and 10% are most frequently used for parenteral nutrition compounding (11). Most commercially available amino acid formulations also contain electrolytes, buffers, or both. Modified or special amino acid products have been formulated for certain disease states and conditions in which conventional amino acid solutions may not be well tolerated (eg, renal failure and hepatic failure) (11). The contribution of these formulas to an improvement in overall clinical outcome is debatable. These formulas usually cost much more than conventional amino acid solutions. Therefore, the clinician should evaluate the cost in light of the potential benefit to the patient.

ASPEN guidelines for nutrition support are the basis for the following discussion of specialized amino acid formulas (1,6):

• **Formulations for liver disease:** Standard formulations should be used in ICU patients with acute and chronic liver disease (6). Protein restriction should be avoided in patients with liver failure (6). Formulas that contain high levels of branched-chain amino acids (BCAA) and low levels of aromatic amino acids are designed to correct abnormal amino acid profiles associated with hepatic encephalopathy, as BCAA are metabolized independently of liver function. ASPEN guidelines recommend that BCAA formulas only be

used in the rare case of a patient who has hepatic encephalopathy that is refractory to standard treatment with luminal-acting antibiotics and lactulose (1,6).

- Formulations for renal disease: Essential amino acids are the primary protein source in formulations designed for patients with renal disease. This formula design is based on the theory that nonessential amino acids can be physiologically recycled from urea, while the essential amino acids must be provided by the diet (11). These formulas have no statistically significant advantages when compared to standard amino acid formulas (1,23). The ASPEN guidelines suggest that ICU patients with acute renal failure receive standard enteral or parenteral formulations and that the standard ICU recommendations for protein and energy provision should be followed (6). A specialty formulation designed for renal failure (with the appropriate electrolyte profile) may be considered for patients who have significant electrolyte abnormalities (6). Patients receiving hemodialysis or continuous renal replacement therapy should receive increased amounts of protein, up to a maximum of 2.5 g/kg per day (6). Protein should not be restricted in patients with renal insufficiency as a means to avoid or delay the initiation of dialysis therapy (6).
- **Concentrated amino acid formulations:** Highly concentrated (15% to 20%) amino acid formulations are available for use when fluid restriction is required. These formulations are similar in composition to standard amino acid formulas, except that they may contain larger amounts of acetate (11). However, chloride salts may be used to balance the chloride-to-acetate ratio in the final PN formulation to avoid iatrogenic acid-base disturbances (11).
- **Pediatric formulations:** Pediatric formulas have been designed to meet the unique amino acid requirements of neonates, infants, and children in order to promote weight gain, nitrogen balance, and growth and to avoid an excess of certain amino acids (eg, phenylalanine and methionine) (1). These products generally contain taurine, tyrosine, histidine, arginine, and L-cysteine (semi-essential to neonates) (1).
- **Glutamine**: Glutamine is a conditionally essential amino acid during times of metabolic stress. There has been considerable interest in the potential beneficial effects of glutamine, including systemic antioxidant effects, maintenance of gut integrity, induction of heat shock proteins, and use as a fuel source for rapidly replicating cells (6). The addition of parenteral glutamine at a dose of 0.5 g/kg per day to a parenteral regimen reduces infectious complications, ICU length of stay, and mortality in critically ill ICU patients, as compared to the same parenteral nutrition regimen without glutamine (6). Currently, crystalline amino acid formulations in the United States and Canada do not contain glutamine (6). Due to stability and compatibility issues, there is no intravenous form of glutamine that is commercially available for admixture in parenteral solutions (6,11). Studies of glutamine-supplemented parenteral formulas have used preparations of powdered L-glutamine sterilized by filtration (6,24,25). ASPEN and AND guidelines currently recommend that supplementation with parenteral glutamine be considered when parenteral nutrition is used in the critical care setting to reduce infectious complications (Grade 1)* (18); or for those with demonstrated benefits eg, surgical patients with extensive abdominal surgery, burn patients, acute pancreatitis (6,24). Providing PN glutamine supplementation early and in doses of > 0.5/kg/day is currently suggested to be effective in critical ill patients (24).

Protein requirements: Protein requirements should be based on the patient's individual needs and disease process. Critical illness and hypermetabolism are associated with increased protein turnover, protein catabolism, and negative nitrogen balance (6,26). During a critical illness, protein requirements can double increasing to approximately 15% to 20% of total energy (26). Protein requirements for the critically ill patient are at least 1.5 g/kg per day, and adequate energy should be provided to meet the estimated needs as determined by indirect calorimetry or predictive equations (26). The protein requirements of critically ill patients who are not obese (BMI <30 kg/m²) are 1.2 to 2.0 g/kg actual body weight per day, although burn patients or multiple-trauma patients may require greater amounts of protein (6). Protein sparing does not typically improve with protein intakes greater than 1.5 g/kg per day, except in severely burned patients (26). Critically ill patients with obesity may have higher protein needs. The ASPEN guidelines for critical care ICU adult patients recommend a daily protein intake of more than 2.0 g/kg ideal body weight for class I and II obese patients (BMI, 30 to 40 kg/m²) and more than 2.5 g/kg ideal body weight for class III obese patients (BMI >40 kg/m²) (6). For all patients, a 24-hour urine collection for urinary urea nitrogen may be collected and used to subsequently adjust the prescription for protein delivery.

Lipid sources: Isotonic lipid emulsions provide energy and essential fatty acids. Lipid sources are derived from soybean oil or a 50:50 mixture of soybean and safflower oils (11). In North America, the choice of parenteral lipid emulsion is severely limited to a soy-based, 18-carbon omega-6 fatty acid preparation that has proinflammatory characteristics in the ICU population (6). During the first 7 days in the ICU setting, a soy-

Parenteral Nutrition Support for Adults

based, lipid-free parenteral nutrition regimen has been shown to be associated with a significant reduction in infectious morbidity (pneumonia and catheter-related sepsis), a decreased length of hospitalization and ICU stay, and a shorter duration of mechanical ventilation as compared to the use of lipid-containing parenteral nutrition (6). ASPEN guidelines for critically ill ICU patients recommend that during the first week of hospitalization when enteral nutrition is not feasible, a parenteral formulation without soy-based lipids should be considered (6). Lipid sources are emulsified with egg yolk phospholipid; therefore, their use may be contraindicated in patients with egg allergies. Lipid emulsions contain glycerol, so lipid emulsion does not provide 9 kcal per gram as it would if it were pure fat. Lipid emulsions are available in 10% (1.1 kcal/mL), 20% (2 kcal/mL), and 30% (3 kcal/mL) concentrations (11). The 30% lipid formulation is approved only for compounding of the total nutrient admixture, not for direct intravenous administration (11).

Investigational intravenous fat emulsion (IVFE) products include physical mixtures of medium-chain triglycerides and long-chain triglycerides. These formulations may be useful for patients who are intolerant to the long-chain triglyceride products during critical illness and metabolic stress and also for patients with carnitine deficiency (11). Structured lipid formulas containing linoleic acid, medium-chain fatty acids, and very long-chain omega-3 fatty acid are being investigated to determine if they produce less inflammatory and nonthrombogenic prostaglandins than standard lipid emulsions (11). These formulas have been studied in patients with sepsis, atopic dermatitis, or severe ulcerative colitis and patients undergoing elective surgery (27,28). These formulas are under investigational study and are not available for intravenous use in the United States (11).

Because of the enhanced microbial growth potential with the infusion of IVFE separately from dextrose and amino acid formulations, the Centers for Disease Control and Prevention recommends a 12-hour hang-time limit for IVFE (29). The United States Pharmacopeial has also endorsed the use of IVFE products within 12 hours of opening the original manufacturer's container if the IVFE products are infused as separate preparations from dextrose and amino acids (30). However, because of the lower pH (5.6 to 6.0) of a total nutrient admixture that contains IVFE, dextrose, and amino acids in the same container, the fat emulsion may be administered over 24 hours when infused in a total nutrient admixture (11). Whether infused separately or as a total nutrient admixture, the infusion rate of IVFEs should not exceed 0.11 g/kg per hour to reduce side effects, such as hypertriglyceridemia, and infectious complications (11,31).

Lipid requirements: When a patient is medically stable, lipids provide an important source of essential fatty acids. A total of 2% to 4% of daily energy needs should be supplied as linoleic acid (1% to 2% of linoleic acid and 0.25% to 0.5% of alpha-linolenic acid) (1) or 25 to 100 mg/kg of essential fatty acids (1,32,33). A minimum of 500 mL of 10% lipid stock solution or 250 mL of 20% stock solution administered over 8 to 10 hours, two to three times per week, is sufficient to prevent a deficiency of essential fatty acids. Alternately, 500 mL of a 20% fat emulsion can be given once a week (34). Excessive amounts of intravenous lipids or rapid infusion rates can cause hyperlipidemia (20). Levels of serum triglycerides should be evaluated before the infusion of intravenous lipids. Four hours after lipid infusion, acceptable serum triglycerides levels are less than 250 mg/dL for piggybacked lipids and less than 400 mg/dL for continuous lipid infusion (1). The infusion of intravenous lipids has been associated with impaired immune responses and vascular integrity (19,32,35). Infusion rates of greater than 110 mg/kg per hour may result in reduced lipid clearance and impaired reticuloendothelial function and pulmonary exchange (32). It is recommended that fat intake be restricted to less than 25% to 30% of total energy, or 1 g/kg per day, and provided slowly over 8 to 10 hours if administered as an intravenous supplement (32,34). No more than 2.5 g/kg per 24 hours should be provided to adult patients (1). The rapid infusion of fat emulsions, regardless of the total amount, should be avoided in patients who have severe pulmonary failure (6).

The recommendations for lipids given to critically ill patients requiring parenteral nutrition are more conservative; the data support the administration of less than 1.0 g/kg per day (1,34). ASPEN guidelines for critically ill ICU patients suggests during the first week of hospitalization (when enteral nutrition is not feasible), the use of a parenteral formulation without soy-based lipids should be considered in order to reduce the risks associated with the proinflammatory effects previously discussed (6). These guidelines acknowledge that parenteral nutrition without lipids might exacerbate stress-induced hyperglycemia; therefore, the recommendation should be cautiously applied with consideration to the individual patient's nutritional needs and acute medical situation (6). Carnitine deficiency can lead to fat deposition in the liver and muscle, impaired ketogenesis, and neurologic symptoms. Parenteral nutrition solutions do not contain carnitine, and the supplemental use of carnitine in adults has not been studied. However, carnitine supplementation has been suggested for neonates who receive parenteral nutrition for more than 2 weeks (36).

Parenteral Intravenous Vitamins and Requirements

In 2000, the Food and Drug Administration (FDA) modified the requirements for adult intravenous multivitamin products. The required amounts of ascorbic acid, thiamin, pyridoxine, and folic acid were increased, and a requirement for vitamin K was added (37) (see Table B-4). With the addition of vitamin K to these formulations, the prothrombin time and international normalized ratio of patients who receive anticoagulant therapy should be closely monitored, and anticoagulant medication should be adjusted as needed (20). One formula without vitamin K is available for patients whose prothrombin time or international normalized ratio levels are difficult to manage (38). Weekly intravenous supply of 250 to 500 mcg phylloquinone is adequate to preserve coagulation in PN patients (38). The adequacy of vitamin D in vitamin preparations for PN may be inadequate for patients requiring long term PN as the daily PN infusion provides a conservative amount of 200 to 400 IU (5 to 10 mcg) vitamin D. An effective method to ensure vitamin D status for patients on PN long term is daily sunlight exposure (38). The vitamin and mineral requirements for parenteral nutrition should be based on the Dietary Reference Intakes for the patient's age and sex (38). Clinicians should consider the deleterious impact of exceeding the FDA-recommended levels of parenteral vitamins, and the potential harm that excessive vitamin intake has on other micronutrients, trace elements, and immune status (38).

Vitamin	Amount
Thiamin (B ₁)	6 mg
Riboflavin (B ₂)	3.6 mg
Pyridoxine (B ₃)	6 mg
Cyanocobalamin (B ₁₂)	5 mcg
Niacin	40 mg
Folic acid	600 mcg
Pantothenic acid	15 mg
Biotin	60 mcg
Ascorbic acid	200 mg
Vitamin A (retinol)	1 mg
Vitamin D	5 mcg
Vitamin E	10 mg
Vitamin K	150 mcg

Table B-4: FDA Requirements for Parenteral Multivitamin Products (37)

Parenteral Intravenous Trace Minerals and Requirements

The ASPEN recommendations for daily parenteral intake of the trace elements zinc, copper, chromium, and manganese were updated in 2004 (Table B-5) and remains the most current (16). These updated recommendations include the addition of selenium supplementation (20 to 60 mcg/day) (16). Trace minerals are available as single-entity and combination products in various concentrations for adults, children, and neonates; products with a combination of trace minerals and electrolytes are also available (11,38). Other elements that may be supplemented on an individualized basis include molybdenum, iodine, and iron. Iron supplementation is generally not required for short-term parenteral nutrition, unless the patient is anemic. Oral iron supplementation is the preferred route. However, if oral supplementation is infeasible, iron dextran can be intravenously administered (38). The addition of iron to total nutrient admixtures is not recommended because of compatibility problems (39). Patients with intestinal fluid losses (eg, patients with ostomies) may require additional 12 mg of zinc per liter of output from the small bowel and an additional 17 mcg of zinc per liter of stool or ileostomy losses (16,40). Chromium requirements may also increase to 20 mcg/day with gastrointestinal losses in adults (16).

Table B-5: Daily Parenteral Trace Element Supplementation for Adults (Dose per Day)

	2004 ASPEN	Gastrointestinal
Trace Element	Recommendation	Losses
Zinc ^a	2.5-5.0 mg ^a	Add ^b
Copper	0.3-0.5 mg	500 mcg/d
Chromium	10-15 mcg	20 mcg ^c
Manganese	60-100 mcg	_
Selenium	20-60 mcg	_

^a Add 2 mg/day for hypermetabolic patients for a total of 6 to 12 mg/day.

^bAdd 12 mg/L of small bowel losses, and add 17 mcg/kg of stool or ileostomy losses.

Parenteral Nutrition Support for Adults

^cCheck hemoglobin A1C every 6 months.

Adapted from the following source with permission from ASPEN. ASPEN does not endorse the use of this material in any form other than its entirety:

Clark SF. Vitamins and trace elements. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:142 (Table 8-6).

Parenteral Electrolytes and Requirements

Therapeutic amounts of electrolytes are added to parenteral formulations depending on the patient's requirements (11). The serum phosphate levels of critically ill patients should be monitored closely, and phosphate should be provided when needed due to its essential role in optimal pulmonary function (6). Electrolytes are available in salt forms, including sodium and potassium as chloride acetate or phosphate; calcium as chloride, gluconate, or gluceptate; and magnesium as sulfate or chloride. Calcium gluconate and magnesium sulfate are the preferred forms of these cations because they produce fewer physiochemical incompatibilities (11). The standard daily electrolyte ranges for adults (Table B-6) should be adjusted as indicated by the clinical situation (11). Acetate and chloride do not have specific ranges for intake; rather, they are adjusted as needed to maintain the acid-base balance (10,11).

Table B-6:	Daily	Electrolyte	Requirements	(11)
------------	-------	-------------	--------------	------

Electrolyte	Requirement
Sodium and potassium	1-2 mEq/kg + replacement losses
Calcium	10-15 mEq
Magnesium	8-20 mEq
Phosphate	20-40 mmol

Total Nutrient Admixture Parenteral Solutions

Total nutrient admixture parenteral solutions, also known as three-in-one or all-in-one solutions, are composed of amino acids, dextrose, lipids, vitamins, trace elements, and electrolytes in one container. This method of nutrient delivery differs from the conventional method (two-in-one) of providing CPN, in which lipids are in a separate container and "piggybacked in" with the amino acid–dextrose solution. Both types of parenteral formulation systems are in use today (11). By definition, the total nutrient admixture includes the lipid emulsion on a daily basis, providing an additional energy source (11). Total nutrient admixtures have decreased the cost of CPN due to the decreased administrative and equipment costs associated with CPN preparation decreased nursing time (39). Total nutrient admixtures may also help prevent excessive dextrose administration in critically ill patients (39,41). Also, lipids are administered over a 24-hour period, which may promote better patient tolerance (41).

One disadvantage of total nutrient admixtures is that they provide a higher bacterial growth medium than the conventional system. Also, most particulate matter in the admixture cannot be visually inspected ($_{39,41,42}$). Conventional solutions use a 0.22-µm in-line filter; however, total nutrient admixtures require a larger in-line filter (1.2 µm) because they contain lipids. This larger filter is sufficient for trapping solution particulates, precipitates, and *Candida albicans*, but it does not protect against contaminates such as *Staphylococcus epidermidis, Escherichia coli*, and bacterial endotoxins (11,39,41,42). Refer to the previous discussion on lipid sources for hang-time and infusion guidelines.

Stability and Compatibility

The concentrations of calcium and phosphate ions are directly related to the risk of precipitation, which can result in serious injury and death (43). As the concentration of either micronutrient rises, the likelihood of precipitation increases (11). The verification of large calcium doses (more than two times the Dietary Reference Intake) can help minimize the risk of precipitation (11). The salt form of calcium added to the PN formulation can have a dramatic impact on the risk of precipitation (11). Calcium gluconate and calcium gluceptate are generally less dissociated salt forms of calcium than the chloride salts (11). As a result, the amount of free calcium available to form insoluble complexes with phosphate is reduced (11).

Metabolic or Clinical Parameter	Monitoring Frequency
Blood glucose	Every 6 hours until stable; 100 to 150 mg/dL in
	critically ill patients (6,16,20,44) and 140 to 180 mg/dL in critically ill diabetic patients (19)
Electrolytes (Na, K, Cl), CO ₂ , blood urea nitrogen, creatinine, Mg, Ca ^a , phosphorus	Baseline, daily until stable, then two or three times per week
Total bilirubin, liver function tests (alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase)	Baseline, daily until stable, then weekly
Complete blood cell count with differential	Baseline, then weekly
Prothrombin time/partial thromboplastin time	Baseline, then weekly
Albumin ^b	Baseline, then as needed (\geq 21 days)
Prealbumin ^b	Baseline, then weekly
Transferrin ^b	Baseline, then weekly
Nitrogen balance	As needed
Weight	Daily until stable, then two or three times per week
Vital signs (temperature, pulse, blood pressure, respiratory rate)	Every 8 hours as needed

Table B-7: Monitoring Hospitalized Patients Receiving CPN (18, 44)

^a Half of total calcium (Ca) is protein bound; therefore, during hypoalbuminemia, the true calcium status may not be represented by measuring serum calcium levels. There is a 0.8 mg/dL decrease in the total concentration of serum calcium for each 1.0 mg/dL decrease in the albumin concentration below 4.0 g/dL. Correct serum calcium can be estimated by the following formula: Ca⁺⁺ (mg/dL) = Measured Ca⁺⁺ (mg/dL) + 0.8 × [4.0 – Albumin (g/dL)] (44). However, this formula provides only an estimate and a review of the evidence suggests this formula may actually overestimate the corrected calcium concentration in critically ill patients (45). When an accurate evaluation is needed, the ionized calcium level should be obtained (44).

^b The levels of acute phase hepatic proteins (albumin, prealbumin, and transferrin) can decrease by as much as 25% as a result of acute or chronic inflammatory conditions and are no longer reliable indicators in critically ill patients. This decrease impacts their usability in determining nutrition repletion. If inflammatory markers (eg, C-reactive protein) indicate an inflammatory metabolism, these proteins may not be reliable indicators of nutritional status. Acute phase hepatic proteins may only be reliable when malnutrition is not complicated by inflammatory metabolism caused by acute or chronic disease.

See Section III: Clinical Nutrition Management, Parenteral Nutrition: Metabolic Complications of Central Parenteral Nutrition and Calculating Total Parenteral Nutrition.

Transitional Feeding (6)

Cyclic CPN: The infusion of CPN over a limited amount of time (usually 12 to 18 hours) is called cyclic CPN. Cyclic CPN is indicated for patients who are metabolically stable and for patients who require long-term CPN, such as patients who receive CPN at home. One advantage of cyclic CPN is that feedings more closely resemble physiologic (discontinuous) feedings, which may reduce the hepatic toxicity associated with continuous feedings. Another advantage is improved quality of life, because the patient is free from CPN equipment during the day.

Transition to cyclic is best done over 2 to 4 days by reducing the infusion time in increments of 4 to 6 hours. Cyclic TPN should be tapered up 1 to 2 hours (for patients with DM or hyperglycemia) to decrease hyperglycemia and hypoglycemia (46).

Parenteral to enteral: When the patient is transitioned from parenteral support to enteral support, the tube feeding can be initiated at full strength (10 to 30 mL/hour). As the rate of tube feeding is increased, the rate of parenteral nutrition is proportionately decreased. Tapering of parenteral formula can begin when enteral tube feedings are providing 33% to 50% of nutrient requirements. Once enteral tube feedings are well tolerated and provide more than 60% of energy requirements and 100% of fluid requirements, parenteral nutrition can be discontinued (6).

Parenteral to oral: When patients are transitioned from parenteral support to an oral diet, oral intake is started as clear liquids and then progressed in a stepwise fashion to an appropriate diet as tolerated. Nutrient intake studies should document the adequacy of oral intake. The total parenteral nutrition should be tapered to half of the original rate when the patient is eating 50% of the total estimated energy needs. The CPN should be discontinued when oral intake consistently meets 60% of estimated nutrient needs and 100% of fluid needs (6). If oral intake does not progress to adequate amounts, enteral nutrition should be considered in lieu of PPN or CPN (6).
Parenteral Nutrition Support for Adults

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. ASPEN Board of Directors and the Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *J Parenter Enteral Nutr.* 2002;26 (suppl)(1):1SA-138SA.
- 2. Trujillo EB, Young LS, Chertow GM, Randall S, Clemons T, Jacobs DO, Robinson MK. Metabolic and monetary costs of avoidable parenteral nutrition use. *J Parenter Enteral Nutr.* 1999;23:109-111.
- 3. Mirtallo JM, Patel M. Overview of parenteral nutrition. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:234-244.
- 4. Koretz RL, Lipman TO, Klein S. AGA technical review: parenteral nutrition. *Gastroenterology*. 2001;121:970-1001.
- 5. Twomey PL, Patching SC. Cost-effectiveness of nutritional support. J Parenter Enteral Nutr. 1985;9:3-10.
- McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; ASPEN Board of Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr. 2009;33:277-316.
- Klein S, Kinney, Jeejeebhoy K, Alpers D, Hellerstein M, Murray M, Twomey P. Nutrition support in clinical practice: review of published data and recommendations for future research directions: summary of a conference sponsored by the National Institutes of Health, American Society for Parenteral and Enteral Nutrition, and American Society for Clinical Nutrition. *J Parenter Enteral Nutr.* 1997;21:133-156.
- 8. Mirtallo JM, Forbes A, McClave SA, Jensen GL, Waitzberg DL, Davies AR; International Consensus Guideline Committee Pancreatitis Task Force. International consensus guidelines for nutrition therapy in pancreatitis. *J Parenter Enteral Nutr.* 2012; 36:284-291.
- 9. McClave SA, Chang WK, Dhaliwal R, Heyland DK. Nutrition support in acute pancreatitis: a systematic review of the literature. *J Parenter Enteral Nutr.* 2006;30:143-156.
- 10. National Advisory Group on Standards of Practice Guidelines for Parenteral Nutrition. Safe practices for parenteral nutrition formulations. *J Parenter Enteral Nutr.* 1998;22:49-66.
- 11. Barber JR and Sacks GS. Parenteral nutrition formulations. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 245-263.
- 12. Singer P, Bursztein S, Kirvela O, Mansour B, Yoshimura N, Blaustein J, Askanazi J. Hypercaloric glycerol in injured patients. *Surgery*. 1992;112:509-514.
- 13. Lev-Ran A, Johnson M, Hwang DK, Askanazi J, Weissman C, Gersovitz M. Double-blind study of glycerol vs. glucose in parenteral nutrition of post-surgical insulin-treated diabetic patients. *J Parenter Enteral Nutr.* 1987;11:271-274.
- 14. McEvoy GK, ed. AHFS Drug Information 1999. Bethesda, Md: American Society of Health-System Pharmacists; 1999.
- 15. Wolfe RR, O'Donnell TF Jr, Stone MD, Richmand DA, Burke JF. Investigation of factors determining the optimal glucose infusion rate in total parenteral nutrition. *Metabolism*. 1980;29:892-900.
- 16. Task Force for the Revision of Safe Practices for Parenteral Nutrition. Safe practices for parenteral nutrition. J Parenter Enteral Nutr. 2004;28 (6 suppl):S39-S70.
- 17. Van de Berg G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in critically ill patients. *N Eng J Med.* 2001;345:1359-1367.
- 18. Critical Illness Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- 19. American Diabetes Association. Standards of medical care in diabetes 2013. *Diabetes Care*. 2013;36(suppl 1):11S-66S.
- 20. Kumpf VJ, Gervasio J. Complications of parenteral nutrition. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum.* 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 285-297.
- 21. Jeejeebhoy KN, Chu RC, Marliss EB, Greenberg GR, Bruce-Robertson A. Chromium deficiency, glucose intolerance, and neuropathy reversed by chromium supplementation, in patient receiving long-term total parenteral nutrition. *Am J Clin Nutr.* 1977;30:531-538.
- 22. Solomon SM, Kirby DF. The refeeding syndrome: a review. J Parenter Enteral Nutr. 1990;14:90-97.
- 23. Melnick G. Value of specialty intravenous amino acid solutions. *Am J Health Syst Pharm*. 1996;53:671-674.
- Vanek VW, Matarese LE, Robinson M, Sacks GS, Young LS, Kochevar M; Novel Nutrient Task Force, Parenteral Glutamine Workgroup; A.S.P.E.N. Board of Directors. A.S.P.E.N. position paper: parenteral nutrition glutamine supplementation. *Nutr Clin Pract.* 2011;26:479-494
- 25. Ziegler TR, Benfell K, Smith RJ, Young LS, Brown E, Ferrari-Baliviera E, Lowe DK, Wilmore DW. Safety and metabolic effects of Lglutamine administration in humans. *J Parenter Enter Nutr*. 1990;14:137S-146S.
- Young LS, Kearns LR, Schoefel SL, Canon CN. Protein. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 94-95.
- 27. Mayser P, Mayer K, Mahloudjian M, Benzing S, Kramer HJ, Schill WB, Seeger W, Grimminger F. A double-blind, randomized, placebocontrolled trial of n-3 versus n-6 fatty acid-based lipid infusion in atopic dermatitis. *J Parenter Enteral Nutr.* 2002;26:151-158.
- 28. Weiss G, Meyer F, Matthies B, Pross M, Koenig W, Lippert H. Immunomodulation by perioperative administration of n-3 fatty acids. Br J Nutr. 2002;87:S89-S94.
- 29. Centers for Disease Control and Prevention. Guidelines for the prevention of intravascular catheter-related infections [published erratum appears in *MMWR Recomm Rep.* 2002;51:711]. *MMWR Recomm Rep.* 2002;51:(RR-10):1-29.
- 30. Chapter <797>. Pharmaceutical compounding-sterile preparations. Physical tests. United States Pharmacopeia 28/National Formulary 23. Rockville, Md: United States Pharmacopeial Convention; 2005:2461-2477.
- 31. Klein S, Miles JM. Metabolic effects of long-chain and medium-chain triglycerides in humans. *J Parenter Enteral Nutr.* 1994;18:396-397.
- 32. Seidner DL, Mascioli EA, Istfan NW, Porter KA, Selleck K, Blackburn GL, Bristrian BR. Effects of long-chain triglyceride emulsions on reticuloendothelial system function in humans. *J Parenter Enteral Nutr.* 1989;13:614-619.
- 33. Holman RT. The ratio of trienoic:tetraenoic acids in tissue lipids as a measure of essential fatty acid requirement. J Nutr. 1960;70:410-415.
- 34. Hisse M, Brown JC. Lipids. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 69-77-.
- 35. Kollef MN, McCormack MT, Caras WE, Reddy VV, Bacon D. The fat overload syndrome: successful treatment with plasma exchange. *Ann Intern Med.* 1990;112:545-546.
- 36. Tibboel D, Delemarre FM, Przyrembel H, Bos AP, Affourtit MJ, Molenaar JC. Carnitine deficiency in surgical neonates receiving total

Manual of Clinical Nutrition Management

parenteral nutrition. J Pediatr Surg. 1990;25:418-425.

- 37. Parenteral multivitamin products; drugs for human use; drug efficacy study implementation; amendment (21 CFR 5.70). *Federal Register.* April 20, 2000;65:21200-21201.
- 38. Clark S. Vitamins and trace elements. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:146-148.
- 39. Driscoll D. Total nutrient admixtures: theory and practice. Nutr Clin Pract. 1995;10:114-119.
- 40. Wolman SL, Anderson GH, Marliss EB, Jeejeebhoy KN. Zinc in oral total parenteral nutrition: requirements and metabolic effects. *Gastroenterology*. 1979;76:458-467.
- 41. D'Angio RG, Reichers KC, Gilsdorf RB, Costantino JM. Effect of the mode of lipid administration on parenteral nutrition-related infections. *Ann Pharmacother*. 1992;26:14-17.
- 42. Erdman SH, McElwee CL, Kramer JM, Zuppan CW, White JJ, Grill BB. Central line occlusion with three-in-one nutrition admixture administered at home. J Parenter Enteral Nutr. 1994;18:177-181.
- 43. ASPEN Board of Directors. *ASPEN's Adult Parenteral Nutrition (PN) Support Pathway*. Silver Spring, Md: American Society for Parenteral and Enteral Nutrition; 1998.
- 44. Langley G, Tajchman S. Fluids, electrolytes, and acid-base disorders. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum.* 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 111.
- 45. Dickerson RN, Alexander KH, Minard G, Croce MA, Brown RO. Accuracy of methods to estimate ionized and "corrected" serum calcium concentrations in critically ill mulitpe trauma patients receiving specialized nutrition support. J Parent and Enteral Nutr. 2004;28:133-141.
- 46. Charney, O, Malone, A, AD Pocket Guide To Parenteral Nutrition, 2007, p126-127.

Bibliography

- Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012.
- *Critical Illness Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- Position of the American Dietetic Association: ethical and legal issues in nutrition, hydration, and feeding. *J Am Diet Assoc.* 2008;108:873-882.
- McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; ASPEN Board of Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr. 2009;33:277-316.
- Joint Standards Task Force of ASPEN and the American Dietetic Association Nutrition Support Practice Group. Russell M, Stieber M, Brantley S, Freeman AM, Lefton J, Malone AM, Roberts S, Skates J, Young LS. American Society for Parenteral and Enteral Nutrition and American Dietetic Association: standards of practice and standards of professional performance for registered dietitians (generalist, specialty, and advanced) in nutrition support. *J Am Diet Assoc.* 2007;107:1815-1822.

I. NORMAL NUTRITION AND MODIFIED DIETS C. Modification of Carbohydrate and Fat

Medical Nutrition Therapy for Diabetes Mellitus	C-1
Table C-1: Summary of Glycemic Recommendations for Many Non-Pregnant Adu	ılts With
Diabetes	C-1
Table C-2: Strategies to Treat Disorders of Lipid Metabolism in Patients With Dia	abetes C-7
Table C-3: Carbohydrate Content of Substitutions	C-9
Medical Nutrition Therapy for Gestational Diabetes	C-15
Table C-4: Recommended Weight Gain for Pregnant Women	C-16
Table C-5: Recommended Daily Energy Intake for Pregnant Diabetic Women	C-17
Table C-6: Blood Glucose Goals in Gestational Diabetes Mellitus	C-18
Dietary Management with the Exchange System	C-21
Sugar in Moderation Diet	C-35
Calorie-Controlled Diet for Weight Management	C-36
Table C-7: Weight Classification by BMI, Waist Circumference, and Associated H	lealth Risks
	C-36
Medical Nutrition Therapy for Disorders of Lipid Metabolism	C-40
Table C-8: LDL Cholesterol Goals	C-40
Table C-9: Clinical Identification of the Metabolic Syndrome	C-41
Table C-10: Nutrient Composition of Therapeutic Lifestyle Changes Diet	C-42
Table C-11: Features Of ATP III Guidelines	C-48
Table C-12: LDL Cholesterol (LDL-C) Goals And Recommendations for Therapeu	itic Lifestyle
Changes and Drug Therapy in Different Risk Categories	C-48
Table C-13: Recommended Approaches for Elevated Triglyceride Levels	C-50
Food Guide - Low Saturated Fat, Low Cholesterol Or TLC Diets	C-53
Fat-Controlled Diet	C-55
Food Guide – Fat-Controlled Diet	C-55
Medium-Chain Triglycerides (MCT)	C-57

MEDICAL NUTRITION THERAPY FOR DIABETES MELLITUS

Description

The goals of medical nutrition therapy for diabetes mellitus are to improve overall metabolic outcomes (glucose and lipid levels), provide appropriate energy to maintain desirable body weight, and improve overall health through optimal nutrition (1). The consistent-carbohydrate meal planning approach incorporates consistent carbohydrate intake, fat intake modifications, and consistent timing of meals and snacks (if needed). The American Diabetes Association recommends the consistent-carbohydrate meal planning approach over the standardized energy-level meal patterns based on the exchange lists (1-3).

Indications and Nutrition Diagnosis

Diabetes is diagnosed and classified based on the results of appropriate medical and laboratory tests. After extensive review of the literature and improved standardization of the assay in 2009, the Expert Committee on Diagnosis and Classification of Diabetes Mellitus has approved the use of the A1C test for diagnosing diabetes mellitus (1). An A1C test of > 6.5% is the threshold used to diagnose diabetes mellitus (1). See Section II: Diagnosis Criteria for Diabetes Mellitus. The classification of diabetes mellitus includes four clinical classes (1):

- type 1 diabetes (caused by beta-cell destruction that usually leads to absolute insulin deficiency)
- type 2 diabetes (caused by progressive insulin secretory defect on the background of insulin resistance)
- other specific types (due to other causes, eg, genetic defects in beta-cell function, genetic defects in insulin action, diseases of the exocrine pancreas, or drug or chemical induced)
- gestational diabetes mellitus (diabetes mellitus diagnosed during pregnancy)

The type of diabetes and the individual patient's needs, as presented by the nutrition signs and symptoms, will determine the nutrition diagnosis, medical nutrition therapy, and approach to self-management training.

The overall goal of nutrition intervention is to assist and facilitate individual lifestyle and behavior changes that will lead to improved metabolic control (2). The following goals of medical nutrition therapy apply to all persons with diabetes (2):

- Attain and maintain optimal metabolic outcomes including (2):
 - a) a blood glucose level in the normal range, or as close to the normal range as safely possible, to reduce the risk of diabetic complications,
 - b) a lipid and lipoprotein profile that reduces the risk of macrovascular disease, and
 - c) blood pressure levels that reduce the risk of vascular disease.
- Prevent, treat, or delay the development of obesity, dyslipidemia, cardiovascular disease, hypertension, nephropathy, retinopathy, and neurologic complications associated with diabetes mellitus.
- Improve health through healthy food choices and physical activity.
- Address individual nutrition needs, taking into consideration the patient's personal and cultural preferences and willingness to change.
- Maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence.
- Contribute to normal outcomes of pregnancies for women with preexisting diabetes and gestational diabetes.
- Provide adequate energy and nutrients for increased needs during pregnancy and lactation.
- Provide adequate energy to maintain normal growth and development rates in children and adolescents with diabetes.

Table C-1: Summary of Glycemic Recommendations for Many Non-pregnant Adults With Diabetes^a Biochemical Index Goal^a

Diochemical muex	Gual-
A1C	<7.0%
Preprandial capillary plasma glucose, mg/dL	70-130 (3.9-7.2 mmol/L)
Peak postprandial capillary plasma glucose ^b , mg/d(mmol/l)	<180 (10.0)
Hemoglobin A _{1c}	

^aThese goals should be individualized based on duration of diabetes, age, life expectancy, cormorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness and individual patient considerations. More or less stringent glycemic goals

may be appropriate for individual patients. Postprandial glucose may be targeted if A1C goals are not met despite reaching preprandial glucose goals.

^bPostprandial glucose measurements should be made 1-2 hours after the beginning of the meal, generally peak levels in patients with diabetes.

Source: American Diabetes Association. Standards of medical care in diabetes-2013. Diabetes Care. 2013; 36(suppl 1):S21.

Nutritional Adequacy

The nutrition prescription can be planned to meet the Dietary Reference Intakes as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

The physician may specify one of the following:

• **Consistent-carbohydrate meal plan:** The meal plan incorporates consistent carbohydrate intake (±15 g of carbohydrate per meal or snack), fat intake modifications, and consistent timing of meals and snacks, not specific energy levels. The plan provides four carbohydrate selections (60 grams) at breakfast and five carbohydrate selections (75 grams) at lunch and dinner with the remaining selections from vegetables, lean meats, and appropriate fats. This diet is the standard hospital meal plan for people with diabetes and does not include in-between or evening snacks. The meal plan provides 1,500 to 1,800 kcal/day, with approximately 50% of the energy from carbohydrate, 20% from protein, and 30% from fat (<7% saturated and trans fat combined) (2-4).

If the patient requests additional food, select choices from the vegetable and lean meat groups. Betweenmeal snacks are given based on the individual patient's nutrition needs and preference or to complement medical treatment (eg, medications) to optimize blood glucose levels; thus, snacks are not automatically provided to patients. Food intake frequency (for example, three meals vs smaller meals and snacks) is not associated with long-term differences in glucose levels, lipid levels, or insulin responses in type 2 diabetes mellitus (2). Therefore, division of food intake should be based on the individual's preferences. Conditions for which an evening snack may be warranted include pregnancy, lactation, diabetes in a person with higher energy and protein needs, or prescription of medications that increase the risk of hypoglycemia.

- Nutrition prescription per a registered dietitian's recommendations: The dietitian plans an individualized diet, taking into account the patient's energy and protein needs, fat restrictions, food preferences, and eating habits. Snacks are planned for patients taking insulin or are served according to facility protocols. Meal plans based on exchange lists or carbohydrate counting may be used with this order. Individualization of the meal pattern is emphasized, rather than a specific standard macronutrient distribution. Fat content of the diet is manipulated according to the blood glucose, lipid, and body weight goals. Protein, carbohydrate, and mineral content of the diet may be manipulated to achieve individual metabolic and clinical goals.
- **Regular diet:** This may be considered as an option for patients with increased needs for energy and protein because of other medical conditions, such as pressure ulcers, cancer, burns, sepsis, or surgery.

Note: "No concentrated sweets" is not recommended, since it conveys the impression that simply avoiding sweets will in itself promote good control of blood glucose (2,3).

See Section III: Clinical Nutrition Management

- Diabetes Mellitus: Considerations For Acute Illness
- Diabetes Mellitus: Gastrointestinal Complications
- Diabetes Mellitus: Oral Glucose-Lowering Medications And Insulin
- Diabetes Mellitus: Considerations For Exercise
- Diabetes Mellitus: Fat Replacers And Nutritive/Nonnutritive Sweeteners

Nutrition Intervention and Planning for Medical Nutrition Therapy Carbohydrates and Diabetes

The following terms are preferred when describing carbohydrates: sugars, starch (eg, amylase, amylopectin, modified starches), and fiber (eg, cellulose, hemicellulose, pectins, hydrocolloids) (1). Regulation of blood glucose levels to achieve near-normal levels is the primary goal in the management of diabetes (1). Dietary techniques that limit hyperglycemia following a meal are important in limiting the complications of diabetes

(1). Both the amount (grams) and type of carbohydrate in a food influence blood glucose levels (Grade I)* (1,4,5). The total amount of carbohydrate consumed at meals and snacks influences the postmeal glucose response to a greater extent than other macronutrients (Grade I) (4,5). There is a direct relationship between the amount of carbohydrate in a meal, postmeal blood glucose response, and premeal rapid-acting or short-acting insulin requirements to maintain desirable blood glucose goals (Grade I) (4,5). Therefore, the total amount of carbohydrate consumed is a strong predictor of glycemic response, and monitoring the total grams of carbohydrate (whether by use of exchanges or carbohydrate counting) remains a key strategy in achieving glycemic control (Grade I) (1,4,5).

Studies have demonstrated that when persons with type 1 or type 2 diabetes mellitus consume a variety of sugars or starches, there is no substantial difference in the glycemic response when the total amount of carbohydrate remains constant (2,5). Sucrose intakes of 10% to 35% of total energy intake do not have a negative effect on glycemic or lipid responses in persons with either type 1 to type 2 diabetes when sucrose is substituted for isoenergetic amounts of starch (Grade I) (4). Based on these findings, sugar (eg, sucrose) intake does not have to be avoided. Instead, sugar intake should be based on the total amount of carbohydrate needed to achieve optimal metabolic control and the nutritional contribution to the diet. Foods containing carbohydrate from whole grains, fruits, vegetables, and low-fat milk are important components and should be included in a healthy diet for persons with diabetes mellitus (Grade I) (2,4).

Recently, the use of low-glycemic index foods or low-glycemic diets has received renewed interest. Factors that influence the glycemic response to food include: the type and amount of carbohydrate, type of sugar, nature of starch, cooking and food processing, as well as other food components (eg, fat and natural substances that slow digestion—lectins, phytates, tannins, and starch-protein and starch-lipid combinations) (2,6-9). Fasting and preprandial glucose concentrations, the severity of glucose intolerance, and the second meal or lente effect are other factors that affect the glycemic response to food (2,10-13). Because of the variety of factors that can influence a food's glycemic response and the limited number of long-term studies, there is still insufficient scientific evidence to support the use or nonuse of low-glycemic diets in improving metabolic outcomes (Grade II) (1.2.4.14). A meta-analysis of low-glycemic index diet trials in diabetic subjects showed that such diets produced a 0.4% decrement in hemoglobin A_{1C} (A1C) when compared with high-glycemic index diets (15). Some studies have shown short-term improvements in glycemic control by incorporating highfiber, low-glycemic index foods in meals or snacks (Grade 1) (4). The glycemic index and/or glycemic load used in conjunction with a consideration of total carbohydrate intake may provide greater benefits than consideration of only the total carbohydrate intake (1,2). Therefore, the consideration of the glycemic index may be helpful as an adjunct for select individuals. Individuals can determine the glycemic index's usefulness in maintaining their glycemic goals only by measuring their premeal and postmeal blood glucose levels (1,14,15).

The amount of total carbohydrate intake should be individualized based on the individual's energy goals to achieve or maintain a desirable body weight, eating habits, and glucose and lipid goals (1-3). In type 2 diabetes mellitus, an individual's metabolic profile and the need for weight loss should be considered when determining the carbohydrate and monounsaturated fat content of the diet (1,2). For weight loss, either a low-carbohydrate or low-fat, energy-restricted diet may provide short-term effectiveness (for up to 1 year) (1,2). For patients on low-carbohydrate diets, it is important to monitor lipid profiles, renal function, and protein intake (in patients with nephropathy) and adjust hypoglycemia therapy as needed (2).

For additional information, refer to:

- Energy Balance, Overweight, and Obesity in Diabetes in this section
- Section III: Clinical Nutrition Management, Diabetes Mellitus: Fat Replacers and Nutritive/Nonnutritive Sweeteners

Fiber

According to The American Diabetes Association and evidence-based nutrition practice guidelines fiber consumption recommendations for people with diabetes are the same as for the general population (2,4). The Dietary Reference Intake (DRI) recommends consumption of 14 g dietary fiber per 1,000 kcal, or 25 g for adult women and 38 g for adult men (2,16). However, emerging evidence suggests persons with diabetes may benefit from higher dietary fiber intake. Based on a current review of evidence, diets providing 30 to 50 g fiber per day from whole food sources consistently produced lower serum glucose levels compared to low-fiber diets (Grade III) (16). The addition of viscous dietary fibers slow gastric emptying rates, digestion, and absorption of glucose to benefit immediate postprandial glucose metabolism and long-term glucose control in

individuals with type 2 diabetes mellitus (16,17). Fiber supplements providing doses of 10 to 29 g/day may also have some benefit in terms of glycemic control (Grade III) (16). There is conclusive evidence that dietary fiber intake from whole foods or supplements may lower blood pressure, improve serum lipid levels, and reduce indicators of inflammation (Grade II) (16). Benefits may occur with intake of 12 to 33 g fiber per day from whole foods or up to 42.5 g fiber day from supplements (Grade II) (16). For patients with diabetes complicated by disorders of lipid metabolism, particularly hypercholesterolemia, lower cholesterol levels are an important benefit of consuming a high-fiber diet (16). Potential gastrointestinal side effects of excessive dietary fiber should be considered when integrating fiber into meal planning. A gradual increase in fiber from whole foods and supplements is suggested within recommended ranges to prevent negative side effects (16). Fiber is not digested and absorbed like sugars or starches. For purposes of carbohydrate counting, when there are more than 5 g of fiber per serving, half the number of grams of fiber should be subtracted from the total grams of carbohydrate to determine the amount of available carbohydrate (5).

Resistant Starch

Resistant starch (nondigestible oligosaccharides and the starch amylase) is not digested and therefore not absorbed as glucose in the small intestine. Legumes are the major food source of resistant starch in the diet; 100 g of cooked legumes contain 2 to 3 g of resistant starch, and 100 g (dry weight) of cornstarch contains about 6 g of resistant starch (18). It has been suggested that resistant starch produces a smaller increase in the postprandial glucose level than digestible starch and corresponds to lower insulin levels (2). Studies of persons with diabetes have focused on uncooked cornstarch and its potential to prevent nocturnal hypoglycemia. Some studies have demonstrated less hypoglycemia when cornstarch snacks are used; however, the evidence is limited. There is currently no established benefit of resistant starch for people with diabetes (2).

Timing of Carbohydrate and Food Intake: Type 1 Diabetes

For individuals requiring insulin, the total carbohydrate content of meals and snacks is the first priority and determines the premeal insulin dosage and postprandial glucose response (1-4,19). Individuals receiving intensive insulin therapy can adjust the premeal insulin dose based on the amount of carbohydrate at meals to maintain their blood glucose goals (Grade 1) (4). Individual needs should dictate the time when meals and snacks are eaten, how much time elapses between insulin injection and food intake, and the number of meals and snacks (1). Self-monitoring of blood glucose levels is necessary to achieve optimal blood glucose control and to prevent or delay the onset of diabetic complications (4). Checking blood glucose levels three to eight times per day has been associated with better glycemic control regardless of diabetes type or therapy (Grade I) (4). The American Diabetes Association recommends that people with type 1 diabetes or pregnant women who take insulin check their blood glucose levels three or more times daily, so that they can adjust food intake, physical activity level, and/or insulin dosage to meet blood glucose goals (1). Day-to-day consistency of food consumption is crucial for individuals who inject a fixed daily dosage of insulin (1,2).

For individuals who are on fixed insulin regimens and do not adjust premeal insulin dosages, consistent carbohydrate intake is the first priority (1,2,20). Individuals receiving insulin therapy should eat at consistent times that are synchronized with the action time of their insulin preparation and with blood glucose results, and insulin doses should be adjusted for the amount of food usually eaten or required (1-3). The decision to adjust insulin doses should be based on a review of blood glucose records and discussion with the patient's physician and coordinating health care team.

Intensified insulin therapy (multiple daily injections or insulin pump therapy): The goal of intensified insulin therapy is to bring the blood glucose levels as close to the normal range as is feasible for the individual. Insulin infusion pumps mimic the normal physiologic insulin delivery and allow flexibility in meal size and timing. Individuals that use rapid-acting insulin by injection or an insulin pump should adjust their meal and snack insulin doses based on the carbohydrate content of the meals and snacks (2). Carbohydrate counting, at an advanced level, can greatly increase flexibility in meal planning (21). The Diabetes Control and Complications Trial found that individuals who adjusted their premeal insulin dosages based on the carbohydrate content of Levels than individuals who did not adjust preprandial insulin dosages (22). Potential problems associated with intensified insulin therapy include hypoglycemia and weight gain (2,23-25). Given the potential for weight gain to adversely affect glycemia, dyslipidemia, blood pressure, and general health, the prevention of weight gain is desirable (2,26). Reductions in blood glucose levels and A1C may cause hypoglycemia, which occurs more frequently in individuals with type 1 diabetes (2). Patients on multiple-dose insulin (MDI) or insulin pump therapy should do self-monitoring blood glucose (SMBG) at least prior to meals and snacks, occasionally postprandially, at

bedtime, prior to exercise (1). In addition SMBG should be done when a patient suspects low blood glucose, after treating low blood glucose until they are normoglycemic, and prior to critical tasks such as driving (1). Adjustments should be made in food intake or medication based on SMBG results. Hypoglycemia should be treated appropriately (2). See discussion on Treatment of Hypoglycemia in this section.

Adjustments for exercise: Because the amount of physical activity may vary considerably from day to day, individuals with type 1 diabetes may need to make adjustments in energy intake and insulin dosage to avoid hypoglycemia. For individuals on these therapies, added carbohydrate should be ingested if pre-exercise glucose levels are < 100 mg/dL (1). When exercise is planned, the insulin dose may need to be adjusted to prevent hypoglycemia (2,27). If exercise is unplanned, additional carbohydrate may need to be consumed (2,27). Carbohydrate supplementation is based on the blood glucose level before exercise, previous experience with the particular form of exercise, and the individual's insulin regimen (1,2,27). Moderate-intensity exercise increases glucose uptake by 2 to 3 mg/kg per minute above the usual requirements. More carbohydrate may be needed for higher intensity activities (1,2,27). See Section III: Diabetes Mellitus: Considerations for Exercise.

Timing of Carbohydrate and Food Intake: Type 2 Diabetes

Food intake frequency—three meals or smaller meals and snacks—is not associated with long-term differences in glucose levels, lipid levels, or insulin responses (28,29). Therefore, division of food intake should be based on individual preferences, the lipid profile, and the type of diabetes medications used (Grade I) (2,4). Preprandial and postprandial blood glucose monitoring data levels can be used to determine if adjustments in food or meal planning will be helpful or if medications need to be combined with nutrition therapy (1,2). If individuals with type 2 diabetes require insulin, the consistency and timing of meals and their carbohydrate content become important, as with type 1 diabetes (1,2). Flexible insulin dosing regimens allow for variations in food intake and a more flexible lifestyle. Treatment with sulfonylureas and other insulin secretagogues also requires consistency in meal timing and the carbohydrate content of meals (1). People with type 2 diabetes are more resistant to hypoglycemia than people with type 1 diabetes; however, when a person with type 2 diabetes who is treated with insulin or insulin secretagogues is unable to eat, dosages may need to be modified (1,2). See discussion on Treatment of Hypoglycemia in this section.

Adjustments for exercise: Supplemental food before and during exercise is not needed to prevent hypoglycemia and is not recommended except under conditions of strenuous, prolonged exercise, such as endurance sports. Individuals taking sulfonylurea agents have a slightly increased risk of hypoglycemia during exercise, and supplemental energy intake may be required in some cases (1.27). The need for supplemental energy intake may be determined by glucose self-monitoring. Individuals with type 2 diabetes who use insulin should also monitor their blood glucose levels closely during and after exercise. Several strategies may be used to avert hypoglycemia during and after vigorous, prolonged, or nonhabitual exercise. These strategies involve the consumption of supplemental carbohydrate-containing foods before, during, and after exercise as well as adjustments in insulin dosage and timing (27).

For further information, refer to Section III: Clinical Nutrition Management

- Diabetes Mellitus: Considerations for Exercise
- Diabetes Mellitus: Oral Glucose-Lowering Medications And Insulin

Protein

The recommended protein intake for individuals with diabetes who have normal renal function is the same as for the general population (1). This recommendation translates into approximately 15% to 20% of daily energy intake from protein, which can be derived from both animal and vegetable sources (1,2,17). Individuals with type 2 diabetes and suboptimal glycemic control may have greater protein requirements due to increased protein turnover. However, the increased requirements do not exceed 20% of total energy intake (2). Intakes of protein that exceed 20% of daily energy may be a risk factor for the development of diabetic nephropathy (1). Based on studies of patients with nephropathy, it seems prudent to limit protein intake to the Recommended Dietary Allowances of 0.8 g/kg of body weight, which corresponds to approximately 10% of total energy (1,4,30). During a catabolic state induced by injury, inflammation, or severe illness, protein needs are 1.0 to 1.5 g/kg of body weight, with the higher end of the range for more stressed patients. Refer to Section II: Estimation of Protein Requirements.

In individuals with type 1 or type 2 diabetes, microalbuminuria predicts the later development of overt nephropathy (2). Microalbuminuria greater than 30 mg/day or 20 μ g/min is an indicator for nephropathy and increased cardiovascular morbidity and mortality (Grade II) (1,4). In patients with diabetic nephropathy,

reduction of dietary protein to 0.8 g/kg of body weight per day (the Recommended Dietary Allowances) may slow the progression of nephropathy (Grade II) (2,4). Along with testing for microalbuminuria, the analysis of a spot urine sample to determine the albumin-to-creatinine ratio is strongly encouraged (1).

Small short-term studies suggest that diets with a protein content >20% of total energy reduce glucose and insulin concentrations, reduce appetite, and increase satiety in patients with diabetes (31,32). The amount of protein consumed at meals has minimal influence on the glycemic response, lipid levels, and hormones and metabolites, and it has no long-term effect on insulin (Grade II) (4). As the percentage of protein increases and the percentage of energy from carbohydrate decreases it is difficult to determine whether the higher protein intakes or the lower carbohydrate intakes are responsible for significant effects on metabolic outcomes in studies (Grade II) (4). The effects of high-protein diets on the long-term regulation of energy intake, satiety, and weight as well as the ability of individuals to follow such diets long term have not been adequately studied; therefore, high-protein diets are not recommended as a strategy to improve glycemic outcomes or promote weight loss (Grade I) (1,2,4).

See the discussion on Energy Balance, Overweight, and Obesity in Diabetes in this section.

Fat Intake and Disorders of Lipid Metabolism

The distribution of energy from fat should be individualized based on the patient's nutrition assessment, cardiac risk assessment, disorders of lipid metabolism, and treatment goals (1-4,17,33,34).

Type 2 diabetes is associated with a twofold to fourfold excess risk of coronary heart disease (CHD) (Grade I) (4,33). The most common disorders of lipid metabolism in patients with type 2 diabetes are elevated triglycerides levels and decreased high-density lipoprotein cholesterol levels (33). The concentration of low-density lipoprotein (LDL) cholesterol in patients with type 2 diabetes is similar to that in nondiabetic individuals (33). The National Cholesterol Education Program (NCEP) Adult Treatment Panel III categorizes persons with diabetes mellitus in the high-risk category with therapeutic goals to reduce LDL cholesterol levels to less than 100 mg/dL through therapeutic lifestyle changes (diet and physical activity) and cholesterol-lowering drug therapy (34). A subcategory of high risk, very high risk, consists of persons with existing cardiovascular disease and diabetes as well as persons with cardiovascular disease and severe or poorly controlled multiple risk factors (34). The very high-risk category has a therapeutic option to reduce LDL cholesterol-lowering drug therapy at lower thresholds, are based on evidence from five randomized controlled trials that demonstrated a significantly reduced risk for cardiac events at these lower thresholds (34). Pharmacologic therapy is integral in achieving these lower LDL cholesterol levels in all high-risk patients (34).

The recommended percentage of energy from fat depends on the patient's lipid levels and treatment goals for glucose, disorders of lipid metabolism, and weight. Because persons with diabetes mellitus are at high risk of CHD and cardiovascular mortality (33,34), they should target the lowest LDL cholesterol goal (<100 mg/dL, or <70 mg/dL if categorized as very high risk) (2,33-35). Based on risk factor assessment, a person with diabetes mellitus should follow the recommendations of the NCEP Adult Treatment Panel III, the American Heart Association Dietary Guidelines 2000, and the American Diabetes Association Standards of Medical Care 2008 (1,35,36). The NCEP recommends that individuals with increased risk and/or disorders of lipid metabolism limit their fat intake to less than 35% of total energy, with saturated and trans fat combined targeting less than 7% of total energy (4), polyunsaturated fat restricted to less than 10% of total energy, and monounsaturated fat targeting 10% to 15% of total energy (Grade I) (4,33).

Several studies have investigated the optimal mix of macronutrients to best support metabolic outcomes in persons with diabetes and cardiovascular disease. Diets that are high in monounsaturated fat have not been shown to improve fasting plasma glucose levels or A1C values (2). Low–saturated fat (<10% of energy), high-carbohydrate diets increase postprandial levels of plasma glucose and insulin and increase plasma triglycerides levels (37); in some studies, these diets decrease plasma high-density lipoprotein cholesterol levels when compared with isoenergetic high–monounsaturated fat diets (2,37,38). When saturated-fat energy is replaced with either energy from carbohydrate or monounsaturated fat, there is a reduction in plasma LDL cholesterol levels (2). In other studies, when energy intake was reduced, the adverse effects of high-carbohydrate diets were not observed (2). Individual variability in response to higher carbohydrate diets (~55% of total energy) suggests that the plasma triglyceride response to dietary modifications should be monitored carefully, particularly in the absence of weight loss (2). An individual's metabolic profile and the need for weight loss should determine the medical nutrition therapy recommendations and nutrition

prescription. Consumption of omega-3 fatty acids from fish or from supplements reduces adverse cardiovascular disease outcomes (2,39). In addition, fish consumption displaces foods that are high in saturated fat from the diet (2). Two or more servings of fish per week (with the exception of fried fish fillets) are recommended for persons with diabetes (2,40,41).

Meta-analyses of prospective studies indicate that elevated triglycerides levels are also an independent risk factor for CHD. The NCEP Adult Treatment Panel III established a classification system and guidelines for intervention and treatment of hypertriglyceridemia (35). Refer to Section IC: Medical Nutrition Therapy for Disorders of Lipid Metabolism for the latest recommendations. Table C-2 outlines strategies to treat disorders of lipid metabolism in patients with diabetes:

ıl: decrease serum LDL cholesterol
way out in Lifestryle Changes Dist.
$2F_{0}$ to $2F_{0}$ on any from fat
25% to 55% energy from raturated and trans fath
<200 mg cholesterol per day
ight reduction and physical activity
ils: decrease LDL cholesterol, decrease
Ingrycer Ides prapautic Lifostylo Changes Diot
25% to 35% energy from fat
<7% energy from saturated and trans fat ^b
>10% energy from monounsaturated fat
Approximately 50% energy from carbohydrate
ight reduction and physical activity
ng therapy
als: decrease triglycerides to prevent acute ancreatitis and chylomicronemia syndrome, ecrease LDL cholesterol t approach: <15% energy from all fat
criglycerides are >1,000 mg/dL, omega-3 fatty cids may be used ^c (2,39). ight reduction and physical activity ig therapy

^a Pharmacologic therapy should be initiated as an adjunct to behavioral interventions to achieve a 30% to 40% decrease in LDL cholesterol from baseline values for moderately high risk, and very high risk patients (34).

^b Trans fatty acids increase LDL cholesterol (4). The American Heart Association (2006) recommends <1% of energy from trans fatty acids.

^c See discussion of omega-3 fatty acids in Section C: Medical Nutrition Therapy for Disorders of Lipid Metabolism.

Hypertension and Sodium

People with diabetes should maintain blood pressure levels less than 130/80 mm Hg (1). People with diabetes and hypertension should be medically treated to a systolic blood pressure goal of < 140 mm Hg and a diastolic blood pressure goal < 80 mm Hg (1). Recommendations regarding dietary sodium are the same for people with diabetes and the general population. Both normotensive and hypertensive individuals should limit sodium consumption to 2,400 mg/day. For people with hypertension, less than 2,400 mg/day of sodium is recommended, as well as lifestyle therapy including applying the principles of the Dietary Approaches to Stop Hypertension Diet, weight reduction if overweight, increasing potassium intake; moderation of alcohol consumption; and increased physical activity to lower blood pressure as an adjunct to pharmacotherapy (2,42). For people with hypertension and nephropathy, less than 2,000 mg/day of sodium is recommended (1,2). (See Section IF: Sodium-Controlled Diets.)

The precautions regarding alcohol consumption that apply to the general population also apply to people with diabetes. The *US Dietary Guidelines for Americans* recommends no more than two drinks per day for men and no more than one drink per day for women (1,2). Abstention from alcohol is advised for people with a history of alcohol abuse or dependence, pregnant women, and people with medical problems such as liver disease, pancreatitis, advanced neuropathy, or severe hypertriglyceridemia (2).

The effect of alcohol on blood glucose levels depends not only on the amount of alcohol ingested but also on the relationship to food intake. Alcohol used in moderation and ingested with food does not affect blood glucose levels when diabetes is well controlled (2). Alcoholic beverages should be considered an addition to the regular food/meal plan for patients with diabetes. Food should not be omitted because of the possibility of alcohol-induced hypoglycemia. When energy from alcohol needs to be calculated as a part of the total energy intake, alcohol should be substituted for fat exchanges or fat energy.

Micronutrients and Diabetes

There is no clear evidence that vitamin or mineral supplementation benefits people with diabetes who do not have underlying deficiencies (2). Chromium supplements have been reported to have beneficial effects on glycemia (1,17). However, due to study limitations and other studies that have not found these benefits in people with diabetes, the benefit of chromium supplementation has not been conclusively demonstrated (1,2,17,43). Increased consumption of folate by women of childbearing age to prevent birth defects as well as calcium consumption for the prevention of bone disease are recommended for people with or without diabetes.

Treatment of Hypoglycemia

Hypoglycemia is primarily an issue for diabetics who take insulin and insulin secretagogues. Changes in food intake, physical activity level, and medications can contribute to hypoglycemia. According to the American Diabetes Association's evidence-based guidelines, a glucose level of less than 70 mg/dL should be treated immediately (eg, carbohydrate ingestion, exercise delay, change in insulin dose) (1,2,44). The primary treatments for hypoglycemia are carbohydrate ingestion and medication adjustment. Glucose ingestion is the preferred treatment for hypoglycemia, although any form of carbohydrate that contains glucose may be used (1,2). The glycemic response has a greater correlation with total glucose content than with total carbohydrate content of food (41). For example, treatment of insulin-induced hypoglycemia with 20 g of glucose results in a greater rise in the plasma glucose level than treatment with 20 g of carbohydrate from orange juice or milk (45). The form of the carbohydrate—liquid or solid—does not impact the outcome of the glycemic response. The addition of protein to the carbohydrate does not assist in the treatment of hypoglycemia or prevent subsequent hypoglycemia episodes (1,2). The amount of protein consumed at meals has a minimal influence on the glucose response; however, the insulin response to protein is similar to carbohydrate (Grade II) (4). The addition of fat may retard the acute glycemic response (2,45). Ingestion of 15 to 20 g of glucose or total carbohydrate is an effective treatment of hypoglycemia; but, the blood glucose level may be only temporarily corrected (1,2). Ten grams of oral glucose raises plasma glucose levels by $\sim 40 \text{ mg/dL}$ over 30 minutes, while 20 g of oral glucose raises plasma glucose levels by $\sim 60 \text{ mg/dL}$ over 45 minutes. The initial response to treatment should be seen in approximately 10 to 20 minutes, and blood glucose levels should be evaluated again in 10- to 15-minute increments and again at 60 minutes when glucose levels often begin to fall to determine if additional treatment is necessary (1). Sulfonvlurea-induced hypoglycemia in patients with type 2 diabetes differs from insulin-induced hypoglycemia. Sulfonylurea-induced hypoglycemia can be prolonged and can recur, and therefore requires more persistent treatment (2). For mild to moderate hypoglycemic reactions, the following items, which contain about 15 g of carbohydrate, may be given (46,47). These food items are used because they are readily available and/or easy to carry when away from home, not because they are fast-acting (46,47):

- 4 to 6 oz fruit juice
- 4 to 6 oz regular soda
- three or four glucose tablets (4 g of glucose each)
- 2 tbsp raisins
- five or six Lifesavers candies
- 1 tbsp honey or corn syrup
- 4 tsp or four packets of granulated sugar

Retest the patient's blood glucose level 15 to 20 minutes after ingestion of the food. If the patient's blood glucose level is still low, give an additional 15 g of carbohydrate and retest in 15-minute increments until stabilized (1). Glucagon should be prescribed for all patients at significant risk of severe hypoglycemia (1). Individuals who have hypoglycemia unawareness or who experience one or more episodes of severe hypoglycemia should be advised to increase their glycemic targets to strictly avoid further hypoglycemia for at least several weeks (1).

Carbohydrate Replacement for Acute Illness, Missed Meals, or Delayed Meals

Acute illness or missed meals: Acute illness in persons with type 1 diabetes can increase the risk of diabetic ketoacidosis (1,2). During acute illness, the need for insulin continues and actually may increase due to an increased level of counterregulatory hormones associated with stress (2). Measuring blood glucose levels, measuring blood or urine ketones levels, drinking adequate amounts of fluids, and ingesting carbohydrate, especially if the blood glucose level is less than 100 mg/dL, are all important during acute illness (2). When illness or diagnostic tests prevent a diabetic individual from consuming the usual diet, systematic replacement of carbohydrate is appropriate. In adults, the daily ingestion of 150 to 200 g of carbohydrate (or 45 to 50 g every 3 to 4 hours), along with medication adjustments, should be sufficient to keep the glucose level in the goal range and prevent starvation ketosis (2). The carbohydrate value of the foods in the missed meal can be replaced with easily consumed liquids or soft foods as tolerated. Usually a missed meal may be satisfactorily replaced by at least 50 g of carbohydrate (or three to four carbohydrate choices) taken by mouth. The consumption of at least 50 g of carbohydrate every 3 to 4 hours has been recommended (1,2). If the patient is incapable of taking food by mouth, alternative nutrition support should be evaluated.

Delayed meals: When the meal is delayed and the blood glucose level is normal, carbohydrate should be given. Usually 15 g of carbohydrate (one fruit or bread exchange) every 30 to 45 minutes until the meal is served, or 15 to 30 g of carbohydrate for a 1- or 2-hour delay, protects the patient from hypoglycemia.

Enteral nutrition: For tube feedings, either a standard enteral formula (50% carbohydrate) or a lower-carbohydrate content formula (33% to 40% carbohydrate) may be used (2). Care should be taken not to overfeed patients because of the risk of exacerbating hyperglycemia (1,2).

Carbohydrate Content of Foods

Foods selected from the following list can be used as substitutes for foods of similar carbohydrate content in the missed meal or during illness.

Table C-3: Carbonyurate Content of Substitutions				
15 g of Carbohydrate				
Apple juice	½ cup	Jelly beans	nine	
Applesauce, sweetened	¼ cup	Jelly, jam	1 tbsp	
Applesauce, unsweetened	½ cup	Lifesavers candy	five or six	
Cooked cereal	½ cup	Orange juice	½ cup	
Cranberry juice	1/3 cup	Pineapple juice	½ cup	
Cream soup, made with water	1 cup	Popsicle bar (3 oz)	one	
Custard	½ cup	Regular soda	½ cup	
Gelatin	½ cup	Sherbet	¼ cup	
Grape juice	1/3 cup	Sugar, granulated	4 tsp	
Ice cream	½ cup	Syrup	1 tbsp	
12 g of Carbohydrate				
Milk (whole, reduced-fat, nonfat	t) 1 (cup		
Eggnog	1/2	cup		
Plain vogurt	1.	านท		

Table C-3: Carbohydrate Content of Substitutions

Note: Patients who experience hypoglycemia and are being treated with acarbose (Precose) or miglitol (Glyset) should be treated with glucose.

Glycemic Goals in a Hospital Setting

A rapidly growing body of evidence supports targeting glucose control in the hospital setting with the potential for reduced mortality and morbidity and improved health care outcomes (1). Studies of surgical patients, neurological patients, and patients acutely managed for myocardial infarction have demonstrated significant improvement in outcomes when glycemic goals are tightly managed (1). Hyperglycemia in the

hospital can result from factors including stress and decompensation of type 1 diabetes, type 2 diabetes, or other forms of diabetes; hyperglycemia may also be jatrogenic due to the administration or withholding of pharmacologic agents, including glucocorticoids and vasopressors (1). Insulin therapy should be initiated for treatment of persistent hyperglycemia starting at a threshold of not greater than 180 mg/dL (1). Once insulin therapy is started, the blood glucose levels of a majority of critically ill patients should target a range of 140 to 180 mg/dL (1). These revised ranges are currently suggested as the protocol for treatment to promote safety in achieving the desired glucose range without increasing risk for severe hypoglycemia (1). For non-critically ill patients' there is no clear evidence for specific blood glucose goals (1). If treated with insulin, the premeal blood glucose levels should generally be < 140 mg/dL with random blood glucose < 180 provided thesetargets can be safely achieved (1). More stringent targets may be appropriate in stable patients with previous tight control. Less stringent targets may be appropriate in those with severe comorbidities (1). Insulin should be initiated when necessary to achieve target values (1). Scheduled prandial insulin doses should be given in relation to meals and should be adjusted according to point-of-care glucose levels. The traditional sliding-scale insulin regimens are ineffective and are not recommended (1). In addition, glucose monitoring should be initiated in any patient not known to be diabetic who receives therapy associated with high risk for hyperglycemia, including high dose glucocorticoid therapy, initiation of enteral or parenteral nutrition, octreotide or immunosuppressive medications. If hyperglycemia is documented and persistent, treatment should be provided using the same glycemic goals as patients without known diabetes (1).

See Section III: Clinical Nutrition Management, Diabetes Mellitus: Considerations for Acute Illness

Diabetes Nutrition Management: Meal Planning Approaches (21)

Familiarity with the variety of meal planning approaches available can help dietitians more effectively teach patients how to reach their nutrition goals. The type of approach selected should depend on the goals for metabolic outcomes and the patient's nutrition needs, literacy, motivation, and lifestyle. The American Diabetes Association patient education publications are based on current diabetes treatment guidelines. Approaches that can be used for teaching meal planning include:

Basic nutrition guidelines	These guidelines provide the patient with an understanding of the basic principles of nutrition and guidance in selecting an adequately balanced diet for optimal health, eg, <i>Dietary Guidelines for Americans, Food Guide Pyramid</i> , and <i>Guide to Good Eating</i> .
Basic diabetes guidelines	These guidelines provide the patient with an understanding of the connection between food intake and metabolic outcomes. They give the patient direction in making appropriate food choices for managing diabetes, eg, the American Diabetes Association/American Dietetic Association's <i>The First Step in Diabetes Meal Planning for the Newly Diagnosed, Diabetes Food Guide Pyramid,</i> and <i>Healthy Food Choices</i> .
Menu approaches to meal planning	These approaches provide simple examples to assist patients with meal planning, eg, <i>The New Family Cookbook for People with Diabetes</i> and the American Diabetes Association's <i>Month of Meals</i> cookbook series and individualized menus.
Exchange lists for meal planning	This approach is designed to provide patient structure and guidance in meal planning. Exchanges are organized by calorie levels which is helpful in designing calorie-controlled meal plans. Food groups are organized based on foods that provide similar amounts of carbohydrate, protein and fat content. This helps to assist the patient in meal planning and meeting target nutrient and carbohydrate intake goals. The most common exchange list is the American Diabetes Association/Academy of Nutrition and Dietetics' <i>Eating Healthy with Diabetes: An Easy Read Guide; Choose Your Foods: Exchange List for Diabetes;</i> and <i>Exchange List for Weight Management.</i>
Counting approaches	These approaches provide structure with specific rules that are clearly identified. They allow optimal flexibility with food choices and meal planning, eg, American Diabetes Association/Academy of Nutrition and Dietetics' <i>Basic Carbohydrate Counting, Advanced Carbohydrate Counting,</i> and <i>The Diabetes Carbohydrate & Fat Gram Guide.</i>

Energy Balance, Overweight, and Obesity in Diabetes

Overweight and obesity affect insulin resistance and metabolic outcomes; therefore, weight loss is recommended for persons with diabetes who are overweight or obese as well as persons at risk for developing diabetes who are overweight or obese (1,2,4). Short-term studies have demonstrated that weight loss in subjects with type 2 diabetes is associated with decreased insulin resistance, improved measures of glycemia and dyslipidemia, and reduced blood pressure (Grade II) (4). The evidence-based nutrition guidelines encourage setting goals for a reasonable body weight, defined as a weight that the patient and the health care team acknowledge as being achievable and maintainable (1,2,4). A weight loss of 5% to 10% from baseline has positive effects on metabolic outcomes. The body mass index may be used to identify healthy weight ranges and estimate the desirable body weight. National guidelines for weight management can be applied to persons with diabetes who are overweight or obese and for whom weight loss is a primary health outcome (1,48). For patients with type 2 diabetes who have a BMI \geq 35 kg/m², bariatric surgery should be considered especially if the diabetes is difficult to control with lifestyle and pharmacologic therapy (1). Refer to Section IB: Nutrition Management of Bariatric Surgery.

A standard weight-loss diet that adjusts total energy intake to achieve an energy deficit of 500 to 1,000 kcal/day will initially achieve 1 to 2 lb of weight loss per week (48). Although many people can lose weight (as much as 10% of initial weight in 6 months) with these standard diets, without continued support and followup, people usually regain the weight that was lost (2,48). Low-fat, low-energy diets have traditionally been promoted for weight loss; however, three randomized controlled trials found that subjects on lowcarbohydrate diets lost more weight at 6 months than subjects on low-fat diets (49-51). A meta-analysis showed that at 6 months, low-carbohydrate diets were associated with greater improvements in triglycerides levels and high-density lipoprotein cholesterol concentrations than low-fat diets; however, the LDL cholesterol level was significantly higher on the low-carbohydrate diets (52). A more recent meta-analysis of restricted-carbohydrate diets in patients with type 2 diabetes revealed similar findings, with the exception of elevated LDL levels (53). The analysis showed that a decrease in carbohydrate intake from 65% to 35% of total energy yields an expected decrease of approximately 23% in the triglycerides level (53). A comparison of the studies demonstrated variable carbohydrate intakes (4% to 45% of total energy), and in most studies the carbohydrate intake fell below the Recommended Daily Allowance of 130 g/day (53). The authors concluded that a lower-carbohydrate diet can be beneficial in treating type 2 diabetes due to beneficial effects on the levels of glucose, A1C, and triglycerides; however, the impact of these diets on cardiovascular outcomes remains to be determined (53).

In a majority of studies, a low-carbohydrate diet begins with an induction phase of <30 g of carbohydrate per day with incremental increases to achieve $\sim 30\%$ to 40% of energy from carbohydrate (54). The American Diabetes Association recommends either a low-carbohydrate or low-fat, energy-restricted diet as an effective option for short-term (up to 1 year) weight loss in overweight and obese persons with type 2 diabetes (1,2). However, the American Diabetes Association does not recommend a low-carbohydrate diet in which the total carbohydrate intake is restricted to less than 130 g/day (2,53). Low-carbohydrate diets are broadly defined in the literature; the macronutrient composition from carbohydrate ranges from 4% to 45% in these diets (53). A review of popular diets by the U.S. Department of Agriculture defined a low-carbohydrate diet as containing <30% of energy from carbohydrate, a medium-carbohydrate diet as 30% to 55% of energy from carbohydrate, and a high-carbohydrate diet as >55% of energy from carbohydrate (54). Because considerable variations exist for low-carbohydrate diets, it is important for the dietitian to work collaboratively with the physician and patient in designing an optimal meal pattern that best supports desired metabolic outcomes. When prescribing a low-carbohydrate diet to diabetic patients, it is prudent to provide a carbohydrate amount that meets the Recommended Daily Allowance of 130 g/day because lower intakes eliminate many foods that are important sources of energy; fiber; water-soluble vitamins folate, thiamin, and pyridoxine; fatsoluble vitamins A and E; and minerals including calcium, potassium, and magnesium (53). The safety concerns that surround low-carbohydrate diets include increased uric acid levels in gout patients as well as other related side effects including constipation, diarrhea, dizziness, halitosis, headaches, and insomnia (55,56). Low-carbohydrate diets may not be suitable for children, reproductive-age women, and hypertensive individuals (55). In addition, the American Diabetes Association does not advocate the use of high-protein diets due to increased risks of glomerular hyperfiltration and accelerated renal complications associated with diabetes (2,53). Therefore, the ratio of protein generally should not exceed 20% of total energy when determining the macronutrient distribution for a low-carbohydrate meal plan. Patients who are prescribed a low-carbohydrate meal plan should be closely monitored and have frequent assessments of their lipid profile, renal function, protein intake, urine levels of ketones and glucose, as well as uric acid levels in patients at risk for gout. In addition, the hypoglycemic risk must be assessed to prevent episodes of hypoglycemia (1,2).

In selected patients, drug therapy as an adjunct to lifestyle change may be appropriate to achieve weight loss (1). However, it is common for patients to regain weight after the discontinuation of medications (1,57). In patients with severe obesity, surgical options, such as gastric bypass and gastroplasty, may be appropriate and allow significant improvement in glycemic control with reduction or discontinuation of medications (1,58).

For further information, refer to:

- Section IB: Nutrition Management of Bariatric Surgery,
- Section IC: Calorie-Controlled Diet for Weight Management,
- Section II: Body Mass Index (BMI),
- Section II: Estimation of Energy Expenditures, and
- Section III: Obesity and Weight Management.

For medical nutrition therapy outcomes monitoring and suggested delivery of care in the acute care setting, refer to "Morrison Nutrition Practice Guideline – Diabetes Mellitus (Uncontrolled and Complications)" (59).

Special Populations

Children and adolescents with diabetes: Nutrient requirements for children and adolescents with type 1 or type 2 diabetes are similar to the requirements for children and adolescents who do not have diabetes. The primary goal for children and adolescents with type 1 diabetes is achieving blood glucose goals that maintain normal growth and development without excessive hypoglycemia. Individualized food/meal plans and intensive insulin regimens can provide flexibility to accommodate irregular meal times and schedules as well as varying appetite and activity levels (1,2). Withholding food or having a child eat consistently without an appetite, in an effort to control blood glucose levels, should be discouraged (1,2). Nutrition for children and adolescents with type 2 diabetes should focus on healthy lifestyle changes that normalize glycemia (1,2). Cessation of weight gain with normal linear growth is a primary outcome that will help achieve glycemic goals in overweight and obese children with diabetes (1,2).

Pregnancy and lactation with preexisting diabetes: Nutrient requirements during pregnancy and lactation are similar for women with and without diabetes (1,2). The distribution of energy intake and carbohydrates in the meal plan of a pregnant woman with preexisting type 1 or type 2 diabetes should be based on her eating habits, blood glucose levels, and stage of pregnancy. Regular meals and snacks are important to avoid hypoglycemia due to the continuous fetal draw of glucose from the mother (1,2). An evening snack is usually necessary to decrease the potential for overnight hypoglycemia and fasting ketosis (1,2). (Refer to the discussion on pregnant women with preexisting diabetes and gestational diabetes, presented later in this section.)

Older adults with diabetes: The American Geriatrics Society emphasizes the importance of medical nutrition therapy for older adults with diabetes (1,2). Obese older adults with diabetes may benefit from modest energy restriction and an increase in physical activity to promote modest weight loss of 5% to 10% of body weight (1,2,59-63). Lifestyle modifications, and weight loss goals established for younger adults are also suggested for older adults. However, an involuntary weight loss of >10 lb or 10% of body weight in <6 months should be addressed in the nutrition assessment and medical nutrition therapy evaluation (2,64). Older nursing home residents who have diabetes tend to be underweight rather than overweight (2,3). Low body weight has been associated with greater morbidity and mortality in this population (3). Therefore, the imposition of dietary restrictions on elderly patients with diabetes in long-term care facilities is not warranted (2). Residents with diabetes should be served a regular menu with consistency in the amount and timing of carbohydrate intake (2). There is no evidence to support prescribing diets such as "no concentrated sweets" or "no sugar added" (2). In the institutionalized elderly, undernutrition is likely and caution should be exercised when prescribing weight loss diets (2). The treatment team should consider the resident's age, life expectancy, comorbidities, and preferences when outlining a plan for care (2). Adjusting the resident's medications to control glucose levels, lipid levels, and blood pressure rather than implementing food restrictions can reduce the risk of iatrogenic malnutrition (2,65).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. American Diabetes Association. Standards of medical care in diabetes—2013. Diabetes Care. 2013;36(suppl 1): 11S-66S.
- 2. American Diabetes Association. Nutrition recommendations and interventions for diabetes. *Diabetes Care*. 2008;31(suppl1): 61S-78S. (under revision in 2013)

- 3. American Diabetes Association. Diabetes nutrition recommendations for health care institutions: position statement. *Diabetes Care.* 2004;27(suppl 1):55S-57S.
- Type 1 and Type 2 Diabetes Evidence-Based Nutrition Practice Guideline for Adults. The Academy of Nutrition and Dietetics, 2008. In: The Academy of Nutrition and Dietetics Evidence Analysis Library at <u>http://www.andevidencelibrary.com</u>. Accessed January 23, 2013.
- 5. Sheard NF, Clark NG, Brand-Miller JC, Franz MJ, Pi-Sunyer FX, Mayer-Davis E, Kulkarni K, Geil P. Dietary carbohydrate (amount and type) in the prevention and management of diabetes. A statement by the American Diabetes Association. *Diabetes Care*. 2004;27:2266-2269.
- 6. Gannon MC, Nuttall FQ, Westphal SA, Fang D, Ercan-Fang N. Acute metabolic response to high-carbohydrate, high-starch meals compared with moderate carbohydrate, low-starch meals in subjects with type 2 diabetes. *Diabetes Care.* 1998;21:1619-1626.
- Wolever TMS, Nguyen P-M, Chiasson J-L, Hunt JA, Josse RG, Palmason C, Rodger NW, Ross SA, Ryan EA, Tan MH. Determinants of diet glycemic index calculated retrospectively from diet records of 342 individuals with non-insulin-dependent diabetes mellitus. *Am J Clin Nutr.* 1994;59:1265-1269.
- 8. O'Dea K, Snow P, Nestel P. Rate of starch hydrolysis in vitro as a predictor of metabolic responses to complex carbohydrate in vivo. *Am J Clin Nutr.* 1981;34:1991-1993.
- 9. Jarvi A, Karlstrom B, Granfeldt Y, Bjorck I, Vessby B. The influence of food structure on postprandial metabolism in patients with NIDDM. *Am J Clin Nutr*. 1995;61:837-842.
- 10. Nielsen OH, Nielsen GL. Preprandial blood glucose values: influence on glycemic response studies. *Am J Clin Nutr*. 1989;53:1243-1246.
- 11. Rasmussen I, Hermansen K. Preprandial blood glucose values and glycemic responses in insulin-dependent diabetes mellitus at constant insulinemia. *Am J Clin Nutr.* 1991;53:520-523.
- 12. Fraser RJ, Horowitz M, Maddox AF, Harding PE, Chatterton BE, Dent J. Hyperglycemia slows gastric emptying in type 1 (insulindependent) diabetes mellitus. *Diabetologia*. 1990;33:675-680.
- 13. Schvarcz E, Palmer M, Aman J, Lindkvist B, Beckman K-W. Hypoglycaemia increases the gastric emptying rate in patients with type 1 diabetes mellitus. *Diabet Med.* 1993;10:660-663.
- 14. Franz MJ. The glycemic index: not the most effective nutrition therapy intervention. Diabetes Care. 2003; 26:2466-2468.
- 15. Brand-Miller J, Hayne S, Petocz P, Colagiuri S. Low-glycemic diets in the management of diabetes: a meta-analysis of randomizedcontrolled trials. *Diabetes Care.* 2002; 26:2261-2267.
- 16. Position of the American Dietetic Association: health implications of dietary fiber. J Am Diet Assoc. 2008;108:1716-1731.
- 17. Chandalia M, Garg A, Lutjohann D, von Bergmann K, Grundy SM, Brinkley LJ. Beneficial effects of high dietary fiber intake in patients with type 2 diabetes mellitus. *N Eng J Med.* 2000;23:1461-1466,
- 18. Englyst HN, Veenstra J, Hudson GJ. Measurement of rapidly available glucose (RAG) in plant food: a potential in vitro predictor of the glycemic response. *Br J Nutr.* 1996;75:327-337.
- 19. Wolever TMS, Hamad S, Chiasson J-L, Josse RG, Leiter LA, Rodger NW, Ross SA, Ryan EA. Day-to-day consistency in amount and source of carbohydrate intake associated with improved glucose control in type 1 diabetes. *J Am Coll Nutr*. 1999;18:242-247.
- Rabasa-Lhoret R, Garon J, Langelier H, Poisson D, Chiasson J-L. Effects of meal carbohydrate content on insulin requirements in type 1 diabetic patients treated intensively with the basal-bolus (ultralente-regular) insulin regimen. *Diabetes Care*. 1999;22:667-673.
- 21. Holler HJ, Pastors JG, eds. Diabetes Medical Nutrition Therapy. Chicago, Ill: American Dietetic Association; 1997.
- 22. Delahanty LM, Halford BH. The role of diet behaviors in achieving improved glycemic control in intensively treated patients in the Diabetes Control and Complications Trial. *Diabetes Care*. 1993;16:905-912.
- 23. DCCT Research Group. Weight gain associated with intensive therapy in the diabetes control and complications trial. *Diabetes Care*. 1988;11:567-573.
- 24. Wing RR, Klein R, Moss SE. Weight gain associated with improved glycemic control in population-based sample of subjects with type 1 diabetes. *Diabetes Care.* 1990;13:1106-1109.
- 25. Carlson MG, Campbell PJ. Intensive insulin therapy and weight gain in IDDM. Diabetes. 1993;42:1700-1707.
- 26. Purnell JQ, Hokanson JE, Marconvina SM, Steffes MW, Cleary PA, Brunzell JD. Effect of excessive weight gain with intensive therapy of type 1 diabetes on lipid levels and blood pressure. *JAMA*. 1998;280:140-146.
- 27. American Diabetes Association. Physical activity/exercise and diabetes mellitus: position statement. *Diabetes Care*. 2004;27(suppl 1);58S-62S.
- 28. Arnold L, Mann J, Ball M. Metabolic effects of alterations in meal frequency in type 2 diabetes. Diabetes Care. 1997;20:1651-1654.
- 29. Beebe CA, Van Cauter E, Shapiro T, Tillel H, Lyons R, Rubenstein A, Polonsky K. Effect of temporal distribution of calories on diurnal patterns of glucose levels and insulin secretion in NIDDM. *Diabetes Care*. 1990;13:748-755.
- 30. Hansen HP, Tauber-Lassen E, Jensen BR, Parving HH. Effect of dietary protein restriction on prognosis in patients with diabetic nephropathy. *Kidney Int.* 2002;62:220-228.
- 31. Gannon MC, Nuttal FQ. Effect of a high-protein, low-carbohydrate diet on blood glucose control in people with type 2 diabetes. *Diabetes*. 2004;53:2375-2382.
- 32. Gannon MC, Nuttall FZ, Saeed A, Jordan K, Hoover H. An increase in dietary protein improves the blood glucose response in persons with type 2 diabetes. *Am J Clin Nutr.* 2003;78:734-741.
- 33. American Diabetes Association. Dyslipidemia management in adults with diabetes: position statement. *Diabetes Care*. 2004;27(suppl 1):68S-71S.
- Grundy SM, Cleeman JI, Merz CN, Brewer HB Jr, Clark LT, Hunninghake DB, Pasternak RC, Smith SC Jr, Stone NJ for the Coordinating Committee of the National Cholesterol Education Program. Implications of recent trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines [published erratum appears in *Circulation*. 2004;110:763]. *Circulation*. 2004; 110:227-239.
- 35. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486-2497.
- 36. AHA Dietary Guidelines: revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation*. 2000;102:2284-2299.
- 37. Garg A, Bantle JP, Henry RR, Coulston AM, Griver KA, Raatz SK, Brinkley L, Chen YD, Grundy SM, Huet BA, Reaven GM. Effects of varying carbohydrate content of diet in patients with non-insulin–dependent diabetes mellitus. *JAMA*. 1994;271:1421-1428.
- 38. Garg A. High-monounsaturated-fat diets for patients with diabetes mellitus: a meta-analysis. Am J Clin Nutr. 1998;67:577S-582S.
- 39. Montori VM, Farmer A, Wollan PC, Dinneen SF. Fish oil and glycemic control in diabetes: a quantitative systematic review. *Diabetes Care*. 2000;23:1407-1415.

- 40. Mozaffarian D, Bryson CL, Lemaitre RN, Burke GL, Siscovick DS. Fish intake and risk of incident heart failure. J Am Coll Cardiol. 2005;45:2015-2021.
- 41. Erkkila AT, Lichtenstein AH, Mozaffarian D, Herrington DM. Fish intake is associated with a reduced progression of coronary artery atherosclerosis in postmenopausal women with coronary artery disease. *Am J Clin Nutr.* 2004;80:626-630.
- 42. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER, Simons-Morton DG, Karanja N, Lin P-H for the DASH–Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med.* 2001;344:3-10.
- 43. Althuis MD, Jordan NE, Ludington EA, Wittes JT. Glucose and insulin responses to dietary chromium supplements: a meta-analysis. *Am J Clin Nutr.* 2002;76:148-155.
- 44. Cryer PE. Hypoglycaemia: the limiting factor in the glycaemic management of type 1 and type 2 diabetes. *Diabetologia*. 2002;45:937-948.
- 45. Brodows RG, Williams C, Amatruda JM. Treatment of insulin reactions in diabetes. JAMA. 1984;252:3378-3381.
- 46. Medical Management of Non-Insulin-Dependent (Type II) Diabetes. 3rd ed. Alexandria, Va: American Diabetes Association; 1994.
- 47. Slama G, Traynard PY, Desplanque N, Pudar H, Dhunputh I, Letanoux M, Bornet FRJ, Tchobroutsky G. The search for an optimized treatment of hypoglycemia. *Arch Intern Med.* 1990;150:589-593.
- 48. National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.* Bethesda, Md: National Institutes of Health; 1998. NIH publication No. 98-4083. Available at: http://www.nhlbi.nih.gov/nhlbi/htm. Accessed September 24, 2002.
- 49. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med.* 2003;348:2082-2090.
- Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF. The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med.* 2004;140:778-785.
- 51. Gardner C, Kiazand A, Alhassan S, Soowon K, Stafford R, Balise R, Kraemer H, King A. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related factors among overweight premenopausal women. *JAMA*. 2007;297:969-977.
- 52. Nordmann AJ, Nordmann A, Briel M, Keller U, Yancy WS Jr, Brehm BJ, Bucher HC. Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. *Arch Intern Med.* 2006; 166:285-293.
- 53. Kirk JK, Graves DE, Craven TE, Lipkin EW, Austin M, Margolis KL. Restricted-carbohydrate diets in patients with type 2 diabetes: a meta-analysis. J Am Diet Assoc. 2008; 108:91-100.
- 54. Kennedy ET, Bowman SA, Spence JT, Freedman M, King J. Popular diets: correlation to health, nutrition, and obesity. J Am Diet Assoc. 2001;101:411-420.
- 55. Crowe TC. Safety of low-carbohydrate diets. *Obes Rev.* 2005;6:235-245.
- 56. Last AR, Wilson SA. Low-carbohydrate diets. Am Fam Physician. 2006;73:1942-1948.
- 57. Leung WY, Neil TG, Chan JC, Tomlinson B. Weight management and current options in pharmacotherapy: orlistat and sibutramine. *Clin Ther.* 2003;25:58-80.
- 58. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, Schoelles K. Bariatric surgery: a systematic review and meta-analysis. *JAMA*. 2004;292:1724-1737.
- Morrison Nutrition Practice Guideline Diabetes Mellitus (Uncontrolled and Complications). In: Inman-Felton A, Smith K. Morrison Nutrition Practice Guidelines. Atlanta, Ga: Morrison Management Specialists; 2009. Available at: www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition.
- 60. Brown AF, Mangione CM, Saliba D, Sarkisian CA. Guidelines for improving the care of the older person with diabetes mellitus. *J Am Geriatr Soc.* 2003;51:265S-280S.
- 61. Miller CK, Edwards L, Kissling G, Sanville L. Nutrition education improves metabolic outcomes among older adults with diabetes mellitus: results from a randomized controlled trial. *Prev Med.* 2002;34:252-259.
- 62. Horani MH, Mooradian AD. Management of obesity in the elderly: special considerations. *Treat Endocrinol.* 2002;1:387-398.
- 63. Heiat A, Vaccarino V, Krumholz HM. An evidence-based assessment of federal guidelines for overweight and obesity as they apply to elderly persons. *Arch Intern Med.* 2001;161:1194-1203.
- 64. Roberts SB, Hajduk CL, Howarth NC, Russell R, McCrory MA. Dietary variety predicts low body mass index and inadequate macronutrient and micronutrient intakes in community-dwelling older adults. *J Gerontol A Biol Sci Med Sci*. 2005;60:613-621.
- 65. Reed RL, Mooradian AD. Management of diabetes mellitus in the nursing home. Ann Long Term Care. 1998;6:100-107.

MEDICAL NUTRITION THERAPY FOR GESTATIONAL DIABETES MELLITUS AND PREGNANCY WITH PREEXISTING DIABETES MELLITUS

Overview

Previously, gestational diabetes mellitus (GDM) was defined as any degree of glucose intolerance with onset or first recognition during pregnancy (1). In 2011, The American Diabetes Association revised the definition along with new diagnostic criteria as a result of an increase in the incidence of pregnant women being identified to have undiagnosed overt diabetes and studies demonstrating adverse maternal, fetal and neonatal outcomes using previously established standards considered to be normal for pregnancy (1). It is now recommended that women found to have diabetes at their first prenatal visit receive a diagnosis of overt, not gestational diabetes (1,2). An increase in insulin-antagonist hormone levels and resulting insulin resistance occurs in the second and third trimester of pregnancy. Women who are unable to produce adequate insulin to maintain normal glucose concentrations during the second and third trimester are then diagnosed with gestational diabetes mellitus (1,2). After delivery, in a majority of women with GDM blood glucose will return to normal but these women are at increased risk of developing type 2 diabetes (1,2).

Nutrition Assessment and Diagnosis

The results of the Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study, a large-scale, multinational, epidemiologic study demonstrated that the risk of adverse maternal, fetal, and neonatal outcomes continuously increased as a function of maternal glycemia at 24 to 28 weeks of gestation, even within ranges previously considered to be normal for pregnancy (1). These results led to reconsideration of the diagnostic criteria for GDM by an international consensus of multiple obstetrical and diabetes organizations (1,2). The new criteria established in 2011 will significantly increase the prevalence of GDM, primarily because only one abnormal value, not two is sufficient to make the diagnosis (1,2). With increasing identification will come a greater demand for management and treatment of GDM. However, it is important to note that 80 to 90% of newly diagnosed cases evaluated in these studies had mild GDM (whose glucose values overlapped with the new thresholds), and likely can be managed with lifestyle therapy alone (1,2).

Women with risk factors for diabetes should be screened for undiagnosed type 2 diabetes at the first prenatal visit using standard diagnostic criteria established by the American Diabetes Association (1,2). Risk factors for screening include (1):

- overweight (BMI > 25 kg/m²)
- first-degree relative with diabetes
- high-risk race/ethnic group for prevalence of diabetes
- delivered a baby weight > 9 lbs or previous diagnosis of GDM
- polycystic ovarian syndrome (PCOS)
- A1C > 5.7%, impaired glucose tolerance (IGT), or impaired fasting glucose (IFG) on previous testing

All pregnant women not known to have diabetes, should be tested for GDM at 24 to 28 weeks of gestation (1,2). The diagnosis of GDM is based on the results of the 75-g 2-hour oral glucose tolerance test (OGTT) with plasma glucose measurement fasting and at 1 and 2 hours (2). The OGTT should be performed in the morning over an overnight fast of at least 8 hours (2). Diagnosis of GDM is made when any of the following plasma glucose values are exceeded (1,2):

- Fasting: > 92 mg/dL (5.1 mmol/L)
- 1 hour: > 180 mg/dL (10.0 mmol/L)
- 2 hour: >153 mg/dL (8.5 mmol/L)

Because the A1C test reflects the glycemic profile in the last 10 weeks, it is not used to determine need for additional therapy in GDM. It can however, be used in the first trimester to diagnose type 2 diabetes (A1C > 6.5%) (1,2). See Section II: Diagnostic Criteria for Diabetes Mellitus.

The American College of Obstetricians (ACOG) continues to recommend a 2-step approach to screening and diagnosis. ACOG recommends all pregnant women be screened for GDM, whether patient history, clinical risk factors, or a 50-g, 1-hour glucose challenge test at 24 to 28 weeks (3). The diagnosis of GDM is made based on the result of the 100-g, 3-hour OGTT that requires two or more thresholds be met or exceed to make the

Medical Nutrition Therapy for Gestational Diabetes Mellitus

diagnosis of GDM. The National Institutes of Health is currently planning a consensus development on these two approaches (3). Likely registered dietitians will see the diagnosis of GDM using either one of the approaches discussed in this section (3).

Clinical Monitoring

Maternal metabolic monitoring should be directed at detecting hyperglycemia severe enough to increase risks to the fetus (4). Fasting hyperglycemia (>105 mg/dL) may be associated with an increased risk of intrauterine fetal death during 4 to 8 weeks of gestation (5). For both GDM and preexisting diabetes, self-monitoring of blood glucose levels is essential for the management of diabetes during pregnancy. Urine should also be tested for ketones on a routine basis, and these test results can be used to detect insufficient energy or carbohydrate intake in women treated with energy restriction (1-5). Monitoring the urine glucose levels is not appropriate in GDM (3,5). The patient's blood pressure and urine protein levels should be monitored to detect hypertensive disorders (3,5). Monitoring schedules for patients with preexisting diabetes have been developed by the American Diabetes Association (1,3,5). Initially it is suggested that women test four times per day in a fasting state and one to two hours after each meal (1).

Nutrition Intervention for Gestational Diabetes Mellitus

All women with GDM should receive nutrition counseling by a registered dietitian, which is consistent with the recommendations of the American Diabetes Association (1,4,5,6). Medical nutrition therapy for GDM primarily involves a carbohydrate-controlled meal plan that promotes optimal nutrition for maternal and fetal health with adequate energy for appropriate gestational weight gain, achievement and maintenance of normoglycemia, and the absence of ketosis (3,4,6). Specific therapeutic goals are based on an individual nutrition assessment and self-monitoring of blood glucose levels. Optimum neonatal outcomes occur more frequently in women who gain the recommended amount of weight based on prepregnancy body mass index (BMI) levels established by the Institute of Medicine (Grade 1)* (6).

Overweight and obese women with GDM benefit from nutrition counseling by a dietitian to decrease the rate of weight gain, decrease the levels of fasting and postpartum serum glucose, and normalize infant birth weight (Grade I) (6). Weight loss is not recommended during pregnancy; however, modest energy and carbohydrate restriction may be appropriate for overweight and obese women with GDM (4). Refer to Table C-4 for the current weight gain guidelines and Section IA: Nutrition Management During Pregnancy and Lactation (7-9).

Table C-4: Recommended weight Gam for Freghant women based on Frepregnancy DMI"			
Prepregnancy Weight Classification	BMI (kg/m ²)	Recommended Total Gain (kg [lb])	
Low BMI	<18.5	12.5-18 (28-40)	
Normal BMI	18.5-24.9	11.5-16 (25-35)	
Overweight	25.0-29.9	7-11.5 (15-25)	
Obese	>30.0	5-9	
For women carrying multiple fetuses, the following weight gain is appropriate ^b			
Twin pregnancy			
Normal BMI	17.0-25.0	0 kg (37-54 lb)	
Overweight	14.0-23.0 kg (31-50 lb)		
Obese	11.0-19.0	0 kg (25-42 lb)	
Triplet pregnancy	20.5-11.3	3 kg (45-55 lb) (9)	
^a Sources: Institute of Medicine of the National Academies. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington, DC:			

Table C-4: Recommended Weight Gain for Pregnant Women Based on Prepregnancy BMI^a

^aSources: Institute of Medicine of the National Academies. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington, DC: The National Academies Press; 2009. Available at <u>http://www.iom.edu/~/media/Files/Report%20Files/2009/Weight-Gain-During-Pregnancy-Reexamining-the-Guidelines/Report%20Brief%20-%20Weight%20Gain%20During%20Pregnancy.pdf</u>. Accessed January 23, 2013.

^bBrown JE, Carlson M. Nutrition and multifetal pregnancy. *J Am Diet Assoc.* 2000;100:343-348.

Energy Requirements

The MNT should include adequate energy and nutrients to meet the needs of pregnancy and should be consistent with the maternal blood glucose goals. Cohort studies show that energy requirements are highly variable and can be met by increasing food intake, decreasing physical activity, or decreasing fat storage. Therefore, recommendations for energy levels are best determined by monitoring weight gain, physical activity, appetite, daily food intake, and glucose and ketone records (Grade I) (6). Refer to Table C-5 for the suggested daily energy intake for pregnant diabetic women (5,10).

Table C-5: Recommended Dail	ly Energy Intake for Pregnant Diabetic Women
------------------------------------	--

Prepregnancy Weight Status	Energy (kcal/kg per day)	Energy (kcal/lb per day)
Desirable body weight	30	13.6
>120% of desirable body weight, BMI >30 kg/m ²	25	10.9
<90% of desirable body weight	36-40	16.3-18.2

Sources: American Diabetes Association. Gestational diabetes mellitus: position statement. *Diabetes Care*. 2004;27(suppl 1):88S-90S. *Medical Management of Pregnancy Complicated by Diabetes*. 2nd ed. Alexandria, Va: American Diabetes Association; 1995.

Managing Ketosis

The prevention of ketosis is a primary outcome of medical nutrition therapy in GDM (5). Case-control and cohort studies have found an association between ketonemia and ketonuria during GDM and a lower intelligence quotient in offspring (Grade II) (6). Ketone testing is an important part of self-monitoring and aids in adjusting the energy intake level, carbohydrate distribution, and physical activity level (Grade II) (6). To prevent ketosis, adequate energy intake and the appropriate distribution of meals and snacks is important. An evening snack may be needed to prevent accelerated ketosis overnight (4). Low-energy diets in obese women with GDM can result in ketonemia and ketonuria (4). Randomized controlled trials have shown that restricting energy intake to 1,200 kcal/day in obese women (BMI >30 kg/m²) with GDM results in ketonemia or ketonuria, whereas restricting the daily energy intake to a more liberal amount of ~1,800 kcal (25 kcal/kg of actual weight) does not result in ketonemia or ketonuria (Grade I) (6). Moderate energy restriction, defined as a 30% reduction in estimated energy needs, in obese women with GDM may improve glycemic control and reduce excessive maternal weight gain without the development of ketonemia; however, insufficient data are available to determine the effect of such diets on perinatal outcomes (4). Daily food records, weekly weight checks, and ketone testing remain paramount in assessing the adequacy of a patient's energy intake (4).

Carbohydrate Intake

The amount and kind of carbohydrate in meals and snacks are key to maintaining optimal blood glucose levels and reducing the need for insulin while controlling maternal weight gain and infant birth weight (Grade II) (4,6,11). The amount and distribution of carbohydrate intake should be based on the clinical outcome measures of hunger, plasma glucose levels, weight gain, and ketone levels (4). A minimum of 175 g of carbohydrate should be provided on a daily basis (Grade II) (3,4,6). A diet comprised of 42% to 45% carbohydrate distributed among six to eight meals and snacks throughout the day with smaller amounts of carbohydrate (15 to 45 g) at breakfast and snacks promotes normal blood glucose levels (Grade II) (6). Lower carbohydrate intake is suggested at breakfast, because carbohydrate is generally less well tolerated at breakfast than at other meals during pregnancy (4). It has been suggested that nonnutritive sweeteners may be used in moderation as a means to control total energy intake and promote blood glucose control in GDM (5). While there are recognizable benefits of the use of nonnutritive sweeteners with maintenance of blood glucose control, there is limited evidence to support the use or nonuse of nonnutritive sweeteners in pregnancy and even less evidence addressing this issue specifically in GDM (Grade IV) (6). Refer to Section IA: Nutrition Management During Pregnancy and Lactation for additional information about the nutrient requirements and use of nonnutritive sweeteners during pregnancy.

Self-Monitoring of Blood Glucose Goals

Self-monitoring of blood glucose levels is an essential component of maintaining desirable blood glucose levels in women with GDM. Studies have shown that the best outcomes are achieved when both fasting and 1- or 2-hour postprandial blood glucose levels are monitored three to eight times per day and used to modify food intake or meal patterns and physical activity levels (Grade I) (6). The American Diabetes Association recommends that people with type 1 diabetes and pregnant women who take insulin check their blood glucose levels three or more times daily so that they can adjust their food intake, physical activity level, and/or insulin dosage to meet blood glucose goals (1). Evidence has shown that mean serum glucose levels of 86 mg/dL increase the risk for small for gestational age infants and that mean glucose levels of 105 mg/dL increase the risk for small (Grade I) (6). The following are glucose goals established for GDM by the American Diabetes Association based on capillary glucose concentrations (1):

Table C-6: Blood Glucose Goals in Gestational Diabetes Mellitus			
Time of Measurement	Capillary-Blood Glucose		
	(mg/dL)		
Fasting	<95		
1-h postprandial	<140		
2-h to 6-h postprandial	<120		

-- ...

Source: Metzger BE, Buchanan TA, Coustan DR, de Levia A, Dunger DB, Hadden DR et al. Summary and recommendations of the Fifth International Workshop Conference on Gestational Diabetes Mellitus. Diabetes Care. 2007;30(suppl. 2): S251-S260.

For women with pre-existing type 1 or type 2 diabetes who become pregnant, the following are suggested optimal glycemic goals, if they can be achieved without excessive hypoglycemia (1):

• Premeal, bedtime, and overnight glucose	60 to 99 mg/dL
 Peak postptrandial glucose 	100 to 129 mg/dL
• A1C	< 6.0%

Source: Kitzmiller JL, Block JM, Brown FM, Catalano PM, Conway DL, Coustan DR et al. Managing preexisting diabetes for pregnancy: summary of evidence and consensus recommendations for care. Diabetes Care. 2008;31:1060-1079.

The newer models of glucose meters are often plasma calibrated. Plasma-calibrated meters usually read 10% to 15% higher than whole-blood glucose meters. Therefore, healthcare providers and patients should be aware of which test is being used to measure glucose levels (4,5). Plasma meters also allow the patient to test glucose levels at other sites, such as the thigh, arm, or calf. To account for differences in blood glucose levels from different sites, plasma glucose is measured instead of whole-blood glucose because the values are more consistent from site to site.

Medication Management

Research indicates that pharmacological therapy, such as the use of insulin, insulin analogs and glyburide, improves glycemic control and reduces the incidence of poor maternal and neonatal outcomes (Grade II) (6). Insulin therapy is recommended if medical nutrition therapy fails to maintain the following self-monitored glucose levels: a fasting plasma glucose level of ≤105 mg/dL, a 1-hour postprandial blood glucose level of <155 mg/dL, and/or a 2-hour postprandial plasma glucose level of $\leq 130 \text{ mg/dL}$ (Grade I) (6). Human insulin should be used when insulin is prescribed, and self-monitoring blood glucose records should guide the dosage and timing of insulin If insulin therapy is added to nutrition therapy, a primary goal must be to maintain consistent therapy. carbohydrate intake at meals and snacks to facilitate insulin adjustments (4). The prevention of ketosis may require multiple daily insulin injections and the distribution of dietary carbohydrate into small frequent meals (three meals and three or four snacks). Insulin requirements normally increase as the pregnancy proceeds, and the insulin regimen must be continually adjusted throughout the pregnancy. Blood glucose monitoring by the patient is an essential part of this process (4,5,11). The heightened insulin requirement will plummet within hours of delivery. Metabolic control during labor, delivery, and the postpartum period should be managed by frequent determinations of blood glucose levels and adjustments to the insulin dose.

A number of drugs commonly used in the treatment of patients with diabetes may be relatively or absolutely contraindicated during pregnancy (1). Statins and angiotensin-converting enzyme inhibitors, which are used to manage disorders in lipid metabolism or hypertension, should be discontinued prior to conception (1). Among the oral antidiabetic drugs, metformin and acarbose are classified as category B drugs (no evidence of risk in humans), while all other oral antidiabetic drugs including sulfonylureas (eg, glyburide) are classified as category C drugs (risk cannot be ruled out) (1,4,5). The potential risks and benefits of oral antidiabetic agents in the preconception period must be carefully evaluated, recognizing that data are insufficient to establish the safety of these agents in pregnancy (1). Particularly for patients with preexisting diabetes, medication evaluation should be carefully assessed as part of preconception care (1).

Physical Activity

Regular physical activity reduces insulin resistance, lowers fasting and postprandial glucose concentrations, and may be used as an adjunct to nutrition therapy to improve maternal glycemia (Grade II) (4.6). The optimal frequency and intensity of exercise for lowering maternal glucose concentrations have not been determined; but, it appears that a minimum of three weekly exercise sessions, each longer than 15 minutes, is required to modify maternal glucose levels. In addition, 2 to 4 weeks of regular exercise may be required before a reduction of glycemia occurs (4). Regular physical activity has also been shown to reduce excessive weight

Medical Nutrition Therapy for Gestational Diabetes Mellitus

gain during pregnancy (Grade II) (6). Persons should frequently monitor their blood glucose levels before and after physical activity. Persons who do exercise should be aware that prolonged exercise (> 60 minutes) is more likely to cause hypoglycemia in pregnancy (Grade II) (6).

Follow-up Evaluation and Monitoring

The recurrence rate of GDM in subsequent pregnancies is 30% to 65% (Grade I) (6). The American Diabetes Association recommends a follow-up evaluation of each woman diagnosed with gestational diabetes (1,4), because these women may be prone to the development of type 2 diabetes later in life (Grade I) (4). Reclassification of maternal glycemic status should be performed 6 to 12 weeks after delivery (1). If postpartum glucose levels are normal, then glycemia should be reassessed at a minimum of 3-year intervals (1). Women with impaired fasting glucose or impaired glucose tolerance in the postpartum period should be annually tested for diabetes (1,5). See Section II: Diagnostic Criteria for Diabetes Mellitus. It is prudent to provide nutrition counseling and guidance to these women after the birth of their children. Lifestyle modifications aimed at reducing weight or preventing weight gain and increasing physical activity after pregnancy are recommended to reduce the risk of developing type 2 diabetes mellitus (1).

Dietary Recommendations for Pregnant Women With Preexisting Diabetes

Preconception care is a key factor in successful pregnancy outcomes for persons with preexisting diabetes (type 1 or type 2). All women with diabetes should be educated regarding the need for good blood glucose control before pregnancy and should participate in family planning (1). A woman's A1C level should be as close to normal as possible (<6.5%) before conception is attempted (1). Medication use should be evaluated before conception because drugs commonly used to treat diabetes and its complications may be contraindicated or not recommended in pregnancy (1). Nutrient requirements during pregnancy and lactation are similar for women with and without diabetes (4). For pregnancy complicated by diabetes, nutrition therapy should attempt to achieve and sustain optimal maternal blood glucose control. A favorable pregnancy outcome is defined as a gestational duration of 39 to 41 weeks and the birth of a live infant weighing 6.8-7.9 lb (3.1-3.6) (8). During pregnancy with prior onset of type 1 or type 2 diabetes, the distribution of energy intake and carbohydrates in the meal plan should be based on the woman's eating habits, blood glucose records, and stage of pregnancy. Regular meals and snacks are important to avoid hypoglycemia due to the continuous fetal draw of glucose from the mother (4). An evening snack is usually necessary to decrease the potential for overnight hypoglycemia and fasting ketosis (4). Energy intake to achieve appropriate weight gain may be estimated based on the percent of desirable body weight before the pregnancy (4.5). Pregravid BMI may be used to estimate a goal for weight gain during pregnancy (7-9).

Lactation

Breast-feeding is recommended for infants of women with preexisting diabetes or GDM. Research indicates that even short duration of breastfeeding results in long-term improvements in glucose metabolism and may also reduce the risk of type 2 diabetes in children (Grade III) (6). Successful lactation requires planning and coordination of care (12). In most situations, breast-feeding mothers require less insulin because of the energy expended by nursing. Lactating women have reported fluctuations in blood glucose levels related to nursing sessions, and they often require a carbohydrate-containing snack before or during nursing sessions (12).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. American Diabetes Association. Standards of medical care in diabetes—2013. Diabetes Care. 2013;36(suppl 1): 11S-66S
- 2. American Diabetes Association. Diagnosis and classification of diabetes mellitus: position statement. *Diabetes Care*. 2013;36(suppl 1): 67S-74S.
- 3. Gestational Diabetes. In: *The Nutrition Care Manual*. The Academy of Nutrition and Dietetics. Updated annually. Available at: nutritioncaremanual.org. Accessed January 23, 2013.
- 4. American Diabetes Association. Nutrition recommendations and interventions for diabetes: position statement. Diabetes Care. 2008;31 (suppl 1):61S-78S.
- 5. American Diabetes Association. Gestational diabetes mellitus: position statement. Diabetes Care. 2004;27(suppl 1):88S-90S.
- 6. *Gestational Diabetes Evidence-Based Nutrition Practice Guideline*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition Dietetics; 2008. Available at: http://www.andevidencelibrary.com. Accessed January 23, 2013.
- 7. Food and Nutrition Board. *Nutrition During Pregnancy*. Part I: Weight Gain. Part 2: Nutrient Supplements. Washington, DC: Institute of Medicine, National Academy of Sciences; 1990.
- 8. Butte NF, King JC. Energy requirements during pregnancy and lactation. Public Health Nutr. 2005;8:1010-1027.
- 9. Brown JE, Carlson M. Nutrition and multifetal pregnancy. J Am Diet Assoc. 2000;100:343-348.
- 10. Medical Management of Pregnancy Complicated by Diabetes. 2nd ed. Alexandria, Va: American Diabetes Association; 1995.
- 11. Major CA, Henry MJ, De Veciana M, Morgan MA. The effects of carbohydrate restriction in patients with diet-controlled gestational diabetes. *Obstet Gynecol.* 1998;91:600-604.
- 12. Reader D, Franz MJ. Lactation, diabetes, and nutrition recommendations. Curr Diab Rep. 2004;4:370-376.

Medical Nutrition Therapy for Gestational Diabetes Mellitus

Bibliography

Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome. *J Am Diet Assoc.* 2008;108:553-561.

Position of the American Dietetic Association and American Society for Nutrition: Obesity, Reproduction, and Pregnancy Outcomes. JAm Diet Assoc. 2009;109:918-927.

Position of the American Dietetic Association: Promoting and supporting breastfeeding. J Am Diet Assoc. 2009;109:1926-1942.

ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists. No. 30, September 2001 (replaces Technical Bulletin No. 200, December 1994). Gestational diabetes. *Obstet Gynecol.* 2001;98:525-538.

DIETARY MANAGEMENT WITH THE EXCHANGE SYSTEM

This system is used in the calculation of energy-controlled and diabetic diets. Various exchange groups within the system can also be used in the calculation of fat-controlled diets.

Each list shows the kinds and amounts of foods to use for one exchange. Exchanges are based upon roughly equivalent carbohydrate, protein, and fat content. Exchanges have been defined in household measures. For food-service portioning, they may be further defined by serving utensils or multiples (or fractions) of the standard portion.

These lists are a reference for initial planning of diets, menu writing, and carbohydrate counting. Additional foods and recipes can be translated into exchanges and/or grams of carbohydrates through the evaluation of macronutrient content. The following chart shows the amount of nutrients in one serving from each list.

Food List	Carbohydrate	Protein	Fat	Energy
	(g)	(g)	(g)	(kcal)
Carbohydrates				
Starch: breads,	15	0-3	0-1	80
cereals and grains;				
starchy vegetables;				
crackers and snacks;				
and beans, peas,				
and lentils				
Fruits	15			60
Milk				
Fat-free, low-fat, 1%	12	8	0-3	100
Reduced-fat, 2%	12	8	5	120
Whole	12	8	8	160
Sweets, desserts, and	15	varies	varies	varies
other carbohydrates				
Nonstarchy vegetables	5	2		25
Meats and Meat Substitutes				
Lean		7	0-3	45
Medium-fat		7	4-7	75
High-fat		7	8+	100
Plant-based proteins	varies	7	varies	varies
Fats			5	45
Alcohol	5	2		100

Choose Your Foods: Exchange Lists for Diabetes. © 2008 American Dietetic Association. Reprinted with permission.

Broade	Amount	Starchy Vagatablas	Amount
Diedus Pagal Jarga (about 4 ag)	Allount	Defried heaveh(, canned	
Bagel, large (about 4 02)	1/4(1 0Z)	Casaawa	$\frac{1}{2}$ cup
Biscuit ^a , 272 inches across	1	Cassava	1/3 cup
Breau	2 alter (11/ar)	Lorin on cob, large	$\frac{1}{2}$ cup
reduced-calorie	Z Slices (1 + 2 OZ)	Hominy ^o , canned	1/2 (5 0Z)
white, whole-grain, pumpernickel,	1 slice (1 oz)	Mixed vegetables with corn,	% cup
rye, unfrosted raisin	1	peas, and pasta ^b	1 cup
Chapatti, small, 6 inches across		Parsnips	17
Cornbread ^a , 1 ³ / ₄ -inch cube	$1(1\frac{1}{2}0Z)$	Peas, green ^b	¹ / ₂ cup
English mutfin	1/2	Plantain, ripe	¹ / ₂ cup
Hot dog bun or hamburger bun	1/2 (1 oz)	Potato	1/3 cup
Naan, 8 inches by 2 inches	1/4	baked with skin	
Pancake, 4 inches across, ¹ / ₄ -inch thick	1	boiled, all kinds	1/4 large (3 oz)
Pita, 6 inches across	1/2	mashed, with milk and fata	$\frac{1}{2}$ cup or $1/2$ medium (3 oz)
Roll, small, plain	1 (1 oz)	French fried (oven baked)	¹ / ₂ cup
Stuffing, bread ^a	1/3 cup	Pumpkin ^b , canned, no sugar	1 cup (2 oz)
Taco shell ^a , 5 inches across	2	added	1 cup
Tortilla, corn, 6 inches across	1	Spaghetti/pasta sauce	
Tortilla, flour, 6 inches across	1	Squash, winter ^b (acorn,	½ cup
Tortilla, flour, 10 inches across	1/3 tortilla	butternut)	1 cup
Waffle ^a , 4-inch square or 4 inches	1	Succotash ^b	½ cup
across		Yam, sweet potato, plain	½ cup
Cereal and Grains	Amount	Crackers and Snacks	Amount
Barley, cooked	1/3 cup	Animal crackers	8
Bran, dry		Crackers	
oat ^b	¼ cup	round butter-type ^a	6
wheat ^b	½ cup	saltine-type	6
Bulgur (cooked)	½ cup	sandwich-style ^a , cheese or	3
Cereals		peanut butter filling	
bran ^b	½ cup	whole-wheat ^a , regular	2-5 (¾ oz)
cooked (oats, oatmeal)	½ cup	whole-wheat ^a , lower fat or	2-5 (¾ oz)
puffed	1½ cups	crispbreads	
shredded wheat, plain	½ cup	Graham crackers, 2½-inch	3
sugar-coated	½ cup	square	³ ⁄4 0Z
unsweetened, ready-to-eat	³ ⁄4 cup	Matzoh	4
Couscous, cooked	1/3 cup	Melba toast, 2- by 4-inch piece	20
Granola		Oyster crackers	3 cups
low-fat	¼ cup	Popcorn	3 cups
regular ^a	¼ cup	with butter ^{a,b}	3 cups
Grits, cooked	½ cup	no fat added ^b	3 cups
Kasha	½ cup	lower fat ^b	³ / ₄ OZ
Millet, cooked	1/3 cup	Pretzels	2
Muesli	¼ cup	Rice cakes, 4 inches across	
Pasta, cooked	1/3 cup	Snack chips	
Polenta, cooked	1/3 cup	fat-free or baked (tortilla,	15-20 (¾ oz)
Quinoa, cooked	1/3 cup	potato), baked pita chips	
Rice, white or brown, cooked	1/3 cup	regular ^a (tortilla, potato)	9-13 (¾ oz)
Tabbouleh (tabouli), prepared	½ cup		
Wheat germ, dry	3 tbsp		
Wild rice, cooked	½ cup		
Beans, Peas, and Lentils	Amount		
Baked beans ^b	1/3 cup	^a Extra fat, or prepared with added fat (C	ount as 1 starch + 1 fat)
Beans ^ь , cooked (black, garbanzo,	½ cup	^b More than 3 g of dietary fiber per servit	ng
kidney, lima, navy, pinto, white)		c480 mg or more of sodium per serving	
Lentils ^b , cooked (brown, green, yellow)	½ cup		
Peas ^b , cooked (black-eyed, split)	½ cup		

STARCH One starch exchange equals 15 g of carbohydrate, 0-3 g of protein, 0-1 g of fat and 80 kcal.

FRUITS	
--------	--

One fruit exchange equals 15 g of carbohydrate and 60 kcal.

Fruits	Amount	Fruits (Cont.)	Amount
Apple, unpeeled, small	1 (4 oz)	Pears	
Apples, dried	4 rings	canned	½ cup
Applesauce,	½ cup	fresh, large	1/2 (4 oz)
unsweetened		Pineapple	
Apricots		canned	½ cup
canned	½ cup	fresh	³ ⁄ ₄ cup
dried	8 halves	Plums	
fresh ^a	4 whole (5½ oz)	canned	½ cup
Banana, extra small	1 (4 oz)	dried (prunes)	3
Blackberries ^a	³ ⁄4 cup	small	2 (5 oz)
Blueberries	³ ⁄4 cup	Raspberries ^a	1 cup
Cantaloupe, small	1/3 melon or 1 cup cubed (11 oz)	Strawberries ^a	1¼ cup whole berries
Cherries		Tangerines, small ^a	2 (8 oz)
sweet, canned	½ cup	Watermelon	1 slice or 1¼ cups cubes (13½ oz)
sweet, fresh	12 (3 oz)		
Dates	3	Fruit Juices	Amount
Dried fruits	2 tbsp	Apple juice/cider	½ cup
(blueberries, cherries,		Fruit juice blends, 100%	
cranberries, mixed		juice	1/3 cup
fruit, raisins)		Grape juice	1/3 cup
Figs		Grapefruit juice	½ cup
dried	1½	Orange juice	½ cup
fresh ^a	1½ large or 2 medium (3½ oz)	Pineapple juice	½ cup
Fruit cocktail	½ cup	Prune juice	1/3 cup
Grapefruit			
large	½ (11 oz)		
sections, canned	³ ⁄ ₄ cup		
Grapes, small	17 (3 oz)		
Honeydew melon	1 slice or 1 cup cubed (10 oz)		
Kiwi ^a	1 (3½ oz)		
Mandarin oranges,			
canned	³ ⁄ ₄ cup		
Mango, small	½ fruit (5½ oz) or ½ cup		
Nectarine, small	1 (5 oz)		
Orange, small ^a	1 (6½ oz)		
Рарауа	1/2 fruit or 1 cup cubed(8 oz)		
Peach			
canned	½ cup		
fresh, medium	½ (6 oz)		
^a More than 3 g of die	tary fiber per serving		

^aMore than 3 g of dietary fiber per serving

One milk exchange equals 12 g of carbohydrate and 8 g of protein.				
Food	Serving Size	Count as		
Fat-free or low-fat (1%)				
Milk, buttermilk, acidophilus milk, Lactaid	1 cup	1 fat-free milk		
Evaporated milk	½ cup	1 fat-free milk		
Yogurt, plain or flavored with an artificial sweetener	2/3 cup (6 oz)	1 fat-free milk		
Reduced-fat (2%)				
Milk, acidophilus milk, kefir, Lactaid	1 cup	1 reduced-fat milk		
Yogurt, plain	2/3 cup (6 oz)	1 reduced-fat milk		
Whole				
Milk, buttermilk, goat's milk	1 cup	1 whole milk		
Evaporated milk	¹ / ₂ cup	1 whole milk		
Yogurt, plain	8 oz	1 whole milk		

MILK One milk exchange equals 12 g of carbohydrate and 8 g of protein

DAIRY-LIKE FOODS

One milk exchange equals 12 g of carbohydrate and 8 g of protein.				
Food	Serving Size	Count as		
Chocolate milk				
fat-free	1 cup	1 fat-free milk + 1 carbohydrate		
whole	1 cup	1 whole milk + 1 carbohydrate		
Eggnog, whole milk	½ cup	1 carbohydrate + 2 fats		
Rice drink				
flavored, low-fat	1 cup	2 carbohydrates		
plain, fat-free	1 cup	1 carbohydrate		
Smoothies, flavored, regular	10 oz	1 fat-free milk + 2½ carbohydrates		
Soy milk				
light	1 cup	1 carbohydrate + ½ fat		
regular, plain	1 cup	1 carbohydrate + 1 fat		
Yogurt				
and juice blends	1 cup	1 fat-free milk + 1 carbohydrate		
low carbohydrate (less than 6 g of carbohydrate per choice)	2/3 cup (6 oz)	½ fat-free milk		
with fruit, low-fat	2/3 cup (6 oz)	1 fat-free milk + 1 carbohydrate		

SWEETS, DESSERTS, AND OTHER CARBOHYDRATES

One carbohydrate exchange equals 15 g of carbohydrate and variable amounts of protein, fat, and energy.

Food	Serving Size	Count as
Reverages Soda and Energy/Sports Drinks		Jount us
Granhanny iviae coalitail	1/	1 composition
	$\frac{4}{2}$ cup	1 carbonyurate
	$1 \operatorname{can}(8.3 \operatorname{0Z})$	2 carbonyurates
Fruit drink or lemonade	1 cup (8 oz)	2 carbonydrates
Hot chocolate		
regular	1 envelope added to 8 oz water	1 carbohydrate + 1 fat
sugar-free or light	1 envelope added to 8 oz water	1 carbohydrate
Soft drink (soda), regular	1 can (12 oz)	2½ carbohydrates
Sports drink	1 cup (8 oz)	1 carbohydrate
Brownies, Cake, Cookies, Gelatin, Pie, and Pudding		
Brownie, small, unfrosted	1¼-inch square, 7/8-inch high	1 carbohydrate + 1 fat
	(about 1 oz)	
Cake		
angel food, unfrosted	1/12 of a cake (about 2 oz)	2 carbohydrates
frosted	2-inch square (about 2 oz)	2 carbohydrates + 1 fat
unfrosted	2-inch square (about 1 oz)	1 carbohydrate + 1 fat
Cookies		
chocolate chip	2 (2¼ inches across)	1 carbohydrate + 2 fats
gingersnap	3	1 carbohydrate
sandwich with crème filling	2 small (about 2/3 oz)	1 carbohydrate + 1 fat
sugar-free	3 small or 1 large (3/4 - 1 oz)	1 carbohydrate + 1-2 fat
vanilla wafer	5	1 carbohydrate + 1 fat
Cuncake frosted	1 small (about 1^{3} / oz)	2 carbohydrates $+ 1-1\%$
Superice, nosteu	1 Shian (abbat 1 /4 02)	fats
Fruit cobbler	$\frac{1}{2}$ (21/2 07)	3 carbobydrates + 1 fat
Colatin regular	¹ / ₂ cup (5 / 2 0 2)	1 carbohydrato
Dio	72 Cup	1 carbonyurate
rie	$1/6$ of θ inch his	2 composition + 2 fata
commercially prepared fruit, 2 crusts	1/0 of 0 in sh mis	3 carboliyurates + 2 rats
pumpkin or custaru	1/8 of 8-men pie	1 ⁴ ₂ carbonyurates + 1 ⁴ ₂
		lats
Pudding	17	
regular (made with reduced-fat milk)	½ cup	2 carbohydrates
sugar-free or sugar and fat-free (made with fat-free	½ cup	1 carbohydrate
milk)		
Candy, Spread, Sweets, Sweeteners, Syrups, and		
Toppings		
Candy bar, chocolate/peanut	2 "fun-size" bars (1 oz)	$1\frac{1}{2}$ carbohydrates + $1\frac{1}{2}$
	2	fats
Candy, hard	3 pieces	1 carbohydrate
Chocolate "kisses"	5	1 carbohydrate + 1 fat
Coffee creamer		
dry, flavored	4 tsp	½ carbohydrate + ½ fat
liquid, flavored	2 tbsp	1 carbohydrate
Fruit snacks, chewy (pureed fruit concentrate)	1 roll (3/4 oz)	1 carbohydrate
Fruit spreads, 100% fruit	1½ tbsp	1 carbohydrate
Honey	1 tbsp	1 carbohydrate
Jam or jelly, regular	1 tbsp	1 carbohydrate
Sugar	1 tbsp	1 carbohydrate
Syrup	*	-
chocolate	2 tbsp	2 carbohydrates
light (pancake-type)	2 tbsp	1 carbohydrate
regular (pancake-type)	1 thsp	1 carbohydrate
regular (palloune type)		2 541 5511 , 41 410

.

SWEETS, DESSERTS, AND OTHER CARBOHYDRATES LIST (Cont.)

One carbohydrate exchange equals 15 g of carbohydrate and variable amounts of protein, fat, and energy.

Food	Serving Size	Count as
Condiments and Sauces		
Barbeque sauce	3 tbsp	1 carbohydrate
Cranberry sauce, jellied	¼ cup	1½ carbohydrates
Gravy ^a , canned or bottled	½ cup	½ carbohydrate + ½ fat
Salad dressing, fat-free, low-fat, cream-based	3 tbsp	1 carbohydrate
Sweet and sour sauce	3 tbsp	1 carbohydrate
Doughnuts, Muffins, Pastries, and Sweet Breads		
Banana nut bread	1-inch slice (1 oz)	2 carbohydrates + 1 fat
Doughnut		-
cake, plain	1 medium (1½ oz)	1½ carbohydrates + 2 fats
yeast-type, glazed	3 ³ / ₄ inches across (2 oz)	2 carbohydrates + 2 fats
Muffin (4 oz)	1/4 muffin (1 oz)	1 carbohydrate + ½ fat
Sweet roll or Danish pastry	1 (2½ oz)	2½ carbohydrates + 2 fats
Frozen Bars, Frozen Desserts, Frozen Yogurt,		
and Ice Cream		
Frozen pops	1	½ carbohydrate
Fruit juice bars, frozen, 100% juice	1 (3 oz)	1 carbohydrate
Ice cream		5
fat-free	¹ ∕₂ cup	1½ carbohydrate
light	¹ / ₂ cup	1 carbohydrate + 1 fat
no sugar added	¹ / ₂ cup	1 carbohydrate + 1 fat
regular	¹ / ₂ cup	1 carbohydrate + 2 fats
Sherbet, sorbet	¹ / ₂ cup	2 carbohydrates
Yogurt, frozen		5
fat-free	1/3 cup	1 carbohydrate
regular	¹ / ₂ cup	1 carbohydrate + 0-1 fats
Granola Bars. Meal Replacement Bars/Shakes.		
and Trail Mix		
Granola or snack bar, regular or low-fat	1 (1 oz)	1½ carbohydrates
Meal replacement bar	1(11/3 oz)	$1\frac{1}{2}$ carbohydrates + 0-1 fat
Meal replacement bar	1 (2 oz)	2 carbohydrates + 1 fat
Meal replacement shake, reduced-energy	1(10-11 oz)	$1\frac{1}{2}$ carbohydrates + 0-1 fat
Trail mix	(,	
candy/nut-based	1 oz	1 carbohvdrate + 2 fats
dried fruit-based	1 oz	1 carbohydrate + 1 fat

^a480 mg or more of sodium per serving

NONSTARCHY VEGETABLES

One vegetable exchange equals 5 g of carbohydrate, 2 g of protein, 0 g of fat, and 25 kcal. Each exchange is a ½-cup portion of cooked vegetables or a 1-cup portion of raw vegetables.

F F F F F F F F F F F F F F F F F F F	- 8
Amaranth or Chinese spinach	Kohlrabi
Artichoke	Leeks
Artichoke hearts	Mixed vegetables (without corn, peas, or pasta)
Asparagus	Mung bean sprouts
Baby corn	Mushrooms, all kinds, fresh
Bamboo shoots	Okra
Beans (green, wax, Italian)	Onions
Bean sprouts	Oriental radish or daikon
Beets	Pea pods
Borscht ^a	Peppers ^b (all varieties)
Broccoli	Radishes
Brussels sprouts ^b	Rutabaga
Cabbage (green, bok choy, Chinese)	Sauerkraut ^a
Carrots ^b	Soybean sprouts
Cauliflower	Spinach
Celery	Squash (summer, crookneck, zucchini)
Chayote ^b	Sugar snap peas
Coleslaw, packaged no dressing	Swiss chard ^b
Cucumber	Tomato
Eggplant	Tomatoes, canned
Gourds (bitter, bottle, luffa, bitter melon)	Tomato sauce ^a
Green onions or scallions	Tomato/vegetable juice ^a
Greens (collard, kale, mustard, turnip)	Turnips
Hearts of palm	Water chestnuts
Jicama	Yard-long beans
Note: Salad greens (like chicory endive escarole lettuc	e arugula radicchio and watercress) are on the Free Foods list

Note: Salad greens (like chicory, endive, escarole, lettuce, arugula, radicchio, and watercress) are on the Free Foods list. ^a480 mg or more of sodium per serving

^bMore than 3 g of dietary fiber per serving

MEATS AND MEAT SUBSTITUTES

One exchange equals 7 g of protein.			
Lean Meats and Meat Substitutes	Amount	Lean Meats and Meat Substitutes (Cont.)	Amount
(0-3 g of fat, 45 kcal per exchange)		Organ meats: heart, kidney, liver	1 oz
		Note: May be high in cholesterol	
Beef: select or choice grades trimmed of fat:	1 oz	Oysters, fresh or frozen	6 medium
ground round, roast (chuck, rib, rump), round,		Pork, lean	
sirloin, steak (cubed, flank, porterhouse, T-bone),		Canadian bacon ^a	1 oz
tenderloin		rib or loin chop/roast, ham, tenderloin	1 oz
Beef jerky ^a	½ 0Z	Poultry without skin: Cornish hen, chicken,	1 oz
Cheeses with 3 g of fat or less per ounce	1 oz	domestic duck or goose (well-drained of	
Cottage cheese	¼ cup	fat), turkey	
Egg substitutes, plain	¼ cup	Processed sandwich meats with 3 g of	1 oz
Egg whites	2	fat or less per ounce: chipped beef,	
Fish, fresh or frozen, plain: catfish, cod, flounder,	1 oz	deli thin-sliced meats, turkey ham, turkey	
haddock, halibut, orange roughy, salmon, tilapia,		kielbasa, turkey pastrami	
trout, tuna		Salmon, canned	1 oz
Fish, smoked ^a : herring or salmon (lox)	1 oz	Sardines, canned	2 small
Game: buffalo, ostrich, rabbit, venison	1 oz	Sausage ^a with 3 g of fat or less per ounce	1 oz
Hot dog ^a with 3 g of fat or less per ounce	1	Shellfish: clams, crab, imitation shellfish,	1 oz
(Eight hot dogs per 14-oz package)		lobster, scallops, shrimp	
Note: may be high in carbohydrate		Tuna, canned in water or oil, drained	1 oz
Lamb: chop, leg, or roast	1 oz	Veal, loin chop, roast	1 oz

^a480 mg or more of sodium per serving (based on the sodium content of a typical 3-oz serving of meat, unless 1 or 2 oz is the normal serving size)

Dietary Management with the Exchange System			
Medium-Fat Meats and Meat Substitutes	Amount	High-Fat Meats and Meat Substitutes	Amount
(4-7 g of fat, 75 kcal per exchange)		(8+ g of fat, 100 kcal per exchange)	
Beef: corned beef, ground beef, meatloaf, prime grades trimmed of fat (prime rib), short ribs, tongue	1 oz	Bacon pork ^a (16 slices per lb, each slice is 1 oz before cooking)	2 slices
Cheeses with 4-7 g of fat per ounce: feta,	1 oz	turkey ^a (½ oz each before cooking)	3 slices
mozzarella, pasteurized processed cheese spread, reduced-fat cheeses, string		Cheese, regular: American, bleu, brie, cheddar, hard goat, Monterey jack,	1 oz
Egg	1	queso, and Swiss	
Note: High in cholesterol, so limit to three per week		Hot dog ^{a,b} : beef, pork, or combination (10 per 1-lb package)	1
Fish: any fried product	1 oz	Hot dog ^a : turkey or chicken	1
Lamb: ground, rib roast	1 oz	(10 per 1-lb package)	
Pork: cutlet, shoulder roast	1 oz	Pork: ground, sausage, spareribs	1 oz
Poultry: chicken with skin, dove, pheasant, wild duck or goose, fried chicken, ground turkey	1 oz	Processed sandwich meats with 8 g of fat or more per ounce: bologna, pastrami, hard salami	1 oz
Ricotta cheese	2 oz or ¼ cup	Sausage ^a with 8 g of fat or more per	1 oz
Sausage ^a with 4-7 g of fat per ounce	1 oz	ounce: bratwurst, chorizo, Italian,	
Veal, cutlet (no breading)	1 oz	knockwurst, Polish, smoked, summer	

^a480 mg or more of sodium per serving (based on the sodium content of a typical 3-oz serving of meat, unless 1 or 2 oz is the normal serving size) ^bExtra fat, or prepared with added fat. (Add an additional fat choice to this food.)

One exchange equals 7 g of protein.

-

PLANT-BASED PROTEINS

One exchange equals 7 g of protein.		
Food	Serving Size	Count as
"Bacon" strips, soy-based	3	1 medium-fat meat
Baked beans ^a	1/3 cup	1 starch + 1 lean meat
Beans ^a , cooked: black, garbanzo, kidney, lima, navy, pinto, white	½ cup	1 starch + 1 lean meat
"Beef" or "sausage" crumbles, soy-based ^a	2 oz	½ carbohydrate + 1 lean meat
"Chicken" nuggets, soy-based	2 (1½ oz)	½ carbohydrate + 1 medium-fat meat
Edamame ^a	½ cup	½ carbohydrate + 1 lean meat
Falafel (spiced chickpea and wheat patties)	3 (about 2 inches across)	1 carbohydrate + 1 high-fat meat
Hot dog, soy-based	1 (1½ oz)	½ carbohydrate + 1 lean meat
Hummus ^a	1/3 cup	1 carbohydrate + 1 high-fat meat
Lentils, brown, green, or yellow ^a	½ cup	1 carbohydrate + 1 lean meat
Meatless burger, soy-based ^a	3 oz	½ carbohydrate + 2 lean meats
Meatless burger, vegetable- and starch-based ^a	1 (about 2½ oz)	1 carbohydrate + 2 lean meats
Nut spreads: almond butter, cashew butter, peanut	1 tbsp	1 high-fat meat
Dutter, soy hut butter	1/	1 starsh + 1 loan most
Peas", cooked: black-eyed and spirt peas	⁺ ∕2 Cup	1 starch + 1 lean meat
"Course as" wetting and hered	$\frac{1}{2}$ cup	1 starch + 1 lean meat
Sausage patties, soy-based	$1(1\frac{1}{2}0Z)$	1 medium-fat meat
Soy nuts, unsalted	³ ⁄ ₄ 0Z	½ carbohydrate + 1 medium-fat meat
Tempeh	¼ cup	1 medium-fat meat
Tofu	4 oz (½ cup)	1 medium-fat meat
Tofu, light	4 oz (½ cup)	1 lean meat

^aMore than 3 g of dietary fiber per serving

^b480 mg or more of sodium per serving

One fat exchange equals 5 g of fat and 45 kcal.

Unsaturated/Monounsaturated Fats	Amount	Polyunsaturated Fats (Cont.)	Amount
		Salad dressing	
Avocado, medium	2 tbsp (1 oz)	reduced-fat ^a	2 tbsp
Nut butters (trans fat-free): almond butter,	1½ tsp	Note: May be high in carbohydrate	
cashew butter, peanut butter (smooth or		regularª	1 tbsp
crunchy)		Seeds	
Nuts		flaxseed, whole	1 tbsp
almonds	6	pumpkin, sunflower	1 tbsp
Brazil	2	sesame seeds	1 tbsp
cashews	6	Tahini or sesame paste	2 tsp
filberts (hazelnuts)	5	Saturated Fats	Amount
macadamia	3	Bacon, cooked, regular or turkey	1 slice
mixed (50% peanuts)	6	Butter	
peanuts	10	reduced-fat	1 tbsp
pecans	4 halves	stick	1 tsp
pistachios	16	whipped	2 tsp
Oil: canola, olive, peanut	1 tsp	Butter blends made with oil	
Olives		reduced-fat or light	1 tbsp
black (ripe)	8 large	regular	1½ tsp
green, stuffed	10 large	Chitterlings, boiled	2 tbsp (½ oz)
Polyunsaturated Fats	Amount	Coconut, sweetened, shredded	2 tbsp
Margarine: lower-fat spread (30%-50%	1 tbsp	Coconut milk	_
vegetable oil, trans fat-free)		light	1/3 cup
Margarine: stick, tub (trans fat-free), or	1 tsp	regular	1½ tbsp
squeeze (trans fat-free)		Cream	
Mayonnaise		half and half	2 tbsp
reduced-fat	1 tbsp	heavy	1 tbsp
regular	1 tsp	light	1½ tbsp
Mayonnaise-style salad dressing		whipped	2 tbsp
reduced-fat	1 tbsp	whipped, pasteurized	¼ cup
regular	2 tsp	Cream cheese	
Nuts		reduced-fat	1½ tbsp (¾ oz)
pignolia (pine nuts)	1 tbsp	regular	1 tbsp (½ oz)
walnuts, English	4 halves	Lard	1 tsp
Oil: corn, cottonseed, flaxseed, grape seed,	1 tsp	Oil: coconut, palm, palm kernel	1 tsp
safflower, soybean, sunflower		Salt pork	¹ ⁄4 0Z
Oil: made from soybean and canola oil	1 tsp	Shortening, solid	1 tsp
(eg, Enova)		Sour cream	
Plant stanol esters		reduced-fat or light	3 tbsp
light	1 tbsp	regular	2 tsp
regular	2 tsp		

FATS

^a480 mg or more of sodium per serving

FREE FOODS

A *free food* is any food or drink that contains less than 20 kcal or no more than 5 g of carbohydrate per serving. Foods with serving sizes should be limited to three servings per day. Be sure to spread them out throughout the day. If you eat all three servings at one time, it could raise your blood glucose level. Foods listed without a serving size can be eaten whenever you like.

Low-Carbohydrate Foods	Serving Size	Condiments	Serving Size
Cabbage, raw	½ cup	Barbecue sauce	2 tsp
Candy, hard (regular or sugar-free)	1 piece	Catsup (ketchup)	1 tbsp
Carrots, cauliflower, or green beans, cooked	¼ cup	Honey mustard	1 tbsp
Cranberries, sweetened with sugar	½ cup	Horseradish	
substitute		Lemon juice	
Cucumber, sliced	½ cup	Miso	1½ tsp
Gelatin		Mustard	
dessert, sugar-free		Parmesan cheese, freshly grated	1 tbsp
unflavored		Pickle relish	1 tbsp
Gum		Pickles	
Jam or jelly, light or no sugar added	2 tsp	dill ^a	1 ½ medium
Rhubarb, sweetened with sugar substitute	½ cup	sweet, bread and butter	2 slices
Salad greens		sweet, gherkin	³ ⁄4 oz
Sugar substitutes (artificial sweeteners)		Salsa	¼ cup
Syrup, sugar-free	2 tbsp	Soy sauce ^a , light or regular	1 tbsp
Modified-Fat Foods with Carbohydrate	Serving Size	Sweet chili sauce	2 tsp
Cream cheese, fat-free	1 tbsp	Taco sauce	1 tbsp
Creamers		Vinegar	
nondairy, liquid	1 tbsp	Yogurt, any type	2 tbsp
nondairy, powdered	2 tsp		
Margarine spread			
fat-free	1 tbsp	Free Snacks	
reduced-fat	1 tsp	These foods in these serving sizes a	are perfect
Mayonnaise		free-food snacks.	
fat-free	1 tbsp	5 baby carrots and celery sticks	
reduced-fat	1 tsp	¹ / ₄ cup blueberries	
Mayonnaise-style salad dressing	1.1	¹ / ₂ oz sliced cheese, fat-free	
rat-iree	1 tosp	10 goldfish-style crackers	
reduced-fat	1 tsp	2 saltine-type crackers	
Salad dressing	1 th an	1 frozen cream pop, sugar-free	
fat free of low-lat	1 tDSp 2 than	² / ₂ OZ lean meat	
rat-iree, italian	2 tDSp	1 cup light popcorn	
Sour cream, lat-free or reduced-lat	1 tosp	i vanna water	
light or fat free	2 th an	Drinks /Mirros	
ngulor	2 tDSp 1 then	Diffices Food on this list without a comving	iza can ha
regulai	1 tosp	consumed in moderate amounts	Size call be
Seasonings		Bouillon, broth, consommé ^a	
Foods on this list can be consumed in moderate		Bouillon or broth, low-sodium	
amounts.		Carbonated or mineral water	
Flavoring extracts (for example, vanilla, almond,		Club soda	
peppermint)		Cocoa powder, unsweetened (1 tbsp)	
Garlic		Coffee, unsweetened or with sugar substitute	
Herbs, fresh or dried		Diet soft drinks, sugar-free	
Nonstick cooking spray		Drink mixes, sugar-free	
Pimento		Tea, unsweetened or with sugar substitute	
Spices		Tonic water, diet	
Hot pepper sauce		Water	
Wine, used in cooking		Water, flavored, carbohydrate-free	
Worcestershire sauce			

^a480 mg or more of sodium per serving

Entrées	Serving Size	Count as
Casserole type ^a (tuna noodle, lasagna, spaghetti with meatballs, chili with beans, macaroni and cheese)	1 cup (8 oz)	2 carbohydrates + 2 medium-fat meats
Stews ^a (beef/other meats and vegetables)	1 cup (8 oz)	1 carbohydrate + 1 medium-fat meat + 0-3 fats
Tuna salad or chicken salad	½ cup (3½ oz)	¹ / ₂ carbohydrate + 2 lean meats + 1 fat
Frozen Meals/Entrées	Serving Size	Count as
Burrito ^{a,b} (beef and bean)	1 (5 oz)	3 carbohydrates + 1 lean meat + 2 fats
Dinner-type meal ^a	generally 14-17 oz	3 carbohydrates + 3 medium-fat meats + 3 fats
Entrée or meal with less than 340 kcal ^a Pizza	about 8-11 oz	2-3 carbohydrates + 1-2 lean meats
cheese/vegetarian, thin crust	1/4 of a 12-inch (4½-5 oz)	2 carbohydrates + 2 medium-fat meats
meat topping, thin crust	1/4 of a 12-inch (5 oz)	2 ¹ / ₂ carbohydrates + 1 medium-fat meat + 3 fats
Pocket sandwich	1 (4½ oz)	2 carbohydrates + 2 medium-fat meats + 1½ fats
Pot pie ^a	1 (7 oz)	3 carbohydrates + 1 lean meat + 1- 2 fats
Salads (Deli-Style)	Serving Size	Count as
Coleslaw	½ cup	1 carbohydrate + 1½ fats
Macaroni/pasta salad	¹ / ₂ cup	2 carbohydrates + 3 fats
Potato salad ^a	½ cup	1 ¹ / ₂ carbohydrates + 1-2 fats
Soups	Serving Size	Count as
Bean, lentil, or split pea ^a	1 cup	1 carbohydrate + 1 lean meat
Chowder ^a (made with milk)	1 cup (8 oz)	1 carbohydrate + 1 lean meat + 1½ fats
Cream ^a (made with water)	1 cup (8 oz)	1 carbohydrate + 1 fat
Instant ^a	6 oz prepared	1 carbohydrate
with beans or lentils ^a	8 oz prepared	2½ carbohydrate + 1 lean meat
Miso soup ^a	1 cup	½ carbohydrate + 1 fat
Oriental noodle ^a	1 cup	2 carbohydrates + 2 fats
Rice (congee)	1 cup	1 carbohydrate
Tomato ^a (made with water)	1 cup (8 oz)	1 carbohydrate
Vegetable beef, chicken noodle, or other broth- type ^a	1 cup (8 oz)	1 carbohydrate

COMBINATION FOODS

^a600 mg or more of sodium per serving (for combination food main dishes/meals)

^bMore than 3 g of dietary fiber per serving
Breakfast Sandwiches Serving Size Count as Egg, cheese, meat, English muffin^a 2 carbohydrates + 2 medium-fat meats 1 Sausage biscuit sandwich^a 1 2 carbohydrates + 2 high-fat meats + 3½ fats Main Dishes/Entrées Serving Size Count as Burrito^{a,b} (beef and beans) 1 (8 oz) 3 carbohydrates + 3 medium-fat meats + 3 fats Chicken breast, breaded and fried^a 1 carbohydrate + 4 medium-fat meats 1 (5 oz) Chicken drumstick, breaded and fried 1 (2 oz) 2 medium-fat meats Chicken nuggets^a 6 (3½ oz) 1 carbohydrate + 2 medium-fat meats + 1 fat Chicken thigh, breaded and fried^a ¹/₂ carbohydrate + 3 medium-fat meats 1 (4 oz) + 1½ fats Chicken wings, hota 6 (5 oz) 5 medium-fat meats + $1\frac{1}{2}$ fats **Oriental Foods** Serving Size Count as Beef, chicken, or shrimp with vegetables in 1 cup (about 5 oz) 1 carbohydrate + 1 lean meat + 1 fat sauce^a Egg roll, meat^a 1 (about 3 oz) 1 carbohydrate + 1 lean meat + 1 fat Fried rice, meatless ½ cup 1¹/₂ carbohydrates + 1¹/₂ fats Meat and sweet sauce^a (orange chicken) 3 carbohydrates + 3 medium-fat meats 1 cup + 2 fats Noodles and vegetables in sauce^{a,b} (chow mein, 2 carbohydrates + 1 fat 1 cup lo mein) Pizza Serving Size Count as Pizza 1/8 of a 14-inch 2¹/₂ carbohydrates + 1 medium-fat cheese, pepperoni, regular crust^a (about 4 oz) meat + $1\frac{1}{2}$ fats 1/4 of a 12-inch 2¹/₂ carbohydrates + 2 medium-fat cheese/vegetarian, thin crust^a (about 6 oz) meats + $1\frac{1}{2}$ fats Sandwiches **Serving Size** Count as Chicken, sandwich, grilled^a 1 3 carbohydrates + 4 lean meats Chicken, sandwich, crispy^a 1 3¹/₂ carbohydrates + 3 medium-fat meats + 1 fat 2¹/₂ carbohydrates + 2 medium-fat Fish sandwich with tartar sauce 1 meats + 2 fats Hamburger large with cheese^a 1 $2\frac{1}{2}$ carbohydrates + 4 medium-fat meats + 1 fat regular 1 2 carbohydrates + 1 medium-fat meat + 1 fat Hot dog with bun^a 1 carbohydrate + 1 high-fat meat + 1 fat 1 Submarine sandwich 6-inch sandwich less than 6 g of fat^a 3 carbohydrates + 2 lean meats 6-inch sandwich 3¹/₂ carbohydrates + 2 medium-fat regular meats + $1\frac{1}{2}$ fats Taco, hard or soft shell (meat and cheese) 1 small 1 carbohydrate + 1 medium-fat meat + 1½ fats

FAST FOODS

^a600 mg or more of sodium per serving (for fast-food main dishes/meals)

^bMore than 3 g of dietary fiber per serving

Salads	Serving Size	Count as
Salad, main dish ^{a,b} (grilled chicken–type, no	salad	1 carbohydrate + 4 lean meats
dressing or croutons)		
Salad, side, no dressing or cheese	small (about 5 oz)	1 vegetable
Sides/Appetizers	Serving Size	Count as
French fries, restaurant style ^c	small	3 carbohydrate + 3 fats
	medium	4 carbohydrates + 4 fats
	large	5 carbohydrates + 6 fats
Nachos, with cheese ^a	small (about 4½ oz)	2½ carbohydrates + 4 fats
Onion rings ^a	1 serving (about 3 oz)	2½ carbohydrates + 3 fats
Desserts	Serving Size	Count as
Milkshake, any flavor	12 oz	6 carbohydrates + 2 fats
Soft-serve ice cream, cone	one small	2½ carbohydrates + 1 fat

FAST FOODS (Cont.)

^a600 mg or more of sodium per serving (for fast-food main dishes/meals)

^bMore than 3 g of dietary fiber per serving

^cExtra fat, or prepared with added fat

ALCOHOL

Alcoholic Beverages	Serving Size	Count as
Beer		
light (4.2%)	12 fl oz	1 alcohol equivalent + ½ carbohydrate
regular (4.9%)	12 fl oz	1 alcohol equivalent + 1 carbohydrate
Distilled spirits: vodka, rum, gin, whiskey (80 or 86 proof)	1½ fl oz	1 alcohol equivalent
Liqueur, coffee (53 proof)	1 fl oz	1/2 alcohol equivalent + 1 carbohydrate
Sake	1 fl oz	1⁄2 alcohol equivalent
Wine		
dessert (sherry)	3 ½ fl oz	1 alcohol equivalent + 1 carbohydrate
dry, red, or white (10%)	5 fl oz	1 alcohol equivalent

SAMPLE EXCHANGE PATTERNS FOR CALCULATED DIETS USING EXCHANGE SYSTEM

1,200 kcal							
	СНО	= Starch ·	+ Fruit	+ Milk	Meat	Vegetable	Fat
Breakfast	3	1	1	1	1		1
Snack							
Lunch	3	2	1		3	1	1
Snack							
Supper	3	1	1	1	2	1	1
Snack							
Totals:	9	4	3	2	6	2	3

1,500 kcal

	СНО	= Starch ·	+ Fruit	+ Milk	Meat	Vegetable	Fat
Breakfast	4	2	1	1	1		1
Snack							
Lunch	4	3	1		3	1	1
Snack							
Supper	3	2		1	3	1	2
Snack							
Totals:	11	7	2	2	7	2	4

1,800 kcal

,							
	СНО	= Starch +	- Fruit	+ Milk	Meat	Vegetable	Fat
Breakfast	4	2	1	1	1		2
Snack							
Lunch	5	4	1		3	1	2
Snack							
Supper	5	3	1	1	3	1	2
Snack			1				
Totals:	14	8	4	2	7	2	6

2,000 kcal

	СНО	= Starch	+ Fruit	+ Milk	Meat	Vegetable	Fat
Breakfast	4	2	1	1	1		1
Snack							
Lunch	5	3	1	1	3	1	2
Snack							
Supper	5	3	1	1	3	1	2
Snack	2	1	1		1		1
Totals:	16	9	4	3	8	2	6

2,200 kcal

	СНО	= Starch +	- Fruit	+ Milk	Meat	Vegetable	Fat
Breakfast	5	3	1	1	1		1
Snack							
Lunch	5	3	1	1	3	2	2
Snack	1	1					
Supper	5	3	1	1	3	1	2
Snack	2	1	1		1		1
Totals:	18	11	4	3	8	3	6

SUGAR IN MODERATION DIET

Description

The diet restricts foods high in added sugar and fat. "Added sugar" is defined as that added to sweeten food at the table or added by the manufacturer, and is chiefly sucrose or corn syrup.

Indications

This is a less restricted diet for people trying to lose weight. It is also used in conjunction with the "Dumping Syndrome Diet." (See Section IB: Dumping Syndrome Diet.) This diet is not meant for people with type 1 diabetes or type 2 diabetes.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "Sugar in Moderation Diet."

Planning the Diet

Exclude the following foods.

FOOD GROUP	FOODS EXCLUDED
Beverages and Milk	Regular carbonated beverages; fruit ades; sugar-sweetened soft drinks; sugar- sweetened iced tea; chocolate milk; milkshake; eggnog; sweetened yogurt; cocoa (sweetened)
Breads and Crackers	Sweet rolls or breads; doughnuts
Cereals and Grains	Sugar-coated cereals; granola-type cereals; presweetened cooked cereals
Meat, Fish, Poultry, Cheese, Eggs, Legumes	Glazed meats
Vegetables and Potatoes	Candied or glazed vegetables; sweet pickled vegetables
Fruits and Juices	Sweetened fruits or juices; candied or glazed fruits
Desserts	Cakes, pies, cookies, pastries, sherbets, puddings, gelatin desserts (regularly sweetened), ice cream
Sugar and Sweets	Candy, chewing gum, sugar, honey, jam, jelly, marmalade, syrup, molasses
Miscellaneous	Sweet relishes

CALORIE-CONTROLLED DIET FOR WEIGHT MANAGEMENT

Description

For the Calorie-Controlled Diet for Weight Management, the Regular Diet is modified by reducing energy intake below what is necessary for maintenance of body weight. Intake of essential protein, vitamins, and minerals is maintained by limiting the amount of fat and sugar in the diet and substituting low-energy foods for foods of similar nutrient content that are higher in energy. Weight loss and weight maintenance therapy should be based on a comprehensive weight management program including diet, physical activity, and behavior therapy. The combination therapy is more successful than any one intervention alone (Grade I)* (1).

Indications

Weight reduction is desirable because obesity is related to increased mortality and because weight loss reduces the risk factors for several chronic diseases. Thus, weight loss may help to both control diseases worsened by obesity and decrease the likelihood of developing these diseases. Strong and consistent clinical evidence supports weight loss in overweight or obese persons who have hypertension, hyperlipidemia, or type 2 diabetes, as well as in overweight or obese persons who are at risk for developing these conditions (Grade I) (1-6). Obesity is a significant risk factor for non-alcoholic fatty liver disease, an emerging condition that is the most common cause of abnormal liver function tests in obese children and adults (7). Non-alcoholic fatty liver disease can lead to significant liver damage including cryptogenic cirrhosis, steatohepatitis, and hepatocellular carcinoma (7). In overweight and obese persons, weight loss is recommended to (1-7):

- lower blood pressure in patients with hypertension (5)
- lower total cholesterol, low-density lipoprotein cholesterol, and triglycerides levels in patients with hyperlipidemia (4)
- lower blood glucose levels in patients with type 2 diabetes (3,4,6)
- prevent liver disease (7)

Fat is lost when the body is in a state of negative energy balance, which is achieved by reduced energy intake, increased energy output (through muscle work), or both. The reduction of total energy intake vs. the macronutrient composition of the diet is the most important component for achieving negative energy balance and subsequent weight loss (8).

Body mass index (BMI), defined as weight (kg) divided by height² (m²), and waist circumference should be used to classify overweight and obesity, estimate risk for disease, and identify treatment options (Grade II) (1-3). The BMI is highly correlated to obesity, fat mass, and risk of other diseases (Grade II) (1-3). For optimal health, a BMI of 18.5 to 24.9 kg/m² is recommended for adults, based on evidence that this range is associated with minimal risk of disease. Table C-7 outlines the health risk classes associated with different BMI levels and waist circumferences in adults aged 18 years and older.

			Health Risk Relative t	o Waist Circumference
Weight	BMI (kg/m²)	Risk Class	Men ≤102 cm (≤40 inches) Women ≤88 cm (≤35 inches)	Men >102 cm (>40 inches) Women >88 cm (>35 inches)
Underweight	<18.5	-	_	_
Normal	18.5-24.9	0	_	Increased
Overweight	25.0-29.9	0	Increased	High
Obesity	30.0-34.9	Ι	High	Very high
	35.0-39.9	II	Very high	Very high
Extreme	<u>≥</u> 40.0	III	Extremely high	Extremely high

Table C-7: Weight Classification by BMI, Waist Circumference, and Associated Health Risks

Source: National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Available at: http://www.nhlbi.nih.gov/nhbli/htm. Accessed January 21, 2004.

Children and adolescents: Complications of obesity in children and adolescents include hypertension, dyslipidemia, orthopedic disorders, sleep disorders, gall bladder disease, and insulin resistance (9). The National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel recommends evaluations and possible treatment for: (1) children and adolescents who have a BMI greater than or equal to the 85th

percentile and complications of obesity; and (2) children and adolescents who have or a BMI greater than the 95th percentile with or without complications of obesity (9). The use of weight maintenance vs weight loss to achieve weight goals depends on the patient's age, baseline BMI percentile, and the presence of medical complications (9). The committee recommends treatment that begins early, involves family, and institutes permanent changes in a stepwise manner (9). (Refer to Section III: Obesity and Weight Management.)

Contraindications

Weight reduction is not recommended for the following groups:

- pregnant women (Energy restriction during pregnancy that is sufficient to produce weight loss can be dangerous for the development of the fetus.) (10)
- patients with unstable mental or medical conditions, unless medically supervised (2)
- patients with anorexia nervosa or a history of this disorder, unless medically supervised (2)
- terminally ill patients (2)

See Section III: Clinical Nutrition Management for additional information on:

- Heart Failure
- Gastroesophageal Reflux Disease (Gerd)
- Hypertension
- Hypertriglyceridemia
- Hypoglycemia
- Obesity And Weight Management

Nutritional Adequacy

The precise level at which energy intake is insufficient for an adequate diet is difficult to define without taking into consideration the age and sex of the individual and the corresponding Dietary Reference Intakes. Diets that provide 1,200 kcal or less of energy are generally inadequate to meet Dietary Reference Intakes. Therefore, a daily multivitamin is recommended when energy levels of this range are prescribed (2,3). Determining the energy level that promotes weight loss is difficult in overweight and obese individuals, and estimated energy needs should be based on resting metabolic rate (RMR) (Grade I) (1). Whenever possible, RMR should be measured (eg, indirect calorimetry). If RMR cannot be measured, the Mifflin-St. Jeor equation using actual weight is the most accurate method for estimating RMR for overweight and obese individuals (Grade I) (1). (Refer to Section II: Estimating Energy Expenditures.) Meta-analysis of the literature has identified that 1,200 kcal/day for women and 1,400 to 1,500 kcal/day for men (2,3,8,11) are acceptable energy intake levels that promote gradual and safe weight loss of 0.5 to 1 lb/week (2,3,8). An individualized reduced energy diet along with energy expended through physical activity should reduce body weight at an optimal rate of 1 to 2 lbs/week for the first 6 months and achieve an initial weight loss goal of up to 10% from baseline. These goals are realistic, achievable, and sustainable (Grade I) (1).

Recent studies have evaluated the impact of total energy and macronutrient composition on weight loss. The US Department of Agriculture has found that diets high in carbohydrate (>55%) and low to moderate in fat (15% to 30%) tend to be lower in total energy and higher in diet quality when compared to lowcarbohydrate diets (<30%). In these studies, the BMI was significantly lower for men and women on the high-carbohydrate diet; the highest BMIs were noted for individuals on a low-carbohydrate diet. Based on these findings, weight loss is independent of macronutrient composition, and energy restriction is the key variable associated with short-term weight reduction (8,11). A randomized controlled trial investigated weight loss outcomes using a low-carbohydrate diet compared to a low-fat, low-energy, high-carbohydrate (conventional) diet. Initial weight loss was significantly greater in the low-carbohydrate group; however, after 1 year the difference between the groups was not statistically significant (Grade II) (1,12). The difference in weight loss between the two groups in the first 6 months was attributed to overall greater energy deficit in the low-carbohydrate group (Grade II) (1,12). The low-carbohydrate diet was associated with a greater improvement in some risk factors for coronary heart disease. Adherence was poor and attrition was high in both groups (12). Results of this study should be interpreted with caution, given the relatively small sample size and short duration (12). Longer and larger studies are required to determine the long-term safety and efficacy of low-carbohydrate, high-protein, high-fat diets (12). A low-glycemic index diet is not recommended for weight loss or weight maintenance because it has not been shown to be effective in these areas (Grade I) (1).

Calorie-Controlled Diet for Weight Management

How to Order the Diet

The physician may specify any of the following:

- "Weight Reduction Diet": The dietitian determines an appropriate weight loss goal and energy level.
- "____kcal Diet": The dietitian plans an individualized meal plan within the energy prescription.
- Restriction of sodium, fat, cholesterol or another dietary component: If a restriction is required, it should be prescribed along with the diet order.

Initiation of a weight reduction diet is not usually recommended in a hospital setting. Weight loss and weight maintenance therapy should be based on a comprehensive weight management program including diet, physical activity, and behavior therapy (Grade I) (1). Medical nutrition therapy for weight loss should last at least 6 months or until weight loss goals are achieved, with implementation of a weight maintenance program after that time (Grade I) (1). A greater frequency of contacts between the patient and practitioner may lead to more successful weight loss and weight loss maintenance (Grade I) (1).

Moderate energy restriction for weight loss is recommended (2,3). Individualized meal plans of 1,200 to 1,500 kcal/day for women and 1,400 to 2,000 kcal/day for men can promote retention of lean body mass while facilitating weight reduction when combined with physical activity and behavioral modification (2,3). Reducing dietary fat and/or carbohydrate is a practical way to create an energy deficit of 500 to 1,000 kcal below estimated energy needs and should result in a weight loss goal of 1 to 2 lbs/week (Grade I) (1). Portion control should be included as part of a comprehensive weight management program. Portion control at meals and snacks results in reduced energy intake and weight loss (Grade II) (1). In addition, total energy intake distributed throughout the day, achieved by the consumption of four or five meals/snacks per day (including breakfast), may be preferable and result in greater weight loss than eating in the evening (Grade II) (1). For people who have difficulty with self selection or portion control, meal replacements (eg, liquid meals, meal bars, energy-controlled packaged meals) may be used as part of a comprehensive weight management program (Grade I) (1). Substituting one or two daily meals or snacks with meal replacements is a successful weight loss and weight maintenance strategy (Grade I) (1). When setting goals with patients, the dietitian should establish a realistic and practical target, such as a 5% to 10% decrease in the baseline weight or a decrease of 2 BMI units (Grade I) (1-3).

Successful weight reduction requires a commitment to behavioral change, family support, and attention to physical activity patterns (Grade I) (1-3). Behavioral therapy should use multiple strategies including self-monitoring, stress management, stimulus control, problem solving, contingency management, cognitive restructuring, and social support (Grade I) (1). Moderate physical activity promotes the maintenance of lean body mass, contributes to the energy deficit needed for weight loss, and may help with the maintenance of weight loss (Grade I) (1). Physical activity should be assessed with individualized long-term goals established to accumulate at least 30 minutes of moderate-intensity physical activity on most, and preferably all, days of the week, unless medically contraindicated (Grade I) (1).

Weight loss medications approved by the Food and Drug Administration may be part of a comprehensive weight management program (1). Clinicians should collaborate with other members of the health care team regarding the use of these weight loss medications for people who meet the criteria. A BMI of 30 kg/m^2 or greater with no comorbid conditions or a BMI exceeding 27 kg/m^2 with comorbid conditions should be one criterion for the use of medications to treat obesity (3). Other criteria include: failure to manage weight with more conservative behavioral methods; number and severity of associated comorbidities; absence of contraindications, such as depression or ischemic heart disease; and the need for short-term weight loss to reduce operative risk (2). Depending on the type of medication, a loss of 10% to 15% of the baseline weight has been observed with the adjunct of lifestyle modification (low-energy diet and increased physical activity) (Grade 1) (1,13). Weight regain occurs after drug withdrawal; thus, long-term use is required to maintain the weight loss (13). Data on the use of weight loss medication for longer than 2 years are limited, and the efficacy and safety of long-term treatment with pharmacologic agents remains unclear (1,13). Pharmacologic agents are a useful adjunct to, but not a substitute for, necessary changes in diet and physical activity. The effectiveness of pharmacologic intervention depends on its use with appropriate dietary nutrition intervention, increased physical activity, and lifestyle change (1,2). Refer to Section IB: Nutrition Management of Bariatric Surgery and Section III: Obesity and Weight Management for more information.

Planning the Diet

The dietitian should plan an energy-controlled diet to meet the individual needs and lifestyle of the client. Suggestions to reduce daily energy intake include (1-5,13):

- Reduce intake of foods with high-energy density (eg, alcohol and fat). Follow the US Dietary Guidelines of less than 30% energy from fat, 10% to 20% from protein, and 50% to 60% from carbohydrates.
- Reduce the total amount of food consumed by decreasing portion size and frequency of consumption. Employ behavior modification techniques to improve control over the food selection process and the act of eating.
- Establish self-management training techniques that will enhance the satiety of meals but reduce the energy intake. For example, encourage the patient to eat slowly so that the brain can register that the stomach is full, or recommend eating ample amounts of low-energy density vegetables (eg, salads with small amounts of salad dressing or fat-free dressing) to provide chewing satisfaction and fill the stomach.

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Adult Weight Management Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2006. Available at: http://www.andevidencelibrary.com. Accessed August 1, 2006.
- 2. Position of the American Dietetic Association: weight management. J Am Diet Assoc. 2009;109:330-346.
- 3. National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.* Bethesda, Md: National Institutes of Health; 1998. NIH publication No. 98-4083. Available at: http://www.nhlbi.nih.gov/nhlbi/htm.
- 4. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486-2497. Available at: http:// nhlbi.nih.gov/guidelines/cholesterol/index.htm.
- Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ, and the National High Blood Pressure Education Program Coordinating Committee. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003; 42:1206-1252.
- 6. Diabetes Type 1 and 2 Evidence- Based Nutrition Practice Guideline for Adults. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.andevidencelibrary.com. Accessed December 10, 2008.
- 7. Festi D, Colecchia A, Sacco T, Bondi M, Roda E, Marchesini G. Hepatic steatosis in obese patients: clinical aspects and prognostic significance. *Obes Rev.* 2004;5:27-42.
- 8. Kennedy ET, Bowman SA, Spence JT, Freedman M, King J. Popular diets: correlation to health, nutrition, and obesity. J Am Diet Assoc. 2001;101:411-420.
- 9. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee recommendations. Pediatrics. 1998;102:1-11.
- 10. Subcommittee on Nutrition Status and Weight Gain During Pregnancy, Committee of Nutritional Status During Pregnancy and Lactation, Food and Nutrition Board, Institute of Medicine. *Nutrition During Pregnancy*. Washington, DC: National Academy Press; 1990.
- 11. Kennedy ET, Bowman SA. Assessment of the effect of fat-modified foods on diet quality in adults, 19 to 50 years, using data from the Continuing Survey of Food Intake of Individuals. *J Am Diet Assoc.* 2001;101:455-460.
- 12. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med.* 2003;348:2082-2090.
- 13. Fabricatore AN, Wadden TA. Treatment of obesity: an overview. Clin Diabetes. 2003;21:67-72.

MEDICAL NUTRITION THERAPY FOR DISORDERS OF LIPID METABOLISM

Description

The dietary approach for the treatment of disorders of lipid metabolism, which include both hypercholesterolemia and hypertriglyceridemia, is a progressive reduction in total fat, saturated fat, trans fat, and cholesterol, coordinated in a plan to obtain or maintain reasonable body weight. The diet follows the recommendations of the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III of the National Institutes of Health and the American Heart Association (AHA) (1-4).

Indications

Medical nutrition therapy (MNT) is recommended as the primary treatment and management tool for disorders of lipid metabolism. MNT is recommended before administration of cholesterol-lowering medication in lower risk cases or in combination with drug therapy in higher risk cases. Recommended therapeutic lifestyle changes and target goals for low-density lipoprotein (LDL) cholesterol levels are based on risk factor assessment. Major independent risk factors (exclusive of LDL cholesterol) that predict the 10-year risk for coronary heart disease (CHD) are based on Framingham risk evaluation scores (1). These risk factors include (1):

- cigarette smoking
- hypertension (blood pressure greater than 140/90 mm Hg and/or receiving antihypertensive medication)
- low level of high-density lipoprotein (HDL) cholesterol (less than 40 mg/dL)
- family history of premature CHD (CHD in male first-degree relative younger than 55 years; CHD in female first-degree relative younger than 65 years)
- age (men, 45 years or older; women, 55 years or older)

The high-risk category consists of persons who have existing CHD, persons who have CHD risk equivalents that confer a 10-year risk of CHD greater than 20%, and persons with diabetes. The target LDL cholesterol level for these individuals is less than 100 mg/dL. A subcategory of high risk, very high risk, contains persons with existing cardiovascular disease and diabetes and persons with cardiovascular disease and severe or poorly controlled multiple risk factors (4). The very high-risk category has a therapeutic option to target the LDL cholesterol to less than 70 mg/dL (4). Persons with more than one risk factor and a 10-year risk of CHD of 20% or less have an LDL cholesterol target goal of less than 130 mg/dL. Persons with one or no risk factors and a 10-year risk of CHD less than 10% have an LDL target goal of less than 160 mg/dL. Primary intervention using therapeutic lifestyle changes and drug therapy should begin after evaluation of the patient's fasting lipid profile, consisting of levels of LDL cholesterol, total cholesterol, HDL cholesterol, and triglycerides and consideration of the risk factor assessment (1,4). LDL cholesterol is the primary target for risk reduction intervention (1,4). Targeting lower LDL goals, in combination with initiating cholesterol-lowering drug therapy at lower thresholds, is based on evidence from five randomized controlled trials demonstrating significant risk reduction for cardiac events at the recommended lower thresholds (4). The NCEP recommends pharmacologic therapy that is sufficient to achieve a 30% to 40% reduction in baseline LDL cholesterol levels in all high risk and moderately high risk patients (4).

Table C-8: LDL Cholesterol Goals (4)

Risk Category ^a	LDL Cholesterol Goal (mg/dL)	LDL Cholesterol Level (mg/dL) to Consider Drug Therapy ^b
High risk and very high risk:	<100	<u>≥</u> 100
CHD or CHD risk equivalent,	<70 optional	< 100 for very high risk
10-year risk >20%		
Moderately high risk:	< 130	<u>></u> 130
Two or more risk factors,		100-129 optional ^c
10-year risk 10%-20%		
Moderate risk:	<130	<u>></u> 160
Two or more risk factors,		
10-year risk <u><</u> 10%		
Lower risk:	<160	<u>>1</u> 90
Zero or one risk factor		160-189 optional

^aRisk factors that modify LDL goals are given in the bulleted list above, under Indications.

^bIt is advised that intensity of drug therapy be sufficient to achieve a 30% to 40% reduction in LDL cholesterol levels (4).

^c For moderately high risk persons, when LDL cholesterol is 100-129 mg/dL at baseline or on lifestyle therapy, initiation of an LDL-lowering drug to achieve an LDL cholesterol level <100 mg/dL is a therapeutic option (4).

For patients hospitalized due to a cardiac event, LDL cholesterol levels begin to decline in the first few hours after an event and are significantly decreased within the first 24 to 48 hours. These levels may remain low for up to 3 months. Thus, the initial LDL cholesterol level obtained in the hospital may be substantially lower than is usual for the patient (1). The NCEP ATP III emphasizes the need to begin drug therapy for high-risk patients (Refer to Table C-8) in addition to the Therapeutic Lifestyle Changes Diet to reach the goal for LDL cholesterol. When drugs are prescribed, the Therapeutic Lifestyle Changes Diet should always be maintained and reinforced (1).

Clinical trials demonstrate that lowering LDL cholesterol reduces total mortality, coronary mortality, major coronary events, coronary artery procedures, and strokes in persons with established CHD (1.4). The ATP III specifies LDL cholesterol less than 100 mg/dL as the goal of therapy in secondary prevention. The ATP III recognizes that the risk of CHD is influenced by other factors not included among the major, independent risk factors listed previously. Life-habit risk factors include obesity, physical inactivity, and atherogenic diet (1). Emerging risk factors, for which scientific evidence demonstrates varying degrees of contribution to CHD risk in select persons, include lipoprotein, homocysteine, prothrombotic and proinflammatory factors, and impaired fasting blood glucose levels (more than 110 mg/dL) (1). At this time, the evidence does not support specific modifications to target LDL cholesterol based on these risks.

Metabolic Syndrome

Metabolic syndrome is a clustering of three or more risk factors that include abdominal obesity, atherogenic dyslipidemia (elevated triglycerides level and low HDL cholesterol), hypertension, and insulin resistance (with or without glucose intolerance) (1). The NCEP ATP III recognizes the need to address metabolic syndrome as a secondary target of risk-reduction therapy, after the primary target of LDL cholesterol lowering (1). Management of metabolic syndrome primarily focuses on reducing underlying causes (eg, obesity and physical inactivity) and to treat associated nonlipid and lipid risk factors. Physical activity at any level (light, moderate, or vigorous) as well as food patterns emphasizing a diet high in fruits, vegetables, and whole grains is associated with reduced incidence of metabolic syndrome (Grade II)* (5). In the metabolic syndrome patient, a cardioprotective dietary pattern (low in saturated fat, trans fat, and cholesterol; limited in simple sugar intake; and increased in consumption of fruits, vegetables, and whole grains) provides the background for modifying the energy balance to achieve weight loss (Grade IV) (5). Extremes in intakes of carbohydrates or fats should be avoided (Grade IV) (5). Restricted energy intake combined with at least 30 minutes of physical activity on most days of the week should be used recommended_for individuals with metabolic syndrome (Grade II) (5). Weight loss of 7% to 10% of body weight should be encouraged if indicated (Grade II) (5).

Risk Factor	Defining Level
Abdominal obesity ^a	Waist circumference
Men	>102 cm (>40 inches) ^b
Women	>88 cm (>35 inches)
Triglycerides	≥150 mg/dL
HDL cholesterol	
Men	<40 mg/dL
Women	<50 mg/dL
Blood pressure	≥130/≥85 mm Hg
Fasting blood glucose	>100 mg/dL ^c

Table C-9: Clinical Identification of the Metabolic Syndrome

^a Overweight and obesity are associated with insulin resistance and the metabolic syndrome. However, abdominal obesity is more highly correlated with the metabolic risk factors than is an elevated body mass index. Therefore, the simple measure of waist circumference is recommended to identify the body weight component of the metabolic syndrome.

^b Some male patients can develop multiple metabolic risk factors when the waist circumference is only marginally increased, such as 94 to 102 cm (37 to 39 inches). These patients may have a strong genetic contribution to insulin resistance. They should benefit from changes in life habits, similar to men with categorical increases in waist circumference.

^c The ATP III criteria for the metabolic syndrome are now the same as the American Diabetes Association's revised definition of impaired fasting glucose. Thus, the threshold for elevated fasting glucose was reduced from 110 mg/dL to 100 mg/dL.

Nutritional Adequacy

The Therapeutic Lifestyle Changes Diet is planned to meet the Dietary Reference Intakes as outlined in Section IA: Statement on Nutritional Adequacy.

The National Institutes of Health maintain that the Therapeutic Lifestyle Changes Diet is consistent with good nutrition, and the aim of this diet is to achieve healthy eating patterns consistent with the Dietary Guidelines for Americans and American Heart Association Diet and Lifestyle Recommendations 2006 (1,3). A diet low in saturated fat, trans fats, cholesterol, and total fat is recommended for all healthy persons 2 years of age and older (1,3).

How to Order the Diet

Specify all that apply:

- Indicate Therapeutic Lifestyle Changes Diet (preferred) or Low-Fat, Low-Cholesterol Diet. The registered dietitian will determine the specific approaches used to implement the Therapeutic Lifestyle Changes Diet and medical nutrition goals based on individualized assessment and target treatment objectives to reduce CHD risk.
- Further reduction in total fat intake may be implemented. This additional reduction depends on the dietitian's assessment and the patient's compliance.
- If required, sodium control or other dietary modification should be specifically ordered.
- If weight reduction is desired, the dietitian should set the weight loss goal with the patient and determine the appropriate weight loss regimen.

Planning the Cardioprotective Diet

The Therapeutic Lifestyle Changes Diet stresses reductions in intake of saturated and trans fat (less than 7%) and cholesterol (less than 200 mg/dL) as the primary dietary modifications to lower LDL cholesterol in patients requiring primary and secondary prevention (1). Based on the response to a low-saturated fat, low-cholesterol diet, additional therapeutic options, such as plant stanols/sterols (2 g/day) and increased viscous (soluble) fiber (10 to 25 g/day), can be implemented as part of MNT self-management training (1). Refer to Disorders in Lipid Metabolism Evidence-Based Nutrition Practice Guideline (5). When metabolic syndrome or its associated lipid risk factors (elevated triglycerides level or low HDL cholesterol level) are present, therapeutic lifestyle changes also should include weight reduction and increased physical activity (1,5).

Table C-10: Nutrient Composition of Therapeutic Lifestyle Changes Diet (1,3,5)

Nutrient	Recommended Intake
Saturated and trans fat ^a	<7% of total energy combined (5) (trans fat <1% of total energy) (3)
Polyunsaturated fat	Up to 10% of total energy
Monounsaturated fat	Up to 20% of total energy
Total fat	25% to 35% of total energy
Carbohydrate ^b	50% to 60% of total energy
Fiber	25 to 30 g/day
Protein	Approximately 15% of total energy
Plant stanol/sterols	2 g/day (optional)
Cholesterol	<200 mg/day
Total energy ^c	Balance energy intake and energy expenditure to maintain desirable body weight and to prevent weight gain

^a Trans fatty acids are an LDL-raising fat; AHA recommends <1% of energy from trans fats (3).

^b Carbohydrate should be derived predominately from foods rich in complex carbohydrates, including grains (especially whole grains), fruits, and vegetables.

^c Daily energy expenditure should include at least moderate physical activity (contributing approximately 200 kcal/day).

Source: Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486-2497. Updates based on: *Disorders in Lipid Metabolism Evidence-Based Nutrition Practice Guideline*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2005. Available at: http://www.andevidencelibrary.com. Accessed June 1, 2006.

Cardioprotective Dietary Approaches in Managing Disorders in Lipid Metabolism

Total fat and saturated fatty acids: Elevated LDL cholesterol is an independent risk factor for CHD (1). Saturated fat is the principal dietary determinant of LDL cholesterol levels (6). The reduction of dietary saturated fat directly decreases clearance of LDL and very-low-density lipoprotein (VLDL) remnants (1). The recommendations for saturated fat are based on existing cardiovascular disease, risk factors, and LDL cholesterol value. A diet consisting of 25% to 35% total fat, <7% saturated and trans fat, and <200 mg cholesterol lowers serum total cholesterol and LDL cholesterol by 9% to 16% and decreases the risk of CHD

(Grade 1) (5). Sources of saturated fatty acids include: butter, lard, vegetable shortening, baked and pastry products, fat in meat, poultry, whole dairy products, palm oil, palm kernel oil, and cocoa butter. Moderate reduction of total fat (25% to 30% of total energy) facilitates a decrease in saturated fatty acids and may also help in weight reduction in overweight patients (1.7). The recommendations for total fat intake are based on the percentage of the patient's total daily energy intake, metabolic profile, and need for weight loss. The AHA does not recommend very-low-fat diets (less than 15% of total energy) (3). Very-low-fat diets may lead to inadequate intake of essential fatty acids. In addition, very-low-fat diets are often associated with the use of processed low-fat foods that are energy dense, compounding the metabolic abnormalities found in persons with high triglycerides levels, low HDL cholesterol levels, or insulin resistance (8.9).

Polyunsaturated fatty acids (PUFA): The two major categories of PUFA are omega-6 and omega-3 fatty acids. Linoleic acid is the primary omega-6 fatty acid and predominates in the American diet. The AHA recommends that less than 10% of total fat energy come from PUFA. The latest World Health Organization's guidelines set a range of 4% to 10% for PUFA intake (10). Isocalorically replacing saturated fatty acids with monounsaturated fatty acids (MUFA) and PUFA is associated with reductions in LDL cholesterol (Grade 1) (5). Studies have demonstrated that intakes of greater than 10% PUFA are associated with decreasing HDL cholesterol level, an independent predictor for CHD (10). Sources of omega-6 fatty acids include corn oil, safflower oil, sunflower oil, soybean oil, nuts, and seeds.

Omega-3 fatty acids: Studies have demonstrated beneficial effects of increased intake of omega-3 fatty acids in patients with coronary artery disease (11-14). Most of these studies used supplements containing long-chain omega-3 fatty acids (eg, eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA] or fish oil) at daily dosages ranging from 850 mg to 2.9 g. Studies that demonstrate a reduction in plasma triglycerides level provide higher dosages (3 to 4 g/day) (15). The GISSI trial demonstrated that high doses of omega-3 fatty acids provide benefits in preventing recurrent myocardial infarction events (1,14). Epidemiological studies indicate that regular consumption of an average of two servings of fatty fish per week (about 3.5 oz) high in long chain omega-3 fatty acids is associated with a 30% to 40% reduced risk of death from cardiac events in subjects with prior disease (Grade II) (5). One serving of fatty fish can provide approximately 1,000 mg of EPA and DHA (3). This amount from a supplement or fish reduces CHD mortality rates in patients with CHD (Grade II) (5). Because of the benefits of omega-3 fatty acids, the AHA Dietary Guidelines 2006 recommends consumption of more than two fish meals per week for the general population. Epidemiological studies indicate that inclusion of vegetable oils and food sources high in alpha-linolenic acid, resulting in a total intake of more than 1.5 g/day, is associated with a 40% to 65% reduced risk of death from cardiac events (Grade III) (5). This amount is equivalent to consuming ¹/₂ to 1 tablespoon ground flaxseed meal, 1 teaspoon flaxseed oil, or 1 tablespoon of canola or walnut oil. The 2006 AHA recommendations advise patients with documented CHD to consume approximately 1 g/day of EPA plus DHA, preferably from oily fish. EPA plus DHA supplements could be considered in consultation with a physician (3). For individuals with hypertriglyceridemia (> 200 mg/dL), 2 to 4 g/day of EPA plus DHA, provided as capsules under a physician's care, are recommended as a therapeutic option (Grade II) (3.5). Sources of omega-3 fatty acids include cold-water fish (salmon, mackerel, Atlantic herring, lake trout, albacore tuna, sardines, swordfish) tofu, soybean and canola oils, flaxseed, and English walnuts.

MUFA: Oleic acid is the primary MUFA. Evidence indicates that oleic acid may cause as great a decrease in LDL cholesterol levels as does linoleic acid when substituted for saturated fatty acids in the diet. Substitution of MUFA for saturated fat lowers LDL cholesterol levels without decreasing HDL cholesterol levels (Grade 1)(5,16). Evidence supports that a diet high in MUFA (up to 30% of total energy) can improve specific dyslipidemias compared with diets of equal energy value that replace fat with carbohydrate. A diet relatively high in unsaturated fat can prevent a decrease in HDL cholesterol levels and an increase in triglycerides levels that can occur in some individuals consuming a high-carbohydrate (more than 60% total energy), low-fat diet (17). Sources of MUFA include canola oil, olive oil, peanut oil, and avocados.

Trans fatty acids: Trans fatty acids are created through hydrogenation, a process in which vegetable oils are heated in the presence of metal catalysts to produce vegetable shortening and margarine. Trans fatty acids increase LDL cholesterol levels and decrease HDL cholesterol levels. Because saturated fats increase LDL cholesterol levels but do not decrease HDL cholesterol levels, trans fatty acids can produce a greater increase in the ratio of LDL cholesterol to HDL cholesterol (Grade I) (5). Population and cohort studies show that a high trans fatty acid intake increases risk of CHD events (Grade II) (5). It is estimated that 5% to 6% of the fat in the American diet is composed of trans fatty acids (16). Sources of trans fatty acids include hardened vegetable fat, stick margarine, shortening, and baked products made with these fats. Public concern has been raised about the use of margarine and whether other options, including butter, might be a better choice. The AHA Nutrition Committee recommends margarine as a preferable substitute for butter. Soft margarine with no more than 2 g

of saturated and trans fat per tablespoon and with liquid oil as the first ingredient is recommended in place of stick margarine. According to the 2005 Academy guidelines, a diet consisting of <7% saturated and trans fat combined should be the therapeutic goal for saturated and trans fat intake (Grade I) (5). According to the 2006 guidelines from the AHA, a diet should consist of <7% saturated fat and <1% trans fat (3).

Cholesterol: Dietary cholesterol is found only in animal products, especially those foods that are high in saturated fatty acids (eg, meats and whole dairy products). There is some evidence that dietary cholesterol enhances the serum cholesterol-raising action of saturated fatty acids, although to a lesser extent than saturated fat (1,3). Most foods high in saturated fat are also sources of dietary cholesterol. Reduced intake of foods high in saturated fatty acids, notably eggs and, to a lesser extent, shellfish, have smaller effects on LDL cholesterol (3). Therefore, periodic consumption of eggs and shellfish can be integrated into the Therapeutic Lifestyle Changes Diet meal plan.

Carbohydrates: Complex carbohydrates should make up the majority of digestible carbohydrates. When fat intake is reduced and nutrient replacement is required to maintain energy balance, the replacement should be complex carbohydrates. Recommended sources include whole grains, legumes, fruits, vegetables, nuts, and low-fat dairy products. Data on the ideal isocaloric substitution of carbohydrate for fat to maximize LDL-cholesterol lowering are presently not available (Grade V) (5).

Total protein and soy protein: Approximately 15% of total energy should be provided as protein. Currently there is no scientific evidence to support the concepts that high-protein diets result in sustained weight loss, significant changes in metabolism, or improved health (7,18). Recent randomized, controlled trials have demonstrated that consumption of 20 to 50 g of soy protein daily may reduce LDL cholesterol levels (5,19-22). Studies vary greatly in their estimation of the effect of diets low in saturated fat and cholesterol containing 26 to 50 g of soy protein, either as food or as a soy supplement with 0 to 165 mg of isoflavones (Grade II), and effects may vary based on initial cholesterol level (Grade III) (5). If consistent with patient preference and not contraindicated by risks or harms, then soy (eg, isolated soy protein, textured soy, tofu) may be included as part of a cardioprotective diet. Consuming 26 to 50 g/day of soy protein in place of animal protein can reduce total cholesterol by 0% to 20% and LDL cholesterol by 4% to 25% (Grade II) (5). Evidence is insufficient to establish a beneficial role for isoflavones as an independent component (Grade III) (5). Soy protein concentrates that remove isoflavones during processing may not be as effective (21). In October 1999, the Food and Drug Administration approved a health claim that allows food label claims for reduced risk of heart disease on foods that contain more than 6.25 g of soy protein per serving (3). The claim states that 25 g of soy protein per day, as part of a diet low in saturated fat and cholesterol, may reduce the risk of heart disease. Sources of soy protein include soy milk, soybeans, tofu, soy-based meat and cheese substitutes, and alfalfa sprouts. Soy protein may not be recommended in some individuals with breast cancer. Individuals with breast cancer or at high risk for breast cancer should speak with their physician (5). Consumption of greater than 50 g of soy protein with isoflavones may cause gastrointestinal distress in some individuals (5). Additionally, care should be taken when introducing soy into a patient's diet, because some individuals have an undiagnosed allergy to soy protein (5).

Total energy: Maintaining a balance between energy intake and expenditure is a goal of MNT. Some patients with high LDL cholesterol levels are sensitive to energy intake. Weight reduction and attainment of a reasonable body weight will completely correct their elevated LDL cholesterol concentrations. In many people, weight reduction will also reduce plasma triglycerides levels and raise HDL cholesterol levels (3).

Fiber: An intake of total dietary fiber 25 to 30 g is recommended for adults and is associated with decreased risk for CHD and cardiovascular disease (Grade II) (3,5,23). Increased intake of foods rich in soluble fiber correlates with decreased serum cholesterol levels (Grade I) (3,5,23). Consuming diet from whole food or supplements in total fiber (17 to 30 g/day) and soluble fiber (7 to 13 g/day) as part of a diet low in saturated fat and cholesterol can further reduce total cholesterol levels by 2% to 3% and LDL cholesterol levels by up to 7% (Grade I) (5). Choosing soluble fibers (notably beta glucan and pectin) found in oats, barley, and pectin-containing fruits and vegetable, beans and legumes provides adjunctive lipid-lowering benefits beyond those achieved through the reduction of total and saturated fat alone (3,23). The AHA recommends the intake of more than 25 g/day of soluble fiber from a variety of sources, including grains, vegetables, fruits, legumes, and nuts (3).

Other Dietary Factors Influencing Blood Lipid Levels and Risk Factors

Antioxidants: Oxidative processes are involved in the development and clinical expression of cardiovascular disease, and dietary antioxidants may contribute to disease resistance (3). Epidemiological data suggest that intake of foods rich in vitamin E, C, and beta carotene as part of a cardioprotective dietary pattern is associated with decreased risk for coronary artery disease (Grade III) (5,24-29). Most studies have involved the consumption of

antioxidant-rich foods, such as fruits, vegetables, and whole grains, from which antioxidants were derived (29). Because so many nutrients are contained in these foods, it is difficult to directly link the antioxidant nutrients to the reduction in CHD risk. Antioxidants such as vitamin E, vitamin C, and beta carotene (or carotenoids) should be specifically planned into a cardioprotective dietary pattern (Grade III) (5). The AHA recommends increased consumption of antioxidant-rich fruits, vegetables, and whole grains, which is associated with reduced disease risk (Grade III) (5,29). There is limited evidence to support the use of antioxidant supplements for disease prevention, even though this topic has been an issue of considerable debate (29). Vitamin E, vitamin C, and beta carotene supplements should not be recommended to reduce the risk of cardiovascular disease because they have shown no protection for cardiovascular disease events or mortality (Grade II) (5). The observation that adequate consumption of vitamin E may be difficult to achieve by dietary means leads the debate regarding vitamin E supplementation (26,27). Recent trials provide stronger evidence that vitamin E supplementation does not reduce cardiovascular disease or all-cause mortality; in some cases, vitamin E supplementation may lead to negative health outcomes including increased risk of death from hemorrhagic stroke (30-34). The Cambridge Heart Antioxidant Study (32) demonstrated a benefit of vitamin E in secondary prevention; however, the GISSI trial (14) and Heart Outcomes Prevention Evaluation Study (33) showed no beneficial effects of vitamin E at doses of 300 mg and 400 mg, respectively. Supplemental vitamin E, vitamin C, beta carotene, and selenium should not be taken with the simvastatin/niacin drug combination because of possible blunting of HDL2 (the HDL subfraction that is thought to be most protective) and an increased percentage of stenosis demonstrated in one study (Grade II) (5). Supplemental beta carotene cannot be recommended for individuals who smoke because of an increased risk for lung cancer in these individuals (Grade II) (5,28,29,34).

Folic acid and vitamins B_6 and B_{12} : Homocysteine, an amino acid in the blood, appears to oxidize LDL cholesterol (16). A high level of serum homocysteine, independent of other cardiac risk factors, is associated with increased risk for coronary artery disease. Conversely, low homocysteine levels are associated with reduced risk. (Grade II) (5,16). An increase in blood total homocysteine levels of 5 µmol/L elevates the risk of coronary artery disease as much as does a 20 mg/dL increase in total cholesterol (16). Factors that influence blood homocysteine levels include: deficiencies of folate, B_6 , and B_{12} ; age; sex; menopausal status; renal function; and certain medications. Folate is required for the conversion of homocysteine levels (35-37). Although supplemental folate (0.5 to 2.5 mg) taken alone or in combination with B_6 (10 to 25 mg) and B_{12} (0.4 mg) reduced homocysteine levels by 17% to 34%, it did not reduce the risk for coronary events after a period of 6 months to 2 years in stable coronary artery disease patients, post-stroke patients, or post-angioplasty patients that had normal baseline homocysteine levels and total cholesterol levels (Grade II) (5,38,39). Folate, vitamin B_6 , and vitamin B_{12} should be included in the cardioprotective dietary pattern to meet the Dietary Reference Intakes. Based on current evidence the supplementation of these vitamins to lower cardiovascular risk is not recommended (5).

Alcohol: Alcohol does not affect LDL cholesterol concentrations, but it does increase serum triglycerides concentrations and HDL cholesterol levels in many individuals. The mechanism for the rise in HDL cholesterol is not known. It is also not known whether the higher level of HDL affords any protection against CHD. Population and cohort studies, primarily of men, suggest that one or two drinks of alcohol-containing beverages per day are associated with reduced risk of cardiovascular disease, while excessive consumption of alcohol is associated with increased CHD (Grade I) (5,40-42). The NCEP report and the AHA do not specifically recommend alcohol consumption for CHD prevention (1,5). Most data do not support an association the between type of alcoholic beverage (wine, beer, or liquor) and protection against cardiovascular disease (Grade II) (5,40-42). The cardiovascular disease benefits of alcohol may be realized when alcohol is consumed with meals (Grade II) (5,40-42). Observational studies have found a relationship between high alcohol intake (more than three drinks per day) and elevated blood pressure (3,5). The AHA Guidelines 2000 are consistent with the US Dietary Guidelines in recommending that daily alcohol consumption be limited to two drinks for men and one drink for women.

Phytochemicals: Phytochemicals are nutritive substances found in plants. Nuts, whole grains, fruits, and vegetables contain a variety of phytochemicals. Epidemiologic studies show a relationship between the intake of phytochemicals, primarily plant sterols, flavonoids, and plant sulfur compounds, and CHD. Plant sterols found in rice bran decrease cholesterol levels (43). Flavonoids found in tea, onions, soy, and wine have antioxidant properties. Plant sulfur substances found in garlic, onions, and leeks decrease serum cholesterol (44). Studies are difficult to interpret because food sources containing phytochemicals have multiple compounds, and results distinguishing a specific cause and effect to the atherosclerotic process have been limited. Studies are currently assessing the relationship of phytochemicals to CHD.

Nuts: Nuts may enhance the cardioprotective dietary pattern because of their beneficial fatty acid profile as well as other favorable nutritional components (5,43). The daily consumption of 50 to 113 g ($\frac{1}{2}$ to 1 cups) of nuts with a diet low in saturated fat and cholesterol decreased total cholesterol by 4% to 21% and LDL cholesterol by 6% to 29% when weight was not gained (Grade II) (5,45,46). Nuts (walnuts, almonds, peanuts, macadamia, pistachios, and pecans) may be isocalorically incorporated into a cardioprotective dietary pattern low in saturated fat and cholesterol (5). Consuming 5 oz/week of nuts is associated with a reduced risk of CHD (5,45,46).

Stanol and sterol ester-containing foods: Plant sterols occur naturally and are isolated from soybean oils (3). Plant sterols are generally esterified, forming sterol esters, to increase solubility; in some cases, plant sterols are saturated to form stanol esters (3). Esterified forms of plant sterols and stanols are potent hypercholesterolemic agents, and the daily consumption of 2 to 3 g (in the form of margarine, lowfat yogurt, orange juice, breads, and cereals) lowers total cholesterol concentrations in a dose-dependent manner by 4% to 11% and LDL cholesterol concentrations by 7% to 15% (Grade II) (5,47,48). The efficacy of the two forms, plant stanol esters and plant sterol esters, is comparable (Grade II) (5, 47, 48). In addition, non-esterified forms of sterols and stanols are equally effective (Grade III) (5,49). The total cholesterol and LDL-cholesterol lowering effects of stanols and sterols are evident even when sterols and stanols are consumed as part of a cholesterol-lowering diet (Grade I) (5,47,48). For patients receiving statin therapy, plant stanols further reduce LDL cholesterol and total cholesterol (Grade III) (5). A limited effect of plant stanols and sterols on HDL cholesterol and triglycerides has been reported. If consistent with patient preference and not contraindicated by risks or harms, then plant sterol ester- and stanol ester-enriched foods may be consumed two or three times per day, for a total of 2 or 3 g/day, in addition to a cardioprotective diet to further lower total cholesterol and LDL cholesterol levels (Grade I) (5). For maximal effectiveness, foods containing plant sterols and stanols (spreads, juices, and yogurts) should be eaten with other foods (5). To prevent weight gain, isocalorically substitute stanol- and sterol-enriched foods for other foods (5). Plant stanols and sterols are effective in people who take statin drugs. Plant sterol and stanol products should not be used in individuals with sitosterolemia (5).

Other Diet Approaches

Very-low-fat, high-carbohydrate (VLFHC) diets: Very-low-fat (up to 15% of total energy), high-carbohydrate (greater than or equal to 60% of total energy) diets have been implemented with patients who are not responsive to diets with more moderate fat intake (eg, 25% to 30%). Progression of coronary atherosclerosis was delayed among patients who consumed a VLFHC diet and engaged in regular exercise (50,51). Replacing both saturated fat and unsaturated fat with carbohydrates can lower total cholesterol and LDL cholesterol levels. In addition, carbohydrate increases VLDL concentrations by adding triglycerides to VLDL particles, a characteristic unfavorable to individuals who have preestablished characteristics of metabolic syndrome. A VLFHC diet also decreases HDL cholesterol levels (1). However, the VLFHC diet–induced lowering of HDL cholesterol levels that are associated with a high-fat diet (52). Decreases in total cholesterol and HDL cholesterol and HDL ratio. This finding was observed in participants in the Lifestyle Heart Trial who demonstrated angiographic regression of their CHD while also demonstrating diet-induced lowering of HDL cholesterol levels (51).

The VLFCH diet plans include the Pritikin program and the Ornish meal plans (50,51). These meal plans limit total fat intake to less than 10% of total energy and encourage the consumption of whole grains, fruits, and vegetables in addition to increased physical activity and reduced stress. In certain individuals under a physician's supervision, very-low-fat diets may lead to weight loss and improved lipid profiles. However, these diets are not recommended by the AHA, as previously mentioned, especially for persons who exhibit characteristics of metabolic syndrome or who have hypertriglyceridemia (3). See "Total fat and saturated fatty acids" earlier in this section.

Mediterranean diet: The traditional Mediterranean diet has been investigated based on favorable effects on the lipid profile and decreased CHD risk in persons who live in Mediterranean countries. The Mediterranean diet is a plant-based diet that emphasizes fruits, vegetables, breads and other cereal grains, potatoes, beans, nuts, and seeds (53). Olive oil is the principal fat source. Cheese and yogurt are the key dairy products consumed daily. Fish, poultry, and eggs are consumed in moderate amounts. Desserts consist of fresh fruit, and concentrated sugars are eaten only a few times per week. Little red meat is eaten, and moderate amounts of red wine can be consumed with meals (53). Total fat content of the diet ranges from 25% to 35% of total energy, and saturated fat contributes less than 8% of total energy. Few studies have examined the Mediterranean diet plan. In one study, recurrent myocardial infarction, all cardiovascular events, and cardiac and other deaths were reduced by 70% in the group consuming a Mediterranean diet (54). Further research is needed to investigate the impact of this diet plan on cholesterol lowering and to determine if it is the specific dietary components (eg,

omega-3 fatty acid), increases in specific fatty acid consumption (eg, oleic acid or linolenic acid), or increased antioxidant consumption (eg, vitamins C and E) that cause the cholesterol-lowering effect (54).

Cardiovascular risk (1,3,4): Increased blood cholesterol levels or, more specifically, increased levels of LDL cholesterol are related to increased risk of CHD. Coronary risk rises progressively with an increase in cholesterol levels, particularly when cholesterol levels are greater than 200 mg/dL. Patients with established CHD are at high risk of subsequent myocardial infarction or CHD death, a risk five to seven times higher than without established CHD.

Substantial evidence indicates that lowering total cholesterol and LDL cholesterol levels (often combined with drug interventions) will decrease the incidence of CHD in both primary and secondary prevention settings (eg, patients with and without evidence of CHD, respectively) (3,4).

Short-term clinical trials support the projection that for individuals with serum cholesterol levels initially in the 250 to 300 mg/dL range, each 1% reduction in serum cholesterol level yields approximately a 2% reduction in CHD rates. Epidemiologic studies suggest that the reduction in CHD rates achievable by a long-term cholesterol-lowering regimen amounts to as much as 3% for each 1% reduction in serum cholesterol level. Thus, it is reasonable to estimate that the 10% to 15% reduction in serum cholesterol level resulting from MNT should reduce CHD risk by 20% to 30%.

Patient-Centered vs Population Approaches (1,3)

The patient-centered and population approaches are complimentary and together represent a coordinated strategy aimed at reducing cholesterol levels and coronary risk.

Patient-centered approach: A clinical or patient-based approach seeks to identify individuals at high risk who will benefit from intensive intervention efforts. Criteria define the candidates for medical intervention. Guidelines describe how to detect hypercholesterolemia, how to set goals for patients, and how to treat and monitor these patients.

Population approach: The population or public health approach attempts to lower blood cholesterol levels in the whole population by promoting healthful changes in dietary habits and physical activity levels. The AHA and the NCEP III take the position that a generalized reduction in cholesterol levels in Americans should decrease the prevalence of CHD. It is widely assumed that the eating habits of Americans are primarily responsible for high cholesterol levels. For this reason, the AHA and the NCEP III recommend that the population at large adopt an eating pattern designed to maintain plasma cholesterol levels near the desirable range.

Special Groups

Older adults: According to the AHA, advanced age does not preclude the need to follow the Therapeutic Lifestyle Changes Diet or heart-healthy guidelines (3,4). Postmenopausal women and older men with elevated LDL cholesterol are at increased risk of developing cardiovascular disease and therefore should be managed based on risk assessment (1,3,4). When older individuals follow a reduced saturated fat and cholesterol diet, LDL cholesterol levels decrease (1,3,55). In addition, drug therapy (eg, statins) can be a beneficial adjunct in high-risk patients and is the preferred therapy in place of hormone-replacement therapy in postmenopausal women (1,4). Because older adults have decreasing total energy needs, they have the added challenge of requiring education on the need for nutrient-dense foods within various food groups to meet nutritional needs (56).

Pregnant women with preexisting lipid disorders: Elevations in blood cholesterol and triglycerides levels may occur during pregnancy, with maximal levels in the third trimester and a return to normal levels after delivery. Generally, the MNT previously prescribed for the preexisting lipid disorder should be continued during pregnancy. If MNT is very restrictive, careful consideration should be given to ensure adequate nutrient intake. Drug therapy should be discontinued during pregnancy, since the effect of lipid-lowering drugs on the fetus has not been carefully studied (1,3).

Children and adolescents: The AHA Guidelines 2000 are indicated for all healthy individuals older than 2 years (3). However, according to the AHA, it should not be assumed that a diet for adults is also appropriate for children. Individual growth and nutritional requirements need to be considered at each stage of development. Studies have demonstrated that diets low in saturated fat can support adequate growth and development in children and adolescents (57,58). The prevalence of obesity and type 2 diabetes mellitus is increasing in the pediatric population (3). Nutrition strategies for this population should focus on appropriate nutritional intake, balancing energy intake, and increasing physical activity (3).

Racial and ethnic populations: African Americans have the highest overall CHD mortality rate and the highest out-of-hospital coronary death rates of any ethnic group in the Unites States, particularly at younger ages (1). The increased incidence is partly attributed to increased prevalence of coronary risk factors, including hypertension, ventricular hypertrophy, diabetes mellitus, cigarette smoking, obesity, and physical inactivity. Other high-risk ethnic groups and minority populations in the United States include Hispanics, Native Americans, Asian and Pacific Islanders, and South Asians. The NCEP ATP III recommends intervention for cholesterol management consistent with recommendations outlined for all other populations (1).

ATP III Guidelines

Criteria that define candidates for MNT intervention and the Therapeutic Lifestyle Changes Diet and lifestyle modifications have been established by the NCEP ATP III, which builds on the previous recommendations of ATP II and ATP I (1). These guidelines emphasize the importance of MNT provided by a registered dietitian in facilitating the behavior changes needed to follow the recommended diet and lifestyle changes before initiating therapy with cholesterol-lowering medications or in adjunct with this therapy. The guidelines are expected to substantially increase the number of Americans being treated for high cholesterol. The number of people receiving dietary treatment is expected to increase from 52 million to 65 million, and the number of people receiving prescribed cholesterol-lowering drugs is expected to increase from 13 million to 36 million (1).

Table C-11: Features of ATP III Guidelines

Focus on Multiple Risk Factors

- Identifies persons with diabetes but without CHD, most of whom have multiple risk factors, as CHD risk equivalent
- Uses Framingham projections of 10-year absolute CHD risk (i.e., the percent probability of having a CHD event in 10 years) to identify certain patients with two or more risk factors for more intensive treatment
- Identifies persons with multiple metabolic risk factors (metabolic syndrome) as candidates for intensified therapeutic lifestyle changes

Lipid and Lipoprotein Classification

- Identifies LDL cholesterol levels <100 mg/dL as optimal
- Categorizes low HDL cholesterol levels as <40 mg/dL
- Specifies lower cutoff levels for triglycerides to increase the attention given to moderate elevations

Support for Implementation

- Recommends a complete lipoprotein profile (total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides) as the preferred initial test, rather than screening for only total cholesterol and HDL
- Encourages use of plant stanols and sterols and viscous (soluble) fiber as therapeutic dietary options to enhance lowering of LDL cholesterol
- Presents strategies for promoting adherence to therapeutic lifestyle changes and drug therapies
- Recommends treatment beyond LDL lowering for persons with triglycerides levels \geq 200 mg/dL

Risk Category	LDL-C Goal (mg/dL)	LDL-C Level (mg/dL) to Initiate Therapeutic Lifestyle Changes	LDL-C Level (mg/dL) to Consider Drug Therapyª
CHD or CHD risk equivalent, 10-year risk >20%	<100 <70 optional	<u>≥</u> 100	≥100 < 100 optional
Two or more risk factors, 10-year risk 10% to 20%	< 130	> 130	≥130 100-129 optional
Two or more risk factors, 10-year risk <u><</u> 10%	<130	<u>></u> 130	<u>></u> 160
Zero or one risk factor ^b	<160	<u>≥</u> 160	≥190 160-189 optional

Table C-12: LDL Cholesterol (LDL-C) Goals and Recommendations for Therapeutic Lifestyle Changes and Drug Therapy in Different Risk Categories (4)

^aIt is advised that the intensity of therapy be sufficient to achieve at least a 30% to 40% reduction in LDL-C levels. If a person has high triglycerides levels and low HDL levels, consider combining nicotinic acid or fibrate with the LDL-lowering drug.

^bAlmost all people with 0 or 1 risk factor have a 10-year risk <10%; thus, 10-year risk assessment in these individuals is unnecessary.

The degree of reduction of LDL cholesterol levels achieved by MNT depends on dietary habits before starting therapy, degree of compliance, and inherent biological responsiveness. In general, patients with high cholesterol levels experience greater absolute reductions in LDL cholesterol levels than do those with low

Manual of Clinical Nutrition Management

cholesterol levels (1). Lowering total cholesterol and saturated fat intake (less than 7% to 8% of total energy) reduces total cholesterol and LDL cholesterol by 13% and 16%, respectively (59). For every 1% decrease in energy consumed from saturated fat, total cholesterol levels and LDL cholesterol levels may each decrease by approximately 2 mg/dL (59). Studies of MNT provided by a registered dietitian indicate that with two to six planned visits patients reported a 15% to 22% reduction in total dietary fat (from 32% to 33% of energy to 25% to 28% of energy) and a 22% to 36% reduction in saturated fat (from 11% to 12% of energy to 7% to 9% of energy). These dietary changes were accompanied by a 6% to 13% reduction in total plasma cholesterol levels and a 7% to 14% reduction in LDL cholesterol levels (Grade I) (5).

Management of Specific Dyslipidemias and Special Considerations

Because the LDL cholesterol level offers more precise risk assessment and is the primary target of medical interventions, treatment decisions are primarily based on LDL cholesterol levels. However, the NCEP ATP III recommends the evaluation of a comprehensive lipoprotein panel that includes fasting blood LDL cholesterol, HDL cholesterol, triglycerides, and total cholesterol. A comprehensive panel provides a more accurate picture so that the Therapeutic Lifestyle Changes Diet and drug therapy can be individualized based on a patient's metabolic characteristics.

Low HDL cholesterol (<40 mg/dL): Low HDL cholesterol levels are a significant risk factor for CHD, independent of LDL cholesterol levels and other risk factors (1). A reduced serum level of HDL cholesterol is defined as a concentration less than 40 mg/dL. For every 1 mg/dL decrease in HDL cholesterol level, the risk of CHD is increased by 2% to 3% (60). Likewise, higher HDL cholesterol levels appear to afford a degree of protection against CHD. This protection warrants considering a high HDL cholesterol level (\geq 60 mg/dL) as a negative risk factor. HDL cholesterol is measured as part of the lipoprotein analyses for primary prevention in adults without CHD. For secondary prevention in adults with evidence of CHD, lipoprotein analysis is required in all patients, and classification is based on only LDL cholesterol. Appropriate advice in treatment includes smoking cessation, weight reduction, and increased physical activity. Avoidance of certain drugs that reduce HDL cholesterol, such as beta-adrenergic blocking agents (beta-blockers), anabolic steroids, and progestational agents, should also be considered (1).

Very high LDL cholesterol (\geq190 mg/dL): Persons with very high LDL cholesterol levels usually have genetic forms of hypercholesterolemia (eg, familial hypercholesterolemia, familial defective apolipoprotein B, and polygenic hypercholesterolemia). Early detection should be completed in young adults to prevent premature CHD. These disorders often require combined drug therapy (statin and bile acid sequestrant) to achieve the goals of LDL-lowering therapy (1).

Elevated serum triglycerides levels (≥200 mg/dL): According to the NCEP ATP III, meta-analyses of prospective studies indicate that elevated triglycerides levels are an independent risk factor for CHD. Factors

that contribute to elevated triglycerides levels include obesity and overweight, physical inactivity, cigarette smoking, excess alcohol intake, high-carbohydrate diets (more than 60% of energy intake), certain diseases (eg, type 2 diabetes mellitus, chronic renal failure, and nephrotic syndrome), certain drugs (eg, corticosteroids, estrogens, retinoids, and higher doses of beta-adrenergic blocking agents), and genetic disorders (1). Generally, elevated serum triglycerides levels are most often observed in persons with metabolic syndrome (1). The NCEP ATP III adopts the following classification of serum triglycerides levels:

Normal triglycerides	<150 mg/dL
Borderline high triglycerides	150 to 199 mg/dL
High triglycerides	200 to 499 mg/dL
Very high triglycerides	<u>></u> 500 mg/dL

The VLDL level is the most readily available measure of atherogenic remnant lipoproteins. The ATP III identifies the sum of LDL and VLDL cholesterol (termed non-HDL cholesterol, which can also be calculated by taking total cholesterol minus HDL cholesterol) as a secondary target of therapy in persons with high triglycerides levels (more than 200 mg/dL) (1). The goal for non-HDL cholesterol in persons with high serum triglycerides levels can be set at 30 mg/dL higher than that for LDL cholesterol (eg, if LDL goal is less than 100 mg/dL, non-HDL goal would be less than 130 mg/dL) (1).

The treatment for elevated triglycerides level depends on the causes and severity (1). The primary aim of therapy is to achieve the target goal of LDL cholesterol. Fibrates and nicotinic acid can be used in combination with LDL-lowering drugs to lower triglycerides and achieve lipid goals (1,4). The ideal macronutrient

composition for lowering triglycerides in patients with hypertriglyceridemia > 150 mg/dL is unclear (Grade III) (5). A calorie controlled, cardioprotective dietary pattern that avoids extremes in carbohydrate and fat intake, limits alcohol, limits refined sugar while increasing complex carbohydrate food and includes physical activity is suggested (Grade III) (5). Weight loss of 7 to 10% of body weight should be encouraged if indicated (5). These lifestyle changes have been shown to lower triglyceride levels (Grade III) (5). If a patient is identified to have very high triglycerides (> 500 mg/dL), the dietitian should refer to the ATP III guidelines described below and recommend a very low-fat diet (< 15% of energy intake) (Grade II) (5). High doses of supplemental EPA/DHA have been shown to lower triglycerides in patients with elevated triglycerides (Grade II) (5). If a patient has high triglycerides (> 200 mg/dL) supplemental EPA/DHA capsules of 2 to 4 grams per day may be prescribed by the physician or be considered a therapeutic intervention (Grade II) (5).

Triglycerides (TG) Level	ATP III Recommended Approaches (1)
Borderline high (150-199	weight reduction
mg/dL)	increased physical activity
High (200-499 mg/dL)	weight reduction
	increased physical activity
	• drug therapy (LDL-lowering drugs and/or nicotinic acid or fibrate to lower
	TG)
Very high (≥500 mg/dL)	• very-low-fat diet (<15% of energy intake) to prevent acute pancreatitis
	weight reduction
	increased physical activity
	• drug therapy (LDL lowering-drugs and/or nicotinic acid or fibrate to lower
	TG)

When low HDL cholesterol (less than 40 mg/dL) is associated with a high triglycerides level (200 to 499 mg/dL), the secondary priority is achieving the non-HDL cholesterol goal (as previously described). The HDL-raising drugs, which include fibrates and nicotinic acid, can be considered in persons with CHD and CHD risk equivalents. Fibrates and nicotinic acid can be used in combination with LDL-lowering drugs to achieve lipid goals (1,4). Also see Section III: Hypertriglyceridemia.

Physical Activity and Weight Management

Excess body fat, or obesity, is a major risk factor for CHD and also contributes to the development of other risk factors, such as diabetes and hypertension. Weight management plays a vital role in achieving and maintaining good health while enhancing the quality of life. Proper nutrition and physical activity are key factors that influence weight control. The National Heart, Lung, and Blood Institute has established clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. These guidelines identify a need for MNT weight management (61).

An important adjunct to long-term change in eating habits and lifestyle is an increase in physical activity. There is evidence that regular exercise alone reduces CHD mortality by increasing HDL cholesterol levels and, in some patients, lowering LDL cholesterol levels. The exercise should emphasize aerobic activity, such as brisk walking, jogging, swimming, bicycling, and tennis. Improvements in cardiovascular fitness result from exercising at moderate intensity for 30 to 45 minutes on most, if not all, days (Grade II) (1,5). Vigorous, high-intensity exercise must be performed with caution in high-risk persons and only with the advice of a physician and under the supervision of trained personnel (1).

Self-Management Training for the Patient with Disorder of Lipid Metabolism

Once the patient's risk assessment, clinical status, motivation, comprehension, and environmental support are assessed, the dietitian should set goals with the patient and/or caregiver and provide self-management training to meet the patient's individualized needs. Refer to the following evidence-based nutrition practice guidelines (5, 62) or a combination of treatment guidelines as needed for treatment and frequency of MNT intervention:

- Morrison Nutrition Practice Guideline Coronary Artery Bypass Graft (CABG) or Angioplasty (63)
- Disorders of Lipid Metabolism Evidence-Based Nutrition Practice Guideline from the Academy of Nutrition and Dietetics (5)

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486-2497.
- Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Final Report. 2002. NIH publication No. 02-5215. Available at: http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3full.pdf. Accessed December 17, 2002.
- 3. AHA Diet and Lifestyle Recommendations Revision 2006: A Scientific Statement From the American Heart Association Nutrition Committee. *Circulation*. 2006;114:82-96.
- 4. Grundy SM, Cleeman JI, Merz CN, Brewer B, Clark LT, Hunninghake DB, Pasternak RC, Smith SC, Stone NJ for the Coordinating Committee of the National Cholesterol Education Program. Implications of recent trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation*. 2004;110:227-239.
- 5. *Disorders in Lipid Metabolism Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2011. Available at: http://www.andevidencelibrary.com. Accessed January 24, 2013.
- 6. Hegsted DM, Ausman LM, Johnson JA, Dallal GE. Dietary fat and serum lipids: an evaluation of the experimental data [published erratum appears in *Am J Clin Nutr.* 1993;58:245]. *Am J Clin Nutr.* 1993;57:875-883.
- 7. Kennedy ET, Bowman SA, Spence JT, Freedman M, King J. Popular diets: correlation to health, nutrition, and obesity. *J Am Diet Assoc.* 2001;101:411-420.
- 8. Kris-Etherton PM, Pearson TA, Wan Y, Hargrove RL, Moriarity K, Fishell V, Etherton TD. High-monounsaturated fatty acid diets lower both plasma cholesterol and triacylglycerol concentrations. *Am J Clin Nutr*. 1999;70:1009-1015.
- 9. Kasim-Karakas SE, Almario RU, Mueller WM, Peerson J. Changes in plasma lipoproteins during low-fat, high-carbohydrate diets: effects of energy intake. *Am J Clin Nutr.* 2000;71:1439-1447.
- 10. Stone NJ, Nicolosi R, Kris-Etherton P, Ernst ND, Krauss RM, Winston M. Summary of the scientific conference on the efficacy of hypocholesterolemic dietary interventions. *Circulation*. 1996;94:3388-3391.
- 11. De Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation*. 1999;99:779-785.
- 12. Singh RB, Niaz MA, Sharma JP, Kumar R, Rastogi V, Moshiri M. Randomized, double-blind, placebo-controlled trial of fish oil and mustard oil in patients with suspected acute myocardial infarction: the Indian experiment of infarct survival-4. *Cardiovasc Drugs Ther*. 1997;1:485-491.
- 13. Von Schacky C, Angerer P, Kothny W, Theisen K, Mudra H. The effect of dietary omega-3 fatty acids on coronary atherosclerosis: a randomized, double-blind, placebo-controlled trial. *Ann Intern Med.* 1999;130:554-562.
- 14. GISSI-Prevenzione Investigators. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. *Lancet.* 1999;354:447-455.
- 15. Harris WS. n-3 Fatty acids and serum lipoproteins: human studies. *Am J Clin Nutr*. 1997;65:1645S-1654S.
- 16. Kwiterovich PO. The effect of dietary fat, antioxidants, and pro-oxidants on blood lipid, lipoproteins, and atherosclerosis. *J Am Diet Assoc.* 1997;97:S31-S41.
- 17. Kris-Etherton PM. AHA Science Advisory: monounsaturated fatty acids and risk of cardiovascular disease. American Heart Association Nutrition Committee. *Circulation*. 1999;100:1253-1258.
- 18. St Jeor ST, Ashley JM. Dietary strategies: issues of diet composition. In: Fletcher GF, Grundy SM, Hayman LL, eds. *Obesity: Impact on Cardiovascular Disease*. Armonk, NY: Futura Publishing Co Inc; 1999: 233-246.
- 19. Anderson JW, Johnstone BM, Cook-Newell ME. Meta-analysis of the effects of soy protein intake on serum lipids. *N Engl J Med*. 1995;333:276-282.
- 20. Baum JA, Teng H, Erdman JW Jr, Weigel RM, Klein BP, Perskey WW, Freels S, Surya P, Bakhit RM, Ramos E, Shay NF, Potter SM, et al. Long-term intake of soy protein improves blood lipid profiles and increases mononuclear cell low-density lipoprotein receptor messenger RNA in hypercholesterolemic, postmenopausal women. *Am J Clin Nutr.* 1998; 68:545-551.
- 21. Crouse JR III, Morgan T, Terry JG, Ellis J, Vitolins M, Burke GL. A randomized trial comparing the effect of casein with that of soy protein containing varying amounts of isoflavones on plasma concentrations of lipids and lipoproteins. *Arch Intern Med.* 1999;159:2070-2076.
- 22. Tonstad S, Smerud K, Hoie L. A comparison of the effects of 2 doses of soy protein or casein on serum lipids, serum lipoproteins, and plasma total homocysteine in hypercholesterolemic subjects. *Am J Clin Nutr.* 2002;76:78-84.
- 23. Position of the American Dietetic Association: Health implications of dietary fiber. J Am Diet Assoc. 2008;108:1716-1731.
- 24. Kritchevsky SB, Tell GS, Shimakawa T, Dennis B, Li R, Kohlmeier L, Steere E, Heiss G. Provitamin A carotenoid intake and carotid artery plaques: the Atherosclerosis Risk in Communities Study. *Am J Clin Nutr*. 1998;68:726-733.
- 25. Kushi LH, Folsom AR, Prineas RJ, Mink PJ, Wu Y, Bostick RM. Dietary antioxidant vitamins and death from coronary heart disease in postmenopausal women. *N Engl J Med.* 1996;334:1156-1162.
- 26. Rimm EB, Stampfer MJ, Ascherio A, Giovannucci E, Colditz GA, Willett WC. Vitamin E consumption and the risk of coronary heart disease in men. *N Eng J Med.* 1993;328:1450-1456.
- 27. Stampfer MJ, Hennekens CH, Manson JE, Colditz GA, Rosner B, Willett WC. Vitamin E consumption and the risk of coronary heart disease in women. *N Engl J Med.* 1993;328:1444-1449.
- Morton SP, Hardy M. Effect of Supplemental Antioxidants Vitamin C, Vitamin E, and Coenzyme Q10 for the Prevention and Treatment of Cardiovascular Disease. Evidence Report/Technology Assessment No. 83 (Prepared by Southern California-RAND Evidence-based Practice Center). Rockville, MD: Agency for Healthcare Research and Quality; 2003. AHRQ publication No. 03-E043.
- 29. Tribble DA. AHA Science Advisory: antioxidant consumption and risk of coronary heart disease: emphasis on vitamin C, vitamin E, and beta-carotene: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1999;99:591-595.
- 30. Miller ER 3rd, Pastor-Barriuso R, Dalal D, Riemersma RA, Appel LJ, Guallar E. Meta-analysis: high-dosage vitamin E supplementation may increase all-cause mortality. *Ann Intern Med.* 2005;142:37-46.
- 31. Lonn E, Bosch J, Yusuf S, Sheridan P, Pogue J, Arnold JM, Ross C, Arnold A, Sleight P, Probstfield J, Dagenais GR, HOPE and HOPE-TOO Trial Investigators. Effects of long-term vitamin E supplementation on cardiovascular events and cancer: a randomized controlled trial. *JAMA*. 2005;293:1338-1347.
- 32. Stephens NG, Parsons A, Scholfield PM, Kelly F, Cheeseman K, Mitchinson MJ. Randomised controlled trial of vitamin E in patients with coronary disease: Cambridge Heart Antioxidant Study (CHAOS). *Lancet.* 1996;347:781-786.
- 33. Lonn E, Yusuf S, Hoogwerf B, Pogue J, Yi Q, Zinman B, Bosch J, Dagenais G, Mann JF, Gerstein HC; HOPE Study; MICRO-HOPE Study. Effects of vitamin E on cardiovascular and microvascular outcomes in high-risk patients with diabetes: Results of the HOPE study and the MICRO-HOPE substudy. *Diabetes Care*. 2002;25:1919-1927.
- 34. Vivekananthan DP, Penn MS, Sapp SK, Hsu A, Topol EJ. Use of antioxidant vitamins for the prevention of cardiovascular disease: metaanalysis of randomized trials. *Lancet.* 2003;361: 2017-2023.

- 35. Gallagher PM, Meleady R, Shield D, Tan KS, McMaster D, Rozen R, Evans A, Graham I, Whitehead A. Homocysteine and risk of premature coronary heart disease. *Circulation*. 1996:2154-2158.
- 36. Morrison H, Schaubel D, Desmeules M, Wigle D. Serum folate and risk of fatal coronary heart disease. JAMA. 1996;275:1893-1896.
- 37. Bautista LE, Arenas IA, Penuela A, Martinez LX. Total plasma homocysteine level and risk of cardiovascular disease: a meta-analysis of prospective cohort studies. *J Clin Epidemiol*. 2002;55:882-887.
- 38. Schnyder G, Roffi M, Flammer Y, Pin R, Hess OM. Effect of homocysteine-lowering therapy with folic acid, vitamin B₁₂, and vitamin B₆ on clinical outcome after percutaneous coronary intervention: the Swiss Heart study: a randomized control trial. *JAMA*. 2002; 288:973-979.
- Toole JF, Malinow MR, Chambless LE, Spence JD, Pettigrew LC, Howard VJ, Sides EG, Wang CH, Stampfer M. Lowering homocysteine in patients with stroke to prevent recurrent stroke, myocardial infarction, and death: the Vitamin Intervention for Stroke Prevention (VISP) Randomized Controlled Trial. JAMA. 2004; 291: 565-575.
- 40. Di Casteinuovo A, Rotondo S, Iacoviello L, Donati MB, deGaetano G. Meta-analysis of wine and beer consumption in relation to vascular risk. *Circulation*. 2002; 105:2836-2844.
- 41. Goldberg IJ, Mosca L, Paino MR, Fisher EA. AHA Science Advisory: wine and your heart: a science advisory for healthcare professionals for the Nutrition Committee, Council on Epidemiology and Prevention, and Council on Cardiovascular Nursing of the American Heart Association. *Circulation*. 2001; 103:472-475.
- 42. Mukamal KJ, Conigrave KM, Mittleman MA, Camargo CA, Stampfer MJ, Willett WC, Rimm EB. Roles of drinking pattern and type of alcohol consumed in coronary heart disease in men. *N Engl J Med.* 2003; 348:109-118.
- 43. Craig,W. Phytochemicals: Guardians of our health. J Am Diet Assoc. 1997: 97:S199-S204.
- 44. Science Advisory and Coordinating Committee of the American Heart Association. Phytochemicals and cardiovascular disease. *Circulation*. 1997;95:2591-2593.
- 45. Kris-Etherton PM, Zhao G, Pelkman CL, Fishell VK, Coval SM. Beneficial effects of a diet high in monounsaturated fatty acids on risk factors for cardiovascular disease. *Nutr Clin Care*. 2000;3:153-162.
- 46. Sabate J, Haddad E, Tanzman J, Rajaram S. Serum lipid response to the graduated enrichment of a Step I diet with almonds: a randomized feeding trial. *Am J Clin Nutr.* 2003;77:1379-1384.
- 47. Hallikainen MA, Sarkkinen ES, Uusitupa MI. Plant stanol esters affect serum cholesterol concentrations of hypercholesterolemic men and women in a dose-dependent manner. *J Nutrition*. 2000;130:767-776.
- 48. Homma Y, Ikeda I, Ishikawa T, Tateno M, Sugano M, Nakamura H. Decrease in plasma low-density lipoprotein cholesterol, apolipoprotein B, cholesteryl ester transfer protein, and oxidized low-density lipoprotein by plant stanol ester-containing spread: a randomized, placebo-controlled trial. *Nutrition*. 2003;19:369-374.
- 49. Vanstone CA, Raeini-Sarjaz M, Parsons WE, Jones PJH. Unesterified plant sterols and stanols lower LDL-cholesterol concentrations equivalently in hypercholesterolemic persons. *Am J Clin Nutr.* 2002;76:1272-1278.
- 50. Ornish D, Scherwitz LA, Billings JH, Brown SE, Gould KL, Merritt TA, Sparler S, Armstrong WT, Ports TA, et al. Intensive lifestyle changes for reversal of coronary heart disease. *JAMA*. 1998;280:2001-2007.
- 51. Gould KL, Ornish D, Scherwitz L, Brown S, Edens RP, Hess MJ, Mullani N, Bolomey L, Dobbs F, et al. Changes in myocardial perfusion abnormalities by positron emission tomography after long-term, intense risk factor modification. *JAMA*. 1995;274:894-901.
- 52. Connor WE, Connor SL. Should a low-fat, high carbohydrate diet be recommended for everyone? N Engl J Med. 1997;337:562-566.
- 53. Willett WC, Sacks F, Trichopoulou A, Drescher G, Ferro-Luzzi A, Helsing E, Trichopoulos D. Mediterranean diet pyramid: a cultural model for healthy eating. *Am J Clin Nutr.* 1995;61(suppl):1402S-1406S.
- 54. Renaud S, deLoregeril M, Delaye J, Gaidollet J, Jacquerd T, Marmelle N, Martin JL, Monjaud L, Salen P, Toubel P. Cretan Mediterranean diet for prevention of coronary heart disease. *Am J Clin Nutr.* 1995;61(suppl 1):1360S-1376S.
- 55. Ginsberg HN, Kris-Etherton P, Dennis B, Elmer PJ, Ershow A, Levevre M, Pearson T, Roheim P, et al. Effects of reducing dietary saturated fatty acids on plasma lipids and lipoproteins in healthy subjects: the DELTA study, protocol 1. *Arterioscler Thromb Vasc Biol.* 1998;18:441-449.
- 56. Russell RM, Rasmussen H, Lichtenstein AH. Modified Food Guide Pyramid for people over seventy years of age. J Nutr. 1999; 129:751-753.
- 57. Obarzanek E, Hunsberger SA, Van Horn L, Hartmuller VV, Barton BA, Stevens VJ, Kwiterovich PO, Franklin FA, Kimm SY, Lasser NL, Simons-Morton DG, Laaer RM. Safety of a fat-reduced diet: the Dietary Intervention Study in Children (DISC). *Pediatrics*. 1997;100:51-59.
- 58. Daniels SR, Greer FR, and the Committee on Nutrition. Lipid screening and cardiovascular health in childhood. *Pediatrics*. 2008;122:198-208.
- Yu-Poth S, Zhao G, Etherton T, Naglak M, Jonnalagadda S, Kris-Etherton PM. Effects of the National Cholesterol Education Program's Step I and Step II Dietary intervention programs on cardiovascular disease risk factors: a meta analysis. *Am J Clin Nutr.* 1999;69:632-646.
- 60. Oster G, Thompson D. Estimated effects of reducing dietary saturated fat intake on the incidence and costs of coronary heart disease in the United States. *J Am Diet Assoc.* 1996;96:127-132.
- 61. National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. Clinical *Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: the Evidence Report.* Bethesda, Md: National Institutes of Health; 1998. NIH publication No. 98-4083. Available at: <u>http://nhlbi.nih.gov/nhlbi/htm</u>.
- 62. Position of The American Dietetic Association and Dietitians Canada: Dietary fatty acids. J Am Diet Assoc. 2007;107:1599-1611.
- 63. Morrison Nutrition Practice Guideline Coronary Artery Bypass Graft (CABG) or Angioplasty. In: *Morrison Nutrition Practice Guidelines*. Atlanta, Ga: Morrison Management Specialists Inc; 2009. Available at: www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition

FOOD GUIDE - LOW SATURATED FAT, LOW CHOLESTEROL OR TLC DIETS

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages	Carbonated beverages, soft drinks, coffee, tea, cocoa mixes	
Breads, Cereals and Grain Products	Most breads, cereals, pasta, rice, dried peas, beans, and potatoes. Low-fat crackers: Rye Crisp, Saltines*, bread sticks, fat-free crackers*, melba toast, English muffins, bagels, pita bread	Commercial baked goods: croissants, cheese breads, doughnuts, muffins, biscuits, butter- type rolls, quick breads, granola- type cereals, snack crackers like cheese crackers, potato chips, egg noodles, creamed potatoes/pasta
Vegetables and Fruits	All fresh, canned, or dried fruits and vegetables	Vegetables prepared in butter, cream, or other sauces
Meat, Fish and Poultry (limit to 5 to 6 oz/day)	Chicken & turkey: remove the skin and any visible fat Fish & shellfish Veal & wild game Beef, pork & lamb: use lean cuts only. Profigure 1 and ground heaf cirlain tip	Poultry: goose, domestic duck Fatty cuts of meat Fried fish Beef: corned beef brisket*, regular ground beef, short ribs
	 Beer: extra real ground beer, smolif up, ground steak, and rump roast. Pork: center-cut ham*, loin chops and tenderloin Luncheon meats at least 95% fat-free 	Processed meats: bacon*, bologna*, salami*, sausage*, hot dogs* Organ meats: liver, gizzard, brains, heart & kidney (limit to once a month)
Eggs	Egg whites, cholesterol-free egg substitute Reduced cholesterol eggs or egg substitutes	Egg whites unlimited, egg yolks (2 to 4 per week based on lipid profile and response to low fat diet)
Milk and Dairy Products (2 to 3 serving/day)	Fat-free or 1% fat milk and buttermilk, low-fat or nonfat yogurt, fruited or frozen (1% or less milk fat)	Whole milk (4%), regular, evaporated, condensed; cream, half & half, reduced fat milk, imitation milk products, nondairy creamers, whipped toppings, eggnog, whole milk yogurt, milkshakes
	Low-fat cheese: any cheese labeled 2 to 6 g of fat per oz, part-skim mozzarella cheese, cottage cheese (1% or 2%) or, part-skim ricotta cheese (1/4 cup)	All natural cheeses (eg, bleu, brie, feta, muenster, Roquefort, camembert, cheddar, Swiss, American)

*High in salt. Individuals limiting their salt intake should avoid these foods.

Food Guide - LOW SATURATED FAT, LOW CHOLESTEROL OR TLC DIETS (Cont.)		
FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Fats & Oils Limit to 6 to 8 tsp or portions a day, including fat used in cooked products.	Unsaturated trans fat free vegetable oils: canola, safflower, sunflower, corn, peanut, olive, soybean. Margarine in which the first ingredient is "liquid" oil. Diet margarine. Nonfat butter substitutes. Nonstick spray. Nonfat or fat-free salad dressing or mayonnaise. Fat-free cream cheese and sour cream	Saturated fats & oils: butter, coconut oil, cocoa butter, palm kernel oil, lard, or bacon fat. Margarine or shortenings in which the first ingredient is a partially hydrogenated oil or animal fat. Margarines containing trans fat. Dressings made with egg yolk, bleu cheese, or sour cream. Coconut, chocolate, commercial dips. Low-fat or "light" cream cheese or sour cream
	 Olives* (5 large) Nuts (1 oz = 3 tsp fat) Avocado (1/8 med) Mayonnaise-type dressing (2 tsp) Salad dressing (1 Tbsp) Peanut butter (2 Tbsp =3 tsp of allowed fat) 	 Foods with hidden fat: Biscuit* (2") 1 tsp of fat Cornbread* (2" cube) 1 tsp of fat Muffin (small) 1 tsp of fat Pancakes (2) 1 tsp of fat Stuffing/dressing (½ cup) 1 tsp of fat Cake (3" square) 1 tsp of fat Fruit pie (1" wedge) 1 tsp of fat Cookies (2 to 2") 1 tsp of fat These foods are acceptable if prepared homemade with oil or "liquid" margarine. Count the "hidden" fat in these foods toward the daily fat allowances.
Soups	Broth type made with allowed ingredients.	Cream soups. Condensed cream soups.*
Sugar & sweets	 "Fat-free" frozen desserts, cakes, and cookies; angel food cake; sherbet, sorbet, gelatin; occasionally ice milk, low-fat yogurt Syrups: chocolate (made with cocoa), strawberry, etc. Cookies: graham crackers, gingersnaps, animal crackers, fig bars, vanilla wafers Pudding made with fat free milk Hard candy 	Commercial baked goods: pies, cakes, cookies, doughnuts, pastries; ice cream, brownies, fudge topping, milk chocolate candy

*High in salt. Individuals limiting their salt intake should avoid these foods.

For patients on sodium restrictions, use the following sample daily meal plans.

Food Group	Number of Servings 2000 mg sodium	Number of Servings 1500 mg sodium
Bread, Cereals, and Grain Products (150 mg/serving)	6 regular bread or cereal items	5 regular bread or cereal items
Vegetables/Fruits (10 mg/ ½ c serving)	5	5
Meat, Fish, Poultry and Eggs (60 mg/ 1 oz)	6	6
Milk and Dairy (150 mg/serving)	3	2
Fats (100 mg/serving)	1	0 (regular)
	7-8 unsalted	7-8 unsalted
Soup (unsalted only)	Calculate into diet	Calculate
Sugar & Sweets	Read label and calculate into diet	Read label and calculate into diet
Total Sodium	1760 mg	1460 mg

FAT-CONTROLLED DIET (50 Grams)

Description

Omitting and/or limiting fat-containing foods restricts the total amount of fat in the diet. The type of fat is not considered.

Indications

A fat-controlled diet is indicated for individuals who are unable to properly digest, metabolize, and absorb fat. Common diseases of the hepatobiliary tract, pancreas, intestinal mucosa, and lymphatic system impair fat digestion, metabolism, and absorption (1-5). A low fat-diet may also be useful in the treatment of patients with gastroesophageal reflux (4.6).

Contraindications

In pancreatic insufficiency, enzyme preparations remain the primary treatment for steatorrhea. As normal a diet as possible is encouraged to increase the likelihood that a nutritionally adequate diet will be consumed (5,7,8). The diet should restrict fat only to the individual's tolerance level.

The treatment of choice for gallstones at the present time, where indicated, is surgery. There is no reason in the postoperative period to restrict or modify fat intake in any way.

Nutritional Adequacy

The Fat-Controlled Diet can be planned to meet the Dietary Reference Intakes (DRIs) for all nutrients as outlined in Section IA: Statement on Nutritional Adequacy. Vitamin E intake will be lower than in a regular diet. However, the requirement for vitamin E is proportional to the intake of polyunsaturated fatty acids, which will also be reduced in a Fat-Controlled Diet.

Ordering the Diet

- Order as "Low-Fat Diet" or "50-Gram-Fat Diet" can be ordered (this is sufficiently restricted for many indications).
- Other levels of fat restriction can be specified, eg, 25 to 35 g fat diet.
- If a cholesterol restriction is desired, the diet ordered should be "Therapeutic Lifestyle Changes Diet" in Section IC: Medical Nutrition Therapy for Disorders of Lipid Metabolism.

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED	
Beverages and Milk	Coffee, tea, carbonated drinks, fat-free milk or buttermilk, evaporated skim, nonfat dry milk, skim or low-fat yogurt	Whole, reduced-fat, evaporated, condensed, or chocolate milk, yogurt made from whole milk, cocoa mixes	
Breads, Cereals and Grains	Whole-grain or enriched breads, dinner rolls, cereals and grains, pasta, plain crackers	Quick breads such as muffins, biscuits, rich or sweet rolls, doughnuts, pancakes, waffles, party crackers, potato chips, granola unless calculated into diet	
Meat, Fish, Poultry, Cheese, Eggs (average 3 to 5 g fat/oz) (limit intake to 5 oz/day)	Lean meat (trimmed of visible fat), fish, and fowl (without skin). The following are equal to 1 oz meat: 1 egg, ¼ cup tuna, salmon (water-packed), or cottage cheese. The following low-fat cheeses are allowed (one serving per day): 1oz low fat or fat free milk cheeses (sapsago, mozzarella, farmer's) or ¼ cup 1% cottage or ricotta cheese	Fried or fatty meats, such as luncheon meats, cured and processed meats, other cheeses	

FOOD GUIDE – FAT-CONTROLLED DIET

FOOD GUIDE – FAT-CONTROLLED DIET (Cont.)		
FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Vegetables	Any prepared without fat.	Vegetables in cream sauces or gravies, fried vegetables including potatoes
Fruits and Juices	All	None
Fats (limit intake to 5 tsp/day; use no more than 2 servings/meal) (Average: 1 tsp fat = 5 g fat)	Butter, margarine, vegetable oil, crisp bacon (1 strip = 1 tsp fat)	Cream, avocado, nuts, coconut, olives, peanut butter
Soup	Any soups made with fat free milk or fat free broth	Commercially canned soups, cream soups, soups containing fat or whole milk
Desserts	Fruit, sherbet, sorbet, fat-free frozen desserts, gelatin, angel food/sponge cake, low fat cookies (gingersnaps, vanilla wafers), fat-free cakes, puddings made with fat free milk, meringues	Ice milk, ice cream, pie, cake, cookies, pastries, any desserts made with shortening, chocolate, cream, nuts, or fat
Sweets	Sugar, jelly, honey, syrups with no fats, molasses, plain marshmallows, hard candy	Any containing chocolate, nuts, cream, coconut, butter-flavored or fudge syrup
Miscellaneous	Vinegar, low-calorie or fat-free dressings, cocoa or carob powder, herbs and spices, salt, pepper, Butter Buds [®]	Chocolate, coconut, gravy

SAMPLE MENU

Breakfast	Noon	Evening
Orange juice	Honey glazed chicken (skinless)	Lean beef tips and noodles
Cream of wheat	Baked potato/margarine	Seasoned green beans
Scrambled egg	Steamed broccoli	Sliced tomato salad
Wheat toast	Fruited gelatin	Fat-free French dressing
Margarine	Dinner roll	Peach halves
Jelly	Margarine	Dinner roll
Fat free milk	Sherbet	Margarine
Coffee	Iced Tea	Fat free milk
Sugar	Sugar	Iced tea
		Sugar

References

- 1. Gastrointestinal Disease/Gallbladder. In: *Academy of Nutrition and Dietetics Nutrition Care Manual*. Chicago: Ill: Academy of Nutrition and Dietetics; 2007. Available at: nutritioncaremanual.org. Accessed January 6, 2007.
- 2. Burch JM. Acute pancreatitis. In: Rakel RE, ed. *Conn's Current Therapy*. Philadelphia: WB Saunders Co; 1993:502-506.
- 3. Chak A, Banwell JG. Malabsorption syndromes. In: Rakel RE, ed. Conn's Current Therapy. Philadelphia: WB Saunders Co; 1993:496-502.
- 4. Dwyer JT, Roy J. Diet therapy. In: Isselbacher KJ, Braunwald E, et al, eds. *Harrison's Principles of Internal Medicine*. 12th ed. New York, NY: McGraw-Hill, Inc; 1991:420-427.
- 5. Marotta RB, Floch MH. Dietary therapy of steatorrhea. Gastroenterol Clin N Am. 1989; 18:485-512.
- 6. Goyal RK. Diseases of the esophagus. In: Isselbacher KJ, Braunwald E, et al, eds. *Harrison's Principles of Internal Medicine*. 13th ed. New York, NY: McGraw-Hill, Inc; 1992:1503-1515.
- 7. Brady MS, Rickard K, Yu PL, Eigen H. Effectiveness of enteric coated pancreatic enzymes given before meals in reducing steatorrhea in children with cystic fibrosis. *J Am Diet Assoc.* 1992;92:813-817.
- 8. Bone R. Cystic fibrosis. In: Wyngaarden JB, Smith LH, Bennett JC, eds. *Cecil Textbook of Medicine*. 19th ed. Philadelphia: WB Saunders Co; 1992;418-421.

MEDIUM-CHAIN TRIGLYCERIDES (MCT)

Description

Commercial medium-chain triglycerides (MCT) are composed of 8 to 10 carbon fatty acids synthesized from palm kernel and coconut oils (1). MCT provide 8.3 kcal per g and 116 kcal per tablespoon (2).

Indications

MCT are indicated in conditions where long-chain triglycerides (LCT) are not well tolerated. MCT are commonly used in fat-controlled diets to provide increased calories and improve the palatability of a reduced-fat diet See Section 1C: Fat-Controlled Diet. The following properties of MCT may make it useful in disorders where LCT are problematic:

- Absorption can occur despite pancreatic lipase deficiency (2).
- Bile salts or micelles are not required for dispersion in water and subsequent absorption (2).
- Transport across the intestinal mucosa occurs more readily than with LCT (2).
- MCT are not dependent upon chylomicrons for transit and consequently do not require lipoprotein lipase for oxidation (2).
- Transport does not occur through the lymphatic system. MCT travel directly to the liver via the portal vein, as free fatty acids bound to albumin (2).
- MCT hydrolyzes to fatty acids more quickly (2) and oxidizes more rapidly and efficiently than LCT (1).

MCT may be adjunctive to a fat-controlled diet in the following conditions:

- pancreatic insufficiency (1)
- cystic fibrosis (1)
- intestinal resection (1)
- hepatobiliary disease (1)
- lymphangiectasia (2)
- chyluria (2)
- chylous ascites (2)
- chylothorax (2)
- secondary carnitine deficiency syndromes (3)
- whipple's disease (4)
- hyperchylomicronemia (4)

MCT may be therapeutically incorporated into the ketogenic diet, which is used to control epileptic seizures (see Section IE: Ketogenic Diet) and may also be used in adjunct with antineoplastic treatment for pediatrics (5).

Contraindications

Under normal physiologic conditions, MCT are ketogenic. Therefore, MCT are contraindicated in persons who are prone to diabetic ketoacidosis (2).

In cirrhosis, MCT accumulate in the blood, resulting in a condition that presents with symptoms similar to hepatic encephalopathy, including hyperlactacidemia, hyperammonemia, hyperventilation, and altered EKG findings (2).

Nutritional Adequacy

MCT are used in conjunction with specific diets, such as fat-controlled or ketogenic diets. Nutritional adequacy will depend on the prescribed diet.

How to Order the Diet

MCT are generally ordered in conjunction with a fat-controlled diet. The order should specify the number of mL or g MCT to be added to the diet. For example: "____ g Fat-Controlled Diet plus ____ mL (g) MCT".

Planning the Diet

Medium-Chain Triglycerides (MCT)

- MCT are available as MCT oil or in formulas containing MCT.
- MCT should be introduced slowly to avoid the abdominal distention and pain, nausea, vomiting, and diarrhea associated with rapid infusion or high dose (2).
- MCT in divided doses of no more than 15 to 20 mL (3 to 4 tsp) at a time are generally well tolerated (2). Patients should initially receive no more than 20 to 30 mL per day, increasing by 5 to 10 mL per day as tolerated until the MCT goal is met.
- To incorporate MCT into the diet, the following are suggested (2):
 - Add 1 tsp MCT oil to 4 oz fat free milk, carbonated beverages, juices, or flavored drinks. If patient is
 prescribed a ketogenic diet, use sugar-free beverages and follow fluid restrictions.
 - Substitute an equal amount of MCT oil for other fats when cooking and baking.
 - Prepare salad dressings with MCT oil.

References

- 1. Babineau TJ, Pomposelli J, Forse RA, Blackburn GL. Specific nutrients: carbohydrates, lipids, nucleic acids. In: Zaloga GP, ed. *Nutrition in Critical Care.* St. Louis, Mo: Mosby; 1994:196-197.
- 2. Nelson JK, Moxness KE, Jensen MD, Gastineau CF, eds. Gastrointestinal diseases and disorders. In: *Mayo Clinic Diet Manual: A Handbook of Nutrition Practices.* 7th ed. St. Louis, Mo: Mosby; 1994:230-232.
- 3. Pons R, De Vivo DC. Primary and secondary carnitine deficiency syndromes. J Child Neuro. 1995;10:S8-S24.
- 4. Long-chain trigylceride restricted medium-chain trigylceride diet. In: *Manual of Clinical Dietetics*. 6th ed. Chicago, Ill: American Dietetic Association; 2000: 725.
- 5. Nebeling LC, Lerner E. Implementing a ketogenic diet based on medium-chain triglyceride oil in pediatric patients with cancer. *J Am Diet. Assoc.*1995 Jun;95(6):693-7.

I. NORMAL NUTRITION AND MODIFIED DIETS D. Modification of Fiber

Fiber-Restricted Diets	D-1
Food Guide - Low Fiber Diet	D-2
High-Fiber Diet	
Food Guide High-Fiber Diet	D-10
Dietary Fiber Content Of Foods	D-11
Gastrointestinal Soft Diet	D-14
Food Guide-Gastrointestinal Soft Diet	D-15

FIBER-RESTRICTED DIETS (Low-Fiber*)

Description

The Low-Fiber Diet restricts dietary fiber and provides less than 10 grams a day $_{(1)}$. Foods that have been defined in qualitative terms as having tough fibers are also eliminated. Animal products, refined grain products and cereals (providing < 2 g/serving), and selected fruits and vegetables are included. Previously this diet had also been titled the "low residue diet". Due to no scientifically acceptable definition of residue and the lack of widespread availability of resources to provide this information, the term is no longer used by the Academy of Nutrition and Dietetics (1,2). Since fiber content of the diet can be estimated from food composition tables, "low fiber diet" is the preferred title of this diet and used throughout this manual (1,2).

Indications

- To prevent the formation of an obstructing bolus when the intestinal lumen is narrowed.
- To delay intestinal transit time in conditions of diarrhea.
- To reduce (not eliminate) the fiber in the colon pre- and postoperatively.
- To allow the bowel to rest during acute exacerbation of inflammatory bowel disease (ulcerative colitis, Crohn's disease), acute phases of diverticulitis, or radiation enteritis.

For patients with acute episodes of ulcerative colitis, Crohn's disease, or diarrhea the low fiber diet may need further modifications for individual symptom management. The avoidance of caffeine, lactose and excess fat may improve tolerance in patients with acute exacerbations of ulcerative colitis and Crohn's disease (3). The low-fiber diet is intended for short-term use. The goal of nutrition therapy is to establish tolerance to a wider variety of foods and to make a transition to a regular diet.

Contraindications

A fiber-restricted diet is contraindicated when a soft stool is desired, as in individuals with diverticulosis. A low-fiber intake may aggravate the symptoms of irritable bowel or constipation. In these cases, a high-fiber intake is recommended. (See Section ID: High-Fiber Diet.). If a soft texture is desired, as in the case of a patient with esophageal narrowing, a mechanical soft diet may be ordered.

Nutritional Adequacy

The Low-Fiber diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy. At least one serving of citrus fruit juice is recommended for daily vitamin C.

How to Order the Diet

Order as "Low-Fiber Diet." Milk and other lactose-containing foods will not be restricted unless ordered. Milk is fiber-free; therefore, it is not necessary to eliminate it (2). However, for individuals with inflammatory bowel disease who present with a primary or secondary lactose deficiency, a low-fiber, low-lactose dietary restriction may be appropriate. In addition, caffeine may also need to be limited or avoided in individuals with IBD, or diarrhea to decrease intestinal transit time (1.3).

Planning the Diet (1):

- 1. Select cereals, grains and bread products, and vegetables that are low in fiber (< 2 g fiber/serving)
- 2. For patients with a lactose intolerance, dairy products may need to be avoided or limited
- 3. For patients with IBD and diarrhea limit or avoid caffeine per individual tolerance (1.3).
- 4. Clean, prepare and store foods using proper sanitation techniques

Reference

- 1. Low Fiber Nutrition Therapy. Gastrointestinal Diseases. In: *The Academy of Nutrition and Dietetics Nutrition Care Manual*. Updated annually. Available at: http://www.nutritioncaremanual.org. Accessed January 25, 2013.
- 2. Cunningham E. Are low-residue diets still applicable? Journal of Academy of Nutrition and Dietetics. 2012; 112:960.
- 3. Brown AC, Rampertab SD, Mullin GE. Existing dietary guidelines for Crohn's disease and ulcerative colitis. *Expert Rev Gastroenterol Hepatol.* 2011;5:411-425.

Fiber-Restricted Diet

FOOD GUIDE - LOW FIBER DIET (1,3)			
FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED	
Beverages	Decaffeinated coffee, tea, and other	Caffeinated beverages (1,3)	
(0 gm fiber/serving)	beverages (1,3)		
No limit of allowed			
1000S Milk Products	Choose vogurt with live active cultures	Milk is avoided only when lactose	
(0 gm fiber/serving)	evaporated skim, and low-fat milk, sov	intolerance is present (1). Yogurt with	
No limit of allowed	milk, powdered milk, cheese, low-fat ice	berries, orange or lemon rind, or nuts.	
foods	cream, sherbet	Whole milk, half and half, cream, sour cream, regular ice cream	
Breads and Starches,	All breads and crackers made from white	Whole-wheat, rye, pumpernickel or	
Pasta, Rice	flour or choose grain foods with less	bran breads, crackers, muffins	
(0.5 gm fiber/ serving	than 2 grams dietary fiber per serving	Buckwheat pancakes	
per allowed foods)	(1) Craham crackers	Rye wafers	
No limit of allowed	Corn and flour tortillas	Breads and crackers containing fruit,	
TOODS	Cornbread	nuts, or seeds Prouge rice: barlow	
	Pasta, noodles, white rice	brown nee, barley	
Breakfast Cereals	Farina, cream of rice, grits,	Wheatena, rolled wheat, and other whole-	
No limit of allowed	white flour or others providing $< 2 \text{ g}$	Ready-to-eat whole-grain oat and bran	
foods	fiber per serving (1)	cereals including bran flakes, granola, Grape-Nuts, oat bran, 100% bran, puffed wheat, shredded wheat, wheat bran, wheat flakes, wheat germ	
Fruit Juices	Fruit juice without pulp (except for prune	Prune juice. Juice with pulp	
(0 gm fiber/serving	Juice).		
No limit of allowed			
foods			
Fruits	Banana, applesauce	All fresh fruits except banana	
(2.0 gm fiber/serving	Canned and well cooked fruits		
per allowed foods)			
of allowed fiber			
containing foods/day			
Vegetables and	Mushrooms (cooked)	Raw or fried vegetables	
Vegetables Juices	Tomato/vegetable juice	Broccoli	
(2.0 gm fiber/ serving	I omato sauce	Lorn Mixed vegetables	
Count in 3-6 servings	excent those on the Foods to Exclude list	Skin of notato	
of allowed fiber	encept those on the roots to Include hot	Brussel Sprouts	
containing foods/day		Cabbage	
		Cauliflower	
		Succotash (also see legumes)	
Moot Fich Doultw-	Tondon well cooled mosts neultry fish	Lollard, mustard, and turnip greens	
Cheese, Eggs	eggs and soy prepared without added	hacon Luncheon meats such as hologna	
(0 gm fiber/serving)	fat. Smooth nut butter	or salami, hot dogs, tough or chewy cuts	
No limit of allowed		of meat, fried eggs, all dried beans, peas,	

foods

and nuts, Chunky nut butters.

Cont. FOOD GUIDE- LOW-FIBER DIET

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Legumes None allowed	None	All legumes: chickpeas, lima beans, black- eyed peas, kidney beans, pinto beans, etc Peanut butter Baked beans
Soup	Meat, rice, noodle soups	Minestrone soup
(2.0 gm fiber/ serving per allowed foods) Count in 3-6 servings of allowed fiber containing foods/day	Soups made from allowed vegetables	Bean, pea, and lentil soups, chili
Fats (0 gm fiber/serving) No limit of allowed foods	Oils, butter, cream, margarine, mayonnaise	Nuts, seeds
Sugar and Sweets (0 gm fiber/serving) No limit of allowed foods	Cranberry sauce, seedless, sherbet	Candy containing fruits, nuts, or coconut Jam, marmalade, relishes containing seeds, or skins
Miscellaneous (0 gm fiber/serving) No limit of allowed foods	Catsup, spices, herbs, seasonings	Pickles

SAMPLE MENU (less than 10 g fiber)					
Breakfast	Noon	Evening			
Orange juice	Chicken rice soup	Baked chicken with gravy			
Grits	Roast beef sandwich on white bread	Whipped potatoes			
Soft-Cooked egg	Mayonnaise	Green beans			
White toast	Tomato juice	White dinner roll			
Margarine	Orange sherbet	Margarine			
Grape jelly	Decaffeinated Coffee and tea	Canned peaches			
Milk	Sugar	Milk			
Decaffeinated Coffee		Decaffeinated Tea			
Sugar		Sugar			

HIGH-FIBER DIET

Description

Dietary fiber is beneficial for health maintenance and disease prevention and is a key nutrition intervention strategy for several chronic diseases including cardiovascular disease, diabetes mellitus, and also effective in weight management, and bowel-related diseases (1). A high-fiber diet emphasizes the consumption of dietary fiber from foods of plant origin, particularly minimally processed fruits, vegetables, legumes, and whole-grain and high-fiber grain products. A plant-based diet may also provide other nonnutritive components such as antioxidants and phytoestrogens that have implications as health benefits (1). Dietary fiber intake in the United States continues to be at less than the recommended levels, with an average daily intake of only 15 g (1,2). Major sources of dietary fiber in the US food supply include grains and vegetables (1,3). White flour and white potatoes provide the most fiber in the US diet, about 16% and 9%, respectively, not because they are concentrated fiber sources, but because they are the most widely consumed (1.3). Legumes only provide about 6% of fiber and fruits provide only 10% of fiber in the overall US diet because of low food consumption (1.3). The Academy recommends that healthy adults consume the Dietary Reference Intakes (DRIs) of 14 g dietary fiber per 1,000 kcal, or 25 g/day for women and 38 g/day for men (1). The American Academy of Pediatrics recommends that children 2 years and older consume a daily amount of fiber equal to or greater than their age plus 5 g (1). In September 2002, the Institute of Medicine's Food and Nutrition Board released the first DRIs for total fiber that are based on life stage (see Table D-1) (2). In addition to the DRIs, the amount and sources of fiber in the diet should be determined by the nutrition objectives for the specific disease state, as outlined below.

Table D-1. DRIS 101 TOtal Fiber by Life Stage (2)					
Age	Male	Female			
0-12 months	ND ^b	ND			
1-3 years	19 g	19 g			
4-8 years	25 g	25 g			
9-13 years	31 g	26 g			
14-18 years	38 g	26 g			
19-50 years	38 g	25 g			
> 50 years	30 g	21 g			
Pregnancy (14-50 years)		28 g			
Lactation (14-50 years)		29 g			
3 DBIs based on 14 g total fiber nor 1 000 keel					

 Table D-1:
 DRIs^a for Total Fiber by Life Stage (2)

^a DRIs based on 14 g total fiber per 1,000 kcal

^bND, not determined

Definitions of Dietary Fiber

There are a variety of definitions of dietary fiber (1,2). Some definitions are based primarily on analytical methods used to isolate and quantify dietary fiber, whereas others are physiologically based (1). Crude fiber is the amount of plant material that remains after treatment with acid or alkali solvents. It is predominantly a measure of the cellulose content of a food and, as such, significantly underestimates the total dietary fiber found in plant food. Many older food composition tables report only crude fiber (4). For labeling purposes in the United States, dietary fiber is defined as the material isolated by analytical methods approved by the Association of Official Analytical Chemists. Although the Institute of Medicine recommends that the terms soluble and insoluble fiber not be used (2), food labels still may include soluble and insoluble fiber data (1).

The Panel on the Definition of Dietary Fiber under the Food and Nutrition Board of the National Academy of Sciences has developed definitions for dietary fiber, functional fiber, and total fiber. *Dietary fiber* is the nondigestible component of carbohydrates and lignin naturally found in plant foods (2,5). *Functional fiber* refers to fiber sources that have similar health benefits as dietary fiber, but are isolated or extracted from natural sources or synthetic sources (2,5). *Total fiber* is the sum of dietary fiber and functional fiber (2,5). The intent of these definitions is to recognize the physiologic actions of fiber and its demonstrable health effects and to reduce the emphasis on dietary fiber as a constituent of food requiring quantification (2,5). There has been a trend to assign specific physiologic effects either to soluble or insoluble fibers (5). This approach makes it difficult to evaluate the effects of fiber provided by mixed diets (5). Dietary fiber provided by mixed diets is two-thirds to three-fourths insoluble; however, the exact distribution between soluble and insoluble

fiber depends on the method of analysis (5). In addition, soluble and insoluble fiber foods often have similar benefits vs independent benefits that affect health outcomes. For example, both psyllium seed husk, an insoluble fiber source, and oat bran, a soluble fiber source, increase stool weight, improve laxation, and lower blood cholesterol levels (1). Only the viscous soluble fibers (not all soluble fibers) are hypocholesterolemic agents (1). Based on the review of evidence, the National Academy of Sciences' panel recommends that the terms *soluble fibers* and *insoluble fibers* gradually be eliminated and replaced by the specific beneficial physiologic effects of fiber (2,5).

Indications and Nutrition Intervention Guidelines

Constipation and normal laxation: Consumption of dietary fiber is a frequently prescribed nutrition intervention for the prevention or treatment of constipation. Fiber supplements may produce benefits in the laxation of healthy individuals (Grade III)* (1). However, more research is needed to clarify the dose and type of fiber needed for gastrointestinal health and management (Grade III) (1). Many fiber sources; including cereal brans, psyllium seed husk, methylcellulose, and a mixed high-fiber diet increase stool weight, thereby promoting normal laxation (1,6). The increase in stool weight is caused by the presence of fiber, the water that the fiber holds, and the partial fermentation of the fiber, which increases the amount of bacteria in stool (7). The large intestine responds to the larger and softer mass of residue produced by a high-fiber diet by contracting, which moves the contents toward excretion (1). Fiber in mixed diets, legumes, and whole-grain and high-fiber grain products are particularly effective promoters of normal laxation (1). A fiber supplement may be needed when food intake is low, as in the case of inactive older adults (1). Common fiber supplements are psyllium seed husk and methylcellulose (1). Many foods are natural laxatives because they contain indigestible carbohydrates and other compounds with natural laxative properties; these foods include cabbage, brown bread, oatmeal porridge, fruits with rough seeds, vegetable acids, aloe, rhubarb, cascara, senna, castor oil, honey (fructose), tamarinds, figs, prunes, raspberries, strawberries, and stewed apples (1). Fluid intake, exercise, stress, and relaxation also influence fecal elimination and should be considered when a dietitian is planning treatment.

Diverticulosis: Diverticular disease of the colon is thought to occur secondary to increased intracolonic pressure caused by hard, dry fecal material and the increased effort necessary to eliminate this type of stool. Well-controlled, experimental studies confirming the benefits of a high-fiber diet in the prevention and management of diverticular disease are relatively few, with less than conclusive results. One study found that 90% of patients with diverticular disease remained symptom-free after 5 years on a high-fiber diet (1,8). This result may be explained by the fact that a high-fiber diet promotes the formation of soft, large stools that are defecated more easily, resulting in lower colonic pressure and less straining during elimination (1). Also, a high-fiber diet may reduce the chance that an existing diverticulum will burst or become inflamed (1). The National Institute of Diabetes and Digestive and Kidney Diseases recommends 20 to 35 g of fiber each day for the management of diverticular disease (1,9). Mild pain medications may help to relieve symptoms; however, many pain medications affect the emptying of the colon, an undesirable side effect for people with diverticulosis (9).

To increase stool bulk, studies suggest increasing the consumption of whole-grain breads, cereals, and brans. In cases of diverticulosis, a common practice has been to provide a high-fiber intake that excludes the hulls of nuts, corn, and seeds because they may get trapped in the diverticula (1). However, a recent study found that the consumption of nuts, corn, and popcorn was not associated with an increased risk of complicated diverticular disease. Instead, the researchers observed inverse relationships between nut and popcorn consumption and the risk of diverticulitis (10). According to the National Institute of Diabetes and Digestive and Kidney Diseases, "foods such as nuts, popcorn hulls, and sunflower, pumpkin, caraway, and sesame seeds have been recommended to be avoided by physicians out of fear that food particles could enter, block, or irritate diverticula, however this is not validated by the research" (9). Poppy seeds and seeds in tomatoes, zucchini, cucumbers, strawberries, and raspberries are generally considered harmless (9). Because of the limited evidence, the dietitian should customize the patient's meal plan taking into account the patient's individual tolerances (1,9-11). The recommendation to avoid nuts, seeds, corn, and popcorn in diverticular disease should be reconsidered (11).

Irritable bowel syndrome: Diagnostic criteria for irritable bowel syndrome (IBS) is intestinal dysfunction of at least 3 months' duration, during which time diarrhea, diarrhea alternating with constipation, and chronic constipation may be experienced in the absence of any underlying disease states (12). IBS is characterized by abdominal discomfort associated with altered bowel function; structural and biochemical

abnormalities are generally absent (12). Patients who present with rectal bleeding, weight loss, iron deficiency anemia, nocturnal symptoms, and a family history of selected organic diseases (colon cancer, inflammatory bowel disease, or celiac disease) should undergo medical testing to exclude underlying causes (12). Microscopic colitis can masquerade as IBS (12). IBS is a prevalent and expensive condition that is associated with a significantly impaired health-related quality of life and reduced work productivity (12). Based on strict criteria, 7% to 10% of people worldwide have IBS (12). Approximately 60% of IBS patients believe that certain foods exacerbate their symptoms, and research suggests that allergies to certain foods could trigger IBS symptoms (12). However, based on an extensive review of the literature, there is no correlation between foods that patients identify as a cause of their IBS symptoms and the results of food allergy testing (12). Psyllium hydrophilic mucilloid (ispaghula husk) is moderately effective and can be given a conditional recommendation for managing IBS (12). Wheat bran or corn bran is no more effective than placebo in the relief of global symptoms of IBS and cannot be recommended for routine use (12). Certain antispasmodics (hyoscine, cimetropium, pinaverium, and peppermint oil) may provide short term relief of abdominal pain and discomfort (12). However, evidence of long-term efficacy, safety, and tolerability is limited (12). Probiotics possess a number of properties that may prove to be beneficial for patients with IBS (12). Although studies of Lactobacilli have repeatedly shown no effect on symptoms, probiotics combinations including strains of Lactobacillis Bifidobacteria infantis (eg, AlignTM) and Saccharomyces boulardii may improve symptoms of IBS (12,13). A position statement from the Academy of Nutrition & Deitetics suggests that fiber intake shows inconsistent results in IBS. Dietary fiber should be considered as a therapy for bowel syndromes, but it should not be regarded as a proven therapy that is suitable for all individuals with bowel syndromes (1). A reduction in lactose and foods sources of fermentable oligo-di-and monosaccharides and polyols (FODMAPs) have also been suggested to improve symptoms. These include fruits, dried fruits, fruit juice, fructose as added sweetener, high fructose corn syrup, honey, coconut, fortified wines, onion, leek, asparagus, artichokes, cabbage, brussel sprouts, beans, legumes, sorbitol, mannitol, isomalt, and xylitol (14, 15).

Cardiovascular disease and hypercholesterolemia: Dietary fiber intake from whole foods or supplements may lower blood pressure, improve serum lipid levels, and reduce indicators of inflammation such as Creactive protein (Grade II) (1). Benefits may occur with daily fiber intakes of 12 to 33 g from whole foods or up to 42.5 g from supplements (Grade II) (1). The DRI recommendations for dietary fiber intake are based on protection against cardiovascular disease (1). The one characteristic common to all cholesterol-lowering fibers is viscosity (16). Fiber that lowers blood cholesterol is found in foods such as apples, barley, beans and other legumes, fruits, vegetables, oatmeal, oat bran and rice hull. Purified sources of cholesterol-lowering fiber include beet fiber, guar gum, karaya gum, konjac mannan, locust bean, gum, pectin, psyllium seed husk, soy polysaccharide, and xanthan gum (1,17). The US Food and Drug Administration has studied two fibers, beta glucan in oats and psyllium husk, to authorize a health claim that foods meeting specific compositional requirements and containing 0.75 g of beta glucan or 1.7 g of psyllium husk per serving can reduce the risk of heart disease (18). Viscous fibers lower cholesterol because their viscosity interferes with bile acid absorption from the ileum. In response, low-density lipoprotein cholesterol is removed from the blood and converted into bile acids by the liver to replace the bile acids lost in the stool. Evidence also indicates that cholesterol synthesis is dampened by changes in the composition of the bile acid pool that accompany the ingestion of viscous fibers (1,19). Fiber ingestion also affects the levels of blood pressure and C-reactive protein, which are both biomarkers linked to the risk of cardiovascular disease (Grade II) (1). A secondary benefit of a high-fiber diet is a lower dietary content of energy, fat, and simple sugars; these reductions are effective dietary intervention strategies for weight management and hypertriglyceridemia associated with cardiovascular disease (1,20).

Diabetes mellitus: Diets providing 30 to 50 g of fiber per day from whole food sources consistently produce lower serum glucose levels compared to a low-fiber diet. Daily fiber supplements providing 10 to 29 g may have some benefit in terms of glycemic control (Grade III) (1). The addition of viscous dietary fibers slows gastric emptying rates, digestion, and the absorption of glucose to benefit immediate postprandial glucose metabolism and long-term glucose control in individuals with diabetes mellitus (1). The American Diabetes Association has determined that the consumption of soluble fiber independent of total fiber has limited documented effects on glycemic control in individuals with diabetes (21). Although large amounts of dietary fiber (>50 g/day) may have beneficial effects on glycemia, insulinemia, and lipemia, it is not known whether such high levels of fiber intake can be maintained long-term (21). According to the American Diabetes Association and evidence-based nutrition practice guidelines, fiber consumption recommendations for people with diabetes are the same as for the general population (1,21). The DRI recommends consumption of 14 g of dietary fiber per 1,000 kcal, or 25 g for adult women and 38 g for adult men. For general health benefits, the

daily consumption of dietary fiber is encouraged from food sources such as whole grains, fruits, and vegetables (21).

Weight management: Dietary fiber intake from whole foods or supplements may have some benefit in terms of weight loss and other health outcomes associated with weight loss (Grade III) (1). Benefits may occur with daily fiber intakes of 20 to 27 g from whole foods or up to 20 g from supplements (Grade III) (1). Fiber acts as a physiological obstacle to energy intake by displacing energy and nutrients from the diet, increasing the expansion of the stomach leading to increased satiety and feelings of fullness, and reducing the absorption efficiency of the small intestine (1). High-fiber foods promote energy balance by providing a significant amount of volume and low-energy density (1).

Cancer: People who eat a greater amount of fruits and vegetables have about one half the risk of developing cancer and a lower mortality rate from cancer (22). Evidence that a high-fiber diet decreases the risk of certain cancers, including large bowel cancers (colon and rectum) and breast cancer, remains inconclusive (1). Although dietary fiber intake may not protect against colorectal cancer in prospective studies, some support exists for the protective properties of whole-grain intake (1,23). There is evidence that vegetables, fruits, and whole grains reduce the risk of chronic diseases including cancer, which provides support for the use of a high-fiber diet in reducing cancer risk (1,22).

Use in enteral formulas: Two types of enteral formulas that contain dietary fiber are currently marketed: blended formulas made from whole foods and formulas supplemented with purified fiber sources (eg, oat, pea, hydrolyzed guar gum, and sugar beet fiber) (1). Dietary fiber added to enteral formulas is thought to aid in normalizing bowel function and reduce the incidence of diarrhea. However, there is no conclusive evidence that fiber-containing enteral formulas prevent diarrhea in tube-fed patients (1). A recent addition to enteral formulas is fructooligosaccharides, which are short-chain oligosaccharides (usually 2 to 10 monosaccharide units). Because they are not digested in the upper digestive tract, fructooligosaccharides have some of the same physiologic effects as soluble fiber (24). Fructooligosaccharides are rapidly fermented by intestinal bacteria that produce short-chain fatty acids, which stimulate water and electrolyte absorption and should aid in the treatment of diarrhea. Although fructooligosaccharides are a preferred substrate for *Bifidobacteria*, they are not used by potentially pathogenic bacteria, thus helping to maintain and restore the balance of healthy gut flora (1). Fructooligosaccharides are not isolated by currently accepted methods to measure dietary fiber, so they cannot technically be called dietary fiber (5). The Association of Official Analytical Chemists has developed newer methods to analyze fructooligosaccharides (1).

Contraindications

Diverticulitis: A high-fiber diet may be contraindicated when inflammation has caused the narrowing or blockage of the intestinal lumen or during acute diverticulitis (9). However, this recommendation is based on individual tolerance.

Infants and children: The American Academy of Pediatrics does not encourage the addition of high-fiber foods to the diets of infants younger than 1 year old. High-fiber foods are filling but contain little energy, potentially causing reduced energy intakes in infants, whose stomach capacities are naturally small. According to the American Academy of Pediatrics, the daily fiber intake of children 2 years and older should be an amount equal to or greater than their age plus 5 g (1).

Phytobezoar formation: Phytobezoars are masses of vegetable matter that become trapped in the stomach. Individuals who experience decreased gastric motility or emptying, such as diabetic gastroparesis, or individuals who have undergone surgical procedures for stomach cancer or peptic ulcer disease may be susceptible (1,25,26). These individuals should be advised to avoid the following foods implicated in phytobezoar formation: apples, berries, brussels sprouts, coconuts, figs, green beans, oranges, persimmons, and potato peels or highly viscous over-the-counter fiber supplements such as glucomannan (1,25,26).

Nutritional Adequacy

The diet can be planned to meet the DRIs as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "High-Fiber Diet."
Planning the Diet

- Promote food intake patterns consistent with the 2010 *Dietary Guidelines for Americans* and MyPlate that encourage a wide variety of plant foods to achieve fiber intakes goals. Emphasize fruits, vegetables, and whole-grain breads and cereals.
- Foods made with whole-grain flours are substituted for foods made with refined flours and starches.
 - People who experience difficulty in chewing fruits and vegetables may increase fiber in their diet by consuming one or more servings daily of a high-fiber cereal, such as bran; substituting whole-wheat bread for white bread; and consuming soft or cooked fruits and vegetables.
- If unprocessed bran is consumed, it must be served thoroughly moistened and mixed with food and be incorporated gradually into the diet. One tablespoon of bran contains 4.5 g of dietary fiber. To incorporate bran into the diet, begin with 1 tsp/day and gradually increase in divided doses, as tolerated, to 4 to 6 tbsp/day. Three tablespoons of bran, consumed daily in divided doses, is adequate to promote normal bowel functioning.
- High-fiber foods should be added to the diet gradually. An increase in fiber consumption may initially generate bloating and flatulence. Patients should be advised that these conditions may occur but will generally subside as the digestive system adjusts to increased fiber consumption.
- Fiber gathers water in the colon, hence its stool-bulking property. For this reason, a high-fiber diet should also include a liberal intake of fluids, consisting of at least 8 cups or 64 oz of extra fluid per day (1). Consuming increased amounts of fiber without increasing fluid consumption can lead to the formation of hard, dry stools that are difficult to eliminate.
- Consider use of probiotics and prebiotics for patients with Irritable Bowel Syndrome (IBS) and Diverticulosis (13).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Slavin, JL. Position of the American Dietetic Association: health implications of dietary fiber. *J Am Diet Assoc.* 2008;108:1716-1731.
- Institute of Medicine's Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. National Academy of Sciences, 2002:265-334. Preprint available at: http://www.nap.edu.books/0309085373/html/index.html. Accessed September 16, 2002.
- 3. Fungwe TV, Bente L, Hiza H. USDA Center for Nutrition Policy and Promotion. *The Food Supply and Dietary Fiber: Its Availability and Effect on Health.* Available at: http://www.cnpp.usda.gov/Publications/NutritionInsights/Insight36.pdf. Accessed July 28, 2008.
- 4. Slavin JL. Dietary fiber: classification, chemical analyses, and food sources. *J Am Diet Assoc.* 1987;87:1164-1171.
- 5. Dietary Reference Intakes: Proposed Definition of Dietary Fiber. Washington, DC: National Academy Press; 2001:1-64.
- 6. Cummings JH. The effect of dietary fiber on fecal weight and composition. In: Spiller GA, ed. *CRC Handbook of Dietary Fiber in Human Nutrition*. 2nd ed. Boca Raton, Fla: CRC Press; 1993:263-349.
- 7. Kurasawa S, Haack VS, Marlett JA. Plant residue and bacteria as bases for increased stool weight accompanying consumption of higher dietary fiber diets. *J Am Coll Nutr.* 2000;19:426-433.
- 8. Salzman H, Lillie D. Diverticular disease: diagnosis and treatment. Am Fam Physician. 2005;72:1229-1234.
- 9. National Digestive Diseases Information Clearinghouse. *Diverticulosis and Diverticulitis*. 2008. Available at: http://digestive.niddk.nih.gov/ddiseases/pubs/diverticulosis/index.htm. Accessed September 4, 2008.
- 10. Strate LL, Liu YL, Syngal S, Aldoori WH, Biovannucci EL. Nut, corn, and popcorn consumption and the incidence of diverticular disease. *JAMA*. 2008;300:907-914.
- 11. Marcason W. What is the latest research regarding the avoidance of nuts, seeds, corn and popcorn in diverticular disease? *J Am Diet Assoc.* 2008;108:1956.
- 12. American College of Gastroenterology Task Force on IBS. An evidence-based systematic review on the management of irritable bowel syndrome. *Am J Gastroenterol.* 2009;104 (suppl 1): S1-S35.
- 13. Douglas LC, Sanders ME. Probiotics and probiotics in dietetics practice. J Am Diet Assoc. 2008; 108:510-521.
- 14. Ong DK, Mitchell SB, Barrett JS, Shepherd S, Irving PM, Biesiekierski JR, Smith S, Gibson PR, Muir JG. Manipulation of dietary short chain carbohydrates alters the pattern of gas production and genesis of symptoms in irritable bowel syndrome. *J Gastroenterology and Hepatology*. 2010; 25: 1366-1373.
- 15. Shepherd SJ, Parker FC, Muir JG, Gibson PR. Dietary triggers of abdominal symptoms in patients with irritable bowel syndrome: randomized placebo controlled evidence. *Clin Gastroenterol Hepatol*. 2008; 6: 765-771.
- 16. Marlett JA. Sites and mechanisms for the hypocholesterolemic actions of soluble dietary fiber sources. In: Kritchevsky D, Bonfield C, eds. *Dietary Fiber in Health and Disease*. New York, NY: Plenum Press; 1997:109-121.
- 17. Marlett JA. Dietary fiber and cardiovascular disease. In: Cho SS, Dreher ML, eds. *Handbook of Dietary Fiber*. New York, NY: Marcel Dekker; 2001:17-30.
- 18. US Food and Drug Administration. Health claims: soluble fiber from certain foods and risk of heart disease. *Code of Federal Regulations*. 2001;21:101.81.
- 19. Marlett JA, Hosig KB, Vollendorf NW, Shinnick FL, Haack VS, Story JA. Mechanism of serum cholesterol reduction by oat bran. *Hepatology*. 1994;20:1450-1457.

- 20. AHA Diet and Lifestyle Recommendations Revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006;114:82-96.
- 21. American Diabetes Association. Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes Care*. 2008;31(suppl1): 61S-78S.
- 22. Craig WJ. Phytochemicals: guardians of our health. J Am Diet Assoc. 1997;97(suppl 2):S199-S204.
- 23. Schatzkin A, Houw T, Park Y, Subar AF, Kipnis V, Hollenbeck A, Leitzmann MF, Thompson FE. Dietary fiber and whole-grain consumption in relation to colorectal cancer in the NIH-AARP Diet and Health Study. *Am J Clin Nutr.* 2007;85:1353-1360.
- 24. Roberfroid M, Slavin JL. Nondigestible oligosaccharides. Crit Rev Food Science Nutr. 2000;40:461-480.
- 25. Vanderbeek PB, Fasano C, O'Malley G, *Hornstein J. Esophageal obstruction from a hygroscopic pharmacobezoar containing glucomannan. Clin Toxicol (Phila).* 2007; 45:80-82.
- 26. Gastroparesis. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed January 25, 2013.

FOOD GUIDE HIGH-FIBER DIET

This list is not meant to be all-inclusive. Only the foods highest and lowest in fiber in each category are listed in the categories Higher-Fiber and Lower-Fiber Foods, respectively.

FOOD GROUP	Emphasize HIGHER-FIBER FOODS	Minimize LOWER-FIBER FOODS
Beverages and Milk		Beverages and milk
Breads and Crackers	100% whole-wheat bread, rolls, muffins Whole-wheat crackers Bran muffins Pumpernickel and rye bread	White bread and rolls Saltine and other refined crackers
Cereals and Grains	100% and 40% bran cereal, whole- wheat and oat cereals, including puffed wheat, shredded wheat, granola,* Grape-Nuts, oatmeal,* oat bran,* rolled wheat, and Wheatena Brown rice, barley	Cereals from refined wheat flours, corn, or rice, including farina, grits, cream of rice, cornflakes, puffed rice, and crisp rice White rice, pasta, noodles
Desserts	Cake, cookies, and pastry made from white flour Ice cream, sherbet Cream or custard pies Pudding, custard Gelatin	
Fats	Peanuts,* tree nuts, seeds	Butter, margarine, oils, mayonnaise, salad dressings, gravy
Fruits	All except juices	Juices
Meat, Fish, Poultry, Cheese, Eggs, Legumes	Chili and other entrees containing legumes* Peanut hutter*	Meat, fish, poultry, eggs, cheese
Soups	Vegetable and other legume [*] soups	Broth, meat, rice, noodle soups
Sugars and Sweets	Candy made primarily from coconut, raisins, or other fruit or nuts	Hard, chocolate, or caramel candy; honey, jam, jelly, molasses, sugar, syrup
Vegetables and Potatoes	All, especially broccoli, corn, greens, legumes,* peas,* sweet potatoes, winter squash	

*Foods containing soluble fibers.

SAMPLE MENU (23 g fiber)

Breakfast	Noon	Evening	Snack
Fresh orange	Split pea soup	Cranberry relish	Milk
40% bran cereal	Roast beef sandwich on whole-	Roast turkey	Grapes
Soft cooked egg	wheat bread with lettuce and	Sweet potatoes	
Whole wheat toast	tomato	Broccoli	
Margarine	Relish plate	Tossed salad with dressing	
Jelly	Apricot halves	Whole wheat bread	
Milk	Coffee or tea	Margarine	
Coffee		Fresh apple	
		Coffee or tea	

DIETARY FIBER CONTENT OF FOODS

Food Group	<1 g	1-1.9 g	2-2.9 g	3-3.9 g	4-4.9 g	5-5.9 g	>6 g
Breads	Bagel (¹ / ₂) Dinner roll French bread Hamburger/hot dog roll (¹ / ₂) Hard roll Italian bread Pancake (1) Graham crackers (2) White bread	Whole-wheat pita bread (5 inches) Raisin Rye Tortilla	Pumpernickel Bran muffin			Rye wafers (3)	
Cereals (3/4 cup cooked; 1 oz dry, unless noted)	Puffed rice Puffed wheat Rice Krispies	Oatmeal Cornflakes Granola Grits	Grape-Nuts Shredded Wheat Wheat Chex	Cheerios Raisin bran Wheat germ Wheaties	Unprocessed bran 40% bran flakes Oat bran Ralston cereal		All-Bran Bran Buds
Pasta, rice	Macaroni Spaghetti Egg noodles Rice, white	Rice, brown					
Vegetables and legumes (½ cup cooked unless noted)	Cabbage, raw Bean sprouts Celery, raw Cucumber, raw Green pepper Lettuce, raw Mushrooms, raw	Asparagus Brussels sprouts Cabbage Carrots, raw Cauliflower Green beans Summer squash Tomatoes, raw Turnips Zucchini squash	Broccoli Carrots Corn Mixed vegetables Okra Potato, no skin Spinach	Baked beans Sweet potato	Baked potato, no skin Kidney beans Lima beans Peas Winter squash	Chickpeas Pinto beans	Lentils
Fruits, canned (unless noted)	Grapefruit, raw Grapes, raw Pineapple Plums Watermelon Fruit juices (including nectars)	Applesauce Apple slices Apricots Cantaloupe Cherries, raw or cooked Cherries, raw Fruit cocktail Peaches Pineapple, raw Prunes (3) Raisins, dried (2 tbsp) Strawberries, raw	Banana Nectarine Papaya Pears	Apple, raw Dates (5) Mango Orange, raw	Pear, raw Raspberries, raw		
Miscellaneous	Olives	Filberts Popcorn Walnuts	Almonds Avocado Fruit pie Peanuts Peanut butter				

Source: Pennington J. Bowes and Church's Food Values of Portions Commonly Used. 17th ed. Philadelphia, Pa: JB Lippincott; 1998.

Dietary Fiber Content of Common Foods

G	rams		Gra
Beverages and Milk		Cheerios	3
Milk, white, nonfat or low-fat	0.0	Cornflakes	
(8 oz)		Granola (1/3 cup)	
Buttermilk	0.0	Grape-Nuts	2
Coffee, tea	0.0	Oat bran (1/3 cup)	4
<u>Bread</u>		Puffed rice (1 cup; 14 g)	(
Bagel (1 whole)	1.6	Puffed wheat (1 cup; 14 g)	(
Bran muffin (1 average size)	2.5	Raisin bran	4
Biscuit	0.5	Rice Krispies	(
Cornbread	1.0	Shredded wheat	
Dinner roll (1)	0.9	Wheaties	3
Doughnut	0.7	Wheat germ $(1/4 \text{ cup})$	3
French bread (1 slice)	0.8	Pasta, Rice, etc (½ cup cooked)	
Hamburger/hot dog roll (½)	0.6	Barley	3
Hard roll (1 white)	0.9	Macaroni; spaghetti	(
Italian bread (1 slice)	0.9	Rice, white	(
Pancakes	1.0	Rice, brown	
Pita bread (5 inches)	1.0	Desserts	
Pumpernickel bread (1 slice)	2.1	Cake, plain, iced (1/12 of	9 (
Raisin bread (1 slice)	1.1	inches)	
Rye bread (1 slice)	1.9	Carrot cake (1/12 of 9 inches)	
Taco shell (1)	1.3	Coffee cake (1/6 of 16 oz)	(
Tortilla, flour	1.2	Cookies (1 oz)	(
White bread (1 slice)	0.6	Gelatin dessert	(
Whole-wheat bread (1 slice)	1.9	Ice cream (½ cup)	(
Crackers		Pie, fruit $(1/8 \text{ of } 9 \text{-inch pie})$	2
Graham	0.25	Pudding	(
Rye wafers (3)	5.7	Yogurt (8 oz) plain or fruit	(
Saltines (2)	0.1	Fats and Nuts	
Triscuits (7)	4.0	Avocado (1/4)	2
Wheat Thins (24)	1.0	Butter; margarine (1 tsp)	(
<u>Snacks</u>		Cream, dairy and nondairy,	all (
Corn chips (1 oz)	1.4	types	
Popcorn (1 cup)	1.0	Mayonnaise; smooth salad	(
Potato chips (1 oz)	1.0	dressing (1 tbsp)	
Pretzels (1 oz)	0.9	Oil; shortening (1 tbsp)	(
Cereals and Grains		Olives (5 medium)	(
(Cooked cereal 3/4 cup unless no	oted)	Tartar sauce; thousand island	(
Cream of rice	1.0	dressing (1 tbsp)	
Farina	2.4	<u>Nuts</u> (1 oz)	
Grits	1.5	Almonds, roasted	3
Oatmeal	3.0	Filberts	1
Ralston	4.6	Peanuts, roasted and salted	2
<u>Dry, Ready-to-Eat Cereal</u>		Peanut butter, chunky (2 tbsp)	2
(1 oz unless noted)		Walnuts (1 oz)	1
All-Bran	10.0	Fruits and Juices	
Bran Buds (1/3 cup)	12.0	(½ cup portion unless noted)	
Bran, unprocessed (1 tbsp)	4.6	Apple, raw with peel	3
40% bran flakes	4.0	(2½-inch diameter)	
		Apple, canned, sliced	1

Gra	ams		Grams
	3.0	Applesauce, canned	1.0
	1.1	Apricots, canned (3 halves)	1.4
	1.8	Banana (1 medium)	2.7
	2.5	Cantaloupe (1/4 melon)	1.3
	4.8	Cherries, sweet (10)	1.6
	0.1	Cherries, canned	1.9
	0.5	Dates, dried (5)	3.1
	4.0	Fig, dried (3)	6.8
	0.5	Fruit cocktail	1.2
	2.8	Grapefruit (½)	1.4
	3.0	Grapefruit sections, canned	0.5
	3.8	Grapes, European (10)	0.8
		Honeydew melon	0.5
	3.0	Mandarin oranges	0.9
	0.9	Mango (1 medium)	3.7
	0.5	Nectarine (1 medium)	2.2
	1.7	Orange (1 small)	3.1
		Papaya (½ medium)	2.5
9	0.5	Peaches, canned (2 halves)	1.6
		Peach, raw (1 medium)	1.7
	1.4	Pear, canned (2 halves)	2.0
	0.8	Pear, raw (2½ per pound)	4.0
	0.5	Pineapple, canned	1.0
	0.0	Pineapple, raw	1.8
	0.0	Plums, raw, 1 medium	1.0
	2.0	Plums, canned (3)	0.9
	0.0	Prunes (3)	1.8
	0.0	Raisins (2 tbsp)	1.6
		Raspberries, raw	4.2
	2.1	Strawberries, raw	1.6
	0.0	Tangerine	1.9
all	0.0	Watermelon	0.4
		<u>Fruit Juices</u> (½ cup)	
	0.0	Apple	0.0
		Apricot nectar	0.8
	0.0	Cranberry	0.0
	0.5	Grapefruit; orange	0.0
	0.0	Grape	0.0
		Pineapple	0.0
	•	Prune	1.3
	3.0	Meat, Fish, Poultry, Cheese, Egg	<u>s</u> 0.0
	1.7	Sugar and Sweets	0 7
	2.3	Jam; preserves (1 tbsp)	0.7
	2.0	Jelly (1 tbsp)	0.0
	1.4	Sugars; honey; syrups	0.0
		Cranberry sauce (1/4 cup)	0.7

3.7

1.7

Dietary Fiber Content of Common Foods (Cont.)

	Grams		Grams
<u>Soups (½ cup)</u>		Green pepper	0.9
Bean with bacon	7.0	Kale	1.3
Beef barley	2.0	Lentils	7.8
Beef noodle	1.0	Lettuce, iceberg (shredded)	0.5
Celery, cream of	1.0	Mixed vegetables	2.5
Cheddar cheese	1.0	Mushrooms, raw	0.4
Chicken gumbo	1.0	Mushrooms, canned	1.9
Chicken noodle	1.0	Okra	2.2
Chicken rice	0.0	Onions, raw, chopped	1.4
Chicken vegetable	2.0	Peas, green, frozen	4.4
Clam chowder, Manhattan	2.0	Pinto beans	5.5
Clam chowder, New England	1.0	Potato, baked, with skin	4.6
Corn chowder	2.0	Potato, boiled (140 g)	2.3
Minestrone	4.0	Potato, french fried (20)	1.6
Mushroom, cream of	1.0	Potato, mashed	1.9
Pea, green	2.5	Radishes	0.7
Pea, split	5.0	Sauerkraut	2.9
Potato, cream of	1.0	Spinach	2.8
Tomato	2.0	Spinach, raw	0.8
Turkey noodle	1.0	Squash, summer	1.3
Vegetable	2.0	Squash, winter	4.5
Vegetable beef	2.0	Squash, zucchini	1.8
Vegetables		Sweet potatoes, mashed	3.0
(¹ / ₂ cup portion cooked or raw unl	ess noted)	Tomato, raw (1medium)	1.4
Asparagus	1.4	Tomato juice	0.7
Baked beans (1/3 cup)	3.0	Tomato sauce	1.7
Bean sprouts	0.6	Turnips	1.6
Beans, green, fresh-cut	2.0	Turnip greens	2.5
Beans, green, cut	1.3	<u>Miscellaneous</u>	
Beans, kidney	4.9	Ketchup (1 tbsp)	0.2
Beans, lima, baby	4.9	Mustard (1 tsp)	0.1
Beans, navy	6.7	Pickle, dill (1 medium)	0.3
Beets	1.4	Pickle, sweet (4 slices)	0.5
Broccoli, raw (1 spear)	1.3	Pickle relish, sweet (1tbsp)	0.5
Broccoli, spears	2.8		
Broccoli, chopped	2.3		
Brussels sprouts	2.0		
Cabbage, cooked	1.7		
Cabbage, raw	0.8		
Carrots, cooked	2.6		
Carrots, raw (1 medium)	2.2		
Cauliflower, cooked	1.7		
Cauliflower, raw	1.3		
Celery, raw (1 stalk)	0.7		
Chard	1.7		
Chickpeas (garbanzo beans)	5.3		
Collard greens	2.4		
Coleslaw	0.9		
Corn kernels	2.3		
Cowpeas (black-eyed peas)	3.7		
Cucumber, raw	0.4		

Source: Pennington J. Bowes and Church's Food Values of Portions Commonly Used. 17th ed. Philadelphia, Pa: JB Lippincott; 1998.

GASTROINTESTINAL SOFT DIET

Description

The Gastrointestinal Soft Diet limits most raw, highly seasoned, and fried foods. The diet contains only moderate amounts of fiber.

Indications

This diet is used as a transitional diet for patients who have undergone surgery that irritates or causes major discomfort to the gastrointestinal tract.

Contraindications

The diet does not necessarily limit fat or the size of meals and may be counterproductive in patients with gastro- esophageal reflux (see Section III: Gastroesophageal Reflux).

The diet is low in fiber and may be contraindicated in disorders, such as diverticulosis, requiring a liberal fiber intake. See Section 1D: Fiber-Restricted Diets and High-Fiber Diet.

The diet may inappropriately limit mealtime variety and thereby limit enjoyment and oral intake.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

Order as "Gastrointestinal (GI) Soft Diet." If between-meal feedings are required, they should be specifically ordered.

SAMPLE MENU

Breakfast	Noon	Evening
Orange juice	Roast beef	Cream of tomato soup
Oatmeal	Whipped potatoes	Baked chicken
Scrambled egg	Cooked carrots	Steamed rice
Toast	Plain roll	Green beans
Margarine	Margarine	Plain roll
Jelly	Sugar cookies	Margarine
Milk	Iced Tea	Sliced peaches
Coffee		Milk

FOOD GUIDE-GASTROINTESTINAL SOFT DIET

Since the food tolerances of patients with gastrointestinal disorders and symptoms can vary considerably, attention should be given to individual food tolerances.

FOOD GROUP	FOOD ALLOWED	FOODS EXCLUDED
Beverages and Milk	Milk and milk drinks Cereal beverages Carbonated beverages Coffee, tea	Alcohol
Breads and Crackers	White, seedless rye, fine whole-wheat bread Plain crackers Graham crackers	Coarse whole-grain breads Breads with seeds, nuts, or raisins Highly seasoned crackers
Cereals and Grains	Cooked and dry cereals unless listed as excluded Plain spaghetti, macaroni, noodles, rice	Bran cereals Cereals with raisins Brown or wild rice
Desserts	Plain cake, cookies, pudding, custard, ice cream, sherbet, gelatin, fruit whips	Pastries, pies, desserts containing nuts, coconut, dried fruits, fruit with seeds or tough skins
Fats	Butter Cream; cream sauce Bacon Margarine Mayonnaise; mild salad dressing	Fried foods Gravy Nuts Olives Spicy salad dressings
Fruits and Juices	All fruit juices Avocado Banana Grapefruit and orange sections without membrane Baked peeled apple; applesauce Canned: apricots, cherries, peaches, pears, pineapple Peeled ripe peaches or pears	Raw fruit not listed as allowed Dried fruits Fruits with edible seeds or tough skins
Meat, Fish, Poultry, Cheese, Eggs, Legumes	Meat, fish, or poultry, not fried Plain cheeses Eggs, except fried Smooth peanut butter	Fried meat, fish, or poultry Highly seasoned cold cuts or sausage Fried eggs
Soup	Cream soups made from foods allowed; meat, rice, noodle soups	Vegetable soups unless made from foods allowed
Sugar and Sweets	Sugar, syrup, honey, clear jelly; plain, sugar candy in moderation	Jam, marmalade, and candies that contain tough skins, seeds or nuts
Vegetables and Potatoes	Tomato juice Cooked asparagus, beets, carrots, green or wax beans, green peas, mushrooms, potatoes, spinach, summer squash, sweet potatoes, tomatoes, winter squash	Raw vegetables All other cooked vegetables Deep-fried vegetables
Miscellaneous	Salt, allspice, cinnamon, paprika, herbs, flavoring extracts, ketchup	Red, black, white pepper; horseradish, mustard, pickles, popcorn, potato chips

I. NORMAL NUTRITION AND MODIFIED DIETS E. Pediatric Diets

Nutrition Management of the Full-Term Infant	E-1
Table E-1: Nutrient Comparison of Breast Milk, Formula, and Cow's Milk	E-2
Table E-2: Infant Feeding Guidelines	E-3
Infant Formula Comparison Chart	E-4
Nutrition Management of the Toddler and Preschool Child	E-7
Table E-3: Food Groups and Recommended Portion Sizes	E-8
Nutrition Management Of The School-Aged Child	E-9
Table E-4: Food Groups and Recommended Portion Sizes	E-10
Nutrition Management Of The Adolescent	E-12
Ketogenic Diet	E-14

NUTRITION MANAGEMENT OF THE FULL-TERM INFANT

Growth and nutrient needs during the first year of life exceed those at any other stage of the life cycle. However, since the organ systems are not fully developed in infancy, special considerations should be given to when and how foods are introduced. While supplying sufficient nutrients to promote growth and maintenance, it is important for the infant's diet to not exceed the requirements or capabilities of the infant's digestive or excretory systems. The optimal feeding regimen is to exclusively breast-feed for six months and breast-feed with complementary foods for at least twelve months (1,2).

Breast-feeding

Breast-feeding is the optimal way to provide food for the health, growth, and development of the infant. In addition to its unique nutrient composition, it offers immunologic and psychosocial benefits that are not provided by any other feeding substance. Human milk is unique in that it provides docosahexaenoic acid (DHA), a long-chain fatty acid that is essential for infant brain and eye development (3.4). Lactoferrin, an iron-binding protein found in whey of human milk, has been observed to inhibit the growth of certain iron-dependent bacteria in the gastrointestinal tract (5). Infants who are breast-fed usually have fewer gastrointestinal and nongastrointestinal infections, including otitis media, pneumonia, bacteremia, diarrhea, and meningitis. They have fewer food allergies and a reduced risk of certain chronic diseases throughout life (eg, type 1 diabetes, lymphoma, and Crohn's disease) (1,2,6-9).

Infants nursed by a vegan mother may be at risk for vitamin B_{12} deficiency. The dietary vitamin B_{12} intake of the mother should be assessed to determine adequacy. Vegan mothers should be instructed to supplement their diets with foods fortified with vitamin B_{12} (10).

Contraindications for Breast-Feeding

Infants with certain inborn metabolism errors, such as phenyalamine, maple syrup urine disease, or galactosemia should not be breastfed (2).

Breast-feeding is contraindicated for women who:

- use addictive drugs, such as cocaine, marijuana, and phencyclidine (PCP)
- drink more than a minimal amount of alcohol
- receive certain therapeutic or diagnostic agents, such as radiation or chemotherapy (11,12)
- are infected with the human immunodeficiency virus (HIV) (2)

Women should not breast-feed when they are receiving certain therapeutic medications. Not only is toxicity to the infant a concern, but research has indicated that some medications affect the infant's metabolism. In addition, some agents (eg, bromocriptine) decrease milk production. Whereas most medications are considered compatible with breast-feeding, there are substances for which the risk of toxicity to the infant is considered to be greater than the benefit to the mother. The most frequently used of these medications to be aware of include (12):

- amphetamine
- bromocriptine
- cyclophosphamide
- cyclosporine
- doxorubicin
- ergotamine
- lithium
- methotrexate
- nicotine
- phenindione

Formula Feeding

The use of commercially prepared infant formula is an acceptable alternative to breast-feeding. These formulas are designed to approximate the composition of human milk as closely as possible. Most commercial infant formulas are composed of milk proteins or soy protein isolate.

Nutrition Management of the Full-Term Infant

Milk-based formulas are generally appropriate for use with the healthy full-term infant. Standard formulas have a 60:40 whey-to-casein ratio, which is desirable in a formula; they provide 20 kcal/oz. Breast milk yields an 80:20 whey: casein ratio with about the same number of calories. Soy-based formulas are often used from birth to prevent allergic disease in infants with a strong family history of allergies (13).

As long as the commercially prepared infant formula with iron is delivered in the appropriate volumes for a term infant, it is not necessary to supplement with additional vitamins or iron. The American Academy of Pediatrics recommends that formula-fed infants be given an iron-fortified cereal or supplemented with iron by 6 months of age. When food is introduced during the second 6 months of life, the combination of food and formula will meet the infant's nutrient requirements (14). Fluoride supplementation may be required if powdered or concentrated formula is used and if the community water supply contains less than 0.3 ppm of fluoride. Fluoride should not be supplemented before 6 months of age (2).

Therapeutic or specialized formulas are indicated for use with premature infants, as well as infants with cow's milk allergy or intolerance, intact protein allergy, or generalized malabsorption. Premature-infant formulas are modified in terms of their energy, macronutrient, and micronutrient content in order to meet the specialized physiologic and gastrointestinal needs of these infants. Premature infants should be discharged home on premature-infant formula and remain on it until 12 months of age. Human milk fortifiers (HMFs) are specially designed to be added to expressed breast milk for the premature infant. HMFs provide protein, energy, calcium, phosphorus, and other minerals needed for rapid growth and normal bone mineralization in the premature infant. Hydrolysate formulas are indicated for the nutrition management of infants with allergies to intact protein from either cow's milk or soy. These hydrolyzed formulas, some of which also contain part of the fat as medium chain triglycerides, may also be used for infants with generalized malabsorption of both protein and fat (eg, short gut syndrome and cystic fibrosis). Fat-modified formulas are indicated for nutrition management of infants with steatorrhea due to their limited bile salt pool, such as those with biliary atresia or other forms of malabsorption or intolerance. Medical formulas for various disorders of inborn errors of metabolism are also available from the major formula manufacturers for disorders such as phenylketonuria and maple syrup urine disease.

Water

If the infant consumes an adequate amount of breast milk, formula, or both, the infant will have an adequate intake of water.

Cow's Milk

Cow's milk should not be introduced until a child is 1 year of age. The nutrient composition of cow's milk varies substantially from that of human milk. Feedings with cow's milk causes a markedly high renal solute load due to its protein and sodium content, and infants are not generally able to concentrate urine well. The ingestion of cow's milk increases the risk for gastrointestinal blood loss and allergic reactions. Whole milk can be introduced after the first year and continued through the second year. After the second year, reduced-fat milk can be served (2).

Products per 100 cc	Energy (kcal)	Protein (g)	Calcium (mg)	Phosphorus (mg)	Iron (mg)	Sodium (mg)
Breast milk	70	1.0	32	14	0.3	8
Milk-based formula (20 kcal/oz)	67	1.5	42-51	28-39	1.2	15-20
Soy-based formula (20 kcal/oz)	67	1.8-2.1	60-71	42-51	1.2	20-30
Whole cow's milk (homogenized)	64	4.9	120	95	Trace	51

Table E-1: Nutrient Comparison of Breast Milk, Formula, and Cow's Milk

Introduction of Solid Food

There is no nutritional need to introduce solid food to infants during the first 6 months of age (1,2). The infant's individual growth and development pattern is the best indicator of when to introduce semisolid and solid foods. Generally, an infant will double his birth weight and be able to sit upright without support by the time semisolid foods are introduced. By 4 to 5 months, the infant has the ability to swallow nonliquid foods. If

solids are introduced before this time, these foods may displace breast milk or formula and the infant may receive inadequate energy and nutrient needs.

No specific schedule of introduction of food other than breast milk or formula must be followed, but certain recommendations exist:

- Iron-fortified infant cereal is commonly suggested as the first food offered. Start with a few spoonfuls of a single-grain, iron-fortified infant cereal such as rice, once or twice a day.
- Introduce single-ingredient foods, one at a time, so that the offending food can be identified if an adverse reaction occurs.
- Vegetables might be accepted more readily if introduced before fruits, since fruits taste sweeter.
- Allow at least 3 days between the introduction of each new food.
- Begin with small amounts of foods, offering seconds as necessary.
- Avoid early introduction of the following common allergens: egg white, cow's milk, citrus, wheat, chocolate, fish, shellfish, tree nuts, and nut butters (eg, no peanut butter until 18 to 24 months of age) because susceptible infants with a family history of allergies may experience allergic reactions.
- Take care to avoid spoilage of home-prepared foods and jars of food once they are opened. Do not feed infants directly from the jar, as saliva added to the jar causes faster spoilage.
- Select appropriate solid foods that require minimal chewing. Foods such as hot dogs, peanuts, grapes, berries, raw carrots and sliced apples, raisins, potato or corn chips, popcorn, seeds, round, hard candies, and gum may cause choking and aspiration in infants and children.

Age (months)										
Food	0-2	2-4	4-6	6-8	9-10	11-12				
Human milk/ formula (oz)	18-28	25-32	27-45	24-32	24-32	24-32				
Iron-fortified cereal (tbsp)			4-8	4-6	4-6	4-6				
Zwieback, dry toast				1	1	1-2				
Vegetable, plain, strained (tbsp)				3-4	6-8	7-8 (soft, cooked, chopped)				
Fruit, plain strained (tbsp)				3-4	6-8	8 (soft, chopped)				
Meat, plain, strained (tbsp)				1-2	4-6	4-5 (ground or chopped)				
Egg yolk (tbsp)					1	1				
Fruit juice (oz)				2-4	4	4				
Potato, rice, noodles (tbsp)						8				

Table E-2: Infant Feeding Guidelines

References

- 1. Position of the American Dietetic Association: breaking the barriers to breast feeding. *J Am Diet Assoc.* 2001;101:1213-1220.
- 2. American Academy of Pediatrics Committee on Nutrition. Breast feeding and the use of human milk (policy statement). *Pediatrics*. 1997;100(6):1035-1039.

3. Jorgensen MH, Hernell O, Lund P, Holmer G, Fleisher-Michaelsen K. Visual acuity and erythrocyte docosahexaenoic acid status in breast-fed and formula-fed infants during the first four months of life. *Lipids*. 1996;31:99-105.

4. Makrides M, Neumann MA, Byard RW, Simmer K, Gibson RA. Fatty acid composition of brain, retina, and erythrocytes in breast- and formula-fed infants. *Am J Clin Nutr*. 1994;60:189-194.

- 5. Lawrence RA. *Breastfeeding: A Guide for the Medical Profession*. 4th ed. St Louis, Mo: Mosby-Year Book; 1994.
- 6. Forsyth JS. The relationship between breast-feeding and infant health and development. *Proc Nutr Soc.* 1995;54:407-418.
- 7. Bruno G. Prevention of atopic disease in high risk babies (long-term follow-up). *Allergy Proc.* 1993;14:181-186.
- 8. Koletzko S. Role of infant feeding practices in the development of Crohn's disease in childhood. *Br Med J.* 1989;298:1617-1618.

9. American Academy of Pediatrics Work Group on Cow's Milk Protein and Diabetes Mellitus. Infant feedings and their possible relationship to the etiology of diabetes mellitus. *Pediatrics*. 1994; 94:752-754.

- 10. Darby ML, Loughead JL. Neonatal nutritional requirements and formula composition: a review. J Obstet Gynecol Neonatal Nurs. 1996;25:209-217.
- 11. Anderson PO. Drug use during breast-feeding. *Clin Pharm.* 1991;10:594-624. (Cited in: Nutrition Management in the Full-Term Infant. *Pediatric Manual of Clinical Dietetics.* Chicago, Ill: The American Dietetic Association; 1997.)
- 12. American Academy of Pediatrics Committee on Drugs. The transfer of drugs and other chemicals into human milk. *Pediatrics*. 1994;93:137-150.
- 13. Iyngkaran N, Yadav M, Looi LM. Effect of soy protein on the small bowel mucosa of young infants recovering from acute gastroenteritis. *J Pediatr Gastroenterol Nutr*. 1988;7:68-75.
- 14. American Academy of Pediatrics Committee on Nutrition. Iron supplementation for infant formulas (policy statement). *Pediatrics*. 1999;104(1):119-123.

Man				Infa	ant For	mula (Con	npa	riso		nart	104		.1.						
ual				Nutrient Co	<u>ontent</u>	or inta	nt r		nuia	IS (per	10	J Ca	II)						
of	Formula	Indications	Calorie/Oz	СНО	Protein	Fat	CI	10	Fa	it ov	Prot	ein	Na	K	Minera	lls mg	C 2	F -	OSM mOom///m	
Clinic							g	of cal	g	of cal	g	of cal	Na	n	CI	Р	Ca	ге	H2O	mOsm
al N	Cow's Milk-Based					•														
Nutritio	Breast Milk	Normal infant feeding	20	Lactose	Lactalbumin 65% Casein 35%	High in oleic; low in volatile fatty acids	9.9	40	5.8	52	2.1	8	37	85	82	19	37	0.18	343	24.5
n Management	Cow's Milk, Whole	Normal feeding >1yr old	19	Lactose	Casein 81% Lactalbumin 19%															
	Enfamil PREMIUM	Normal infant feeding	20	Lactose	Nonfat milk	Palm olein, soy, coconut, sunflower oils	11.2	43.5	5.3	48	2.1	8.5	27	108	63	43	78	1.8	300	19.1
	Enfamil A.R.	Need for thickened formula	20	Lactose, rice starch, maltodextrin	Nonfat milk	Palm olein, soy, coconut, sunflower oils	11	44	5.1	46	2.5	10	40	108	75	53	78	1.8	230	23
E-4	Enfamil Gentlease	Reduced lactose	20	Corn syrup solids	Partially hydrolyzed nonfat milk and whey protein concentrate	Palm olein, soy, coconut, sunflower oils	10.8	43	5.3	48	2.3	9	36	108	63	46	82	1.8	230	21
-	Similac Advance	Normal infant feeding	20	Lactose, Galactooligosaccharides (GOS)	Nonfat milk, whey protein concentrate	Safflower, soy coconut oils	10.7	43	5.6	49	2.07	8	24	105	65	42	78	1.8	310	18.7
	Similac Advance Organic	Normal infant feeding	20	Organic Corn Maltodextrin, Organic Lactose, Organic Sugar	Organic nonfat milk	Organic sunflower, soy, coconut oils	10.6	42	5.5	49	2.07	8	24	105	65	42	78	1.8	225	18.8
	Soy-Based				1														1	
	Enfamil Prosobee	Normal infant feeding – soy, lactose and sucrose free	20	Corn syrup solids	Soy protein isolate	Palm olein, soy, coconut, sunflower oils	10.6	42	5.3	48	2.5	10	36	120	80	69	105	1.8	180	23
Copyrig	Similac Sensitive Isomil Soy (concentrate)	Normal infant feeding – soy	20	Corn syrup solids, sugar	Soy protein isolate, L- methionine	High Oleic Safflower, Soy oil, Coconut Oil	10.4	41	5.46	49	2.45	10	44	108	62	75	105	1.8	200	154.5
ht © 201	Similac Sensitive	Lactose intolerance	20	Corn Maltodextrin, Sugar, Galactooligosaccharides (GOS)	Milk protein isolate	Safflower, soy coconut oils	11.1	43	5.4	49	2.14	9	30	107	65	56	84	1.8	200	19.9
3 (Premature (NICU only)	-									-									
Compass (All rights	Enfamil Premature 20 Cal Low Iron (RTF)	Prematurity	20	Corn syrup solids, lactose	wontat milk, whey protein concentrate	MCT oil, soy, sunflower, safflower oil	11	44	5.1	44	3	12	58	98	90	83	165	0.5	240	27
Group, Inc 3 reserved	Enfamil Premature 20 Cal Iron Fortified (RTF)	Prematurity	20	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, sunflower, safflower	11	44	5.1	44	3	12	58	98	90	83	165	1.8	240	27
• •	Enfamil 24 Premature	Prematurity	24	Corn syrup solids,	Nonfat milk,	MCT oil,	11	44	5.1	44	3	12	58	98	90	83	165	0.5	300	27

			Inf	ant For	mula (Con	npa	riso	n Cl	har	t								
			Nutrient C	ontent	of Infa	nt F	For	nula	ıs (per	10	0 ca	al)						
Formula	Indications	Calorie/Oz	СНО	Protein	Fat	C	НО	Fa	it N	Pro	tein		- /	Minera	lls mg			OSI	M
						g	% of cal	g	% of cal	g	% of cal	Na	к	CI	P	Са	Fe	mOsm/Kg H2O	PRSL, mOsm
24 Cal Low Iron (RTF)			lactose	whey protein concentrate	soy, sunflower, safflower oil														
Enfamil 24 Premature 24 Cal Iron Fortified (RTF)	Prematurity	24	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, sunflower, safflower oil	11	44	5.1	44	3	12	58	98	90	83	165	1.8	300	27
Enfamil Premature High Protein 24 Cal (RTF)	Prematurity	24	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, sunflower, safflower oil	10.5	42	5.1	44	3.5	14	58	98	90	83	165	1.8	300	30
Similac Special Care 20 with Iron (RTF)	Prematurity	20	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, coconut oils	10.3	41	5.4	47	3	12	43	129	81	100	180	1.8	235	27.8
Similac Special Care 24 with Iron (RTF)	Prematurity	24	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, coconut oils	10.3	41	5.4	47	3	12	43	129	81	100	180	1.8	280	27.8
Similac Special Care 24 High Protein (RTF)	Prematurity	24	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, coconut oils	10	40	5.4	47	3.3	13	43	129	81	100	180	1.8	280	29.5
Similac Special Care 30 with Iron (RTF)	Prematurity, formula or breast milk supplement	30	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, coconut oils	7.7	31	6.6	57	3	12	43	129	81	100	180	1.8	325	27.8
Premature Discharge/Tr	ansitional	•																	
Enfamil EnfaCare	Premie discharge formula	22	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	Sunflower, safflower, soy, coconut oils, MCT oil	10.4	42	5.3	42	2.8	11	37	105	78	66	120	1.8	310	25
Similac Expert Care Neosure	Premie discharge formula	22	Corn syrup solids, lactose	Nonfat milk, whey protein concentrate	MCT oil, soy, safflower, coconut oils	10.1	40	5.5	49	2.8	11	33	142	75	62	105	1.8	250	25.2
Human Milk Fortifiers	Promoturity	n/2	Corp syrup colida	Milk protoin	MCT all	<0.4	6	1	62	1 1	22	16	20	10	50	00	1 4 4	35	0.0
Fortifier (powder) per 4 packets	Frematurity	11/a	Com syrup solids	whey protein isolate	soybean oil	<0.4	Ö		02	1.1	32	10	29	13	50	90	1.44	30	9.0
Similac Human Milk Fortifier (powder) per 4 packets	Prematurity	n/a	Corn syrup solids	Nonfat milk, whey protein concentrate	MCT oil	1.8	51	0.36	23	1.0	28	12	63	33	79	138	0.42	95	10.6
Nutramigen	Cow's Milk Protein Allergy	20	Corn syrup solids, modified corn starch	Casein hydrolysate (from milk)	Palm olein, soy, coconut, sunflower oils	10.3	41	5.3	48	2.8	11	47	110	86	52	94	1.8	260-320	25
Nutramigen with Enflora LGG	Allergy	20	Corn syrup solids, modified corn starch	Casein hydrolysate (from milk)	Palm olein, soy, coconut,	10.3	41	5.3	48	2.8	11	47	110	86	52	94	1.8	300	25

Manual of Clinical Nutrition Management

E-5

Copyright © 2013 Compass Group, Inc. All rights reserved.

			Infa	ant For	mula	Con	npa	riso	n C	har	t								
			Nutrient Co	ontent	of Infa	int I	For	mula	as (per	10	0 ca	l)						
Formula	Indications	Calorie/Oz	СНО	Protein	Fat	С	но	Fa	at	Pro	tein			Minera	ls mg			OS	М
	-					g	% of cal	g	% of cal	g	% of cal	Na	к	CI	P	Са	Fe	mOsm/Kg H2O	PRSL, mOsm
					sunflower oils														
Pregestimil	Fat malabsorption	20	Corn syrup solids, modified corn starch	Casein hydrolysate (from milk)	MCT oil, soy, corn, safflower or sunflower oils	10.2	41	5.6	41	2.8	11	47	110	86	52	94	1.8	290-320	25
Similac Expert Care Alimentum	Fat malabsorption	20	Corn maltodextrin, sugar	Casein Hydrolysate, L-Cystine, L- Tyrosine, L- Tryptophan	MCT oil, safflower, soy oils, DATEM	10.2	41	5.5	48	2.75	11	44	118	80	75	105	1.8	320	25.3
Nutramigen AA	Protein allergy	20	Corn syrup solids, modified tapioca starch	Amino acids	Palm olein, soy, coconut, sunflower oils	10.3	41	5.3	48	2.8	11	47	110	86	52	94	1.8	350	25
EleCare	Allergy	20	Corn syrup solids	Free L- amino acids	MCT oil, safflower, soy oils	10.7	43	4.8	42	3.1	15	45	150	60	84	116	1.5	350	28
Neocate Infant	Allergy, GI 5% MCT oil	20	Corn syrup solids	Free L- amino acids	MCT oil palm, coconut, sunflower oils	11.7	47	4.5	41	3.1	12	37	155	77	93	124	1.8	375	Not noted
Neocate Infant DHA and ARA	33% MCT oil	20	Corn syrup solids	Free L- amino acids	Safflower, coconut, soy oils	11.7	47	4.5	41	3.1	12	37	155	77	93	124	1.5	375	Not noted
Similac PM 60/40	Lower phosphorus and potassium content	20	Lactose	Whey Protein Concentrate, Sodium Caseinate	Safflower, soy, and coconut oils	10.2	41	5.6	50	2.2	9	24	80	59	28	56	0.7	280	18.3
Enfaport	Chylothorax, LCHAD Deficiency	30	Corn syrup solids	Calcium caseinate and sodium caseinate (from milk)	MCT oil and soy oil	10.2	41	5.4	45	3.5	14	30	115	87	52	94	1.8	280	28
Portagen	Chylothorax	30	Corn syrup solids, sugar	Sodium caseinate	MCT and corn oils	11.5	46	4.7	40	3.5	14	55	126	87	70	94	1.8	350	33
Rehydration Solutions																-			
Enfamil Enfalyte per 8 ounces	Light cherry flavor	3.4	Rice syrup solids	None	None	7.2		0		0		12 mEq	6 mEq	10.8 mEq				170	
Peulaiyte per 8 ounces	Unflavored	3	Dextrose	INONE	ivone	5.9		U		U		mEq	4.7 mEq	8.3 mEq				250	

Manual of Clinical Nutrition Management

E-6

Copyright © 2013 Compass Group, Inc. All rights reserved.

NUTRITION MANAGEMENT OF THE TODDLER AND PRESCHOOL CHILD

Description

The Regular Diet for the Toddler (1 to 3 years of age) and the Preschool Child (4 to 5 years of age) includes a wide variety of foods to promote optimal growth and development. The diet consists of foods of different textures, tastes, and colors provided throughout the day. Snacks may be required to meet the nutrient needs, since the toddler and preschooler have small stomach capacities.

Indications

The diet is served when specific dietary modifications are not therapeutically required.

Nutritional Adequacy

The diet can be planned to meet the Dietary Reference Intakes (DRIs) for the specific age as outlined in Section IA: Statement on Nutritional Adequacy. Actual nutrient requirements may vary widely among children of the same age, depending on the rate of growth and stage of development. Critical nutrition concerns of US children include excessive intake of dietary fat, especially saturated fats as well as inadequate intakes of foods rich in calcium, fiber, vitamin E, folate, iron, magnesium, and potassium (1). The most recent prevalence estimates from the National Health and Nutrition Examination Survey 2003-2004 indicate that 33.6 % of individuals age 2 to 19 years were at risk of overweight and 17.1% were overweight compared to 28.2% and 13.9 % respectively in 1999-2000 (1,2). This trend has led for the need to broaden dietary guidance from not only focusing on under-consumption but also overconsumption and decreased energy expended as a result of decreased physical activity (1). The attainment of optimal health by improving the quality of the diet an increasing physical activity will promote decreases in chronic disease in children 2 years and older (1).

How to Order the Diet

Order as "Pediatric Regular Diet" or "Regular Diet for Age _____." The age of the patient will be taken into consideration in implementing the diet order. Any specific instructions should be indicated.

Planning the Diet

Energy needs vary with the growth rate, body size, and physical activity of the child. The average daily energy requirement for ages 1-3 years is 1046 kcal for males and 992 kcal for females (3). The estimated daily energy needs for ages 4 to 5 years is 1742 kcal for males and 1642 kcal for females (3). The Institute of Medicine's Food and Nutrition Board have established acceptable macronutrient distribution ranges (ADMR) for children and include 45 to 65% of total calories from carbohydrates, 5 to 20% of total calories from protein for young children, and 30% to 40% of total calories from fat for 1 to 3 years and 25% to 35% of total calories from fat for 4 to 18 year olds (1,3).

The recommended protein (RDA) intake is 13 g/day (or 1.1 g/kg) for 1- to 3-year-olds and 19 g/day (or 0.95 g/kg) for 4- or 5-year-olds (3). Adequate protein intake may be difficult to obtain if chewing skills are limited or milk intake is inadequate. Cheese, peanut butter, and yogurt may be considered to help promote adequate protein intake. Dietary reference intakes that limit added sugars, defined as sugars and syrups that are added to food during processing or preparation, have been established (1,3). The daily intake of added sugars should be limited to 25% of the total energy consumed by a child (3). Twenty-five percent is a maximum limit; the recommended amount of added sugar in a healthy diet is 6% to 10% of total energy (1,3). Fruit juices can provide a substantial amount of sugar and energy in the diet of children. Currently it is recommended that daily fruit juice consumption be limited to 4 to 6 ounces per day for children 1 to 6 years of age (4).

The toddler and preschool child have distinct developmental and nutrition needs. After the first year of life, a time of rapid growth and development, the growth rate slows, but there is a steady increase in body size. Along with the decrease in growth rate, the appetite decreases. However, there is an increased need for protein and many vitamins and minerals (1). Failure to meet calcium requirements in combination with sedentary lifestyle in childhood can impede the achievement of maximal skeletal growth and bone mineralization, thereby increasing risk for osteoporosis later in life (1).

The toddler and preschool child is striving for independence. Self-feeding is important, although the child may not physically be able to handle feeding utensils or have good hand-eye coordination. At this age, food likes and dislikes become prominent, and food acquires a greater social significance. Children's food preferences are learned through repeated exposure to foods. A minimum of 8 to 10 exposures to a food, is often required for a child to overcome their neophobic response and develop an increased preference for that food $_{(1,5)}$. Family involvement and family mealtimes play a key factor affecting children's nutrition, health, and overall well-being $_{(1)}$.

Nutrition Management of The Toddler and Preschool Child

Beginning at 2 years of age, recommendations from the *Dietary Guidelines* (6) and *MyPyramid* for Kids (2 to 5 years) (7) should be applied for healthy children (1,6,7). See Table E-3. Current guidelines recommend total fat intake between 30 to 35 percent of calories for children 2 to 3 years of age and between 25 to 35 percent of calories for children 4 years and older (6). Most fats should come from sources of polyunsaturated and monounsaturated fatty acids, such as fish, nuts, and vegetable oils (6) The DRIs have established an adequate intake (AI) of fiber which represents a higher than estimated requirements due to the known health benefits of fiber. For children 1 to 3 years 19 g fiber/day is recommended and for ages 4 to 8 years 25 g/day of fiber is recommended (3).

Food Group	Daily Servings	Portion Size	Portion Size		
		1-3 years	4-5 years		
Grains, Breads,	>6 servings				
Cereals	Bread	1⁄4 - 1⁄2 slice	³ ⁄ ₄ -1 slice		
	Dry cereal	¼ - 1/3 cup	½ cup		
	Cooked cereal, noodles, rice	¼ - 1/3 cup	1/3 - ½ cup		
	Crackers	2-3	4-6		
Fruits	≥2 servings				
	Fresh fruit	½ small	1⁄2 -1 small		
	Cooked, canned, or raw, (chopped)	1/3 cup	½ cup		
	Juice	1⁄4- 1⁄2 cup	½ cup		
Vegetables	<u>≥</u> 3 servings				
	Cooked, canned, or raw, (chopped)	¼ cup	½ cup		
		¹ ⁄ ₄ - ¹ ⁄ ₂ piece	½-1 piece		
	Whole	¹ / ₄ cup	½ cup		
	Juice	-	-		
Milk	3-4 servings				
	Milk	½ cup	³ ⁄4 cup		
	Yogurt	½ oz (2-4 tbsp)	¾ oz (4-6 tbsp)		
	Cheese				
Meat	2 servings				
	Egg	1	1		
	Cooked meat	1-3 tbsp	3-5 tbsp		
	Dried beans, peas	1-3 tbsp	2-4 tbsp		
Fat	3-4 servings				
	Margarine; butter; oil	1 tsp	1 tsp		

Children should be supervised during meals and snacks. A child who is choking may not be able to make noise or to attract attention. Foods that may cause choking include hot dogs, chunks of meat, nuts, peanut butter, raw apples, jelly beans, hard candy, gum drops, popcorn, raw carrots, raisins, grapes, berries, and potato or corn chips. By changing the form of some of these items, these foods are less likely to cause choking, such as serving peanut butter with jelly, not by the spoonful, or cutting hot dogs or grapes in small pieces.

References

1. Position of the American Dietetic Association. Nutrition guidance for healthy children aged 2 to 11 years. *J Am Diet Assoc.* 2008;108:1038-1047.

 Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. JAMA. 2006;295:1549-1555.

3. Institute of Medicine's Food and Nutrition Board. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids (Macronutrients)*. Washington, DC: National Academy of Science; 2002.

6. Dietary Guidelines for Americans 2010. Available at:

http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf. Accessed Jan 31, 2011.

7. My Pyramid for Kids. US Department of Agriculture. Available at http://mypyramid.gov/kids/ (2 to 5 years). Accessed January 23, 2009.

^{4.} Committee on Nutrition of the American Academy of Pediatrics. Policy Statement: the use and misuse of fruit juice in pediatrics. *Pediatrics*. 2001;107:1210-1213.

^{5.} Birch LL, Marlin DW. I don't like to; I never tried it: Effects of exposure on two-year-old children's food preferences. *Appetite*. 1982;3: 353-360.

NUTRITION MANAGEMENT OF THE SCHOOL-AGED CHILD

Description

The Regular Diet for the School-Aged Child (6 to 11 years old) includes a wide variety of foods to promote optimal growth and development. Nutrition during this stage should supply adequate nutrients to support physical activity, attain a healthy weight, and ensure that the growth demands of adolescence are met (1). The most recent prevalence estimates from the National Health and Nutrition Examination Survey 2003-2004 indicate that 33.6 % of individuals age 2 to 19 years were at risk of overweight and 17.1% were overweight compared to 28.2% and 13.9 % respectively in 1999-2000 (1.2). This trend has led for the need to broaden dietary guidance from not only focusing on under-consumption but also overconsumption and decreased energy expended as a result of decreased physical activity (1) Healthy eating habits and regular participation in physical activity should be established to reduce the risk of chronic disease and achieve optimal physical and cognitive development (1). Foods are provided based on the Dietary Guidelines (3) and MyPyramid for Kids (6 to 11 years) (4) and the National Cholesterol Education Program (5). Three meals per day plus one to three planned snacks are recommended.

Indications

This diet is served when specific dietary modifications are not therapeutically required.

Nutritional Adequacy

The Regular Diet for the School-Aged Child meets the Dietary Reference Intakes (DRIs) for specific ages as outlined in Section IA: Statement on Nutritional Adequacy, provided that a variety of foods is consumed. Energy and protein requirements vary with the child's age, growth rate, and physical activity. Nutrition concerns of US children include excessive intake of dietary fat, especially saturated fats as well as inadequate intakes of foods rich in calcium, fiber, vitamin E, folate, iron, magnesium, and potassium (1)

How to Order the Diet

Order as "Pediatric Regular Diet" or "Regular Diet for Age _____." The patient's age will be taken into consideration in implementing the diet order. Any specific instructions should be indicated.

Planning the Diet

Energy needs vary with the growth rate, body size, and physical activity of the child. The average energy requirement for children aged 4 to 8 years is 1,742 kcal for boys and 1,642 kcal for girls. For children aged 9 to 11 years, the average daily energy requirement is 2,279 kcal for boys and 2,071 kcal for girls (6). The Institute of Medicine's Food and Nutrition Board has established acceptable macronutrient distribution ranges for school-aged children. These guidelines indicate that carbohydrates should provide 45% to 65% of total energy, proteins should provide 10% to 30% of total energy, and fat should provide 25% to 35% of total energy (6). The recommended dietary allowance (RDA) for protein is 0.95 g/kg for children aged 4 to 13 years. This RDA is met by children aged 4 to 8 years who consume 19 g of protein per day and children aged 9 to 13 years who consume 34 g of protein per day (6). Dietary reference intakes that limit added sugars, defined as sugars and syrups that are added to food during processing or preparation, have been established (1,6). The daily intake of added sugars should be limited to 25% of the total energy consumed by a child (6). Twenty-five percent is a maximum limit; the recommended amount of added sugar in a healthy diet is 6% to 10% of total energy (1,6). Fruit juices can provide a substantial amount of sugar and energy in the diet of school-aged children. Currently it is recommended that daily fruit juice consumption be limited to 4 to 6 oz for children aged 1 to 6 years and 8 to 12 oz for children and adolescents aged 7 to 18 years (7).

The DRI for calcium in children aged 8 years or younger is 500 mg. The DRI increases to 1,300 mg for children aged 9 years or older (8). The requirement for calcium increases with the growth of lean body mass and the skeleton. The higher DRI for calcium was established because evidence indicates that calcium intakes at this level can increase bone mineral density in children, thus decreasing their risk of developing osteoporosis later in life (1). Failure to meet calcium requirements in combination with sedentary lifestyle in childhood can impede the achievement of maximal skeletal growth and bone mineralization, thereby increasing risk for osteoporosis later in life (1).

Older children (9 to 11 years) will have a natural increase in appetite. Between the ages of 8 and 11 years some children (primarily girls), may be at risk for developing eating disorders due to an overemphasis on body image and low intake (9).

Nutrition Management of The School-Aged Child

Recommendations from the *Dietary Guidelines*, National Cholesterol Education Program, and the American Academy of Pediatrics should be applied to the diet of healthy children. These recommendations include an intake of fat between 25 to 35% of total energy, limiting saturated fat to less than 10% of total energy, limiting dietary cholesterol to less than 300 mg/day, and limiting the intake of *trans* fatty acids to as low as possible (3,5,10). Lower targets may need to be achieved based on risk factor assessment of the child's cardiovascular risk profile (10). The recommended daily fiber intake for children aged 6 to 11 years is equal to or greater than the child's age plus 5 g (11). The DRIs have established adequate intakes of fiber that are higher than the estimated requirements due to the health benefits of fiber. The adequate intake of fiber is 25 g/day for children aged 4 to 8 years, 31 g/day for boys aged 9 to 13 years, and 26 g/day for girls aged 9 to 13 years (6).

Food Group	Daily Servings	Portion Size
Grains, Breads, Cereals	More than six servings	
	Bread	1 slice
	Dry cereal	1 oz or ¾ cup
	Cooked cereal	½ cup
	Noodles	4-6
	Rice	½ cup
	Crackers	4 to 6
Fruits	Two or more servings	
	Fresh fruit	1 whole medium
	Cooked, canned, or raw (chopped)	½ cup
	Juice	½ cup
Vegetables	Three or more servings	
	Cooked, canned, or raw (chopped)	½ cup
	Juice	³ ⁄4 cup
Milk	Three servings	
	Milk	1 cup
	Yogurt	1 oz
	Cheese	1 oz
Meat	Two to three servings	
	(a total of 5-6 oz/day)	
	Egg	1
	Cooked meat	2-3 oz
	Dried beans, peas	½ cup
	Peanut butter	2 tbsp
Fats, Sweets	As needed to provide	
	energy	

Table E-4: Food Groups and Rec	ommended Portion Sizes f	for the School-Aged Child (3)
---------------------------------------	--------------------------	-------------------------------

See Section III: Clinical Nutrition Management Obesity and Weight Management.

References

- 1. Position of the American Dietetic Association. Nutrition guidance for healthy children aged 2 to 11 years. *J Am Diet Assoc.* 2008;108:1038-1047.
- 2. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA*. 2006;295:1549-1555.
- 3. Dietary Guidelines for Americans 2010. Available at:
- http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf. Accessed Jan 31, 2011.
- 4. My Pyramid for Kids. US Department of Agriculture. Available at http://mypyramid.gov/kids/ (6 to 11 years). Accessed January 23, 2009.
- 5. National Cholesterol Education Program. *Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents*. Washington, DC: US Dept of Health and Human Services; 1991. NIH publication 91-2732.
- 6. Institute of Medicine's Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids,

Cholesterol, Protein and Amino Acids (Macronutrients). Washington, DC: National Academy of Science; 2002.

- 7. American Academy of Pediatrics Committee on Nutrition. Policy statement: the use and misuse of fruit juice in pediatrics. *Pediatrics*. 2001;107:1210-1213.
- 8. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride.* Washington, DC: National Academy Press; 1997.
- 9. Nutrition Management of the School-Age Child. *Pediatric Manual of Clinical Dietetics*. 2nd ed. Chicago, Ill: American Dietetic Association; 2003.
- 10. Daniels SR, Greer FR, and the Committee on Nutrition. Lipid screening and cardiovascular health in childhood. *Pediatrics*. 2008;122:198-208.
- 11. Position of the American Dietetic Association: Health implications of dietary fiber. J Am Diet Assoc. 2008;108:1716-1731.

NUTRITION MANAGEMENT OF THE ADOLESCENT

Description

The Regular Diet for the Adolescent (11 to 19 years of age) includes a wide variety of foods to promote normal growth and development during puberty and to maintain a good nutritional status for health and disease prevention after the physiological growth has occurred. Foods are provided based on the *Dietary Guidelines for Americans*, the American Heart Association, the American Cancer Association, and the National Cholesterol Education Program (1,2).

Indications

The diet is served when specific dietary modifications are not therapeutically required.

Nutritional Adequacy

The Diet for the Adolescent is adequate to meet the Dietary Reference Intakes (DRIs) for the specific age as outlined in Section IA: Statement on Nutritional Adequacy, provided that a variety of foods is consumed. Energy and protein requirements vary with the adolescent's age, sex, stage of growth, and physical activity. Special attention may be required to ensure adequate intake of iron, zinc, and calcium.

How to Order the Diet

Order as "Regular Diet" or "Regular Diet for Age _____." The age of the patient will be taken into consideration in implementing the diet order. Any specific instructions should be indicated.

Planning the Diet

Energy needs vary with the sex, stage of growth, and physical activity of the adolescent. See Section IA: Estimated Energy Requirement (EER) for Male and Females Under 30 Years of Age. An initial estimate for energy that relates more closely to physiological age can be obtained by calculating kilocalories divided by height in centimeters (3). This is determined by dividing the DRI for energy for the child's age and sex by the reference height (listed on the Estimated Energy Requirement (EER) for Male and Females Under 30 Years of Age table and then multiplying kilocalories per centimeter by the adolescent's height (4). If the height is unavailable or cannot be measured accurately, the DRI for the kilocalories per day may be used (4). Therefore, periodic adjustments in energy intake may be necessary to maintain an appropriate weight for height.

Protein needs for adolescents also relate more to the physiological age than chronological age. The RDA for protein is 0.95 g/kg weight for ages 11-13 then decreases slightly to 0.85 g/kg/day at the age of 14 to 18. Adequate intake ranges from 34 g/day (9 to 13 years) to 52 g/day (14 to 18 years) (4). See Section IA: Dietary Reference Intake Values for Protein by Life Stage Group.

Girls generally begin puberty around 10 to 12 years of age and boys begin between 11 and 13 years of age. Likewise, girls usually have a peak height velocity around age 12 and boys around age 14. Young men often achieve an adult height greater than young women do because boys grow prepubertally 2 years more than girls do and have a longer period of growth once puberty starts. Girls generally stop growth at 16 years of age and boys at 18 years of age.

During puberty, body composition changes. Boys double their lean body mass between 10 and 17 years of age and maintain about 12% body fat by late puberty. Girls gain more fat during puberty and usually have 23% body fat by late puberty.

Vitamins and mineral needs increase as the adolescent grows. Calcium, iron, and zinc are particularly important for growth, and dietary intake is frequently inadequate. Careful food selection is required to meet the DRIs. Accepting changes that will improve nutrient intake seems to be most successful when the change is related to physical development, appearance, and sports performance.

The DRI for calcium is 1,300 mg for both sexes between the ages of 9 and 18 years (5). The accelerated skeletal and muscular development during adolescence makes this stage of life a critical time for bone growth and deposition of calcium.

The DRI for iron 14- to 18-year-olds is 11 mg/day for males and 15 mg/day for females (4). The need for iron increases during puberty with the increase in muscle mass and blood volume.

The RDA for zinc for males and females 14 to 18 years is 11 mg/day and 9 mg/day, respectively (4). Zinc is especially important during adolescence because of its role in growth and sexual maturation.

Recommendations from the *Dietary Guidelines*, the American Heart Association, the American Cancer Association, and the National Cholesterol Education Program should be applied for healthy adolescents. Refer to the Regular Diet-Adult in Section IA for recommendations and guidelines.

Also refer to Section IA: Regular Diet in Pregnancy and Lactation and III: Clinical Nutrition Management, Obesity and Weight Management.

References

- 1. Dietary Guidelines for Americans 2010. Available at:
- http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf. Accessed Jan 31, 2011.
- 2. National Cholesterol Education Program. *Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents*. Washington, DC: US Dept of Health and Human Services; 1991. NIH publication 91-2732.
- 3. Nutrition Management of the Adolescent. *Pediatric Manual of Clinical Dietetics*. 2nd ed. Chicago Ill: The American Dietetic Association; 2003.
- 4. Institute of Medicine's Food and Nutrition Board. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids (Macronutrients)*, Washington, DC, National Academy of Science, 2002.
- 5. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride.* Washington, DC: National Academy Press; 1997.

KETOGENIC DIET

Description

The ketogenic diet is designed to establish and maintain ketosis. The diet is very high in fat and severely restricted in carbohydrates. This is done by calculating the diet to provide 3 to 4 grams of fat for each 1 gram of protein and carbohydrate combined, thus converting the fuel burned by the body from carbohydrate to fat. A physician prescribes the ratio of 3:1 or 4:1 as appropriate for each individual patient. The diet is calculated to meet the specific needs of each individual for calories and protein, and provides little to no carbohydrate depending on protein requirements. Even with the high fat content of the diet, weight is usually maintained with very little gain. This is possible because calories are calculated to meet only 75% of the individual's Dietary Reference Intake (DRI) for energy. The foundation of the diet is either heavy whipping cream or MCT oil. The diet using whipping cream is described below.

Indications

The diet serves as an adjunct to anti-convulsant medications in controlling intractable seizures. It is used in cases of resistance to medications or drug toxicity (1,2). Sustained ketosis appears to be important in modifying the convulsive threshold (1,3). The diet seems to be most effective in children 18 months to 10 years of age (4), although it can be used with older children and adults with varying degrees of success. The diet is administered to those who have myoclonic absence (drop) and atonic seizures, which are difficult to control with medications. It may also benefit children with generalized tonic-clonic (grand mal) seizures and seizures of the Lennox-Gestault Syndrome. The ketogenic diet can be used for all types of seizures, especially if medication therapy is not effective (5).

The diet requires a trial period of 2 to 3 months during which effectiveness is assessed and the diet is adjusted to maintain strong ketosis. Once it is determined that the diet is effective on controlling seizure activity, a commitment of 1 to 2 years is required after which weaning is done gradually. Because of the extreme dietary regimens involved in this diet, the Johns Hopkins Pediatric Epilepsy Center recommends use of the ketogenic diet for those individuals who have more than 2 seizures a week despite treatment with at least 2 different anticonvulsant medications (6).

Nutritional Adequacy

The ketogenic diet is inadequate in vitamin B-complex vitamins, folate, iron, calcium, and zinc. The diet must be supplemented with vitamins, iron and calcium in forms that are sugar-free.

How to Order the Diet

Order as "Ketogenic Diet." A nutrition consult by a registered dietitian must accompany the diet order, as the diet has to be precisely calculated. All medications must be carbohydrate free, as well as toothpaste. The diet must be initiated in a hospitalized setting under close supervision.

Planning the Diet

A gram scale and a copy of the Epilepsy Diet Treatment book (6) are paramount in administering this diet effectively.

Calculation (5)

1. Sample patient: age and weight

Age5Height43 inchesWeight in kilograms18.46 (40.6 lb)Ideal weight18.46 (50th percentile)

Ketogenic Ratio (fat calories:nonfat calories ratio)

Up to 2 years	3:1
2 years to 12 years	4:1
Over 12 years	3:1

A 4:1 Ketogenic diet is prescribed for the patient, which at 50th percentile matches the ideal weight for his age and size.

2. Calories per kilogram: Calculate the ideal body weight for the child's height using the NCHS growth charts. Determine the number of calories per kilogram based on the child's age and ideal weight from the following chart (7). Additional adjustments for caloric needs will need to be individualized based on patient's activity level.

Un to 1 wood	00 least /lea
Up to 1 year	80 kcal/kg
12 - 18 months	75 kcal/kg
18 months - 3 years	70 kcal/kg
4 - 6 years	65 kcal/kg
7 - 8 years	60 kcal/kg
9 - 10 years	55 kcal/kg
11 - 14 years	40 kcal/kg or less

3. Total calories: Determine the total number of kcal in the diet by multiplying the child's ideal weight by the number of calories required per kilogram.

The patient, age 5 and weighing 18.46 kg, needs a total of 65 x 18.46 or 1,200 kcal per day.

4. Dietary unit composition: Dietary units are the building blocks of the ketogenic diet. A 4:1 diet has dietary units made up of 4 gm of fat to each 1 gm of protein plus carbohydrates. Because fat has 9 calories/g (9 x 4 = 36), and protein and carbohydrates each have 4 kcal/g (4 x 1 = 4), a dietary unit at a 4:1 diet ratio has 36 + 4 = 40 kcal. The caloric value and breakdown of dietary units vary with the ketogenic ratio.

Ratio	Fat Calories	Carbohydrates plus Protein Calories	Calories per Dietary Unit
2:1	2 g x 9 kcal/g = 18	1 g x 4 kcal/g = 4	18 + 4 = 22
3:1	3 g x 9 kcal/g = 27	1 g x 4 kcal/g = 4	27 + 4 = 31
4:1	4 g x 9 kcal/g = 36	1 g x 4 kcal/g = 4	36 + 4 = 40
5:1	5 g x 9 kcal/g = 45	1 g x 4 kcal/g = 4	45 + 4 = 49

The patient's dietary units will be made up of 40 calories each because he is on a 4:1 ratio.

5. Dietary unit quantity: Divide the total calories allotted by the number of calories in each dietary unit to determine the number of dietary units to be allowed daily.

Each of the patient's dietary units on a 4:1 ratio contains 40 calories, is allowed a total of 1200 kcal/day, so he receives 1200/40 = 30 dietary units per day.

6. Fat allowance: Multiply the number of dietary units' times the units of fat in the prescribed ketogenic ratio to determine the number of fat grams permitted daily.

On his 4:1 diet, with 30 dietary units per day, the patient will have 30 x 4 or 120 g of fat per day.

7. Protein and carbohydrate allowance: Multiply the number of dietary units times the number of protein plus carbohydrate in the prescribed ketogenic ratio, usually one, to determine the combined daily protein plus carbohydrate allotment.

On his 4:1 diet, the patient will have 30 x 1 or 30 g of protein and carbohydrate per diet.

8. Protein allowance: To maintain health, a 5-year-old child should eat a minimum of 1 g of protein for every kilogram of weight and/or meet the DRI for protein for age.

At 18.56 kg, the patient should eat 18.5 g of protein per day out of his total protein and carbohydrate allotment of 30 g.

9. Carbohydrate allowance: Determine the grams of carbohydrate allotted by subtracting the protein allotment from the total protein plus carbohydrate allotment. Carbohydrates are the diet's filler and are always determined last.

The patient's carbohydrate allotment is 30 - 18.5 = 11.5 gm carbohydrate daily.

10. Meal Order: Divide the daily fat, protein and carbohydrate allotments into 3 equal meals. It is essential that the proper ratio of fat to protein plus carbohydrate be maintained at each meal.

The patient's diet order reads:

	Daily	Per Meal
Protein	18.5 g	6.2 g
Fat	120 g	40 g
Carbohydrate	11.5 g	3.8 g
Kcal	1,200	400

11. Liquids: Multiply the child's ideal weight by 65 to determine the daily cubic centimeter allotment of liquid. As few as 60cc/kg but as many as 70cc may be adequate, depending on the child's activity level and the climate in which they live. Liquid intake should be spaced throughout the day with no more than 120 - 150 cc being given at any one time. Liquids should be non-caloric such as water, herbal or decaffeinated tea or decaffeinated sugar-free diet soda. Sugar free soda should be limited to no more than 1 calorie per day. In hot climates, the cream may be excluded from the fluid allotment. The liquid allotment may also be set equal to the number of calories in the diet.

The patient, who lives in New York and gets 1200 kcal per day on the diet, is allowed 1200 cc of fluid per day, including his allotted cream.

12. Every child on the ketogenic diet should take a daily dose of a sugar-free vitamin/mineral supplement. For infants or children who have difficulty chewing, 600 to 650 mg of oral calcium, in a sugar-free form, such as calcium gluconate or calcium carbonate or calcium magnesium liquid and a sugarless multi-vitamin with iron, such as Poly-Vi-Sol® liquid or drops can be used. A sugar free multivitamin mineral Chew Tab is a better choice for children over 1 year of age that can chew.

Introducing The Ketogenic Diet

The diet must be introduced in the hospitalized care setting. Initially "ketogenic eggnog" is given after the initial two-day fast or when the ketones have reached the 160 level (4+).

To introduce to children, a ketogenic eggnog is provided a sample full meal recipe follows. The child should receive 1/3 of the child's full meal recipe first meal, 2/3's of the full meal recipe the second meal, and progress to the full recipe by the third meal.

Calculating The Ketogenic Eggnog

Step 1: Calculate the recipe based on 1/3 of the child's total allotted calories. Select an amount of cream that contains close but not equal to the amount of total allotted fat.

	Weight	Protein	Fat	Carbohydrate
Cream	97 g	1.9 g	34.9 g	2.9 g
Egg				
Should b	e	6.2 g	40.0 g	3.8 g

Step 2: Subtract the carbohydrate in the cream used from the total allotted carbohydrate: 3.8 g - 2.9 g = 0.9 g.

Step 3: Add the remaining amount of carbohydrate to the total allotted protein: 6.2 g + 0.9 g = 7.1 g.

Step 4: Subtract the protein used in the cream from the sum in Step 3. 7.1 g - 1.9 g = 5.2 g.

Step 5: Using the food values chart (8), give the amount of egg that contains 5.2 g of protein.

	Weight	Protein	Fat	Carbohydrate
Cream	97 g	1.9 g	34.9 g	2.9 g
Egg	43 g	5.2 g	5.2 g	
Actual total Should be		7.1 g 6.2 g	40.1 g 40.0 g	2.9 g 3.8 g

Recipe for 1 Full Meal

In the ketogenic eggnog, the carbohydrate will be lower than the allotment and the protein will be higher than the allotment. The amount of fat should always be within a close proximity to the allotment. On occasion, depending on different ketogenic ratios used, small amounts of oil may be needed.

The 4:1 ketogenic ratio may be double-checked by adding the grams of protein and carbohydrate in the meal and multiplying by four (4). The sum should be the amount of fat in the meal, in this case, 40.0 g. Since (7.1 g + 2.9 g) x 4 = 40.0 g, the ratio is correct.

When the full quantity is reached, real food may be served or the child may be given eggnog again.

The Ketogenic Eggnog Recipe

Ketogenic eggnog is the only meal that does not need to be eaten all at once. This way the child sipping eggnog will not be under as much pressure as when he/she is faced with a plate of unfamiliar food. At home, the parents can prepare more appetizing, familiar meals. However, it is important that parents be given enough training in preparing solid food meals so they will be able to do it comfortably at home. Ingredients required for the ketogenic eggnog are:

Heavy Cream Egg Vanilla Extract Saccharin (optional)

The patient's first meal of eggnog will be 1/3 of the full meal recipe:

32 g	Heavy Cream
14 g	Egg
up to 5 drops	Vanilla
up to ¼ grain	Saccharin
46 cc	Total

The patient's second meal of eggnog will be 2/3 of the full meal recipe:

64 g	Heavy Cream
28 g	Egg
up to 5 drops	Vanilla
up to ¼ grain	Saccharin
92 cc	Total

Ketogenic Diet

The patient's third meal of eggnog will be the full meal recipe.

92 g 14 g	Heavy Cream
up to 5 drops	Vanilla
up to ¼ grain	Saccharin
140 cc	Total

Regular meals are provided to the patient usually by the third meal and/or prior to discharge.

Calculating Meal Plans

When calculating the meal plan, divide the total protein, fat and carbohydrate allotted for the day by three and provide 1/3 of the allotment per meal. For example:

	Weight	Protein	Fat	Carbohydrate
Cream	65 g	1.3 g	23.4 g	1.9 g
Fruit	19 g	0.2 g		1.9 g
Meat	20 g	4.7 g	3.3 g	
Fat	18 g		13.3 g	
Actual Total Should be		6.2 g 6.2 g	40.0 g 40.0 g	3.8 g 3.8 g

Calculate the whipping cream first. Heavy whipping cream (36%) should take up no more than half of the carbohydrate allotment in the meal.

The patient is allowed a total of 3.8 g carbohydrates per meal. Referring to the food value charts (9), to use half of this allotment of cream, he should eat 65 g of 36% cream, which contains 1.9 g carbohydrates.

Calculate the rest of the carbohydrate (fruits or vegetables) by subtracting the carbohydrate contained in the cream from the total carbohydrate allotment.

Referring to the food value charts, the patient can eat the remaining 1.9 g carbohydrates as 19 g of 10% fruits. The percent equals the percent of carbohydrate in the fruit (7).

15% Carbohydrate Fruits
Apple
Apricot
Blackberries
Blueberries
Figs
Nectarine
Pear
Pineapple
Plums (Damson)
Raspberries (black)
Raspberries (red)
Grapes
Mango

Calculate the remaining protein (meat/fish/poultry, cheese or egg) by subtracting the protein in the cream and vegetable from the total protein allotment. The 65 g of 36% cream and the 19 g of 10% fruits contain a total of 1.5 g of protein.

The patient is allowed 6.2 gm of protein per meal, so he can eat 4.7 g of protein from meat, fish or poultry. Referring to the food value charts (9), this calculates to be 20 g of meat, fish or poultry.

Calculate the amount of fat to be allowed in the meal by subtracting the fat in the cream and protein from the total fat allotment.

The patient has to eat 40 g of fat with each meal. The cream and meat contain 26.7 g of fat, leaving 13.3 g of fat to be mixed with his meal.

Butter, margarine or mayonnaise are more frequently used because of their palatability. However, they contain only 74% fat. Therefore, the remaining grams of fat are divided by 0.74. 13.3/.74 = 17.9 or 18 g of butter, margarine or mayonnaise

Oil is not included but can be used. Oil would raise the average up higher but is not used as often as butter, margarine or mayonnaise.

Other Considerations

Because the diet may induce hypoglycemia, blood glucose levels need to be monitored during the fasting period (3). All IV's must be glucose free. If the blood sugar drops at or below 25-mg % with symptoms of hypoglycemia, administer 15 to 30 cc (1.8 to 3.75 g carbohydrate) of orange juice. Monitor closely and administer more juice if necessary, but be aware that too much carbohydrate will delay ketosis. (See reference 5 for complete hypoglycemia plan.) Another alternative is to administer 1 oz. Pulmocare® plus 5-cc corn/safflower oil. This provides a 4.3:1 ratio and 1.25 g carbohydrate in 30 cc, therefore, treating the hypoglycemia but not interrupting ketosis (7).

Food Guide

All foods must be weighed precisely on a gram scale. *Bowes & Church's Food Values of Portions Commonly Used* (9) is a useful reference for meal planning.

The following foods and products are eliminated from the diet because they contain an appreciable amount of carbohydrates.

Foods to Avoid		
Bread	Jam	Potatoes
Cake	Sugar sweetened Ketchup	Puddings
Candy	Marmalade	Rice
Carbonated beverages,	Medicines containing sugar	Rolls
Cereals, sugar coated	Molasses	Sherbet
Chewing gum	Muffins	Sugar
Cookies	Pancakes	Syrup
Cough drops or cough syrups that contain	Pastries	Toothpaste
sugar	Peas	Waffles
Crackers	Pies	
Honey	Jelly Preserves	
Ice cream, commercial		

Ketogenic Diet

References

- 1. Clark BJ, House FM. Medium Chain Triglyceride Oil Ketogenic Diets in the Treatment of Childhood Epilepsy. J. Hum Nutr, 1978; 32:111.
- 2. Withrow, CD. The Ketogenic Diets: Mechanism of Anticonvulsant Action. In: Glaser GH, Penry JK, Woodbury DM. Anti-epileptic Drugs: Mechanisms of Action. New York, NY: Raven Press; 1980.
- 3. Huttenlocher PR, Wilbourn AJ, Signore JM. Medium Chain Triglycerides as a Therapy for Intractable Childhood Epilepsy. *Neurology* 1971; 21:1097.
- 4. Gordon N. Medium Chain Triglycerides in a Ketogenic Diet. Dev Med Child Neurol. 1977; 19:535.
- 5. *Calculating and Administering the Ketogenic Diet in the Treatment of Pediatric Epilepsy.* University of California Conference, Redondo Beach, CA: September 22-23, 1995.
- 6. Gershoff SN ed. A diet for epilepsy provides new hope. Tufts University Health & Nutrition Letter. 1997;15:6.
- 7. Freeman JM, Kelly MT. *The Epilepsy Diet Treatment, An Introduction to the Ketogenic Diet.* New York, NY: Demos Publication; 1996. 1-800-532-8663.
- 8. Personal Communications. St. Joseph Hospital and Medical Center Nutrition Service, Hospital and Medical Center, Phoenix, Az: 1996.
- 9. Pennington JA. Bowes and Church's Food Values of Portions Commonly Used. 17th ed. Philadelphia, PA: Lippincott; 1998.

I. NORMAL NUTRITION AND MODIFIED DIETS F. Modification of Minerals

Sodium-Controlled Diet	F-1
No Added Salt Diet (4,000-Mg Sodium Diet)	F-4
Food Guide — 3,000-Mg Sodium Diet	F-5
2,000 Mg And 1,500 Mg Sodium Restricted Diet Patterns	F-6
Food Guide — 1,000-Mg Sodium Diet	F-8
Nutrition Management of Potassium Intake	F-10
Table F-1: Potassium Content of Common Foods	F-11
Nutrition Management of Phosphorus Intake	F-12
Table F-2: Phosphorous Content of Common Foods	F-13
Nutrition Management of Calcium Intake	F-14
Table F-3: Calcium Content of Common Foods	F-15

SODIUM-CONTROLLED DIET

Description

The Sodium-Controlled Diet limits sodium intake. Foods and condiments high in sodium are eliminated or restricted at suggested levels to optimally manage blood pressure and underlying medical conditions associated with hypertension or chronic organ damage.

The average dietary sodium intake is approximately 4,100 mg/day for American men and 2,775 mg/day for American women (1). The consumption of processed foods accounts for 75% of the daily sodium intake (2). The minimum daily sodium requirement for healthy adults is 500 mg (3). In 2002, The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure and the National Research Council (JNC 7) recommended daily intake of sodium be limited to 2,400 mg. (4). However, based on emerging data and evaluation of scientific data, more recent recommendations from The American Heart Association, *Dietary Guidelines for Americans*, and National Institutes of Health National Heart Lung and Blood Institute (NIH/NHLBI), and Dietary Approaches to Stopping Hypertension study outcomes suggest targeting less than 2,300 mg/day for healthy adults. High risk populations including African Americans, middle age, older adults and persons with existing hypertension should target a lower sodium intake of 1,500 mg/day in adults (2,4,5,6). Refer to Section III: Hypertension.

Indications

The Sodium-Controlled Diet is used in the treatment of conditions characterized by edema (water retention), including the following:

- cirrhosis of the liver with ascites
- heart failure
- hypertension
- renal disease

Under normal physiologic conditions, the body responds to an increase in sodium consumption with an increase in sodium excretion, generally eliminating the excess sodium within 24 hours (3,7). However, certain diseases or conditions impair the body's ability to maintain a normal sodium and water balance, necessitating a reduction in sodium intake. Excess sodium in the body caused by one of the conditions listed above can lead to edema, increased blood pressure, thirst, and shortness of breath.

Cirrhosis of the liver with ascites: Ascites, an accumulation of nutrient-rich fluid in the peritoneal cavity, often occurs as a result of hepatic cirrhosis. A small percentage of patients with this condition lose weight and reduce their fluid volume by adhering to a sodium-controlled diet (8). Almost 90% of patients respond to combination therapy consisting of a sodium-controlled diet and diuretics, whereas the other 10% of patients are resistant to combination therapy and require further medical intervention (9). Although fluid restrictions often accompany sodium-controlled diets, the efficacy of this practice in the treatment of patients with ascites has been challenged. Fluid restriction may not be necessary unless the serum sodium level drops below 128 mEq/L (8.9). In patients with ascites, the treatment goal is to achieve a negative sodium balance and a weight loss of 0.5 kg/day (8). Sodium-controlled diets that provide less than 2,000 mg of sodium per day, depending on the patient's fluid volume, are recommended (8).

Heart failure: In patients with heart failure, the kidneys respond to a decrease in systemic blood flow by increasing the absorption of sodium and fluids, leading to edema and worsening heart failure. To promote diuresis, a sodium-controlled diet accompanied by diuretic use is the preferred method of treatment (2,3,10). In heart failure, sodium intake should be less than 2,000 mg (2 g) per day. Sodium restriction will improve clinical symptoms and quality of life (Grade II)* (10) According to the *Comprehensive Heart Failure Practice Guidelines*, dietary sodium restriction of 2- to 3- g daily is recommended for patients with the clinical syndrome of heart failure and a preserved or depressed left ventricular ejection fraction (LVEF). Further restriction (< 2 g daily) may be considered in moderate to severe heart failure (13). (See Section III, Heart Failure.) The fluid intake of patients with hyponatremia (plasma sodium concentration <130 mEq/L) may be restricted to 1.4 to 1.9 L/day, depending on the clinical signs and symptoms (Grade III) (10).

Hypertension: Sodium-sensitive individuals have an impaired ability to excrete large concentrations of sodium, leading to increased serum sodium levels, hypervolemia, and hypertension. Between 20% and 50% of individuals with hypertension, particularly the elderly and African Americans, respond to an increase in

Sodium-Controlled Diet

sodium consumption with an increase in blood pressure (7). Other lifestyle modifications that can help prevent hypertension include losing excess body weight, following the Dietary Approaches to Stop Hypertension (DASH) eating plan, increasing physical activity, and avoiding excess alcohol intake (2). The DASH collaborative intervention studies have demonstrated that a reduced sodium diet of <2,400 mg/day, which includes increased intakes of fruits, vegetables, potassium-rich foods, and low-fat dairy foods and decreased intakes of total fat (27%), saturated fat (6%), and cholesterol (<150 mg) has a significant effect on lowering blood pressure (12,13). Patients who followed the DASH eating plan experienced an 8- to 14-mm Hg reduction in systolic blood pressure (Grade IV) (12,13,14). The greatest blood pressure reductions occurred in patients who followed the DASH eating plan at a sodium intake level of 1,500 mg/day (12,14). The Academy of Nutrition and Dietetics' Hypertension Evidence Based Nutrition Practice Guideline suggests that sodium intake be limited to no more than 2,300 mg/day (Grade 1)(15). Reduction of dietary sodium to the recommended levels lowers systolic blood pressure by 2- to 8-mm Hg (Grade I) (14). If the patient demonstrates good adherence to a 2,300 mg sodium diet but has not achieved the treatment goal, then the dietitian should recommend the DASH dietary pattern and/or a reduction in daily sodium intake to 1,600 mg to further reduce blood pressure (Grade 1)(14). Sodium-controlled diets also enhance the effectiveness of diuretic therapy (2,3) and may help individuals remain normotensive after the cessation of pharmacologic therapy (2,16).

If a potassium-wasting diuretic, such as thiazide or a loop diuretic, is prescribed, a diet containing increased amounts of potassium may be necessary to avoid hypokalemia (3). Patients should be advised to consume adequate food sources of potassium as part of medical nutrition therapy to reduce blood pressure. Research suggests that potassium intake lower than the recommended Dietary Reference Intakes is associated with increased blood pressure (Grade II) (14). (See Nutrition Management of Potassium Intake later in this section and Hypertension, including the DASH Eating Plan, in Section III.)

Renal disease: See Section IG: Medical Nutrition Therapy for Chronic Kidney Disease.

Contraindications

Under normal conditions, the dietary restriction of sodium intake should not cause sodium depletion. However, a sodium-controlled diet is contraindicated in the presence of the following:

- conditions that promote sodium depletion (profuse perspiration, vomiting, and diarrhea)
- impaired mechanisms of sodium conservation (colectomy and ileostomy in the postoperative period)
- conditions that conserve sodium as a normal physiologic adjustment (pregnancy)
- lithium carbonate therapy (The kidney does not always discriminate between sodium and lithium. Therefore, with a low sodium intake, the kidney may conserve both sodium and lithium, causing an increased serum lithium level and the potential for lithium toxicity (7)).

Nutritional Adequacy

Sodium-controlled diets can be planned to meet the Dietary Reference Intakes as outlined in Section IA: Statement on Nutritional Adequacy.

How to Order the Diet

- Order the diet in terms of sodium, not salt.
- Order the amount of sodium that should not be exceeded in the diet. Different levels of sodium-controlled diets limit the daily sodium intake to 1,500mg (65 mEq), 2,000 mg (87 mEq), 3,000 mg (130 mEq) or 4,000 mg (174 mEq).
- The dietitian may allow certain higher sodium foods to be added to the patient's diet if the patient's sodium intake falls below the prescribed range due to low energy intake.

Note that diets containing less than 2,000 mg/day of sodium are difficult to sustain outside of the hospital environment for reasons of palatability and convenience (3).

Planning the Diet

Salt substitutes: Salt substitutes will not be offered unless a physician, standing order, or an organization's policy designates their use. Salt substitutes may contain potassium chloride, which could be contraindicated under certain conditions. Some salt substitutes also contain various amounts of sodium.

Sodium in medications: Patients on a sodium-restricted diet should be made aware that certain over-thecounter medications (eg, seltzers and some antacids) contain high quantities of sodium and that they should consult their physician if the medications are used on a regular basis. For more information, refer to Section III: Clinical Nutrition Management:

- Heart Failure
- Corticosteroid Therapy
- Hypertension
- Nephrotic Syndrome

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. US Department of Agriculture, Agricultural Research Service. Nutrient Intakes from Food: Mean Amounts Consumed per Individual, One Day, 2005-2006. Available: www.ars.usda.gov/ba/bhnrc/fsrg.
- Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ, and the National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42:1206-1252.
- 3. Whitmere S. Water, electrolytes, and acid-base balance. In: Mahan KL, Escott-Stump S, eds. *Krause's Food, Nutrition and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders; 2000:159.
- 4. Whelton PK, He J, Appel LJ, Cutler JA et al. National High Blood Pressure Education Program Coordinating Committee. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education program. *JAMA* 2002 Oct 16;288(15):1882-8.
- 5. Appel LJ; American Society of Hypertension Writing Group, Giles TD et al. DASH Position Paper: Dietary approaches to lower blood pressure. *J Clin Hypertens* 2009 Jul;11(7):358-68.
- 6. Lloyd-Jones DM. Cardiovascular risk prediction: basic concepts, current status, and future directions. *Circulation* 2010 Apr 20; 121(15):1768-77.
- 7. Haddy FJ, Pamnani MB. Role of dietary sodium in hypertension. J Am Coll Nutr. 1995;14:428-438.
- 8. Gines P, Cardenas A, Arroyo V, Rodes J. Management of cirrhosis and ascites. *N Engl J Med.* 2004;350:1646-1654.
- 9. Aiza I, Perez GO, Schiff ER. Management of ascites in patients with chronic liver disease. *Am J Gastroenterol.* 1994;89:1949-1956.
- 10. *Heart Failure Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.adaevidencelibrary.com. Accessed December 3, 2010.
- 11. Lindenfeld J, Albert NM, Boehmer JP, et al. Executive Summary: HFSA 2010 Comprehensive Heart Failure Practice Guideline. *J Card Fail* 2010;16:475-539.
- 12. Sacks FM, Svetkey LP, Vollmer WM, Obarzanek E, Conlin PR, Miller ER 3rd, Simons-Morton DG, Karanja N, Lin PH. DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med.* 2001;344:3-10.
- 13. Svetkey LP, Simons-Morton D, Vollmer WM, Appel LJ, Conlin PR, Ryan DH, Ard J, Kennedy BM, for the DASH Research Group. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomized clinical trial. *Arch Intern Med.* 1999;159:285-293.
- 14. *Hypertension Evidence-Based Nutrition Practice Guideline*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.adaevidencelibrary.com. Accessed January 10, 2009.
- 15. Vollmer WM, Sacks FM, Ard J, Appel LJ, Bray GA, Simons-Morton DG, Conlin PR, Svetkey LP, Erlinger TP, Moore TJ, Karanja N. DASH-Sodium Trial Collaborative Research Group. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASHsodium trial. *Ann Intern Med*. 2001;135:1019-1028.
- 16. Alderman MH. Non-pharmacological treatment of hypertension. *Lancet.* 1994;344:307-311.
NO ADDED SALT DIET (4,000-mg Sodium Diet)

FOODS EXCLUDED

Bacon* Barbecue sauce* Buttermilk, cultured (limit to 1 cup/day) Ketchup (limit to 1 tbsp/day) Cheese, processed Chili sauce Commercially canned products, frozen products, or convenience products (unless <600 mg of sodium per entree or <350 mg of sodium per single food items) Corned beef* Fish, salty or smoked (eg, anchovies, salted cod, herring, sardines) Frankfurters* Ham* Meat extracts Meat, luncheon* Meat, smoked, cured, canned, or pickled Meat tenderizers Olives Party spreads and dips Salted potato chips, corn chips Salt pork Salted bouillon cubes Salted crackers Salted nuts Soups, canned, frozen, or dehydrated (unless reduced-sodium) Sauerkraut or pickled vegetables Sausage* Spices and herbs that contain salt (eg, garlic salt, celery salt, onion salt, and lemon pepper) Soy sauce Tuna canned in oil (tuna can be used if rinsed)

*May be calculated into the diet. Select only one serving daily from the entire list.

Note: Foods with sodium contents greater than 350 mg per serving should be calculated into the diet.

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages	Low-sodium carbonated beverages Coffee, tea	
Breads, Cereals, Grain Products (150 mg sodium/ serving)	Enriched white, wheat, rye, and pumpernickel bread Hard rolls, dinner rolls Muffins, corn bread Waffles, pancakes Most dry and hot cereals Crackers and breadsticks with unsalted tops Tortillas Enriched unsalted rice, barley, noodles, spaghetti, macaroni, and other pastas Unsalted tortilla chips, pretzels, potato chips, or popcorn Homemade bread stuffing	Breads, rolls, and crackers with salted tops Commercially prepared rice and pasta mixes Salty snack foods Stuffing mixes
Vegetables (10 mg sodium/ 1/2 c serving)	All fresh and frozen vegetables Canned, drained vegetables White and sweet potatoes Squash Low-sodium tomato sauce and tomato paste	Sauerkraut, pickled vegetables, and others prepared in brine Vegetables seasoned with ham, bacon, salt pork, cheese, or cheese sauces Commercially prepared potato mixes Regular tomato sauce and puree
Fruits and Juices (10 mg sodium/ 1/2 c serving)	All fruits and fruit juices Low-sodium or salt-free vegetable juices	Fruits dried with sodium sulfate Regular vegetable juices
Milk (150 mg sodium/serving)	Milk, buttermilk (limit to 1 cup/day), chocolate milk Yogurt, frozen yogurt Regular ricotta cheese (1/4 cup); Swiss or mozzarella cheese (1 oz)	Instant milk beverages, instant cocoa mix, malted milk
Meats and Meat Substitutes (60 mg sodium/ 1 oz serving)	Fresh or frozen beef, lamb, pork, and poultry Fish and most shellfish; canned tuna or salmon, rinsed Eggs and egg substitutes Low-sodium cheese Regular peanut butter (3 times weekly) Dried peas and beans Frozen dinners (<600 mg sodium)	Smoked, cured, salted, koshered, or canned fish, poultry, or meat, including bacon, chipped beef, cold cuts, ham, hot dogs, sausage, sardines, anchovies, marinated herring, and pickled meats Frozen breaded meats Pickled eggs Processed cheese; cheese spreads and sauces
Fats (100 mg/tbsp)	Butter or margarine Vegetable oils Salad dressings in limited amounts (2 tbsp) Light, sour, and heavy cream Mayonnaise Unsalted nuts	Bacon, salad dressings containing bacon fat, bacon bits, and salt pork Snack dips made with instant soup mixes or processed cheese Salted nuts Olives Canned gravy and mixes

FOOD CLUDE 2 000 M/C CODILINA DIET

Sodium-Controlled Diet

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Soups	Homemade broth Soups without added salt and made with allowed vegetables Reduced-sodium canned soups and broths	Regular canned or dehydrated soups Bouillon cubes
Desserts and Sweets	All	None
Miscellaneous	Limit added salt to ¼ tsp/day used at the table or in cooking Use a salt substitute with physician's approval Pepper, herbs, spices Vinegar Ketchup (1 tbsp), mustard (1 tbsp) Lemon or lime juice Hot pepper sauce, low-sodium soy sauce (1 tsp), Worcestershire sauce (1 tsp) Salsa (¼ cup)	Seasonings made with salt, including garlic salt, celery salt, onion salt, and seasoned salt; sea salt; rock salt; kosher salt; lemon pepper Meat tenderizers Monosodium glutamate Olives Regular soy sauce, teriyaki sauce, barbecue sauce

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Honey Clozed Chicken	Unsalted Beef Tips and Noodles
Unsalted Cream of Wheat	Steamed Rice	Seasoned Green Beans
Unsalted Scrambled Egg	Steamed Broccoli	Sliced Tomato Salad
Wheat Toast	Fruited Gelatin	French Dressing
Margarine, Jelly	Dinner Roll	Dinner Roll
Milk (1 cup)	Margarine	Margarine
Coffee	Frosted Banana Cake	Peach Halves
Sugar, Creamer	Milk (1 cup)	Iced Tea
	Tea	Sugar
	Sugar	

2,000 mg and 1,500 mg Sodium Restricted Diet Patterns (Sample Patterns)

	(Sample Fatterns)	
Food Group	Number of Servings 2,000 mg sodium	Number of Servings 1,500 mg sodium
Bread, Cereals, and Grain Products (150 mg/serving)	6 regular bread or cereal items	5 regular bread or cereal items
Vegetables/Fruits (10 mg/ ½ c serving)	5	5
Meat, Fish, Poultry and Eggs (60 mg/ 1 oz)	6	6
Milk and Dairy (150 mg/serving)	3	2
Fats (100 mg/1 Tbsp serving)	1	0
	6 tsp unsalted	7-8 tsp unsalted
Soup (unsalted only)	Calculate into diet	Calculate into diet
Sugar and Sweets	Read label and calculate into diet	Read label and calculate into diet
Total Sodium	1760 mg	1460 mg

Breakfast	Noon	Evening
Orange Juice	Honey Glazed Chicken	Unsalted Beef Tips and Noodles
Stewed Prunes	Steamed Rice	Seasoned Green Beans
Unsalted Cream of Wheat	Steamed Broccoli	Seasoned Carrots
Unsalted Scrambled Egg	Tossed Salad with Fat-Free	Sliced Tomato Salad
Wheat Toast	Dressing	Dinner Roll
Margarine, Jelly	Dinner Roll	Margarine
Nonfat Milk (1 cup)	Margarine	Peach Halves
Coffee	Fresh Banana	Iced Tea
Sugar, Creamer	Nonfat Milk (1 cup)	Sugar
	Tea, Sugar	
SAMPLE MENU (1,500 mg sodium)		
Breakfast	Noon	Evening

SAMPLE MENU (2,000 mg sodium)

DICANIASI	NUUII	Evening
Orange Juice	Honey Glazed Chicken	Unsalted Beef Tips and Noodles
Stewed Prunes	Steamed Rice, unsalted	Seasoned Green Beans, unsalted
Unsalted Cream of Wheat	Steamed Broccoli	Seasoned Carrots, unsalted
Unsalted Scrambled Egg	Tossed Salad with Fat-Free,	Sliced Tomato Salad
Wheat Toast	Low -sodium Dressing	Dinner Roll
Margarine, unsalted	Dinner Roll	Margarine, unsalted
Jelly	Margarine, unsalted	Peach Halves
Nonfat Milk (1 cup)	Fresh Banana	Iced Tea
Coffee	Nonfat Milk (1 cup)	Sugar
Sugar, Creamer	Tea, Sugar	

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages	Coffee; tea	Gatorade
Breads, Cereals, Grain Products	Low-sodium carbonated beverages Hot cereal without salt Puffed rice, puffed wheat, shredded wheat, and low- sodium dry cereals Low-sodium bread Low-sodium crackers, melba toast, and matzo Tortilla Enriched unsalted rice, barley, and pastas Unsalted tortilla chips, pretzels, potato chips, or popcorn	Breads, rolls, and crackers with salted or unsalted tops Quick breads; biscuits; cornbread; muffins Frozen waffles; pancakes Regular dry cereal; instant hot cereals Self-rising flour Commercially prepared rice or pasta mixes Potato chips; salty snack foods
	<i>Limit to 2 servings per day:</i> Enriched white, wheat, rye, and pumpernickel bread or breadsticks; hard rolls and dinner rolls; homemade bread stuffing	
Vegetables	All fresh, unsalted frozen vegetables Low-sodium canned vegetables White or sweet potatoes Squash Unsalted tomato paste	Regular canned vegetables, sauerkraut, pickled vegetables, and others prepared in brine Vegetables seasoned with ham, bacon, or salt pork Tomato sauce, puree, and regular paste Commercially prepared potato mixes Frozen peas, lima beans, and mixed vegetables All frozen vegetables in sauce
Fruits and Juices	All fruits and fruits juices Low-sodium, salt-free vegetable juices	Regular vegetable juices Fruits processed with salt or sodium compounds, eg, some dried fruits
Milk	<i>Limit to 2 servings per day</i> Milk Yogurt	Malted milk; milk shake; buttermilk; chocolate milk
Meats and Meat Substitutes	Any fresh or frozen beef, lamb, pork, and poultry Fish and most shellfish; low-sodium canned tuna or salmon Eggs Low-sodium cheese, cottage cheese, and ricotta cheese Low-sodium peanut butter Dried peas and beans	Any smoked, cured, salted, koshered, or canned meat, fish, poultry including bacon, chipped beef, cold cuts, ham, hot dogs, sausage, sardines, anchovies, marinated herring, and pickled meats Frozen breaded meats Egg substitutes; pickled eggs Regular hard and processed cheese; cottage cheese; cheese spreads and sauces Regular peanut butter Frozen dinners
Fats	Unsalted butter or margarine Vegetable oils Low-sodium salad dressing; low-sodium mayonnaise Nondairy creamer (≤1 oz/day) Unsalted nuts Low-sodium cream cheese	Bacon, bacon bits, and salt pork; regular salad dressings; snack dips made with instant soup mixes or processed cheese; canned gravies and mixes; tartar sauce, salted nuts; olives

FOOD GUIDE — 1,000-MG SODIUM DIET

F-8

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Soups	No-added-salt broths and soups made with allowed vegetables Low-sodium canned soups and broths Low-sodium cream soups made with milk allowance	Regular canned or dehydrated regular soups
Desserts and Sweets	Ice cream Low-sodium pudding Frozen yogurt (count as part of milk allowance) Fruit ice Gelatins and sherbet (not to exceed ½ cup/day) Jam; jelly Syrup	Instant puddings Commercial cake, cookie, and brownie mixes Cheesecake
Miscellaneous	Salt substitute with physician's approval Pepper; herbs; spices Vinegar Low-sodium condiments (ketchup, mustard) Lemon or lime juice Hot pepper sauce Fresh ground horseradish Salsa (¼ cup)	Any seasoning made with salt, including garlic salt, celery salt, onion salt, and seasoned salt; kosher salt Meat tenderizers Monosodium glutamate Worcestershire sauce; regular and low- sodium soy sauce; chili sauce, teriyaki sauce; barbecue sauce Most flavored vinegars Regular condiments Commercial salsa

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Honey Glazed Chicken	Unsalted Reef Tins & Noodles
Unsalted Cream of Wheat	Steamed Rice	Unsalted Green Beans
Unsalted Scrambled Egg	Steamed Broccoli	Sliced Tomato Salad
Melba Toast	Fruited Gelatin	Dinner Roll
Margarine, Jelly	Dinner Roll	Unsalted Margarine
Milk (1 cup)	Unsalted Margarine	Peach Halves
Coffee	Fresh Banana	Iced Tea
Sugar, Creamer	Milk (1 cup) Tea, Sugar	Sugar

NUTRITION MANAGEMENT OF POTASSIUM INTAKE

Description

The medical condition and nutritional requirements of the patient influence whether the dietary intake of potassium is adequate. The amount of potassium in the diet may need to be either increased or decreased, depending on the patient's condition.

Indications

A diet with an increased potassium content is prescribed to retain body potassium stores in the following:

- patients whose long-term use of potassium-losing diuretics, combined with a marginal potassium intake, contributes to potassium depletion
- patients who have increased urinary and gastrointestinal potassium losses resulting from certain diseases or conditions, eg, edema associated with certain cardiac or hepatic disorders, dehydration, the diuretic stage of nephritis

A potassium-supplemented diet may be used in conjunction with pharmaceutical potassium supplements, or alone, in individuals with a mild potassium depletion who are not able to tolerate potassium supplements. However, without supplements, it may be difficult for a patient to consistently increase dietary potassium intake over his or her usual level of intake.

A diet restricting potassium intake is usually required for patients with hyperkalemia, which commonly is caused by renal disease or certain medications. See Dietary Management in Section IG. For patients requiring a Simplified Renal Diet, refer to Simplified Renal Diet in Section IG.

Nutritional Adequacy

Increased potassium intake: The diet is planned as a Regular Diet with an increase in foods that are high in potassium. The diet is planned to meet the Dietary Reference Intakes (DRIs) as outlined in the *Statement on Nutritional Adequacy* in Section IA.

Decreased potassium intake: See Dietary Management Using the Health Food Guide for People with Chronic Kidney Disease in Section IG.

How to Order the Diet

To increase potassium intake: Order the diet as "Regular Diet with high potassium foods." If a specific potassium level is desired, specify the level in grams.

Individual potassium intake varies. To determine the patient's current potassium intake, the physician should order a nutrition consult, including a diet recall of the patient's intake of potassium. From this evaluation, the dietitian can make appropriate recommendations for the patient to increase potassium intake.

To decrease potassium intake: See Dietary Management with Renal Choice System and Simplified Renal Diet in Section IG.

Planning the Diet

To increase potassium intake, refer to the Table F-1: Potassium Content of Common Foods.

See Section IG: Modification of Protein, Dietary Management Using the Healthy Food Guide for People With Chronic Kidney Disease.

FOOD ITEM	SERVING SIZE	POTASSIUM (mg)
Dairy Products		
Cheese, American	1 oz	101
Cheese, Cheddar	1 oz	127
Ice Cream	³ /4 cup	192
Milk	1 cup	422
Yogurt, Fruited	1 cup	441
Dried Beans and Peas		
Great Northern Beans	½ cup	344
Lima Beans	½ cup	369
Pinto Beans	½ cup	397
Peas	½ cup	216
Empite		
Apricate Dried	F	241
Ranana	5 16 modium	241
Dallalla	⁷² medium 1 cup of piocos	404
Datos	1 cup of pieces	200
Granefruit	⁷⁴ cup 16 small	156
Honeydew Melon	1 cup of pieces	461
Orange Juice	¹ / ₄ cup	236
Orange	1 small 21/2-inch diameter	230
Prune Juice	¹ / ₂ cun	353
Strawherries	³ / ₂ cup	185
Watermelon	1 cup	185
watermeion	Teup	105
Vegetables		
Broccoli	¹∕₂ cup	227
Brussels Sprouts	¹ ∕₂ cup	247
Mushrooms, Cooked	½ cup	278
Potato, Baked in Skin	$1-2 1/3 \times 4 \frac{3}{4}$ inches	609
Potato, Mashed With Margarine	¹∕₂ cup	244
Spinach	½ cup	419
Sweet Potatoes	½ cup	348
Tomato, Fresh	2 slices	109
Tomato Sauce	¼ cup	226
Breads and Cereals	4.40	404
Bran Buds	1/3 cup	421
Bran Flakes	¹ / ₂ cup	123
Uatmeal, Cooked	¹ / ₂ cup	200
Raisin Bran	14 OZ (1 DOX)	184
Whele Wheet Breed	1 tDSp	134
whole wheat Bread	1 slice	26
Meats, Fish, Poultrv		
Beef; Chicken	1 oz	79 (average)
Tuna	¼ cup	89
Nuts		
Peanut Butter	2 tbsp	91
Peanuts, Dry Roasted	1 OZ	230
Pecans	1 OZ	105

Table F-1: POTASSIUM CONTENT OF COMMON FOODS

Source: USDA Handbook No. 8. Washington, DC: US Dept of Agriculture; 1986.

.

NUTRITION MANAGEMENT OF PHOSPHORUS INTAKE

Description

Phosphorus intake is limited to the prescribed level.

Indications

Hyperphosphatemia can lead to secondary hyperparathyroidism, resulting in bone disease. To prevent hyperphosphatemia, a phosphorus-restricted diet may be adjunctive to the use of agents that bind phosphorus in the gastrointestinal tract for individuals with chronic renal failure. Generally, phosphorus is restricted to 600 to 1200 mg/day. However, when a simultaneous restriction of protein is ordered, such as in renal disease, the phosphorus level is generally lowered enough to be within the desired range. With a glomerular filtration of 25 mL/min, phosphate binding substances alone are usually sufficient to control the serum phosphorus level. See Medical Nutrition Therapy for Chronic Kidney Disease in Section IG.

Nutritional Adequacy

If the phosphorus level is restricted to a level below 800 mg, the Dietary Reference Intakes (DRIs) for phosphorus will not be met. If milk products are restricted in order to achieve this level of phosphorus, the DRI for calcium, vitamin D, and riboflavin may not be met; calcium supplementation may be indicated. See Medical Nutrition Therapy for Chronic Kidney Disease in Section IG for a discussion of nutritional adequacy for patients with renal disease.

How to Order the Diet

Specify the desired intake of phosphorus in milligrams and any other restrictions, eg, _____ Diet, ___ mg phosphorus.

Planning the Diet

Generally, the phosphorus restriction can be met by:

- limiting the intake of foods containing milk
- eliminating legumes, nuts, chocolate, and cola from the diet
- substituting refined grains for whole grains

Refer to Table F-2: Phosphorus Content of Common Foods, for additional foods that may warrant restriction.

See Section IG: Moderation of Protein, Medical Nutrition Therapy for Chronic Kidney Disease.

FOOD ITEM	SERVING SIZE	PHOSPHORUS (mg)
Dairy Products		
Cheese, American	1 oz	112
Cheese, Cheddar	1 oz	143
Cheese, Cottage	½ cup	69
Ice Cream	³ ⁄4 cup	101
Milk	1 cup	250
Yogurt, Fruited	1 cup	271
Dried Beans and Peas		
Great Northern Beans	¹ / ₂ cup	145
Lima Beans	¹ / ₂ cup	100
Pinto Beans	¹ / ₂ cup	136
Peas	¹ / ₂ cup	94
Breads and Cereals		
Bran Buds	1/3 cup	218
Bran Flakes		96
Oatmeal. Cooked		122
Raisin Bran	$1 \frac{1}{4} \text{ oz} (1 \text{ box})$	132
Wheat Germ	1 thsp	162
Whole Wheat Bread	1 slice	64
Meats, Fish, Poultry		
Beef: Chicken	1 oz	65 (average)
Egg	1	90
Tuna	¹ / ₄ cup	53
Nuts		
Peanut Butter	2 thsp	103
Peanuts Dry Roasted	1 oz	100
Pecans	1 oz	86
Miscellaneous		
Cola	12 07	45
Chocolate	1 oz	40

Table F-2: PHOSPHOROUS CONTENT OF COMMON FOODS

Source: USDA Handbook No. 8. Washington, DC: US Dept of Agriculture; 1986.

NUTRITION MANAGEMENT OF CALCIUM INTAKE

Description

The medical condition and nutritional requirements of the patient influence whether the dietary intake of calcium is adequate. The amount of calcium in the diet may need to be either increased or decreased, depending on the patient's condition.

Indications

Calcium restriction may be indicated for the following:

- to control hypercalciuria
- in conjunction with overall treatment for urolithiasis

An adequate intake of calcium has been associated with a reduced risk of osteoporosis. The Dietary Reference Intakes (DRIs) includes the amount of calcium needed to reduce the risk of osteoporosis (1). However, it is difficult for many women to consume these levels without supplementation. In addition, after gastric bypass procedures, calcium supplementation will be required to maintain serum levels and prevent metabolic bone disease.

Nutritional Adequacy

Calcium-Restricted Diet: The diet is inadequate in calcium, vitamin D, and riboflavin.

Calcium-Enhanced Diet: The diet meets the DRIs as stated in the *Statement on Nutritional Adequacy* in Section IA.

How to Order the Diet

To decrease calcium in the diet: Specify the desired level of calcium intake in milligrams. Include any other necessary restrictions. Order _____Diet, _____ mg calcium.

To increase calcium in the diet above the DRI: Specify the desired level of calcium in milligrams. The DRI for calcium for males and females is as follows (1):

Age (years) Calcium (mg/day)

9 to 18	1300
19 to 50	1000
>51	1200

Planning the Diet

To restrict calcium: Eliminate milk and all milk products.

To encourage increase in calcium intake: Refer to Table F-3: Calcium Content of Common Foods, for additional foods to encourage eating. If supplementation is required, recommend supplements with calcium carbonate, since this form contains the most available amount of elemental calcium. Refer to the supplement's label to determine the actual amount of calcium, which usually is referred to as elemental calcium (2,3) Elemental calcium is highest in calcium carbonate (40%). Other calcium supplements contain lesser amounts of elemental calcium, eg, calcium phosphate (38%), calcium citrate (21%), calcium lactate (13%), and calcium gluconate (9%). To calculate the amount of elemental calcium in a supplement, identify the number of milligrams the supplement contains. For example: 1 pill of 650 mg of calcium carbonate [650 mg x 40%] provides 260 mg of elemental calcium (2). Approximately 4 tablets per day of calcium carbonate are needed to meet the RDI for most age-groups. Calcium supplements of 1200 to 1500 mg/day should be provided to all patients after gastric bypass surgery (Roux-en-Y and bilio-pancreatic diversion (BPD)) (4). In the cases of gastric bypass, calcium citrate with vitamin D is the preferred preparation because it is more soluble than calcium carbonate in the absence of gastric cacid production (5). For patients with the BPD procedure who have clinical steatorrhea, a high dose calcium supplementation regimen (2000 mg/day) along with monthly intramuscular vitamin D is recommended to reduce the risk of metabolic bone disease (1).

Adequate intake or synthesis of vitamin D is critical to ensure adequate absorption of calcium. The DRI for vitamin D for men and women is as follows (1):

 Age (years) Vitamin D (IU)

 19 to 50
 200

 51 to 70
 400

 ≥71
 600

Although vitamin D is synthesized in the skin from exposure to sunlight, studies have shown that older adults usually do not have adequate exposure to sunlight to synthesize the necessary vitamin D. This problem is compounded by increased use of sunscreens with high sun protection factors and an inefficiency of the skin to manufacture vitamin D as adults age. For adults over 50 years of age and younger adults who spend little time outside, it may be advised to take a daily multivitamin with vitamin D (which typically contains 400 IU of vitamin D) (6-8).

FOOD ITEM	SERVING SIZE	CALCIUM (mg)	
Milk and Dairy Products			
Cheese			
American	1 oz	174	
Cheddar	1 oz	204	
Cottage, Creamed	1 oz	68	
Mozzarella, Part Nonfat	1 oz	183	
Parmesan Cheese	1 tbsp	70	
Swiss	1 oz	272	
Hot Cocoa	1 cup	106-300	
Ice Cream	½ cup	88	
Ice Milk	½ cup	102	
Milk, Whole, Nonfat, Chocolate	1 cup	287-300	
Pudding	½ cup	125-187	
Sherbet	½ cup	52	
Yogurt, Fat-Free	8 oz	314	
Yogurt, Frozen	4 oz	105	
Yogurt, Fruit-Flavored	8 oz	415	
Yogurt, Plain	8 oz	415	
Fich			
Fish Sardines Canned With Bones	1 07	101	
Salmon Canned With bones	1 02	74	
Samon, canned with bones	1.02	7 1	
Vegetables			
Broccoli	½ cup cooked	89	
Collard Greens	½ cup cooked	178	
Kale	¹ / ₂ cup cooked	90	
Turnin Greens	¹ / ₂ cup cooked	125	
rump dreens	72 cup cooncu	120	
Legumes			
Great northern beans	1 cup cooked	121	
Navy beans	1 cup cooked	128	
Pinto beans	1 cup cooked	82	
Fruits			
Dried figs	5	258	
Calcium-fortified orange juice	1 cup	300	

Table F-3: CALCIUM CONTENT OF COMMON FOODS

Sources: USDA Handbook No. 8. Washington DC: US Dept of Agriculture; 1986.

Position of The American Dietetic Association and Dietitians of Canada: vegetarian diets. JAm Diet Assoc. 003;103(6):748-765.

Nutrition Management of Calcium Intake

References

- 1. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride*. Washington, DC: National Academy Press; 1997.
- 2. Calcium Supplements (Systemic). MedlinePlus Health Information Available at
- http://www.nlm.nih.gov/medlineplus/druginfo/uspdi/202108.html. Accessed September 16, 2002.
- 3. National Osteoporosis Foundation. How calcium helps. Available at: http://www.nof.org/other/calcium.html. Accessed April, 27, 1998.
- 4. Kushner R. Managing the obese patient after bariatric surgery: a case report of severe malnutrition and review of the literature. *JPEN*. 2000;24:126-132.
- 5. Levenson DI, Bockman RS. A review of calcium preparations. Nutr Rev. 1994;52:221-232.
- 6. National Osteoporosis Foundation. How can I prevent osteoporosis? Available at: http://www.nof.org/PreventOsteo.html. Accessed April 27, 1998.
- 7. Thomas MK, Lloyd-Jones DM, Thadhani RI, Shaw AC, Deraska DJ, Kitch BT, Vamvakas EC, Dick IM, Prince RL, Finkelstein JS N Engl J Med. 1998;338:777-783.
- 8. Malabanan A, Veronikis IE, Holick MF. Lancet. 1998;351:805-806. Letter.

I. NORMAL NUTRITION AND MODIFIED DIETS G. Modification of Protein

Protein-Controlled Diet for Acute and Refractory Hepatic Encephalopathy	G-1
Sample Menu	G-3
Protein-Based Exchanges	G-5
Medical Nutrition Therapy for Chronic Kidney Disease	G-7
Table G-1: Definition and Stages of Chronic Kidney Disease	G-7
Table G-2: Recommended Lipid Levels for Adults With Chronic Kidney Disease	G-11
Table G-3: Daily Nutritional Requirements for Adults With Renal Disease Based on Ty	pe Of
Therapy	G-15
Tables G3.1-3.5: Healthy Food Guide (Pre-ESRD)	G-18
Tables G4.1-G4.6: Healthy Food Guide For People On Dialysis	G-23
Meal Patterns Using Healthy Food Guide	G-27
Simplified Renal Diet	G-28

PROTEIN-CONTROLLED DIET FOR ACUTE AND REFRACTORY HEPATIC ENCEPHALOPATHY

Description

Adjustment of the amount and type of protein characterizes the Protein-Controlled Diet for Hepatic Encephalopathy. Energy and protein are provided to attempt maintenance of nitrogen balance and support liver regeneration.

Indications

The diet is used in the treatment of acute and refractory hepatic encephalopathy associated with hepatic disorders, which may include the following:

- hepatitis
- cholestatic liver disease
- cirrhosis with acute and/or chronic encephalopathy

Liver disease causes numerous metabolic problems that can affect all major nutrients and the assessment parameters commonly used to evaluate nutritional status of the patient with hepatic disease. The classic signs of liver disease are anorexia, weight loss, and nausea with marked deficiencies in energy, protein, vitamins, and minerals (1,2). Because of the high risk for malnutrition in persons with hepatic diseases the American Society for Enteral and Parenteral Nutrition (ASPEN) recommends protein restriction be no less than 0.6 to 0.8 g/kg and reserved to those patients during acute or refractory episodes of encephalopathy. Normal protein intake should be resumed of 1 to 1.2 g/kg after the cause of encephalopathy has been identified and treated (3). The widespread practice of protein restriction for all patients with cirrhosis is not justified and often leads to iatrogenic protein malnutrition (3).

Although malnutrition does not correlate with the type of liver disease, therapeutic modifications vary according to the type and severity of hepatic insufficiency. Generally, fatty liver requires little to no nutrition intervention, while cirrhosis necessitates major changes in the patient's food intake. A major goal of medical nutrition therapy in liver disease is to prevent and treat hepatic encephalopathy (1,3).

Hepatic disease can profoundly affect the nutritional status of the patient because of its effects on carbohydrate, fat, protein, vitamin, and mineral metabolism. Metabolic disorders of the following are commonly seen in the clinical setting of patients with hepatic insufficiency:

- Carbohydrates: Adverse effects can include hypoglycemia or hyperglycemia. Hypoglycemia is most frequently seen in acute hepatitis or fulminant liver disease, probably due to impaired gluconeogenesis (1,3). Hyperglycemia is commonly observed secondary to counteracting catabolic hormones and insulin resistance when superimposed by acute stress and injury (1). Soluble fiber may be beneficial in managing hepatic encephalopathy. Soluble fiber is fermented in the colon by the same mechanism as lactulose, which eliminates ammonia in the form of ammonium ion and bacterial proteins (3).
- Fats: Malabsorption may occur because of inadequate production of bile salts. This may lead to steatorrhea, which could lead to deficiencies in fat-soluble vitamin and calcium levels. Researchers have found an increase in serum lipids, reflecting lipolysis (1,3).
- Protein: The effect of hepatic injury on protein metabolism is more dramatic than is carbohydrate or fat metabolism. There is a decrease in synthesis of serum albumin, the transportation of proteins, and the clotting factors (1,3). The ability of the liver to synthesize urea decreases, which results in an accumulation of ammonia and a decrease in serum urea level. This derangement in metabolism elevates the serum aromatic amino acids (AAAs) (phenylalanine, tryptophan, and tyrosine) and methionine and decreases the serum branched-chain amino acids (BCAAs) (valine, isoleucine, and leucine). The only enzymes that metabolize AAAs are located in the hepatocytes. In hepatic insufficiency, there is a decrease in hepatic oxidation of AAAs, leading to an increase in circulation of AAAs in the plasma. In contrast, BCAAs are metabolized primarily by the skeletal muscle. There is an increase in BCAA oxidation in the peripheral tissue during stress, causing a drop in plasma circulation (1).

Protein-Controlled Diet

• Vitamins and minerals: Hepatic injury results in decreased absorption, transport, and storage and may alter the metabolism of vitamins and minerals. Cirrhotic livers have been reported to store decreased levels of thiamin; folate; riboflavin; niacin; pantothenic acid; vitamins B₆, B₁₂, and A; zinc; and cobalt (1.4). In chronic liver disease, the hydroxylation of dietary and endogenous vitamin D to the active form (25-hydroxy derivative) is impaired and may lead to a deficiency state with concomitant osteomalacia. Although there are possibilities of vitamin and mineral deficiencies, supplementation should be administered only when a specific nutrient deficiency is identified. Supplementation should be monitored. Vitamin K deficiency may be induced from malabsorption with steatorrhea, dietary deficiency, impaired hepatic storage, and/or decreased production of gut flora due to intake of antibiotics. If vitamin K deficiency occurs, the rate at which prothrombin is converted to thrombin is affected, thus hampering the coagulation process and producing inadequate clotting factors (1). Intravenous or intramuscular vitamin K often is given for 3 days to rule out hypopothrombinemia due to deficiency (4).

Nutritional Adequacy

Diets containing less than 50 g of protein may be inadequate in thiamin, riboflavin, calcium, niacin, phosphorus, and iron based on the Statement on Nutritional Adequacy in Section IA. Supplementation may be indicated but should be assessed on an individual basis. This diet should be considered a transitional diet. Normal protein intake should be resumed soon after the cause of encephalopathy has been identified and treated. Long-term protein restriction should only be considered in patients with refractory encephalopathy (3).

How to Order the Diet

The diet order should specify the grams of protein required from food. Base the grams of protein ordered on the patient's actual weight or use ideal body weight in cases where weight cannot be measured or accurately accessed due to fluid issues (e.g, with ascites). To calculate weight, see Section II (Estimation of Energy Expenditures, or Weight for Height Calculation – 5' Rule). If a special formula is requested, the amount should be specified. Specify any restriction such as sodium, fluid, or other nutrients.

Planning the Diet

The table below outlines the recommended nutrient prescription according to type of hepatic disease (3,5,6).

Type of Hepatic Disease	Nutrient Prescription
Fatty liver/steatosis	Abstinence from ethanol Weight reduction, if attributable to obesity Reduced energy and dextrose intake, especially if patient is receiving total parenteral nutrition (PN)
Hepatitis (acute/chronic/alcoholic)	Energy: 30 – 35 kcal/kg Protein: 1 – 1.2 g/kg 50 mg elemental Zinc may improve hepatic fibrosis ⁽³⁾
Cirrhosis (uncomplicated)	Energy: $30 - 35 \text{ kcal/kg}$ or RMR x 1.2 to 1.4 ⁽³⁾ Protein: $1 - 1.2 \text{ g/kg}$ Supplementing 50 mg elemental Zinc may improve hepatic fibrosis ⁽³⁾ Evaluate Vitamin D and thiamine for supplementation ⁽³⁾
Cirrhosis (complicated)	Energy: 30 – 35 kcal/kg or RMR x 1.2 to 1.4 ⁽³⁾ Protein: 1 – 1.5 g/kg (with malnutrition) Supplementing 50 mg elemental Zinc may improve hepatic fibrosis ⁽³⁾ Evaluate Vitamin D and thiamine for supplementation ⁽³⁾
Esophageal varices	Liberal diet consistency, normal consistency is encouraged as tolerated

Type of Hepatic Disease	Nutrient Prescription
Ascites	Sodium restriction: 2 g/day with diuretics Fluid restriction: use clinical judgment Fat-soluble vitamin supplement up to 100% RDA may be necessary in cholestatic cirrhosis (see steatorrhea)
Hepatic encephalopathy	Energy: 35 kcal/kg Protein: 0.6 – 1.2 g/kg. Start at 0.6 g/kg per day and progress to 1 – 1.2 g/kg as tolerated. Do not give products enriched with glutamine. Consider high soluble fiber diet. Zinc may be beneficial.
Hepatic coma	Use tube-feeding Protein: Start at 0.6g/kg per day and progress to 1 – 1.2 g/kg day as tolerated. Do not give products enriched with glutamine.
Steatorrhea >10 g/day or Cholestatic liver disease with weight loss	 Fat: 40 g/day (long-chain triglycerides) or < 30% total fat intake, Supplement with medium-chain triglycerides to provide additional energy. Oral supplement with calcium, 1,25 hydroxy-vitamin D, and calcitonin may be required. May require supplementation of fat-soluble vitamins.

Meal size and frequency: Some patients require small portions and frequent feedings because ascites limits the capacity for gastric expansion. Studies have shown that the metabolic profile after an overnight fast in patients with cirrhosis is similar to normal individuals undergoing prolonged starvation without any associated stress. Cirrhosis can be considered a disease of accelerated starvation with early recruitment of alternative fuels. A small-scale study showed patients with cirrhosis who received an evening snack to supply energy during sleeping hours were able to maintain a greater positive nitrogen balance than did other patients who were fed less frequently (2).

Commercial supplements: Supplementation with enteral formulas is often necessary to increase the patient's intake. Modular products of carbohydrates and fat can increase energy intake without increasing protein intake. The usefulness of special products containing BCAAs is controversial, and these products generally have a higher cost. The guidelines for nutrition therapy in liver disease developed by the American Society for Enteral and Parenteral Nutrition (ASPEN) restrict the use of BCAA enriched formulas to patients with refractory encephalopathy not responding to medical therapy (7).

SAMPLE MENU					
Brookfast	(50 g of protein)				
Orange Juice (½ c)	Garden Green Salad (1 oz)	Cranberry Juice Cocktail (½ cup)			
Oatmeal (½ c)	with Dressing (1 Tbsp)	Oven Fried Chicken (2 oz)			
Toast (2 slices)	Roast Beef Sandwich	Buttered Rice (½ c)			
Margarine (2 tsp)	Roast Beef, Shaved (1 oz)	Seasoned Green Beans (1/2 c)			
Jelly (1 Tbsp)	Bread (2 slices)	Dinner Roll (1)			
Milk (½ c)	Mayonnaise (2 Tbsp)	Margarine (2 tsp)			
Sugar	Sliced Tomato (1 oz)	Sliced Peaches (½ c)			
Coffee; Tea	Fresh Fruit Salad (½ c)	Lemonade			
Nondairy Creamer	Fruit Punch				
Snack	Snack	Snack			

Snack	Snack	Snack
Hard Candy (6 pieces)	Fruit Ice (3 oz)	Banana (1)
Jelly Beans (1 oz)		Dry Cereal (¾ oz)
		Milk (½ c)

References

Manual of Clinical Nutrition Management

^{1.} Wong K, Klein B, Fish J. Nutrition Management of the Adult with Liver Disease. In: Skipper A, ed. *Dietitian's Handbook of Enteral and Parenteral Nutrition*. 2nd ed. Gaithersburg, Md: Aspen Publishers; 1998.

^{2.} Levinson M. A practical approach to nutritional support in liver disease. *Gastroenterologist*. 1995;3:234-240.

^{3.} Frazier TH, Wheeler BE, McClain CJ, Cave M Liver Disease. In: Mueller CM, ed. A.S.P.E.N. Adult Nutrition Support Core Curriculum,

Protein-Controlled Diet

2nd ed.. Silver Spring , Md: American Society of Parenteral and Enteral Nutrition. ; 2012 (pages 454-469.

- 4. Hasse JM, Matarese LE. Medical nutrition therapy for liver, biliary system, and exocrine pancreas disorders. In: Mahan K, Escott-Stump E, eds. *Krause's Food, Nutrition and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders; 2000:710.
- 5. Corish C. Nutrition and liver disease. *Nutr Rev.* 1997;55:17-19.
- 6. Shronts EP, Fish J. Hepatic failure. In: Merrit RJ, ed. *The A.S.P.E.N. Nutrition Support Practice Manual*. Silver Spring, Md: Aspen Publishers;1998.
- 7. ASPEN Board of Directors and the Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients [published erratum appears in JPEN J Parenter Enteral Nutr. 2002;26:144]. JPEN J Parenter Enteral Nutr. 2002;26 (suppl 1):1SA-138SA.

PROTEIN-BASED EXCHANGES

Exchange	Protein (g)	
Meat and Meat Substitutes	7	
Milk	4	
Starch/Bread	2.5	
Vegetables	2	
Fruit	Negligible	
Low-Protein Products	0.2	
Sweets	Negligible	
Fats	Negligible	

	Portion	Variances in Portion or
		Protein Content (0.2 g) Noted
Meat and meat substitutes (7 g protein)		
Egg	1 large	1 medium egg = 5.7 g
Cheese (natural hard or semisoft)	1 oz	
Cheese, processed (eg, American)	1 oz	6.6 g
Cottage Cheese	¼ cup	
Meat, fish, poultry (lean portion, cooked)	1 oz	
Meat (ground or flaked)	¼ cup (1 oz)	
Legumes (cooked):	½ cup	
Black beans		7.6 g
Garbanzo beans		7.3 g
Kidney beans		6.7 g
Lentils		8.9 g
Lima beans		7.3 g
Pinto beans		7 g
Black-eyed peas (Cow Peas)		5.7 g
Peanut butter	2 Tbsp	7.9 g
Milk (4 g protein)		
Cream, Half-and-Half	½ cup	3.6 g
Cream, light	½ cup	3.3 g
Cream, heavy (whipping)	³₄ cup	3.7 g
Cream, heavy (fluid)	³ ⁄4 cup	3.6 g
Cream cheese	2 Tbsp	2.1 g
Milk, whole, low-fat, nonfat, or chocolate	½ cup	
Yogurt, fruited	½ cup	4.5 g
Yogurt, plain, low-fat, vanilla	1/3 cup	
Custard	1/3 cup	
Pudding	$\frac{1}{2}$ cup	
Starch/Bread (2.5 g protein)	•	
Bread, white, rye, whole wheat	1 slice	
Biscuit	1	Approx. 1 oz biscuit = 2 g
Cereal (cooked)		
Cream of rice	6 oz	1.6 g
Farina	6 oz	2.6 g
Grits	6 oz	2.7 g
Maltex	4 oz	2.9 g
Oatmeal	4 oz	3 g
Ralston	4 oz	2.8 g
Rolled wheat	4 oz	2.5 g
Wheatena	4 oz	2.8 g
Cereal (ready-to-eat)	-	0
40% Bran Flakes	1 oz	3.6 g

	Portion	Variances in Portion or Protein Content (0.2 g) Noted	
Corn flakes	1 box (¾ oz)	1.7 g	
Crisp rice	1 box (5/8 oz	1.2 g	
Puffed rice	½ oz	0.9 g	
Puffed wheat	½ oz	2.1 g	
Shredded wheat	1 oz	3.1 g	
Crackers		0	
Graham	4 squares	2.3 g	
Saltines	6	3 g	
Muffin, corn	1	Approx. $1\frac{1}{2}$ oz = 2.8 g	
Pasta, rice, noodles (cooked)	⅓ cup		
Ice cream	¹ / ₂ cup	2.4 g	
Ice milk	¹ / ₂ cup	2.6 g	
Starchy Vegetables (2.5 g protein)		C C	
Corn	½ cup		
Peas, green	½ cup	½ cup = 4.1 g	
Potato (baked)	1 (5 oz)	3.2 g	
Potatoes, french fried (2 – 3 inches long)	10	3.2 g	
Potato (mashed)	⅓ cup	2 g	
Potato (peeled and boiled)	1 small (5 oz)	-	
Sweet potato or yam (canned)	½ cup		
Winter squash	½ cup	1.5 g	
Other Vegetables (2 g protein)	-	-	
All others (cooked)	⅓ cup		
Except those in <i>Starch/Bread</i> and	-		
Fruits (nogligible protoin)			
All			
Low-Protein Products (each exchange contains 0.2	g protein)		
Low-protein bread	1 slice (1½ oz)		
Low-protein rusks	2 slices		
Low-protein macaroni or noodles	½ cup, cooked (¼ cup dry)	
Low-protein gelatin	½ cup, prepared	l (negligible protein)	
Low-protein cookies	2		
Sweets (negligible protein)	Fats (negligible	e protein)	
Candy: hard candy, lollipops, jelly beans, gum	Butter or Mai	rgarine	
drops, marshmallows	Oil or Shorter	ning	
Carbonated beverages	Mayonnaise		
Lemonade; Limeade	Salad Dressing (except sour cream based or cream		
Noncarbonated soft drinks	cheese)		
Jam; jelly	Nondairy Cre	Nondairy Creamer	
Popsicles; fruit ice, italian ice	Gravy (meat drippings with fat, thickened with		
Sugar; syrup; honey	cornstarch)	

MEDICAL NUTRITION THERAPY FOR CHRONIC KIDNEY DISEASE

Description

The approach to medical nutrition therapy for chronic kidney disease (CKD) is based on the stage and progression of kidney disease, comorbid conditions (eg, diabetes mellitus, hypertension, or cardiovascular disease), and renal replacement therapy (RRT). Medical nutrition therapy and nutrition intervention are provided based on the individualized needs of the patient. The dietary approach is modified in one or more of the following constituents: protein, sodium, potassium, total fluid, and phosphorus. The diet may also be modified to provide adequate amounts of energy, vitamins, and minerals. The Academy of Nutrition and Dietetics' *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* and recommendations from the National Kidney Foundation serve as a framework for providing care to renal patients based on disease stage and the requirement for RRT (1,2). These guidelines and resources, in addition to the guidelines cited below, are consistent with the language and terms used for reimbursement in Medicare beneficiaries.

Indications

Management of CKD: This disease causes a progressive reduction in renal function for more than 3 months that results in a reduced ability to control body water volume, acid-base balance, hormonal regulation, and electrolyte concentrations (3). The five stages of CKD are described in Table G-1: Definition and Stages of Chronic Kidney Disease (2). Stage 1, which is the least severe stage, is characterized by kidney damage with a glomerular filtration rate (GFR) greater than 90 mL/min per 1.73 m²; stage 5, the most severe stage, is characterized by kidney failure with a GFR less than 15 mL/min per 1.73 m² (3). The leading causes of CKD are diabetes mellitus and hypertension, which account for 65% to 70% of all new cases of end-stage renal disease requiring RRT (3). Other causes of CKD include vascular disease, urologic disorders, and primary glomerular or interstitial kidney diseases (3). Symptoms of uremia, such as nausea, anorexia, and altered taste sensation, can lead to reduced oral intake and an increased risk of malnutrition in patients with CKD (3,4). The goals for dietary management in CKD are to minimize uremic toxicity, prevent wasting and malnutrition, and complement the prescribed RRT regimen.

GFR	With Kidney Damage*		Without Kidı	ney Damage*
(mL/min/1.73 m ²)	With HBP**	Without HBP**	With HBP**	Without HBP**
≥90	1	1	"High Blood Pressure"	"Normal"
60-89	2	3	"High Blood Pressure with↓ GFR"	"↓GFR" ^α
30-59	3	3	3	3
15-29	4	4	4	4
<15 (or dialysis)	5	5	5	5

Table G-1: Definition and Stages of Chronic Kidney Disease

Shaded area represents chronic kidney disease; numbers designate stage of chronic kidney disease.

*Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies. **High blood pressure (HBP) is defined as ≥140/90 mm Hg in adults and >90th percentile for height and gender in children. «May be normal in infants and the elderly.

8. Resource: Definition and Stages of Chronic Kidney Disease. In: Part 4. Definition and Classification of Stages of Chronic Kidney Disease. KDOQI Clinical Practice Guidelines for Chronic Kidney Disease Evaluation, Classification and Stratification. Available at http://www.kidney.org/professionals/kdoqi/guidelines_ckd/p4_class_g1.htm. Accessed January 10, 2011.

Typically, CKD progresses until treatment by RRT (dialysis) or transplantation is required. Dietary modifications and practice guidelines outlining the scope of nutrition therapy are based on the classification or stage of the disease (1,2). Patients with CKD are generally classified in two groups (2,3):

- Patients in stages 1 to 5 who do not yet require RRT; management is primarily by diet modifications and medication, or
- Stage 5 patients who require RRT (hemodialysis, peritoneal dialysis, or other types of RRT).

This section focuses on medical nutrition therapy for CKD. The Academy's *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* provides the most current recommendations for CKD patients (stages 1 to 5) who are managed primarily by diet modifications and do not require dialysis (1). For stage 5 patients who require RRT, the clinician should refer to recommendations outlined in the National Kidney Foundation's *Pocket Guide to Nutrition Assessment of the Patient with Chronic Kidney Disease* (2). Both of these resources provide specific guidelines for the nutrition care of adult kidney transplant recipients (1,2). The American

Kidney Foundation has published diagnostic criteria and management strategies for acute kidney injury, which replaces the term *acute renal failure*. Nutrition intervention strategies for acute kidney injury are described in Table G-3 of this section and in Section III: Nutrition Management of Acute Kidney Injury and Chronic Kidney Disease.

RRTs

The three primary types of RRTs are hemodialysis, peritoneal dialysis, and continuous RRT (2,3).

Hemodialysis: Hemodialysis accounts for 80% of all RRTs used to manage the complications of renal disease (3). Hemodialysis uses an artificial kidney (hemodialyzer) to cleanse the blood. This process returns the body to a more normal state by removing excess fluid and waste products. However, it does not replace the endocrine functions of the kidney. The average treatment lasts 3 to 5 hours and is performed three times a week. Treatment is based on adequate urea clearance to equal a urea reduction goal of greater than 65% or a Kt/V (clearance of the dialyzer × time/volume) of greater than 1.2 (2,3). Hemodialysis removes some watersoluble vitamins such as vitamin C, folic acid, and pyridoxine. Hemodialysis also removes certain minerals and electrolytes. Potassium is efficiently removed by hemodialysis, while phosphorus and magnesium are removed to a lesser degree (3). Hemodialysis may increase energy requirements because of lymphocyte stimulation and complement activation (3). Hemodialysis can be performed at a dialysis center or at home. There are two types of more frequent hemodialysis, short daily dialysis and nocturnal dialysis. These types of hemodialysis provide more treatment time with fewer side effects and risks; patients who receive more frequent hemodialysis may consume a more liberal diet than patients who receive thrice weekly conventional hemodialysis (2).

Peritoneal dialysis: This type of dialysis involves the removal of waste products and water within the peritoneal cavity by using the peritoneal membrane as a filter. The dialysis solution (dialysate) is instilled through the peritoneal catheter into the peritoneal cavity or peritoneum. The many blood vessels and capillaries throughout the peritoneum are separated from the peritoneal cavity by a layer of mesothelium. Passive movement from the peritoneal capillaries into the dialysate removes the uremic toxins. The high osmolality of the dialysate due to the high dextrose concentration results in the removal of extracellular fluid. This type of RRT may place the patient at increased risk for infection (eg, peritonitis) and hyperglycemia (3). There are two major types of peritoneal dialysis:

- Continuous ambulatory peritoneal dialysis (CAPD) is a manual dialysis process, whereby a continuous presence of a dialysate in the peritoneal cavity is interrupted intermittently for drainage and instillation of fresh dialysate. The dialysate is usually exchanged four times a day, with only a 30- to 35-minute interruption of daily activity during each exchange. The dialysate remains in the peritoneal cavity for 3½ to 4 hours during the day and 8 to 10 hours at night.
- Continuous cyclic peritoneal dialysis (CCPD) or automated peritoneal dialysis provides more daytime freedom and a decreased risk of infection by decreasing the number of catheter connections to two per day. A cycler machine delivers three or four dialysate exchanges each night, lasting 2½ to 3 hours each. Approximately 2 L of dialysate remains in the peritoneal cavity during the day for 12 to 15 hours and is then drained when the patient begins the nightly routine.

Intermittent peritoneal dialysis is also available; however, it is not a standard treatment.

Patients who receive peritoneal dialysis may develop hypokalemia, since commercially available solutions do not contain potassium (3). Potassium can be liberalized in the diet or supplemented orally if needed. The peritoneal dialysate can provide a substantial amount of energy from glucose to the patient when hypertonic solutions are needed for increased fluid removal (3). Diabetic patients may have a greater risk for hyperglycemia, and all patients can develop hypertriglyceridemia. A low-sodium, fluid-restricted diet can help eliminate the need for higher dextrose concentration solutions. The amount of total energy absorbed depends on the infusion volume, dwell time, and dextrose concentration (3). (See Determination of Glucose Absorption in Peritoneal Dialysis later in this section.) The nutritional intake of patients who receive peritoneal dialysis may be affected by bloating, abdominal fullness, and loss of appetite due to the indwelling dialysate (3,4). The protein needs of patients who receive peritoneal dialysis are increased, and it is important to encourage a high-protein diet to minimize the risks of malnutrition and infection. Some patients may require protein or protein-energy supplementation to meet their daily estimated protein needs of 1.2 to 1.3 g/kg (2).

Continuous RRT: Continuous RRTs include continuous venovenous hemofiltration, continuous venovenous hemodialysis, continuous hemodiafiltration, and slow continuous ultrafiltration (3). Each method uses a

different mechanism for fluid and solute clearance (3). Continuous arteriovenous hemofiltration and continuous arteriovenous hemodialysis rely on arterial and venous cannulation and the patient's own blood pressure to provide the gradient for blood flow and dialysis (3). Although continuous RRT has a lower clearance rate than standard hemodialysis, it provides superior clearance of fluids, nitrogenous waste, and other metabolic by-products and improved hemodynamic stability because the process is continuous (3). This procedure is often used in the critical care setting when patients are hemodynamically unstable. In addition. continuous RRT has become the preferred method of dialysis for acute kidney injury in some centers (3). As compared to hemodialysis, continuous RRT is more expensive, immobilizes the patient, and requires continuous anticoagulation (3). The amount of nitrogen loss is dependent on the type of continuous RRT; however, the differences in losses between methods are not significant (3). Daily protein requirements for adults on continuous RRT therapy are between 1 and 2.5 g/kg (3.4). The daily protein requirement for patients who receive continuous arteriovenous hemofiltration is from 1.5 to 1.8 g/kg, because the losses of small peptides and amino acids can be high (3,5). Fluid losses can be as great as 20 L/day, therefore fluid replacement is necessary to prevent hypovolemia, and electrolytes should be frequently monitored (3). Energy needs for acute kidney injury patients receiving continuous RRT are approximately 1.3 times greater than their resting energy expenditure requirements, or an average of 25 to 35 kcal/kg per day (3). The dialysate and replacement fluids used in some types of continuous RRT provide dextrose, therefore dextrose energy should be included in calculations determining total energy (3).

Transplantation

A transplant offers a relatively favorable long-term outlook and improves the quality of life for many individuals with CKD, especially young children. A functioning transplanted kidney performs the excretory and regulatory functions of a normal kidney. Successful transplantation frees the patient from the time-consuming demands of dialysis and a strict dietary regimen. Refer to the Academy of Nutrition and Dietetics' *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* and the National Kidney Foundation's *Pocket Guide to Nutrition Assessment of the Patient with Chronic Kidney Disease* for specific nutrition guidelines before and after transplantation for adults (1,2).

Nutritional Adequacy

Because individual diets for renal disease vary widely as to the nutrients controlled, a general statement on nutritional adequacy is not given. Refer to statements for each constituent in the respective sections:

Section IF: Nutrition Management of Potassium Intake
 Sodium-Controlled Diet
 Nutrition Management of Phosphorus Intake
 Section III: Clinical Nutrition Management, Management of Acute Kidney Injury and Chronic Kidney Disease

How to Order the Diet

Refer to the "How to Order the Diet" instructions for each of the components required in the respective chapters. See Nutritional Adequacy in this section. Also, refer to Nutrition Management of Fluid Intake in Section IA.

Nutrition Assessment and Nutrition Intervention

Planning the Diet

Refer to Table G-3: Daily Nutritional Requirements for Adults with Renal Disease Based on Type of Therapy.

Body weight estimates are used to calculate the nutritional needs of patients with CKD (1). There are no standard reference norms in the CKD population (including kidney transplant recipients). Therefore, the registered dietitian should use clinical judgment to determine which data to include in estimations of body weight, including actual measured weight; history of weight changes; serial weight measurements, and adjustments for the suspected impact of edema, ascites, and polycystic organs (Grade IV)* (1). Body weight can be difficult to determine because as kidney function decreases, the ability to regulate fluid balance may be compromised, and multiple factors must be considered (1). A variety of published weight norms can be used in the anthropometric assessment of individuals with CKD (including kidney transplant recipients); however, each norm has significant drawbacks and should be used with caution (Grade IV) (1). The assessment of standard body weight can be derived from National Health and Nutrition Examination Survey (NHANES II) weight table, which is based on sex, age, and frame size. (Refer to Section II: Standard Body Weight (SBW) Determination Based on NHANES II.) Energy, protein, and trace mineral recommendations use standard body weight as the basis for determining the nutrient requirements for CKD patients (1,2,5,6). Although standard

body weight data is validated and standardized and uses a large database of ethnically diverse groups, data provides only actual weights, not ideal weights for the reduction of morbidity and mortality (Grade IV) (5).

Energy

CKD (stages 1 to 5): The energy requirements of CKD patients who do not receive dialysis are similar to the requirements of healthy individuals and are influenced by age, sex, and physical activity level (1.5). Resting metabolic rates determined by direct and indirect calorimetry are similar for patients with CKD and healthy control subjects. Studies of individuals who consumed less than 0.8 g protein per kg of ideal body weight reported a neutral or positive nitrogen balance when energy intakes were between 35 and 45 kcal/kg of ideal body weight and a negative nitrogen balance when energy intakes were 15 to 25 kcal/kg of ideal body weight (1,7). Therefore, energy intakes should be greater for patients who consume less than the Recommended Daily Allowance for protein (Grade 1) (1). Five randomized controlled trials published between 2001 and 2007 examined patients with a normal body weight and found that a total energy intake of 23 to 35 kcal/kg of body weight (when consuming a protein-restricted diet with a daily protein intake of 0.3 to 0.7 g/kg of body weight) is adequate to maintain a stable body mass index in adult nondiabetic patients with CKD (Grade II) (1). Overweight patients with CKD and type 1 or type 2 diabetes who receive a total energy intake of 1,780 to 1,823 kcal (when consuming protein-restricted diets with a daily protein intake of 0.68 g to 0.86 g/kg body weight) can decrease body weight without signs of malnutrition (Grade II) (5). When prescribing energy requirements for persons with CKD, the primary goals should be to provide an adequate amount of total energy to maintain or achieve a reasonable body weight and positive nitrogen balance (1). Based on this emerging evidence, the 2010 Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline recommends a daily energy intake of 23 to 35 kcal/kg body weight for all adults with CKD, including patients who have recovered from kidney transplantation surgery (Grade II)(1). The registered dietitian should consider weight status, nutrition goals, age, sex, physical activity level, and metabolic stressors when determining energy requirements (Grade II)(1).

Hemodialysis: For patients 60 years and older with stage 5 disease who receive dialysis, an energy intake of 30 to 35 kcal/kg of ideal body weight or standard body weight is suggested (2,5,6). For patients younger than 60 years of age, energy needs should be calculated at a minimum of 35 kcal/kg of ideal body weight or standard body weight (2). The Academy has explored evidence regarding the accuracy and application of methods to measure energy expenditure. For additional information, refer to Section II: Estimating Energy Expenditure.

Peritoneal dialysis: In peritoneal dialysis, glucose is absorbed from the dialysate. Therefore, the dietary energy intake may need to be decreased to prevent excess weight gain and obesity. Glucose absorption varies among patients due to differences in peritoneal permeability. Some patients who receive CAPD or CCPD absorb more than 800 kcal/day from the dialysate, depending on the exchange concentrations (5). (See Determination of Glucose Absorption in Peritoneal Dialysis later in this section.) Energy absorbed from the dialysate should be subtracted from the daily energy intake from the diet (2,5,6).

Protein

CKD (stages 1 to 5): The Modification of Diet in Renal Disease (MDRD) trial found that a low protein intake reduces intraglomerular pressure, solute load, and overall nephron activity and may preserve renal function or delay its progressive decline. (3,6,7). However, more recent trials with larger samples of subjects and a longer duration (up to 2 years) have demonstrated that a low-protein diet (0.6 to 0.897 g/kg of body weight per day without ketoacid supplementation) did not significantly alter the decline in GFR when compared to a typical level of protein intake (1.0 to 1.4 g/kg per day), regardless of the stage of CKD or the type of diabetes among patients with diabetic nephropathy (Grade II)(1). A few studies have demonstrated that protein-restricted diets (0.7 g dietary protein per kg of body weight per day) with adequate total energy intake can slow the GFR decline and maintain a stable nutrition status in adult nondiabetic patients with CKD (Grade II)(1,7).

Nutrition practice guidelines recommend that the protein intake be based on the patient's creatinine clearance, estimated GFR, and urinary protein losses (1,2,4,6). A protein-controlled diet with a daily protein intake of 0.6 to 0.8 g/kg of body weight is recommended for nondiabetic adults with CKD who are not on dialysis and have an estimated GFR that is less than 50 mL/minute/1.73 m² (Grade II)(1). Clinical judgment should be used when recommending lower protein intakes; the patient's level of motivation, willingness to participate in frequent follow-up testing, and risk for protein-energy malnutrition should be considered (Grade II) (1).

Diabetic nephropathy: For adults with diabetic nephropathy, a protein-controlled diet providing a daily protein intake of 0.8 to 0.9 g/kg of body weight is recommended by the 2010 *Chronic Kidney Disease Evidence*-

Based Nutrition Practice Guideline (Grade II)(1). Dietary protein intake of 0.7 g/kg of body weight per day may cause hypoalbuminemia in some patients (Grade II)(1,7). However, a few studies have found that protein-restricted diets may improve microalbuminuria (1).

Hemodialysis: The recommended protein intake for patients who receive hemodialysis three times per week is at least 1.2 g/kg of standard body weight per day (2,5,6). A nonfasting patient loses 10 to 13 g of amino acids and small peptides during a single hemodialysis treatment (3). Approximately 30% to 40% of the amino acids lost during hemodialysis are essential. Therefore, high–biological value protein should provide at least 50% of the total protein in the diet (2,5-7). The reuse of artificial dialyzer membranes may increase amino acid losses, depending on the composition of the dialyzer.

Peritoneal dialysis: The protein recommendations for patients who receive peritoneal dialysis are 1.2 to 1.3 g/kg of standard body weight (2,4-6). Protein requirements may be even higher, depending on the patient's stress level or metabolic needs. When used for long-term management of CKD, peritoneal dialysis is associated with progressive wasting and malnutrition (3). Factors that contribute to wasting include: anorexia caused by inadequate dialysis, additional and secondary illnesses, discomfort, fullness, or severe dietary restriction; the loss of protein, amino acids, and vitamins to the dialysate; and peritonitis leading to catabolism.

Kidney transplant: For adult kidney transplant recipients who have recovered from surgery and have an adequately functioning allograft, a daily protein intake of 0.8 to 1.0 g/kg of body weight is recommended (Grade IV)(1). The registered dietitian should consider the medical status of each patient, addressing individual issues as needed (1). Adequate but not excessive protein intake supports allograft survival and minimizes the impact on comorbid conditions (Grade IV)(1).

Fat

Elevated levels of lipoproteins and abnormalities in lipid metabolism are common in patients with CKD (2). The National Kidney Foundation Task Force on Cardiovascular Disease has recommended the use of the National Cholesterol Education Program (NCEP) Adult Treatment Panel III guidelines for patients with chronic renal disease, including kidney transplant recipients (Grade II)(1,2). For patients with renal disease, the target goals for cholesterol are modified slightly based on data from morbidity and mortality studies (2,5,8). For therapeutic lifestyle diet modifications, see Section C: Medical Nutrition Therapy for Disorders of Lipid Metabolism.

Stage of Renal Failure	Recommended Lipid Levels ^a
CKD (stage 1-5)	Cholesterol <200 mg/dL Low-density lipoprotein cholesterol <100 mg/dL High-density lipoprotein cholesterol >40 mg/dL Triglycerides (fasting) <150 mg/dL
CKD with maintenance dialysis (stage 5)	Cholesterol 180-200 mg/dL Low-density lipoprotein cholesterol <100 mg/dL

Table G-2: Recommended Li	pid Levels for Adults with	Chronic Kidney Disease (8)
---------------------------	----------------------------	----------------------------

^aLevels may be measured as nonfasting levels except where indicated.

Source: National Kidney Foundation, Kidney Disease Outcomes Quality Initiative. *Guideline 4: Management of Dyslipidemias in Diabetes and Chronic Kidney Disease*. National Kidney Foundation; 2007. Available at:

www.kidney.org/professionals/kdoqi/guideline_diabetes/guide4.htm. Accessed February 20, 2009.

Sodium and Fluid

CKD (stages 1 to 5): For adults with CKD, including kidney transplant recipients, the 2010 *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* suggests a sodium intake of less than 2.4 g/day with adjustments for blood pressure, medications, kidney function, hydration status, acidosis, glycemic control, catabolism, and gastrointestinal issues (eg, vomiting, diarrhea, constipation, or gastrointestinal bleeding) (Grade II)(1). The prescribed fluid intake should be sufficient to maintain_appropriate hydration (1,4-6).

Hemodialysis: The daily sodium allowance for patients who receive hemodialysis is 2 g/day with adjustments based on urine output (2,4-6). The more urine that the patient produces, the more sodium the patient may eliminate via the urine. Under steady-state conditions, urinary output usually provides a good guide for fluid intake. The volume of urine output per day plus 1,000 mL of fluid is recommended to maintain fluid weight gain of less than 3% to 5% of the interdialytic weight between hemodialysis treatments (2,4-6). If

the patient is anuric, 1,000 to 1,500 mL/day of fluid is recommended (3,5,6).

Peritoneal dialysis: Sodium balance and blood pressure can be well controlled with CAPD or CCPD. As much as 5,700 mg/day of sodium can be removed with CAPD. Patients must understand the symptoms of hypotension as well as the methods to avoid it. The appropriate sodium requirement for each patient should be determined from an evaluation of parameters including weight (dry weight vs fluid weight), blood pressure (hypotension or hypertension), shortness of breath, and edema (5). The sodium intake for most patients should be 2 g/day (2). The suggested fluid intake for patients who receive CAPD or CCPD it is 2,000 mL/day (2). Patients should monitor their weight and blood pressure and adjust their sodium and fluid intake as necessary. Adjustments in fluid balance can be made by altering the quantity or concentration of hypertonic solutions. Patients must measure their own blood pressure and weigh themselves regularly to determine the glucose concentration of dialysate necessary to maintain fluid balance (2,5,6).

Potassium

CKD (stages 1 to 5): The 2010 *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* suggests that CKD patients (stages 3 and 4), including kidney transplant recipients) who exhibit hyperkalemia should be prescribed less than 2.4 g/day of potassium, with adjustments based on serum potassium levels, blood pressure, medications, kidney function, hydration status, acidosis, glycemic control, catabolism, and gastrointestinal issues (eg, vomiting, diarrhea, constipation, or gastrointestinal bleeding) (Grade II)(1). Dietary and other therapeutic lifestyle modifications are recommended as part of a comprehensive strategy to reduce cardiovascular disease risk in adults with CKD (1). The degree of hypokalemia or hyperkalemia can have a direct effect on cardiac function and should be carefully monitored and adjusted based on biochemical values (1,2).

Hemodialysis: For patients who receive hemodialysis, a potassium intake of 40 mg/kg of standard body weight is recommended; or, the potassium intake can be determined from laboratory values (2,5,6). An intake of 2 to 3 g/day has also been suggested (3,6). Hemodialysis removes potassium; therefore, monitoring potassium levels and ensuring adequate intake is important (3). Inadequate dialysis, gastrointestinal bleeding, hyperglycemia, infection, and catabolism can cause hyperkalemia, which can lead to life-threatening medical problems (2). Adjustments in potassium intake (either from the diet or from the dialysate bath) can be made to achieve target potassium levels (2).

Peritoneal dialysis: Patients who receive CAPD or CCPD may not need potassium restrictions; however, an assessment should be based on the patient's laboratory values (2,5,6). Peritoneal dialysis can increase the risk for hypokalemia, since most commercially available solutions do not contain potassium (3). If needed oral supplementation and/or dietary intake can be adjusted to compensate for low potassium levels. A target intake of 3 to 4 g/day of potassium is suggested (3,5,6).

Phosphorus

Alterations in calcium, phosphorus, and vitamin D metabolism result in secondary hyperparathyroidism, causing renal osteodystrophy and cardiac and extraskeletal calcification (5,6,9). Derangements in mineral and bone metabolism common to CKD are associated with increased morbidity and mortality (9). This association prompted the development of the KDOQI (Kidney Disease Outcomes Quality Initiative) Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease (9). These guidelines provide recommendations for the evaluation of phosphorus, calcium, plasma intact parathyroid hormone, and alkaline phosphorus and for the management and treatment of abnormalities with vitamin D, phosphate binders, and dialysate baths (9). Phosphorus control is the cornerstone for the treatment and prevention of secondary hyperparathyroidism, bone disease, and soft-tissue calcification in CKD (1).

CKD (stages 1 to 5): The 2010 *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* recommends that adults with CKD (stages 3 and 4) receive a low-phosphorus diet providing 800 to 1,000 mg/day or 10 to 12 mg/g of protein (Grade II) (1). Serum phosphorus levels are difficult to control with diet alone, therefore a phosphate binder may be required (1,5,6). For adults with CKD (stages 3 and 4), the dose and timing of phosphate binders should be individually adjusted according to the phosphate content of meals and snacks to achieve the desired serum phosphorus levels (Grade IV) (1).

Hemodialysis and peritoneal dialysis: Phosphorus requirements should be individualized or limited to10-12 mg/kg of standard body weight or 800-1000 mg when serum phosphorus levels approach the upper limit of normal range (1,5,6).

A phosphorus restriction is advised when the intact parathyroid hormone level is greater than 70 pg/mL in

stage 3 CKD patients; greater than 110 pg/mL in stage 4 CKD patients; or greater than 300 pg/mL in stage 5 CKD patients (3,5,9). Control of serum phosphorus levels is usually not possible by diet alone (5,9). Calciumbased, or non-calcium-based, non-aluminum-based phosphate binders are given at mealtimes to bind the phosphate from food. The prescribed amount of phosphate binders should be individualized according to the amount of phosphate present in a meal. Approximately 60% to 70% of ingested phosphorus is absorbed (10). However, 100% of additive phosphorus from food enhancers or preservatives is absorbed, and phosphorus is frequently added to many foods that were once considered to be low-phosphorus foods. One gram of calcium carbonate (CaCO₃) binds roughly 39 mg of phosphorous, and 1 g of calcium acetate binds 40 to 60 mg of phosphorous. Whereas $CaCO_3$ contains 40% elemental calcium, calcium acetate is composed of 25% elemental calcium. Calcium acetate contains 167 mg of elemental calcium in each tablet, and CaCO₃ contains 500 mg. With an increased calcium-phosphorous product, sevelamer hydrochloride, sevelamer carbonate, or lanthanum carbonate may be more effective and will not contribute to elevated phosphorus and calcium levels. One 800-mg tablet of sevelamer hydrochloride or sevelamer carbonate replaces a calcium acetate tablet (11,12). Typical starting doses of lanthanum carbonate are 500 to 1,000 mg with a designated meal. If calcium and phosphorus levels are at the high end of the normal range, a calcium binder may increase these levels to exceed the normal range and contribute to soft-tissue calcification. The goal is for the serum calcium-phosphorus product to be less than 55 mg²/dL² (5,6,10). Aluminum-containing phosphate binders are not recommended due to the risk for aluminum toxicity, which can lead to osteodystrophy, anemia, and encephalopathy (3,9).

Calcium

CKD (stages 1 to 4): The secretion of parathyroid hormone, which affects bone integrity and soft tissue calcification, is primarily regulated by serum calcium (1). The *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline* recommends that the total elemental calcium intake in adults with CKD (stages 3 and 4, including kidney transplant recipients) not exceed 2,000 mg/day (including dietary calcium, calcium supplementation, and calcium-based phosphate binders) to decrease the predisposition for mineral and bone disorders (Grade IV)(1). The use of vitamin D and other supplements that impact the calcium level should also be considered in the nutrition assessment of calcium needs (1,5,6,9,13). Refer to Section III: Nutrition Management of Acute Kidney Injury and Chronic Kidney Disease for an expanded discussion on evaluating calcium in CKD including suggested monitoring parameters and correcting total calcium for low albumin in the assessment of calcium.

Hemodialysis and peritoneal dialysis: Calcium intake should be less than 2,000 mg/day or individualized based on calcium, phosphorus, and parathyroid hormone levels and the use of vitamin D supplementation (2,5,6,9). Intestinal absorption of calcium is impaired in uremia due to the lack of the active form of vitamin D (3). Also, diets prescribed for patients with CKD tend to be low in calcium because of the restriction of dairy products (5). Calcium supplementation is not typically recommended due to an association between increased calcium load and cardiovascular calcification in CKD patients. As in CKD (stages 1 to 4), the general dietary recommendation for patients who receive RRT depends on serum calcium levels and other factors. If calcium supplements are needed, they should be taken between meals and not be confused with those supplements used to bind phosphorus. An activated form of vitamin D can also be used to enhance calcium absorption. It is important to use an adjusted calcium level in patients with low albumin levels.

Magnesium

The kidney is the organ primarily responsible for the maintenance of serum magnesium. Patients with uremia should be wary of laxatives, enemas, antacids, or phosphate binders (eg, MagneBindTM, which combines magnesium and CaCO₃) that contain magnesium (3,4). If these products are used, magnesium levels should be monitored (2). Excess magnesium mainly accumulates in bone tissue, where it is deleterious to bone metabolism. Symptoms of excess magnesium include muscle weakness, hypotension, electrocardiographic changes, sedation, and confusion. If the patient's magnesium levels are too high, the level of magnesium in the dialysate can be decreased.

Guidelines for Vitamin and Trace Mineral Supplementation in CKD

Vitamins: The diet for CKD contains less than the Dietary Reference Intakes (DRIs) for several water-soluble vitamins because of the restricted intake of foods high in protein and potassium (5). Other causes of vitamin deficiencies include impaired food intake as a result of uremia and alterations in the absorption, metabolism, or activity of some vitamins. The most common vitamin deficiencies in CKD include vitamin D, folic acid, vitamin B₆, and vitamin C (5). Studies do not support the routine supplementation of fat-soluble vitamins other than vitamin D for patients consuming well-balanced, adequate diets (5,9). Patients with CKD have a

predisposition for mineral and bone disorders, as well as other conditions that may be affected by insufficient vitamin D (1). Registered dietitians should recommend nutritional vitamin D supplementation to adults with CKD, including kidney transplant recipients, whose serum levels of 25-hydroxyvitamin D are less than 30 ng/mL (75 nmol/L) (Grade IV) (1). Supplements of 1,25-dihvdroxyvitamin D (calcitriol), the active metabolite of vitamin D, can be provided to maintain normal calcium homeostasis and prevent osteomalacia (3). Supplementation with vitamin D analogs, paricalcitol (Zemplar) and doxercalciferol (Hectorol), can be used to treat secondary hyperparathyroidism. The advantage of using the analogs as opposed to calcitriol is the decreased absorption of phosphorus and calcium in the gut (13). Supplementation with 1,25dihydroxycholecalciferol, the active form of vitamin D, in the presence of CaCO₃, must be individualized, and its effects on calcium levels must be frequently monitored (9). Deficiencies of water-soluble vitamins, especially vitamin C, folate, and vitamin B_6 , may occur secondary to poor appetite, altered metabolism, uremia, removal by dialysis, and a restricted diet (3,5,6). Each patient should be evaluated and treated with vitamins according to individual need and after the appropriate assessment of biochemical levels (1,2,4-6,9). In adults with CKD (including kidney transplant recipients), the clinician should recommend vitamin B₁₂ and folic acid supplementation if the mean corpuscular volume is greater than 100 ng/mL and the serum levels of these nutrients are less than normal values (Grade IV) (1). Vitamin C supplementation at a level greater than the DRI is not recommended to manage anemia in patients with CKD (stages 1 to 4) due to the risk of hyperoxalosis (Grade IV) (1). If vitamin C supplementation is proposed to improve iron absorption in stage 1 to 4 CKD patients (including kidney transplant recipients) who are anemic, the registered dietitian should recommend the DRI for vitamin C (Grade IV) (1). Vitamin C supplementation (60 to 100 mg/day) is recommended for adults who receive RRT (hemodialysis and peritoneal dialysis) (5,6); however, doses greater than 200 mg/day increase blood oxalate levels, which can result in the deposition of oxalate in the heart, kidney, and blood vessels (3). Thiamin supplementation of 1.5 to 2 mg/day is suggested for patients on CAPD because of dialysis loss (5,6). Supplementation with folic acid (1 mg/day), vitamin B_6 (2 mg/day), and vitamin B_{12} (3 mcg/day) is suggested for patients who receive hemodialysis or peritoneal dialysis (3.5.6).

Trace minerals: Patients with CKD experience alterations in the metabolism of trace minerals, especially zinc and iron. Serum or tissue levels of these trace minerals can be high or low. Trace minerals should be supplemented or restricted only after performing the appropriate biochemical assessments (5). Zinc supplementation (15 mg/day) is suggested for patients who receive hemodialysis or peritoneal dialysis (5). Iron status should be routinely evaluated and supplemented based on individual need (5,6). In adults with CKD (stages 1 to 4, including kidney transplant recipients), the dietitian should recommend oral or intravenous iron administration if the serum ferritin level is less than 100 ng/mL and the transferrin saturation, referred to as TSAT, is less than 20% (Grade IV)(1). Sufficient iron should be recommended to maintain adequate levels of serum iron to support erythropoiesis (Grade IV)(1).

See Section III: Clinical Nutrition Management, Management of Acute Kidney Injury and Chronic Kidney Disease.

Diabetes Management in Patients with CKD

Medical nutrition therapy for people with diabetes mellitus and kidney disease is complex and requires an individualized approach (1,2,5,6,8). For adults with diabetes and CKD (including kidney transplant recipients), the clinician should implement medical nutrition therapy to manage hyperglycemia with a target hemoglobin A1C level of approximately 7% (Grade I) (1). Intensive treatment of hyperglycemia, while avoiding hypoglycemia, prevents the development of diabetic kidney disease and may slow the progression of established kidney disease (Grade I)(1). In addition to the nutrient modifications required for managing renal disease, consistent carbohydrate intake is a primary goal for persons with diabetes mellitus complicated by CKD. The treatment approach should follow the guidelines outlined in Section IC: Medical Nutrition Therapy for Diabetes Mellitus. The 2002 versions (second editions) of *A Healthy Food Guide for People With Chronic Kidney Disease* and *A Healthy Food Guide for People on Dialysis* focus on complementing the patient's existing diabetes meal planning approaches (eg, constant carbohydrate meal plan, carbohydrate counting meal plan, or exchange meal plan) (2,6). These publications also recommend strategies that best meet the nutritional needs of the patient and that promote or maintain glucose tolerance.

Table G-3: Daily Nutritional Requirements for Adults with Renal Disease Based onType of Therapy

Diagnosis/	Energy	Protein	Fluid	Sodium	Potassium	Phosphorus
Therapy						
Acute kidney injury (5)	25-35 kcal/kg ^a , or determine via indirect calorimetry Consider stress level. Include energy from continuous RRT, if applicable.	0.8-1.2 g/kg ^a with no dialysis 1.2-1.5 g/kg ^a with dialysis or if catabolic	Anuric/ oliguric phase ^c : 500 mL + total output (urine, vomitus, and diarrhea) Diuretic phase: Large volume of fluids may be needed. Assess frequently.	Anuric/ oliguric phase: 2-3 g; based on blood pressure and edema Diuretic phase: Replace based on urine output, edema, need for dialysis, and serum sodium levels.	Anuric/oliguric phase: 2-3 g Diuretic phase: Replace losses depending on urine volume, serum potassium levels, and need for dialysis and medication.	8 to 15 mg/kg ^a Individualize based on serum values.
Stage 1-4 CKD (1)	23-35 kcal/kg ^a (Grade II)(1) 30-35 kcal/kg ^a if >60 years old (6,14)	0.6-0.8 g/kg ^a without diabetes (Grade II)(1) 0.8-0.9 g/kg ^a with diabetic nephropathy (Grade II)(1)	Individualize to maintain appropriate hydration status.	Individualize or <2.4 g (Grade II) (1)	Individualize based on laboratory values. If hyperkalemia, <2.4 g (for stages 3 and 4) (Grade II)1)	Based on serum value For stages 3 and 4, 800- 1,000 mg or 10-12 mg/g of protein (Grade II)(1) Dose and timing of phosphate binders individualized (Grade IV)(2)
Stage 5 CKD/ Hemodialysis (5,6,9,14)	35 kcal/kg ^a <60 years old 30-35 kcal/kg ^a if >60 years old	>1.2 g/kg ^a with >50% HBV ^b	Urine output + 1,000 mL 1,000-1,500 mL if anuric	Individualize or 2 g	2-3 g Individualize based on laboratory values.	Individualize, or <10-12 mg/kg ^a or 800- 1,000 mg when serum phosphorus levels approach the upper limit of normal range (2) Usually requires phosphate binder (9)
Stage 5 CKD/ Peritoneal dialysis (5,6,9,14)	35 kcal/kg ^a 30-35 kcal/kg ^a if >60 years old Subtract energy absorbed from dialysate from daily energy prescription.	1.2-1.3 g/kg ^a with >50% HBV ^b	Individualize to maintain fluid balance and blood pressure.	Individualize or 2 g	3-4 g Adjust to serum levels.	Individualize, or <10-12 mg/kg ^a or 800- 1,000 mg when serum phosphorus levels approach the upper limit of the normal range (2) Usually requires phosphate binder (9)

^aTo calculate these requirements, use standard body weight derived from the NHANES II weight table based on sex, age, and frame size. (See Section II: Standard Body Weight (SBW) Determination Based on NHANES II.) In some cases, actual body weight or adjusted dry weight may be more appropriate than standard body weight (152). In all cases, individual practitioners should use their own clinical judgment and expertise in selecting a method (1,2,5,9).

^bHBV, high-biological value protein

^cAnuric/oliguric phase refers to less than 500 mL of urine output per 24 hours (1,14).

Medical Nutrition Therapy for Chronic Kidney Disease Estimates of Calories Absorbed in Peritoneal Dialysis

Energy requirements and nutrient intake calculations for patients who receive peritoneal dialysis should include carbohydrate absorption from the dialysate. The most accurate method of determining the energy load is to measure the grams of glucose in the effluent and compare that with the grams of glucose infused (2).

Simple estimate of glucose absorption in peritoneal dialysis: The following quick estimate does not consider the peritoneal equilibration test (2)

For CCPD: [(dextrose % x L) x 3.4] x 40% = energy (kcal)(2)

For CAPD: [(dextrose % x L) x 3.4] x 60% = energy (kcal)(2)

Glucose absorbed (kcal)₍₂₎ = Glucose infused (mL) x 40% APD* or Glucose infused (mL) x 60% absorption (CAPD). For a 1-L volume of dialysate in CAPD:

1.5% = 15 g x 3.4 kcal/g = 51 kcal x 60% = 31 kcal/L2.5% = 25 g x 3.4 kcal/g = 85 kcal x 60% = 51 kcal/L4.25% = 42.5 g x 3.4 kcal/g = 144.5 kcal x 60% = 86.7 kcal/L*APD-automated peritoneal dialysis

Example (2): A patient on CAPD uses 4 L of 1.5% solution and 4 L of 4.25% solution.

4L, 1.5% = 124 kcal 4L, 4.25% = 346 kcal Total = 470 kcal

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2010. Available at: www.andevidencelibrary.com. Accessed February 4, 2013.
- 2. McCann L, ed. *Pocket Guide to Nutrition Assessment of the Patient with Chronic Kidney Disease*. 4th ed. New York, NY: National Kidney Foundation; 2009. Available at www.kidney.org/professionals/crn/pocketGuide/index.cfm.
- 3. Wolk R, Moore E, Foulks C. Renal disease. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum: A Case-Based Approach—The Adult Patient.* Silver Spring, Md: American Society For Parenteral and Enteral Nutrition; 2012:492-510.
- 4. Kopple JD, Massry SG, eds. *Nutrition Management of Renal Disease*. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2004.
- 5. Chronic kidney disease. In: *The Nutrition Care Manual*. Updated annually. The Academy of Nutrition and Dietetics. Available at: www.nutritioncaremanual.org. February 4, 2013.
- 6. Renal Practice Group of the American Dietetic Association. *National Renal Diet: Professional Guide*. 2nd ed. Chicago, Ill: American Dietetic Association; 2002.
- 7. Caggiula AW, Levey AS. MDRD Study data suggest benefits of low-protein diets. J Am Diet Assoc. 1995;95:1289.
- 8. National Kidney Foundation. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Diabetes and Chronic Kidney Disease. *Am J Kidney Dis.* 2007;49(suppl 2): S12-S154.
- National Kidney Foundation. KDOQI Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease. Am J Kidney Dis. 2003;42(suppl 3): S1-S201.
- 10. Hsu CH. Are we mismanaging calcium and phosphate metabolism in renal failure? Am J Kidney Dis. 1997;29:641-649.
- 11. Malluche HH, Monier-Faugere MC. Hyperphosphatemia: pharmacologic intervention yesterday, today and tomorrow. *Clin Nephrol.* 2000;54:309-317.
- 12. Renagel [package insert]. Cambridge, Mass: Genzyme Corp; 2001.
- 13. Slatopolsky E., Martin KJ, Sherrard DJ. Should vitamin D analogs be the therapy of preference for ESRD patients with secondary hyperparathyroidism? *Dial Transplantation*. 2001;30:190-195.
- 14. Wiggins KL, ed. Guidelines for Nutrition Care of Renal Patients. 3rd ed. Chicago, Ill: American Dietetic Association; 2002.

Bibliography

Centers for Medicare & Medicaid Services. *End Stage Renal Disease (ESRD) Program Interpretive Guidance Version 1.1.* October 2008 update. Baltimore Md: Dept of Health and Human Services; 2008. Ref: S&C-09-01.

National Kidney Foundation. KDOQI Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification, and Stratification. *Am J Kidney Dis*. 2002;39(suppl 1):S1-S266.

DIETARY MANAGEMENT USING THE HEALTHY FOOD GUIDE FOR PEOPLE WITH CHRONIC KIDNEY DISEASE

Before determining a patient's diet prescription and calculating his or her meal plan, the dietitian should perform a complete nutrition assessment, with special attention to the following factors:

- 1. Medical history.
- 2. Physician's orders.
- 3. Treatment modality (pre-end-stage renal disease [pre-ESRD], hemodialysis, or peritoneal dialysis). Nutrition management of the renal patient depends on the method of treatment as well as on medical and nutritional status. A comparison of treatment methods and primary concerns in each is summarized in the table below.
- 4. Presence of other chronic diseases that may affect the nutritional status. As a result, the diet prescription also will be affected.

Stage of CKD	Treatment	Renal Replacement Therapy (RTT)	Duration	Metabolic Concerns
CKD Stage 1- 5 (without dialysis)	Diet and medications	None	Indefinite	Glomerular hyperfiltration; rise in BUN; bone disease
				Hypertension; glycemic control in diabetes
Hemodialysis	Diet and medications; hemodialysis	Dialysis using vascular access for waste product and fluid removal	3-5 h 2-3 d/wk	Amino acid loss; interdialytic electrolyte and fluid changes
				Bone disease; hypertension
CAPD or CCPD	Diet and medications; peritoneal dialysis	Dialysis using peritoneal membrane for waste product and fluid removal	3-5 exchanges 7 d/wk	Protein loss into dialysate; glucose absorption from dialysate Bone disease; weight
aCAPD indicator or	ntinuous ambulatory	paritoneal dialysis: CCPD, continuous cyclic p	oritonoal dialucis:	gain; hyperlipidemia; glycemic control in diabetes

Comparison of Treatment Approaches for Patients With CKD

^aCAPD indicates continuous ambulatory peritoneal dialysis; CCPD, continuous cyclic peritoneal dialysis; and BUN, blood (serum) urea nitrogen.

The second edition of the National Renal Diet (1) and educational guides, *Healthy Food Guide For People With Chronic Kidney Disease* (2), and *Healthy Food Guide for People on Dialysis* (3) is recommended by the Renal Practice Group of the Academy of Nutrition and Dietetics as the meal planning approach for persons with CKD (1-3). This edition uses an approach that is flexible and encourages self-management training and individualization for both the patient and registered dietitian (1). Foods are divided into groups or "choices" according to nutrient content and are categorized based on the amount of protein, energy, sodium, potassium, and phosphorus content.

The following information and tables are reprinted with permission from the Academy of Nutrition and Dietetics, *National Renal Diet: Professional Guide* (1), *Healthy Food Guide for People With Chronic Kidney Disease* (2), and *Healthy Food Guide for People on Dialysis* (3), 2002.

Overview of the National Renal Diet

The *National Renal Diet*, second edition, version 2002, simplifies the approach to medical nutrition therapy management for persons with CKD. The newer versions focus on two primary diet approaches, one for use with CKD patients stages 1-5 without dialysis (*Healthy Food Guide for People with Chronic Kidney Disease [Pre-ESRD]* (2) and one for use with patients on dialysis (*Healthy Food Guide for People on Dialysis*) (3). The intent of

the revised version is to simplify the diet approach and allow for more flexibility and self-management training opportunities with the patient. The *National Renal Diet Professional Guide*, second edition, can be used to provide detail review of these two diet approaches (1).

The guides for CKD (stages 1-5 without dialysis) and dialysis are very similar but differ somewhat in how foods are grouped and categorized. Differences in how foods are grouped are based on the unique needs of persons with stage 1-5 CKD compared with those with CKD stage 5 on dialysis. A summary can be reviewed in Tables G3.1 to G3.5: Healthy Food Guide for People With Chronic Kidney Disease (stage 1-5) (1,2) and Tables G4.1 to G4.6: Healthy Food Guide for People on Dialysis (1,3). In both guides, food lists that are provided are limited to the most common foods. The dietitian will need to work with the patient to address serving limits, serving sizes, and additional food choices that may not be included on the lists provided. Food choices in both guides are grouped according to the amount of protein, calories, sodium, potassium, and phosphorus. Nutrient composition of foods can vary greatly, depending on the size, variety, growing conditions, processing, packaging, and final preparation (1). Nutritionists IV and V (First Data Bank) were used to update food lists for the revised National Renal Diet guides (1).

Tables G3.1-3.5: Healthy Food Guide for People with Chronic Kidney Disease (Pre-ESRD)

Table G3.1: High-Protein Foods

High-Protein Food Choices: The high protein food list includes sources of protein from both animals and vegetables that provide a high-biological source of protein (providing 6 to 8 g protein per serving). Foods that provide a high source of phosphorus and sodium are identified (see footnotes).

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
High protein	6-8	50-100	20-150	50-150	50-100
Higher phosphorus proteins	6-8	50-100	20-150	50-350	100-300 (if marked a *)
Higher sodium proteins	6-8	50-100	200-450 (if marked with b **)	50-150	50-100

*a—food contains 100-300 mg phosphorus per serving.

**b—food contains 200-450 mg sodium per serving.

Table G3.2: Low-Protein Foods

Lower-Protein Food Choices: The low-protein food choices include vegetables as well as breads, cereals, and other grain foods, and desserts that provide 2 to 3 g protein per serving. The foods contained in this group help to complete the protein, nutrient, and calorie needs of the patient. Most CKD patients do not need to monitor potassium intake, but if necessary, vegetables are grouped by potassium content.

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Vegetables	2-3	10-100	0-50	1) 20-150	10-70
(separated by				2) 150-250	
potassium				3) 250-550	
content)					
Breads, rolls,	2-3	50-200	0-150	10-100	10-70
cereals, grains,					
crackers,					
snacks,					
desserts					
Higher sodium	2-3	50-200	150-400 (if	10-100	100-200 (if marked
and/or			marked with		with a*)
phosphorus			b **)		
grain foods					

*a—food contains 100-200 mg phosphorus per serving.

**b—food contains 150-400 mg sodium per serving.

Table G3.3: Fruit Choices

Fruit Choices: Fruits add very little protein to the diet (0 to 1 g per serving) but provide necessary vitamins, calories, fiber, and flavor. The fruit lists are grouped according to potassium content for those needing to monitor potassium intake.

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Fruits	0-1	20-100	0-10	1) 20-150	1-20
(grouped by				2) 150-250	
potassium				3) 250-550	
content)				-	

Table G3.4: Calorie and Flavoring Choices

Calorie and Flavoring Choices: Foods grouped in this category help to add extra calories and flavor to foods to help enhance caloric intake and can be added to the diet to prevent weight loss.

Food List	Protein (g/serving)	Calories (kcal/serving)	Sodium (mg/serving)	Potassium	Phosphorus (mg/serving)
Calorie choices	0-1	100-150	0-100	0-100	0-100
Flavor choices	0	0-20	250-300	0-100	0-20

Table G3.5: Vegetarian Protein Choices

Vegetarian Choices: The section on vegetarian choices is intended for patients who avoid animal foods. It can replace the protein choices section (Tables 3.1 to 3.2). Table 3.5 provides nutrient values of vegetarian proteins and foods categorized in this group. Choosing vegetarian proteins over animal proteins may result in a higher phosphorus load. If this is a concern, phosphorus binders may be needed, or the patient may need to limit other high-phosphorus foods (See the dairy and phosphorus choices, Table 4.3, in A *Healthy Food Guide for People on Dialysis*.)

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Protein foods	6-8	70-150	10-200	60-150	80-150
Higher sodium, potassium, or phosphorus proteins	6-8	70-150	250-400 (marked with b**)	250-500 (marked with c***)	200-400 (marked with a *)

*a—food contains 200-400 mg phosphorus per serving.

**b—food contains 250-400 mg sodium per serving.

***c—food contains 250-500 mg potassium per serving.

Calculating Food Choices for People with CKD (Stages 1-5) (1,2)

- 1. Refer to Section IG, Table G2: Nutritional Requirements for Adults with Renal Disease Based on Type of Therapy to determine nutrition needs.
- 2. Once nutrition needs are known, calculate and identify protein needs and determine choices from Tables G3.1 to G3.2. At least 50% of the protein should come from the High-Protein Food List (Table G3.1) to ensure high-biological proteins are consumed. Choices from the higher phosphorus and sodium groups can be included as needed by the dietitian's discretion to meet the patient's nutrition needs.
- 3. Lower-protein foods can then be selected to fulfill protein and nutrient requirements (refer to Table G3.2). Most CKD patients do not need to monitor potassium intake, but if necessary, vegetables are grouped by potassium content.
- 4. After protein needs have been met, fruit choices (also grouped by potassium content) and calorie and flavoring choices can be used to provide balance, flavor, and additional calories to meet nutrition needs and complete the patient's meal plan (refer to Tables G3.3 to G3.4).

HIGH-PROTEIN FOOD LIST Serving 1 oz

Serving 1 02					
Protein	Calories	Sodium	Potassium	Phosphorus (mg/serving)	
(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)		
6-8	50-100	20-150	50-150	50-100	
Beef (1 oz)					
Egg substitutes	(¼ cup)				
Eggs (1 large)					
Fish (1 oz)					
Lamb (1 oz)					
Pork (1 oz)					
Poultry (1 oz)					
Shellfish (1 oz)					
Veal (1 oz)					
Wild game (1 oz	:)				

High-Protein and High-Phosphorus Food Lists

Protein	Calories	Sodium	Potassium	Phosphorus (mg/serving)	
(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)		
6-8	50-100	20-150	50-350	100-300 (if marked a *)	
Cheese (1 oz) ^a					
Cooked dried be	ans and peas (½ cu	p) ^a			
Evaporated mill	ĸ (½ cup) ª				
Milk, all kinds (1	l cup) ^a				
Nut butters (2 tl	bsp) ^a				
Nuts (¼ cup) ^a					
Organ meats (1	oz) ^a				
Soy milk (1 cup) ^a					
Sweetened condensed milk (½ cup) ^a					
Tofu (¼ cup) ^a					
Yogurt (1 cup) ^a					

*a—food contains 100-300 mg phosphorus per serving.

High-Protein and High-Sodium (Salt) Food Lists

ingii i i otem a	ingi i i otem and ingi sourani (sare) i ood insts						
Protein	Calories	Sodium	Potassium	Phosphorus (mg/serving)			
(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)				
6-8	50-100	200-450 (if marked	50-150	50-100			
		with b**)					
Bacon (4 slices)	b						
Breakfast sausa	ge (3 links or 1½ pa	tties) ^b					
Canned tuna, sa	lmon (1 oz or ¼ cup) ^b					
Cottage cheese (¹ / ₄ cup) ^b						
Deli-style roast beef, ham, turkey (1 oz) ^b							
Frankfurters, bratwurst, Polish sausage (2 oz) ^b							
Luncheon meats, bologna, liverwurst, salami, etc (2 oz) ^b							
	C I						

**b—food contains 200-450 mg sodium per serving.

LOWER-PROTEIN FOOD LISTS: VEGETABLES Serving: ¹/₂ cup unless otherwise noted

Protein	Calories	Sodium	Potassium	Phosphorus (mg/serving)
(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	
2-3	10-100	0-50	1) 20-150	10-70
			2) 150-250	
			3) 250-550	

Dower I roteni i obu hists. Vegeta		
Group 1: 20-150 mg	Group 2: 150-250 mg	Group 3: 250-550 mg
Alfalfa sprouts	Asparagus	Artichokes
Bamboo shoots (canned)	Broccoli	Avocado
Bean sprouts	Celery	Bamboo shoots (fresh, raw)
Beets	Kale	Beets (fresh)
Cabbage	Mixed vegetables	Brussels sprouts
Carrots	Peas	Chard
Cauliflower	Peppers	Greens (beet, collard, mustard, etc)
Corn	Summer squash, boiled	Kohlrabi
Cucumber	Turnips	Okra
Endive	Zucchini	Parsnips
Eggplant		Potatoes
Green beans		Pumpkin
Lettuce		Rutabagas
Mushrooms		Spinach
Onions		Sweet potatoes
Radishes		Tomatoes
Summer squash, raw		Tomato sauce, puree
Water chestnuts (canned)		V-8 juice
Watercress		Wax beans
		Winter squash
		Yams

Lower-Protein Food Lists: Vegetables (Cont.)

LOWER-PROTEIN FOOD LISTS: BREADS AND ROLLS, CEREALS AND GRAINS, CRACKERS AND SNACKS, AND DESSERTS

Protein	Calories	Sodiu	m (mg/serving)	Potassium	Phosphorus
(g/serving)	(kcal/serving)			(mg/serving)	(mg/serving)
2-3	50-200	0-150		10-100	10-70
2-3	50-200	150-4	00 (if marked with b**)	10-100	100-200 (if marked with a *)
Breads and Rolls:			Cereals:		
Bagel (½ small)			Low-salt cereals (Corn Pops, Coca Puffs, Sugar Smacks, Fruity		
Bread, all kinds (1 slice or 1 oz)			Pebbles, Puffed Wheat, Puffed Rice) (1 cup or 1 oz)		
Bun, hamburger or hot dog type (½)			Cereals, cooked (Cream of Rice or Wheat, Farina, Malt-o-Meal) (½ cup)		
Cornbread, homemade (1 piece or 2 oz)			Grits, cooked (½ cup)		
Danish pastry or sweet roll (½ small)			Pasta, cooked (noodles), macaroni, spaghetti) (½ cup)		
Dinner roll or hard roll (1 small)			Rice, cooked (½ cup)		
Doughnut (1 small)			Crackers, unsalted (4 2-inch crackers)		
English muffin (½)			Graham crackers (3 squares)		
Pita or pocket bread (½ 6-inch			Melba toast (3 oblong)		
diameter)			Popcorn, unsalted (1½ cups, popped)		
Tortilla, flour (1- to 6-inch diameter)			Pretzel, unsalted sticks or rings (3/4 oz, 10 sticks)		
			Tortilla chips, unsalted (3/4 oz, 9 chips)		
Desserts:			Added salt and phosphorus:		
Sugar cookie (4 cookies)			Biscuits, muffins (1 small) ^{a,b}		
Shortbread cookie (4 cookies)			Cake $(1/20 \text{ round cake or } 2 \times 2 \text{-inch square})^{a,b}$		
Sugar wafer (4 cookies)			Cornbread, from mix (1 piece or 2 oz) ^{a,b}		
Vanilla wafer (10 cookies)			Fruit pie (1/8 pie) ^b		
		Granola, oatmeal (½ cup) ª			
		Pancakes, waffles (1-4 inches) ^{a,b}			
		Pretzels, salted sticks or rings (3/4 oz or 10 sticks) ^b			
		Dry cereals, most brands (3/4 cup) ^b			
		RyKrisp (3 crackers) b			
		Sandwich cookie (4 cookies) b			
			Whole-wheat cereals, bran cereals (1/2 cup) a,b		

*a—food contains 100-200 mg phosphorus per serving.

**b—food contains 150-400 mg sodium per serving.
FRUIT LISTS

Serving: ¹/₂ cup unless otherwise noted

	Protein	Calories	Sodium	Potassium	Phosphorus	
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)	
Fruits	0-1	20-100	0-10	1) 20-150	1-20	
(grouped by				2) 150-250		
potassium				3)250-550		
content)						
Group 1: 20-15	50 mg	Group 2: 150-25	50 mg	Group 3: 250-5	50 mg	
Apple, raw (with	10ut skin)	Apple, raw (with s	skin)	Gooseberries (ra	w)	
Apple juice (½ c	up)	Grape juice (cann	ed/bottled)	Peach, dried (5)		
Applesauce		Peach , raw (with	skin)	Pear, dried (5)		
Apricot nectar		Pear, raw (with sl	kin)	Figs, dried (5)		
Blackberries		Cherries, raw (10))	Apricots, dried (10)	
Blueberries		Cantaloupe		Apricots, raw (3)		
Cranberries		Figs (2 whole)		Banana (1 small)		
Cranberry juice	cocktail	Grapefruit, raw		Dates (¼ cup)		
Fruit cocktail		Grapefruit juice		Honeydew melo	n	
Gooseberries, ca	nned	Mango		Kiwifruit		
Grape juice (froz	zen	Рарауа		Nectarine		
concentrate)						
Grapes		Rhubarb		Orange juice		
Lemon, lime (1 i	raw)			Orange		
Lemon, lime juic	ce			Prune juice		
Papaya nectar				Prunes (5)		
Peach nectar				Raisins		
Peach, canned						
Pear, canned						
Pear nectar						
Pineapple, fresh	or canned					
Plum, raw or car	nned					
Raspberries, rav	v or frozen					
Strawberries, ra	w or frozen					
Tangerine, raw						
Watermelon, ray	W					

CALORIE AND FLAVORING CHOICES FOOD LISTS

	Protein	Calories	Sodium	Potassium	Phosphorus	
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)	
Calorie choices	0-1	100-150	0-100	0-100	0-100	
Flavor choices	0	0-20	250-300	0-100	0-20	
Chewy fruit snacks	and candies (1	oz)	Mints, peppermi	nt patties (13 min	ts or ½ large)	
Cranberry sauce or	relish (¼ cup)		Nondairy creame	ers, half-and-half (¼ cup)	
Cream cheese (2 tb	sp)		Nondairy creame	ers, half-and-half		
Fruit chews (4 or 1	oz)		Nondairy whippe	ed topping (½ cup)	
Fruit drinks (1 cup)		Popsicles, juice bars (1 bar)			
Fruit roll up (2)			Salad dressing (1½ tbsp)			
Gumdrops (8)			Soda pop (1 cup)			
Hard candy (4 piec	es)		Sorbet (½ cup)			
Honey (2 tbsp)			Sour cream (¼ c	up)		
Jam or jelly (2 tbsp)		Sugar, brown or	white (2 tbsp)		
Jelly beans (15)			Sugar, powdered	Sugar, powdered (3 tbsp)		
Lifesavers (13)			Syrup (2 tbsp)			
Margarine or butter (1 tbsp)			Tartar sauce (2 tbsp)			
Marmalade (2 tbsp) Vegetable oil (1 tbsp)						
Marshmallows (5 large) Whipped cream (¼ cup)						
Mayonnaise (1½ th	osp)					

VEGETARIAN FI	NOTEIN FOOD I	1313 (ALSO KEFEI	N TO LOWER-T NO	TEIN FOOD LIST	3]		
	Protein	Calories	Sodium	Potassium	Phosphorus		
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)		
Protein foods	6-8	70-150	10-200	60-150	80-150		
Higher sodium,	6-8	70-150	250-400	250-500	200-400 (marked with		
potassium, or			(marked with	(marked with	a*)		
phosphorus			b **)	C ***)			
proteins							
Cheese, all kinds	(1 oz)		Okra (1 cup) ^c				
Eggs (1 large)			Soy cheese (1 oz)) a,b			
Nut butters (1½	tbsp)		Soy milk (1 cup)	с			
Tempeh (½ cup)			Soy nuts (2 tbsp)	b			
Tofu, firm type (1	¼ cup)		Soy protein isolate (½ oz) ^c				
Tofu, soft type (1/2	∕₂ cup)		Soy sprouts (1 cup) ^c				
			Soy yogurt (1 cup) ^c				
Higher phosphore	us (a), sodium (b), or potassium	Tofu hotdog (1 oz) ^b				
(c):			Vegetarian meat analogs (Gardenburgers, Bocaburgers) (2				
Cottage cheese (1	¼ cup) ^b		oz) ^b				
Dried beans, pea	s (½ cup)º		Yogurt (1 cup) ^{a,c}				
Milk (1 cup) ^{a,c}							
Miso (¼ cup) ^b							
Natto (¼ cup) ^c							
Nuts (¼ cup) ^{b,c}							

VEGETARIAN PROTEIN FOOD LISTS (ALSO REFER TO LOWER-PROTEIN FOOD LISTS)

*a—food contains 200-400 mg phosphorus per serving.

**b—food contains 250-400 mg sodium per serving.

***c—food contains 250-500 mg potassium per serving.

Tables G4.1-G4.6: Healthy Food Guide for People on Dialysis

Table G4.1: Protein Choices

Protein Choices: The foods included in this list include sources of protein from both animals and vegetables that provide a high-biological source of protein (generally 6 to 8 g protein per serving). Foods that provide a high source of sodium, potassium, and/or phosphorus are identified.

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Animal protein	6-8	50-100	20-150	50-150	50-100
Higher sodium, potassium, or	6-8	50-100	200-500 (if marked b**)	250-450 (if marked c***)	100-300 (if marked a*)
phosphorus proteins					

a*—food contains 100-300 mg phosphorus per serving.

**b—food contains 200-500 mg sodium per serving.

***c—food contains 250-450 mg potassium per serving.

Table G4.2: Fruit and Vegetable Choices (grouped by potassium content)

Fruit and Vegetable Choices: Fruits and vegetables are grouped by potassium content. Most patients can choose one high-potassium food, two medium-potassium foods, and three low-potassium foods per day. Choices will vary depending on the serum potassium level and dialysis therapy.

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Low potassium	0-3	10-100	1-50	20-150	0-70
Medium potassium	0-3	10-100	1-50	150-250	0-70
High potassium	0-3	10-100	1-50	250-550	0-70

Table G4.3: Dairy and Phosphorus Choices

Dairy and Phosphorus Choices: The foods in this group contain 100 to 120 mg phosphorus per serving, and 2 to 8 g protein per serving. Most patients can choose one or two high-phosphorus foods a day, depending on lab values, use of phosphate binders, and type/frequency of dialysis therapy.

Food List	Protein	Calories	Sodium	Potassium	Phosphorus
	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Dairy and phosphorus	2-8	100-400	30-300	50-400	100-120

Table G4.4: Bread, Cereal, and Grain Choices

Bread, Cereal, and Grain Choices: This group of foods generally provides 2 to 3 g protein per serving. Grain foods with higher values of sodium, potassium, and/or phosphorus are identified. These foods can be integrated in the meal plan to meet nutrition needs. Laboratory values, use of phosphate binders, and type/frequency of dialysis therapy should be considered to determine servings recommended per day.

Food List	Protein (g/serving)	Calories (kcal/serving)	Sodium (mg/serving)	Potassium (mg/serving)	Phosphorus (mg/serving)
Breads, rolls, cereals, grains, crackers, snacks, desserts	2-3	50-200	0-150	10-100	10-70
Higher sodium and/or phosphorus grain foods	2-3	50-200	150-400 (if marked with b**)	10-100 (if marked with c***)	100-200 (if marked with a *)

a*—food contains 100-200 mg phosphorus per serving.

**b—food contains 150-400 mg sodium per serving.

***c—food contains 10-100 mg potassium per serving.

Table G4.5: Calorie and Flavoring Choices

Calorie and Flavoring Choices: Foods grouped in this category help to add extra calories and flavor to foods to help enhance caloric intake and can be used to help prevent weight loss.

Food List	Protein (g/serving)	Calories (kcal/serving)	Sodium (mg/serving)	Potassium (mg/serving)	Phosphorus (mg/serving)
Calorie choices	0-1	100-150	0-100	0-100	0-100
Flavor choices	0	0-20	250-300	0-100	0-20

Table G4.6: Vegetarian Protein Choices

Vegetarian Choices: The section on vegetarian choices is intended for patients who avoid animal foods. It can replace the protein choices section (Table G4.1). Table G4.6 provides nutrient values of vegetarian proteins and foods categorized in this group. Choosing vegetarian proteins over animal proteins may result in higher intakes of potassium and phosphorus per gram of protein. Higher sodium, potassium, and phosphorus foods are identified and often can be used once or twice per week depending on lab values, fluid weight gains, and dialysis therapy.

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Protein foods	6-8	70-150	10-200	60-150	80-150
Higher sodium,	6-8	70-150	250-400	250-500	200-400
potassium, or				(marked with b)	(marked with a)
phosphorus					
proteins					

Calculating Food Choices for People on Dialysis (1,3)

1. Refer to Table G2: Nutritional Requirements for Adults With Renal Disease Based on Type of Therapy to determine nutrition needs. Priorities in meal planning for people on dialysis will vary with their nutrition status, laboratory values and type/frequency of dialysis therapy.Once nutrition needs are

known, calculate and identify protein needs and determine choices. At least 50% of the protein should come from the Protein Choices Food List (Table G4.1) to ensure high-biological proteins are consumed. Choices from the higher sodium, potassium, and phosphorus groups can be included as needed by the dietitian's discretion to meet the patient's nutrition needs.

- 2. Other protein foods can then be selected to fulfill protein and nutrient requirements (refer to Tables G4.2 to G4.4.). The amount of sodium, potassium, and phosphorus should be determined based on the patient's laboratory values, medications (eg, phosphorus binders) and type/frequency of dialysis.
- 3. Calorie and flavoring choices can be used to provide balance, flavor, and additional calories to meet nutrition needs and complete the patient's meal plan (refer to Table G4.5).

Animai Protein: Serving 1 oz							
Protein	Calories	Sodium		Potassium	Phosphorus		
(g/serving)	(kcal/serving)	(mg/servii	1g)	(mg/serving)	(mg/serving)		
6-8	50-100	20-150		50-150	50-100		
Beef (1 oz)			Poult	try (1 oz)			
Egg substitutes	(¼ cup)		Shellfish (1 oz)				
Eggs (1 large)			Veal (1 oz)				
Fish (1 oz)			Wild game (1 oz)				
Lamb (1 oz)							
Pork (1 oz)							

PROTEIN CHOICES FOOD LIST

High Sodium, Potassium, Phosphorus, Protein Food Lists

Protein	Calories	Sodium	Potassium	Phosphorus			
(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)			
6-8	50-100	200-500 (if	250-450 (if marked	100-300 (if marked a *)			
		marked b**)	c ***)				
Bacon, breakfas	t sausage ^b (4 slices, 1 ¹ ⁄	2 patties, or 3 links)				
Canned tuna, ca	nned salmon, or sardin	es ^{a,b} (¼ cup)					
Cheeses, all kind	ls ^{a,b} (1 oz)						
Cooked, dried b	eans and peas ^{a,c} (½ cup)					
Cottage cheese ^b	(¼ cup)						
Deli-style roast	beef, ham, turkey (1 oz)	b					
Frankfurters, br	atwurst, polish sausage	e (2 oz) ^b					
Luncheon meats	s, bologna, liverwurst, s	alami, etc (2 oz) ^b					
Milk (1 cup) ^{a,b}							
Nut butters (2 th	osp) ^a						
Nuts (¼ cup) ^{a,c}							
Organ meats (1	0 Z) ^a						
Soy milk (1 cup)	с						
Tofu (¼ cup) ^a							
Vegetarian meat analogs (garden burgers, soy burgers, etc) (2 oz) ^b							
Yogurt (1 cup) ^{a,}	b,c						

*a- food contains 100-300 mg phosphorus/serving **b- food contains 200-500 mg sodium/serving ***c- food contains 250-450 mg potassium/serving

FRUIT AND VEGETABLE CHOICES FOOD LISTS (grouped by potassium content)

Food List*	Protein (g/serving)	Calories (kcal/serving)	Sodium (mg/serving)	Potassium (mg/serving)	Phosphorus (mg/serving)
Low potassium	0-3	10-100	1-50	20-150	0-70
Medium potassium	0-3	10-100	1-50	150-250	0-70
High potassium	0-3	10-100	1-50	250-550	0-70

*Refer to Vegetable Lists and Fruit Lists under Healthy Food Guide for People With Chronic Kidney Disease (Pre-ESRD), this section.

DAIRY AND PHOSPHORUS CHOICES FOOD LISTS

Protein	Calories	Sodium	Potassium	Phosphorus	
(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)	
2-8	100-400	30-300	50-400	100-120	
Biscuits, muffins (1 sn	nall)	Organ meats (1 oz)			
Cake (1 slice, 2×2 inc	ches)	Pancakes, waffles (1-4	4 inches)		
Cheese (1 oz)		Pudding, custard (½ c	cup)		
Cooked dried beans an	nd peas (½ cup)	Sardines (1 oz)			
Condensed and evaporated milk (¼ cup)		Soy milk (1 cup)			
Cottage cheese (¼ cup)		Tofu (¼ cup)			
Granola, oatmeal (½ cup)		Tortillas, corn (2- to 6	-inch diameter)		
Ice milk or ice cream (½ cup)		Vegetarian meat anal	ogs (Garden burgers,	Bocaburgers, etc)	
Light cream or half-and-half (½ cup)		(2 oz)			
Milk, all kinds (½ cup)		Whole-wheat cereals, bran cereals (½ cup)			
Milkshake (½ cup)		Yogurt, plain or fruit flavored (½ cup)			
Nut butters (2 tbsp)		Nondairy milk substitutes (1 cup)			
Nuts (¼ cup)					

BREAD, CEREAL, AND GRAIN CHOICES FOOD LISTS

Food List***	Protein (g/serving)	Calories (kcal/serving)	Sodium (mg/serving)	Potassium (mg/serving)	Phosphorus (mg/serving)
Breads, rolls, cereals, grains, crackers, snacks, desserts	2-3	50-200	0-150	10-100	10-70
Higher sodium and/or phosphorus grain foods	2-3	50-200	150-400 (if marked with b **)	10-100	100-200 (if marked with a*)

*a—food contains 100-200 mg phosphorus per serving.

**b—food contains 150-400 mg sodium per serving.

***Refer to Bread, Cereal, and Grain Choices Food Lists under Healthy Food Guide for People With Chronic Kidney Disease (Pre-ESRD), this section.

CALORIE AND FLAVORING CHOICES

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List*	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Calorie choices	0-1	100-150	0-100	0-100	0-100
Flavor choices	0	0-20	250-300	0-100	0-20

*Refer to Calorie and Flavoring Choices Food List under Healthy Food Guide for People With Chronic Kidney Disease (Pre-ESRD), this section.

VEGETARIAN PROTEIN CHOICES

	Protein	Calories	Sodium	Potassium	Phosphorus
Food List****	(g/serving)	(kcal/serving)	(mg/serving)	(mg/serving)	(mg/serving)
Protein foods	6-8	70-150	10-200	60-150	80-150
Higher sodium,	6-8	70-150	250-400	250-500	200-400
potassium, or				(marked with	(marked with
phosphorus proteins				c ***)	a*)

*a-food contains 200-400 mg phosphorus per serving.

***c-food contains 250-500 mg potassium per serving.

****Refer to Vegetarian Protein Food Choices Lists under Healthy Food Guide for People With Chronic Kidney Disease (Pre-ESRD), this section.

References

- 1. National Renal Diet Professional Guide. 2nd ed. Chicago, Ill: Renal Practice Group of the American Dietetic Association; 2002.
- 2. Schiro Harvey K. *A Healthy Food Guide for People With Chronic Kidney Disease*. 2nd ed. Chicago, Ill: Renal Practice Group of the American Dietetic Association; 2002
- 3. Schiro Harvey K. A *Healthy Food Guide for People on Dialysis*. 2nd ed. Chicago, Ill: Renal Practice Group of the American Dietetic Association; 2002.

MEAL PATTERNS USING HEALTHY FOOD GUIDE (SAMPLE)

Sample Meal Pattern for CKD (Pre-ESRD)

Based on 70 kg reference person	1
---------------------------------	---

Food Choice	Number of	Breakfast	Noon	Evening
	Choices			
High-Protein	3	1	1	1
Choices				
High-Protein, High	1	1		
Phosphorus Choices				
Vegetable Choices				
Group 1	1		1	
Group 2	1		1	
Group 3	1			1
Bread, Cereal and	8	3	2	3
Grain Choices				
Fruit Choices				
Group 1	1			1
Group 2	1		1	
Group 3	1	1		
Calorie and	6-7	2-3	2	2
Flavoring Choices				

Approximate Totals:

61 g Protein 2,130 calories 2,090 mg sodium

2,160 mg potassium

840 mg phosphorus

Sample Meal Pattern for CKD (Dialysis)

Based on 70 kg reference person

Food Choice	Number of Choices	Breakfast	Noon	Evening
High-Protein	8	2	3	3
Choices				
Dairy and	1	1		
Phosphorus Choices				
Vegetable Choices				
Group 1	1		1	
Group 2	2		1	1
Group 3				
Bread, Cereals and	9	3	3	3
Grain Choices				
Fruit Choices				
Group 1	1			1
Group 2	1		1	
Group 3	1	1		
Calorie and	6-7	2-3	2	2
Flavoring Choices				
Fluid Choices	3			

Approximate Totals: 84 g Protein

2350 calories 2150 mg sodium 2220 mg potassium 1100 mg phosphorus 960 cc fluid

SIMPLIFIED RENAL DIET

Description

The Simplified Renal Diet mildly restricts sodium, potassium, phosphorus, and fluid intake.

Indications

- patients in predialysis stage
- patients receiving hemodialysis and having difficulty adhering to the Renal Diet

Nutritional Adequacy

The diet is inadequate in calcium according to the Dietary Reference Intakes (DRIs) as outlined in the Statement on Nutritional Adequacy in Section IA.

How to Order the Diet

The diet should be ordered "No Added Salt Diet (NAS) _____ cc fluid restriction, Simplified Renal Diet." For patients who require a renal-diabetic restriction, order "Consistent Carbohydrate, NAS Diet with _____ cc fluid restriction, Simplified Renal Diet."

Planning the Diet

Guidelines for the Simplified Renal Diet follow:

- 1. Limit milk and milk products to $\frac{1}{2}$ cup/day.
- Limit foods high in potassium to one serving per day. Such foods include cantaloupe and honeydew, potatoes, prunes, oranges, orange juice, prune juice, dried beans and peas, nuts and peanut butter, chocolate, bananas, apricots, and tomatoes. (The renal choice list may be used for guidelines of a serving.)
 Eliminate salt substitutes and light salt
- 3. Eliminate salt substitutes and light salt.
- 4. If phosphorus restriction is required, limit bran cereal, whole wheat bread, nuts, and dried beans to one serving per day.
- 5. For protein requirements, provide 6 oz of meat or meat entree per day. Offer an egg for breakfast at least every other day.

Bibliography

Ecklund K. Handling the dialysis diet in long-term care. *Consultant Dietitian*. 1992; 17 (1): 1.

I. NORMAL NUTRITION AND MODIFIED DIETS H. Diets for Sensitivity/Miscellaneous Intolerances

Gluten-Free Diet Food Guide – Gluten-Free Diet	H-1 H-6
Tyramine-Restricted Diet.	H-10
Food Guide – Tyramine Restriction	H-11
Lactose-Controlled Diet	H-12
Lactose Content of Milk Products	H-14
Food Guide — Lactose-Controlled	H-14
Nutrition Management Of Food Hypersensitivities	H-16
Corn-Free Diet	H-17
Egg-Free Diet	H-17
Milk-Free Diet	H-17
Peanut-Free Diet	H-18
Soy-Free Diet	H-18
Wheat-Free Diet	H-18
Shellfish-Free Diet	H-19
Tree Nut-Free Diet	H-19

GLUTEN-FREE DIET

Description

The Gluten-Free Diet is the primary treatment for celiac disease, which is also called gluten-sensitive enteropathy or celiac sprue. The only treatment for celiac disease is lifelong adherence to a gluten-free meal pattern, including strict avoidance of prolamins, which are proteins found in wheat, rye, barley, and triticale (1). Dermatitis herpetiformis is the term for the skin manifestation of celiac disease. The Gluten-Free Diet also helps to control most cases of dermatitis herpetiformis associated with gluten-sensitive enteropathy (1-6).

Indications

Celiac disease is an immune-mediated disease characterized by chronic inflammation of the small intestine mucosa that results in malabsorption due to atrophy of the intestinal villa (1-6). Although celiac disease was once thought to be a rare childhood disease, it is now recognized as a fairly common multisystem disorder that occurs in one in 133 people (1,6). Individuals with celiac disease have an immunologic reaction to proteins termed prolamins, which are found in wheat, rye, and barley (1). When foods containing gluten are consumed by a person with celiac disease, the digestive process fails and an immunologically reactive protein fragment remains (1). Research suggests that a 33–amino acid molecule may be the cause of the inflammatory response (1,4-6). This molecule_enters the intestinal mucosa and cannot be degraded by digestive enzymes or pancreatic enzymes. The molecule then enters the lamina propria, where it causes the release of T cells (1). The presence of the T cells in the lamina propria triggers cytokine activation, antibody production, and inflammatory responses (1). The resulting villous atrophy and inflammation of the mucosa result in malabsorption (1-6). The proximal bowel (duodenum) is the first area of the gastrointestinal tract to be exposed to the immunologically reactive peptide. Therefore, it is exposed to the highest concentration of the peptide and is often the most severely injured section of the small intestine. The jejunum and occasionally the ileum may also be affected.

Although the classic presentation of celiac disease is diarrhea, wasting, malabsorption, failure to grow, bloating, and abdominal cramps, not all individuals with celiac disease have these symptoms. Many individuals with celiac disease are diagnosed when seeking medical care for other problems such as anemia, osteoporosis, peripheral neuropathy, and fatigue (1,6). Celiac disease is categorized into four main classes according to the National Institutes of Health Consensus Conference Statement (6):

Classical celiac disease: This class is characterized by predominant gastrointestinal symptoms and sequelae including malabsorption, significant weight loss or gain, failure to grow (in children), diarrhea, constipation, excessive gas, bloating, and abdominal pain. The diagnostic testing reveals positive serologic test results and biopsy-proven intestinal atrophy. Symptoms improve after a patient adopts a gluten-free diet.

Celiac disease with atypical symptoms: This class is characterized by predominantly extraintestinal manifestations and few or no gastrointestinal symptoms. Non-gastrointestinal symptoms include anemia, osteoporosis, peripheral neuropathy or neurological symptoms, dental enamel defects, and fatigue. The diagnostic test results and treatment response are consistent with classical celiac disease.

Silent celiac disease: This disease is characterized by a lack of clinical symptoms in spite of positive serologic test results and biopsy-proven villous atrophy. Diagnosis of silent celiac disease usually results from screening high-risk individuals, eg, family members and individuals with associated conditions such as type I diabetes mellitus, Down syndrome, or Williams syndrome. A clear outcome benefit of treating these individuals has not emerged from current data analysis.

Latent celiac disease: This class is characterized by positive serologic test results, the absence of villous atrophy on intestinal biopsy, and no clinical symptoms of celiac disease. These individuals may develop intestinal changes and symptoms of celiac disease later in life.

Dermatitis herpetiformis: This condition is the skin manifestation of celiac disease, which is characterized by a bilateral, symmetric rash or eruptions primarily on pressure points of the skin that may evolve into blisters or bullae (fluid-filled sacs). These lesions are painfully itchy and do not respond well to topical treatment. Dermatitis herpetiformis is diagnosed from a skin biopsy taken from a site next to a lesion. Ninety percent of individuals with dermatitis herpetiformis have no gastrointestinal symptoms characteristic of celiac disease, but 75% have biopsy-proven villous atrophy that responds well to a gluten-free dietary pattern. Topical treatment of the lesions with sulfapyridine is effective in treating this form of bullous atopic

Gluten-Free Diet

dermatitis. Although oral medications may also be used, adherence to a gluten-free diet is the most effective way to prevent dermatitis herpetiformis.

Compliance with a gluten-free dietary pattern reduces the prevalence of diarrhea, constipation, abdominal pain and bloating, nausea or vomiting, reduced gut motility, delayed gastric emptying, and prolonged transit time (Grade II)* (7). Evidence is limited regarding the effect of a gluten-free dietary pattern on indigestion, dysphagia, and reflux (Grade II) (7). Individuals who comply with a gluten-free dietary pattern have substantial improvement in villous atrophy; however, mucosal abnormalities may persist in some individuals (Grade II) (7). Although normalization of abnormalities may occur within 1 year, it generally takes longer, depending on the severity of villous atrophy, level of compliance, and age at diagnosis (Grade II) (7). Recovery in children may progress faster and more completely than in adults (Grade II) (7). People with celiac disease are more likely than healthy controls to experience neurological symptoms such as depression, cerebellar ataxia, headaches, migraines, and neuropathy (Grade II) (7). Early diagnosis and compliance with a gluten-free dietary pattern may reduce the prevalence of symptoms related to cerebellar ataxia, headaches, and migraines (Grade II) (7). The evidence is less conclusive or limited regarding the effect of a gluten-free diet on depression, anxiety, and epilepsy (Grade II) (7).

Nutrition Assessment and Diagnosis

Biopsy of the small intestine is the gold standard for diagnosing celiac disease (1-6). Several biopsies should be taken because mucosal abnormalities may be localized (6). Criteria for diagnosis include mucosal abnormalities (eg, increased density of intraepithelial lymphocytes, partial to total villous atrophy, and crypt hyperplasia) and clinical improvement after a period of time on a gluten-free nutrition prescription (6.8). Tests for genetic markers are available to determine the likelihood that a person has celiac disease (1). The DQ2 and DQ8 markers are highly correlated with celiac disease and are tools for assessing a person's risk for celiac disease (6). Persons who exhibit symptoms of irritable bowel syndrome or who have undiagnosed gastrointestinal complaints (eg, diarrhea, bloating, gas, and abdominal pain), especially when accompanied by fatigue and weight loss, should be assessed for celiac disease. In a survey of adults with celiac disease, 37% of cases reported an initial diagnosis of irritable bowel syndrome (9). Serologic markers that can be used by dietitians to screen for celiac disease include immunoglobulin A, antihuman tissue transglutaminase, and immunoglobulin A endomysial antibody (1). Tests for these markers have a high sensitivity and specificity and are the best available tests in terms of diagnostic accuracy (1,6).

A comprehensive nutritional assessment is critical in determining whether recurrent symptoms are related to gluten sensitivity or to an unrelated problem. Damage to the intestinal mucosa may cause various degrees of malabsorption that leads to deficiencies of key vitamins and minerals, including calcium, vitamin D, iron, and folate (4). The following discussion reviews the evidence regarding the long-term effects of following a gluten-free dietary pattern after a diagnosis of celiac disease (7).

Calcium: Clinical trials and cross-sectional studies have found reduced bone mineral content and bone mineral density in untreated children, adolescents, and adults (7). Both of these parameters improve significantly with compliance to a gluten-free dietary pattern for at least 1 year (Grade I) (7). Compliance with dietary treatment initiated during childhood or adolescence allows the achievement of normal bone mineralization (Grade I) (7). However, in adults who received no treatment or delayed treatment in childhood or adolescence, a gluten-free meal pattern may improve bone density but not normalize bone mineral density (Grade I) (7). Successful treatment depends on the age at diagnosis, as patients who do not receive treatment in childhood and adolescence may never reach peak bone mass (Grade I) (7). Further studies are needed to evaluate the effects of calcium and vitamin D supplementation on bone mineral content and bone mineral density, as well as the effects of hormone replacement therapy for postmenopausal women (7). Adults with celiac disease should have a bone density test (dual energy X-ray absorptiometry scan) at the time of the diagnosis (6).

Iron: For most children and adults with celiac disease, compliance with a gluten-free dietary pattern results in significant improvement in hematological parameters including serum hemoglobin, iron, ferritin, mean corpuscular volume, mean corpuscular hemoglobin, and red cell distribution width (Grade II) (7). Recovery from anemia, as indicated by the normalization of hemoglobin concentrations, generally occurs within 6 months; recovery from iron deficiency, as indicated by the normalization of ferritin concentrations, may take longer than 1 year (Grade II) (7). Iron supplementation in the form of a multivitamin with iron may be necessary to achieve normal values for these hematological variables within these time periods (Grade II) (7).

Lactose: Patients may need to be evaluated for lactose intolerance, which can appear secondary to celiac disease. If the patient is lactose intolerant, see the discussion of the Lactose-Controlled Diet later in this section. Usually lactose intolerance will normalize within weeks to months of adopting a gluten-free diet pattern (1).

Contraindications

One form of celiac disease, refractory sprue, does not respond to the Gluten-Free Diet or responds only temporarily.

Nutritional Adequacy

The Gluten-Free Diet can be planned to meet the Dietary Reference Intakes as outlined in the Statement on Nutritional Adequacy in Section IA. Compliance with a gluten-free dietary pattern results in significant improvements in nutritional laboratory values, such as serum hemoglobin, iron, zinc, and calcium, as a result of intestinal healing and improved absorption (Grade II) (7). Often, supplementation may be required to treat deficiencies secondary to celiac disease (1). Anemia may be treated with folate, iron, or vitamin B₁₂. Patients who are dehydrated due to severe diarrhea require electrolytes and fluids. Vitamin K may be prescribed for patients who develop purpura, bleeding, or prolonged prothrombin time. Calcium and vitamin D supplementation may be necessary to correct osteomalacia. Vitamins A and D may be necessary to replenish stores depleted by steatorrhea. Daily consumption of a gluten-free, multivitamin-mineral supplement containing the Dietary Reference Intakes is recommended for patients who continue to have suspected deficiencies or malabsorption (1,7).

How to Order the Diet

Order as "Gluten-Free Diet."

Nutrition Intervention and Prescription

The Gluten-Free Diet is based on the avoidance of the grains, chemicals, and natural or artificial ingredients that are toxic for patients with celiac disease or dermatitis herpetiformis (1). This diet eliminates all foods containing wheat, rye, barley, triticale, and their derivatives (1,6). Derivatives of these grains include wheat-based spelt, semolina, and kamut. Quinoa, buckwheat, amaranth, and teff are allowed on a gluten-free diet, based on plant taxonomy and limited scientific evidence for the need to exclude these items (1). Millet, sorghum, Job's tears, teff, ragi, and wild rice are more closely related to corn than to wheat. The American Dietetic Association, Dietitians of Canada, and other organizations such as the Gluten Intolerance Group and the Celiac Disease Foundation consider these plants to be acceptable in a gluten-free diet (1,6,10). The following grains and plant foods can be included in a gluten-free prescription (1):

- Rice, corn, amaranth, quinoa, teff (or tef), millet, finger millet (ragi), sorghum, Indian rice grass (Montina), arrowroot, buckwheat, flax, Job's tears, sago, potato, soy, legumes, tapioca, wild rice, cassava (manioc), yucca, and nuts
- Nonmalt vinegars, including cider vinegar, wine vinegar, and distilled vinegar

Oats: Studies have shown that incorporating oats uncontaminated with wheat, barley, or rye into a glutenfree dietary pattern at intake levels of approximately 50 g of dry oats per day is generally safe for people with celiac disease and improves their compliance (Grade II) (7,11-15). However, the introduction of oats may result in gastrointestinal symptoms such as diarrhea and abdominal discomfort (7,16-18). Additional adverse effects include dermatitis herpetiformis, villous atrophy, and an increased density of intraepithelial lymphocytes, indicating that some persons with celiac disease may be unable to tolerate oats (Grade II) (7). The risk of crosscontamination with gluten-containing products remains a substantial concern in the United States. Some food companies such as Gluten Free Oats and Cream Hill Estates are attempting to improve the purity of oat production and may be a resource for persons with celiac disease (1). Until oats are proven safe, the inclusion of oats in a gluten-free diet should be at the discretion of patients in consultation with their physicians and dieititians (1). Patients who consume oats should be advised to limit their daily consumption to approximately 50 g of dry oats, an amount found to be safe in studies (19). Ideally, patients should only consume oats that have been tested and found to be free of gluten contamination (1,19).

Wheat starch-based gluten-free foods: Both natural and wheat starch-based gluten-free foods (as defined by the Codex Alimentarius (20)) produce similar histological and clinical recovery in people with celiac disease (Grade III) (7). Overall compliance with a gluten-free diet may be more important than the specific type of diet (eg, natural or wheat starch-based), as evidenced by the incomplete bowel mucosal recovery and positive serological test results generally seen in study subjects who have dietary lapses (Grade III) (7).

Gluten-Free Diet

Alcohol: Beer, ale, porter, stout, and other fermented beverages should be avoided because they are derived from barley (1). Distilled alcoholic beverages (eg, gin and vodka) may be included in a gluten-free nutrition prescription. Although these beverages may be derived from gluten-containing grain, the process of distillation should prevent any protein from remaining in the final distillate (1). Checking the manufacturer's label is important with all types of alcoholic products because gluten-containing additives may be added after the alcohol is distilled.

The following guidelines should be considered when determining the nutrition prescription (1):

- The daily protein intake for adults should be 1 to 2 g/kg of body weight (1). Use high-biological value proteins.
- Adequate energy intake should be determined by using the Ireton-Jones or Mifflin–St. Jeor equations, or as outlined in Section II: Estimation of Energy Expenditures. Consider the need for weight gain if weight loss is unintentional and associated with disease.
- Evaluate the need for gluten-free vitamin and mineral supplementation.
- Evaluate the need for a medium-chain triglyceride supplement in adults who are diagnosed with steatorrhea.

The_long-term nutritional adequacy of a gluten-free diet has been investigated. Adherence to the gluten-free dietary pattern may result in a diet that is high in fat and low in carbohydrates and fiber, as well as low in iron, folate, niacin, vitamin B_{12} , calcium, phosphorus, and zinc (Grade II) (7). A food intake survey of persons with celiac disease found that less than 50% of the female participants consumed the recommended amounts of fiber, iron, and calcium (21). A small number of studies of adults show a trend toward weight gain after diagnosis (Grade II) (7). These factors may need to be considered during long-term patient management. The following dietary guidelines may be suggested to persons with celiac disease (1):

- Consume 5- to 10-oz equivalent servings from the grain food group each day.
- Choose whole grain, gluten-free products whenever possible.
- Choose enriched, gluten-free products over refined, unenriched products whenever possible.
- Increase intake of gluten-free products made from alternative plant foods (eg, amaranth, buckwheat, quinoa, teff, and flaxseed) to provide good sources of fiber, iron, and some B vitamins.
- Increase intake of other enriched, gluten-free foods, such as rice and energy bars.
- Increase intake of noncereal sources of thiamin, riboflavin, niacin, folate, iron, and fiber.
- Consider taking a gluten-free multivitamin and mineral supplement. Gluten-free brands include Freeda (www.freedavitamins.com) and Nature's Bounty (www.naturesbounty.com).

Cross-contamination with gluten-containing grains or gluten-containing products during processing, preparation, or food handling should be avoided. Patients will need to become proficient in the evaluation of food and manufacturers' labels to screen the ingredients in food products, dietary or medical food supplements, and medications. Hidden sources of gluten in food products include: hydrolyzed vegetable protein, flavorings, malt flavoring (includes malt syrup, malt extract, malt milk, and malt vinegar), brown rice syrup, modified food starch, dextrin, caramel color, vegetable gum, soy sauce, monoglycerides and diglycerides in dry products, emulsifiers, alcohol-based extracts (eg, vanilla extract), prepared meats, and flavored coffees. The following additional components contain gluten and are often overlooked: broth, breading, croutons, pasta, stuffing, flours, sauces, coating mixes, marinades, thickeners, roux, soup base, self-basting poultry, imitation seafood, and imitation bacon (1). According to the U.S. Food and Drug Administration's Code of Federal Regulations (21CFR137), the following terms on a food label or ingredient list indicate the presence of wheat (22):

• flour, white flour, plain flour, bromated flour, enriched flour, phosphate flour, self-rising flour, durum flour, farina, semolina, and graham flour (1)

Effective January 1, 2006, under the Food Allergen Labeling and Consumer Protection Act of 2004 (FALCPA), if a food or an ingredient contains wheat or protein derived from wheat, the word "wheat" must be clearly stated on the food label (1,22). FALCPA applies not only to food products but also to dietary supplements, infant formulas, and medical foods. This regulation includes products that contain dextrin, caramel color, or modified food starch found in food products containing protein derived from wheat (1,22). All prescribed and over-the-counter medications should be evaluated by a knowledgeable pharmacist or physician prior to use. Ingredients used as part of the packaging are not required to be listed on the label. Persons with celiac disease should be aware of the potential for cross-contamination with gluten-containing foods that may occur as result of preparing or cooking foods (eg, frying or grilling).

Nutrition Evaluation and Monitoring

Persons with celiac disease may experience an improvement in symptoms after 3 to 6 days of consuming a gluten-free diet, with full improvement of the intestinal mucosa within 6 months (23). Individuals with celiac disease demonstrate improved quality of life after compliance with a gluten-free dietary pattern for at least 1 year (Grade II) (7). Celiac disease is a chronic disease. An asymptomatic state depends on lifelong maintenance of the Gluten-Free Diet. Patients should be cautioned against ingesting gluten once they start to gain weight and feel better. The ingestion of gluten damages the mucosa and causes recurrent symptoms, although several weeks may lapse before the patient observes symptoms. Villous atrophy is significantly associated with dietary compliance (Grade II) (7). Therefore, an assessment of dietary adherence is critical in determining whether recurrent symptoms are related to gluten sensitivity or to an unrelated problem. Individuals who are diagnosed with celiac disease and are not treated or do not adhere to a gluten-free diet are at greater risk of developing osteoporosis and benign and malignant complications including lymphoma and other autoimmune diseases such as type 1 diabetes mellitus (1-6).

|--|

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Beverages	Brewed coffee (regular and decaffeinated), tea Instant and freeze-dried Sanka, Maxwell House, and Brim coffee Cocoa Carbonated beverages, except some root beer Artificially flavored fruit drinks, cider Wine, rum	Some herbal teas Other instant coffees Some fruit-flavored drinks ^a Some cocoa or chocolate mixes ^a Some root beer Beer ^a , ale, and other distilled spirits made from cereal grains ^b
Breads, Cereals, and Grain Products	Specially prepared breads, crackers, cakes, cookies, pasta, and other products made with these flours and starches: cornflower, cornstarch, cornmeal, potato starch, rice flour, soy flour, soybean starch, tapioca, arrowroot starch, whole-bean flour, sago, rice bran, buckwheat, millet, flax, teff, sorghum, amaranth, and quinoa Pure corn tortillas Potato chips made only of potato; corn chips made only of corn Plain popcorn Rice cakes (check label) Hominy grits Rice, rice noodles Corn or rice cereals containing malt flavoring derived from corn; cream of rice; puffed rice; puffed millet	 Breaded foods Breads, crackers, muffins, pizza crust, and other products made from barley, oat^a, rye, or wheat flour; triticale; graham flour; gluten flour; bulgur; farina; wheat-based semolina; spelt; kamut Bran or wheat germ Commercial "gluten" bread Commercially prepared mixes for buckwheat pancakes or corn bread Cracker crumbs Pretzels, chips, and other snack foods, except those allowed Pasta and noodles made from barley, oat, rye, or wheat flour Communion wafers
Vegetables	Plain, fresh, frozen, or canned vegetables, except those excluded	Commercially prepared vegetables and salads (eg, some restaurant french fries or battered vegetables) ^a Vegetables prepared with sauces Canned baked beans
Fruits and Juices	Fresh, frozen, canned, or dried fruit Fruit juices	Prepared fruits with excluded flours or grains (eg, some pie fillings and thickened fruits)
Milk	Milk Chocolate milk (check label)	Cereal beverages such as Ovaltine Some commercial chocolate milk ^b , malted milk, instant milk mixes
Meat and Meat Substitutes	 Pure meat, fish, poultry, eggs, bacon, and ham Pure cottage cheese; natural hard and semisoft cheeses Peanut butter, soybeans, dried beans, and other legumes; tofu Cold cuts, frankfurters, or sausage without fillers^a 	Breaded meat, fish, or poultry Canned or frozen meat dishes, stews, chili Patties, loaves, and croquettes made with bread crumbs or flour Some prepared meats such as cold cuts, frankfurters, sausages, and some hamburgers ^{a b} Processed cheese, cheese food, and cheese spreads Textured or hydrolyzed vegetable or plant protein products (TVP, HVP, HPP) Self-basting turkey with HVP added Cheese products containing oat gum as an ingredient Imitation crab containing wheat, starch, or other unacceptable fillers
 nems may or may n 	iot contain giuten or narmful prolamins. Verify ingredient l	ist and purity of product with supplier.

^b Check product label and contact manufacturer to clarify questionable ingredients, especially the source of flavoring in meat and poultry products.

FOOD GROUP	FOODS ALLOWED	FOODS EXCLUDED
Meat and Meat Substitutes		Frozen individual fish (may be dusted with flour); tuna canned with hydrolyzed protein
Fats	Butter, margarine, lard, cream, shortening, oils Mayonnaise Nuts Olives Gravy and sauce made with allowed thickening agents Salad dressings that do not contain a gluten stabilizer Cream cheese	Some commercial salad dressings (consult label) Cream sauce thickened with flour Nondairy cream substitute Commercially prepared gravy and sauce Coated and flavored nuts
Soups	Homemade broths; vegetable or cream soups made with allowed ingredients	Commercially prepared soups, bouillon, or broth with HVP or HPP ^b Soups containing barley, pasta, noodles, HVP, or HPP
Desserts and Sweets	Cakes, cookies, or pastries made from allowed flours or starches and cereal-free baking powder Custard Cornstarch, rice, and tapioca puddings Gelatin desserts Kozy Shack puddings and flans Ice cream with a few simple ingredients (usually brands that are expensive) Sorbet, frozen yogurt, and sherbet (check labels) Coconut Marshmallows Hard candy Commercial and homemade candies free from excluded grains Sugar, honey, corn syrup, maple syrup, jam, jelly, molasses	Ice cream containing stabilizers ^a Commercially made puddings Cookies, cakes, pies, pastry, and other baked items, unless specially prepared Doughnuts Bread pudding Products made with brown rice syrup prepared with barley malt enzyme Flavored syrups Chocolate and other candy containing excluded ingredients Desserts with malt, malt flavoring, or natural flavoring Chocolate-covered nuts that may have been rolled in wheat flour
Miscellaneous	Salt, monosodium glutamate, tamari, spices, herbs, flavoring extracts, dry mustard Dry yeast Pure cocoa and chocolate Cider Vinegar, except malt vinegar Pickles, olives	Soy sauce ^b , commercial catsup ^b , chili sauce ^b , barbecue sauce, Worcestershire sauce, horseradish, seasoning mixes Cake yeast, baking powder Some pizzas ^a Licorice Chewing gum ^b Malt vinegar

Malt vinegar ^a Items may or may not contain gluten or harmful prolamins. Verify ingredient list and purity of product with supplier. ^b Check product label and contact manufacturer to clarify questionable ingredients, especially the source of flavoring in meat and poultry products.

SAMPLE MENU				
Breakfast	Noon	Evening		
Orange Juice	Baked Chicken	Braised Beef Tips		
Cream of Rice	Steamed Rice	Whipped Potatoes		
Soft-Cooked Egg	Steamed Broccoli With Carrots	Green Beans		
Gluten-free Bread	Gluten-free Bread	Sliced Tomato Salad		
Margarine, Jelly	Margarine	Peach Halves		
Milk	Pineapple Chunks	Gluten-free Bread		
Coffee	Milk	Margarine		
Sugar, Creamer	Iced Tea, Sugar	Iced Tea, Sugar		
Milk Coffee Sugar, Creamer	Pineapple Chunks Milk Iced Tea, Sugar	Gluten-free Bread Margarine Iced Tea, Sugar		

CAMDI E MENII

Substitutions for Wheat Flour

Most patients find special cookbooks helpful. Recipes can be modified by the following substitutions:

For baking, 1 cup wheat flour may be replaced by:

1 cup corn flour (finely milled)
 1 scant cup fine cornmeal
 ³/₄ cup coarse cornmeal
 5/8 cup (10 tbsp) potato starch flour
 7/8 cup (14 tbsp) rice flour (white or brown)
 1 cup soy flour plus ¼ cup potato starch flour
 ½ cup soy flour plus ½ cup potato starch flour

For thickening, 1 tbsp of wheat flour may be replaced by:

1¹/₂ teaspoons of cornstarch, potato starch, rice, flour arrowroot starch, or gelatin

2 teaspoons of quick-cooking tapioca

1 tbsp rice flour (white or brown)

Support Groups

American Celiac Society PO Box 23455 New Orleans, LA 70183 (504) 737-3293 www.americanceliacsociety.org

Celiac Sprue Association PO Box 31700 Omaha, NE 68131-0700 (877) CSA-4CSA www.csaceliacs.org Celiac Disease Foundation 13251 Ventura Blvd., #1 Studio City, CA 91604 (818) 990-2354 www.celiac.org

Gluten Intolerance Group of North America 31214 124th Ave SE Auburn, WA 98092-3667 (253) 833-6655 www.gluten.net

Suppliers of Gluten-Free Products

Bob's Red Mill (800) 553-2258 www.bobsredmill.com

Gluten Solutions (888) 845-8836 www.glutensolutions.com

Health Valley* (800) 434-4246 www.healthvalley.com

Kinnikinnick Foods* (877) 503-4466 www.kinnikinnick.com

Miss Roben's (800) 891-0083 www.missroben.com Ener-G Foods* (800) 331-5222 www.ener-g.com

Gluten-Free Mall (866) 575-3720 www.glutenfreemall.com

Heartland's Finest (888) 658-8909 www.heartlandsfinest.com

Maple Grove Food and Beverage* (323) 322-0501 www.maplegrovefoods.com

Pamela's Products (707) 462-6605 www.pamelasproducts.com Enjoy Life Foods* (888) 503-6569 www.enjoylifefoods.com

The Gluten-Free Pantry/Glutino (800) 291-8386 www.glutino.com

Kingsmill Foods (416) 755-1124 www.kingsmillfoods.com

Med-Diet (800) 633-3438 www.med-diet.com

Perky's Natural Foods* (888) 473-7597 www.perkysnaturalfoods.com

*Provides enriched gluten-free bread products or baking mixes

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Celiac disease. The American Dietetic Association Nutrition Care Manual. Updated Annually. Available at: nutritioncaremanual.org. Accessed October 26, 2010..
- 2. Abdulkarim AS, Murray JA. Review article: the diagnosis of coeliac disease. Aliment Pharmacol Ther. 2003;17:987-995.
- 3. Alaedini A, Green PH. Narrative review: celiac disease: understanding a complex autoimmune disorder. *Ann Intern Med.* 2005;142:289-298.
- 4. Farrell RJ, Kelly CP. Celiac sprue. N Engl J Med. 2002;346:180-188.
- 5. Fasano A, Catassi I. Current approaches to diagnosis and treatment of celiac disease: an evolving spectrum. *Gastroenterology*. 2001;120:636-651.
- National Institutes of Health Consensus Development Conference Statement. NIH Consensus Development Conference on Celiac Disease. June 28-30, 2004; Bethesda, Md. Final Statement August 9, 2004. Available at: http://consensus.nih.gov/2004/2004CeliacDisease118html.htm. Accessed November 27, 2007.
- Celiac Disease Evidence Based Nutrition Practice Guidelines. American Dietetic Association Evidence Analysis Library. The American Dietetic Association; 2009. Available at: http://www.adaevidencelibrary.org. Accessed October 25, 2010.
- 8. Maki M, Collin P. Coeliac disease. *Lancet.* 1997;349:1755-1759.
- 9. Zipser RD, Patel S, Yahya KZ, Baisch DW, Monarch E. Presentations of adult celiac disease in a nationwide patient support group. *Dig Dis Sci.* 2003;48:761-764.
- 10. Thompson T. Case Problem: questions regarding the acceptability of buckwheat, amaranth, quinoa, and oats from a patient with celiac disease. *J Am Diet Assoc.* 2001;101:586-587.
- 11. Janatuinen EK, Kemppainen TA, Pikkarainen PH, Holm KH, Kosma V-M, Uusitupa MIJ, Maki M, Julkunen RJK. Lack of cellular and humoral immunological responses to oats in adults with coeliac disease. *Gut.* 2000;46:327-331.
- 12. Hoffenberg EJ, Haas J, Drescher A, Barnhurst R, Osberg I, Bao F, Eisenbarth G. A trial of oats in children with newly diagnosed celiac disease. *J Pediatr.* 2000;137:361-366.
- 13. Janatuinen EK, Kemppainen TA, Julkunen RJK, Kosma V-M, Maki M, Heikkinen M, Uusitupa MIJ. No harm from five year ingestion of oats in coeliac disease. *Gut.* 2002;50:332-335.
- 14. Storsrud S, Olsson M, Arvidsson LR, Nilsson LA, Kilander A. Adult coeliac patients do tolerate large amounts of oats. *Eur J Clin Nutr.* 2003;57:163-169.
- Hogberg L, Laurin P, Faith-Magnusson K, Grant C, Grodzinsky E, Jansson G, Ascher H, Browaldh L, Hammersjo JA, Lindberg E, Myrdal U, Stenhammer L. Oats to children with newly diagnosed coeliac disease: a randomised double blind study. *Gut.* 2004;53:649-654.
- 16. Lundin KE, Nilsen EM, Scott HG, Loberg EM, Gjoen A, Bratlie J, Skar V, Mendez, E, Lovik A, Kett K. Oats induced villous atrophy in coeliac disease. *Gut.* 2003;52:1649-1652.
- 17. Peraaho M, Kaukinen K, Mustalahti K, Vuolteenaho N, Maki M, Laippala P, Collin P. Effect of oats-containing gluten-free diet on symptoms and quality of life in coeliac disease: a randomized study. *Scand J Gastroenterol.* 2004;39:27-31.
- 18. Arentz-Hansen H, Fleckenstein B, Molberg O, Scott H, Koning F, Jung G, Roepstorff P, Lundin KEA, Sollid LM. The molecular basis for oat intolerance in patients with celiac disease. *PLoS Med.* 2004;1:e1.
- 19. Thompson T. Oats and the gluten-free diet. J Am Diet Assoc. 2003;103:376-379.
- 20. Joint FAO/WHO Food Standards Program. Codex Committee on Nutrition and Foods for Special Dietary Uses. Draft revised standard for gluten-free foods. *CX/NFSDU 98/4*. July 1998:1-4.
- 21. Thompson T, Dennis M, Higgins L, Lee A, Sharrett M. Gluten-free diet survey: are Americans with coeliac disease consuming recommended amounts of fibre, iron, calcium and grain foods? *J Hum Nutr Diet.* 2005;18:163-169.
- 22. US Food and Drug Administration's (FDA) Code of Federal Regulations (21CFR137). Available at: http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=108_cong_bills&docid=f:s741es.txt.pdf. Accessed November 29, 2007.
- 23. Godkin A, Jewell D. The pathogenesis of celiac disease. *Gastroenterology*. 1998;115:206-210.

Bibliography

Green PH, Jabri B. Coeliac disease. Lancet. 2003;362:383-391.

- Fasano A, Berti I, Gerarduzzi T, Not T, Colletti RB, Drago S, Elitsur Y, Green PH, Guandalini S, Hill ID, Pietzak M, Ventura A, Thorpe M, Kry D, Fornardi F, Wasserman SS, Murray JA, Horvath K. Prevalence of celiac disease in at-risk and not-at-risk groups in the United States: a large multicenter study. *Arch Intern Med.* 2003;163:286-292.
- Murray JA. The widening spectrum of celiac disease. Am J Clin Nutr. 1999;69:354-365.

Thompson T. Thiamin, riboflavin, and niacin contents of the gluten-free diet: is there cause for concern? *J Am Diet Assoc.* 1999; 99: 858-862. American Gastroenterological Association medical position statement: Celiac Sprue. *Gastroenterology.* 2001; 120:1522-1525.

TYRAMINE-RESTRICTED DIET

Description

Foods containing tyramine and other vascoconstrictive amines are eliminated from the Tyramine-Restricted Diet.

Indications

The Tyramine-Restricted Diet is indicated when patients are receiving monoamine oxidase inhibitors (MAOIs) and the medication Zyvox (Linezolid), an oxazolidinone antibiotic possessing weak, reversible monoamine oxidative inhibitor activity. (1,2). MAOI's treat anxiety and depression by inhibiting the inactivation of neurotransmitters. Therapy with MAOIs is used to prevent the catabolism of dietary tyramine, which normally is metabolized in the gastrointestinal tract. The result is an increased concentration of tyramine in the body, causing the release of norepinephrine and an elevation of mood. Increase amounts of tyramine, however, can cause an excess amount of norepinephrine to be released, which may result in a hypertensive crisis. This is characterized by severe headaches, palpitation, neck stiffness or soreness, nausea or vomiting, sweating, fever, and visual disturbances.

Many foods normally contain small amounts of tyramine and other vasopressor amines. Large amounts have been reported only in aged, fermented, pickled, smoked, or bacterially contaminated products. When fresh foods are stored, especially meat, poultry, fish, and related items such as pâté, gravy, and soup stock, fermentation occurs and the tyramine content of the food increases. Since heat does not destroy tyramine, all foods should be fresh, fresh frozen, or canned and should be handled, prepared, stored, and served in ways that maximize freshness.

The consequences of tyramine intake are dose-related. Therefore, reactions can be prevented without total abstinence from tyramine-containing foods. As an example, Zyvox (Linezolid) is usually administered as an IV antibiotic in the hospital setting when typically lower tyramine foods are consumed. A recent study showed dietary restriction was not necessary due to lower tyramine content of the hospital meals (3). However, the FDA continues to recommend avoiding consuming large amounts of foods or beverages with high tyramine content while consuming Zyvox (2). A rational approach to diet compliance could best be achieved by emphasizing the most crucial items to avoid.

Caffeine does not contain tyramine, but excessive amounts may precipitate hypertensive crisis. Therefore, foods containing caffeine such as chocolate (1) should be ingested with caution. In addition, using the herb ginseng with MAO inhibitors such as Nardil or Parnate may cause headache, trouble sleeping, nervousness, and hyperactivity (1).

Nutritional Adequacy

The diet, a variation of the Regular Diet, can be planned to meet the DRIs as outlined in the Statement on Nutritional Adequacy in Section IA.

How to Order the Diet

Order as "_____ Diet, Tyramine Restricted."

Planning the Diet

Guidelines for dietary counseling in MAOI use include the following:

- 1. Begin nutrition counseling before medication therapy.
- 2. Monitor patient compliance.
- 3. Recommend preparation and consumption of only fresh foods.
- 4. Continue the diet 4 weeks beyond medication therapy.

Resynthesis of monoamine oxidase occurs slowly, and food interactions may occur up to 3 weeks after withdrawal of some MAOI medications. Prudent practice is to start the tyramine-restricted diet when the medication therapy is begun and to continue the diet for 4 weeks after the medication regimen is withdrawn.

Reference

- 1. Drug safety data: Linezolid. Available at:
 - http://www.accessdata.fda.gov/drugsatfda_docs/label/2005/021130s008,009,021131s009,010,021132s008,009lbl.pdf(200 5 data report). Accessed 8/6/2010.
- 2. Zyvox In: Drug Library. Drug Digest. Express Scripts, Inc. @ www.drugdigest.org, accessed October 1, 2005.
- 3. Rumore MM, Roth M, Orfanos A. Dietary tyramine restriction for hospitalized patients on linezolid: an update. *Nutr Clin Pract.* 2010;25:265-269.

Bibliography

Tyramine-controlled diet. In: *The Academy of Nutrition and Dietetics Nutrition Care Manual*. Updated annually. Available at: http://www.nutritioncaremanual.org. Accessed October 26, 2010.

TYRAMINE RESTRICTION – FOOD GUIDE

Beverages	Wine; beer; champagne (regular, alcohol-free, or reduced alcohol) Caffeine-containing beverages (eg, coffee, tea, or soft drinks) should b limited to two 8-oz Servings per day	
Milk	Cheese or cheese products except cottage cheese, cream, ricotta, or processed (American)	
Meats & Fish	Aged, cured, smoked, pickled, or salted meats and fish Liver; pate Hot dogs; sausage; salami; pepperoni; bacon	
Vegetables	Sauerkraut Pickled vegetables such as pickles; chili pepper Broad beans	
Fruits	None	
Miscellaneous	Soy sauce; teriyaki sauce; black bean sauce Meat tenderizers Bleu cheese, ranch, or other cheese-containing salad dressings Brewer's yeast Olives Chocolate	

Note: Patients should be reminded to consult their physician or pharmacist before taking new medications, especially cold tablets, decongestants, most allergy and asthma medications, hypertensive medications, diet pills, and sleeping pills.

MONOAMINE OXIDASE INHIBITOR (MAOI) DRUGS

GENERIC NAME	GENERAL USE
Tranylcypromine sulfate	Antidepressant
Phenelzine sulfate	Antidepressant
Isocarboxazid	Antidepressant
Furazolidone	Antimicrobial
Procarbazine hydrochloride	Anticancer

FOODS EXCLUDED

LACTOSE-CONTROLLED DIET

Description

The Lactose-Controlled Diet limits intake of milk and milk products to the amount tolerated by the individual. Refer to Lactose Maldigestion medical nutrition therapy protocol for medical nutrition intervention strategies ⁽¹⁾.

Indications

The Lactose-Controlled Diet is indicated in patients who are lactose intolerant; they are deficient in the enzyme lactase and are unable to tolerate ingested lactose. Lactose maldigestion occurs when digestion of lactose is reduced as a result of low activity of the enzyme lactase, as determined by the breath hydrogen test (2). Interpretation of the terms used to describe lactose maldigestion varies. For example, lactose intolerance refers to the gastrointestinal symptoms resulting from consumption of too much lactose relative to the body's ability to break it down by the intestinal enzyme lactase (1). Lactose maldigestion or its symptoms (lactose intolerance) should not be confused with a milk allergy, which is an allergy to milk proteins, not lactose. Lactose maldigestion is present in 70% of the world's adults and 20% to 25% of the US population. It is most prevalent among African-Americans, Asians, Hispanics, Native Americans, and people of Jewish descent. Lactose not hydrolyzed by lactase in the small intestine passes into the large intestine, where it is broken down by bacteria. The products of bacterial degradation can irritate the mucosa and raise the osmolality of the intestinal contents, causing a net secretion of fluid. Symptoms include bloating, abdominal pain, flatulence, and diarrhea, usually within 30 minutes after ingestion of lactose-containing foods.

Lactose maldigestion is not a disease, but a normal physiologic pattern (3). Primary lactase deficiency is the most common type and occurs as a normal physiological process in which lactase production in the brush border of the small intestine is reduced (3). Lactase deficiency may be secondary (secondary lactase deficiency) to significant protein-energy malnutrition, acquired immunodeficiency syndrome (AIDS), or iron deficiency anemia. Secondary lactase deficiency has also been observed following the use of antibiotics and anti-inflammatory drugs for arthritis. A transient secondary lactase deficiency may occur following viral gastroenteritis. It has been observed following surgical resection of the stomach or small bowel when there is a decrease in the absorptive area, following radiation therapy to the gastric or pelvic area, and after prolonged disuse of the gastrointestinal tract (eg, with total parenteral nutrition). However, the lactase activity may return to normal in the latter conditions over time. In children, it is typically secondary to infections or other conditions, such as diarrhea, AIDS, or giardiasis. Lactose intolerance may also be secondary to conditions that produce intestinal damage, such as celiac sprue, regional enteritis, Crohn's disease, and gluten-sensitive enteropathy.

Treatment is aimed at the underlying disorder in order to restore the patient's tolerance to lactose and to eliminate lactose restrictions. Evidence suggests that people with medically confirmed lactase maldigestion can include the recommended number of servings of milk and other dairy foods in their diet, which may actually improve their tolerance to lactose (1-3).

In feeding malnourished hospitalized patients and other patients with lactose intolerance, intolerance to 12 g of lactose can be clinically relevant. The following are used to determine the presence of lactose intolerance:

- *A diet history* can reveal symptoms of lactose intolerance following ingestion of lactose. Relief of symptoms following trial of a reduced lactose intake also indicates lactose intolerance.
- *A breath hydrogen analysis test* is the gold standard, or method of choice, to diagnose lactose maldigestion, especially in children. An increase in breath hydrogen concentration, generally 10 to 20 ppm above baseline, warrants a diagnosis of lactose maldigestion.
- *A lactose tolerance test* gives an oral dose of lactose equivalent to the amount of 1 quart of milk (50 g). In the presence of lactose intolerance, the blood glucose level increases less than 25 mg/dL of serum above the fasting level, and gastrointestinal symptoms may appear.
- *A biopsy* of the intestinal mucosa to determine lactase activity.

Congenital lactose intolerance is a rare condition. It is commonly diagnosed during the newborn period by intestinal biopsy and enzyme assay. Congenital lactose intolerance can cause life-threatening diarrhea and dehydration in the newborn. A lactose-free formula is indicated as soon as the diagnosis is made.

Nutritional Adequacy

The Low-Lactose Diet can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in the Statement on Nutritional Adequacy in Section IA. Adequate calcium can be obtained through the inclusion of dairy products, including cheese, yogurt, and milk or lactose-hydrolyzed milk.

When dairy products are limited, adequate intake of calcium, phosphorus, vitamins A and D, and riboflavin may be difficult to obtain. Because of the increased importance of calcium and its relationship to various diseases (eg, osteoporosis, hypertension) maintaining calcium intake of 1000 to 1300 mg/day for adults is a primary goal (4). Vitamin D fortified milk is the most dependable source of vitamin D. A vitamin D supplement may be indicated if exposure to sunlight is not ensured and if other foods fortified with vitamin D are not included in the diet.

How to Order the Diet

Order as "Lactose-Controlled Diet".

Planning the Diet

The important consideration is how much lactose can be tolerated without developing intestinal symptoms.

Between 80% and 100% of people with lactase deficiency experience the symptoms described if they drink 1 quart of milk a day. Research indicates that most people with low levels of lactase can comfortably ingest at least 1 cup (8 oz) of milk (12 g of lactose) with a meal and even 2 cups of milk in a day (5,6). One study has found that people with lactose maldigestion can consume 1500 mg of calcium per day if the dairy products are distributed between the three meals and provided partially in the form of yogurt and cheese (2 cups of milk, 2 oz of cheese, and 8 oz of yogurt) (7). Tolerance to milk products is greater when they are consumed with other foods and spaced throughout the day. Whole milk is better tolerated than lower fat milk, and chocolate milk is better tolerated than unflavored milk (8,9). Generally, cheeses and ice cream are better tolerated than milk because of its lower lactose content. Adults with lactose intolerance can usually tolerate the amounts of milk in many prepared foods, such as breads, luncheon meats, and creamed foods, if these foods are given at intervals throughout the day.

Milk contributes a number of important nutrients to the diet, and dairy products are a major source of calcium, protein, and riboflavin. The maximum amount of milk products that can be taken without adverse effects should be included in the diet of persons with lactose maldigestion. Tolerance to lactose can be improved by gradually increasing intake of lactose-containing foods such as dairy products (3).

Commercial lactase enzyme preparations (eg, Lactaid[®] and Dairy Ease[®]) will hydrolyze 70% to 90% of the lactose in milk depending on the amount added. Lactose-reduced milks (reduced-fat, nonfat, calcium-fortified, and chocolate) with 70% to 100% of their lactose hydrolyzed are available. Lactose-reduced cottage cheese, pasteurized processed cheese, and some ice creams are available in some markets. Lactaid[®] caplets and Dairy Ease[®] tablets, which can be taken before ingestion of milk or milk products, are also available. Products made from soy, eg, tofu, calcium and vitamin fortified soy milk, tofu-based ice cream substitutes, and pasta entrees, are also available.

The following ingredients contain lactose and can be identified on the product's food label: (dry) milk solids/curds, casein, whey (solids), and lactose.

Other compounds that may appear on the food label but do not contain lactose are calcium compounds, kosher foods marked "pareve" or "parve," lactate, and lactic acid.

10 – 15 g	1 - 6 g	<1 g ^a
Milk, fluid, 1 cup	Pudding, ½ cup	Processed American Cheese, 1 oz
	Ice Cream, ½ cup	Cream cheese, 1 oz
	Ice Milk, ½ cup	
Yogurt ^b , 1 cup	Sherbet, ½ cup	Natural Hard and Semisoft Cheeses, 1
	Processed Cheese Spread, 1 oz	OZ
	Cottage Cheese, ½ cup	Half-and-Half, 1 tbsp
	Lactaid [®] and Dairy Ease [®] Milk (<100%	Sour Cream, 1 tbsp
	reduced), 1 cup	

LACTOSE CONTENT OF MILK PRODUCTS

^a These foods are processed with small amounts of milk, milk products, milk solids, or lactose and can be considered to have minimal to undetectable amounts of lactose.

^bOnly yogurt with active cultures is well tolerated by persons with a lactase deficiency. Yogurt with active cultures is labeled "live and active culture."

FOOD GUIDE — LACTOSE-CONTROLLED

FOOD GROUPSFOODS THAT MAY CAUSE DISTRESS	
Beverages and Milk	Milk (including acidophilus milk) and milk products except yogurt ^a ; however, 4 to 8 oz of milk can usually be tolerated with meals several times per day Mocha mix
Fruits and Juices	None
Vegetables	Any prepared with milk or cheese Instant mashed potatoes containing lactose Creamed, scalloped, or commercial products containing milk
Breads and Cereals	Instant Cream of Wheat; high-protein cereals; cereals with milk
Meat, Fish, Poultry, Cheese	 Meats and meat substitutes in cream sauce Cold cuts, luncheon meats, sausage, processed meats that contain milk, nonfat milk solids or lactose filler Cottage cheese; processed cheese spread (Hard, aged cheeses, eg, bleu, brick, Camembert, cheddar, Colby, Edam, provolone, and Swiss, and processed cheeses, eg, American, Swiss are low in lactose and usually do not present a problem.)
Fats	Cream; half-and-half; whipping cream Gravies made with milk
Soups	Cream soups; chowder; commercially prepared soups that contain milk or milk products
Desserts	Ice cream Pudding, custard, and other desserts containing milk or milk products
Sugar and Sweets	Candy containing milk or cocoa Butterscotch candies, caramels, chocolate

^a Only yogurt with active cultures is well tolerated by persons with a lactase deficiency. Yogurt with active cultures is labeled "live and active culture."

SAMPLE MENU

Breakfast	Noon	Evening
Orange Juice	Honey Glazed Chicken	Braised Beef & Noodles
Oatmeal	Baked Potato With Margarine	Seasoned Green Beans
Hard-Cooked Egg	Steamed Broccoli	Sliced Tomato Salad
Biscuit	Fruited Gelatin	French Dressing
Margarine; Jelly	Dinner Roll	Peach Halves
Coffee	Margarine	Dinner Roll
Sugar; Nondairy creamer	Frosted Banana Cake	Fruited Yogurt
Milk (½ cup if tolerated) or	Milk (½ cup if tolerated)	Margarine
Lactose-Reduced Milk	Tea; Sugar	Tea; Sugar

References

- 1. Inman-Felton A. Overview of lactose maldigestion (lactose nonpersistence). J Am Diet Assoc. 1999;99:481-489.
- 2. Lactose Intolerance. Washington, DC: National Digestive Disease Information Clearinghouse; 1994. NIH Publication No. 94-2751.
- 3. McBean LD, Miller GD. Allaying fears and fallacies about lactose intolerance. J Am Diet Assoc. 1998;98:671-676.
- 4. Yates AA, Schlicker SA, Suitor CW. Dietary Reference Intake: The new basis for recommendations for calcium and related nutrients, B vitamins and choline. J Am Diet Assoc. 1998;98:699-706.
- 5. Suarez F, Savaiano D, Levitt MD. A comparison of symptoms after the consumption of milk or lactose-hydrolyzed milk by people with self-reported severe lactose intolerance. *N Engl J Med.* 1995;333:1-4.
- 6. Suarez F, Savaiano D, Arbisi P, Levitt MD. Tolerance to the daily ingestion of two cups of milk by individuals claiming lactose intolerance. *Am J Clin Nutr.* 1997;65:1502-1506.
- 7. Suarez F, Adshead J, Furne J. Levitt MD. Can lactose maldigesters tolerate the ingestion of a dairy-rich diet containing approximately 1500 mg calcium/day? New Orleans, La: American Gastroenterological Association Digestive Disease Week Syllabus. 1998;A-520, #2086.
- 8. Dehkordi N, Rao DR, Warren AP, Chawan CB. Lactose malabsorption as influenced by chocolate milk, skim milk, sucrose, whole milk, and lactose cultures. *J Am Diet Assoc.* 1995;95:484-486.
- 9. Hertzler SR, Levitt MD, Savaiono PA. Colonic adaptation in the daily lactose feeding in lactose maldigesters reduces lactose intolerance. J Am Clin Nutr. 1996;64:1232-1236.

Bibliography

Escott-Stump S. Nutrition and Diagnosis-Related Care. 5th ed. Baltimore, Md: Lippincott Williams & Wilkins; 2002.

- Hermans MM, Brummer RJ, Ruijgers AM, Stockbrugger RW. The relationship between lactose tolerance test results and symptoms of lactose intolerance. *Am J Gastroenterol* 1997;92:981-984.
- Hetzler S, Huynh BL, Savaiano DA. How much lactose is low lactose? J Am Diet Assoc. 1996;96:243-246.
- Lactose-controlled diet. Manual of Clinical Dietetics. Chicago, Ill: American Dietetic Association; 1996.
- Lactaid. Available at: http://www.jnj.merck.com. Accessed April 28, 1998.

Lee MF, Krasinsk SD. Human adult-onset lactase decline: an update. Nutr Rev. 1998;98:1-8.

Lin MY, Yen CI, Chen SH. Management of lactose maldigestion by consuming milk containing lactobacilli. Dig Dis Sci. 1998;43:133-137.

Beyer PL. Medical nutrition therapy for lower gastrointestinal tract disorders. In: Mahan KL, Escott-Stump S, eds. *Krause's Food, Nutrition and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders; 2000:679-680.

National Digestive Diseases Information Clearinghouse. Lactose intolerance. Available at: http://www.gastro.com. Accessed April 28, 1998.

Ramirez FC, Lee K, Graham DY. All lactase preparations are not the same: results of a prospective, randomized, placebo-controlled trial. *Am J Gastroenterol*. 1994;89:566-570.

Savaiano DA, Abou A, Anouar EI, Smith DE, Levitt MD. Lactose malabsorption from yogurt, pasteurized yogurt, sweet acidophilus milk, and cultured milk in lactase-deficient individuals. *Am J Clin Nutr.* 1984;40:1219-1223.

Vesa TH, Korpela RA, Sahi T. Tolerance to small amounts of lactose in lactose maldigesters. Am J Clin Nutr. 1996;64:197-201.

NUTRITION MANAGEMENT OF FOOD HYPERSENSITIVITIES

Description

This diet eliminates the offending food or foods that cause an adverse reaction. Generally, the diet is the Regular Diet with the omission of the offending food. Each individual's sensitivity to the food determines the degree to which the particular food must be omitted.

Indications

Food hypersensitivity is an immune response, generally from IgE, to food components. The reaction results from an antigen of food source (usually protein) and may occur immediately (1 minute to 2 hours) or as a delayed reaction (2 to 48 hours) (1). Allergic tendencies are inherited, but not necessarily to a specific antigen. Foods most commonly reported to cause allergic reactions in children are cow's milk, chicken eggs, peanuts, soy, and fish; in adults, the most common are tree nuts, peanuts, fish, shellfish and wheat (2-4). The most common reactions to food allergies are gastrointestinal (eg, diarrhea, nausea, vomiting, cramping, and abdominal distention and pain), skin-related, and respiratory responses as well as systemic anaphylaxis with shock.

No simple test can be used to accurately diagnose the presence of a true food hypersensitivity. Unidentified or misdiagnosed food hypersensitivities can cause fatal reactions, result in inappropriate treatments, and threaten nutritional status. For the diagnosis of hypersensitivity, the following measures should be taken: a food reaction history, a physical examination, a 1- to 2-week diary recording foods eaten and symptoms, biochemical testing, immunologic testing, eg, skin tests such as, radioallergosorbent test (RAST) and the enzyme-linked immunosorbent assay (ELISA), a trial elimination diet for 2 weeks or until symptoms are clear, and a food challenge (2,5,6).

The history, used to identify the suspected food, should include detailed descriptions of symptoms, amount of food ingested, time of intake, and time of onset of symptoms.

A trial elimination diet removes all suspected foods and reintroduces them one at a time; if the symptoms are reduced by 50% or more while the patient is on the diet, that food is suspected (5). The food challenge is made after symptoms are cleared. Although challenges can be open, single-blind, or double-blind, the double-blind, placebo-controlled food challenge (8) is the preferred method for diagnosis of food hypersensitivity. Foods are provided in a pure form, and challenged one at a time, one per day. After the trial elimination diet and food challenge, the patient's diet should be altered eliminating the response-related food for 6 to 8 weeks (5). These foods are challenged again, and if the patient does not react to them, the foods are returned to the diet on an occasional basis.

Nutritional Adequacy

The trial elimination diet is intended to be short term because of its nutrient inadequacies. Most eliminations that involve a single food can be planned to meet the Dietary Reference Intakes (DRIs) as outlined in the Statement on Nutritional Adequacy in Section IA. However, diets that eliminate cow's milk may be low in calcium, vitamin D, and riboflavin. If children must eliminate cow's milk, the diet may also be low in protein and vitamin A. Diets that restrict or eliminate eggs, meats, and fish may be deficient in protein. Grain-free diets may be deficient in B vitamins, iron, energy, and carbohydrates. Citrus-free diets may be deficient in vitamin C and folic acid. Diets that eliminate multiple foods can be deficient in certain nutrients and should be evaluated, so that appropriate alternatives are recommended to supply nutrients that are lacking. No food group should be completely eliminated on a permanent basis unless absolutely necessary.

How to Order the Diet

Order as "_____-Free Diet" (specify food to eliminate).

Planning the Diet

The basic diet should be the appropriate diet for the patient's age. Only foods confirmed by the food challenge should continue to be restricted. It is important to personalize the patient's diet based on food preferences.

Labels and recipes should be carefully read to avoid ingestion of the food that causes a reaction. Teaching the patient to read food labels, make appropriate substitutions, and purchase foods free of the suspected allergen, is the most helpful component to the self-management training. Often this training will require

more than one session. Patients should be encouraged to contact food manufacturers with questions about ingredients. The Food Allergy & Anaphylaxis Network (FAAN) has a Grocery Manufacturer's Directory and small, pocket-laminated cards listing food terminology.

These resources are available for purchase directly from FAAN (10400 Eaton Place, Suite 107, Fairfax, VA 22030, 703/691-3179, Fax, 703/691-2713, email: fan@world web.net) (5).

The following section lists ingredients and terms found on food labels, which indicate the presence of specific food allergens.

Corn-Free Diet

Ingredients to avoid:

- Baking powder
- Corn, all types
- Corn flour
- Corn grits
- Corn malt
- Corn meal
- Corn starch
- Corn sugar; corn sweeteners
- Corn syrups
- Dextrin; dextrose
- Equal[®] sugar substitute

Egg-Free Diet

- Ingredients to avoid:
 - Albumin
 - Apovitellin
 - Cholesterol-free egg substitute
 - Egg
 - Egg powder
 - Egg whites, all forms
 - Globulin
 - Livetin
 - Mayonnaise

Milk-Free Diet

Ingredients to avoid:

- Artificial butter flavor; butter-flavored oil
- Butter, butter solids
- Buttermilk
- Casein; caseinates (ammonium, calcium, magnesium, potassium, sodium)
- Cheese, all types; cheese flavor; cheese sauce; cottage cheese; cream cheese
- Cream; sour cream; whipped cream
- Curds
- Custard
- Ghee
- Goat's milk
- Half-and-half
- Hydrolysates (casein, milk, protein, whey, whey protein)
- Ice cream

Manual of Clinical Nutrition Management

- Lactalbumin; lactalbumin phosphate;
 - H-17

- Fructose
- Glucose
- Hominy
- Lactic acid
- Maize
- Maltodextrin
- Modified food starch
- Popcorn
- Sorbitol
- Vegetable gum; vegetable starch
- Meringue (meringue powder)
- Ovalbumin
- Ovoglobulin
- Ovomucin
- Ovomucoid
- Ovovitellin
- Simplesse
- Surumi
- Malted milk
- Milk: whole, low-fat, reduced fat, and nonfat
- Milk chocolate
- Milk derivative; milk powder; milk protein; milk solids; milk solid pastes
- Nonfat milk solids; nonfat dry milk
- Nougat
- Pudding
- Rennet casein
 - Simplesse
 - Sour milk solids
 - Sweetened condensed milk
 - Whey: curd, lactose-free, demineralized, sweet dairy; whey protein concentrate; whey solidsYogurt; frozen yogurt; yogurt powder
 - Foods that may indicate the presence of milk protein: caramel candies, high

Nutrition Management of Food Hypersensitivities

- lactoglobulin
- Lactate solids
- Lactose
- Lactulose

Note: The designation "pareve" on food labels indicates that the product does not contain milk.

Peanut-Free Diet

Ingredients to avoid:

- Artificial nuts
- Beer nuts
- Cold pressed or extruded peanut oil
- Ground nuts
- Mandelonas
- Mixed nuts

Soy-Free Diet

Ingredients to avoid:

- Eda-Mame (soybeans in pods)
- Hydrolyzed soy protein
- Kinnoko flour
- Kyodofu (freeze-dried tofu)
- Miso; soy miso
- Modified food starch
- Natta
- Okara (soy pulp)
- Shoyu sauce
- Soy albumin
- Soy concentrate
- Soy flour; soybean flour

Wheat-Free Diet

Ingredients to avoid:

- All-purpose flour, enriched flour
- Bran
- Bread; bread crumbs
- Bulgur
- Cake flour
- Cereal extract
- Couscous
- Crackers; cracker meal
- Durum; durum flour; durum wheat
- Farina
- Flour, wheat, bran, graham
- Food starch
- Gluten; high-gluten flour
- Graham flour
- Malt; malt extract

- Nuts; flavored nuts, nutmeat, pieces
- Peanuts
- Peanut butter; peanut butter chips
- Peanut flour
- Peanut syrup
- Foods may have peanut protein presence: baked goods, candy (including chocolate, egg rolls, chili, enchilada sauce, flavoring, marzipan, nougat
- Soy milk; soybean milk
- Soy nuts
- Soy protein; soy protein isolate
- Soy sauce
- Soy sprouts
- Soybean granules
- Supro
- Tamari
- Tempeh
- Textured Vegetable Protein (TVP)
- Tofu
- Yakidofu
- Noodles
- Pasta
- Pastry flour
- Semolina
- Spelt
- Soy Sauce
- Starch
- Surumi
- Wheat; wheat bran; wheat flour
- Wheat germ
- Wheat gluten
- Wheat malt
- Wheat starch
- Whole-wheat berries

protein flour, non-dairy products

Shellfish-Free Diet

Ingredients to Avoid:

- Abalone
- Clams
- Crab
- Crawfish
- Lobster
- Mollusks
- Oysters

Tree Nut-Free Diet

Ingredients to Avoid:

- Almonds
- Artificial nuts
- Brazil nuts
- Caponata
- Cashews
- Filbert/hazelnuts
- Gianduja (nut mixture found in chocolate)
- Hickory nuts
- Nougat
- Natural nut extract (i.e, almond, walnut)

- Prawns
- Scallops
- Shrimp
- Foods that may indicate the presence of shellfish protein: fish stock, flavoring (including natural or artificial), seafood flavoring (such as crab or clam exgtract), surimi
- Nutmeal
- Nut oil
- Nut paste (i.e, almond paste)
- Nut pieces
- Pecans
- Pesto
 - Pine nuts (also known as Indian, pinon, pinton, pignoli, pignolia and pignon nuts
- Pralines
- Walnuts

Bibliography

- 1. Escott-Stump S. Nutrition and Diagnosis-Related Care. 5th ed. Baltimore, Md: Lippincott Williams & Wilkins; 2002:76.
- 2. Wilson SH. Medical nutrition therapy for food allergy and food intolerance. In: Mahan KL, Escott-Stump S, eds. *Krause's Food, Nutrition and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders; 2000. p 916-924.
- 3. American Academy of Allergy, Asthma, and Immunology. Position statement on anaphylaxis in school and other childcare settings. *J Allergy Clin Immunol.* 1998;102:173.
- 4. Nutrition management of food hypersensitivities. In: *Pediatric Manual of Clinical Dietetics*. 2nd ed. Chicago, Ill: American Dietetic Association; 2003.
- 5. Watson WT. Food allergy in children. Clin Rev Allergy Immunol. 1995;13:347-359.
- 6. Bischoff SC, Mayer J, Wedemeyer J, Meier PN, Zeck-Kapp G, Wedi B, Kapp A, Cetin Y, Gebel M, Manns MP. Colonoscopic allergen provocation (COLAP): a new diagnostic approach for gastrointestinal food allergy. *Gut.* 1997;40:745-753.
- 7. Plant M. New directions in food allergy research. J Allergy Clin Immunol. 1997;100:7-10.

II. NUTRITION ASSESSMENT/INTERVENTION

Body Weight Evaluation and Indicators of Nutrition-Related Problems	II-1
Table II-1: Weight Loss as an Indicator of Malnutrition	II-2
Table II-2: Body Mass as an Indicator of Lower Than Recommended Weight Levels	;II-2
Table II-3: Evaluation of Weight Loss in Adult Malnutrition (Undernutrition)	II-2
Table II-4: Indicators of the Nutrition Diagnosis of Malnutrition	II-2
Table II-5: ICD-9-CM Codes for Nutrition-Related Disorders	II-3
Stature Determination	II-5
Body Mass Index (BMI)	II-6
Determining Ideal Body Weight (IBW) Based on Height to Weight: Hamwi Method	II-7
Standard Body Weight (SBW) Determination Based on NHANES II	II-8
Determination of Frame Size	II-9
Estimation of Ideal Body Weight and Body Mass Index For Amputees	II-10
Estimation of Energy Expenditures	II-12
Estimation of Protein Requirements	II-17
Laboratory Indices of Nutritional Status	II-18
Protein Status	II-18
Vitamin Status	II-19
Hematologic Status	II-19
Classification of Some Anemias	II-20
Diagnostic Criteria for Diabetes Mellitus	II-21
Major Nutrients: Functions and Sources	II-23
Physical Signs of Nutritional Deficiencies	II-26
Food and Medication Interactions ^a	II-27
Herb and Medication Interactions	II-34

BODY WEIGHT EVALUATION and INDICATORS OF NUTRITION-RELATED PROBLEMS

Overview

Weight evaluation is part of the nutrition assessment. Weight should also be routinely evaluated to determine the outcome of nutrition interventions. If possible, a patient should be weighed on admission to any clinical setting and monitored frequently throughout the length of stay. For the most reliable assessment, the patient should be weighed on the same scale for each evaluation, preferably by using a beam scale with nondetachable weights. In addition, the patient should be weighed while fasting, after voiding, and without drainage bags and dressings. If drainage bags and dressings cannot be removed, weigh them separately and deduct their weight.

Body Weight as an Indicator of Nutrition-Related Problems

Body weight and changes in body weight are two of the most reliable indicators of declining or improving nutritional status. The three methods of evaluating body weight described by Blackburn are listed below (1-3). The "percent usual weight", "percent of recent weight change", and the body mass index (BMI) are strongly correlated with morbidity, mortality, and severity of illness in hospitalized patients and long-term care residents (1,2). Unintended weight loss is a well-validated indicator of nutritional compromise and can be used to diagnose nutrition-related problems, such as adult malnutrition (3). For the most complete evaluation of weight status, the dietitian should use the three assessment parameters described below in addition to the BMI level (3). For the evaluation of BMI height and weight should be measured rather than estimated (3). For the dehydrated or edematous patient, the measured weight must be intuitively increased or decreased, respectively, prior to evaluation (3).

The criteria listed in Tables II: 1-3 can be used in determining ICD-9-CM (International Classification of Diseases, 9th Revision, Clinical Modification) diagnostic codes when clinically indicated. It is important to clarify that existing ICD-9-CM codes may not be applicable to patients seen by acute and chronic care clinicians in developed countries (3). The Academy advises that these codes will likely change some of the definitions in the 10th revision cycle (3). In the interim, the Academy of Nutrition and Dietetics and A.S.P.E.N. recently published guidelines for using weight in addition to five other nutrition related indicators for the identification and documentation of adult malnutrition (undernutrition) (3). Because there is no single parameter that is definitive of adult malnutrition, identification of at least two or more of the six characteristics is recommended for diagnosis including, insufficient energy intake; weight loss; loss of muscle mass; loss of subcutaneous fat; localized or generalized fluid accumulation; and diminished functional status as measured by hand grip (3). Criteria specific to the evaluation of weight loss is outlined in Table II-3: Evaluation of Weight Loss in Adult Malnutrition (3). For the complete list of characteristics of adult malnutrition refer to Table II-3. In addition to these resources, the dietitian can refer to the Academy of Nutrition and Dietetics International Dietetics & Nutrition Terminology (IDNT) Reference Manual (4). The Academy cites BMI levels less than 18.5 kg/m² for adults and less than 23 kg/m² for older adults (>65 years of age) as indicators of lower than recommended weight levels when determining a nutrition-related diagnoses (4).

 Percent ideal body weight (1) = <u>Actual weight</u> × 100 Ideal weight See Determining Ideal Body Weight Based on Height to Weight: The Hamwi Method (page II-6).
 Percent usual body weight (1) = <u>Actual weight</u> × 100 Usual weight
 Percent recent weight change (1) = <u>(Usual weight – Actual weight)</u> × 100

Usual Weight

Body Weight Evaluation

Table II-1. Weight Loss as an indicator of Manutrition(1,2)			
Time	Significant Weight Loss (%)	Severe Weight Loss (%)	
1 week	1 to 2	> 2	
1 month	5	>5	
3 months	7.5	>7.5	
6 months	10	>10	

Table II-1: Weight Loss as an Indicator of Malnutrition(1,2)

Table II-2: Body Mass as an Indicator of Lower than Recommended Weight Levels (4)			
Lower than Recommended Body Mass Index			
Weight Levels	(kg/m^2)		
Adult	< 18.5		
Older Adults (> 65 years)	< 23		

Table II-3: Evaluation of Weight Loss in Adult Malnutrition (Undernutrition)(3)

Frame Size	Females	Males
	Allow 100 lb for first 5 ft of height	Allow 106 lb for first 5 ft of height
Madium	plus 5 lb for each additional inch.	plus 6 lb for each additional inch.
Medium	Subtract 2.5 lb for each inch less	Subtract 2.5 lb for each inch under
	than 5 ft.	5 ft.
Small	Subtract 10%	Subtract 10%
Large	Add 10%	Add 10%

Table II-4: Indicators^a of the Nutrition Diagnosis of Malnutrition(4)

Nutrition Diagnosis	Etiologies (Causes)	Nutrition Assessment Indicators
Malnutrition (NI-5.2) Inadequate intake of protein and/or energy over prolonged periods of time resulting in loss of fat stores and/or muscle wasting	 Physiological causes Lack of food or limited access to food Food and nutrition- related knowledge deficit concerning the amount of energy and protein required Psychological (eg, depression, eating disorders) 	Anthropometric:BMI < 18.5 kg/m² adults (< 23 kg/m² for adults 65 years and older)Inadequate maternal weight gainBMI < 5th percentile in children

Nutrition Diagnosis	Etiologies (Causes)	Nutrition Assessment Indicators
Inadequate protein-	Same as malnutrition	Biochemical:
energy intake (NI-		Normal albumin (in the setting of normal liver function
5.3)		despite decreased protein-energy intake)
Inadequate intake of		
protein and/or		Anthropometric:
energy compared to		Inadequate maternal weight gain
established reference		Weight loss of 7.5% during past 3 months, > 5% in one
standards or		month, 1 to 2 % in one week in adults, any weight loss
recommendations		or failure to gain weight in children.
based on		
physiological needs		Nutrition-focused physical finding:
of short or recent		Slow or delayed wound healing
duration		
		Food/nutrition and client history:
		Intake analysis less than estimated or measured energy
		requirements
		Restriction or omission of food groups such as dairy or
		meat groups (protein) or bread or milk group (energy)
		Nutrient malabsorption (eg, bariatric surgery)

^a Two or more indicators must be present to determine a nutrition diagnosis of malnutrition (3).

Adapted from: International Dietetics & Nutrition Terminology (IDNT) Reference Manual. 4th ed. Chicago, Ill: Academy of Nutrition and Dietetics; 2012.

ICD-9-CM	Diagnosis ^a	Criteria ^b
Code Number		
260.0	Kwashiorkor	Nutritional edema with dyspigmentation of skin and hair
261.0	Nutritional marasmus	Nutritional atrophy; severe, chronic calorie deficiency; severe malnutrition
262.0	Other severe protein-energy malnutrition	Nutritional edema without mention of dyspigmentation of skin and hair
263.0	Protein-energy malnutrition of moderate degree	Refer to Tables II: 1-3 for nutritional indicators of malnutrition.
263.1	Protein-energy malnutrition of mild degree	Refer to Tables II: 1-3 for nutritional indicators of malnutrition.
263.8	Other protein-calorie malnutrition	Criteria is not available. Refer to Tables II: 1-3 for nutritional indicators of malnutrition.
263.9	Unspecified protein-calorie malnutrition, calorie malnutrition	A disorder caused by a lack of proper nutrition or an inability to absorb nutrients from food An imbalanced nutritional status caused by insufficient intake of nutrients to meet normal physiological requirements
579.3	Other and unspecified postsurgical nonabsorption	Hypoglycemia or malnutrition following gastrointestinal surgery
783.2	Abnormal loss of weight and underweight	BMI <18.5 kg/m ² (<23 kg/m ² in older adults 65+ years)(5); also refer t Table II:1
783.21 and/or V85.0	Loss of weight	BMI <18.5 kg/m ² ; also refer to Table II: 1
783.22 and/or V85.0	Underweight	BMI <18.5 kg/m ²

^aMalnourished individuals may meet criteria for these billable ICD-9-CM codes.

^b The 10th revision (ICD-10) of the codes is scheduled for implementation in 2013 and will change some of the definitions as shown in the Table II-5. The Academy of Nutrition and Dietetics and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) have proposed new malnutrition codes to the National Center for Health and Vital Statistics for inclusion. At this time acute-phase hepatic

Body Weight Evaluation

proteins (albumin, prealbumin, transferrin, and retinol binding protein) should be interpreted with caution or evaluated simultaneously with indicators of inflammation (eg, C-reactive protein, white blood cell count, and blood glucose levels) in determining etiologically based diagnosis (3).

References

- 1. Blackburn GL, Bistrian BR, Maini BS, Schlamn HT, Smith MF. Nutritional and metabolic assessment of the hospitalized patient. J Parenter Enteral Nutr. 1977;1:11-22.
- 2. Splett PL, Roth-Yousey LL, Vogelzang JL. Medical nutrition therapy for the prevention and treatment of unintentional weight loss in residential healthcare facilities. *J Am Diet Assoc.* 2003;103:352-362.
- White J, Guenter P, Jenson G, Malone A, Schofield M; the Academy Malnutrition Work Group; the A.S.P.E.N. Malnutrition Task Force; and the A.S.P.E.N. Board of Directors. Consensus Statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Characteristics recommended for the identification and documentation of adult malnutrition (Undernutrition). J Acad Nutr Diet. 2012;112:730-738.
- 4. International Dietetics & Nutrition Terminology (IDNT) Reference Manual. 4th ed. Chicago, Ill: Academy of Nutrition and Dietetics; 2012.

Bibliography

American Medical Association. AMA ICD-9-CM 2007: Physician, International Classification of Diseases: Clinical Modification. 9th rev.ed. Chicago, IL: American Medical Association, 2006. Under review and revision in 2013.

STATURE DETERMINATION

Method 1: Height

Height should be taken with the subject in stocking feet and standing against a vertical measuring board. (For patients with severe curvature of the spine, other measurements of stature may be more accurate.)

Procedure: Have the subject stand erect with weight equally distributed on both feet and the heels together and touching the vertical board. Where possible the head, shoulder blades, buttocks, and heels should all touch the vertical board. Arms should be hanging free at the sides with palms facing the thighs. Subject should look straight ahead, take a deep breath, and hold position while the horizontal headboard is brought down firmly on top to the head. Measure to the nearest 0.1 cm.

Method 2: Arm Span

Measurement of arm span is roughly equal to height. The span measurement remains constant despite decreasing height with age and is an acceptable alternative method for establishing height.

Procedure: Position the subject with his or her feet against a flat surface, usually a wall. Fully extend the subject's upper extremities (including hands) at shoulder level with palms facing forward. Place a tape measure against the wall to measure the distance between the tip of one middle finger to the tip of the other middle finger (exclude fingernails). Arm span must be done supine between birth and three years of age.

Note: Measurement of arm span may be difficult in elderly persons due to an inability to adequately stretch out their arms, and chest measurements may be altered by lung disease or osteoporosis. Arm span may be used in the elderly to estimate maximum stature at maturity before occurrence of age-related bone loss.

Method 3: Knee Height

Knee height provides a method to measure stature of persons who cannot stand upright. Unlike overall height, knee height changes little with age. The measurement is highly correlated with stature.

Estimation of Stature From Knee Height			Factor*
White male	6 – 18 years	2.22 (Knee Height) + 40.54	<u>+</u> 8.42 cm
	18 – 60 years	1.88 (Knee Height) + 71.85	<u>+</u> 7.94 cm
	60 – 80 years	2.08 (Knee Height) + 59.01	<u>+</u> 15.68 cm
Black male	6 – 18 years	2.18 (Knee Height) + 39.60	<u>+</u> 9.16 cm
	18 – 60 years	1.79 (Knee Height) + 73.42	<u>+</u> 7.2 cm
	60 – 80 years	1.37 (Knee Height) + 95.79	<u>+</u> 16.8 cm
White female	6 – 18 years	2.15 (Knee Height) + 43.21	<u>+</u> 7.8 cm
	18 – 60 years	1.87 (Knee Height) + 70.25 – (0.06 age)	<u>+</u> 7.2 cm
	60 – 80 years	1.91 (Knee Height) + 75 – (0.17 age)	<u>+</u> 17.64 cm
Black female	6 – 18 years	2.02 (Knee Height) + 46.59	<u>+</u> 8.78 cm
	18 – 60 years	1.86 (Knee Height) + 68.10 – (0.06 age)	<u>+</u> 7.6 cm
	60 – 80 years	1.96 (Knee Height) + 58.72	<u>+</u> 16.5 cm

The following formulas are used to compute stature from knee height:

"The stature of an individual will have a 95% chance of falling within the boundaries represented by the formula with the appropriate correction factor.

Adapted from: Chumlea W, Guo S, Steinbaugh M. Prediction of stature from knee height for black and with adults and children with application to mobility-impaired or handicapped person. *J Am Diet Assoc.* 1994;94:1385-1388. From: Grant A, DeHoog S. *Nutrition Assessment Support and Management.* Seattle, Wash: DeHoog/Grant; 1999. Reprinted by permission.

Procedure: The knee length measurement is made with a sliding, broad-blade caliper similar to the apparatus used to measure the length of infants.

Bibliography

Grant A, DeHoog S. Nutritional Assessment Support and Management. 5th ed. Seattle, Wash: Grant/DeHoog; 1999.
BODY MASS INDEX (BMI)

										We	eight (b)										
Height	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205
5'0"	20	21	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40
5'1"	19	20	21	22	23	24	25	26	26	27	28	29	30	31	32	33	34	35	36	37	38	39
5'2"	18	19	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	36	37	37
5'3"	18	19	19	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	35	36
5'4"	17	18	19	20	21	21	22	23	24	25	26	27	27	28	29	30	31	32	33	33	34	35
5'5"	17	17	18	19	20	21	22	22	23	24	25	26	27	27	28	29	30	31	32	32	33	34
5'6"	16	17	18	19	19	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32	33
5'7"	16	16	17	18	19	20	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32
5'8"	15	16	17	17	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30	31
5'9"	15	16	16	17	18	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30
5'10"	14	15	16	17	17	18	19	19	20	21	22	22	23	24	24	25	26	27	27	28	29	29
5'11"	14	15	15	16	17	17	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	29
6'0"	14	14	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28
6'1"	13	14	15	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27
6'2"	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	22	23	24	24	25	26	26
6'3"	12	13	14	14	15	16	16	17	17	18	19	19	20	21	21	22	22	23	24	24	25	26
6'4"	12	13	13	14	15	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	24	25

The BMI score means the following:	Underweight	Below 18.5
	Normal	18.5 – 24.9
	Overweight	25.0 - 29.9
	Obesity	30.0 - 39.9
	Extreme obesity	<u>></u> 40

BMI levels less than 18.5 kg/m² for adults and less than 23 kg/m² for older adults (>65 years of age) are indicators of lower than recommended weight levels (1).

Source: National Heart, Lung, and Blood Institute Obesity Education Initiative. Expert Panel. Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults. Available at: http://www.nhlbi.nih.gov. Accessed November 8, 1999.

Reference

1. International Dietetics & Nutrition Terminology (IDNT) Reference Manual. 4th ed. Chicago, Ill: Academy of Nutrition and Dietetics; 2012.

DETERMINING IDEAL BODY WEIGHT (IBW) BASED ON HEIGHT TO WEIGHT: THE HAMWI METHOD

Frame Size	Females	Males
Medium	Allow 100 lb for first 5 ft of height plus 5 lb for each additional inch. Subtract 2.5 lb for each inch less than 5 ft.	Allow 106 lb for first 5 ft of height plus 6 lb for each additional inch. Subtract 2.5 lb for each inch under 5 ft.
Small	Subtract 10%	Subtract 10%
Large	Add 10%	Add 10%

Source: Nutrition and Your Health: Dietary Guidelines for Americans. 3rd ed. Washington, DC: US Depts of Agriculture and Health and Human Services; 1990. Home and Garden Bulletin No. 232.

Hamwi GJ. Changing dietary concepts. In: Danowski TS (ed). Diabetes Mellitus: Diagnosis and Treatment, Vol. 1. New York: American Diabetes Association, Inc; 1964:73-78.

The above method of calculating Ideal Body Weight (IBW) is also referred to as the 5 foot rule.



HEALTHY WEIGHT CHART

*Without Shoes

**Without Clothes

Source: Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2000.vailable at: http://www.ars.usda.gov/dgac/dgacguideexp.pdf. Accessed February 23, 2000.

STANDARD BODY WEIGHT (SBW) DETERMINATION BASED ON NHANES II

Men		25-54 Year	rs		54-74 Years				
Height (in)	Small	Medium	Large	Small	Medium	Large			
62	64	68	82	61	68	77*			
63	61	71	83	62	70	80			
64	66	71	84	63	71	77			
65	66	74	79	70	72	79			
66	67	75	84	68	74	80			
67	71	77	84	69	78	85			
68	71	78	86	70	78	83			
69	74	78	89	75	77	84			
70	75	81	87	76	80	87			
71	76	81	91	69	84	84			
72	74	84	91	76*	81	90			
73	79*	85	93	78*	88	88			
74	80*	88	92	77*	95	89			

Based on NHANES II weight table, 50th percentile, kg

Women		25-54 Years		54-74 Years			
Height (in)	Small	Medium	Large	Small	Medium	Large	
58	52	63	86	54	57	92	
59	53	66	78	55	62	78	
60	53	60	87	54	65	78	
61	54	61	81	56	64	79	
62	55	61	81	58	64	82	
63	55	62	83	58	65	80	
64	57	62	79	60	66	77	
65	60	63	81	60	67	80	
66	58	63	75	68	66	82	
67	59	65	80	61*	72	80	
68	62	67	76	61*	70	79	
69	63*	68	79	62*	72*	85*	
70	64	70	76	63*	73*	85*	

Note: The clinician's judgment should be used in assigning these weights. Some categories are based on a small sample size of patients or estimated* by linear regression equation.

Reference

Frisancho AR. New Standards of weight and body composition by frame size and height for assessment of nutritional status of adults and the elderly. *Am J Clin Nutr.* 1984; 40:808-819. McCann L (ed) *Pocket Guide to Nutrition Assessment of the Patient with Chronic Kidney Disease (3rd Ed)*. National Kidney Foundation Council on Renal Nutrition, 2002: 11.

DETERMINATION OF FRAME SIZE

Method 1	Wrist Measurement
----------	-------------------

Frame Size (*r* values) = Height (cm)

Wrist Circumference (cm)

<i>r</i> values			
Females	Males	Interpretatio	Method
		n	
>11.0	>10.4	Small frame	1. Measure individual height in centimeters (cm)
10.1 – 11	9.6 - 10.4	Medium frame	2. Measure the smallest part of the individual's wrist in centimeters.
<10.1	<9.6	Large frame	3. Divide the height by the wrist circumference to derive <i>r</i> value for frame size. Look at table to the left to interpret frame size of individual.

Method 2: Elbow Breadth (1,2)

Frame size is influenced by soft tissue and fat but elbow breadth is a good index of skeletal or frame size and is less affected by fat than wrist circumference. It is also closely associated with lean body mass. Elbow breadth is the distance between the epicondyles of the humerus and should be measured with either sliding or spreading calipers. To measure:

- 1. Extend one arm in front of the body and bend the forearm upward at a 90° angle. Keep the fingers straight and turn the inside of the wrist toward the body.
- 2. Place the thumb and index finger of the other hand on the two prominent bones (epicondyles of the humerus) on the right side of the elbow. For greatest accuracy, use sliding calipers. (Sliding calipers can be obtained from Lafayette Instrument, PO Box 5729, 3700 Sagamore Pkwy N, Lafayette, IN 47903; telephone: 800/428-7545; fax: 765-423-4111; e-mail: rehab@licmef.com.)
- 3. Place the blades of the sliding caliper (blades pointing up) or the tips of the spreading caliper on the epicondyles. Exert firm pressure to compress the soft tissues and record in the measurement to the nearest 0.1 cm.
- 4. Frisancho developed a frame index based on elbow, breadth, height, and age. "Frame Index 2" was derived using data from the National Health and Nutrition Examination Survey III (NHANES) and accounts for age-related changes to height and weight. Plug the value into the following formula:

Frame Index 2 = Elbow Breadth (mm) divided by Height (cm) × 100

5. Use the table below to identify frame size for age.

Frame Size Daseu on Stature and Age								
		<u>Men</u>		Women				
Age (yi)	Small	Medium	Large	Small	Medium	Large		
18 – 25	<38.4	38.4 - 41.6	>41.6	< 35.2	35.2 - 38.6	>38.6		
25 - 30	<38.6	38.6 - 41.8	>41.8	<35.7	35.7 - 38.7	>38.7		
30 - 35	<38.6	38.6 - 42.1	>42.1	<35.7	35.7 - 39.0	>39.0		
35 - 40	<39.1	39.1 - 42.4	>42.4	<36.2	36.2 - 39.8	>39.8		
40 - 45	<39.3	39.3 - 42.5	>42.5	<36.7	36.7 - 40.2	>40.2		
45 - 50	<39.6	39.6 - 43.0	>43.0	<36.7	37.2 - 40.7	>40.7		
50 - 55	<39.9	39.9 - 43.3	>43.3	<37.2	37.2 - 41.6	>41.6		
55 - 60	<40.2	40.2 - 43.8	>43.8	<37.8	37.8 - 41.9	>41.9		
60 - 65	<40.2	40.2 - 43.6	>43.6	<38.2	38.2 - 41.8	>41.8		
65 – 70	<40.2	40.2 - 43.6	>43.6	<38.2	38.2 - 41.8	>41.8		
70 – 75	<40.2	40.2 - 43.6	>43.6	<38.2	38.2 - 41.8	>41.8		

Frame Size Based on Stature and Age

Adapted from: Frisancho AR. *Anthropometric Standards for the Assessment of Growth and Nutritional Status*. Ann Arbor, Mich: University of Michigan Press; 1990. In: Grant A, DeHoog S. *Nutrition Assessment Support and Management*. 5th ed. Seattle, Wash: Grant/DeHoog; 1999. Reprinted by permission.

Bibliography

Frisancho R. New standards of body weight and composition by frame size and height for assessment of nutritional status of adults and elderly. *Am J Clin Nutr.* 1984;40:808

Grant A, DeHoog S. *Nutrition Assessment Support and Management*. 5th ed. Seattle, Wash: Grant/DeHoog; 1999. Grant J. *Handbook of Total Parenteral Nutrition*. 2nd ed. Philadelphia, Pa: WB Saunders Co; 1992:19.

ESTIMATION OF IDEAL BODY WEIGHT AND BODY MASS INDEX FOR AMPUTEES

In the case where a patient had an amputation, ideal body weight (IBW) cannot be compared to the standards for normal adults. Although body proportions vary from individual to individual, segmental weights can be used to prove an approximation of IBW.

Percent Total Body Weight by Individual Body Parts ⁽¹⁾



Source: Osterkamp, Linda. Current perspective on assessment of human body proportions of relevance to amputees. *J Am Diet Assoc.* 1995:95;215-218.

Determining Adjusted Body Weight for the Amputee

Using the IBW of the patient before the amputation, subtract the percentage of the body limb or part that was removed to obtain an adjusted IBW. For a method of determining IBW, see Section II: Determining Ideal Body Weight (IBW) Based on Height and Weight: The Hamwi Method.

Example: Determine the adjusted IBW for a woman 5'5" with a below-knee amputation of the right leg.

IBW (female 5'5")	125 lb
Right below the knee (calf 4.4% + foot 1.5% = (-5.9%)	7.5 lb
Adjusted IBW	117.5 lb

Formula for Body Mass Index (BMI) for Amputees (2)

Body mass index can be calculated for persons with amputees by using the following formula described by Himes (2). Total body weight (TBW) or referred to as "WtE" is calculated by taking the "WtO" or observed body weight which is the current measured body weight (after the amputation) and divide by one minus the total percentage of limb(s) removed. The percentage of limbs missing can be derived from the Table above.

Formula for BMI in Amputees: $WtE = WtO/(1-P)_{(2)}$

WtE is estimated total body weight (TBW); WtO is the observed body weight (measured weight after the amputation); and P is the percentage of the missing limb segment (s) which can be estimated based on the Table above.

Example: A below-the-knee amputation is 5.9% or 0.059 of TBW. Observed (measured) weight is 70 kg.

WtE = 70 kg /(1-0.059) = 70 kg/(0.941) = 74.4 kg

To obtain height for use in the BMI equation, use height prior to amputation, current height if it can be obtained, or derived from the arm span (for formula see, Section II: Stature Determination). The WtE or adjusted Total

Estimation of Ideal Body Weight and Body Mass Index for Amputees

Body Weight (TBW) derived from this formula can be then used in the BMI formula. A low body mass index has a predictor of mortality in a 15-month period after an amputation in the elderly population (3).

References

- 1. Osterkamp L. Current perspective on assessment of human body proportion of relevance to amputees. *J Am Diet Assoc.* 1995;95: 215-218.
- 2. Himes JH. New equation to estimate body mass index in amputees {letter] J Am Diet Assoc. 1995;95:646.
- 3. Coletta EM. Care of the elderly patient with lower extremity amputation. J Am Board Fam Pract. 2000;13:23-34.

ESTIMATION OF ENERGY EXPENDITURES

Discussion

Since the early 1900s, various formulas have been employed to estimate energy expenditure. Since the advent of the doubly labeled water (DLW) technique in the 1980s, scientists have begun to more accurately determine total energy expenditure (TEE) in free-living persons (1). Unfortunately, due to its high cost and the limited number of laboratories that perform the DLW technique, this application is not currently accessible in the clinical setting. Most recently, the Academy of Nutrition and Dietetics (AND) explored evidence that reported the accuracy and application of various methods used to measure energy expenditure, particularly indirect calorimetry and predictive formulas for various population groups (2,3). These reports provide evidence that can be used by dietetic professionals to make informed clinical decisions regarding whether to measure or estimate resting metabolic rate (RMR), also known as resting energy expenditure (REE) in patients (2). The predictive equations that have been evaluated include: the Harris-Benedict equation (4), Mifflin–St. Jeor equation (MSJE) (5), Owen equations (6,7), and equations used by the World Health Organization and the Dietary Reference Intakes (8). In 2012, the Swinamer equation, Ireton-Jones equations, Penn State equations, MSJE, Brandi equation, and Faisey equation were evaluated for their application in estimating energy expenditure in critically ill patients (3). The equations found to most reliable and validated by the Academy Evidence Analysis Library are addressed in this publication (2,3).

The Dietary Reference Intakes for energy, which are based on studies using the DLW technique, are considered the most accurate references for estimating TEE in free-living persons (2.9). These values can serve as a resource for the assessment of patients who are not critically ill or do not have multiple disease processes (2.8), (Refer to Section A: Estimated Energy Requirements (EER) for Male and Female Under 30 Years of Age.) The Mifflin-St. Jeor equation predicts RMR with the most consistency and the least percentage of error in the ambulatory population (2). Multiple studies have reported variable accuracy with the Harris-Benedict equation; this equation accurately predicts RMR only 45% to 81% of the time in healthy non-obese subjects (2). The accuracy of all predictive equations decreases when applied to the obese population. In studies of obese patients, the Harris-Benedict equation accurately predicted RMR only 33% to 64% of the time, while the Mifflin-St. Jeor equation accurately predicted RMR 70% of the time (2). Because of the variations reported with the use of the Harris-Benedict formula, evidence provides limited support for its use in estimating the energy expenditure of ambulatory or hospitalized critically ill population groups, unless otherwise specified in this section (2, 3, 9). Energy expenditure depends on factors including age, gender, height, weight, and physical activity. In the hospital setting where patients generally have multiple complications and the potential for rapid changes in medical status, predictive formulas that include not only determinants of RMR, but also modifiers for illness severity, inflammatory state, and respiratory demands may be needed (3,9,10). The clinician should realize that any method used to estimate energy expenditure only provides an approximation (2). These equations should be used only as a guide or starting point, after which the patient must be closely monitored and interventions must be devised based on individual needs that promote the attainment of nutritional status.

Recommended Formulas to Calculate RMR in Critical Care Patients

Indirect calorimetry is the standard for determination of RMR in critically ill patients because RMR based on measurement is more accurate than estimation using predictive equations (Grade I)* (3). If predictive equations are needed in non-obese adult and older adult critically ill patients, the best prediction accuracy of equations studied (listed in order of accuracy) include the Penn State equation (PSU 2003b version) (69% < 60 years old, 77% > years old), Brandi (61% <60 years old, 61% > 60 years old), MSJE x 1.25 (54% < 60 years old, 54% > 60 years old), and Faisey equation (65% <60 years old, 37% > 60 years old) (Grade II) (3,9,11). The Harris-Benedict equation (with or without activity and stress factors), Ireton-Jones equations, and Fick equation should not be used to determine RMR in critically ill patients, as these equations do not have adequate prediction accuracy (Grade I) (3). If predictive equations are needed for critically ill individuals who are obese and < 60 years the evidence recommends Penn State equation (PSU 2003b) worked best and predicted RMR with 70% accuracy (Grade II) (3). In obese critically ill patients > 60 years old, a modified Penn State equation [PSU (2010)] predicted RMR with 74% accuracy (Grade II) (3). All other predictive equations tested had lower accuracy rates (3). Refer to the *Critical Illness Evidence-Based Nutrition Practice Guideline* (2012) in the ADA Evidence Analysis Library for detailed information (3).

The formulas described below are considered the most reliable and valid formulas used for patients in the critical care and hospital setting (2,3,9,10). These equations have easily measured variables (height, weight, age, gender, diagnosis, presence of obesity, and ventilatory status) that are used in the equation to estimate RMR. . The variables in these some of the equations take into account the health,mobility status, and ventilator status of a critical care patient. The common practice of multiplying additional physical activity level (PAL) factors or injury factors is not validated with these formulas unless specified (3,10). All equations described below were developed using actual weight (3). At this time there is no evidence that substituting adjusted or ideal weight in these calculations results in improved accuracy (2, 3, 9).

Penn State equations (3):

The latest version referred to as Penn State Equation (PSU 2003b) was validated in 2009 $_{(Grade II) (3)}$. This version of the formula is currently recommended for critically patients of any age with BMI below 30, or for patients who are younger than 60 years with BMI over 30 $_{(Grade II) (3)}$. The Penn State equation originated in 1998 and was modified again in 2003. The PSU 1998 version used the Harris-Benedict equation and was replaced with the Mifflin St Jeor equation using actual weight in 2003 to improve accuracy $_{(3, 10)}$. The equation was further revised to improve performance for the older adult obese population $_{(10)}$. The 1998 and 2003a versions should no longer be used and were invalidated in 2007 and 2009 by the Academy Evidence Analysis Library $_{(3)}$:

PSU 2003 b: RMR = Mifflin^a (0.96) + V_E (31) + T_{max} (167) - 6212

^aMifflin = resting energy expenditure calculated using the Mifflin St. Jeor Equation (5)

V_E = minute ventilation (L/min)

T_{max} = maximum temperature (degrees Celsius)

Modified Penn State Equation, also known as PSU 2010 was validated in 2010 by the Academy $_{(Grade II)}$ (3). This formula is recommended to be used for patients with BMI over 30 and older than 60 years of age (3).

PSU 2010: RMR = Mifflin^a (0.71) + V_E (64) + T_{max} (85) - 3085

^aMifflin = resting energy expenditure calculated using the Mifflin St. Jeor Equation (5) V_E = minute ventilation (L/min) T_{max} = maximum temperature (degrees Celsius)

Brandi Equation (3, 12)

Upon evaluation by the Academy Evidence-Based Library, the Brandi equation was shown to be unbiased toward non-obese patients with an accuracy of 61% (Grade III) (3). It was biased for obese patients with accuracy of 48% (Grade III) (3, 12).

HBE (0.96) + HR (7) + Ve (48)-702

HBE = Harris Benedict Equation V_E = Expired minute ventilation

Harris-Benedict equation (4) for use in Brandi equation or otherwise specified: kcal/day (male) = 66 +13.8 (W) + 5.0(H) – (6.8 x A) kcal/day (female) = 655 + 9.6 (W) + 1.8 (H) – (4.7 x A), where W = actual weight (kg) H = height (cm) A = age (years)

Faisey Equation (3,12, 13)

In the two studies evaluating the Faisey equation it was shown the equation was unbiased but inaccurate (Grade III) (3). The equation predicted RMR +/- in 53% of subjects (Grade III) (3).

W (8) + H (14) + V_E (32) + T (94) -4,834

Equation uses weight (W) in kilograms and height (H) in centimeters. V_E is expired minute ventilation and T is body temperature in degrees centigrade (3).

Swinamer Equation (3, 14)

Published in 1990, was based on observations in 112 mechanically ventilated, critically ill trauma, surgical, and medical patients within the first 2-days of admission to critical care unit $_{(14)}$. Studies comparing RMR and Swinamer equation have demonstrated accuracy rates ranging from 45% to 61% with a trend toward overprediction $_{(Grade I)}$ (3, 12-15).

Energy Expenditure (EE) = 945 (BSA) - 6.4 (age) +108 (T) + 24.2 (breaths/min) + 81.7 (VT) -4349

Equation includes body surface area (BSA) in squared meters (m2), age (A), temperature (T) in degrees Celsius, breaths per minute (BPM), and and tidal volume (VT) in liters per minute (L/min).

Mifflin-St. Jeor Equation and Recommended Formulas to Calculate RMR in Ambulatory Patients

The Mifflin–St. Jeor equation (MSJE) was designed to estimate RMR in the ambulatory population (5, 10). Actual body weight is used in these equations, regardless of BMI (5). It is generally recommended that a PAL factor be multiplied to obtain TEE. It has been recommended that the RMR be multiplied by a PAL factor of 1.3 for sedentary individuals; however, this recommendation has not been validated in studies (9). If needed, use a higher activity factor to correct for active individuals engaging in purposeful activity (9). See the following table as a guideline, or refer to PAL described in Dietary Reference Intakes in Section A to determine appropriate PAL estimate (16). Injury factors have not been validated for use with these equations and therefore are not recommended as part of TEE calculations $_{(3,10)}$. The Academy's practice guidelines for Critical Illness recently reviewed the usability of the MSJE in the critical care setting. The evaluation showed the Mifflin St. Jeor equation is applicable to non-obese adults and older adults when estimating resting metabolic rate in the critical care setting (3). When RMR was determined by MSJE and multiplied by 1.25 the equation accurately predicted energy requirements in 54% of the subjects (Grade II) (3).

The Mifflin-St. Jeor equation for men is:

RMR = 10(weight^a in kg) + 6.25(height in cm) – 5(age in years) + 5

The corresponding equation for women is: $RMR = 10(weight^a in kg) + 6.25(height in cm) - 5(age in years) - 161$

^aUse total body weight, regardless of BMI.

PAL Factor
1.2
1.3
1.6-1.7
1.8-1.9
2.0-2.4

Estimating Energy Expenditure in the Obese Population

Ideally, the RMR of an obese patient should be based on lean body mass that is determined by methods such as dual x-ray absorptiometry, underwater weighing, or gold-standard energy expenditure prediction models (eg, DLW technique) (9). However, these methods are not practical in most clinical settings. The common clinical practice of using an adjusted body weight to estimate the metabolically active tissue mass does not improve the accuracy of predicting the metabolic rate in obese patients (3,10). The Adjusted Body Weight for Obesity formula, a well-known formula developed by Cunningham (17), is not considered applicable in current clinical practice and therefore should not be used in any predictive equations (5,9,10,18). The consensus of literature supports the use of actual body weight in predictive formulas for estimating RMR in obese patients (18,19). Formulas like the Mifflin–St. Jeor, and if the patient is critically ill, Penn State (PSU 2003b and PSU 2010 version) equations have utilized obese subjects in the validation of the equations and therefore are an option for predicting RMR in obese patients (3,5,10). According to the ADA's Weight Management Practice Guidelines, estimated energy requirements should be based on resting metabolic rate (RMR) (20). If possible, RMR should be measured (eg, indirect calorimetry). If RMR cannot be measured, then the Mifflin–St. Jeor

equation using actual body weight is the most accurate formula for estimating RMR for overweight and obese healthy individuals (Grade I) (20).

Estimating Energy Requirements for Spinal Cord Injury

People with spinal cord injury tend to have reduced metabolic activity due to denervated muscle. Measured energy expenditure is at least 10% below predicted; therefore, caloric needs of spinal cord injured patients should be based on measured energy expenditure (Grade III) (21). If indirect calorimetry is not available during the acute phase (0 - 4 weeks post-injury using prediction equations based on critical care level using admission weight in the Harris Benedict formula and multiplying by an activity factor of 1.1 and an injury/stress factor of 1.2 is suggested (Grade III) (21). During the rehabilitation phase, one study reports initial caloric needs can be estimated using 22.7 kcal/kg body weight for individuals with tetraplegia and 27.9 kcal/kg for those with paraplegia (21). When estimating caloric needs of individuals with spinal cord injury, acuteness of injury, level of injury, gender, and physical activity level should be taken into consideration (Grade III) (21).

Measuring RMR by Indirect Calorimetry

Indirect calorimetry is an indirect measurement of REE based on quantification of an individual's respiratory gas exchange (ratio of oxygen consumed to carbon dioxide produced). From respiratory gas exchange measurements, a respiratory quotient can be obtained that can provide additional information about individual substrate utilization (10). Many stress factors and kilocalorie ranges proposed for estimating energy expenditure for specific disease states are based on indirect calorimetry studies; however, the accuracy of these formulas for estimating expenditure for the individual patient can vary (9,10). Factors that affect energy expenditure and impact the outcome of indirect calorimetry results include: changes in medications that act as a stimulant or a sedative, changes in the degree or type of ventilator support, and day-to-day variations in the metabolic stress level (10). These factors should be considered when monitoring and interpreting measured REE. Indirect calorimetry remains a viable option for estimating energy requirements in the critical care setting and can be useful in the prevention of overfeeding the critical care patient. Precise guidelines and more in-depth considerations for the use of indirect calorimetry have been published (2,10).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Black AE, Coward WA, Cole TJ, Prentice AM. Human energy expenditure in affluent societies: an analysis of 574 doubly-labelled water measurements. *Eur J Clin Nutr.* 1996;50:72-92.
- 2. Energy Expenditure Evidence Analysis Project. Academy of Nutrition and Dietteics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2006. Available at: http://www.andevidencelibrary.org. Accessed January 20, 2013.
- 3. Critical Illness Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: http://www.andevidencelibrary.com. Accessed January 16, 2013.
- 4. Harris J, Benedict F. *A Biometric Study of Basal Metabolism in Man.* Washington, DC: Carnegie Institute of Washington; 1919. Publication No. 279.
- 5. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. *Am J Clin Nutr.* 1990;51:241-247.
- 6. Owen OE, Holup JL, D'Alessio DA, Craig ES, Polansky M, Smalley KJ, Kavle EC, Bushman MC, Owen LR, Mozzoli MA. A reappraisal of the caloric requirements of men. *Am J Clin Nutr*. 1987;46:875-885.
- 7. Owen OE, Kavle E, Owen RS, Polansky M, Caprio S, Mozzoli MA, Kendrick ZV, Bushman MC, Boden G. A reappraisal of caloric requirements in healthy women. *Am J Clin Nutr.* 1986;44:1-19.
- Institute of Medicine's Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. National Academy of Sciences; 2002:265-334. Preprint available at: http://www.nap.edu.books/0309085373/html/index.html. Accessed April 20, 2005.
- 9. Calculations for nutrition assessment. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: http://www.nutritioncaremanual.org. Accessed January 30, 2013.
- 10. Wooley JA, Frakenfield DC. Energy. In: . In: Mueller CM ed. *The A. S. P. E. N. Adult Nutrition Support Core Curiculum.* 2nd ed.. Silver Spring, MD: American Society of Enteral and Parenteral Nutrition; 2012:22-35.
- 11. Frankenfield D, Hise M, Malone A, Russell M, Gradwell E, Compher C, For the Evidence Anlaysis Work Group. Prediction of resting metabolic rate in critically ill adult patients: Results of a systematic review of the evidence. *J Am Diet Assoc.* 2007;107:1552-1561.
- 12. Frankenfield DC, Coleman A, Alam S, Cooney R. Analysis of estimation methods for resting metabolic rate in critically ill adults. J Parenter Enteral Nutr. 2009;33;27.
- 13. Savard JF. Faisy C, Lerolle N, Guerot E, Diehl JL, Fagon JY. Validation of a predictive method for an accurate assessment of resting energy expenditure in mechanically ventilated patients. *Critical Care Medicine*. 2008;36:1,175-1,183.
- 14. Swinamer DL, Grace MG, Hamilton SM, Jones R, Roberts P, King EG. Predictive equation for assessing energy expenditure in mechanically ventilated critically ill patients. *Crit Care Med.* 1990;18:657-661.
- 15. Anderegg BA, Worrall C, Barbour E, SimpsonKN, Delegge M. Comparison of resting energy expenditure prediction methods with measure resting energy expenditure in obese, hospitalized adults. J Parenter Enterl Nutr. 2009; 33:168-175.

Estimation of Energy Expenditures

- 16. Shetty PS, Henry CJ, Black AE, Prentice AM. Energy requirements of adults: an update on basal metabolic rates (BMRs) and physical activity levels (PALs). *Eur J Clin Nutr.* 1996;50(suppl 1):S11.
- 17. Cunningham JJ. An individualization of dietary requirements for energy in adults. J Am Diet Assoc. 1982;80:335-338.
- 18. Frankenfield DC, Muth ER, Rowe WA. The Harris-Benedict studies of human basal metabolism: history and limitations. J Am Diet Assoc. 1998;98:439-445.
- 19. Ireton-Jones CS, Turner WW Jr. Actual or ideal body weight; which should be used to predict energy expenditure? *J Am Diet Assoc.* 1991;91:193-195.
- 20. Adult Weight Management Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2006. Available at: http://www.andevidencelibrary.com. Accessed January 30, 2013.
- 21. Spinal Cord Injury Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2009. Available at: http://www.andevidencelibrary.com. Accessed January 30, 2013.

22.

ESTIMATION OF PROTEIN REQUIREMENTS

The following methods can be used to estimate protein requirements based on life stage. Use of actual body weight or when weight cannot be obtained, ideal body weight (IBW), is suggested for all equations because protein requirements relate to lean body mass. In the underweight, malnourished patient, use of actual body weight has been suggested in equations using anabolic protein levels in order to avoid the consequences of overfeeding in these patients. A nitrogen balance test may be employed to evaluate adequacy of protein intake in either obese or undernourished patients. Refer to Section III: Burns for information on the nitrogen balance test.

Adult Maintenance: Recommended Dietary Allowances (RDA): 0.8 to 1.0 g/kg ideal body weight (1)

Older Adults Maintenance: Emerging evidence recommends protein be increased to 1.0 to 1.25 g/kg daily (2-5) or 12% to 14% of total energy intake for the elderly.

Adult Critical Illness Normal Weight and Obesity

Critical illness and the stress response to illness and trauma is associated with increased protein turnover, protein catabolism, and negative nitrogen balance (6). Protein requirements double in critical illness to approximately 15% to 20% of total calories (6). In critically ill patients with a body mass index (BMI) < 30, protein requirements should be in the range of 1.2 to 2.0 g/kg actual body weight per day. Protein requirements may be even higher in burn or multi-trauma patients (3, 6). 2009 ASPEN guidelines suggest even higher ranges for the obese critically care ICU adult patient. The guidelines recommend protein in a range of > 2.0 g/kg ideal body weight per day for Class I and II obese patients (BMI 30 to 40), > 2.5 g/kg ideal body weight per day for Class III obese patients (BMI > 40) (6). The best nutrition assessment indicator to determine adequacy of protein intake in critical illness is nitrogen balance evaluation (6). Refer to Section III: Burns for detailed overview for when nitrogen balance may be warranted.

Spinal Cord Injury: The acute phase of spinal cord injury results in an obligatory negative nitrogen balance that may persist for 7 weeks or more, as nitrogen excretion increases with changes in body weight and loss of lean body mass (7). Efforts to achieve positive nitrogen balance with aggressive nutrition support are generally unsuccessful and may result in overfeeding (7). Although a protein intake of 2.4 grams/kg IBW/day may lessen the negative nitrogen balance, 2 g protein/kg IBW/day may be more appropriate given potential concerns of substrate overload (Grade III)* (7). Acute phase hypoalbuminemia may not be indicative of malnutrition, but a rising albumin level within 3 weeks of injury generally indicates adequate nutritional intake (7). For a person with spinal cord injury, 0.8 - 1.0 g protein/kg body weight/day may be required for maintenance, with an increase to 1.0 - 1.5 g protein/kg body weight/day if pressure ulcers or infection are present (Grade III)(7).

Refer to Criteria and Dietary Reference Intake Values for Protein by Life Stage Group in Section 1A or Section III for disease-specific information.

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- Institute of Medicine's Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. National Academy of Sciences, 2002: 265-334; preprint at http://www.nap.edu.books/0309085373/html/index.html. Accessed September 16, 2002.
- 2. Institute of Medicine's Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. (Macronutrients). Washington, DC: National Academy of Sciences, 2005: 107-180.
- 3. Campbell WW, Crim MC, Dallal GE, Young VR, Evans WJ. Increased protein requirements in elderly people: new data and retrospective reassessments. Am J Clin Nutr. 1994;60:501-509.
- Harris NG. Nutrition in aging. In: Mahan LK, Escott-Stump S. Krause's Food, Nutrition, and Diet Therapy. 10th ed. Philadelphia, Pa: 4. WB Saunders Co; 2000:294.
- Evans WJ, Cyr-Campbell D. Nutrition, exercise, and healthy aging. J Am Diet Assoc. 1997;97:632-638. 5.
- McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; A.S.P.E.N Board of 6. Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A. S. P. E. N.). JPEN J Parenter Enteral Nutr. 2009;33:277-316.
- 7. Spinal Cord Injury Evidence Analysis Project. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2007. Available at: http://www.andevidencelibrary.com. Accessed January 29, 2013.

LABORATORY INDICES OF NUTRITIONAL STATUS

Laboratory values can be useful in assessing nutritional status or identifying those at high risk that may require nutrition intervention. However, caution is necessary when interpreting laboratory values, and results from single laboratory values should be interpreted carefully. The laboratory tests listed below are commonly used to evaluate either a direct or indirect relationship to a patient's nutritional status. The negative acute-phase hepatic proteins albumin, pre-albumin, transferrin and retinol-binding protein are now considered better indicators of inflammatory metabolism, morbidity, mortality and severity of illness than nutritional status (1-4). These proteins can decrease by as much as 25% as a result of inflammatory metabolism caused by acute or chronic disease (1). The Academy of Nutrition and Dietetics evidence analysis indicates that these acute-phase proteins do not consistently or predictably change with weight loss, calorie restriction, or nitrogen balance (5). Considering this emerging evidence, the Academy and A.S.P.E.N. do not recommend the evaluation of acute phase proteins for use in the identification and documentation of adult malnutrition (Undernutrition) (5).

Test	Purpose/Definition	Normal Range	Discussion
Protein Statu	S		
Albumin	Indicator of inflammatory metabolism, morbidity, mortality, or severity of illness (1-4)	3.5 – 5.0 g/dL	Should not be used as an indicator of nutritional status Use as an indicator of inflammatory metabolism, morbidity, mortality, or severity of illness (1-4) Elevated levels occur in dehydration. Low in uncomplicated malnutrition (without existing acute or chronic disease) (1)
Pre-albumin	Indicator of inflammatory metabolism, morbidity, mortality, or severity of illness (1-4)	19 - 43 mg/dL	Should not be used as an indicator of nutritional status Use as an indicator of inflammatory metabolism, morbidity, mortality, or severity of illness (1-4) More sensitive to dietary change than albumin post fasting, (4). Low in uncomplicated malnutrition (without existing acute or chronic disease) (1)
Protein, total	Total protein is of little value as a sensitive index for estimating protein nutritional status	Serum value 6.4 – 8.3 g/dL	Decreased values occur with: nephrosis severe burns malnutrition overhydration hepatic insufficiency Increased values occur with: multiple myeloma dehydration
Transferrin	Indicator of inflammatory metabolism, morbidity, mortality, or severity of illness (1-4)	200 – 400 mg/dL	Should not be used as an indicator of nutritional status Use as an indicator of inflammatory metabolism, morbidity, mortality, or severity of illness (1-4) Decreases with anemia and protein-energy malnutrition (uncomplicated by acute or chronic disease) Increases with iron deficiency, infection, oral contraceptives, and pregnancy
Urea nitrogen	Urea is the principal end product of protein catabolism	10 – 20 mg/dL Values may be slightly higher in the elderly	Decreased values occur with: liver impairment decreased protein intake overhydration malabsorption high-carbohydrate, low-protein diets Increased values occur with: renal insufficiency GI bleeding dehydration lower urinary tract infection diabetes mellitus obstruction starvation congestive heart failure excessive protein intake or protein catabolism

Test	Purpose/Definition	Normal Range	Discussion
Vitamin Status			
25- Hydroxyvitamin D	Vitamin D status	30 to 70 ng/mL ⁶	Decreased values occur with: Hypocalcemia Osteomalacia Osteoporosis Increased values occur with: Hypercalcemia, hypercalcuria, soft tissue calcification
Hematologic St	atus		
Red blood cells (RBCs)	Measures the number of RBCs in whole blood	M: 4.5 - 6.0 million/mm ³ F: 4.0 - 5.5 million/mm ³	Decreased values occur with: anemia chronic infection leukemia Increased values occur with: dehydration
Hemoglobin (Hgb)	Part of the red blood cells that carries oxygen and carbon dioxide in the blood	M: 13 – 18 g/100 dL F: 12 – 16 g/100 dL	Men's Hgb may drop $1 - 2 g/100 mL$ with age. Women have no documented change. Although Hgb declines with age, other signs should be reviewed, eg, pale skin, pale conjunctiva.
Hematocrit (HCT)	Measures the percent of RBCs in the total blood volume	M: 42% – 52% F: 37% – 47%	Values may decrease slightly in the elderly
Mean corpuscular hemoglobin concentration (MCHC)	Measures the concentration of Hgb per unit of red blood cells	32% - 36%	Values <30 indicate advanced iron deficiency anemia
Mean corpuscular volume (MCV)	Measures the average size of the RBC	80 – 95 mm ³	Increased values indicate pernicious anemia. Decreased values indicate iron deficient anemia.
Ferritin	Provides an index of iron stores in iron deficiency and iron overload	M: 12 – 300 μg/L F: 10 – 150 μg/L	Significantly higher in mend and post menopausal women. Decreased values occur with iron or protein deletion. Increased values occur with iron excess.
Prothrombin time (PT)	Measures velocity of blood clotting and is an indirect measure of vitamin K status	Adults: 11 – 12.5 seconds 85% – 100% of control	Increased values occur with: vitamin K deficiency (common in elderly and hospitalized) liver disease fat malabsorption medication therapy (antibodies, anticoagulants, aspirin) PT >25 seconds is associated with major bleeding

References

- 1. Fuhrman MP, Charney P, Mueller CM. Hepatic proteins and nutrition assessment. J Am Diet Assoc. 2004;104:1258-1264.
- 2. Gabay C, Kushner I. Acute phase proteins and other systemic responses to inflammation. N Eng J Med. 1999; 340:448-454.
- 3. Mueller C. True or false: serum hepatic protein concentrations measure nutritional status. Support Line. 2004;26:8-16.
- 4. International Dietetics & Nutrition Terminology (IDNT) Reference Manual. 4th ed. Chicago, Ill: Academy of Nutrition and Dietetics; 2012.
- White J, Guenter P, Jenson G, Malone A, Schofield M; the Academy Malnutrition Work Group; the A.S.P.E.N. Malnutrition Task Force; and the A.S.P.E.N. Board of Directors. Consensus Statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Characteristics recommended for the identification and documentation of adult malnutrition (Undernutrition). J Acad Nutr Diet. 2012;112:730-738.

6. Javorsky B, Maybee N, Padia SH, Dalkin AC. Vitamin D deficiency in gastrointestinal disease. *Practical Gastroenterology*. March 2006;52-72.

CLASSIFICATION OF SOME ANEMIAS

Test	B12 Deficiency	Folate Deficiency	Iron Deficiency	Anemia of Chronic Disease
RBC count	D	D	D	D
Hemoglobin	D	D	D	Slight D
Hematocrit	D	D	D	D
MCV	Ι	Ι	D	N
МСН	Ι	Ι	D	N
MCHC	Ν	Ν	D	Ν
Reticulocyte count	N or D	N or D	N or D	N or D
RDW	N or I	N or I	Ι	N
Serum ferritin	Ι	Ι	D	N or I
TIBC	Ν	Ν	N or I	N or D
Transferrin	Ν	Ν	N or I	N or D
Transferrin	Ν	Ν	D	N or D
saturation (%)				
Serum iron	Ν	N	D	D
Serum folate	N or I	D	Ν	Ν
Red cell folate	D	D	Ν	Ν
Vitamin B12	D	Ν	Ν	Ν
Red blood cells	Normochromic,	Normochromic,	Hypochromic,	Hypochromic, microcytic
	macrocytic	macrocytic	microcytic	(both mild)
Other	Hypersegmented	Hypersegmented	Anisocytosis	Poikilocytosis (slight),
	neutrophils,	neutrophils,		anisocytosis (moderate)
	macro-ovalocytes	macro-ovalocytes		

I = increased; N = normal; D = decreased; TIBC = total iron-binding capacity

Source: Grant A, DeHoog S. Nutrition Assessment Support and Management. 5th ed. Seattle, Wash: Grant/DeHoog; 1999:183. Reprinted by permission.

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

The diagnostic criteria for diabetes are issued by the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (1). In 2009, the Expert Committee on Diagnosis and Classification of Diabetes Mellitus approved the use of the A1C test for diagnosing diabetes mellitus after extensive review of the literature and improved standardization of the assay (1). This system of classification of diabetes is based on the cause of the disease, as opposed to the therapy used to treat the hyperglycemia.

Diagnosis of Diabetes

The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus advocates use of the following laboratory criteria for nonpregnant adults (1):

- 1. Symptoms of diabetes plus casual plasma glucose concentration greater than or equal to 200 mg/dL (11.1 mmol/L). Casual is defined as any time of the day without regard to the time since the last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.
- 2. Fasting plasma glucose (FPG) concentration greater than or equal to 126 mg/dL (7.0 mmol/L). Fasting is defined as no energy intake for at least 8 hours.
- 3. Plasma glucose concentration 2 hours after glucose ingestion greater than or equal to 200 mg/dL during an oral glucose tolerance test (OGTT). The test should be performed, as described by the World Health Organization (WHO), using a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water.
- 4. A1C test greater than or equal to 6.5%. Test should be performed in a laboratory using a method that is National Glycohemoglobin Standardization Program (NGSP) certified and standardized to the Diabetes Control and Complications Trial (DCCT) assay.

In the absence of unequivocal hyperglycemia with acute metabolic decompensation, these criteria should be confirmed by repeated testing on a different day. The OGTT is not recommended for routine clinical use (1).

Impaired Fasting Glucose (IFG) and Impaired Glucose Tolerance (IGT)

The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus endorses the following criteria for the diagnosis of diabetes mellitus and gestational diabetes (1). The Expert Committee recognizes an intermediate group of patients whose glucose levels, although do not meet the criteria for diabetes, are too high to be considered normal. This group is now referred to as "pre-diabetes" indicating the relatively high risk for development of diabetes in these patients (1):

	"Normal"	"Pre-diabetes"	"Diabetes"
Fasting plasma glucose	<u><</u> 100 mg/dL	Impaired Fasting Glucose (IFG) ≥100 and <125 mg/dL (IFG)	<u>></u> 126 mg/dL
Glucose tolerance, at 2 hours after glucose load (during OGTT)	<140 mg/dL	Impaired Glucose Tolerance (IGT) ≥140 to 199 mg/dL (IGT)	>200 mg/dL
A1C Test	4.5 to 5.5 %	5.7 % to 6.4%	<u>≥</u> 6.5 %

Based on the Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study, the International Association of Diabetes and Pregnancy Study Group (IADPSG) has modified diagnostic criteria for gestational diabetes (1-3). These new criteria will significantly increase the prevalence of GDM, primarily because only one abnormal value, not two is sufficient to make the diagnosis (1).

Screening and Diagnosis of Gestational Diabetes Mellitus (GDM) (1-3)

Plasma Glucose	75-g Diagnostic Test* (mg/dL)
Fasting	<u>></u> 92
1 h postprandial	<u>></u> 180
2 h postprandial -	<u>≥</u> 153

Note: High risk women (e.g., marked obesity, history of GDM, glycosuria, or strong family history of diabetes) should undergo glucose testing during the initial prenatal care visit for a diagnosis of overt diabetes. All women not known to have diabetes should undergo a 75-g OGTT at 24 to 28 weeks of gestation.

"The 75-g oral-glucose tolerance test should be performed on pregnant women not known to have diabetes at 24 to 28 weeks of gestation. The diagnosis of GDM requires any one of the three plasma glucose values obtained during the test to meet or exceed the values shown above. The test should be done in the morning after an overnight fast of between 8 hours.

References

- 1. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus: position statement. *Diabetes Care*. 2013;36 (suppl 1):67S-74S.
- 2. Metzger BE, Lowe LP, Dyer AR, Trimble ER, Chaovarindr U, Coustan DR, Hadden DR, McCance DR, Hod M, McIntyre HD, Oats JJ, Persson B, Rogers MS, Sacks DA. Hyperglycemia and adverse pregnancy outcomes. *N Engl J Med.* 2008;358:1991-2002.
- Metzger BE, Gabbe SG, Persson B, Buchanan TA, Catalano PA, Damm P, Dyer AR, Leiva A, Hod M, Kitzmiler JL, Lowe LP, McIntyre HD, Oats JJ, Omori Y, Schmidt MI. International Association of Diabetes and Pregnancy Study Groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. *Diabetes Care*. 2010;33:676-682.

Bibliography

- The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the Expert Committee on Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care.* 1997;20: 1183-1197.
- American Diabetes Association. Implications of the diabetes control and complications trial: position statement. *Diabetes Care*. 2002;25 (suppl 25S-27S.
- American Diabetes Association. Implications of the United Kingdom Prospective Diabetes Study. *Diabetes Care*. 2002;25: (suppl 1): 28S-32S.

MAJOR NUTRIENTS: FUNCTIONS AND SOURCES

Fat-Soluble Vitamins	Important Sources	Physiological Roles
Vitamin A (retinol, beta carotene)	Milk, butter, fortified margarine, whole milk Cheese, liver, egg yolk (retinol) Green leafy and stem vegetables, yellow fruits and vegetables (carotene), eg, spinach, asparagus, broccoli, carrots, apricots, and cantaloupe	Maintains normal vision in dim light, healthy skin, and mucous membranes Essential for normal skeletal and tooth development
Vitamin D (calciferol)	Exposure to sunlight, fortified foods, fish liver oils	Maintains blood calcium and phosphorus levels Required for proper bone development
Vitamin E (tocopherol)	Vegetable oils Whole grains, wheat germ Leafy vegetables Egg yolk Legumes, nuts (especially almonds, peanuts, pecans, walnuts), sunflower seeds	Protects the integrity of normal cell membranes Assists in prevention of hemolysis of red blood cells Protects vitamin A, acting as an antioxidant
Vitamin K	Lettuce, spinach (green leafy vegetables), kale, cauliflower, cabbage Egg yolk Soybean oil Liver	Produces prothrombin in normal blood clotting
Water-Soluble Vitamins	Important Sources	Physiologic Roles
Ascorbic acid (vitamin C)	Citrus fruits, strawberries, cantaloupe, tomatoes, sweet peppers, cabbage, potatoes, kale, parsley, turnip greens, broccoli	Maintains integrity of capillaries Promotes healing of wounds and fractures Aids tooth and bone formation Increases iron absorption Protects folic acid Helps form collagen for healthy connective tissue
Thiamin (vitamin B1)	Pork, liver, chicken, fish, beef Whole grains, wheat germ, dried yeast, enriched cereal products Nuts and lentils	Metabolizes carbohydrates for energy Provides function of nerve cell membranes
Riboflavin (vitamin B2)	Milk Liver, meat, fish, eggs Enriched cereal products Green leafy vegetables	Metabolizes carbohydrates, proteins, and fats for energy Closely related to the metabolism of protein
Niacin	Liver, poultry, meat, fish, eggs Whole grains, enriched cereal products Peanuts, peanut butter	Metabolizes carbohydrate for energy
Pyridoxine (vitamin B6)	Pork, organ meats, meat, poultry, fish, legumes, seeds Whole grains	Metabolizes protein Converts tryptophan to niacin Synthesizes hemoglobin Maintains integrity of central nervous system

Water-Soluble Vitamins	Important Sources	Physiologic Roles	
Vitamin B12 (cyanocobalamin)	Animal foods only: liver, meat, salt-water fish, oysters, eggs Milk	Essential for red blood cell maturation and normal function of all body cells (especially nervous system, gastrointestinal tract and bone marrow)	
Folate	Green leafy vegetables Liver, beef, fish, dry beans, lentils Whole grains	Essential for DNA synthesis and synthesis and maturation of red blood cells	
Pantothenic acid	Animal sources (esp. organ meats, egg yolk, and meat) Whole grains Legumes Yeast	Responsible for_metabolism of carbohydrates, proteins, and fats for energy; formation of some hormones, hemoglobin, and nerve-regulating substances	
Biotin	Organ meats, egg, yolk, legumes, nuts	Synthesizes fatty acids Helps in metabolism of carbohydrates for energy	
Minerals	Important Sources	Physiologic Roles	
Calcium	Milk Hard cheeses, eg, cheddar, Swiss, mozzarella, and provolone Yogurt, ice cream, cottage cheese Turnip and mustard greens, collards, kale, broccoli, cabbage	Maintains strength of bones and teeth Involved with transmission of nerve impulses, muscle contractions and relaxation, blood clotting, structure and function of cell membranes, and absorption of vitamin B12	
Phosphorus	Milk and milk products Meat, poultry, fish and eggs Whole grain cereals and flours Nuts and legumes	Essential for structure of bones and teeth; release of stored energy; structure of RNA and DNA; cell permeability; and metabolism of carbohydrates, fats, and proteins	
Magnesium	Whole grain breads and cereals Soybeans, nuts, dry beans and peas, green leafy vegetables	Fundamental to the production of energy, calcium, and phosphorus metabolism in bone; maintenance of the function and structural integrity of heart muscle as well as other muscles and nerves	
Sodium	Use of salt at the table and in cooking Processed foods Milk Eggs, meat, poultry, fish Smoked meats Olives, pickles, soy sauce	Maintains normal osmotic pressure water balance, normal irritability of nerve cells and contraction of muscles, and permeability of the cell membrane	
Potassium	Meats, poultry, fish, (especially veal and salmon) Fruits and vegetables (especially bananas, potatoes, tomatoes, and citrus fruits) Whole grain cereals	Maintains normal osmotic pressure and fluid balance Required to store energy within the cell Key to transmission of nerve impulse and contraction of muscle fibers, especially the heart muscle	
Chloride	Use of salt at the table and in cooking	Regulates osmotic pressure, water balance, and acid-base balance of extracellular fluid Is a component of hydrochloric acid in the gastric juice	

Trace Elements	Important Sources	Physiologic Roles
Iron	Liver, meat, fish and poultry Whole grain and enriched cereals Legumes Green leafy vegetables Eggs Dried fruit Foods cooked in iron pots and skillets (especially foods with a high acid content)	Essential to the formation of hemoglobin in blood and myoglobin in muscles, which supply oxygen to cells
Zinc	Animal products (especially liver and oysters) Beef, lamb, pork Whole grain cereals Legumes Peanuts Peanut butter	Essential in wound healing, synthesis of proteins, mobilization of vitamin A from liver, normal cellular immune functions, and normal growth of genital organs
Copper	Organ meats Shellfish (especially oysters and crabs) Whole grain cereals Hickory and brazil nuts, sesame and sunflower seeds Legumes (soybeans, kidney, navy, lima beans)	Essential for formation of red blood cells and the utilization of iron, production of energy, cell protection against oxidative damage, and synthesis of connective tissue
Iodine	Iodized salt used at the table and in cooking	Part of thyroid hormones Influences physical and mental growth, functioning of nervous and muscle tissues, circulatory activity, and metabolism of all nutrients
Fluoride	Fluoridated water Seafood	Increases deposit of calcium, which strengthens the bone and reduces the acid in the mouth, therefore decreasing tooth decay
Chromium Manganese Molybdenum Selenium Nickel Silicon Vanadium	Present in very small amounts in plant foods (ie, whole grains, dried beans and peas, nuts, seeds, fresh fruits and vegetables) Animal foods (meat, fish, poultry, eggs)	Essential as components of enzymes and hormones

PHYSICAL SIGNS OF NUTRITIONAL DEFICIENCIES

Body Part	Signs	Deficiencies
Hair	Color change Easy pluckability, sparseness Alopecia Brittle Dryness	Protein-energy malnutrition Biotin, zinc, vitamins A and E
Skin	Acneiform lesions Follicular keratosis (scalelike plaques) Xerosis (dry skin) Ecchymoses; petechiae (hemorrhagic spots) Thickening and hyperpigmentation of pressure points Scrotal dermatosis	Vitamin A Vitamin A or essential fatty acids Vitamin A Vitamins C and K Niacin Niacin and riboflavin
Eyes	Pale conjunctiva (pale coloring of eyelid lining and whites of the eyes) Bitot's spots (foamy spots on the whites of the eyes) Conjunctival xerosis (inner lids and whites appear dull, rough) Angular palpebritis (corners of eyes are cracked, red)	Iron, folate, or vitamin B ₁₂ Vitamin A Vitamin A Riboflavin and niacin
Mouth	Decreased production of salivary fluids Angular stomatitis (cracked, red, flaky at corner of mouth) Bleeding gums Cheilosis (vertical cracks of lips)	Vitamin A Vitamin B ₁₂ Vitamin C Riboflavin
Tongue	Atrophic papillae (smooth, pale, slick tongue) Glossitis (red, painful tongue) Magenta tongue (purplish, red tongue)	Folate, niacin, riboflavin, iron, or vitamin B ₁₂ Folate, niacin, and vitamin B ₁₂ Riboflavin
Nails	Koilonychia (concave, spoon-shaped)	Iron
Extremities	Genu valgum or varum (knocked knees or bowed legs) Loss of deep tendon reflexes of lower extremities	Vitamin D or calcium Thiamin and vitamin B ₁₂

Medication Classification	Effect of Food on Medication	Effect of Medication on Nutritional Status	Patient Guidelines
Analgesic Acetaminophen (Tylenol) Ibuprofen (Motrin)	Food delays but does not ↓ absorption	GI bleeding is possible Decreased platelet levels of vitamin C Decreased serum folate due to competing for serum protein binding sites	If medication (any analgesic) upsets stomach, take with meals
Aspirin/salicylate	None	Fecal iron loss; potassium depletion	Vitamin C supplementation is recommended for individuals receiving salicylates for treatment of rheumatoid arthritis May take with low-mineral carbohydrate snack
Antacid Magnesium trisilicate (Trisogel)	None	May ψ iron absorption	Take after meals with water Evaluate iron status regularly
Calcium carbonate (Tums) Sodium bicarbonate		↑ thirst; ↑ weight (edema)	Take iron supplements separately 1 hour before or 2 hours after eating
Anticonvulsant Diphenylhydantoin (Dilantin)	Administer separately from tube feeding due to possible effects of bioavailability	Possible megaloblastic anemia with long- term therapy (responds to 25 μ g/day of folate) Increased turnover of vitamin D Blocks conversion of vitamin D by liver; osteomalacia may result (responds to vitamin D) Increased vitamin C requirements Reduction in vitamin K-dependent coagulation factors Reduced serum B ₁₂ status Can \checkmark taste acuity Hyperglycemia has been reported Folate need \uparrow with long-term therapy; however, \uparrow folate will \checkmark absorption	Take with food or milk (medication may cause gastric irritation) Stop tube feeding 1 hour before and 1 hour after medication intake (1) Liberal intake of dairy products is advised Vitamin D or folate supplement may be needed Take Ca or Mg supplement or antacids 2 hours before or after medication If patient has loss of seizure control, may need to ψ folic acid supplement
Antibiotic/Anti- Infective Erythromycin Penicillin or Ampicillin Tetracycline (Achromycin)	Delayed absorption when taken with food Absorption impaired by concurrent intake of food and antacids containing	Anorexia; oral candidiasis; abdominal stress Can promote negative nitrogen balance	Take on empty stomach with full glass of water Allow 1 hour to elapse between penicillin dose and consumption of fruit juice or other acidic beverage Take on empty stomach with full glass of water to avoid nausea. Avoid milk products, iron-fortified cereals, and iron supplements within 2 hours of dosage

FOOD AND MEDICATION INTERACTIONS^a

<code>^aGI</code> indicates gastrointestinal; $\boldsymbol{\uparrow}$, increase; and $\boldsymbol{\downarrow}$, decrease

l

Medication Classification	Effect of Food	Effect of Medication on	Patient Guidelines
Linezolid (Zyvox)	Possible interaction with foods and beverages with high tyramine content (2)	Linezolid is an oxazolidinone antibiotic possessing weak, reversible monoamine oxidative inhibitor activity.	Avoid high tyramine containing foods during use. This IV antibiotic is primarily used in the hospital setting when typically lower tyramine foods are consumed. A recent study showed dietary restriction is often not necessary due to lower tyramine content of the hospital meals (3). However, FDA recommends avoiding consuming large amounts of foods or beverages with high in tyramine content during use (2).
Ciprofloxacin (Cipro) Sulfasalazine		Patients taking sulfasalazine may require a supplement of 1 mg/day of folic acid to prevent vitamin deficiency associated with competition of the medication for absorption of folate Possible \checkmark vitamin K and vitamin B absorption	Take multivitamins with minerals, Ca, Fe, Mg separately by 2 hours Stop tube feeding 2 hours before and 2 hours after medication intake
Antifungal Griseofulvin (Fulvicin)	Fulvicin absorption improves with a fatty meal or whole milk	Taste loss; oral candidiasis; dry mouth; stomach pain	Take with whole milk or meal
Antihyperlipidemic Clofibrate (Atromid-S) Colestipol (Cholestid; Probucol) Cholestyramine (Questran, Cuemid) Atorvastatin (Lipitor) Lovastatin, (Mevacor, Altocor) Simvastatin (Zocor)	Consuming high- fiber foods at same time medication is taken ↓ absorption of medication Grapefruit juice may increase medication availability and absorption (4-7) St. John's Wort can reduce the concentration of	Nausea Reported decreased absorption of vitamins A, D, K, B ₁₂ , folate, and Fe Constipation common	Take with food or milk Follow a low-cholesterol and low-fat diet Increase intake of water and high- fiber foods Take with meals Take vitamin/mineral supplements 1 hour before or 4 hours after medication intake Mevacor: Do not consume with high- fiber foods Avoid grapefruit juice (4-7). Mevacor and Altocor: Avoid St. John's Wort (8)

Medication	Effect of Food	Effect of Medication on	Patient Guidelines
Classification	on Medication	Nutritional Status	r attent durachines
^a GI indicates gastrointestinal	; $igtharpoon$, increase; and $igstyle$, decr	ease	
Antihypertensive			
Propranoioi beta	Food may	Dry mouth; diarrnea; nausea;	Take with food
(Inderal	decrease,	May prevent the appearance of	Avoid natural licorice
Lopressor)	delay absorption	certain premonitory signs and	Avoid natural neorice
портеззот ј	(depending on	symptoms of acute hypoglycemia in	
	which beta	type 1 diabetes	
	blocker is used)		
Calcium channel	Grapefruit juice		Avoid grapefruit juice (4-7).
blockers	may increase		
(Nifedipine,	medication		
Felodipine,	availability and		
Nicardipine,	absorption (4-7)		
Nimodipine,			
Nisoldining) and			
Phenylalkylamine			
beta blocker			
(Verapamil)			
Bronchodilator	Medication effect	May occasionally act as a GI irritant	Avoid large amounts of caffeine and
Theophylline	is increased by	Anorexia	chocolate
(Theo-24,	caffeine; toxicity	Bitter aftertaste	Take with food to help reduce GI
Theo-Dur,	can result	Raises glucose with high dosage	irritation
Theolair,	When plasma		Avoid major changes in
Slo-bld)	levels are		carbonydrate and protein
	tea cola		Avoid excessive intake of
	chocolate and		charbroiled meats
	acetaminophen		
	and xanthine		
	contribute to		
	high values; may		
	↑ risk of		
	cardiovascular		
	and central		
	side effects		
	Medication effect		
	may be		
	decreased by		
	ingestion of		
	charcoal-broiled		
	meats		
	High-		
	protein/low-		
	diet is associated		
	with decreased		
	medication level		

^aGI indicates gastrointestinal; \uparrow , increase; and \downarrow , decrease

Medication Classification	Effect of Food on Medication	Effect of Medication on Nutritional Status	Patient Guidelines
Cardiovascular Digoxin (Lanoxin)	Food delays but does not inhibit absorption except for bran, which may reduce absorption. Avoid Licorice (8). St. John's Wort can reduce the concentration of medications in blood (8)	Digitalis may exacerbate metabolic effects of hyperkalemia, especially with respect to myocardial activity May cause GI irritation Anorexia; weight loss Diarrhea. Some forms of licorice may increase risk for toxicity (8).	Take 2 hours after meals to lessen gastric irritation Take medication between meals if meals are high in bran; bran decreases effects and level of medication Diet should provide liberal potassium, Mg, and Ca intake Avoid natural licorice (8) (imported) ^b and low Na intake Avoid taking St. John's Wort (8) Avoid grapefruit juice (4-7)
Arrhythmia Specific: Amiodarone (Cordarone) Disopyramide (Norpace)	Grapefruit juice may increase medication availability and absorption (4-7)		
Diuretic Thiazides (Diuril, Hydrodiuril) Furosemide (Lasix) Triamterene with hydrochloro- thiazide (Dyazide, Maxzide)	Food increases absorption. Mg supplement will \checkmark absorption of medication Licorice may reduce effects (8), particularly hydrodiuruil and aldactone (8)	Natriuresis may be accompanied by loss of potassium Also increases K, Mg, Zn, and B ₆ excretion Will ↓ utilization of folate Hypokalemia is uncommon, but hyperkalemia may occur	Do not take with magnesium supplement Take with meal or milk Limit intake of foods high in sodium; sodium-restricted diet may be preferred Increase intake of foods high in potassium and folate Limit use of natural licorice (8) (imported) ^b Avoid use of salt substitutes Increase potassium intake only when necessary and then cautiously
GI Stimulant Cisapride (Propulsid)	Grapefruit juice may increase medication availability and absorption (4,5).		Avoid grapefruit juice (4,5).
Laxative Bisacodyl (Dulcolax)	Milk can raise pH of stomach sufficiently to dissolve enteric coating prematurely	Misuse may cause hypokalemia and weight loss	Take on empty stomach with 8 oz of water or juice Drink plenty of fluids
Bisacodyl (Dulcolax)	Milk can raise pH of stomach sufficiently to dissolve enteric coating prematurely	Misuse may cause hypokalemia and weight loss	Take on empty stomach with 8 oz of water or juice Drink plenty of fluids

^aGI indicates gastrointestinal; \uparrow , increase; and \downarrow , decrease

Medication Classification	Effect of Food on Medication	Effect of Medication on Nutritional Status	Patient Guidelines
Thyroid Preparation Synthroid		Goitrogenic substances naturally present in some foods can interfere with iodine uptake by the thyroid	Take iron supplement separately by 4 hours Take on empty stomach If hypothyroidism is induced by goitrogenic foods, one should encourage thorough cooking to inactivate the goitrogens in some vegetables
Haloperidol (Haldol)	May cause additive hypotension with alcohol Coffee or tea may precipitate liquid form of medication	Appetite ↓; weight loss; anorexia Dry mouth Constipation	Take Fe supplement separately by 4 hours Avoid consumption of coffee, tea, or fruit juice 1 hour before or 2 hours after taking liquid form Take with food or milk
Miscellaneous			
Cimetidine (Tagamet)	Food delays medication absorption and allows maintenance of an effective blood concentration between doses	Aplastic anemia; ↓ absorption of vitamin B ₁₂	Take with or directly after meals Advise concerning taste changes
Protease Inhibitor (Fortorase)	Grapetruit juice may increase medication availability and absorption (4,5).		Avoid grapefruit juice (4,5).
Benzodiazepines Midazolam, and Triazolam (Halcion, Clomipramine, Anafranil)	Grapefruit juice may increase medication availability and absorption (4,5,8).		Avoid grapefruit juice (4,5,8).

Vitamin Supplements Vitamin A	Adequate fat, protein, and vitamin E needed for absorption ↑ calories for carbohydrate intake; ↑ thiamin requirement	Toxic in excess doses	Avoid excessive intake of raw fish

^aGI indicates gastrointestinal; ↑, increase; and √, decrease ^bNatural licorice contains a corticosteroid pressor substance (carbenoxolone), which can interfere with the effect of antihypertensive medications (beta blockers/hydralazine /thiazides/spironolactone). Advise patients receiving antihypertensive therapy to eat no more than an occasional piece of natural licorice. See Section II: Herb and Medication Interaction.

Medication Classification	Effect of Food on Medication	Effect of Medication on Nutritional Status	Patient Guidelines
Thiamin (B ₁)	Foods high in	Large doses may ↑ blood glucose	Take with food or milk to ψ GI
Niacin (B ₃)	thiaminase may ↓ thiamin activity	and cause jaundice and G disturbances; 3 – 9 g/day produces toxicity	distress For capsules, do not mix contents with iam or jelly
Pyridoxine (B ₆)			
Vitamin C		Large doses may \uparrow red blood cell hemolysis and destroy dietary vitamin B ₁₂ when taken with food May be relevant to kidney stone formation	Take vitamin B-12 supplement separately by 1 hour Take with iron supplement to 个 iron absorption
Vitamin D		Toxic in excess doses	
Vitamin E		Large doses may induce vitamin K deficiency	
Mineral		>20 mg/day will produce severe	Do not take with high-fat, low-sugar
Supplements		skeletal fluorosis	(rich) foods
Potassium	Decreased	will ↑ fluorosis	Keep Ca supplement and albumin hydroxide separate of fluoride by 2 hours
K-Lyte)	taken with dairy products		Do not take with dairy products

^aGI indicates gastrointestinal; $\hat{\mathbf{\Lambda}}$, increase; and $\mathbf{\psi}$, decrease

Also find reference to the following medications:

Anticoagulants	See Section III: Anticoagulant Therapy
Corticosteroids	See Section III: Corticosteroid Therapy
Calcium supplements	See Section IF: Nutrition Management of Calcium Intake
Chemotherapeutic agents	See Section III: Cancer
Monamine oxidase inhibitors	See Section IH: Tyramine Restricted Diet

Manual of Clinical Nutrition Management

Oral glucose lowering medications See Section III: Diabetes: Oral Glucose Lowering Medications and Insulin

References

- 1. Doak KK, Haas CE, Dunnigan KJ, Reiss RA, Reiser JR, Huntress J, A Havela JL. Bioavailability of pheyntoin acid and pheyntoin sodium with enteral feeding. *Pharmacotherapy*. 1998; 18: 636-645.
- Drug safety data: Linezolid. Available at http://www.accessdata.fda.gov/drugsatfda_docs/label/2005/021130s008,009,021131s009,010,021132s008,009lbl.pdf(2005 data report). Accessed 8/6/2010.
- 3. Rumore MM, Roth M, Orfanos A. Dietary tyramine restriction for hospitalized patients on linezolid: an update. *Nutr Clin Pract.* 2010;25:265-269.
- 4. Baily DG, Malcolm J, Arnold O, Spence JD. Grapefruit juice-drug interactions. Br J Clin Pharmacol. 1998;46:101-110.
- Huang SM, Hall SD, Watkins P, Love LA, Serabjit-Singh C, Betz JM, Hoffman FA, Honig P, Coates PM, Bull J, Chen ST, Kearns GL, Murray MD. Drug interactions with herbal products and grapefruit juice: A conference report. *Clin Pharmacol Ther.* 2004;75:1-12.
- 6. Stump AL, Mayo T, Blum A. Management of grapefruit-drug interactions. Am Fam Phy. 2006;74: 605-608.
- 7. Mertens-Talcott SU, Zadezensky I, DeCastro WV, Derendorf H, ButterweckV. Grapefruit-drug interactions: can interactions with drugs be avoided. *J Clin Pharm*. 2006;46:1390-1416.
- 8. U.S. Department of Health and Human Services and U.S. Food & Drug Administration. Drugs with Food and Beverages. Available at http://www.fda.gov/ForConsumers/ConsumerUpdates/ucm096386.htm . Accessed October 1, 2010.

Bibliography

U.S. Department of Health and Human Services and U.S. Food & Drug Administration. Drugs with Food and Beverages. Available at http://www.fda.gov/ForConsumers/ConsumerUpdates/ucm096386.htm . Accessed October 1, 2010.

II cl-	C anno 11 a 11		II al-	Dationt Codd alter
Herb Classification -	Lommon	Possible Side	HerD- Modication	Patient Guidelines
Classifications	indications	Enects	Interactions	
Corman chamomile	Internal	Allergy (raro) (1)	Avoid use with	Avoid taking with other
Matricaria recutita	*Cough/bronchitis (2)	Anergy (rate) (1)	coumarin	sedatives, such as
	*Fevers and colds (2)	Patients with severe	anticoagulants (2).	benzodiazepines and
(Note: Various	*Inflammation or	allergies to ragweed	Chamomile may	alcohol (1).
types are available,	spasms of the	should be warned	exacerbate the	Decommonded deily decomp
chamomile, with	*Inflammation of the	reactivity to	effects of warfarin	for internal use: 3 g (2) as an
varying properties.)	mouth and pharynx (2)	chamomile and other	(4).	infusion; 3 g in 150 mL
	*Tendency for infection	members of the aster	Amaid was with	water 3 times daily for
		feverfew, and milk	alcohol and	(8): 1 to 4 mL of liquid
	Topical:	thistle) (2).	benzodiazepines	extract or 1 cup of tea
	Mild inflammation of		(2).	administered three to four
	the skin (2)			times daily (2).
	Contraindications:			
	Pregnancy and			
Echinacea	Internal:	Possible suppression	Immunostimulatin	Dosage is dependent on
Echinacea	*Prevention and	of immunity with	g effects of	for short torm use (<2
Echinacea	cough/bronchitis. and	fiabitual use (1-2)	the	weeks) (1.2.4) are $\frac{1}{2}$ to 1 tsp
angustifolia,	urinary tract infections	Parenteral doses may	immunosuppressiv	liquid (expressed juice of
Echinacea pallida	*Inflammation of the	cause fever, nausea,	e effects of	the herb stabilized in 22%
	mouth and pharynx *Stimulates immune	and vomiting (2).	corticosteroids and	alcohol) or one 88.5-mg
	system (2)	diabetes should	cyclospornic (2,3).	or 900 mg daily (2).
	m ·)	avoid parenteral		
	Topical: Promotes wound	administration (2).		
	healing (1-3)			
	Contraindications: Multiple sclerosis			
	leukosis, collagen			
	disease, AIDS,			
	tuberculosis, and			
	pregnancy			
Feverfew	Prevention and	Potential	May interact with	Recommended daily dosage:
Tanacetum	treatment of migraines	sensitization via skin	thrombolytics,	Recommended daily dosage:
purtnenium	and migrame- associated nausea and	medication (2)	and medications	standard content of 0.2%
	vomiting (2)		that decrease	parthenolide (3). A 4- to 6-
	Antionthritic co	Inflammation of	platelet	week course of continual
	Anuarunnuc <u>(</u> 1)	mouth and tongue (3)	aggregation (eg,	is suggested to improve
	Contraindications:			migraines.
	Pregnancy and			
				A 4- to b-week course of
				improve migraines.

HERB AND MEDICATION INTERACTIONS

Herb	Common	Possible Side	Herb-	Patient Guidelines
Classifications	Indications	Effects	Medication Interactions	
Garlic Allium sativum	*Antiatherosclerosis (lipid-lowering anti- thrombotic, fibrinolytic, antihypertensive) (1-2) <i>Contraindications:</i> Lactation; prolongs bleeding time, and should be discontinued 1-2 weeks prior surgery (3)	Stomach upset, headache, myalgia, fatigue, and vertigo ⁽²⁾ Sulfuric odor, contact irritation, and dermatitis (2)	May increase the effect of antihypertensive medications and anticoagulant medications, such as aspirin, NSAIDs, or warfarin (2,5)	Recommended daily dosage: a commercial preparation of 600 to 900 mg (containing 3 mg of allicin or a total allicin potential of 5,000 μ g) in an enteric-coated form QD (2) or one clove of raw garlic (equal to 4 g) QD or BID (2).
Ginger Zingiber officinale	*Loss of appetite, nausea, travel sickness, and dyspeptic complaints (1,2) <i>Contraindications:</i> Germany's Commission E contraindicates the use of ginger for morning sickness associated with pregnancy (2); gallstone conditions; and persons at risk for hemorrhage (2).	Heartburn (1) Doses >6 g/day may lead to ulcer formation (2). Allergic reaction (rare) (1)	May exacerbate the anticoagulant effects of warfarin (2,3) May decrease effectiveness of antacids, H2 blockers, and proton pump inhibitors May interfere with diabetic and blood pressure medicines (3)	Use only briefly during pregnancy. May prolong bleeding, so do not use after surgery. Patients receiving anticoagulant medications or patients with a history of gallstones should not take ginger (2,3,4). Usual dose for antiemetic is 1 to 2 g freshly ground ginger taken with liquid and in two divided doses (2)
Ginkgo Ginkgo biloba	*Symptomatic relief of organic brain dysfunction and intermittent claudication (dementia, peripheral occlusive arterial disease [POAD]) (1,2); improves memory *Vertigo (vascular origin) (2) *Tinnitus (vascular origin) (2)	Gastrointestinal tract disturbance, headache, and (rarely) contact dermatitis (1-3); blood pressure irregularities (2); blood glucose level changes (3) Patients with known risk factors for intracranial hemorrhage (eg, systemic arterial hypertension, diabetes amyloid senile plaques) should avoid use of ginkgo (2).	May exacerbate the effects of antithrombolytic agents (eg, anticoagulants, antiplatelets, aspirin, or acetaminophen) as a result of a potent inhibitory effect on platelet-activating factor (2,4) May cause hypomania if taken with fluoxetine May interact with medicines that lower seizure threshold and thiazide diuretics (3)	Ginkgo is available in a capsule, tablet, or liquid form. Absorption is unaffected by food intake (1). Recommended daily dosage for cerebrovascular insufficiency (eg, dementia, POAD, and equilibrium disorders) is 120 to 240 mg of standardized dried extract in 2 or 3 oral doses (2,6). A 6- to 8-week course is advised to determine effectiveness of therapy. Avoid taking with Tegretol, Equetro, Carbatrol (cabamazepine), and Depakote (valproic acid) (7)

Herb Classifications	Common Indications	Possible Side Effects	Herb-Medication	Patient Guidelines
Ginseng Panax ginseng Panax quinquefolius	*Stimulates the central nervous system, reduces fatigue, and improves concentration (1-3) Anticancer effects (1,2); antioxidant effects (2); antiplatelet effects (2); antiviral effects (2); hypolipidemic/cardiac effects (2); and hypoglycemic effects (2) <i>Caution in use with:</i> Cardiac disorders, including hypertension (6), and diabetes (2,4) <i>Contraindications:</i> Pregnancy and	Tachycardia and hypertension (1,2) Insomnia, epistaxis, headache, nervousness, and vomiting (2) Reports of mastalgia and postmenopausal vaginal bleeding (2) Overdose can cause hypertension, insomnia, hypertonia, and edema (2).	Avoid concomitant use with warfarin, NSAIDs, and antiplatelet agents (2,5) due to anticoagulant effects. Caution should be taken with diabetic agents/insulin due to hypoglycemic effects (2). Patients taking steroids, MAOIs, or loop diuretics should not use ginseng (2,4).	Recommended daily dosage (usually capsule form) for cognitive function is 400 mg (2); for hypoglycemic effects, 100 to 200 mg (2); for antiviral effects, 100 to 200 mg (2); for physical and psychological performance, 100 mg twice a day (2). Other recommendations: 100 mg QD or BID of 4% to 7% ginsengosides (3) Limit continuous use to less than 3 months (3).
Green tea Camellia sinensis	lactation (2). Prevents cancers of the pancreas, colon, small intestine, stomach, breast, and lung (2) Dental caries (2) Diarrhea (2)	Excess consumption (>5 cups/day) can cause gastrointestinal tract irritation (related to hyperacidity) and excitability or anxiety (related to caffeine) (2). Pregnant women should not exceed a dosage of 300 mg/day (2). Microcytic anemia has been reported in infants fed 250 mL of green tea daily (2).	Resorption of alkaline medications can be delayed because of chemical bonding with tannins (2). May increase risk of bleeding with anticoagulant medications May interact with verapamil by increasing plasma caffeine levels (3)	Available as an infusion or capsule form for internal use. Recommended daily dosage is 300 to 400 mg of polyphenols or 3 cups of green tea (which contains 240 to 320 mg of polyphenols) (2). Avoid concomitant use with grapefruit juice (3).
English hawthorn Crataegus laevigata	*Decreased cardiac output, senile heart, chronic cor pulmonale, and mild forms of bradycardial arrhythmias (2) <i>Contraindications:</i> Acute angina (because herb action is too slow) (1,2); avoid during first trimester of pregnancy; children younger than 12 years should avoid this product	Hawthorn supplements should be prescribed and monitored by a physician (2). During treatment, heart rate and blood pressure should be monitored on a regular basis (2).	Use with antiarrhythmics is discouraged due to similar modes of action (2). English hawthorn may potentiate the effects of cardiac glycosides. Therefore, if initiated in patients taking digoxin, digitoxin, or g- strophanthin, the glycoside dosage should be adjusted (2).	Recommended daily dosage is 5 g of medication or 160 to 900 mg of hawthorn extract (standardized to 20% procyanidins or 2.2% flavonoid content) administered in divided doses three times daily (2,3). Treatment duration is a minimum of 6 weeks (2).

Herb Classifications	Common Indications	Possible Side Effects	Herb-Medication Interactions	Patient Guidelines
English hawthorn <i>Crataegus laevigata</i> (Continued) Kava kava	*Suppresses anxiety	In rare cases, kava	Can cause a hypertensive effect when used in combination with beta-blockers (2) Should be avoided with cisapride and other medications in a similar medication class as cisapride (2) May potentiate the	Recommended dosage is
Piper methysticum	and the central nervous system (2) *May relieve mild anxiety and sleeplessness/ restlessness (2,3) <i>Contraindications:</i> Pregnancy and lactation; patients with endogenous depression (2,8)	kava may cause dry patchy skin and a temporary yellow discoloration of skin, hair, and nails (1). Overdose can result in disorders of complex movement (without a disturbance of consciousness), followed by tiredness and tendency to sleep (2).	effectiveness of substances that affect the central nervous system (eg, alcohol, barbiturates, and psychopharmacologic agents) (2) Kava kava is reported to antagonize the effect of dopamine. Patients with Parkinson disease who take levodopa should avoid kava kava (2).	150 to 300 mg of root extract taken twice daily, with a daily dose of preparations equivalent to 50 to 240 mg kava pyrones (2). The herb should be taken with food or liquid due to its lipid solubility (2). Avoid concomitant use of kava kava with alcohol (3)
Licorice Glycyrrhiza glabra	*Soothing stomach irritations/gastritis (2) *Cough remedy and expectorant/bronchi tis (2) <i>Contraindications:</i> Natural licorice (except deglycyrrhizinated licorice) is not recommended for people with high blood pressure, heart disease, diabetes, cholestatic liver disorders, liver cirrhosis, hypertonia, hypokalemia, or severe kidney insufficiency (6); or for pregnant or lactating women (2,3);	Large amounts may lead to potassium loss, sodium retention, edema, high blood pressure, and cardiac complaints (2,5).	Avoid with thiazide medications, as licorice may counteract the effects of thiazide medications (2). Increases potassium losses, which may increase toxicity to digitalis glycosides (2,4) May interfere with ani-arrhythmic agents (eg, procainamide, quinidine) (2). Prolongs half-life of cortisol, which may lead to hypokalemia, high blood pressure, and edema (2)	Recommended daily dosage is 5 to 15 g (1 to 2 tsp) of dried root, containing 200 to 600 mg of glycyrrhizin (2). Should not be used more than 4 to 6 weeks, otherwise the risk of side effects and overdose increases (2,9) Avoid concomitant use with grapefruit juice (3).

Herb Classifications	Common Indications	Possible Side Effects	Herb-Medication	Patient Guidelines
Milk thistle	*Dyspentic	No known side affects	Concomitant use with	Recommended daily
Silybum marianum	complaints (2)*Used as a tonic, as a stimulant, and for relief of functional disorders of the liver and gallbladder (2)	if properly administered (2)	butyrophenones or phenothiazines results in reduced lipid peroxidation (2) Antagonistic effect with yohimbine and phentolamine (2)	dosage is a 140 mg to 420 mg capsule (standardized to 70% silymarin) BID or TID (1,2); or 400 mg of concentrated extract (which equals 140 mg of silymarin).
Saw palmetto Serenoa repens	*Prostate complaints (relieves the difficulties caused by an enlarged prostate without reducing the enlargement) (2) *Irritable bladder (2) Inhibits male hormones; has some effects on estrogen; may be anti- inflammatory (3) <i>Contraindications:</i> Pregnancy and lactation (due to potential hormonal effects) (2)	Rare cases of gastrointestinal tract upset (1,2)	May exert estrogen, androgen, and alpha- adrenergic blocking effects; therefore, the concomitant use of hormones, hormone- like medications, or adrenergic medications may need to be adjusted (2) No significant adverse effects have been reported in clinical trials (2). Might increase risk of bleeding if taken with anticoagulant medications (3)	Prostate enlargement requires diagnosis and follow-up by a physician (5). Recommended daily dosage is 160 mg BID or 320 mg one time per day of an extract standardized to contain 85% to 95% fatty acids and steroids (2,4).
Valerian Valerian officinalis	*Nervousness and insomnia (1,2) Relieves pain, reduces spasms (6), and stimulates appetite (6) <i>Contraindications:</i> Pregnancy and lactation	Heart palpitations and insomnia occur rarely with long-term use (1,2).	Avoid use with alcohol (2). Potentiates the effect of central nervous system depressants and not recommended for use with sedatives or antidepressants (2). May exacerbate the side effects (drowsiness and fatigue) of medications used to treat allergies or anxiety (eg, antihistamines) (5)	Recommended daily dosage is 400 to 900 mg of standardized valerian root 30 minutes before bedtime to treat insomnia or 220 mg in extract three times daily to treat restlessness (2).

*Indications for use have been approved by the Commission E, Germany's regulatory authority on herbal and botanical products, which is currently recognized as the best expert consensus on medicinal herbs (2).

NSAIDs = nonsteroidal anti-inflammatory drugs; MAOI = monoamine oxidase inhibitor; SSRI = selective serotonin reuptake inhibitor; TID = three times a day; BID = two times a day; QD = every day; a.m. = morning; p.m. = evening.

References

1. O'Hare M, Kiefer D, Farrell M], Kemper K. A review of 12 commonly used medicinal herbs. Arch Fam Med. Available at:

http://www.ama-assn.org/sci-pubs/journals/archive/fami/vol_7/no_6/fsa8005.htm. Accessed February 12, 1999.

2. *PDR for Herbal Medicines*. 2nd ed. Montvale, NJ: Thomson Medical Economics Co; 2000.

3. Therapeutic Research Faculty. Natural Medicines: Comprehensive Database. Stockton, Ca.; 2004.

- 4. Mathai K. Integrative medicine and herbal therapy. In: Mahan K, Escott-Stump S, eds. *Krause's Food, Nutrition, and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders Co; 2000;415-429.
- 5. Ginseng, cardiac drugs don't mix: herbal interactions you should know. *Environ Nutr.* 1999;22:1,4-5.
- 6. Blumenthal M, Goldberg A, Brinckmann J. Herbal Medicine: Expanded Commission E Monographs. Newton, Mass: Integrative Medicine Communication; 2000.
- 7. U.S. Department of Health and Human Services and U.S. Food & Drug Administration. Drugs with Food and Beverages. Available at http://www.fda.gov/ForConsumers/ConsumerUpdates/ucm096386.htm . Accessed October 1, 2010.
- 8. EN's herbal medicine cabinet: top 10 herbs you can trust. *Environ Nutr.* 1998;21:1,4.
- 9. Licorice: sweet candy, soothing remedy, but easy to overdo. Environ Nutr. 1999;22:8.

Bibliography

A Healthcare Professional's Guide to Evaluating Dietary Supplements. Chicago, Ill: The American Dietetic Association and American Pharmaceutical Association; 2000.

III. CLINICAL NUTRITION MANAGEMENT

Introduction	III-1
Table III-1: Conclusion Grading Characteristics	III-1
Table III-2: Grading Implications For Practice	III-2
Anticoagulant Therapy	III-3
Table III-3: Foods Rich in Vitamin K	III-4
Burns	III-6
Cancer	III-10
Table III-4: Cancer Treatments with Potential to Affect Nutritional Status	III-11
Table III-5 : Suggested Nutrition Interventions	III-12
Chronic Obstructive Pulmonary Disease	III-16
Corticosteroid Therapy	III-20
Table III-6: Dietary Interventions for Side Effects of Corticosteroids	III-20
Monitoring in Diabetes Mellitus	III-21
Diabetes Mellitus: Considerations for Exercise	III-23
Diabetes Mellitus: Considerations for Acute Illness	III-25
Diabetes Mellitus: Gastrointestinal Complications	III-27
Diabetes Mellitus: Oral Glucose-Lowering Medications And Insulin	III-29
Table III-7: Glucose-Lowering Agents	III-29
Table III-8: Human Insulins.	
Diabetes Mellitus: Fat Replacers and Nutritive/Nonnutritive Sweeteners	111-32
Dysphagia	III-35
Relationship of Dysphagia to the Normal Swallow	III-37
Enteral Nutrition: Management of Complications	III-38
Gastroesophageal Reflux Disease (GERD)	III-40
Table III-9: Therapeutic Approaches For GERD	III-40
Heart Failure	III-42
HIV Infection and AIDS	III-46
Table III-10: Evaluation Criteria for Weight and Body Composition Changes	III-49
Table III-11: HIV Medications	III-52
Table III-12:-Nutrition Management Strategies	III-56
Hypertension	III-62
Table III-13: JNC 7 Classification of Blood Pressure for Adults	
Table III-14: JNC / Classification and Management of Blood Pressure For Adults	111-03 III_63
Table III-16: Evidence Supporting Lifestyle Modifications to Manage Hypertension	III-65
Table III-17: DASH Eating Plan	III-66
Table III-18: DASH Eating Plan–Number of Servings for Other Energy Levels	III-68
Hypertriglyceridemia	III-70
Table III-19: Recommendations for Elevated Triglyceride Levels	III-71
Hypoglycemia	III-72
Inborn Errors of Metabolism	III-74
Herb and Drug Interactions

Table III-20: Metabolic Disorders That Respond to Dietary Treatment	III-74
Iron Deficiency Anemia Table III-21: Iron Content of Common Foods	 III-77 III-78
Nephrotic Syndrome	III-79
Obesity And Weight Management Table III-22: Classification of Overweight and Obesity and Associated Disease Risk Table III-23: A Guide to Selecting Treatment for Obesity and Overweight	 III-81 III-82 III-82
Pancreatitis	III-89
Table III-24: Common Assessment Parameters for Pancreatitis Table III-25: Guidelines for Using Enteral Nutrition in Severe Acute Pancreatitis Table III-26: Nutrition Intervention Strategies for Pancreatitis	III-89 III-91 III-92
Paranteral Nutrition (PN): Matabolic Complications	111.94
rarenteral Nutrition (TN). Metabolic complications	111- 74
Calculating Total Parenteral Nutrition	III-94
Calculating Total Parenteral Nutrition Peptic Ulcer	III-94 III-99 III-103 III-103
Calculating Total Parenteral Nutrition Peptic Ulcer	III-99 III-103 III-103 III-104
Calculating Total Parenteral Nutrition Peptic Ulcer	III-99 III-103 III-103 III-104 III-106 III-108
Calculating Total Parenteral Nutrition Peptic Ulcer. Table III-27: Medical Approaches for Peptic Ulcer Disease Pneumonia Pressure Ulcers Table III-28: Guidelines for Vitamin C and Zinc Supplementation. Management of Acute Kidney Injury and Chronic Kidney Disease (Stage V) Table III-29: Classification/Staging System For AKI Table III-30: Vitamin and Mineral Supplementation in AKI Managed With CRRT. Nutrition Care Outcomes and Interventions in CKD Renal Replacement Therapy.	III-99 III-103 III-103 III-104 III-106 III-108 III-110 III-110 III-111 III-111

CLINICAL NUTRITION MANAGEMENT A REFERENCE GUIDE

INTRODUCTION

The material in this section:

- provides the dietitian with relevant evidence-based information to consider in the development of the nutrition care plan
- forms the basis for the development of disease- or condition-specific nutrition interventions and protocols as required by the organization

As part of Morrison Management Specialists' strategic plan to support and assimilate evidence-based research into clinical practice, the *Manual of Clinical Nutrition Management* integrates the Academy of Nutrition and Dietetic's (AND or Academy) recommendations and conclusion-grading statements established as part of the Academy's Evidence Analysis Library and evidence-based analysis process. The Academy's Evidence Analysis Library (www.andevidencelibrary.com) is an online library that includes a synthesis of the best, most relevant research on important dietetic practice questions. The library's resources include conclusion statements that provide a concise summary of the research on a given question. The Academy has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. These grades, which are based on the quality and extent of the research, are a tool for practitioners to use when determining the certainty of information.

The Academy's grades are integrated throughout the *Manual* to assist the dietitian in interpreting the strength and relevance of evidence on a particular topic. The criteria and characteristics of the five grades are described in Table III-1.

Tuble III II conclusion draung characteristics			
Grade I Good/strong	Quality of studies is strong and free from design flaws, bias, and execution problems. Uses large number of subjects; outcomes		
	directly related to question; statistical difference is large and meaningful; can be generalized to population of interest.		
Grade II	Quality of studies is strong, however, with minor methodological		
Fair	concerns; inconsistency among results of studies, or studies		
	evaluated have weaker design; doubts about adequate sample size;		
	doubts about statistical significance; minor doubts about		
	generalizability to population of interest.		
Grade III	Studies of weak design; inconclusive findings due to design flaws, bias,		
Limited/weak	or execution problems; inconsistency among results that cannot be		
	explained; inadequate sample size; serious doubts about		
	generalizability to population of interest.		
Grade IV	No studies available; conclusion based on usual practice, expert		
Expert opinion only	consensus, clinical experience, opinion, or extrapolation from the		
	research.		
Grade V	No evidence pertains to the question being addressed.		
Insufficient evidence			

Table III-1: Conclusion Grading Characteristics^a

^aThe grading system is based on the grading system from: Greer N, Mosser G, Logan G, Halaas GW. A practical approach to evidence grading. *Jt Comm J Qual Improv.* 2000;26:700-712. In September 2004, the Academy's Research Committee modified the grading system to this current version.

When necessary, the practitioner can use grading information to assist in clinical decision-making as described in Table III-2.

Introduction

Table III-2: (Grading	Implications	for	Practice ^a
----------------	---------	--------------	-----	------------------------------

Table III =: araang Imprice	
Grade I	Practitioners should follow recommendation unless a clear and
Good/strong	compelling rationale for an alternative approach is present.
Grade II	Practitioners should generally follow recommendation but remain
Fair	alert to new information and be sensitive to patient preferences.
Grade III	Practitioners should be cautious in deciding whether to follow
Limited/weak	recommendation, and should exercise judgment and be alert to
	emerging publications that report evidence. Patient preference
	should have a substantial influencing role.
Grade IV	Practitioners should be flexible in deciding whether to follow
Expert opinion only	recommendation, although the recommendation may set
	boundaries on alternatives. Patient preference should have a
	substantial influencing role.
Grade V	Practitioner should exercise judgment and be alert to emerging
Insufficient evidence	publications that report evidence that clarifies the balance of
	benefit vs. harm. Patient preference should have a substantial
	influencing role.

^aAdapted by the Academy of Nutrition and Dietetics from: American Academy of Pediatrics Steering Committee on Quality Improvement and Management. Classifying recommendations for clinical practice guidelines. *Pediatrics*. 2004;114:874-877.

Grading information in the *Manual* appears in parentheses, as seen in the following example:

Evidence shows that physical activity at any level, light, moderate, or vigorous, as well as food patterns emphasizing a diet high in fruits, vegetables, and whole grains is associated with reduced incidence of metabolic syndrome (Grade II) (1).

64. *Disorders of Lipid Metabolism Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: http://www.andevidencelibrary.com. Accessed January 16, 2013.

Because the Academy's Evidence Analysis Library is an evolving project, not all sections and recommendations in the *Manual* contain grading information. Also, the recommendations that are graded may be frequently updated as evidence emerges. The practitioner is encouraged to refer to the Academy's online library for updates on emerging topics. The grading information is provided to assist practitioners in making decisions about clinical care and interventions. Grading information should complement clinical decision-making, not replace sound clinical judgment or expertise.

This section also contains information (eg, medical diagnostic tests or laboratory indexes) that may not necessarily be mentioned in the nutrition assessment care plan. The evidence that supports the nutrition-specific information is included to strengthen the dietitian's role as a participating member of the health care team. In developing the individual patient care plan, the dietitian selectively discusses the assessment parameters and interventions that are pertinent to improving the patient's nutrition care.

In conclusion, the approaches mentioned for each condition are suggestions that should not be interpreted as definitive nutrition therapy for the given condition. The evidence grades are provided to guide clinical decision-making and the selection of optimal nutrition approaches. Medical approaches are listed with medical nutrition therapy approaches to create an awareness of coordinated therapies. Diets approved by the organization's medical staff are included in Section I: "Normal Nutrition and Modified Diets". Condition-specific protocols, if developed by the organization from the following material, should be approved by the appropriate committee and placed in the organization's practice guidelines manual.

Additional information can be obtained at www.andevidencelibrary.com

The material in this section is intended:

- To provide the dietitian with relevant information that may be considered in the development of the nutrition care plan.
- To form the basis for the development of disease- or condition-specific protocols and nutrition prescriptions as required by the organization.

ANTICOAGULANT THERAPY

Discussion

Oral anticoagulants are used to create a partial deficiency of the active form of vitamin K, which is responsible for maintaining normal blood coagulation. By inhibiting the action of vitamin K, there is a reduced risk of abnormal blood clotting.

Indications

Oral anticoagulants are typically prescribed for the primary and secondary prevention of the following conditions:

- venous thrombosis
- pulmonary embolism
- myocardial infarction

Persons with prosthetic heart valves, atrial fibrillation with embolization, or heredity disorders that result in a hypercoagulant state may be treated with anticoagulants indefinitely (1,2).

A major complication of anticoagulant therapy is hemorrhage. The therapeutic index and safety of anticoagulation therapy is assessed through the measurement of the prothrombin time (PT), which is expressed as the international normalized ratio (INR) (2). An INR of 2.0 to 3.0 is generally considered in the therapeutic range, and the risk of bleeding increases when the INR exceeds 4.0 (2).

Nutrition Implications of Anticoagulant Therapy

The goal of medical nutrition therapy for persons receiving anticoagulant therapy is to provide a consistent intake of vitamin K. The Daily Value for vitamin K is 80 mcg for adults (3). The Daily Value can be used as an appropriate goal for persons on anticoagulant therapy (3,4). The average dietary intake of vitamin K for adults in the United States is estimated to be 90 to 118 mcg/day (5-7). Although most patients' intake will fall into this acceptable range, all patients should have their diets assessed for typical sources and patterns of foods containing vitamin K. Persons who receive anticoagulant therapy should limit their consumption of foods that have a high level of vitamin K.

The list of drugs that interact with vitamin K antagonists is constantly expanding (7). Drug-drug interactions that increase or decrease the effect of anticoagulant therapy should be evaluated before concluding that dietary intake is responsible for a change in the anticoagulant response (2,5,7). Drugs that increase the anticoagulant effect are agents for gout treatment, anabolic steroids, antiarrhythmic agents, antibiotics, antifungal agents, antihyperlipidemic agents, cimetidine, disulfiram, isoniazid, omeprazole, sulfonylureas, and tamoxifen citrate. Drugs that decrease the anticoagulant effect are anticonvulsant agents, cholestyramine, griseofulvin, oral contraceptives, rifampin, sucralfate, and vitamin K (5,8).

Nutrition Assessment and Diagnosis

The oral anticoagulant dose should be established based on the patient's normal vitamin K intake. After the dose is established, a reasonable goal is to maintain the daily vitamin K intake within 250 mcg of baseline (1,9). If major changes in food intake occur, the anticoagulant level may need to be reestablished. Vitamin K intake may increase when a patient starts a weight-reduction diet and includes a greater number of vegetables that are high in vitamin K or begins a high-protein, low-carbohydrate diet (see discussion below). Other reasons for an increased vitamin K intake may include an adjustment in diet because of hospitalization or a change in seasonal eating patterns (1). Unlike other fat-soluble vitamins, stores of vitamin K are rapidly depleted if intake is deficient (1). This information should be considered when assessing the vitamin K level of a patient who has had a low intake of food for a week or longer, as may occur in the hospital setting.

Nutrition Intervention and Monitoring

Patients should be educated about the dietary changes that impact anticoagulant therapy. They also should be informed of foods that are high in vitamin K (III-3). Patients should be encouraged to keep their diet consistent with their present pattern. However, if there is a change in diet that includes vitamin K-rich foods, patients should contact their physician and have their INR/PT monitored.

Anticoagulant Therapy

Table III-3: Foods Rich in Vitamin K (9)

Food	Serving Size	Vitamin K (mcg)
Kale, cooked	½ cup	531
Spinach, cooked	¹ ∕₂ cup	444
Collards, cooked	¹ ∕₂ cup	418
Swiss chard, raw	1 cup	299
Swiss chard, cooked	¹⁄₂ cup	287
Mustard greens, raw	1 cup	279
Turnip greens, cooked	½ cup	265
Parsley, raw	1⁄4 cup	246
Broccoli, cooked	1 cup	220
Brussels sprouts, cooked	1 cup	219
Mustard greens, cooked	½ cup	210
Collards, raw	1 cup	184
Spinach, raw	1 cup	145
Turnip greens, raw	1 cup	138
Endive, raw	1 cup	116
Broccoli, raw	1 cup	89
Cabbage, cooked	½ cup	82
Green leaf lettuce	1 cup	71
Prunes, stewed	1 cup	65
Romaine lettuce, raw	1 cup	57
Asparagus	4 spears	48
Avodaco	1 cup(cube, slice, puree)	30-48
Tuna, canned in oil	3 ounces	37
Blue/black-berries, raw	1 cup	29
Peas, cooked	½ cup	21

Some evidence suggests that cranberry products interact with anticoagulants to increase their effects (10). However, a prospective randomized study of patients taking warfarin demonstrated that their INR levels were not adversely affected by consumption of 1 cup of cranberry juice daily (11). Until further evidence is available on a variety of cranberry products, patients consuming cranberry juice or products should be educated, and their INR/PT should be monitored for the potential interaction. Although iceberg lettuce, red cabbage, asparagus, cauliflower, and soybean oil are often reported as being high in vitamin K, these foods contain much smaller amounts of vitamin K than the foods listed in Table III-3. Therefore, these foods and other foods and beverages not listed (including coffee and tea) may be consumed as desired (9).

Special Considerations

Alcohol: Alcohol has shown to adversely affect the PT/INR ratio (5). Consuming more than 3 servings of alcoholic beverages per day can increase the effect of warfarin (9). Limiting or avoiding alcohol may be advised, and persons who do consume alcohol should consult with their physician.

High-protein, low-carbohydrate diets: A high-protein, low-carbohydrate diet pattern decreases the INR/PT ratio (12). Case reports have demonstrated a decrease in the INR/PT ratio after initiation of a high-protein, low-carbohydrate diet (12). The INR/PT ratio returned to a normal level after the diet was stopped and the warfarin dose was decreased to the original dose. High-protein diets rapidly increase serum albumin levels. This increase may result in more warfarin binding to serum albumin, thereby decreasing the anticoagulant effect of warfarin (12). Patients receiving warfarin therapy should be monitored and educated about the potential interaction that occurs with warfarin and high-protein, low-carbohydrate diets (12).

Dietary and herbal supplements: Several dietary and herbal supplements can interact with anticoagulants and alter the INR/PT ratio (9). Dietary supplements that affect the INR/PT ratio include arnica, bilberry, butcher's broom, cat's claw, dong quai, feverfew, forskolin, garlic, ginger, ginkgo, ginseng, horse chestnut, inositol hexaphosphate, licorice, melilot (sweet clover), pau d'arco, red clover, St. John's wort, sweet woodruff, turmeric, willow bark, and wheat grass (9). In addition, persons who take vitamin and mineral supplements containing vitamin K should be monitored. Vitamin and mineral supplements that are taken sporadically (9). Vitamin E intakes greater than 1,000 International Units (IU) may increase the risk of excess bleeding. Research suggests that

doses up to 800 IU may be safe for individuals taking warfarin, but the evidence is not conclusive (9). Persons taking or considering taking vitamin E supplements should consult with their physician.

Enteral nutrition: Patients who are receiving enteral nutrition support while on anticoagulant therapy should be monitored closely. Significant vitamin K intake from enteral formulas can antagonize the effect of the anticoagulant drug warfarin and result in treatment failure (6). Most enteral formulations contain modest amounts of vitamin K and provide daily vitamin K intake similar to the average dietary intake from foods (6). Consistent intake of an enteral formulation containing less than 100 mcg of vitamin K per 1,000 kcal is not expected to cause warfarin resistance (6,8). However, warfarin resistance can occur in patients on enteral nutrition support whose intake of vitamin K is substantially low (13). This resistance may occur as a result of warfarin binding to protein contained in the enteral formula; however, this mechanism has not been substantiated by clinical data (13,14). A reasonable approach to treating warfarin resistance associated with a low vitamin K intake is to initiate a trial of holding the enteral nutrition regimen for at least 1 hour before and after the warfarin dose (13,14).

- 1. Harris J. Interaction of dietary factors with oral anticoagulants: review and applications. J Am Diet Assoc. 1995; 95:580-584.
- 2. Levine MN, Raskob G, Landfeld S, Kearon C. Hemorrhagic complications of anticoagulant treatment. *Chest.* 2001; 119 (suppl 1):108S-121S.
- 3. Reference values for nutrition labeling. In: *A Food Labeling Guide*. US Food and Drug Administration Center for Food Safety and Applied Nutrition; 1994 (Editorial revisions, 1999). Available at: www.cfsan.fda.gov/~dms/flg-7a.html. Accessed October 8, 2007.
- 4. Booth SL, Centurelli MA. Vitamin K: a practical guide to the dietary management of patients on warfarin. *Nutr Rev.* 1999; 57(9 pt 1):288-296.
- 5. Coumadin [package insert]. Princeton, NJ: Bristol-Myers Squibb Company; 2006.
- 6. Rollins CJ. Drug-Nutrient interactions. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society for Parenteral and Enteral Nutrition; 2012; page 309.
- Vitamin K intake in micrograms by sex, age, and race/ethnicity: United States, 1988–94. In: *Dietary Intake of Macronutrients, Micronutrients, and Other Dietary Constituents: United States, 1988–94.* National Center for Health Statistics. *Vital Health Stat.* 2002; 11(245):102. Available at: www.cdc.gov/nchs/data/series/sr_11/sr11/245.pdf. Accessed October 7, 2007.
- 8. Schulman S. Care of patients receiving long-term anticoagulant therapy. N Engl J Med. 2003; 349:675-683.
- 9. Important Information To Know When You Are Taking: Warfarin (Coumadin) and Vitamin K. Bethesda, Md: National Institutes of Health Clinical Center; 2012.
- 10. Committee on Safety of Medicine and the Medicines and Healthcare Products Regulatory Agency. Possible interaction between warfarin and cranberry juice. *Curr Probl Pharmacovigilance*. 2003; 29:8.
- 11. Li Z, Seeram NP, Carpenter CL, Thames G, Minutti C, Bowerman S. Cranberry does not affect prothrombin time in male subjects on warfarin. *J Am Diet Assoc.* 2006; 106:2057-2061.
- 12. Beatty SJ, Mehta BH, Rodis JL. Decreased warfarin effect after initiation of high-protein, low-carbohydrate diets. *Ann Pharmacother*. 2005; 39: 744-777.
- 13. Petretich DA. Reversal of osmolite-warfarin interaction by changing warfarin administration time. Clin Pharm. 1990; 9:93. Letter.
- 14. Penrod LE, Allen JB, Cabacungan LR. Warfarin resistance and enteral feedings: two case reports and a supporting in vitro study. *Arch Phys Med Rehabil.* 2001; 82:1270-1273.

BURNS

Discussion

Thermal trauma results in marked hypermetabolism and hypercatabolism. Aggressive nutritional support is required to meet metabolic demands, prevent the depletion of body energy and nitrogen stores, support wound healing, enhance immunity, and improve survival (1-3). Energy requirements increase linearly in proportion to burn size to a maximum of approximately twice the normal levels (1). Factors such as agitation, pain, and heat loss during dressing changes are associated with a large increase in energy expenditure (1).

Approaches

Energy requirements in adults: Many formulas are available to determine energy requirements. Unfortunately, many of these formulas have not been validated for the burn population (1,3). The expert consensus is that indirect calorimetry should be used to evaluate resting energy expenditure (REE), also refered to as resting metabolic rate (RMR), on admission to the hospital and at least once weekly until the patient is stabilized (1,3). Indirect calorimetry should be performed late at night or early in the morning (before daily activities) to obtain a more accurate assessment of RMR (1). In addition, indirect calorimetry is recommended when the patient's condition is complicated by infection, sepsis, poor wound healing, obesity, or ventilator dependency (1). The RMR obtained from indirect calorimetry may need to be multiplied by a factor of 1.3 (or 20% to 30%) to account for activity, physical rehabilitation, wound care, and stress of treatments (1,4,5). This figure provides the total energy expenditure for which the clinician would base the nutrition prescription.

If indirect calorimetry is not available, evidence based guidelines recommend using predictive equations considering age (< 60 years or age, or > 60 years of age) and whether the patient is obese or non-obese. The Academy currently suggests (listed in order of accuracy) Penn State (2003b), Brandi equation, Mifflin St Jeor Equation x 1.5, and Faisey equation for use in calculating the RMR in non-obese adult critically ill patients (6). (See Section II: "Estimation of Energy Requirements".) The Curreri formula has also been suggested specifically for burn patients (3). However, the Curreri equation has not been recently tested for measures of reliability and validity (6). Clinicians should recognize the values obtained from all predictive equations are approximate and should be used only as guidelines in predicting energy requirements in burn patients (1-3).

The clinician should be aware that the practice of adding injury and stress factors has not been validated and may lead to the over estimation of the patient's needs (6). Patient's who are mechanically ventilated, sedated or paralyzed due to severity of injury often have reduced energy needs (3). Chemical neuromuscular paralysis decreases energy requirements of critically ill patients by as much as 30% (3).

As previously mentioned, The Curreri formula is a tool for specifically deriving the energy needs of burn patients (7,8). The equation has been shown to overestimate the patient's nutrition needs, particularly during convalescence (1,3). The Curreri equation appears to be most accurate in assessing energy requirements during the early postburn phase (7-to-10 days postburn), when energy expenditure is at its maximum (7,8). Because the equation has not been validated in recent years, the clinician should consider using multiple equations known to be validated (eg, Brandi equation) and compare averages with Curreri if it is used.

Curreri equation for patients aged 16 to 59 years (7): TEE: 25 kcal x kg actual body weight + (40 kcal x % TBSAB^a) aIf percent TBSAB > 50%, use a maximum value of 50%

Curreri Example: 30 year male weighing 70 kg with burns involving 50% TBSA. TEE: 25 kcal x 70 kg + (40 kcal x 50) = 1750 kcal + 2000 kcal= 3750 kcal as total energy expenditure (9)

Source for example: Spodaryk M, Kobylarz K. The usability of harris-benedict and curreri equations in nutritional management of thermal injuries. *Annals of Burns and Fire Disasters*. 2005;18:118.

Energy requirements in children: Indirect calorimetry, if available, should be used on admission to the hospital and twice weekly thereafter until the patient is healed. The RMR should be multiplied by a factor of 1.3 (or 20% to 30%) to provide total energy needs (1,10).

For less than 30% TBSAB, use the Dietary Reference Intakes (DRI) for energy, per age group, as a starting point to provide adequate energy intake (5). (See "Dietary Reference Intakes" in Section IA) For greater than

30% TBSAB, use the following formulas, where BSA = Body Surface Area and BSAB = Body Surface Area Burned (11):

Galveston infant	0 to 12 months	2,100 kcal/m ² BSA + 1,000 kcal/m ² BSAB
Revised Galveston	1 to 11 years	1,800 kcal/m ² BSA + 1,300 kcal/m ² BSAB
Galveston adolescent	12 to 18 years	$1,500 \text{ kcal/m}^2 \text{ BSA} + 1,500 \text{ kcal/m}^2 \text{ BSAB}$

The Curreri formula, which was proposed to calculate the energy needs of the burned adult, has been modified for pediatric patients by using balance studies of weight in burned children (12). The Curreri junior formula is designed for burns of less than 50% total body surface area. It typically overestimates energy requirements in burns exceeding 50%.

Age 0 to 1 year:	Basal kcal + 15 kcal x % Burn
Age 1 to 3 years:	Basal kcal + 25 kcal x % Burn
Age 3 to 15 years:	Basal kcal + 40 kcal x % Burn

Protein requirements: Protein needs of burn patients are directly related to the size and severity of the burn. The increased protein demand is necessary to promote adequate wound healing and to replace nitrogen losses through wound exudate and urine. Failure to meet heightened protein needs can yield suboptimal clinical results in terms of wound healing and resistance to infection. Infants and children further adapt to inadequate protein intake by curtailing growth of cells, conceivably sacrificing genetic growth potential. Most sources currently suggest the following for adult burn patients using actual weight, unless otherwise specified (1.3):

- Adults with TBSA < 10% 1.2 g/kg/day (1,3)
- Adults with TBSA > 10% 1.5 g to 2.0 g/ kg/day (1, 3)
- Adults with large surface area burns may require higher protein intake of 3.0 to 4.0 g/kg/day (3)
- Adults (with BMI > 30) 2.0g/kg/IBW (3)
- Adults target 120 to 150 Non Protein Calorie:1 g nitrogen (9)

In addition, the following has been suggested in the literature: *Adults* (1,13,14)

<10% TBSAB	1.2 to 1.5 g/kg of actual or ideal body weight ^a
10% to 15% TBSAB	1.5 to 2.0 g/kg of actual or ideal body weight ^a
15% to 35% TBSAB	2.0 to 2.5 g/kg of actual or ideal body weight ^a
>35% TBSAB	23% to 25% of total energy
^a Consider using ideal body weight when an ad	tual weight cannot be evaluated or measured, or in cases of severe obesity in which protein

requirements may be overestimated if the actual body weight is used.

Children (10,15,16)3 to 4 g/kg<1% TBSAB</td>3 to 4 g/kg1% to 10% TBSAB15% of total energy or non-protein calories to nitrogen ratio (NPC:N) of
150:1>10% TBSAB20% of total energy or NPC:N of 100:1

Assessment of Protein and Energy Intake: Nitrogen balance in adult patients using the standard equation of nitrogen intake (g) – urinary urea nitrogen + 4 (for obligatory losses of skin, sweat, epithelial) is a reasonable indicator of adequacy of protein and energy intake as long as normal renal status is maintained and accurate intake and output of nitrogen is collected and analyzed (3). The clinician should realize daily variances that cause protein breakdown such as surgery, infections and sepsis. During the first 7 weeks postburn the adult patient's nitrogen balance results should target an anabolic range +5 to +10 g/day (3). Most likely, aggressive nutrition support (eg, enteral nutrition feedings) will be necessary to achieve this goal. Over time as wound closure is achieved, protein needs can taper and a range of +2 to +4 is acceptable for a wound < 5% open (3).

Calculate nitrogen balance from a 24-hour urine collection analyzed for urea urinary nitrogen (UUN):

Nitrogen balance = Nitrogen In (Intake analysis) - Nitrogen Out (see below)

Nitrogen out can be estimated using the following formula for children (13):

[(24-hour UUN collection \times 1.1^a) + (1 g for stool losses) + (estimated wound nitrogen loss^b)] divided by

6.25 g of protein per gram of nitrogen (9) = estimated nitrogen losses (output) per day

^arepresents obligatory losses (skin, sweat, epithelial)

1 0 5	· ·	
wound nitrogen (N) loss:		≤10% open wound = 0.02 g N/kg actual weight per day
		11% to 30% open wound = 0.05 g N/kg actual weight per day
		≥31% open wound = 0.12 g N/kg actual weight per day

Parenteral Nutrition

Carbohydrate	(3,4,14,17)
Adults	3 to 4 mg/kg per minute parenteral glucose infusion or approximately 50 to 60% of total energy requirements in critically ill burn patients (3,18). Insulin should be used to maintain normoglycemia (3,19-20).
Children	Initiate dextrose at 7 to 8 mg/kg per minute and advance as needed to maximum of 20% dextrose solution.
Infants	Initiate dextrose infusion at 5 mg/kg per minute and advance to 15 mg/kg per minute over a 2-day period.

For all burn patients, carbohydrates should account for approximately 50% of total energy.

Fat (3,16,17)	
Adults	10% to 30% of total energy in critical care with 2 % to 4% as essential fatty acids
	to prevent deficiency (3)
Children >1 year	30% to 40% of total energy
Children <1 year	Up to 50% of total energy

Percent TBSAB	Feeding Approach
<20% (if not complicated by facial injury, inhalation injury, or preburn malnutrition)	High-energy, high-protein oral diet is generally sufficient.
>20%	Nocturnal tube feeding to supplement dietary intake during the day may be adequate to meet needs; use nutrient intake analysis to ensure adequate intake.

If feeding is to be given totally by nutrition support, the enteral route is preferred over total parenteral nutrition (3). Starting an intragastric feeding immediately after the burn injury (6 to 24 hours) has been shown to be safe and effective. Total parenteral nutrition should be reserved for only those patients with prolonged alimentary tract dysfunction. Gastric ileus is common in centrally injured burn patients. For these patients, a transpyloric feeding may be indicated.

Electrolytes	Provide based on serum and urine data and fluid	
	needs.	
Minerals	DRI	
Trace elements	DRI	
Minor burns (<20% TBSAB) in all children and adults (3,17)	One multivitamin daily	
Major burns (> 20% TBSAB) in children younger	One multivitamin daily	
than 3 years (3,17)	Vitamin C 250 mg twice daily	
	Vitamin A 5,000 IU daily	
	Zinc sulfate 110 mg daily	

Micronutrient Requirements (1, 3, 17)

Major burns (>20% TBSAB) in adults and children at	One multivitamin daily
least 3 years old	Vitamin C 500 mg twice daily ^a
	Vitamin A 10,000 IU daily
	Zinc 45-50 mg daily or 220 mg (once daily) ^a
^a Recommended delivery in suspension for tube feeding because oral	vitamin C and zinc in large doses may precipitate nausea or

^aRecommended delivery in suspension for tube feeding because oral vitamin C and zinc in large doses may precipitate nausea or vomiting (3).

- 1. Burns. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: nutritioncaremanual.org. Accessed February 4, 2013.
- 2. Mayes T, Gottschlich MM. Burns and wound healing. In: Gottschlich MM, ed. *The Science and Practice of Nutrition Support: A Case-Based Core Curriculum*. Dubuque, Iowa: American Society of Enteral and Parenteral Nutrition; 2001:338-341.
- 3. Collier BR, Cherry-Bukowiec, Mills ME. Trauma, Surgery, and Burns. In: Mueller CM ed. *The A. S. P. E. N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, MD: American Society of Enteral and Parenteral Nutrition; 2012:392-411.
- 4. Waymack JP, Herndon DN. Nutritional support of the burned patient. World J Surg. 1992;16:80-86.
- 5. Rodriguez DJ. Nutrition in major burn patients: state of the art. Support Line: A Publication of Dietitians in Nutrition Support. 1995;7:1-8.
- 6. Critical Illness Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: http://www.andevidencelibrary.com. Accessed January 16, 2013.
- 7. Curreri PW, Richmond D, Marvin J, Baxter CR. Dietary requirements of patients with major burns. *J Am Diet Assoc.* 1974; 65:415-417.
- 8. Gottschlich MM, Ireton-Jones CS. The Curreri formula: a landmark process for estimating the caloric needs of burn patients. *Nutr Clin Pract.* 2001;16:172-173.
- 9. Spodaryk M, Kobylarz K. The usability of harris-benedict and curreri equations in nutritional management of thermal injuries. Annals of Burns and Fire Disasters. 2005;18:117-121.
- 10. Mayes T, Gottschlich M, Khoury J, Warden GD. Evaluation of predicted and measured energy requirements in burned children. J Am Diet Assoc. 1996;96:24-29.
- 11. Hildreth M, Herndon DN, Desai MH, Broemeling LD. Caloric requirements of patients with burns under one year of age. *J Burn Care Rehabil.* 1993;14:108-112.
- 12. Curreri PW, Richmond D, Marvin J, Baxter CR. Dietary requirements of patients with major burns. J Am Diet Assoc. 1974;65:415-417.
- 13. Hildreth M, Gottschlich M. Nutritional support of the burned patient. In: DN Herndon, ed. *Total Burn Care*. Philadelphia, Pa: WB Saunders; 1996.
- 14. Deitch EA. Nutritional support of the burn patient. *Crit Care Clin.* 1995;11:735-750.
- 15. O'Neil CE, Hutsler D, Hildreth MA. Basic nutritional guidelines for pediatric burn patients. J Burn Care Rehabil. 1989;10:278-284.
- 16. Farrell K, Bradley S. Estimation of nitrogen requirement in patients with burns. J Burn Care Rehabil. 1994;15:174.
- 17. Gottschlich MM, Warden GD. Vitamin supplementation in the patient with burns. J Burn Care Rehabil. 1990;11:257-279.
- 18. A.S.P.E.N. Board of Directors. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *J Parenter Enteral Nutr.* 2002;26(suppl): 88SA-93SA.
- 19. Hansen T, Thiel S, Wouters P, Christiansen JS, Van de Berghe G. Intensive insulin therapy exerts anti-inflammatory effects in critically ill patients, as indicated by circulating mannose-binding lectin and C-reactive protein levels. *J Clin Endocrinol Metab.* 2003;88:1082-1088.
- 20. Van den Berghe G, Wouters PJ, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferninande P, Lauwers P, Bouillon R. Intensive insulin therapy in critically ill patients. *N Engl J Med.* 2001;345:1359-1367.

CANCER

Discussion

A cancer patient's nutritional status and well-being are greatly impacted by the type of cancer and the treatment methods (Table III-4). In turn, nutritional status and overall health affect the patient's ability to tolerate treatment and achieve the desired clinical outcome. To optimize clinical outcomes, patients who are diagnosed with cancer should receive early nutrition intervention with a complete nutritional assessment and a plan of care (1). When patients with colorectal cancer who are undergoing pelvic radiation receive individualized nutrition counseling, they experience improvements in energy and protein intake, nutritional status, and quality of life and reductions in symptoms of anorexia, nausea, vomiting, and diarrhea (Grade II)* (1). Similar findings are seen in patients who are receiving chemotherapy for esophageal cancer, head and neck cancer, lung cancer, or acute leukemia (1). Patients who receive a pretreatment nutrition evaluation and weekly visits during chemoradiation and chemotherapy experience reduced weight loss, improved energy and protein intake, and improved quality of life; these patients may also have fewer unplanned hospitalizations, shorter hospital stays, and improved tolerance to treatments for a variety of cancers (Grade III) (1).

Approaches

The Academy of Nutrition and Dietetics has published evidence-based guidelines that address the nutrition interventions that are used in the management of specific types of cancers including:

- breast cancer
- colorectal cancer
- esophageal cancer
- head and neck cancer
- hematological malignancies
- lung cancer
- pancreatic cancer

Because the evidence is limited (Grade III or IV) for many of the current recommendations, a comprehensive overview is not presented; rather, a summary is provided in the following paragraphs. The clinician, however, can refer to this resource for guidance when determining if specific therapeutic nutrition interventions should be initiated or discontinued (1). Parenteral nutrition support is generally not recommended for patients with the types of cancer listed above because of the risks of metabolic and infectious complications and the limited evidence that parenteral nutrition affects the length of hospital stay or patient survival (Grade III) (1). Enteral nutrition may be used to increase the energy and protein intake and maintain the weight of esophageal cancer patients undergoing chemoradiation therapy (Grade III) (1). In addition, the use of enteral nutrition to increase the energy and protein intake of outpatients who are undergoing intensive radiation therapy (Grade II) (1). Medical food supplements that are used to improve the energy and protein intake of patients who are undergoing radiation therapy for head and neck cancer are associated with fewer treatment interruptions and reduced mucosal damage and may minimize weight loss (Grade II) (1).

Vitamin and mineral supplements, special foods, and alternative health products such as herbal products are commonly used by patients diagnosed with cancer. The following discussion is based on the *Oncology Evidence-Based Nutrition Practice Guideline* from the Academy of Nutrition and Dietetics (1). Limited evidence supports the use of topical honey for the treatment of mouth sores in persons who are receiving radiation for head and neck cancer (Grade III) (1). The limited evidence shows that the topical use of honey has been associated with a decreased incidence of severe mucositis as well as weight gain and fewer treatment interruptions (Grade III) (1). Oral arginine supplements, which are used in an attempt to improve the clinical response, are not recommended prior to neoadjuvant chemotherapy for breast cancer (Grade III) (1). In addition, arginine-enhanced medical food supplements or enteral nutrition is not recommended for head and neck cancer patients, because data have demonstrated no improvements in nutrition-related outcomes or treatment complications (Grade II) (1).

Vitamin E (in the form of 670 to 1,000 mg of alpha tocopherol) has not been shown to promote tolerance or reduce the late effects of radiation in patients with breast cancer; rather, vitamin E may have adverse effects such as nutrient-nutrient interactions, drug-nutrient interactions (eg, anticoagulant and anti-hypertensive medications or herbal supplements), and disease-related complications (Grade III) (1). Vitamin E oral

All rights reserved.

supplements are not recommended for persons with head and neck cancer who are receiving radiation therapy, because these supplements increase the risk of developing a second primary cancer and decrease the survival rate (Grade III) (1). Doses of antioxidants (eg, vitamin C, vitamin E, beta-carotene, and selenium) that are greater than the tolerable upper intake level, which are used in an attempt to improve treatment outcomes, are not recommended for patients who are receiving chemotherapy for advanced non-small cell lung cancer. Multiple, high-dose oral antioxidants do not significantly influence the treatment response, survival rate, survival time, or toxicity in this patient population (Grade III) (1). Supplements of omega-3 fatty acids, which are used in an attempt to improve weight gain, are not recommended for pancreatic cancer patients due to limited data and the potential for drug-nutrient interactions (Grade III) (1). Refer to Table III-5 for suggested nutrition interventions and approaches to common problems experienced by cancer patients as a result of the disease or adjunctive treatments.

Evidence remains limited as to the best methods for calculating enery and protein needs in patients with cancer. In general, the Academy evidence anlaysis library suggests indirect calorimetry is the best method for assessing resting metabolic rate (1). If indirect calorimetry is not available, using the Harris Benedict Equation (HBE) has been suggested as it is one of the few equations that have been studied in this population (1). Using the HBE in lung cancer patients receiving chemotherapy showed the HBE to underestimate energy needs by an average of 12 to 13% (Grade III) (1). Limited evidence also suggests the HBE underestimates RMR in head and neck cancer patients (Grade III) (1). Also refer to Section II: "Estimation of Energy Expenditures". Protein requirements vary depending on type of cancer and adjunctive treatment (1). Limited evidence a significant decrease in weight and lean body mass during treatment indicating these patients may need higher intakes of protein (Grade III) (1). Also refer to the discussion on energy and protein requirements for patients with hematologic malignancies undergoing allogeneic Hematopoietic Cell Transplant (HCT) later in this section.

Treatment	Nutrition-Related Adverse Effects
Chemotherapy	
Corticosteroids (eg, cortisone, hydrocortisone methylprednisolone, prednisone, prednisolone, triamcinolone)	e, Abdominal distention, anorexia, increased appetite, diarrhea, ulcerative esophagitis, gastrointestinal bleeding, hypocalcemia, hyperglycemia or hypoglycemia, hypokalemia, hypertension, muscle- mass loss, nausea, osteoporosis, pancreatitis, sodium and fluid retention, vomiting, weight gain
Hormones/analogs (eg, androgens, estrogens progestins) Immunotherapies (eg, B-cell growth factor, interferon, interleukin)	 Anorexia, anemia, increased appetite, diarrhea, edema, fluid retention, glossitis, nausea, vomiting, weight gain Anorexia, diarrhea, edema, nausea, vomiting, stomatitis, taste perversion, weight loss
General chemotherapeutic agents (eg, alkylating agents, antibiotics, antimetabolites, mitotic inhibitors, radiopharmaceuticals, other cytotoxic agents)	Abdominal discomfort, anorexia, diarrhea, oral and gastrointestinal ulceration, nausea, stomatitis, vomiting (Premedication with antiemetics will sometimes relieve or decrease severity of symptoms.)
Radiation therapy	
Head, neck, chest Abdomen, pelvis	Dysgeusia, dysosmia, dysphagia, esophagus, fibrosis, fistula, hemorrhage, odynophagia, stomatitis, stricture, trismus, xerostomia, tooth decay, tooth loss (Tooth decay and loss can be prevented by an aggressive program of dental hygiene.) Bowel damage, diarrhea, fistulization, malabsorption, nausea, obstruction, stenosis, vomiting
Surgary	
Radical head/neck	Altered appearance, chewing or swallowing difficulty, chronic aspiration, dysgeusia, dysphagia, impaired speech, odynophagia, voice loss
Esophagectomy	Diarrhea, early satiety, gastric stasis, hypochlorhydria, regurgitation, steatorrhea
Gastrectomy	Abdominal bloating and cramping, achlorhydria with
Manual of Clinical Nutrition Management	III-11 Copyright © 2013 Compass Group, Inc.

 Table III-4: Cancer Treatments With Potential to Negatively Affect Nutritional Status

Cancer			
Table III-4: Cancer Treatments With Potential to Negatively Affect Nutritional Status			
Treatment	Nutrition-Related Adverse Effects		
	lack of intrinsic factor, diarrhea, dumping syndrome,		
	early satiety, hypoglycemia, mineral deficiencies, fat		
	malabsorption, fat-soluble vitamin deficiency		
Intestinal resection ^a	Vitamin B ₁₂ deficiency, dehydration, diarrhea, fluid or		
	electrolyte imbalance, hyperoxaluria, malabsorption,		
	mineral depletion, renal stone formation, steatorrhea		
^a Problems that develop are determined by the nature and exter	nt of resection; nutritional intervention must be highly individualized.		

Source: Barrocas A. Cancer. In: White J, ed. *The Role of Nutrition in Chronic Disease Outcome*. Washington, DC: Nutrition Screening Initiative; 1997. Reprinted by permission from the Nutrition Screening Initiative, a project of the American Academy of Family Physicians, the Academy of Nutrition and Dietetics, and the National Council on Aging, and funded in part by a grant from Ross Products division, Abbott

Laboratories.

Problem (Signs and Symptoms)	Nutrition Intervention
Chewing or swallowing difficulty	Modify consistency (See "Full Liquid Blenderized Diet";
(secondary to surgery, radiation therapy)	"Nutrition Management of Dysphagia" in Section IB).
Dryness, soreness, or inflammation of oral	Evaluate effect of medications.
mucosa (secondary to tumor, chemotherapy,	Consume fluids with meals.
radiation therapy)	Avoid acidic foods.
	Avoid very coarse foods that do not soften in the mouth.
	Cut foods into small pieces.
	Moisten dry foods; modify diet consistency.
	Use oral mouth rinses.
	Evaluate use of glutamine (10 g orally three or four times per day) (3).
	Patients applying topical honey experienced a significant reduction in grade 3/4 mucositis with 54% either
	maintaining or gaining weight (Grade III) (1,4).
	Try artificial saliva products.
	For prevention of dental caries, between-meal candies and
	gum should be sugarless.
	Avoid hot foods to reduce the risk of burning the mouth.
	Cold foods may be soothing.
	Avoid alcohol.
	Lidocaine (Xylocaine) can be used to relieve pain before
	eating.
	Use a straw or spoon for consuming liquids.
	Saliva stimulants, such as sugarless candy or gum, may be
	beneficial.
Anorexia and altered taste perception	Determine if other problems, such as pain, fear of vomiting,
(secondary to systemic effects of cancer,	medication, or constipation, could be factors.
radiation therapy to head and neck,	Recommend modifications in diet order as necessary.
chemotherapeutic agents)	Monitor intake.
	Take a nutrition history to identify well-liked foods.
	factors causing anarouic and tasts abnormalities
	Consider the use of pharmacetherapy (og progestational
	agents cannabinoids anabolic agents provinctic agents
	agents, cannabilious, anabolic agents, proxinetic agents,
	Cold foods may be more accentable than warm foods
	Chocolate and fruit-flavored supplements are well accented
	Recommend well-seasoned foods (liberal use of herbs, spices
	flavorings).
	Patient should rinse mouth with tea, ginger ale, or salt water
	before and after eating.
	Use zinc supplementation only if there is a clinically validated
Manual of Clinical Nutrition Management	III-12 Convergent @ 2012 Compass Group Inc
manual of Gimical Natition Management	All rights reserved.

Table III-5 : Suggested Nutrition Interventions (1.2)

Problem (Signs and Symptoms)	Nutrition Intervention
Lower threshold for bitterness (meat rejection,	Use nonmeat sources of protein: eggs, dairy products,
especially beef)	poultry, or vegetable sources; poultry or fish may be better
	tolerated than red meat; fish with a strong aroma may not
	be accepted.
	Eat with plastic utensils rather than metal utensils.
Elevated threshold for sweetness	Add sugar to foods (sweet sauces, marinades).
Early satiety	Eat small, frequent meals with high-energy, nutrient-dense
(secondary to malnutrition, obstruction,	foods (addition of glucose polymers).
pain, effects of decreased secretions and	Drink liquids between meals rather than with meals.
peristalsis, chemotherapeutic effects on	Keep head elevated following a meal; avoid meals at bedtime.
digestive tract)	Low-fat foods may be better tolerated; avoid fatty, greasy
	foods.
	Light exercise is allowed if tolerated.
	Intake may be best at breakfast.
	Keep high-protein, high-energy snacks on hand for hibbling.
	Chew thoroughly and eat slowly.
Nausea and vomiting (associated with	Evaluate effects and timing of medications.
chemotherapy, radiation therapy to	Use antiemetic drugs $\frac{1}{2}$ nour before meals.
abdominal and gastric areas, partial	Take deep breaths, sip a carbonated beverage, or suck on ice
obstruction of the gastrointestinal tract)	Chips. Try a dry diet (liquid between meals).
(Note: Nausea and vemiting are usually over	Avoid cooking odors
within 24-48 hours after chemotherany and	Avoid favorite foods when nauseated to prevent development
24 hours after total body irradiation)	of permanent dislike for such foods
2 i nours alter total body infatiation.j	Eat foods without strong odors
	Cold foods are often better accented than hot foods.
	Vigorous nutritional intervention may reverse atrophy due to
	malnutrition.
	Enteral route may be preferable to the parenteral route, as it
	supplies nutrition directly to mucosal cells.
	Use lactose-free supplements.
	Decrease fiber content.
Steatorrhea and diarrhea (secondary to	Decrease proportion of energy from fat.
thoracic esophageal resection, gastric	Use medium-chain or long-chain triglycerides if diarrhea
resection, cancerous involvement of	related to malabsorption (2).
lymphatics, blind loop syndrome,	Decrease fiber content as needed; however, bulking agents
obstruction of the pancreatic or bile ducts)	and foods high in water-soluble fiber may be helpful if
	diarrhea is secondary to radiation.
	Promote adequate fluid intake.
	Recommend a lactose-controlled diet, if required.
	Evaluate all medications (eg, magnesium-containing
	feature and the second second second supplements
	(especially link tilistic, aloc, cayenne, saw painletto, and ginsong) that can cause diarrhoa (a)
	gillselig) that call cause that filed (2).
	may cause diarrhea if consumed in large amounts
	Prohiotic supplementation (vogurt with live cultures) may be
	appropriate if diarrhea is related to altered microflora from
	antibiotics (2.6).
	Glutamine, a powdered protein supplement, has been
	recommended to help control cancer treatment-related
	diarrhea. Not all studies have demonstrated efficacy, but it
	may be related to the dose (7,8). In general, a dose of 10 g
	three times per day has been recommended (2,7,8).
Protein-losing enteropathy	Recommend a high-protein intake.
(secondary to fistula, disruption of intestinal	Monitor nutritional status.
epithelium or lymphatics)	

Table III-5 : Suggested Nutrition Interventions (1,2)			
Problem (Signs and Symptoms)	Nutrition Intervention		
Weight loss (secondary to increased basal	Provide high-energy, nutrient-dense foods.		
metabolic rate, catabolism, decreased food	Use above strategies to promote food intake.		
intake, decreased absorption)	Consider use of pharmacotherapy (eg, progestational agents,		
Anorexia-cachexia syndrome (9)	cannabinoids, anabolic agents, prokinetic agents,		
	antiserotonergic agents, branched-chain amino acids,		
	melatonin) with anorexia-cachexia syndrome (2).		
	Note: Energy expenditure may be increased or decreased;		
	diseases of long duration are associated with		
	hypermetabolism.		
Constipation	Increase fiber and fluid intake; limit gas-forming foods.		
	Light exercise is advised, if tolerated.		
	Evaluate medications as underlying cause (eg, narcotics).		
Heartburn	See Gastroesophageal Reflux Disease later in this section.		

Bowel obstruction: Patients who have had radiation or surgery to the pelvic area are at risk for bowel obstruction. Symptoms of bowel obstruction include cramping abdominal pain, diarrhea, and constipation. Patients who have a partial bowel obstruction may have thin, pencil-like stools or sloughing of necrotic tissue, which may be mistaken for diarrhea. Patients should not take over-the-counter medications without their physicians' approval (2). Patients should consume a low-fiber diet and reduce their intake of bowel-stimulating foods, such as caffeine and sorbitol (2). Symptoms of complete bowel obstruction include cramping that is often accompanied by nausea and vomiting. Diarrhea may precede the complete cessation of bowel movements. The physician should be contacted immediately.

Hematopoietic Cell Transplant: Patients with hematologic malignancies undergoing allogeneic Hematopoietic Cell Transplant (HCT) have shown to have higher energy and protein needs depending on the phase of the HCT and if complications arise such as graft-versus-host disease (GVHD). Limited evidence indicates that the estimated energy requirements are 30 to 35 kcal per kg per day during the first month post-transplant and may be higher during acute GVHD and/or for patients receiving > 75% of their total daily energy intake by parenteral nutrition (Grade II) (1). Protein needs are also higher than the RDA for these patients. Limited evidence suggests that more than 2.2 g protein per kg may be needed to maintain nitrogen balance (Grade II) (1). Further research is needed to define protein requirements in this population.

In the past, patients undergoing HCT or chemotherapy were often prescribed a neutropenic diet which was based on the premise of limiting foods with high bacteria content (2). Neutropenia, a potentially serious side effect of both HCT and chemotherapy, and major risk for infections can be life threatening in oncology patients. It has been suggested that low bacterial foods and beverages can prevent the occurrence of infections and infection-related deaths in cancer patients receiving chemotherapy or HCT. The evidence supporting this practice is lacking and the actual efficacy of the neutropenic diet remains unknown (2, 10). In one study it was shown that a higher rate of infections was observed in the HCT group of patients who received the neutropenic diet compared to HCT patients receiving a regular diet (10). In addition, there is a wide variability in defining the foods allowed on the diet and inconsistency in applying the diet among institutions (2,10,11). Considering the evidence, the neutropenic diet has been removed from the Morrison Manual of Clinical Nutrition. The evidence now suggests dietitians should advocate and educate patients undergoing HCT or chemotherapy on standard food safety guidelines to reduce risk and exposure to foods, beverages, and food preparation methods that could place them at increased risk for infection and food-borne illness. The dietitian should advocate the selection of low-microbial foods and beverages while encouraging proper washing and handling of fresh fruits and vegetables (2). High-microbial foods such as those unpasteurized or raw (uncooked or undercooked foods) should be avoided until immunity is restored or treatment is complete (12). If graft-versus-host disease (GVHD) symptoms are present it is prudent to avoid lactose, high amounts of fat (fried foods, rich sauces, and rich desserts), and high amounts of fiber (including legumes, nuts, and whole grain cereals with > 3 grams fiber/serving) (12).

Cancer

^{*}The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

- Oncology Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Chicago, Ill: Academy of Nutrition and Dietetics; 2007. Available at: http://www.andevidencelibrary.com. Accessed January 16, 2013. Under revision.
- 2. Oncology. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: nutritioncaremanual.org. Accessed January 16, 2013.
- 3. Coghlin Dickson TM, Wong RM, Offrin RS, Shizuru JA, Johnston LJ, Hu WW, Blume KG, Stockerl-Goldstein KE. Effect of oral glutamine supplementation during bone marrow transplantation. *J Parenter Enteral Nutr.* 2000;24:61-66.
- 4. Biswal B, Zakaria A, Ahmad N. Topical application of honey in the management of radiation mucositis: a preliminary study. *Support Care Cancer.* 2003;11:242-248.
- Ripamonti C, Zecca E, Brunelli C, Fulfaro F, Villa S, Balzarini A, Bombardieri E, De Conno F. A randomized, controlled clinical trial to evaluate the effects of zinc sulfate on cancer patients with taste alterations caused by head and neck irradiation. *Cancer*. 1998;82:1938-1945.
- 6. Del Piano M, Ballare M, Montino F, Orsello M, Garello E, Ferrari P, Masini C, Strozzi G, Sforza F. Clinical experience with probiotics in the elderly on total enteral nutrition. *J Clin Gastroenterol.* 2004;38(6 Suppl):S111-S114.
- Kozelsky T, Meyers G, Sloan J, Shanahan T, Dick S, Moore R, Engeler G, Frank A, McKane T, Urias R, Pilepich M, Novotny P, Martenson J. Phase III double-blind study of glutamine versus placebo for the prevention of acute diarrhea in patients receiving pelvic radiation therapy. J Clin Oncol. 2003;21:1669-1674.
- 8. Savarese D, Savy G, Vahdat L, Wischmeyer P, Corey B. Prevention of chemotherapy and radiation toxicity with glutamine. *Cancer Treat Rev.* 2003;29:501-513.
- 9. Inui A. Cancer anorexia-cachexia syndrome. Current issues in research and management. CA Cancer J Clin. 2002;52:72-91.
- 10. Trifilio S, Helenowski I, Giel M, Gobel B, Pi J, Greenberg D, Mehta J. Questioning the role of a neutropenic diet following hematopoetic stem cell transplantation. *Biol Blood Marrow Transplant.* 2012; 9:1385-1390.
- 11. Moody K, Finlay J, Mancuso C, Charlson M. Feasibility and safety of a pilot randomized trial of infection rate: neutropenic diet versus standard food safety guidelines. *J Pediatr Hematol Oncol.* 2006;28:126-132.
- 12. Low Microbial Nutrition Therapy. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: nutritioncaremanual.org. Accessed February 5, 2013.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Discussion

Chronic obstructive pulmonary disease (COPD) is an incurable condition that results in progressive obstruction and inflammation of the airways. COPD is the umbrella term for chronic bronchitis, emphysema, and a range of lung disorders. COPD results from airway obstruction and reduced expiratory flow (1). As COPD progresses, the work of breathing increases to 10 to 20 times that of a person with normal lung function (2). The main symptoms of COPD include dyspnea, possibly accompanied by wheezing, and a persistent cough with sputum production (2). Other symptoms include physical signs, such as a barrel chest related to hyperinflation of the lungs, and hypoxemia and hypercapnia (2). Severe COPD can lead to cyanosis caused by a lack of oxygen in the blood. In some cases, cyanosis can lead to heart failure as a result of the extra work required by the heart to get blood flow to the lungs (3). Patients with COPD often experience compromised nutrition status caused by inadequate nutritional intake and the inability to meet energy expenditure requirements (2). In addition, fat-free mass and bone mineral density are lower in people with COPD (4). The malnutrition associated with COPD has been termed pulmonary cachexia syndrome (2). Patients with pulmonary cachexia syndrome have a progressive reduction in lean body mass due to factors associated with medical management, including medications, and changes in metabolism and energy intake (2). The prevalence of malnutrition, as indicated by a body mass index (BMI) less than 20 kg/m², may be as high as 30% in persons with COPD, and the risk of COPD-related death doubles with weight loss (5).

The most common cause of COPD is exposure to tobacco smoke. Tobacco smoking accounts for an estimated 80% to 90% of the risk for developing COPD (6-8). Other risk factors are secondhand smoking, air pollution, and occupational exposure. Alpha₁-antitrypsin deficiency, the only known genetic abnormality that causes COPD, accounts for less than 1% of COPD cases in the United States (7). Pulmonary function tests, or spirometry, are used to diagnose COPD (2). Forced vital capacity (FVC) is the amount of air that can be forcibly blown out after full inspiration, and forced expiratory volume in one second (FEV₁) is the amount of air that can be forcibly blown out in one second. Both measurements are used to determine airway obstruction. A ratio of FEV₁ to FVC that is less than 0.70 is a diagnostic indicator of COPD (8).

The major treatment goals for persons with COPD are to maximize functional capacity, prevent secondary medical complications, and improve quality of life (2,8). To achieve these treatment goals, medical management of COPD includes smoking cessation or avoidance of environmental smoke and pollution; pharmacologic therapy (eg, bronchodilators, corticosteroids or steroids, antibiotics, and diuretics); pulmonary rehabilitation through aerobic exercise and upper extremity strength training or oxygen therapy; and maintenance of nutritional status (2,7-9).

Nutrition Assessment and Diagnosis

Malnutrition is associated with the wasting and subsequent weakness of respiratory muscles (2,8). Eight studies of the weight and body composition of persons with COPD were recently reviewed (9). The prevalence of malnutrition (as defined by a BMI <20 kg/m²) was as high as 30%, and the risk of COPD-related death doubled with weight loss (Grade II) (9). Even in the 70% of COPD patients with BMIs greater than 20 kg/m², the fat-free mass index and bone mineral density were lower than in healthy controls (Grade II) (9). Long-term corticosteroid therapy, which compromises immune function, combined with respiratory muscle weakness caused by malnutrition predisposes patients with COPD to respiratory tract infections such as pneumonia. Corticosteroids play an important role in wasting syndromes by inhibiting protein synthesis and promoting protein catabolism (2). The wasting effects of steroids seem to be dose dependent; doses greater than 60 mg/day lead to reduced respiratory muscle strength and delayed recovery of muscle function (2). Patients with COPD who are malnourished may have lower lung function measurements, more dyspnea, and lower nutritional intakes than patients who are not malnourished (Grade II) (9). Lastly, patients with COPD may have more impairment with activities of daily living (Grade II) (9).

A comprehensive nutritional assessment that includes a physical assessment and assessments of energy intake (by using indirect calorimetry), biochemical values, medications, and anthropometrics is needed to identify relevant nutrition diagnoses (2). An evaluation of BMI and muscle mass or muscle strength is a useful indicator of malnutrition in COPD patients (2,9). Clubbing, which is a thickening of the flesh under the toenails and fingernails, is a common physical trait found in patients with COPD. The nail curves downward, similar to the shape of the round part of an upside-down spoon. Although the cause of clubbing is still unknown, it is thought that COPD causes vasodilation in the distal circulation that leads to hypertrophy of the tissue of the

nail beds (2). Another physical sign of COPD, cyanosis, is a blue coloration of the skin and mucous membranes caused by the presence of deoxygenated hemoglobin in blood vessels near the skin surface (2). An assessment of muscle mass (eg, arm circumference) and an evaluation of signs of muscle wasting or atrophy should be performed. Patients who take steroids may have decreased respiratory strength, decreased bone mineral density, increase fracture risk, and hyperglycemia (2). A detailed food and nutrition history focusing on total energy and carbohydrate intake; intake of omega-3 fatty acids, calcium, and vitamin D; and use of medical food or other supplements is relevant in the COPD patient population (2,9). Refer to Morrison Nutrition Practice Guideline – Chronic Obstructive Pulmonary Disease for a review of common associated nutrition diagnoses in the acute care setting (10).

Nutrition Intervention

The primary goals of medical nutrition therapy in the management of COPD are to preserve lean body mass, prevent involuntary weight loss, and maintain nutritional status (2,9). Nutrition intervention strategies should be determined based on the patient's nutrition diagnosis, primary medical diagnosis, coexisting diseases, level of care, and risk factors identified during the comprehensive nutrition assessment. Refer to the Morrison Nutrition Practice Guideline – Chronic Obstructive Pulmonary Disease (10) for detailed nutrition assessment and nutrition intervention strategies for the acute care setting. Nutritional supplementation with medical food supplements increases the energy intake and promotes the weight maintenance of hospitalized patients with malnutrition or compromised nutritional status (Grade II) (9). In the ambulatory care setting, nutritional supplementation may result in increased energy intake, with weight gain more likely when combined with exercise (Grade II) (9). The ideal macronutrient composition of medical food supplements to support lung function has not been validated (9); therefore, the selection of supplements should be based on the patient's taste preference and the adequacy to meet individualized nutritional needs (2,9).

Acute respiratory distress syndrome is a secondary complication of COPD that requires hospitalization (2,8). Mechanical ventilation is the primary management, with the objective to keep the lungs at high volume and prevent airway closure (2). Acute respiratory failure or distress syndrome occurs frequently in patients undergoing complicated surgery or as a result of trauma, septic shock, or multiorgan failure (2). Early nutrition intervention and access for enteral feeding support are recommended to prevent further deterioration of the nutritional status of patients who receive mechanical ventilation. Depending on the functional status of the gastrointestinal tract, parenteral nutrition may be indicated. (Refer to "Enteral Nutrition Support for Adults" in Section IB.)

Energy expenditure: The total daily energy needs of people with COPD are highly variable due to differences in resting energy expenditure and physical activity levels (Grade III) (9). Inflammation present during stable or exacerbated COPD increases the resting energy expenditure (Grade III) (9). One small study of ten patients with severe COPD demonstrated that the measured energy expenditure was 50% higher than the World Health Organization predictive equation and that the total daily expenditure was widely variable, ranging from 26 to 48 kcal/kg (11). The Academy of Nutrition and Dietetics performed a comprehensive review of the literature and concluded that further research on the influences of the thermic effect of food, breathing efficiency, and medications on the energy needs of COPD patients is needed (9). Indirect calorimetry is the best available method for assessing energy expenditure and establishing energy requirements in COPD patients (2.9). If indirect calorimetry cannot be performed, energy requirements should be estimated by predictive equations based on the level of care (2). The energy requirements of most adult COPD patients range from 25 to 35 kcal/kg, depending on weight, coexisting disease processes, and nutritional deficits (2). Patients who are clinically overfed develop hypercapnia due to increased carbon dioxide production. Hypercapnia increases the demands of ventilation, which worsens the respiratory status, delays weaning from mechanical ventilation, or both. Overfeeding, defined as energy intake in excess of metabolic demands, should be avoided (2). Weight loss is recommended for overweight patients with COPD. In these patients, weight loss improves respiratory muscle function and decreases shortness of breath (2).

Protein: Provide enough protein to maintain visceral protein status and meet the demands of metabolic stress. Protein requirements do not increase with COPD (2). Protein increases minute ventilation, oxygen consumption, and ventilatory response to hypoxia and hypercapnia. In patients with Acute Respiratory Distress Syndrome, high levels of protein may cause further fatigue, and protein requirements may need to be temporarily reduced (6).

Carbohydrate and fat: It has been proposed that patients with COPD might benefit from a high-fat, moderate-carbohydrate diet (eg, 40% to 55% carbohydrate, 30% to 40% fat, and 15% to 20% protein) (12). The rationale is that carbohydrate as a fuel substrate increases the respiratory quotient. This quotient

represents gas exchange and is defined as carbon dioxide produced divided by oxygen consumed. A lower respiratory quotient indicates better gas exchange and an easier capacity for a patient to breathe. The type of energy substrate (fat, protein, or carbohydrate) and how the body utilizes the substrates determine the respiratory quotient (2). When oxidized for energy production, protein has a respiratory quotient of 0.8, fat has an respiratory quotient of 0.7, and carbohydrate has an respiratory quotient of 1 (2). The clinical benefits of altering fat-to-carbohydrate ratios in patients with COPD when the energy supplied is appropriate have not been demonstrated (2,9,13). The respiratory quotient can be affected by a number of variables other than substrate utilization (13,14). Current practice emphasizes the provision of measured or estimated energy requirements with the emphasis of preventing overfeeding rather than altering substrate by providing high-fat or low-carbohydrate formulations (13).

Omega-3 fatty acids: A review of studies on the influence of omega-3 fatty acids on airway responsiveness demonstrated inconclusive findings regarding a relationship between overall fish intake and COPD mortality, pulmonary function, and symptoms (Grade III) (9). In one study, dietary fish oil consumption by cigarette smokers provided protective effects against COPD (Grade III) (9,15). Further investigation is required to assess the relationship between omega-3 fatty acids and COPD. Currently, supplementation with fish oil is not recommended (Grade III) (9).

Electrolytes and trace elements: Disturbances of electrolytes are common in critically ill patients with COPD. Patients with cor pulmonale or pulmonary edema may require sodium and fluid restriction. Hypophosphatemia, hypokalemia, hyporkalemia, hypocalcemia, and hypomagnesemia are associated with diminished diaphragmatic function (8). Respiratory function improves with the repletion of these nutrients. Phosphorus deficiency reduces the blood's ability to deliver oxygen to tissues and decreases the contractility of respiratory muscles. Magnesium deficiency compromises respiratory muscle strength. The dietary intake of these key nutrients should be monitored (2). Reduced bone mass, as measured by dual-energy x-ray absorptiometry, has been demonstrated in patients with COPD; this finding provides evidence for nutritional concerns related to bone mineral density, fracture risk, and osteoporosis (Grade II) (9). The prevalence of osteoporosis and/or vertebral fractures ranges from 25% to 60% in COPD patients (Grade II) (9). Patients who are treated with steroids (greater than 1,000 mg/day of inhaled or oral steroids) are at an increased risk; changes in biochemical bone markers, decreased bone mineral density, and increased fracture risk are associated with higher steroid intake (Grade II) (9). Low body weight and low BMI are positively correlated with decreased bone density in patients with COPD (Grade II) (9). Emerging research shows associations between hypercapnia, vitamin D status, and bone mineral density (Grade II) (9). Until further research is available, the dietitian should carefully assess the patient's risk factors (eg, older age, corticosteroid use, low BMI, and smoking) and dietary intake of calcium, phosphorus, and vitamin D. In consultation with the physician, supplementation with these key vitamins and minerals should be considered based on the patient's risk level assessment or evidence of need.

Antioxidants: The effects of antioxidants (flavonoids and vitamins A, D, and E) on the pathogenesis and exacerbation of COPD have recently been reviewed (9). Seven studies found reduced serum or tissue levels of antioxidant vitamins in people with COPD (Grade III) (9). However, studies of supplementation report insignificant effects (Grade III) (9). Ongoing studies are investigating the relationship between nutrients and lung function and COPD (9). Food sources rich in antioxidants, vitamins, and minerals are currently recommended in place of supplementation (2).

Mucus production and dairy consumption: Some patients with COPD perceive increased mucus production after consuming milk and dairy products. However, a narrative review concluded that milk and dairy product consumption does not significantly affect lung function parameters (Grade III) (9). More research is needed on this topic (Grade III) (9).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

- 1. Snider GL. Nosology for our day: its application to chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 2003;167:678-683.
- 2. Chronic obstructive pulmonary disease. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.http://nutritioncaremanual.org. Accessed February 5, 2013.
- 3. Bauldoff GS, Diaz PT. Improving outcomes for COPD patients. Nurse Pract. 2006;31:26-43.

- 4. Vestbo J, Prescott E, Almdal T, Dahl M, Nordestgaard BG, Andersen T, Sorensen TI, Lange P. Body mass, fat-free body mass, and prognosis in patients with chronic obstructive pulmonary disease from a random population sample: findings from the Copenhagen City Heart Study. *Am J Respir Crit Care Med.* 2006;173:79-83.
- 5. Chailleux E, Laaban JP, Veale D. Prognostic value of nutritional depletion in patients with COPD treated by long-term oxygen therapy: data from the ANTADIR observatory. *Chest.* 2003;123:1460-1466.
- 6. American Thoracic Society Board of Directors. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 1995;152:S77-S120.
- 7. Global Initiative for Chronic Obstructive Lung Disease. *Executive Summary: Global Strategy for the Diagnosis, Management, and Prevention of COPD.* Available at: www.goldcopd.com. Accessed January 10, 2009.
- 8. Celli BR, MacNee W. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. *Eur Respir J.* 2004;23:932-946.
- Chronic Obstructive Pulmonary Disease Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.andevidencelibrary.com. Accessed February 5, 2013.
- 10. Morrison Nutrition Practice Guideline Chronic Obstructive Pulmonary Disease. In: Inman-Felton A, Smith KG. *Morrison Nutrition Practice Guidelines*. Atlanta, Ga: Morrison Management Specialists Inc; 2012. Available at: www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition.
- 11. Slinde F, Ellegard L, Gronberg AM, Larsson S, Rossander-Hulthen L. Total energy expenditure in underweight patients with severe chronic obstructive pulmonary disease living at home. *Clin Nutr.* 2003;22:159-165.
- 12. Kuo CD, Shiao GM, Lee JD. The effects of high-fat and high-carbohydrate diet loads on gas exchange and ventilation in COPD patients and normal subjects. *Chest.* 1993;104:189-196.
- 13. Schwartz DB, DiMaria R. Pulmonary and cardiac failure. In: Gottschlich MM, ed. *The A.S.P.E.N. Nutrition Support Core Curriculum: A Case-Based Approach—The Adult Patient*. Silver Spring, MD: American Society of Parenteral and Enteral Nutrition; 2007: 496-501.
- 14. McClave SA, Lowen CC, Kleber MJ, McConnell JW, Jung LY, Goldsmith LJ. Clinical use of the respiratory quotient obtained from indirect calorimetry. *J Parenter Enteral Nutr.* 2003;27:21-26.
- 15. Briton JR, Pavord ID, Richards KA, Knox AJ, Wisniewski AF, Lewis SA, Tattersfield AE, Weiss ST. Dietary antioxidant vitamin intake and lung function in the general population. *Am J Respir Crit Care Med*. 1995;151:1383-1387.

CORTICOSTEROID THERAPY

Discussion

Corticosteroids, in the synthetic forms of natural hormones, are potent anti-inflammatory medications that can be given orally or by injection. They can produce severe side effects, such as hypertension, high blood glucose levels, an increased risk of infection, osteoporosis, fluid retention, decreased ability to heal wounds and fragile skin. Widely used corticosteroid preparations are prednisone, prednisolone, hydrocortisone, methylprednisone, and dexamethasone (1).

Indications

Corticosteroids are used as (1):

- immunosuppressive therapy for organ transplant recipients
- short term management of various inflammatory and allergic disorders: rheumatoid arthritis, collagen disease, dermatologic disease and autoimmune disorders (eg, systemic lupus erythematosus)
- treatment of ulcerative colitis, acute exacerbations of multiple sclerosis, and palliation in some leukemias and lymphomas

Approaches

Dietary interventions may be needed for side effects of corticosteroids.

Table III-6: Dietar	y Interventions	for Side Effects of	of Corticosteroids
---------------------	-----------------	---------------------	--------------------

Problem	Recommendation
Decreased calcium absorption	Ensure calcium intake (dietary or by supplementation) to meet Dietary Reference
Osteroporosis ("glucocorticoid arthritis"), thought to result from decreased intestinal absorption and increased renal	Intake for age.
excretion of calcium	Increase sunshine exposure or dietary vitamin D.
Hyperglycemia (steroid induced glucose intolerance)	Adjust diet accordingly.
	May require insulin or oral glucose lowering medication.
Edema; hypertension due to water retention	May need sodium-restricted diet (2).
Weight gain due to increased appetite	Behavioral strategies for dealing with increased appetite.
	See "Calorie-Controlled Diet for Weight Management", in Section IC.
	Exercise.
Negative nitrogen balance secondary to increased protein catabolism	High-protein diet: 1 to 2 g/kg
	Adequate energy: 30 to 35 kcal/kg

References

1. Corticosteroids. Nursing 2003 Drug Handbook. 23rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins Publishers 2003: 701-721.

2. Escott-Stump S. Nutrition and Diagnosis-Related Care. 5th ed. Baltimore, Md: Williams & Wilkins; 2002: 317.

MONITORING IN DIABETES MELLITUS

Nutrition Evaluation and Monitoring

The following information provides relevant outcomes used in the determination of nutrition diagnosis and evaluation and monitoring of medical nutrition therapy. The comprehensive list provides the suggested monitoring parameters used to assess and evaluate the management and progression of diabetes mellitus (type 1, type 2, or gestational diabetes mellitus [GDM]). The information below provides the objective outcomes that support the rationale for providing medical nutrition therapy and self-management training to persons with diabetes mellitus. The outcome parameters that the dietitian will select to evaluate and monitor are determined by the patient's stage of disease (new diagnosis vs. follow-up), type of diabetes, and presenting signs and symptoms. The dietitian can strengthen his or her role as a participating member of the health care team by understanding the impact and interpretation of these outcomes. When developing the individual patient care plan, the dietitian selectively discusses only those assessment and monitoring outcome parameters that are pertinent to the patient's signs and symptoms or nutrition-related diagnosis.

Laboratory studies ⁽¹⁾ :	• Fasting plasma glucose (plasma glucose values are 10% to 15% higher than whole blood glucose values)
	 Serial blood glucose levels before each meal, before evening snack, 3 AM, postprandial
	 A1C test or A1C, also referred to as glycated hemoglobin (GHb),*
	glycohemoglobin, glycosylated hemoglobin, HbA _{1c} , or HbA ₁
	 Glycated serum protein (GSP; shorter half-life [1 to 2 weeks] than GHb [2 to 3 months]; preferred to GHb when anemia is present)
	• Fasting lipid profile, including high-density lipoprotein (HDL), low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), triglycerides, and total cholesterol Renal function indexes (microalbuminuria, serum creatinine in adults, in children if proteinuria is present)
	• Test for microalbuminuria (eg, timed specimen or urine albumin-to-creatinine ratio (UACR)
	• Glucose in urine (limited use and not recommended for use in GDM),
	• Urine and blood ketone testing (recommended in type 1 diabetes, pregnancy with existing diabetes and GDM)
	• Electrolytes
	• Thyroid function tests (for type 1 diabetes at diagnosis and then every 1-2
	years)
	• C-peptide
	 Immunoglobulin A (IgA), tissue transglutaminase (tTg) antibodies or antiendomysial antibodies (anti-EMA) for persons with type 1 diabetes who
	present with signs or symptoms of celiac disease
Medical-clinical (1):	 Insulin regimen and/or oral agent
	Blood pressure
	Comprehensive medical review
	Review of coexisting medical conditions
	 Current weight, body mass index, weight history, desirable weight (mutually agreed on goal) goal weight, growth and development pattern (children, adolescents) Activity level (currence pattern)
	• Activity level (exercise pattern)
	• Nutrition history and typical lood intake (meal and shack times; percent of kilocalories from protein carbohydrate and fat)
	 Self-monitoring of blood glucose (SMBC) level and pattern (Plasma glucose values)
	are 10% to 15% higher than whole blood glucose values and it is crucial that
	neonle with diabetes know whether their monitor and strins provide whole blood
	or plasma results.)
Social:	Relevant social factors, such as access to health care, employment schedule/school,
	culture, literacy level, family support, financial resources, possibly alcohol or other
	substance abuse, self-monitoring strategies (eg, SMBG records), and previous
	treatment programs, including nutrition and diabetes self-management training
*17-1	

*Values in the reference range are different for HbA_1 vs HbA_{1c} (2).

Diabetes Mellitus

For medical nutrition therapy approaches, see "Medical Nutrition Therapy for Diabetes Mellitus" in Section IC.

Routine Monitoring (outcome assessment parameters) (1):

Body weight

Food records

Blood pressure

Self-monitoring of blood glucose (SMBG) records

Self-monitoring of blood glucose (SMBG) should be carried out three or more times daily for patients using multiple insulin injections or insulin pump therapy (1).

Fasting lipid profile

For most adults with diabetes evaluate annually (1).

Glycated hemoglobin,* also referred to as A1C test

Perform A1C test twice per year in stable patients meeting treatment goals. Assess quarterly in patients whose therapy has changed, or are not meeting goals (1).

Serum creatinine

Measure serum creatinine at least annually in all adults with diabetes regardless of the degree of urine albumin excretion. The serum creatinine should be used to estimate glomerular filtration rate (eGFR) and stage the level of chronic kidney disease (CKD) (1).

Urine (ketones, glucose [except with GDM], protein

Annual screening for microalbuminuria, with random spot urine sample for microalbumin or urine albumin-to-creatinine ratio (UACR) should be initiated annually in type 1 diabetes and type 2 diabetes who have been diagnosed for 5 years or more (1).

*Values in the reference range are different for $HbA_1 vs HbA_{1c}$ (2).

Education and self-management training as appropriate: See "Diabetes Nutrition Management: Meal Planning Approaches" in Section IC: "Medical Nutrition Therapy for Diabetes Mellitus" for discussion of teaching materials to use with various meal planning approaches. Also refer to Morrison Nutrition Practice Guideline – Diabetes Mellitus (Uncontrolled and Complications) (4) and Morrison Nutrition Practice Guideline - Gestational Diabetes Mellitus (5) for acute care; and Type 1 and Type 2 Diabetes Mellitus evidence based nutrition practice guidelines for adults in the Academy's Evidence Analysis Library (6).

References

- 1. American Diabetes Association. Standards of medical care in diabetes-2013. *Diabetes Care*. 20102013;36(suppl 1):11S-66S.
- 2. Santiago J. Lessons from the Diabetes Control and Complications Trial. Diabetes Care. 1993;42:1549.
- 3. American Diabetes Association. Nutrition recommendations and interventions for diabetes. Diabetes Care. 2008;31(suppl 1): 61S-78S.
- 4. Morrison Nutrition Practice Guideline Diabetes Mellitus (Uncontrolled and Complications). In: Inman-Felton A, Smith K, eds. *Morrison Nutrition Practice Guidelines*. Atlanta, Ga: Morrison Management Specialists; 2012. Available at: <u>www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition</u>.
- Morrison Nutrition Practice Guideline Gestational Diabetes Mellitus. In: Inman-Felton A, Smith K, eds. Morrison Nutrition Practice Guidelines. Atlanta, Ga: Morrison Management Specialists; 2012. Available at: www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition.
- 6. Type 1 and Type 2 Diabetes Evidence-Based Nutrition Practice Guideline for Adults. The Academy of Nutrition and Dietetics, 2008. In: The Academy of Nutrition and Dietetics Evidence Analysis Library at <u>http://www.andevidencelibrary.com</u>. Accessed February 5, 2013.

Bibliography

American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013;36(suppl 1): S67-S74. American Diabetes Association. Nephropathy in diabetes: position statement. *Diabetes Care*. 2004;27(suppl 1):79S-83S.

American Diabetes Association. Gestational diabetes mellitus: position statement. *Diabetes Care*. 2004;27(suppl 1):88S-90S.

American Diabetes Association. Hypertension management in adults with diabetes: position statement. *Diabetes Care.* 2004; 27(suppl 1):65S-67S.

DIABETES MELLITUS: CONSIDERATIONS FOR EXERCISE

Discussion

It is becoming increasingly clear that exercise may be a therapeutic tool in a variety of patients with, or at risk for diabetes (1). Before beginning an exercise program, the individual with diabetes should undergo a detailed medical evaluation with appropriate diagnostic studies (1). Some types of activities may be contraindicated for people with hypertension, retinopathy, neuropathy, foot ulcers, and other complications of diabetes mellitus. A detailed discussion of issues related to each type of complication is presented in reference 1. For individuals with type 2 diabetes, exercise in the amount of 90 to 150 minutes a week (both aerobic and resistance/strength training) reduces A1C, improves insulin sensitivity, decreases dyslipidemia, and decreases blood pressure (Grade I)* (2,3). Regular physicial activity also has shown to help persons with type 2 diabetes achieve and maintain weight loss goals (2,3). Physical activity provides a way to create an energy deficit which can facilitate and maintain weight loss. Research has shown the combination of diet, exercise, and behavior modification to be the most effective method for reaching and maintaining weight loss goals (1,2,3).

Alteration in Energy and Nutrient Requirements

For individuals wishing to maintain their weight, increases in activity require increases in caloric intake. For adult men engaging in light activities, 30 kcal/kg body weight may suffice, while those who engage in heavy activity may need 50 kcal/kg. For women, light activity may increase need to 30 kcal/kg, while heavy activity may elevate needs to 44 kcal/kg (4). Protein needs may be increased with physical activities to a level of 1.2 grams protein/kg body weight for both men and women (4). Fluid replacement is also an important consideration (4).

Prevention of Exercise Induced Hypoglycemia

Type 1 Diabetes: Because physical activity may vary considerably from day to day, adjustments in energy intake and insulin dosage may be required to avoid hypoglycemia in individuals with type 1 diabetes. Several strategies may be used to avert hypoglycemia during or after exercise. When exercise is planned, insulin dose should be adjusted to prevent hypoglycemia (1,2). If exercise is not planned, additional carbohydrate may need to be consumed (1,2). Carbohydrate supplementation is based on the blood glucose level before exercise, previous experience with the particular form of exercise, and the individual's insulin regimen (1,2). Moderate intensity exercise increases glucose uptake by 2-3 mg/kg/min above usual requirements. Thus, a 70 kg person would need 8.4 to 12.6 [10 to 15] g of carbohydrate per hour of moderate physical activity (2). More carbohydrate may be needed for higher intensity activities or prolonged exercise (eg, > 60 minutes) (1,2). An individual may be at risk for hypoglycemia up to 24 hours after the exercise bout (1,2). Individuals who prefer to consume carbohydrates to prevent hypoglycemia during exercise should test their blood glucose prior to exercising, and consume an amount of food (15 grams carbohydrate) which will prevent hypoglycemia but not cause hyperglycemia (2). The following general guidelines can be helpful in regulating the glycemic response to exercise in type 1 diabetes (1):

- Avoid exercise if fasting glucose levels are > 250 mg/dL and ketosis is present, and use caution if glucose levels are > 300 mg/dL and no ketosis is present
- Ingest added carbohydrate if pre-exercise glucose levels are < 100 mg/dL

Type 2 Diabetes: Supplemental food before and during exercise is not needed to prevent hypoglycemia and is not recommended, except under conditions of strenuous, prolonged exercise, such as endurance sports (2). For individuals taking sulfonylureas, there is a slightly increased risk of hypoglycemia during exercise, and supplemental energy intake may be required in some cases (2). The need may be determined by glucose self-monitoring. Individuals with type 2 diabetes who use insulin should also monitor their blood glucose levels closely during and after exercise. Several strategies may be used to avert hypoglycemia during or after vigorous, prolonged, or nonhabitual exercise. These involve the consumption of supplemental carbohydrate-containing foods before, during, and after exercise, as well as adjustment of insulin dosage and timing (1,2).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

- 1. Physical activity recommendations. In: Standards of medical care in diabetes-2013. Diabetes Care. 2013;36(suppl 1):24S-25S.
- 2. American Diabetes Association. Nutrition recommendations and interventions for diabetes. *Diabetes Care*. 2008;31 (suppl 1): 61S-78S.

Diabetes Mellitus

- 3. Diabetes Mellitus Type 1 & 2 Evidence-Based Nutrition Practice Guideline. The Academy of Nutrition and Dietetics, 2008. In: The Academy of Nutritin and Dietetics Evidence Analysis Library at <u>http://www.andevidencelibrary.com</u>. Accessed Feburary 5, 2013.
- 4. Delvin JT, Ruderman N. Diabetes and exercise: the risk-benefit profile revisited. In *Handbook of Exercise in Diabetes*. Ruderman N, Devlin JT, Shcneider SH, Krisra A, eds. Alexandria, Va: American Diabetes Association; 2002.

Bibliography

Colberg SR, Sigal RJ, Fernhall Bo, Regensteiner JG, Blissmer BJ, Rubin RR, Chasan-Taber L, Albright AL, Braun B. Exercise and type 2 diabetes: The American College of Sports Medicine and the American Diabetes Association: Joint Position Statement. *Diabetes Care.* 2010;33:e147-e167.

DIABETES MELLITUS: CONSIDERATIONS FOR ACUTE ILLNESS

Monitoring

Achieving stringent glycemic control during concurrent illness (eg, acute illness, trauma, or surgery) may reduce mortality and morbidity in the hospital setting (1). Therefore, targeting glucose control in the hospital setting has the potential for improved mortality, morbidity, and health care outcomes (1).

During acute illness, records should be kept of blood glucose levels and ketone tests as well as weight loss, temperature, insulin dose and time given, and any other medications that were given (1).

Individuals with diabetes must be appropriately monitored for diabetic ketoacidosis (acidosis accompanied by the accumulation of ketone bodies in the tissues and fluids). Although diabetic ketoacidosis occurs most often in people with type 1 diabetes, people with type 2 diabetes can develop ketoacidosis during illness. Ill patients with type 1 diabetes should check urine ketones and blood glucose every 3 to 4 hours, more frequently if the blood glucose level is high or if the patient is pregnant. If patients with type 1 diabetes have blood glucose levels that are higher than 240 mg/dL two times in a row, the urine or blood should be tested for ketones. Blood glucose readings >240 mg/dL and moderate to large amounts of ketones are a danger signal for diabetic ketoacidosis. Patients with diabetic ketoacidosis (DKA) require additional insulin and immediate management of fluids and electrolytes (2). Persons with type 2 diabetes are vulnerable to hyperosmolar hyperglycemic nonketotic state (HHS) (3). This condition presents with a glucose of > 600 mg/dL, serum osmolality > 320 mOsm, serum bicarbonate > 15 mEq/L along with lethargy and possible coma (3). Ketones in the urine and blood are trace or absent because the presence of some insulin inhibits lipolysis. Patients should be immediately managed with intravenous fluids, electrolyte management and insulin (3).

Approaches for the Hospital Setting

Guidelines for patients with type 1 or type 2 diabetes mellitus:

- 1. Contact physician when vomiting or diarrhea continues for 3 to 4 hours (1).
- 2. For insulin-requiring patients or patients who are pregnant, test urine for ketones. Contact physician when test shows a moderate to large amount of ketones (1,2).
- 3. For diabetic critical-care patients, contact physician when blood glucose level remains above 180 mg/dL even after supplemental insulin (as arranged with physician). In critically ill patients insulin therapy should be initiated for treatment of persistant hyperglycemia starting at a threshold of no greater than 180 mg/dL (1). Once insulin therapy is started, a glucose range of 140 to 180 mg/dL is recommended for the majority of critically ill patients (1). Critical-care patients will usually require intravenous insulin protocol that has demonstrated efficacy and safety in achieving desired glucose range without increasing risk for hypglycemia (1).
- 4. For diabetic noncritically ill patients there is no clear evidence for specific blood glucose goals (1). If treated with insulin, the premeal blood glucose levels should generally be < 140 mg/dL with random blood glucose < 180 mg/dL provided these targets can be safely achieved without risking hypoglycemia (1). More stringent targets may be appropriate in stable patients with previous tight control. Less stringent targets may be appropriate in those with severe comorbidities (1).</p>
- 5. Contact physician when signs of ketoacidosis—dehydration, drowsiness, abdominal or chest pain, difficulty breathing, sunken eyes, or fruity breath—are present (2).
- 6. Contact physician when temperature is greater than 100°F or when the patient is unable to take fluids for 3 to 4 hours (1).
- 7. The patient should avoid physical exertion and rest at a comfortable room temperature.

Management

Medication: The patient should take insulin or oral glucose-lowering medication regardless of the ability to eat normal amounts (1). During acute illness, an associated increase in levels of counterregulatory hormones may increase insulin requirements (1). Scheduled prandial insulin doses should be given in relation to meals and should be adjusted according to point-of-care glucose levels. The traditional sliding-scale insulin regimens are ineffective and not recommended (1). For people with type 2 diabetes who normally do not need insulin, the presence of infection may necessitate short-term use of insulin. Records should be kept of blood glucose levels and ketone tests as well as weight loss, temperature, insulin dose and time given, and any other medicines that were given (1). The patient should follow the physician's instructions for changing the insulin or medication regimen.

Diabetes Mellitus

Intake of food and fluid: Ingestion of carbohydrate, especially if the blood glucose level is less than 100 mg/dL, is important during acute illness (1). In adults, ingestion of approximately 150 to 200 g carbohydrate daily (at least 50 g, or three to four carbohydrate choices, every 3 to 4 hours) should be sufficient, along with medication adjustments to keep glucose in the goal range, prevent hypoglycemia, and prevent starvation ketosis (1). (See "Carbohydrate Replacement for Acute Illness, Missed Meals, or Delayed Meals" in Section IC: "Medical Nutrition Therapy for Diabetes Mellitus".) Adequate intake of fluids is also very important.

Electrolytes, especially sodium and potassium, may be lost after vomiting, diarrhea, and diaphoresis. Salty liquids, such as soup and broth, can replenish sodium. Fruit juices, milk, yogurt, ice cream, and cream soups (made with milk) can supply potassium. Caffeinated beverages should be avoided, as these can worsen dehydration.

- 1. American Diabetes Association. Standards of medical care in diabetes-2013. Diabetes Care. 2013;36(suppl 1):11S-66S.
- American Diabetes Association. Hyperglycemic crises in diabetes: position statement. *Diabetes Care*. 2004: 27(suppl 1):S94-S102.
 Type 2 Diabetes. In: *Nutrition Care Manual*; Updated annually. The Academy of Nutrition and Dietetics. Available at:
- www.http://nutritioncaremanual.org. Accessed February 5, 2013.

DIABETES MELLITUS: GASTROINTESTINAL COMPLICATIONS

Gastroparesis

Gastroparesis, also known as diabetic gastropathy, is characterized by an abnormal delay in the emptying of foods, particularly solid foods, from the stomach (1). Gastroparesis occurs when the vagus nerve, which controls the movement of food from the stomach through the digestive tract, is damaged (1). As a result, the muscles of the stomach and intestines do not work normally and food moves slowly or stops moving through the digestive tract (1). The most common cause of gastroparesis is diabetes mellitus, and it is usually attributed to autonomic neuropathy (1). Symptoms associated with gastroparesis include nausea, vomiting undigested food, early satiety, bloating, abdominal pain, and wide fluctuations in blood glucose levels (2).

Approaches: The following approaches are used for the medical management of gastroparesis (1).

- Improve the patient's glycemic control, as hyperglycemia slows the rate of gastric emptying (1,2).
- Make changes to the patient's insulin therapy, such as providing a bolus of insulin after eating instead of before eating (3).
- Consider the use of metoclopramide (Reglan), a dopamine antagonist with a central antiemetic effect. This medication stimulates stomach muscle contractions to assist in stomach emptying and reduces nausea and vomiting (1). Gastrointestinal side effects associated with metoclopramide include diarrhea.
- Consider the use of erythromycin (EryPed), an antibiotic and motilin receptor agonist that increases stomach muscle contractions. Side effects associated with erythromycin are nausea, vomiting, diarrhea, abdominal pain and cramps, increased liver function tests, and jaundice.
- In severe cases, consider jejunostomy enteral feeding or gastric neurostimulators (1).
- Recommend postprandial exercise, such walking, because exercise increases solid-meal gastric emptying in healthy individuals (1).

Because of limited evidence for the nutrition management of gastroparesis, dietary approaches and recommendations are based on professional judgement and logical interpretation of gastric physiology (1). Patients vary tremendously in their abilities to tolerate different types of foods, so recommendations must be individualized. A certain amount of trial and learning is involved. Common food modifications for patients with gastroparesis are designed to speed-up gastric emptying. These modifications include:

- Lower the fiber content of the diet; especially avoid fibrous vegetables (such as oranges and broccoli) and poorly digestible solids with limited gastric motility to reduce the risk of bezoar formation (1). Eat small, frequent, balanced meals (six to seven per day) and avoid large meals.
- Replace solid foods with liquid foods or blenderized meals (4). Puree or grind up solid foods, such as meats, so that they may be better tolerated. Some individuals tolerate solids for the first one to two small meals and then do better with liquids for the remainder of the day.
- Avoid high-fat foods and extra fats such as butter, margarine, gravy, or mayonnaise that are added to foods. Some individuals tolerate high-fat liquids such as whole milk and ice cream. Fat appears to be a potent inhibitor of gastric emptying (1).
- Sit up while eating and for 30 minutes after meals. Walking after meals may enhance stomach emptying.

Nausea and Vomiting

Possible causes of nausea and vomiting include neuropathy (gastroparesis), ketosis, and morning nausea secondary to nocturnal hypoglycemia.

Approaches: The following approaches are used to relieve nausea and vomiting.

- Morning nausea caused by overnight hypoglycemia will usually be relieved by eating breakfast.
- Nausea and vomiting caused by ketosis will improve with metabolic stabilization.
- For patients with nausea and vomiting caused by gastroparesis, an antiemetic drug should be part of the treatment plan.

Constipation

The incidence of constipation is believed to be much higher in individuals with diabetes than in nondiabetic individuals. Constipation in diabetes is related to problems with the autonomic nervous system (5). The *Nutrition Care Manual* from the Academy of Nutrition and Dietetics provides a detailed discussion of constipation in people with diabetes (1).

Diabetes Mellitus

Approaches: The following approaches should be used for diabetic patients with constipation.

- To determine the cause of constipation, assess the patient's fluid intake and the insoluble fiber content of the patient's diet. Also, assess the physical activity level and general physical and mental well-being of the patient.
- Review the patient's list of medications (prescription and nonprescription) for medicines that cause constipation. Note a history of laxative use (frequency and duration).
- Consider recommending a formal evaluation if constipation is potentially secondary to other endocrinologic, neurologic, or gastrointestinal disorders.

If laxatives are recommended, carefully consider which type to recommend. Refer to the paper by Haines for a discussion of treating constipation in patients with diabetes (5).

- 1. Gastroparesis in diabetes mellitus. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: http://www.nutritioncaremanual.org. Accessed February 5, 2013.
- 2. Camilleri M. Diabetic gastroparesis. N Engl J Med. 2007;356:820-829.
- 3. Pharmacotherapy in type 1 and type 2 diabetes. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: http://www.nutritioncaremanual.org. Accessed February 5, 2013.
- 4. National Digestive Disease Information Clearinghouse. Gastroparesis. Available at: http://www.digestive.niddk.nih.gov. Accessed December 12, 2008.
- 5. Haines S. Treating constipation in the patient with diabetes. *Diabetes Educ.* 1995;21:223-232.

DIABETES MELLITUS: ORAL GLUCOSE-LOWERING MEDICATIONS AND INSULIN

Table III-7: Glucose-Lowering Agents (1-3)				
Generic Name	Trade	Classification	Onset (h)	Duration (h)
	Name(s) ^a			
Chlorpropamide ^b	Diabinese	Sulfonylurea	1	24-72
Glyburide ^b	DiaBeta,	Sulfonylurea	1.5	12-24
	Micronase,		1.5	12-24
	Glynase		1.5	24
Glipizide ^b	Glucotrol,	Sulfonylurea	1	12-16
	Glucotrol XL		1	24
Glimepiride ^b	Amaryl	Sulfonylurea	2-3	12-24
Nateglinide ^b	Starlix	Nonsulfonylurea	0.33	1-4
Repaglinide ^b	Prandin	Nonsulfonylurea	0.25-0.5	1
Metformin	Glucophage	Biguanide	1-3	6-12
	Glucophage XR	Biguanide	0.5-3	12-24
Glyburide-Metformin (combination drug)	Glucovance	Sulfonylurea-Biguanide	Half-life, 6.2	24-48
Pioglitazone	Actos	Thiazolidinedione	0.5, peak 2-4	24
Rosiglitazone	Avandia	Thiazolidinedione	1	12-24
Acarbose	Precose	α -glucosidase inhibitor	Immediate	6
Miglitol	Glyset	α -glucosidase inhibitor	Immediate	6
Sitagliptin	Januvia	Dipeptidyl peptidase IV inhibitor	1	24
Exenatide	Byetta	Incretin mimetic	0.5-1	6
Pramlintide	Symlin	Amylinomimetic	0.5	6

Table III-7: Glucose-Lowering Agents (1-3)

^aAll product names are registered trademarks of their respective companies.

^bThese oral hypoglycemic agents have the potential to cause hypoglycemia, since their mode of action increases the release of insulin from the pancreas. Patients who take these oral agents may require snacks if there is more than 4 to 5 hours between meals. A bedtime snack may also be necessary and should be evaluated as part of the individualized meal plan (4).

Discussion

Newer oral or injectable glucose-lowering medications, alone or in combination, provide numerous treatment options for achieving glycemic control (1,2). A combination of two or three medications may be used by persons with type 2 diabetes that is not adequately controlled by nutrition therapy (1,2). If glycemic control is not attained, insulin, either alone or in combination with oral medication, may be necessary. The transition to insulin often begins with long-acting insulin given at bedtime to control fasting glucose levels and glucose-lowering medication given during the day to control daytime and postprandial glucose levels (1,2). Many patients with type 2 diabetes require two or more insulin injections daily to achieve glycemic control (1). Some of these patients will attain better glycemic control with three or four daily insulin injections or with an insulin pump (1-3). An algorithm for the treatment of type 2 diabetes has been established by the American Diabetes Association and European Association for the Study of Diabetes (4). This algorithm recommends the initiation of metformin therapy at diagnosis, along with lifestyle intervention that includes medical nutrition therapy (1,4,5). The algorithm calls for the addition of another oral agent or insulin if the percentage of hemoglobin A1c goal exceeds 7% (1,5).

Glucose-Lowering Medications

Commonly used glucose-lowering medications and their onset and duration are listed in Table III-7. Each class of drug has a different mechanism of action, as described below (1-3).

Insulin secretagogues (sulfonylureas and nonsulfonylureas): These drugs promote insulin secretion by the beta cells of the pancreas. Sulfonylureas are metabolized by the liver and cleared by the kidney (except glimepiride); therefore, caution is needed for patients who have impaired renal function (2). Patients should be informed that missed meals or snacks could cause hypoglycemia (2). In addition, these medications are associated with an increase in appetite and possible weight gain (2). Compared with sulfonylureas, nonsulfonylureas cause less weight gain and have an earlier onset, shorter duration (1 to 6 hours), and a *Manual of Clinical Nutrition Management* **III-29** Copyright © 2013 Compass Group, Inc.

Diabetes Mellitus

reduced risk of hypoglycemia (2). Nonsulfonylureas are an option for patients who have erratic meal schedules and are concerned about weight gain (2).

Insulin sensitizers (biguanides, thiazolidinediones, and α **-glucosidase inhibitors):** These drugs enhance insulin action. To be effective, these drugs require the presence of endogenous or exogenous insulin.

Biguanides provide enhanced insulin sensitivity by inhibiting hepatic gluconeogenesis and, to a lesser extent, glycogenolysis. Metformin, the only biguanide, causes weight loss and does not cause hypoglycemia when it is used as a monotherapy (2). Metformin can cause gastrointestinal side-effects such as abdominal pain, nausea, diarrhea, and metallic taste. Starting with small doses of medication and limiting foods such as cauliflower, cabbage, broccoli, lentils, and legumes may lessen these side effects (2). Patients who have compromised renal function or who are at risk for dehydration or chronic heart failure should be closely monitored for the risk of lactic acidosis associated metformin (2).

Thiazolidinediones enhance insulin sensitivity by increasing the efficiency of the glucose transporters. Thiazolidinediones, particularly pioglitazone, beneficially affect lipids by decreasing triglyceride levels, increasing the high-density lipoprotein cholesterol level, and increasing the particle size of low-density lipoprotein cholesterol (2). The main side effects of thiazolidinediones are weight gain and mild edema; therefore, an energy-controlled diet and sodium modification may be warranted (2).

The α -glucosidase inhibitors inhibit enzymes that digest carbohydrates in the small intestine. This inhibition results in delayed carbohydrate absorption and lowered postprandial glucose responses. The α -glucosidase inhibitors should be taken before carbohydrate-containing meals or snacks and should not be taken when meals are missed (2). These medications should be avoided by persons who have severe renal or hepatic impairment or any gastrointestinal disease. The side effects of α -glucosidase inhibitors include bloating, abdominal cramps, flatulence, and diarrhea (2).

Dipeptidyl peptidase IV inhibitors: These drugs inhibit dipeptidyl peptidase IV, the enzyme that degrades endogenously secreted incretins (2). Because of this mechanism, increased levels of incretin hormones result in increased insulin secretion. The most common side effects are headaches and nasopharyngitis (2). Sitagliptin, the first dipeptidyl peptidase inhibitor IV approved for use as a monotherapy or in combination with a thiazolidinedione or metformin, is metabolized in the liver but excreted in the urine. In patients with renal insufficiency, the dose should be decreased by 50% to 75% (2).

Incretin mimetics: These drugs mimic the glucose-lowering effects of the incretin hormone, a glucagon-like peptide that occurs naturally in the body. Exenatide (Byetta) represents a new class of injectable medications for people with type 2 diabetes who take metformin or sulfonylurea agents. Exenatide is an amino-acid peptide that has many of the glucose-lowering effects of incretin (1,3). The glucose-lowering effects of this drug result from a delay in gastric emptying and enhanced glucose-dependent insulin secretion from the beta cells. But, these effects occur only in the presence of elevated glucose levels and decreased insulin production (3). Exenatide is usually injected twice a day, at breakfast and the evening meal. It can be taken up to 60 minutes before a meal, but taking the medication 30 or more minutes prior to a meal may increase the sense of satiety early in the meal, resulting in decreased energy intake (1,3). Because the mechanism slows gastric emptying, a feeling of fullness may be experienced during meals and can cause nausea or early satiety (2). Exenatide is associated with a degree of weight loss over a 2-year period (3).

Amylinomimetics: These drugs counter the effects of amylin deficiency. Amylin is a hormone secreted with insulin by the pancreatic beta cells in response to food intake. Amylin lowers glucose levels by slowing gastric emptying and decreasing postprandial hepatic glucose release, which is related to a decrease in glucagon production from the pancreatic alpha cells and is dependent on the glucose level (2,3). Pramlintide (Symlin), an injectable drug, is a form of amylin. Pramlintide is approved as an adjunct therapy to insulin therapy in patients who have type 1 or type 2 diabetes and have not achieved optimal glucose control (3). Pramlintide, which must be injected separately from insulin, should be given with each meal or snack that exceeds 250 kcal of energy or 30 g of carbohydrate (3).

Insulin ^a	Onset (h)	Peak (h)	Effective	Maximum
			Duration (h)	Duration (h)
Rapid-Acting:				
Lispro (Humalog)	0.25-0.5	1-2	1-4	4
Aspart (NovoLog)	0.25-0.5	1-2	1-4	4
Glulisine (Apidra)	0.25-0.5	1-2	1-4	4
Short-Acting:				
Regular	0.5-1	2-3	6	6-8
Intermediate-Acting:				
NPH ^b (Humulin N)	2-4	6-10	10-16	12-16
Long-Acting:				
Glargine (Lantus) (3)	1.1	None	24	24+
Detemir (Levemir)	1-2	5-14	14-23	14-23
Mixtures:				
NPH ^b / Regular: 70/30; 50/50	0.5-1	Dual	10-16	14-18
Aspart (NovoLog Mix) 70/30	0.1-0.2	1-3	3-5	5
Lispro (Humalog Mix) 70/30	0.1-0.2	1-3	3-5	5

Table III-8: Human Insulins (1-3)

^aGeneric names are listed first, and representative trade names are listed in parentheses. All product names are registered trademarks of their respective companies.

^bNPH (neutral protamine Hagedorn) is also called isophane insulin suspension.

Insulins

Persons with type 1 diabetes obtain the best glycemic control with replacement insulin that mimics normal insulin action (1). The basal or background insulin dose is the amount of insulin required in the postabsorptive state to restrain endogenous glucose output, primarily from the liver (1). The bolus (mealtime) insulin doses mimic what happens when individuals without diabetes eat the normal physiologic pattern of insulin secretion; the plasma glucose and insulin concentrations increase rapidly, peak in 30 to 60 minutes, and return to basal concentrations within 2 to 3 hours (1).

Insulin glargine (Lantus) is a basal insulin that is injected once a day, often at bedtime (3). However, it can be injected at any time during the 24-hour period if it is given consistently at the chosen time. Glargine cannot be mixed with other insulins. Insulin detemir (Levemir) has a duration of action of approximately 17 hours, and therefore may need to be given twice a day. Detemir does have a slight peak, but the peak is extremely predictable, unlike the sometimes erratic peak of NPH (1,3). Rapid-acting insulins are given at meals, often with the use of an insulin pen. On occasion, rapid-acting insulin may be given after meals, most often for children with unpredictable eating habits or adults with gastroparesis (1). Insulin-pump therapy provides basal rapid-acting or short-acting insulin pumped continuously by a mechanical device in micro amounts through a subcutaneous catheter. In addition, bolus doses are given before meals.

Intensive insulin therapy increases the risk of hypoglycemia and weight gain in persons with type 1 diabetes mellitus (6). Skipped or delayed meals, reduced carbohydrate intake at meals, or increased physical activity without the appropriate insulin adjustments are the major causes of hypoglycemia (6). Alcohol may also induce hypoglycemia and mask symptoms related to hypoglycemia (6). Weight gain occurs with intensive insulin therapy because less energy is lost from glycosuria as glycemic control improves (6). Frequency of hypoglycemia and the extra energy used for its treatment may also contribute to weight gain (6). Strategies to prevent weight gain include more frequent weight checks, a decreased daily energy intake, additional physical activity, and review of the appropriate treatment of hypoglycemia.

- 1. Pharmacotherapy in type 1 and type 2 diabetes. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: http://www.nutritioncaremanual.org. Accessed February 5, 2013.
- 2. Fonseca VA, Kulkarni KD. Management of type 2 diabetes: oral agents, insulin, and injectables. J Am Diet Assoc. 2008;108:S29-S33.
- 3. Stuart N, ed. Medications and diabetes: new helps and old friends. *On the Cutting Edge.* [Diabetes Care and Education newsletter.] 2006; 27:1-32.
- 4. American Diabetes Association. Standards of medical care in diabetes-2013. Diabetes Care. 2013;36(suppl 1):11S-66S.
- 5. Nathan DM, Buse JB, Davidson MB, Heine RJ, Holman RR, Sherwin R, Zinman B. Management of hyperglycemia in type 2 diabetes: a consensus algorithm for the initiation and adjustment of therapy: a consensus statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care*. 2006;29:1963-1972.

^{6.} 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;329:977-986.

DIABETES MELLITUS: FAT REPLACERS AND NUTRITIVE/NONNUTRITIVE SWEETENERS

Discussion

The goal of medical nutrition therapy for diabetes mellitus is to improve overall metabolic outcomes (glucose and lipids levels), provide appropriate energy to maintain desirable body weight, and improve overall health through optimal nutrition (1). The appropriateness of specific modified food products for a given individual should depend on the relative priority of lipid management, control of carbohydrate intake, and the need for weight management (2).

Fat Replacers

Fat replacers are compounds that replicate the functional and sensory properties of fats or mimic one or more characteristics of fat in a food. There are four categories of fat replacers: carbohydrate-based molecules composed of simple or complex carbohydrate, gums, and gels (also carbohydrate-based); protein-based molecules composed of whey protein or milk and egg protein; fat-based molecules, which may include chemical alteration of fatty acids to provide fewer calories or no calories; and a combination, usually consisting of a carbohydrate and protein or carbohydrate and lipids (2,3). Fat replacers vary in energy density. Carbohydrate-based fat replacers provide up to four kcal/g, but because they are often mixed with water they typically provide one to two kcal/g (3). Protein-based replacers provide one to four kcal/g, while fat-based replacers provide zero to nine kcal/g (3). Olestra, a noncaloric fat-based ingredient, was approved by the Food and Drug Administration (FDA) in January 1996 as a fat substitute. Because the molecule used in olestra is too large to be absorbed by the gastrointestinal tract, it adds no energy to food. For a period of time, products that contained olestra, such as potato chips, were required to display a list of possible side effects. These possible side effects included decreased absorption of certain nutrients (vitamins A, D, E, and K and carotenoids), loose stools, and abdominal cramping (3). However, scientific review has led the FDA to conclude that the warning is no longer warranted (3.4).

It is the position of the Academy of Nutrition and Dietetics that, within the context of a healthy dietary pattern, fat substitutes, when used judiciously, may provide some flexibility in dietary planning, although additional research is needed to fully determine their long-term health effects (2,3). Fat replacers that are fibers, such as inulin, lupin fiber, or B-glucan, may increase diet quality in that they add to the intake of dietary fiber (3). In one study of men with diabetes, diets encouraging foods containing fiber-based fat and sugar replacers, together with lifestyle changes, caused a greater increase in high-density lipoprotein cholesterol and larger decreases in hemoglobin A1C, weight, and body mass index than were seen with the standard treatment plan (5). On a population level, replacing one to two g fat/day with fat replacers and fat-modified foods can potentially prevent weight gain and associated chronic disease and assist in promoting healthful eating behaviors (2,3). Although fat replacers are used to replace the fat content of foods, these foods are not always consistently lower in energy content because some of the fat in the foods may be replaced by increasing the sugar content of the food (3). Individuals with diabetes should be used in the context of a well-balanced eating program (3).

Sweeteners

Sweetening agents may be categorized as nutritive (those containing energy) and nonnutritive (those that do not contain energy). Nutritive sweeteners include glucose, galactose, maltose, sucrose, fructose, corn-based sweetner, agave nectar and sugar alcohols (eg, sorbitol, mannitol, xylitol, isomalt, maltitol, lactitol, and starch hydrolysates). Seven nonnutritive sweeteners have been approved by the FDA: acesulfame K, aspartame, Luo han guo extract, neotame, saccharin, , stevia, and sucralose (6).

Nutritive sweeteners: The available evidence from clinical studies demonstrates that sucrose does not increase glycemia any more than isocaloric amounts of starch (2,6). People with diabetes do not need to restrict sucrose and sucrose-containing foods based on a concern about aggravating hyperglycemia. However, if sucrose is included in the food or meal plan, it should be substituted for other carbohydrate sources or, if added, be adequately covered with insulin or other glucose-lowering medication (2).

Fructose produces a smaller rise in plasma glucose than sucrose and other starches (2). Fructose reduces postprandial glycemia when it replaces sucrose or starch in the diabetic diet (2). However, fructose-sweetened products may make a major contribution of energy to the daily intake and cannot be considered

"free" foods. Consumption of large amounts of fructose (15% to 20% of daily energy intake [90th percentile of the usual intake]) has been shown to increase fasting total and low-density lipoprotein cholesterol in subjects with diabetes (2). Therefore, consumption of fructose in large amounts may have adverse effects on plasma lipids. Using fructose as a sweetening agent is not recommended for people with diabetes because of this effect (2).

Sorbitol, mannitol, isomalt, maltitol, lactitol, and starch hydrolysates are considered polyols and are frequently listed on the product's nutrition facts label as "sugar alcohols" (2). Sugar alcohols are used in food as sweeteners and bulking agents. Because sugar alcohols are only partially absorbed from the small intestine, the claim of reduced energy values per gram is allowed (2). In some studies, ingestion of sugar alcohols (approximately 50 g) by healthy and diabetic patients produced a lower glycemic response after ingestion of fructose, sucrose, or glucose (2). Consumption in larger than recommended amounts of 10 to 15 g/day (eg, >30 g or greater of polyols or sugar alcohols including lactitol, isomalt, and xylitol) may result in significant increases in flatulence, borborygmus, colic, defecation frequency, loose watery stools (Grade* III) (2,6). Use of sugar alcohols as sweetners has also shown to reduce the risk of dental caries (2). Kilocalories from sugar alcohols vary but average about two kcal/g on food labels. When calculating carbohydrate content of foods containing sugar alcohols, substraction of half the sugar alcohol grams from total carbohydrate grams is appropriate (2,7).

Nonnutritive sweeteners: Acesulfame-K (Sunette, Sweet One, Sweet & Safe), Aspartame (NutraSweet, Equal, Sugar Twin), Luo han guo extract, neotame, saccharin (Sweet'n Low, Sweet Twin, Necta Twin), Stevia (Truvia, PureVia), sucralose (Splenda), are approved by the FDA for use in the United States (6). The FDA also establishes the acceptable daily intake (ADI) for all food additives. It is defined as "the amount of a food additive that can be safely consumed on a daily basis over a person's lifetime without any adverse effects and includes a 100-fold safety factor." Actual intake by individuals with diabetes for all nonnutritive sweeteners is well below the ADI (2). It is unknown whether use of nonnutritive sweeteners improves long-term glycemic control or assists in weight loss (2). In a limited number of studies, nonnutritive sweetners had no effect on changes in blood lipid profiles and glycemic response in adults with diabetes (Grade 11)* (8). No studies in children were identified (Grade III) (8). Studies to determine the effects of nonnutritive sweeteners during pregnancy and lactation have been conducted in animals. No adverse effects have been reported (6.9). Nonnutritive sweeteners are safe for people with diabetes when consumed within the ADI levels established by the FDA (Grade II) (2.8). Limited research in humans, from peer reviewed journals, supports the safety of nonnutritive sweeteners for the general population. Considering the lack of high quality studies, continuing postmarket surveillance of the safety of non-nutritive sweeteners is prudent (Grade III) (8). Using non-nutritive sweeteners in either a calorie restricted or ad libitum diet will affect overall energy balance only if the nonnutritive sweeteners are substituted for higher calorie food or beverages (Grade II) (8).

Technically, aspartame should not be listed as a noncaloric sweetener since it is equivalent in kilocalories to table sugar. However, aspartame is so sweet (about 160 to 220 times sweeter than sucrose) that the small amount consumed in normal use has virtually no kilocalories to consider (6). Aspartame is a dipeptide formed by the synthetic combination of two amino acids. After it has been metabolized, aspartame converts into phenylalanine, aspartic acid, and methanol. Because aspartame is a phenylalanine source, it should not be consumed by individuals with phenylketonuria (6). Neotame is similar to aspartame but is 30 to 50 times sweeter and does not require special labeling for phenylketonuria since a small percentage (<20%) of the phenylalanine from the ingested neotame may be released into the plasma (6,10).

Use in pregnancy and during breastfeeding: The FDA has approved seven nonnutritive sweeteners for general use: acesulfame K, aspartame, Luo han guo extract, neotame, saccharin, , stevia, and sucralose. The studies of the effects of these sweeteners on the reproductive abilities of women and men, as well as on the developing fetus, have been reviewed and deemed safe by numerous regulatory bodies and expert committees around the world (6). Thus, consumption of these nonnutritive sweetners within the ADI levels is safe during pregnancy and for lactating women (1,6). Although saccharin can cross the placenta and remain in fetal tissues because of slow fetal clearance, there is no evidence that saccharin causes ill effects (11). If a woman chooses to use saccharin during pregnancy, the evidence suggests it is safe (1,6). Aspartame does not cross the placenta at intake levels less than enormous amounts (100 times normal) (12). Use of aspartame within the FDA guidelines appears safe for pregnant women (6). Multigenerational studies of rats that received acesulfame-K, neotame, and sucralose have shown no adverse effects on fertility, number of offspring, birth weight, mortality, and fetal development, and thus both sweeteners are considered safe during pregnancy (13).

Diabetes Mellitus

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

- 1. American Diabetes Association. Standards of medical care in diabetes-2013. Diabetes Care. 2013;36(suppl 1):11S-66S.
- 2. American Diabetes Association. Nutrition recommendations and interventions for diabetes. *Diabetes Care*. 2008;31 (suppl 1): 61S-78S.
- 3. Position of the American Dietetic Association: fat replacers. J Am Diet Assoc. 2005;105:266-275.
- 4. US Food and Drug Administration. FDA Talk Paper. FDA Changes Labeling Requirement for Olestra, 2003. Available at: http://www.fda.gov/bbs/topics/ANSWERS/2003/ANS01245.html. Accessed April 18, 2005.
- 5. Reyna NY, Cano C, Bermudez VJ, Medina MT, Souki AJ, Ambard M, Nunez M, Ferrer MA, Inglett GE. Sweeteners and B-glucans improve metabolic and anthropometrics variables in well-controlled type 2 diabetic patients. *Am J Ther.* 2003;10:438-443.
- 6. Position of the Academy of Nutrition and Dietetics: use of nutritive and nonnutritive sweeteners. *J Am Diet Assoc.* 2012;112:739-758.
- 7. Wheeler M, Franz M, Barrier P. Helpful hints: using the 1995 exchange lists for meal planning. Diabetes Spectrum. 1995;8:325-326.
- 8. Non-nutritive Sweetener Evidence Analysis Project. Chicago, Ill: The Academy of Nutrition and Dietetics, 2008. In: Academy of Nutrition and Dietetics Evidence Analysis Library at http://www.andevidencelibrary.com. Accessed February 6, 2013.
- 9. World Health Organization Expert Committee on Food Additives. *Toxicological Evaluation of Certain Food Additives and Food Contaminants*. Geneva, Switzerland: World Health Organization; 1981, 1983:16:11-27, 18:12-14.
- 10. Stargel WW, Mayew DA, Comer P, Andress SE, Butchko HH. Neotame. In: Nabors LO, ed. *Alternative Sweeteners*. New York, NY: Marcel Dekker; 2001:129-145.
- 11. Pitkin RM, Reynolds WA, Filer LJ, Kling TG. Placental transmission and fetal distribution of saccharin. *Am J Obstet Gynecol*. 1971;111:280-286.
- 12. London RS, Rorick JT Jr. Safety evaluation in pregnancy. In: Tschanz C, Butchko HH, Stargel WW, Kotsonis FN, eds. *The Clinical Evaluation of a Food Additive: Assessment of Aspartame*. New York, NY: Marcel Dekker; 2001:115-123.
- 13. Grice HC, Goldsmith LA. Sucralose: an overview of the toxicity data. Food Chem Toxicol. 2000;38(suppl 2):1S-6S.

DYSPHAGIA

Discussion

Causes of dysphagia are classified as mechanical (trauma or surgical resection of one or more of the organs of swallowing) or paralytic (lesions of the cerebral cortex or lesions of cranial nerves of the brain stem).

•

•

Diseases and conditions in which dysphagia may result include the following:

- Head injury
- Brain tumors
- Multiple sclerosis
- Parkinson's disease
- Huntington's chorea
- Myasthenia gravis
- Dementia

Signs and symptoms of dysphagia include:

- Drooling
- Retention of food in mouth
- Coughing before, during, or after swallowing
- Gurgly voice qualities
- Feeling of a lump in the throat
- Pneumonia
- Aspiration of food or saliva

- Choking
- Squirreling of food in cheeks

Cancer of head or neck

Alzheimer's disease

Auds (oral candidiasis)

Laryngectomy (full or partial)

Cerebral palsy

Stroke

• Anorexia, weight loss, or malnutrition

Amyotrophic lateral sclerosis (ALS)

- Fatigue during meals
- Spiking temperatures
- Dehydration

To define the therapeutic regimen, the multidisciplinary care team performs a comprehensive patient evaluation, which may include assessment of the following:

- Diagnosis, treatments, surgical reports, and medications
- Protein-energy malnutrition and other nutrient deficits
- Energy and protein needs
- Indications for enteral feeding
- Olfactory and gustatory sensation
- Excessive salivation
- Food preferences and dislikes and typical meal pattern, elicited through patient and/or family interviews
- Ability to self-feed
- Dentition
- Visual acuity
- Paralysis or paresis
- Obstruction
- Respiratory status
- Orientation, alertness, comprehension, memory, cooperation, motivation, emotional state, and fear of choking
- Structure and function of all muscle groups involved in chewing and swallowing
- Pain associated with food ingestion or swallowing
- Onset, duration, and severity of swallowing problems
- Food consistencies that can be consumed safely, as determined by clinical evaluation at bedside or by video swallow analysis

Approaches

See "Nutrition Management of Dysphagia" in Section IB.

Other Considerations

• In some patients with muscle weakness, avoid sticky foods, as they can adhere to the roof of the mouth, thus causing fatigue. For example, bread may tend to "ball up in the mouth." If this happens, bread can be torn into small pieces and sprinkled into foods. Note: For some patients, sticky foods (eg, peanut butter, caramels) may be used for exercise to improve tongue control, as recommended
Dysphagia

by the speech-language pathologist (SLP). Concentrated sweets may cause increased salivation. Certain foods (eg, Popsicles) may be used to practice sucking, as prescribed by the SLP.

- Offer a variety of food items to reduce boredom and possible reliance on certain foods.
- Foods should be served either warm (not tepid, but not hot enough to risk burning the mouth, secondary to loss of sensations), or cold (for increased stimulation).
- Offer foods in small amounts so the sight of large quantities of food does not overwhelm the patient. Select nutrient-dense foods.
- Do not use liquids to clear the mouth of food; the patient should drink liquids only after food has been cleared.
- The SLP should determine proper positioning: Some patients with neurologic impairments should sit in an upright position with hips flexed to a 90° angle, back straight, feet flat on the floor, and head bent slightly forward. This allows the tongue to facilitate laryngeal elevation and subsequently protect the airway. An upright position also helps to close the glottis, decreasing risk of aspiration. Patient should be sitting up for 15 to 30 minutes before and after meals to prevent aspiration of any residue potentially remaining in the glottis area.
- Ensure a quiet atmosphere, free of distractions while the patient eats.
- Ensure patient comfort. Some patients may require medication to alleviate painful swallowing.
- It is important that the patient be encouraged to communicate problems and successes with the staff.
- It may be beneficial to stir medications into pudding if the patient has fluid restrictions.
- Allow the patient to set his or her own pace while eating.
- Have the patient or caregiver cleanse the patient's mouth before eating.

Bibliography

Dysphagia. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics: Updated annually. Available at: nutritioncaremanual.org. Accessed September 20, 2010.

Mahan K, Escott-Stump S. Medical nutrition therapy for neurologic disorders (Chapter 41). Krause's Food & Nutrition therapy, 12th Edition. St. Louis, MO: Saunders; 2008:1074-1077.

Phase **Description of Normal Swallowing Dysphagia Signs and Dietary Considerations Symptoms** Oral preparatory phase: food is Use semisolid consistencies 1. Drooling manipulated in the mouth and that form a cohesive bolus: masticated if necessary avoid thin liquids 2. Oral or voluntary phase: the tongue Inability to form bolus Use semisolid consistencies propels the food posteriorly to form a bolus; use moist, well-lubricated foods Pocketing food Avoid foods with more than one texture; position food in sensitive areas; use cold, highly seasoned, flavorful food; try dense foods. Prolonged chewing and Use highly textured foods swallowing latency (eg, diced, cooked vegetables and diced fruit): try dense cohesive foods; avoid sticky or bulky foods; assess ability to control liquids Marked prolongation of the feeding process Use cohesive foods 3. Pharyngeal phase: begins with the Choking or coughing on Use cohesive foods triggering of the swallow reflex liquid and/or solids Elevation and retraction of the Wet and gurgly vocal a. soft palate and complete closure quality Include cohesive semisolid of the velopharyngeal port to Nasal regurgitation foods and thickened prevent material from entering liquids the nasal cavity b. Initiation of pharyngeal Struggle behavior (feel for Use soft solids and thick to movement laryngeal elevation) spoon-thick liquids; avoid Elevation and closure of the sticky and bulky foods that c. larvnx and all three sphincters tend to fall apart (epiglottis, false folds, and true folds) d. Relaxation of the crycopharyngeal sphincter to allow the material to pass from the pharynx to the esophagus 4. Esophageal phase: bolus moves from Indigestion Avoid sticky and dry foods; the esophagus to the stomach try dense food followed by liquids Note: The esophageal phase of the swallow is Reflux not amenable to any kind of therapeutic Use semisolid, moist foods exercise regimen. The videofluoroscopic Sensation of food lodged in that maintain a cohesive study of deglutition generally does not the chest bolus involve examination of the esophagus.

RELATIONSHIP OF DYSPHAGIA TO THE NORMAL SWALLOW

Bibliography

Diet for Dysphagia. In: Manual of Clinical Dietetics. Chicago, Ill: American Dietetic Association; 2000.

O'Gara J. Dietary adjustments and nutritional therapy during treatment for oral-pharyngeal dysphagia. *Dysphagia*. 1990;4:209-212.

ENTERAL NUTRITION: MANAGEMENT OF COMPLICATIONS (1-7)

Problem	Approaches
Diarrhea	 Evaluate medication profile (eg, laxatives, stool softeners, antibiotics, medications containing magnesium, or elixirs containing sorbitol, such as acetaminophen or theophylline). Check for <i>Clostridium difficile</i>. Try soluble fiber-containing formula with guar gum (4), or add soluble fiber to the medication regimen (1) for patients with a low risk for bowl ischemia or bowel dysmotility. Consider antidiarrheal medications such as loperamide, diphenoxylate, or paregoric if <i>C. difficile</i> or other infectious complications are ruled out (1). Use continuous infusion administration. Implement continuous enteral feedings (4). Try isotonic or peptide-based formula (4). Observe proper sanitation. Consider use of prebiotics and probiotics (1).
Nausea, gastroparesis/ delayed gastric emptying (1-6)	 Evaluate medication profile (eg, opiate analgesics or anticholinergics). Consider low-fat or isotonic formula. Reduce the rate of infusion by 20 to 25 mL/hour, or try small bolus feedings of 50 to 100 mL (1,6). Try motility medications such as a prokinetic agent (eg, metoclopramide or erythromycin) (4). Administer formula at room temperature (4). Check and evaluate gastric residual volume prior to each bolus feeding or every 4 hours for continuous feeding (1,4). If GRV consistently ranges 200 to 500 mL consider promotility agent if no contraindications (Grade* II) (2). Check for fecal impaction. Try antiemetic medications if gastric residual volumes are normal.
Hyperglycemia	 Monitor blood glucose levels. The target glucose goal is 100 to 150 mg/dL for nondiabetic critically ill patients (4) and 140 to 180 mg/dL for diabetic critically ill patients (2,7). The target glucose goal for medically stable patients with diabetes is <140 mg/dL with random blood glucose levels <180 mg/dL (7). Most critically ill patients with diabetes require intravenous insulin to achieve the desired glucose range without increasing the risk for hypoglycemia (4,7). More stringent targets may be appropriate in stable patients with severe comorbidities (7). Avoid overfeeding. Evaluate total energy compared to estimated requirements (4). Consult with physician regarding the need for intravenous insulin administration in patients who experience persistent hyperglycemia (4).
Dehydration	Use less concentrated formula. Supplement with additional water as needed.
Clogged tube	Check for proper tube size (viscous formulas should be administered through a >10-French catheter).Flush tube with warm water (usually 20 to 30 mL) regularly and before and after administration of medicines.
Constipation	Monitor hydration status. Add free water. Consider fiber-containing formula with extra free water (>1 mL free water/kcal) (1). Consider adding soluble or insoluble fiber medication with extra free water (1). Increase physical activity if possible. If hydration is adequate and other causes are ruled out, consider a stool softener (docusate sodium or docusate calcium), emollients, or laxative (1).
Essential fatty acid deficiency	Add 5 mL of safflower oil daily (1), or provide at least 4% of energy needs as linoleic acid (1). Change formula to one that contains essential fatty acid.

Problem	Approaches
Overhydration, rapid/excessiv e weight gain	Check fluid input and output. A weight change of 0.2 kg/day or more reflects a change in extracellular fluid volume (1). Consider fluid-restricted formula based on fluid/volume status and medical issues.
Abdominal distention	 Check gastric residual volume. If two consecutive measurements are > 500 mL, decrease or hold feedings until possible causes are assessed (Grade III) (2), or follow organization-specific protocols for gastric residual volume targets and intervention strategies (2-5). Check for constipation, fecal impaction, or obstruction. Place feeding tube in distal duodenum or proximal jejunum (Grade II) (1,2).
Aspiration risk	 Maintain the head of the patient's bed at a 30° to 45° angle during feedings (Grade III) (3-5). Post-pyloric placement of the feeding tube tip at or below the Ligament of Treitz may be beneficial for patients who are supine or heavily sedated (Grade II) (2-5). Consider the use of motility agents such as prokinetic medications (metoclopramide and erythromycin) or narcotic agonists if clinically feasible (5). Implement continuous enteral feedings (4).

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Malone AM, Seres DS, Lord L. Complications of enteral nutrition therapy. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:218-233.
- 2. *Critical Illness Evidence-Based Nutrition Practice Guideline.* Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. January 16, 2013.
- 3. Kattelmann K, Hise M, Russell M, Charney P, Stokes M, Compher C. Preliminary evidence for a medical nutrition therapy protocol: enteral feedings for critically ill patients. *J Am Diet Assoc.* 2006; 106:226-241.
- 4. McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; ASPEN Board of Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr. 2009;33:277-316.
- Bankhead R, Boullata J, Brantley S, Corkins M, Guenter P, Krenitsky J, Lyman B, Metheny NA, Mueller C, Robbins S, Wessel J; ASPEN Board of Directors. Enteral Nutrition Practice Recommendations. *J Parenter Enteral Nutr*. 2009;33:122-167. Also available at: www.eatright.org ("Evidence-Based Practice" link). Accessed September 20, 2010.
- 6. Malone AM, Brewer CK. Monitoring for efficacy, complications and toxicity. In: Rolandelli RH, ed. *Clinical Nutrition: Enteral and Tube Feeding*. 4th ed. Philadelphia, Pa: Saunders; 2005: 276-290.
- 7. Executive summary: standards of medical care in diabetes—2010. Diabetes Care. 2010;33 (suppl 1): S4-S10.

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

Discussion

GERD involves the symptomatic reflux of gastric contents- particularly acid, pepsin, and bile- into the esophagus which results in damage to the esophageal mucosa and leads to esophagitis, regurgitation, and heartburn. Heartburn is often elicited by lying flat or bending over. If the reflux is severe enough, the same positions may evoke actual regurgitation of gastric fluid into the mouth, causing choking, coughing, and possible pulmonary aspiration. Other symptoms may include dysphagia, pain on swallowing and water brash (when the mouth suddenly fills with a large amount of fluid secreted from the salivary glands) (1).

Ordinarily the esophagus is protected from reflux of gastric contents by contraction of the lower esophageal sphincter (LES). In persons with chronic esophageal reflux, the sphincter pressure tends to be lower. Either increased intragastric pressure or decreased LES pressure causes GERD.

Treatment is aimed at modifying the factors that promote gastroesophageal reflux and irritation. Treatment requires a multifactorial approach and is aimed at nutrition and lifestyle modifications, drug therapy, consisting of antacids and hydrogen antagonists and, rarely, surgery.

Management goals are as follows:

- 1. Limit intragastric pressure.
- 2. Avoid substances that decrease the LES.
- 3. Decrease acidity of refluxed material to prevent irritation of the esophagus.

Therapeutic treatment is usually provided in three phases.

Table III-9: Therapeutic Approaches for GERD

Approaches	Rationale
Consume small-volume meals; this may necessitate dividing meals into smaller meals and midmorning and midafternoon snacks, or consuming fluids between meals	
Maintain upright posture during and after eating	Intragastric pressure is increased by mechanical and postural factors
Reduce weight if needed (see "Calorie-Controlled Diet for Weight Management" in Section IC)	Regression of symptoms is likely to accompany weight loss
Avoid tight fitting clothing, frequent bending	
Avoid lying down after eating; consume bedtime snacks or meals at least 2 hours before retiring	
Elevate head of bed at least 6 inches when sleeping	
Limit fat in diet (see "Fat-Controlled Diet" in Section IC)	Intragastric pressure can be reduced if stomach emptying is enhanced; fat decreases LES pressure.
Avoid peppermint and spearmint	These substances decrease LES pressure
 Avoid gastric stimulants: Cigarette smoking Alcohol Chocolate Coffee, regular Caffeine 	Goal: decrease acid production; these substances also decrease LES pressure

Phase 1	
Approaches	Rationale
Limit food constituents that the patient claims cause discomfort; these may include citrus fruits and juices, tomato products, and carbonated beverages	
Treat with antacids containing aluminum hydroxide and Magnesium trisilicate (Gaviscon)	May reduce symptoms by forming a viscous barrier in the stomach that impedes reflux
Dhase 2	

1 11436 2		
Approaches	Rationale	
Medical Approaches	Prescribed to decrease acidity	
• Treat with Histamine H ₂ antagonists		
Cimetidine, rantidine		
Omeprazole (Prilosec)		
Bethanechol (Urecholine)	Increases LES pressure	
Metoclopramide (Reglan)		

Approaches Rationale	
Antireflux surgery Occasional use for the patie medical therapy is not suc severe symptoms and cor Although, significant impo postoperatively, recurren histologic evidence of eso time progresses (1).	nt in which maximal ccessful, and persistent pplications are present. ovement is seen ce of symptoms as well as phagitis is reported as

Reference

1. Draganescu JM, Lipshutz WH. Esophagus, stomach, and intestines. In: Skipper A, ed. *Dietitian's Handbook of Enteral and Parenteral Nutrition*. 2nd ed. Gaithersburg, Md: Aspen Publishers; 1998.

Bibliography

Escott-Stump S. Nutrition and Diagnosis-Related Care. 5th ed. Baltimore, Md: Williams & Wilkins; 2002.

Beyer PL. Medical nutrition therapy for upper gastrointestinal tract disorders. In: Mahan KL, Escott-Stump S, eds. *Krause's Food, Nutrition and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders; 2000: 650-652.

Gastroesophageal reflux disease. In: Manual of Clinical Dietetics. Chicago, Ill: American Dietetic Association; 2000.

Richter JE. A critical review of current medical therapy for gastroesophageal reflux disease. J Clin Gastroenterol. 1986;8:72-80.

HEART FAILURE

Discussion

Heart failure, which is a syndrome characterized by left ventricular dilation or hypertrophy, is caused by cardiac dysfunction that results from myocardial muscle dysfunction or loss (1). Heart failure leads to neurohormonal and circulatory abnormalities that cause fluid retention, shortness of breath, and fatigue (1). The leading causes of heart failure include hypertension, coronary heart disease, and diabetes mellitus (1). Other causes include cardiomyopathy, valvular heart disease, arrhythmias, congenital heart disease, thyroid disease, obesity, alcohol abuse, human immunodeficiency virus, acquired immune deficiency syndrome, and illicit drug use (1,2). Heart failure is common in older adults; the prevalence increases from 2% to 3% at age 65 to more than 80% in persons older than 80 years (2). Heart failure is the most common reason for hospitalization, morbidity, and mortality in the elderly (1,2). Referral to a registered dietitian for medical nutrition therapy is recommended for individuals who have heart failure (Grade II)* (3). A minimum of four visits with a registered dietitian can lead to an improved dietary pattern and quality of life and decreased edema and fatigue (Grade II) (3). Medical nutrition therapy in conjunction with optimal pharmacological management may also reduce hospitalizations (Grade II) (3).

Indications

Heart failure precipitates the onset of sodium retention and edema due to the inability of the body to excrete sodium at a rate in equilibrium with dietary sodium intake (1). The primary objectives in managing the signs and symptoms of heart failure include the achievement of fluid homeostasis by using medications such as loop diuretics and the implementation of dietary interventions to reduce fluid retention and increase the excretion of sodium and water (1).

Signs and symptoms of heart failure include (1):

- difficulty breathing, especially when lying flat in bed or with exertion
- waking up breathless at night
- frequent, dry, hacking cough
- poor tolerance to exercise, or dyspnea on exertion
- sudden weight gain caused by edema or ascites
- frequent urination
- swelling in the lower extremities (especially the ankles)
- fatigue, dizziness, weakness, or fainting
- early satiety, nausea, and abdominal swelling or bloating

The medical diagnosis is verified by echocardiography or the assessment of left ventricular function by measuring the ejection fraction. A laboratory test for B-type natriuretic peptide can indicate a diagnosis of heart failure in the clinical setting (2). Heart failure is classified into one of four stages (with stage IV being the most severe) based on its severity and physiological impact (1).

Medical management of heart failure involves a combination of drugs including diuretics, angiotensinconverting enzyme inhibitors, and beta blockers; dietary modifications; exercise recommendations; and symptom and risk factor management (eg, blood pressure control and lipid management) (1-4). Behavioral compliance to the treatment regimen, especially physical exercise, is correlated with successful outcomes. A multidisciplinary approach to treatment, including medical nutrition therapy, decreases hospital utilization and medical costs and improves the quality of life of elderly persons who have heart failure (1-3).

Adverse health outcomes associated with heart failure are (5):

- reduced tolerance to exercise or activity
- stroke
- peripheral vascular disease
- renal failure

Nutrition Assessment and Diagnosis

Referral to a registered dietitian for medical nutrition therapy is recommended for individuals who have heart failure (Grade II) (3). The initial assessment should include a comprehensive evaluation of nutritional

status that evaluates the patient's weight and/or muscle mass (especially if weight is difficult to assess) by using skinfold measurements or grip strength evaluation (4). A careful analysis of biochemical parameters and nutritional intake should assess the adequacy of energy and protein intake, the sodium and fluid balance, and the adequacy of vitamins and minerals that can be impacted by polypharmacy. Folate, thiamine, vitamin B_{12} , calcium, and magnesium are key nutrients compromised by medications such as diuretics and digoxin (1,3).

Patients who have heart failure require limited sodium and fluid intake and adequate total energy intake to meet their increased energy expenditure. Patients who have severe heart failure may develop malnutrition and cardiac cachexia due to the increased energy requirements associated with the increased lung function needed to produce oxygen (2,3). Compromised nutritional intake is also related to the fatigue and shortness of breath associated with fluid retention (2,3). The mechanism of cardiac cachexia includes hypermetabolism, muscle wasting, altered intake, and functional impairments (4). Involuntary weight loss associated with heart failure may be masked by chronic fluid retention. The use of weight as the only parameter to diagnosis a nutrition-related problem may underestimate the incidence of malnutrition (4,5). Intestinal malabsorption, especially of fat, is common in heart failure as a result of mucosal and intestinal congestion related to edema in the large intestine (6). Patients should be monitored for signs and symptoms of malabsorption as part of the nutrition assessment (4).

Nutrition Intervention

Specific nutrition interventions are effective in managing the signs and symptoms of heart failure (Grade II) (1-3). The primary objective of medical nutrition therapy is to complement pharmacotherapy in maintaining fluid and electrolyte balance while preventing malnutrition and cardiac cachexia. Nutrition interventions should be customized based on the patient's individualized needs and the nutrition diagnoses identified by the comprehensive nutrition assessment. Refer to Morrison Nutrition Practice Guideline – Heart Failure and heart failure evidence-based nutrition practice guidelines as needed (3.7). Hypertension is often associated with heart failure; therefore, dietary and lifestyle management strategies for treating hypertension should be applied to heart failure patients who have hypertension (1-3,8). (Refer to "Hypertension", Section III.)

Energy expenditure: Indirect calorimetry is the best method to determine the energy needs of patients who have heart failure (Grade III) (3). When indirect calorimetry is not possible, the clinician should use predictive equations based on the patient's level of care and adjust for an increased catabolic state (Grade III) (3). A primary goal is to provide enough energy to maintain a reasonable body weight and visceral protein status. In some cases, the basal metabolic needs may be 18% higher than age-matched controls; this increased metabolic need can contribute to malnutrition and cardiac cachexia (9). In obese patients, weight loss improves cardiac output and shortness of breath (4).

Protein: The daily protein intake should be at least 1.37 g/kg in clinically stable depleted patients and 1.12 g/kg in normally nourished patients to preserve their actual body composition or limit the effects of hypercatabolism (Grade III) (3). The literature suggests that patients with heart failure have significantly higher protein needs than patients without heart failure, as measured by negative nitrogen balance (Grade III) (3). The patient's nitrogen balance should be evaluated if the adequacy of protein intake is in question (3,4).

Sodium: Limit sodium to 2,000 mg/day (Grade III) (3) and do not exceed 3,000 mg/day (1). Severe heart failure may warrant a lower sodium intake. (Refer to "Sodium-Controlled Diet" in Section I-F.) Sodium restriction will improve the patient's quality of life and clinical symptoms such as edema and fatigue (Grade III) (3). Urinary sodium levels can be assessed to determine the adherence to a low-sodium diet (2). Severely restricted sodium intake (1,000 mg or less) is discouraged for home use. Dietary restriction at this extreme may be unrealistic, leading to reduced patient compliance and compromised nutritional intake.

Fluid: Fluid requirements are based on the presence of edema, ascites, shortness of breath, and hyponatremia and the frequency of weight fluctuations. Fluid restriction improves these clinical symptoms and the patient's quality of life (Grade II) (3). For patients with heart failure, daily fluid intake should be between 1.4 and 1.9 L (48 to 64 oz), depending on clinical symptoms (Grade III) (3). Fluid should be restricted if serum sodium levels fall below 130 mEq/L (1). Sudden increases in body weight of 3 to 5 lb suggest marked fluid retention (4). (Refer to "Nutrition Management of Fluid Intake and Hydration" in Section IA.)

Alcohol: Alcohol provides limited nutrients and should be avoided or limited to one drink per day for women and two drinks or less per day for men; each drink is the equivalent of 1 oz of alcohol (Grade II) (3). Research

Heart Failure

demonstrates that this level of alcohol consumption is not harmful to heart failure patients (Grade II) (3). Patients who have alcoholism should avoid alcohol and seek alcohol rehabilitation.

Thiamin: Individuals who take more than 80 mg/day of loop diuretics, such as furosemide, have increased urinary excretion of thiamin and may develop clinically significant thiamine deficiency. Thiamin deficiency causes high-output cardiac failure (beriberi) and may exacerbate cardiac function in patients who have heart failure (10). The patient should consume at least the Dietary Reference Intake (DRI) for thiamin through food or supplements (Grade III) (3). Food sources high in thiamin include fortified cereals, bran, bread, and meats.

Folate: Heart failure patients should consume at least the DRI for folate through food or a combination of vitamin B_6 , vitamin B_{12} , and folate supplementation. Folate supplementation given with other vitamins and minerals has beneficial clinical outcomes for patients who have heart failure (Grade II) (3).

Vitamin B₁₂: A multivitamin and mineral supplement containing vitamin B₁₂ or a combination of vitamin B₆, vitamin B₁₂, and folate is recommended for heart failure patients. Vitamin B₁₂ supplementation (200 to 500 mcg/day) provided with other vitamins and minerals has beneficial clinical outcomes for heart failure patients (Grade II) (3).

Minerals: Dietary minerals, including potassium, magnesium, and calcium, may be depleted due to diuretic therapy. Food sources of these minerals include low-fat dairy products, fruits, vegetables, and whole grain products. Heart failure patients should consume at least the DRI for these minerals from food sources or supplements and should place a special emphasis on their magnesium intake (Grade II) (3). Low levels of magnesium may be present in patients who have heart failure and can result in irregular heart rhythms (Grade II) (3). The recommended potassium intake is 2 to 6 g/day, unless the patient has renal impairment or receives a potassium-sparing diuretic such as spironolactone. The need for additional magnesium requirements in heart failure patients is being evaluated (3).

Herbal supplements and over-the-counter dietary supplements: Heart failure patients should be carefully evaluated for their use of herbal supplements and over-the-counter dietary supplements. The most commonly used supplements used by heart failure patients include L-arginine, carnitine, coenzyme Q10, and hawthorn (3). Limited evidence is available regarding clinical heart failure outcomes and the use of these supplements (Grade III; except coenzyme 010, grade II) (3). The risks and harms of taking supplements in different disease states and with various medications should be thoroughly examined (3). For example, patients who take warfarin (Coumadin) should be aware that coenzyme Q10 is chemically similar to vitamin K and can decrease the effectiveness of warfarin (3). Hawthorn should be used cautiously in patients who take beta-blockers and calcium channel blockers, as hawthorn may decrease blood pressure (3). In addition, hawthorn in combination with digoxin can increase digoxin levels and increase the risk of side effects. Hawthorn in combination with nitrates, which increase blood flow, may cause or worsen dizziness and lightheadedness (3). Lastly, ephedra (ma huang), ephedrine, or its metabolites should be avoided due to an increased risk for mortality and morbidity in heart failure patients (1). The clinician should use additional resources in conjunction with evidence analysis documents for information regarding the potential side effects of these supplements (3). Refer to Section II: "Food and Medication Interactions" and "Herb and Medication Interactions" as needed.

Caffeine: Some studies have demonstrated that caffeine increases the heart rate and blood pressure and causes dysrhythmias. More research is needed to assess the effect of caffeine on specific conditions. The effects of caffeine intake on heart failure outcomes have not been studied. In addition, the latest guidelines for blood pressure management do not address limiting caffeine as a recommended modifiable lifestyle factor to reduce blood pressure (8). Because information is limited, it is recommended that heart failure patients use caffeine in moderation and do not exceed 300 mg/day.

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Adams KF, Lindenfield J, Committee Members of the Heart Failure Society of America. Heart Failure Society of America 2006 Comprehensive Heart Failure Practice Guideline. *J Card Fail*. 2006;12:e1-e122.
- 2. Heart failure. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed February 7, 2013.

- 3. *Heart Failure Evidence-Based Nutrition Practice Guideline*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.andevidencelibrary.com. Accessed February 7, 2013.
- 4. Schwartz DB, DiMaria R. Pulmonary and cardiac failure. In: Gottschlich MM, ed. The A.S.P.E.N. Nutrition Support Core Curriculum: A Case-Based Approach—The Adult Patient. Silver Spring, MD: American Society of Parenteral and Enteral Nutrition; 2007: 496-501. [April- the ASPEN 2012 Core Curriculum does no longer address cardiac failure (only Pulmonary)- so retaining this reference to support content]
- 5. Schwengel RH, Gottlieb SS, Fisher ML. Protein-energy malnutrition in patients with ischemic and nonischemic dilated cardiomyopathy and congestive heart failure. *Am J Cardiol.* 1994;73: 908-910.
- 6. Nicol SM, Carroll DL, Homeyer CM, Zamagni CM. The identification of malnutrition in heart failure patients. *Eur J Cardiovasc Nurs*. 2002;1:139-147.
- 7. Morrison Nutrition Practice Guideline Heart Failure. In: Inman-Felton A, Smith KG. *Morrison Nutrition Practice Guidelines*. Atlanta, Ga: Morrison Management Specialists Inc; 2012. Available at:
- www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition
- 8. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT, Roccella EJ, the National High Blood Pressure Education Program Coordinating Committee. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003; 42:1206-1252.
- 9. Poehlman ET, Scheffers J, Gottlieb SS, Fisher ML, Vaitekevicius P. Increased resting metabolic rate in patients with congestive heart failure. *Ann Intern Med.* 1994;121:860-862.
- 10. Brady JA, Rock CL, Horneffer MR. Thiamin status, diuretic medications, and the management of congestive heart failure. *J Am Diet Assoc.* 1995;95:541-544.

HIV INFECTION AND AIDS

Discussion

The human immunodeficiency virus (HIV) is a retrovirus that is transmitted through contact with blood or body fluids (semen, vaginal secretions, or breast milk) from an infected person (1). This virus attacks helper T lymphocytes in the blood, often referred to as CD4⁺ T cells. The systematic destruction of these cells weakens the body's immune system, which increases the host's vulnerability to opportunistic infections. Acquired immunodeficiency syndrome (AIDS) is a specific stage of HIV infection in which the progression of the virus has advanced and the immune system becomes compromised. Due to effective primary prevention and the life-prolonging effects of antiretroviral therapies (ARTs), the number of reported HIV and AIDS cases has leveled off at less than 1% of the global population, according to the 2008 *Report on the Global AIDS Epidemic* (1,2). In the United States, prevalence rates increased between 2003 and 2006 as a result of improved survival and efforts to encourage testing and awareness of HIV infection status (1,3,4). Recent data on HIV infection in the United States indicate that more than two thirds of HIV-infected people are between 25 and 49 years of age, while one quarter of infected people are older than 50 years, and 5% of infected people are between 13 and 24 years of age (5). The most vulnerable groups at risk for infection are ethnic minorities, women, children, and adolescents (1).

Overview and Classification of AIDS

The Centers for Disease Control and Prevention (CDC) defines AIDS by the following criteria (6):

- a positive antibody test for HIV, and
- a CD4 count less than 200/mm³ or less than 14% of the total white blood cell count, or
- a clinical diagnosis of one of the 25 AIDS-defining diseases (7)

In addition to the CDC system, the World Health Organization Clinical Staging and Disease Classification System can be used readily in resource-constrained settings without access to CD4 cell count measurements or other diagnostic and laboratory testing methods (8).

A variety of metabolic and physiologic changes are caused by the effect of HIV on the immune system (1). As a result, persons with HIV infection or AIDS are susceptible to disorders, including opportunistic infections, wasting syndrome, neurological dysfunctions, and gastrointestinal ailments, that challenge their nutritional status and quality of life (9). When AIDS is advanced, it increases the patient's susceptibility to neoplasms such as Kaposi sarcoma and non-Hodgkin lymphoma (9). The CD4 cell count is used as a marker of HIV disease progression. The HIV viral load, which is expressed as copies of HIV ribonucleic acid per milliliter of blood, is used to evaluate the amount of virus in the body (9). The viral load test reflects the level of active virus replication throughout the body and is used for evaluating the efficacy of HIV therapies, predicting the progression of HIV infection to AIDS, and assessing the efficacy of new antiviral medications (9). As the viral load increases, the risk for clinical deterioration also increases (7,9). Although a person at the onset of HIV infection may be asymptomatic, the HIV is not dormant; rather, the virus is actively reproducing. In addition to decreasing CD4 counts, symptoms of HIV disease progression include fatigue, weight loss, body composition changes, and diarrhea. Other signs of disease progression are associated with opportunistic symptoms and include night sweats, mouth sores, rashes, and fever (9). Highly active antiretroviral therapy (HAART), which is a combination therapy consisting of several antiretroviral drugs, is effective in reducing and controlling viral burden and improving immune function. The use of HAART has dramatically reduced the mortality rate of HIV-infected persons and the incidence of AIDS wasting syndrome (10). However, the longterm use of HAART has increased the prevalence of complications such as insulin resistance, diabetes, cardiovascular disease, renal disease, cancers, neurologic disease, and bone density loss (1.9).

Nutritional Implications of HIV and AIDS

The impact of nutrition on HIV and AIDS is significant and multi-factorial (1,9). The HIV infection promotes a vicious cycle, as the infection can cause malnutrition, which exacerbates immune dysfunctions and thus increases the vulnerability to opportunistic infections (1,9). The HIV infection and its treatments may initiate a complex dysregulation of the metabolism associated with changes in nutritional status, such as energy expenditure, lipid metabolism, hormonal balances, and immune function (1,11-14). Even in well-nourished individuals, the hormonal response to infection can lead to changes in hormonal sensitivity (eg, insulin, growth hormone, and sex hormones), tissue catabolism, and a reduction in appetite and food intake (1,9). Poor nutritional status, including both undernutrition and overnutrition, can affect immune function independent of HIV infection (1). Reducing or eliminating malnutrition has the potential to significantly slow the

progression of disease, decrease the disease severity, and improve longevity (1). The treatment and management of HIV with ARTs present many nutritional challenges as the antiretroviral medications can alter body composition and metabolic pathways (1,9). Common nutrition diagnosis and nutrient deficiencies that occur in HIV and AIDS include protein-energy malnutrition, various forms of anemia, and alterations in nutrients such as zinc, iron, selenium, vitamin B_{12} , carbohydrate, and fat (1,15-17). Nutrition is paramount in supporting the health and quality of life of HIV-infected persons (1). The registered dietitian should complete routine comprehensive nutrition assessments to identify nutrition alterations and manage the diverse complications associated with HIV or AIDS and its treatments (1). The most common nutrition complications experienced by persons with HIV or AIDS are comprehensively discussed here.

Weight, Body Composition, and AIDS-related Wasting Syndrome

One of the leading nutrition indicators correlating with survival in HIV is weight status (1). Body mass index (BMI) and adequate body cell mass (BCM) are reliable indicators used to determine acute changes in weight and lean tissue in persons with HIV infection or AIDS (1,9,18). The HIV infection leads to an inflammatory response and challenges to the maintenance of weight and lean tissue stores (9). Even small losses of lean tissue are associated with an increase in morbidity and mortality for HIV-infected persons (9,10,19). With a loss of BCM to a level of 54% of the expected value based on height, death is likely to occur in patients with HIV infection regardless of the presence or absence of infectious complications (20). A major component of the clinical syndrome in HIV infection and AIDS is AIDS-related wasting syndrome (6).

The CDC defines AIDS-related wasting syndrome as an involuntary weight loss of greater than 10% of baseline body weight accompanied by one of the following criteria (6):

- chronic diarrhea (at least two diarrheal stools per day for 30 days or longer), or
- chronic weakness and documented fever for 30 days or longer in the absence of a concurrent illness or condition other than HIV infection that could explain the findings

Although the number of AIDS cases has dramatically decreased since the introduction of ARTs, the wasting syndrome continues to occur in approximately 20% of AIDS cases in the United States (9,10). More recent information suggests that weight loss or wasting syndrome occurs in more than 30% of HIV-infected patients regardless of anti-HIV treatment with a total prevalence rate of nearly 40% in the infected population (9,10). The causes of wasting syndrome and malnutrition in HIV disease are complex and multi-factorial (1,9). Suspected mechanisms of weight and protein losses include compromised food intake caused by anorexia and increased utilization of nutrients associated with inflammatory responses (1). Other causes may include reduced intestinal absorption, which can affect the absorption of carbohydrate and fat, resulting in lactose and fat malabsorption. Diarrhea, a symptom of malabsorption, is associated with the AIDS-related wasting syndrome (6). Diarrhea and malabsorption can lead to nutrient deficiencies and compromised energy intake that adversely affect weight status, immune functions, and other normal body processes (1). Further research to support dietary treatment of diarrhea and malabsorption in HIV and AIDS is needed (Grade II)* (1,21). Research on effective treatments, such as amino acid-based elemental diets, probiotics, pancreatic enzyme therapy, calcium carbonate, glutamine, and the BRAT diet (bananas, rice, applesauce, and toast), as well as the effects of medications is warranted based on a review of current evidence (Grade II) (1,21). For people with HIV infection who have diarrhea/malabsorption, the registered dietitian (RD) should encourage the consumption of soluble fiber, electrolyte-repleting beverages and medium-chain triglycerides (MCT) and decrease the consumption of foods that may exacerbate diarrhea (Grade II) (21). Studies of fat malabsorption reported that consumption of MCT resulted in fewer stools, decreased stool fat and weight and increased fat absorption (Grade II) (21). Both unintentional weight loss and lean tissue loss require strategies to ensure that adequate macronutrients are consumed, absorbed, and assimilated to prevent and reverse weight loss and wasting syndrome (1). Considering the critical importance of weight and maintenance of lean body stores, the routine monitoring of body composition by using anthropometric and other measures is recommended in determining nutritional risk and applying appropriate nutrition interventions (Grade I) (1.21).

Lipodystrophy and Metabolic Disease in HIV and AIDS

With the development of new combinations of medications referred to as ARTs, people with HIV or AIDS are living longer (1-5,9). These medication regimens, however, create nutritional challenges including dyslipidemia, insulin resistance and glucose intolerance, and various types of anemias (1,22,23). Lipodystrophy, the abnormal metabolism and deposition of fat, is a common side effect of ARTs (1). Lipodystrophy includes lipoatrophy, which is the loss of subcutaneous fat, and lipohypertrophy, which is the gain of truncal fat (24). The lipodystrophy-associated alterations in body composition have metabolic consequences that lead to dyslipidemia and insulin resistance (1,24). A higher prevalence of risk factors for cardiovascular disease

Manual of Clinical Nutrition Management

HIV Infection and AIDS

related to ART is likely to require exercise and lipid-lowering medications in addition to dietary modifications (1). A heart-healthy diet and regular exercise reduce blood lipid levels in HIV-positive patients (1,25). Research on several lifestyle modification interventions for the treatment of hyperlipidemia in people with HIV infection found improvements in serum lipid profiles (Grade I) (1,21). Patients with lipodystrophy often require nutrition intervention to support a healthy body weight and reduce their intake of saturated fat, *trans* fatty acids, salt, and dietary cholesterol (1). Patients with HIV and hypertriglyceridemia benefit from increasing fiber intake, limiting simple carbohydrates, and avoiding alcohol (1,25). Routine anthropometric measures, blood lipid levels, and blood pressure monitoring should be used to evaluate and monitor lipodystrophy in HIV-infected patients. Applying practice guidelines for cardiovascular disease risk-factor management is the current recommended approach for treatment (1).

Abnormal glucose intolerance has been associated with HIV and AIDS medication therapies (1,26). Patients with insulin resistance may benefit from diabetes education programs, which provide strategies for regulating blood glucose levels through diet and exercise (1). The treatment of insulin resistance with oral antidiabetic medications has been explored with mixed results (1). Metformin and glitazones are being investigated for their potential to improve insulin sensitivity, but their effect on peripheral subcutaneous fat losses in patients receiving ART is still being researched (27-29). In addition to nutrition therapy strategies, medication support may be indicated to help reduce insulin resistance and increase lean body mass (LBM) (1).

Nutrition Assessment and Diagnosis

All patients who are newly diagnosed with HIV infection should receive a comprehensive nutrition assessment. Early referral of HIV-infected patients to medical nutrition therapy can improve their nutritional status and outcomes (1). A review of evidence has shown that medical nutrition therapy (in the form of an increased number of nutrition counseling sessions) prevents progressive weight loss, improves cardiovascular risk indexes, and improves energy intake and other symptoms (Grade I) (1). Studies have also reported improved outcomes related to weight gain, CD4 count, and quality of life with nutrition counseling (Grade I) (1). Symptoms that may affect nutritional status and that are used as indicators for identifying nutrition diagnoses include nausea, vomiting, diarrhea, anorexia, pain, chewing or swallowing difficulties, taste changes, and weight changes (1.9). Providing specific nutrition intervention strategies to resolve the nutrition diagnoses and support patients through these challenges is an important part of medical nutrition therapy (1.9). The following types of nutrition assessment data should be collected and used to determine nutrition diagnoses, nutrition interventions, and counseling strategies.

Biochemical assessment: The levels of serum proteins, fasting lipids (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides), fasting glucose, and micronutrients should be routinely evaluated due to metabolic and immune abnormalities associated with HIV and AIDS (1,9). Altered levels of plasma proteins, micronutrients, and other nutrition-related markers have been documented early in the disease process and are associated with increased risk of mortality in HIV infection (1). Alterations in zinc, iron, selenium, vitamin B₁₂, carbohydrate, and fat have been reported in symptomatic and asymptomatic disease states (1,30,31). Indicators of disease complications and prognosis include nutrition-related laboratory values such as albumin, transthyretin, hemoglobin, hematocrit, creatinine, urea nitrogen, transferrin, glucose, vitamin B₁₂, and Creactive protein (1,32,33). Serum iron, total iron-binding capacity, folate, and vitamin B₁₂ are measured to distinguish types of anemias, including anemia of chronic disease or anemia related to medication therapy (1,34). Serum lipids in men and women and levels of total and free testosterone in men should be monitored regularly for changes indicative of lipodystrophy and decreases in LBM (1). Alterations in nutrition-related laboratory values may reflect inflammatory responses rather than purely nutritional compromise (1). Levels of zinc and albumin, which are both acute-phase reactants, may fall rapidly during the physical stress of infection and quickly increase when the infection is resolved (1). Therefore, biochemical values should be used in conjunction with other nutrition assessment parameters, such as weight, body composition, and nutrient intake (1).

Anthropometry: Routine anthropometric measures, including height, weight, BMI, waist-to-hip ratio, waist circumference, hip circumference, and other circumference measures, should be used to evaluate and monitor risk for wasting syndrome and lipodystrophy (1,9). Body composition measures such as skin folds and BCM, a component of LBM composed of highly functional protein stores (muscles and organs), are also important to monitor (1,9). In a review of 27 studies evaluating the assessment of body composition in persons with HIV infection, the majority of studies reported that fat-free mass and fat mass are generally lower in people (men, women, children, and adolescents) with HIV infection (Grade I) (1,21). Assessment of BCM can detect early changes in LBM and alterations in fat patterns and potential muscle wasting that may not be reflected by weight change or weight records (1,9). Body composition, particularly BCM, can be evaluated by bioelectric impedance analysis,

bioimpedance spectroscopy, skinfold thickness measurements, and dual energy x-ray absorptiometry (Grade II) (1,21). Further research is needed to determine the optimal methodology for body composition measurement in women, children, different ethnic groups, and patients with lipodystrophy (Grade II) (1,21). Results of bioelectrical impedance analysis may vary with the prediction equation used and the equipment manufacturer (1). Skinfold thickness measurements may also vary with the number of sites measured and the prediction equation used (1). Refer to Table III-10.

Table III-10:	Evaluation Criteria for	Weight and Body C	omposition	Changes in HIV	and AIDS (9)
Anthrono	notria	Crit	orio for Evol	untion	

Antinopometric	CITETIA IOI EVALUATION		
Measure			
BMI	Normal BMI is 18.5 to 24.9 kg/m ²		
	<18.5 kg/m ² suggests high risk for morbidity, mortality, and the development or		
	presence of wasting or lipoatrophy		
	>24.5 kg/m ² suggests potential for obesity-related diseases and central fat		
	accumulation		
BCM	Ideal BCM is 100%		
	<95% suggests wasting and associated complications of reduced body functions		
	related to muscle degeneration and mass (eg, ability to sit, swallow, and breathe)		
	and reflects changes in hormonal stasis		
	<55% is associated with the timing of death (9,20)		
Weight Change	>5% unintentional weight loss is associated with increased risk of morbidity and		
	mortality		
	>5% unintentional weight gain is associated with increased risk for central fat		
	accumulation		

Adapted with permission from the Academy of Nutrition and Dietetics from: Nutrition-focused physical findings in HIV/AIDS. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed November 15, 2010.

Energy requirements: The resting energy expenditure (REE) is increased with HIV infection, and this increase may contribute to weight loss. Numerous studies have found that HIV-infected adults have greater REE as compared to healthy controls (1,21,35). However, the total energy expenditure was similar to that of control subjects (Grade II) (1,21). Higher REE has been correlated with fat-free mass, but it has not been consistently correlated with weight or disease status." (1,21,36). Factors related to energy needs in people with HIV infection include stage of disease, opportunistic infections and co-morbidities, inflammation, and effects of medications (Grade II) (1,21). Alterations in endocrine function and reduction of energy intake are associated with wasting (37). The impact of ARTs on energy requirements is related to the patient's response to treatment. Successful treatment with ART decreases REE and promotes weight gain, whereas a lack of response to treatment is associated with wasting (1,38,39). Further research is needed to determine the exact energy requirements of patients with HIV or AIDS (Grade II) (1,21). The REE can be estimated by using predictive formulas and considering additional energy needs associated with fever, infection, diarrhea or malabsorption, weight loss or loss of BCM, and physical activity (1,9). The energy requirements of patients who receive ART and patients who have lipodystrophy, glucose intolerance, or obesity may need to be decreased (1,9). (Refer to Section II: "Estimation of Energy Expenditures".)

Protein requirements: Protein is essential for the maintenance of BCM and normal body functions, including immunity (1,40). Although increased protein intake may have beneficial effects (eg, the maintenance of BCM) for individuals with HIV; the specific protein requirements, protein turnover, and the effects of increased protein intake in persons with HIV have not been adequately studied (Grade III) (1,21). Protein requirements should be based on the disease stage, BCM, nitrogen balance studies, and physical activity level (1,9). Coexisting complications to HIV or AIDS should also be considered; these complications include malabsorption, infection, wasting syndrome, impaired renal function, and impaired hepatic function.

Fat requirements: High-fat, low-fiber diets are fairly common in both the general population and in HIVinfected persons (1). Studies have found that HIV-infected people generally consume diets that are high in total fat, saturated fat, and cholesterol (Grade II) (1,21). Evidence supports a relationship between diets that are high in saturated fat and total fat and hyperlipidemia, particularly hypertriglyceridemia (1). Studies indicate that diets that are low in saturated fat and total fat and that include omega-3 fatty acids result in reduced triglyceride levels, increased high-density lipoprotein cholesterol levels, and lower risk of lipodystrophy (Grade II) (1,21,25). Although the

HIV Infection and AIDS

type of fat consumed may be related to lipodystrophy, further research is needed to examine this possibility (Grade II) (1,21). Omega-3 fatty acid supplementation may affect several types of metabolic modulations, but additional research is needed to determine its role in the management of hypertriglyceridemia (Grade II) (1). Research has shown that lifestyle modifications adopted by HIV-infected persons with hyperlipidemia result in improved serum lipid profiles (Grade I) (1,21). Because of the increased prevalence of lipodystrophy in HIV patients, especially patients who receive ART, the National Cholesterol Education Panel recommends comprehensive lifestyle modifications including lowered fat intake, increased physical activity, and the use of lipid-lowering medications (1,9). In addition, for persons with diarrhea and malabsorption studies of fat malabsorption have shown that consumption of medium chain triglycerides (MCTs) resulted in fewer stools, decreased stool fat and weight and increased fat absorption (Grade II) (21).

Carbohydrate and fiber requirements: Recommendations have been made for increasing fiber intake toward the levels suggested in general nutrition guidelines because of the association with lower prevalence of lipodystrophy (1,41). Limited evidence supports a relationship between low-fiber diets or high–glycemic index diets and increased risk of fat deposition (Grade III) (1,21). Further investigation regarding the dietary intake of carbohydrate in people with HIV infection is warranted (1,21).

Fluid requirements: Water requirements for patients with normal fluid status can be estimated from the Dietary Reference Intakes (9). It may be prudent for patients with HIV or AIDS to avoid carbonated beverages and alcoholic beverages, as well as caffeinated beverages because they act as gastrointestinal stimulants and may contribute to dehydration and diarrhea or interact with ARTs. Consider increasing fluid requirements in patients who develop fever, nausea, vomiting, or diarrhea; the initiation of medication, physical activity, and inclement hot or dry weather may also necessitate increased fluid intake (9). Fluid restrictions may be indicated for patients with renal or hepatic failure (9).

Vitamin and mineral recommendations: Micronutrients, including both vitamins and minerals, play a key role in the maintenance of immune function, reduction of mortality from disease and treatment-related symptoms, and rehabilitation of nutritional status in HIV-infected persons (1). Studies have found that micronutrient deficiencies are common in individuals with HIV infection (Grade II) (1,21). Zinc, selenium, B vitamins, vitamin C, and vitamins A, E, and D may be at risk for deficiency due to inflammatory and metabolic responses and/or interactions with ARTs (1,15-17,21,42). However, it is difficult to adequately study these nutrients effectively due to the inability to separate the effects of individual nutrient deficiencies from the effects of generalized malnutrition on the immune system (1,21,42). A recent review yielded no conclusive evidence to show that micronutrient supplementation reduces morbidity and mortality among HIV-infected adults; however, there is evidence that vitamin A supplementation is beneficial for HIV-infected children (Grade II) (1,21). Further research regarding type, dose, and duration of micronutrient supplementation is needed before recommendations can be provided to persons with HIV or AIDS (Grade II) (1,21). Routine biochemical assessment of vitamin and mineral levels is recommended to determine the best treatment options if symptoms are present and deficiencies are suspected (1,9). Supplementation based on levels described in the Dietary Reference Intakes that remain below the upper limits of safety seems prudent in the absence of sufficient evidence (9).

Bone mineral density recommendations: Patients with HIV may experience a progressive loss of bone mineral density that leads to osteopenia or osteoporosis (1). Patients with HIV often have multiple risk factors for the loss of bone mineral density, including low BMI, weight loss, steroid use, nucleoside reverse transcriptase inhibitor use, and smoking (1,21). Bone density should be monitored with routine bone density tests (1). Bone density can be preserved through the maintenance of optimal weight and the prevention of rapid weight loss (1). Diet modifications that promote the maintenance of bone density include: reducing alcohol and caffeine consumption; choosing calcium-rich beverages (such as milk or fortified soy beverages) instead of high-phosphorus carbonated beverages; eating a variety of protein foods; eating calcium-rich and vitamin D–fortified foods, and taking a daily calcium supplement of 500 to 1,200 mg (1,9,43). Vitamin K, vitamin C, and zinc are also important for bone formation and should be included in an adequate diet (1,43). Assess the ART regimen and suggest adjustments to regimens so as to minimize side effects In addition, encourage physical activity and regular weight-bearing or resistance exercise (1).

Use of herbal supplementation: Supplemental nutrients, herbs, and other medications may be processed by the pathways used by antiretroviral medications. As a result, the levels of the supplements or medications may be greater or less than the expected levels (1). Potential interactions include the reduction of drug efficacy during the concomitant use of St. John's wort, garlic, and echinacea with protease inhibitors or non-nucleoside reverse transcriptase inhibitor antiretroviral drugs (1). Other herbal substances with a potential for drug interactions

include ginseng, melatonin, milk thistle, geniposide, and skullcap (44). Unless carefully tested for safety and medication interactions in persons with HIV or AIDS, herbal supplementation should not be recommended in this population (1,9). (Refer to Table III-11: HIV Medications: Names, Forms, Interactions, and Potential Side Effects (9).)

Physical activity recommendations: Exercise has been recommended as a strategy to maintain body function, restore and maintain adequate nutritional status, and assist in the management of altered glucose, lipid, and bone mineral metabolism (1). A review of evidence found that 20 or more minutes of constant or interval aerobic exercise, progressive resistant exercise, or a combination of both at a frequency of three times per week appeared to be safe in adults with HIV infection and may lead to significant improvements in cardiopulmonary fitness and a reduction in depressive symptoms (Grade I) (1,21). The impact of physical activity on immune status remains unclear (1). Special considerations should be given to patients with HIV who have active infection, reduced aerobic capacity, metabolic changes, or increased pain, fatigue, or impairment while exercising (1). Further research is needed on the effects of exercise on the serum lipid profiles of persons with HIV infection (Grade I) (1,21).

HIV and AIDS in children and adolescents: Children with HIV experience the same nutritional issues as adults who have the disease, but because of the added demands of growth and development, the effects in children are often more devastating (1). Early nutrition intervention is very important, and routine nutrition assessment should include monitoring of height, weight, and head circumference with comparison to growth standards for age and sex (1). Additional serial measures for anthropometry may include thigh circumference and mid-upper arm circumference (1). Deleterious nutritional outcomes commonly experienced by children with HIV or AIDS include the inability to achieve a normal weight for height, growth stunting, failure to thrive, malnutrition, impaired cognitive development or developmental and oral-motor feeding skill delay due to HIV encephalopathy, and wasting (1). Children infected with HIV are considered at high nutritional risk and should be referred for ongoing nutrition assessment and counseling (1).

Medications

There have been many advances in the pharmacologic treatment of HIV infection (1,9). The advent of HAARTs and combination medication therapies has reduced viral loads and increased the quality and length of life in patients with HIV or AIDS (1,10). Treatment of HIV infection with combination ART has been associated with improved nutritional indicators, such as BMI and BCM, as well as negative consequences such as detrimental toxicities and compromised bone mineral density affecting nutritional status (1.45-47). The benefits of ARTs (eg, maintaining BMI and BCM) must be evaluated against the potential for negative health outcomes. The registered dietitian must consider the adverse influences of various medications on indicators of nutrition status and metabolic indicators of disease risk (1). The clinician must recognize that nutrients and nutritional status can affect medication absorption, utilization, elimination, and tolerance (1,48). Potential nutritionrelated adverse effects that are related to ART include dyslipidemia, insulin resistance and glucose intolerance, and anemias (1,22,23). (Refer to the discussion of Lipodystrophy and Metabolic Disease in HIV and AIDS earlier in this section.) Patient adherence to prescribed medication regimens is needed to optimize treatment outcomes and should be assessed (1). Patient adherence to the prescribed medication regimen is affected by negative side effects, changes in body composition (eg, body fat changes as seen in lipodystrophy), and body image issues (1). (Refer to Table III-11: HIV Medications: Names, Forms, Interactions, and Potential Side Effects (9).)

There are currently six classes of antiretroviral medications (1,9):

- Nucleoside reverse transcriptase inhibitors
- Non-nucleoside reverse transcriptase inhibitors
- Protease inhibitors
- Fusion inhibitors
- Entry inhibitors
- Integrase inhibitors

There are also dual-class, fixed-dose, combination drugs that allow for fewer pills or once daily doses (1). Emerging drugs under investigation include a class of maturation inhibitors and other medications that boost the levels of antiretroviral medications (1). Most HIV-infected patients will require life-long pharmacotherapy for disease management (1). Response to ART can vary according to sex, and men and women with HIV infection may experience problems associated with medication interactions differently (1). For example, as

HIV Infection and AIDS

compared to men, women experience greater increases in blood lipid levels with certain medications (1). When using ritonavir and nelfinavir, men experience more diarrhea, while women experience nausea, vomiting, and abdominal pain more frequently than men (1). It is important for the clinician to consider these differences when assessing nutritional status and medication therapies.

Table III-11: HIV Medications: Names, I	forms, Interactions, and	Potential Side Effects (9)
Generic Name (commercial name); Class of Drug (manufacturer); Forms (tablets, capsules, oral solutions, or injections)	Food, Drink, and Other Interactions	Potential Side Effects
Abacavir (Ziagen); NRTI (Glaxo SmithKline); Tablets, oral solution (strawberry- banana)	Take with or without food; Use caution with alcohol (increases amount of time a drug is active in your body)	Nausea and vomiting, loss of appetite, abdominal pain, diarrhea, anemia, pancreatitis, lactic acidosis (rare)
Abacavir/Lamivudine/Zidovudine (Trizivir); NRTI ¹ combination (Glaxo SmithKline); Tablets	Take with or without food	See side effects on drug labels for Abacavir, Lamivudine, Zidovudine
Amprenavir (Agenerase); Protease inhibitor (Glaxo SmithKline); Capsules (contains sorbitol, vitamin E), oral solution (grape, bubble gum, peppermint)— contains acesulfame potassium, saccharin, vitamin E	Take with or without food; Do not take with high- fat meal; Do not take with a vitamin E supplement; If taking antacids, take Amprenavir 1 hour before or after; Avoid grapefruit juice; Increase fluid intake	Diarrhea, nausea and vomiting, taste changes, stomach upset, diabetes, fatigue, increased cholesterol levels, increased triglyceride levels, fat maldistribution, anemia. Do not take if you have kidney or liver failure.
Tipranavir (Aptivus); Protease inhibitor (Boehringer Ingelheim Pharmaceuticals); Capsules	Take with a high-fat meal	Hypertriglyceridemia, hyperglycemia, fat maldistribution
Atazanavir (Reyataz) Protease inhibitor (Bristol-Myers Squibb) Capsules (contain lactose)	Take with food	Nausea, increased indirect bilirubin, lactic acidosis (rare); Be careful if you have liver problems, may require dose change
Darunavir (Prezista) Protease inhibitor (Tibotec Therapeutics) Capsules	Take with or without food	Nausea, diarrhea, headache, cold-like symptoms (runny nose and sore throat)
Delavirdine (Rescriptor); NNRTI ² (Agouron); Tablets (contain lactose);	Take with or without food; Do not take with antacids or magnesium- containing supplements; May take with acidic drinks (such as cranberry juice); Avoid drinking alcohol	Increased thirst, loss of appetite, dry mouth, nausea and vomiting, inflamed stomach, diarrhea, constipation, passing gas; Be careful using this drug if you have liver problems

Generic Name (commercial name); Class of Drug (manufacturer); Forms (tablets, capsules, oral	Food, Drink, and Other Interactions	Potential Side Effects
solutions, or injections) Didanosine, ddI (Videx, Videx EC); NRTI (Bristol-Myers Squibb); Videx: chewable tablets (orange, contains sorbitol, aspartame); powder; Videx EC: capsules	Take without food on an empty stomach ½ hour before or 2 hours after a meal; Do not take with acidic drinks or foods, aluminum- containing antacids, or magnesium- containing supplements; Avoid drinking alcohol	Loss of appetite, diarrhea, nausea and vomiting, abdominal pain, constipation, dry mouth, taste changes, pancreas infection, (increased risk if you drink alcohol), lactic acidosis (rare), problems with feeling in your arms and legs; Be careful if you have kidney problems
Efavirenz (Sustiva); NNRTI (Bristol-Myers Squibb); Capsules, tablets (both contain lactose)	Take on an empty stomach; A high-fat meal increases the risk for side-effects; Avoid drinking alcohol	Loss of appetite, nausea and vomiting, diarrhea, taste changes, increased good and bad cholesterol levels, increased triglyceride levels
Emtricitabine (Emtriva); NRTI (Gilead); Capsules	Take with or without food; Eating a high-fat meal lowers the highest drug levels	Nausea and vomiting, diarrhea, lactic acidosis (rare); Be careful if you have kidney problems; may require a dose change
Enfuvirtide (Fuzeon); Fusion inhibitor (Hoffman-LaRoche); Powder for injection	No diet restrictions	Diarrhea, nausea, fatigue, loss of appetite, constipation, inflamed pancreas, increased triglyceride levels, increased lipase, increased amylase; Low weight decreases clearance of drug from your blood, but no dose adjustment is recommended
Fosamprenavir (Lexiva); Protease inhibitor (Glaxo SmithKline); Tablets	Take with or without food; Avoid vitamin E supplementation	Nausea, vomiting, diarrhea, increased triglyceride levels, fat maldistribution
Indinavir (Crixivan); Protease inhibitor (Merck); Capsules (contain lactose)	Take on an empty stomach or with very-low- calorie/low-protein snack (Note: no food restriction when taken with ritonavir); Take with plenty of fluids (at least 1.5 liters per day); avoid grapefruit juice	Nausea, vomiting, acid reflux, increased or decreased appetite, abdominal pain, taste changes, diarrhea, kidney stones, diabetes (rare), increased blood liver enzyme levels or pancreas enzymes, increased muscle damage, red blood cells are destroyed faster than your body can make them, impaired liver functioning; Dose is changed in cirrhotic liver disease; hyperlipidemia; fat maldistribution

Generic Name (commercial name); Class of Drug (manufacturer); Forms (tablets, capsules, oral solutions. or injections)	Food, Drink, and Other Interactions	Potential Side Effects
Lamivudine, 3TC (Epivir); NRTI (Glaxo SmithKline); Tablets; oral solution (strawberry- banana flavor)	Take with or without food	Nausea and vomiting, abdominal cramps, diarrhea, pancreatitis, lactic acidosis (rare); Dose is changed for kidney problems; Note: also used for hepatitis B in lower doses as Epivir HBV
Lamivudine/Zidovudine, 3TC/ZDV, 3TC/AZT (Combivir); NRTI combination (Glaxo SmithKline); Tablets	Take with or without food	See side effects on drug labels for Lamivudine, Zidovudine
Lopinavir/Ritonavir (Kaletra); Protease inhibitor (Abbott); Soft gel capsules (contain sorbitol), oral solution (cotton candy or vanilla; contains alcohol and saccharin)	Take with food	 Abdominal pain, diarrhea, nausea, increased triglyceride levels; increased cholesterol levels, fat maldistribution; inflamed pancreas; hyperglycemia; Be careful if you have liver problems; if taken with Didanosine, must be taken 2 hours apart
Nelfinavir (Viracept); Protease inhibitor (Agouron); Tablets; powder (contains aspartame)	Take with fatty food; May crush tablets, mix with water, and take immediately after mixing; Mixing powder with acidic food or drink results in bitter taste	Diarrhea, gas passing, nausea, abdominal pain, hyperlipidemia; fat maldistribution; diabetes (rare), increased liver enzymes; Be careful if you have liver problems
Nevirapine (Viramune); NNRTI (Roxane); Tablets; oral suspension (contains sorbitol)	Take with or without food	Nausea and vomiting, abdominal pain, fatigue, toxic to the liver
Ritonavir (Norvir); Protease inhibitor (Abbott); Soft gel capsule; oral solution (contains saccharin, alcohol; peppermint, caramel)	Take with food	Nausea and vomiting, diarrhea, taste changes, loss of appetite, upset stomach, diabetes, inflamed pancreas, increased triglyceride levels, increased liver enzymes, increased muscle damage, increased uric acid
Saquinavir (Invirase, Fortovase); Protease inhibitor (Roche); Invirase: capsules; Fortovase: soft gel capsules	Take within 2 hours of a high-calorie, high- fat meal	Nausea, abdominal discomfort, gas, diarrhea, low blood sugar, mouth/esophageal ulcers; Be careful if you have liver disease, check triglyceride levels
Stavudine, d4T (Zerit, Zerit XR); NRTI (Bristol-Myers Squibb); Zerit: capsules or powder (fruit); Zerit XR: capsules	Take with or without food; Avoid drinking alcohol	Nausea and vomiting, diarrhea, loss of appetite, mouth/esophageal ulcers, lipoatrophy, hyperlipidemia, problems with feeling in your arms and legs, increased liver enzymes, increased pancreas enzymes; May change dose for kidney problems
Tenofovir (Viread); NRTI (Gilead); Tablets (contain lactose)	Take with food; Avoid St John's wort, garlic supplements, and milk thistle	Nausea, vomiting, diarrhea, passing gas, abdominal pain, lactic acidosis (rare), increased muscle damage, increased triglyceride levels; Do not take if you have kidney problems

Generic Name (commercial name); Class of Drug (manufacturer); Forms (tablets, capsules, oral solutions, or injections)	Food, Drink, and Other Interactions	Potential Side Effects
Zalcitabine, ddC (Hivid); NRTI (Roche); Tablets (contain lactose)	Take on an empty stomach; Avoid drinking alcohol	Loss of appetite, mouth sores, nausea and vomiting, diarrhea, constipation, problems with feeling in your arms and legs, lactic acidosis (rare), inflamed pancreas (rare), increased triglyceride levels, anemia Dose may be changed for kidney problems
Zidovudine, AZT, Compound S, Azidothymidine (Retrovir); NRTI (Glaxo SmithKline); Tablets, capsules, syrup (strawberry), injections	Take with or without food; Do not take with a high-fat meal	Loss of appetite, nausea and vomiting, upset stomach, constipation, taste changes, anemia, muscle disease in long-term use; Dose may be changed in impaired liver or kidney function

¹NRTI = nucleoside reverse transcriptase inhibitor

²NNRTI = non-nucleoside reverse transcriptase inhibitor

Sources: Compiled from manufacturer information

Drug Facts and Comparisons. Drug Facts and Comparisons 2004. 58th ed. 2003.

Pronsky ZM, Meyer SA, Fields-Gardner C. *HIV Medications-Food Medication Interaction Guide*. 2nd ed. 2001.

DHHS Panel on Antiretroviral Guidelines for Adults and Adolescents—A Working Group of the Office of AIDS Research Advisory Council. Guidelines for the Use of Antiretroviral Agents in HIV-1-Infected Adults and Adolescents. October 10, 2006.

©Academy of Nutrition and Dietetics. Reprinted with permission. HIV medications in HIV/AIDS. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed November 15, 2010.

Medications for symptom management: Symptom management is a key strategy in maintaining both nutritional status and ART regimen adherence (1). Medications may be used to control symptoms and conditions including nausea, vomiting, diarrhea, mouth and throat sores, and organ diseases (1). Two commonly used medications, megestrol acetate (Megace) and dronabinol (Marinol), are currently approved for appetite stimulation. It should be noted, however, that the weight gain associated with these appetite stimulants is typically in the form of fat mass and not the desired LBM (9). Megestrol acetate also compromises testosterone balance, making it difficult to maintain and restore BCM and possibly exacerbating diabetes mellitus (9). Coadministration of testosterone with megestrol acetate has not been shown to increase lean tissue accrual. Testosterone replacement and anabolic steroids have been explored to assist with the restoration of body weight and BCM in addition to improving strength and quality of life (49). However, anabolic steroids can cause liver toxicity and negative changes in lipid profiles (1). The use of testosterone and recombinant human growth hormone to treat wasting and central fat accumulation has been explored (50). Higher doses of growth hormone promote recovery from HIV-related wasting by restoring BCM, while lower doses of growth hormone reduce central fat accumulation (1). Anti-cytokine therapies, such as thalidomide, have been explored for treatment of tuberculosis and HIV-related wasting (1). More recently, thalidomide has been used to treat recurrent aphthous ulcers and HIV-related colitis; however, its use is limited due to teratogenicity, peripheral neuropathy, and other adverse effects (51).

Nutrition Intervention and Monitoring

Symptom management is an important component of nutrition intervention and monitoring (1). Nutrition interventions should support the patient's medical treatment goals while reducing any negative nutrition-related health effects of the disease and medication regimens (1). Complications are diverse and develop frequently, interfering with nutritional intake and outcomes. Some of the more common complications, as well as nutrition management strategies to optimize nutritional status, are listed in Table III-12: Nutrition Management Strategies for People With HIV Infection or AIDS.

Complication	Descible Causes	Nutrition Internetions
	Possible Causes	Nutrition Interventions
Anorexia	Medication	Review medications.
	Infection	Consider appetite stimulants.
	Fever	Recommend small, frequent, nutrient-dense
	Nausea	foods; eating in a pleasant atmosphere;
	Vomiting	and easy-to-prepare food or assistance
	Diarrhea	with meals.
	Pain	Consider medical nutritional supplements.
	Anxiety	Consider vitamin and mineral supplements if
	Depression	symptoms or biochemical tests indicate
	Other medical therapies	deficiency
	other medical meraples	denciency.
Nausea and vomiting	Medication	Review medications.
0	Infection	Consider antiemetics.
	Anxiety	Consider altering medication times
	Fover	Becommend small frequent feedings: dry
	Modical thorapies	foods soft foods cold or room
	Metical therapies	tomporature foods and salty foods
		elevation of unnon hody during and often
		elevation of upper body during and after
		meals; and liquids between meals.
		Ensure proper food-medication schedule.
		Consider medical nutritional supplements.
		Consider vitamin and mineral supplements if
		symptoms or biochemical tests indicate
		deficiency.
		Recommend oral rehydration.
Diarrhea	Medication	Review medications.
	Antibiotic therapy	Recommend oral rehydration; replace
	Infection	electrolytes; increase soluble-fiber foods;
	Foodborne or waterborne illness	evaluate tolerance to gas-forming foods,
	Food intolerance	fat. and lactose.
	Other medical conditions	Consider intravenous rehydration
	Medical therapy	Consider vogurt or probiotics if long-term
	Anviety	antibiotic therapy is required
	Stroop	Consider vitamin and minoral supplements if
	50.655	
		symptoms or biochemical tests indicate
		deficiency.
		Consider pancreatic enzymes.
Oral and econhageal lesions	Medication	Review medications
or all and esophagear resions	Infection	Recommend soft nonsnicy nonacidic foods:
	Malnutrition	nursed foods or thickened liquids, and oral
		pureeu loous or unickeneu nyulus; anu orar
	Urai and esophageal candidiasis	supplements.
	Kaposi sarcoma and other	Consider a topical analgesic to decrease
	malignancies	mouth pain.
		Consider medical nutritional supplements.
		Consider vitamin and mineral supplements if
		symptoms or biochemical tests indicate
		deficiency.

Table III-12:-Nutrition Management Strategies for People With HIV Infection or AIDS (1)

Complication	Bassible Causes	Nutrition Interventions
	Possible Causes	Desting the sting
Early satiety	Medication	Review medications.
	Infection	Recommend, small, frequent feedings;
	Nausea	nutrient-dense foods; liquids between
		meals; and avoidance of greasy, fried foods
		and gas-forming foods.
		Consider medical nutritional supplements as between-meals snacks.
		Consider vitamin and mineral supplements if
		symptoms or biochemical tests indicate
		deficiency.
Food intolerances	Medication	Review medications.
	Infection	Recommend alternative foods and textures;
	Gastrointestinal disturbance	evaluate patient for nutrient deficiencies.
	Poor dentition	Consider medical nutritional supplements as
	Genetic cause	tolerated.
		Consider vitamin and mineral supplements if
		symptoms or biochemical tests indicate
		denciency.
Tasta and small shanges	Madication	Investigate and treat cause
l'aste allu sillell challges	Medication	Change initiate or discontinue modication
		Decommond small frequent foodings
		eventiment with a wide variety of foods
		and sansonings and alternative protein
		and seasonings and alternative protein
		sources.

Nutrition prescription: The registered dietitian should determine the appropriate mode of nutrition support based on the nutrition assessment and diagnosis. A combination of approaches may be necessary to address the nutrition-related problems faced by persons with HIV (1). Meal planning using guidelines established for lipid management and diabetes management may also be necessary in patients who develop lipodystrophy or glucose intolerance related to medication management (1,9). For these patients refer to Section C: "Modification of Carbohydrate and Fat". Consider the following information when determining the nutrition prescription:

- Oral feedings are preferred over any other feeding method. Efforts to maintain the oral feeding route should be maximized. Nutrient-dense foods and supplements should be used to support maintenance and restoration of nutritional status and body weight. Appetite stimulants may be indicated for patients who experience anorexia (1,9).
- The enteral feeding route is preferred over parenteral administration in order to preserve gut structure and function. Assess patients carefully and reassess them on a regular basis. Patients with AIDS-related wasting syndrome should be carefully monitored for refeeding syndrome (1,9). (See Section B: "Specialized Nutrition Support".)
- Parenteral nutrition may become necessary when a patient meets the criteria for initiation of total parenteral nutrition (9). Continual assessment and routine monitoring of laboratory values is essential. Patients with AIDS-related wasting syndrome should be carefully monitored for refeeding syndrome (9). (See Section B: "Specialized Nutrition Support".)

Pediatric-specific interventions: The goals of intervention for children with HIV infection are similar to the goals for adults with the added dimension of supporting adequate growth and development (1). Mothers with HIV should be made aware of the risks and benefits of different infant-feeding options, including the risk of HIV transmission through breastfeeding (1). The World Health Organization has issued recommendations for infant feeding (52). The World Health Organization recommends the continued use of prophylactic antiretrovirals for mothers and children to reduce the risk of HIV transmission through breast milk (52).

Patient education and food safety: Education and counseling are essential features of medical nutrition therapy for people infected with HIV (1). Patients infected with HIV have weakened immune systems and are more susceptible to contracting foodborne illnesses, as shown by a recent review by the Academy of Nutrition and

HIV Infection and AIDS

Dietetics Evidence Analysis Library (Grade I) (1,21). Foodborne illnesses often cause symptoms similar to those of influenza (diarrhea, nausea, vomiting, fever, and cramping). The severity of symptoms ranges from mild to life-threatening. One study demonstrated a decrease in the number of symptoms and eating difficulties in people with HIV infection when foodborne illness education was a component of an HIV health management program (Grade I) (1,21). In addition, two studies showed that when education was part of a home-delivered meal program, there was strong adherence to food safety guidelines (Grade I) (1,21). Food and water safety education is of special importance to patients experiencing immune dysfunction, especially for patients with low CD4 counts (9,21). Patients should be provided with information that fits their individual lifestyles in terms of shopping, cooking, storing food, and dining out (9). Practical food safety guidelines for patients are as follows (53):

Shopping for Groceries

- Pay attention to "sell by" and "use by" dates on perishable products. Do not purchase or use outdated foods.
- Put raw meat, fish, and poultry in separate plastic bags before placing them in your grocery cart with other foods.
- Avoid cans that have dents, bulges, or leaks.
- Avoid luncheon meats and cheeses from the deli case, as they may have been contaminated from improper food handling. Instead, use prepackaged processed meats and cheeses.
- Select food items that require refrigeration immediately before checking out. If food will be in the car for longer than 30 minutes, use a cooler to keep it cold.
- Buy only pasteurized dairy products.

Storing and Saving Food at Home

- Check refrigerator and freezer temperatures. Refrigerators should be kept at or below 40°F, and freezers should be kept at or below 0°F.
- Keep the interior of the refrigerator and freezer clean.
- After returning home from grocery shopping, immediately place perishable foods in the refrigerator or freezer.
- Place uncooked meat, fish, and poultry products in separate plastic bags, and then set them on a plate on the lowest shelf of the refrigerator to prevent raw juices from dripping onto other foods.
- Use ground beef, ground poultry, and fresh fish within 1 to 2 days. Use beef steaks, roasts, and poultry within 3 or 4 days.
- Label all leftovers with the date. Wrap leftovers or store in a closed container.
- Refrigerate leftovers as soon as possible. Foods left at room temperature for longer than 2 hours are susceptible to bacterial growth.
- Store leftovers in shallow containers rather than narrow, deep bowls. Shallow containers will help the leftovers cool more quickly to the proper temperature.
- Leftovers should be wrapped securely before refrigeration and should be eaten within 3 or 4 days.

Freezing and Defrosting Foods

- Thaw all frozen foods in the refrigerator. Place the frozen foods in a plastic bag or on a plate to prevent juices from dripping onto other foods. Frozen food can be defrosted in the microwave oven, according to the manufacturer's directions, and then cooked immediately.
- Thawing food on the kitchen counter or in warm water is dangerous. Many harmful bacteria grow rapidly between 70°F and 120°F.
- If in doubt about the safety of a food, throw it out.

Preparing Food

- Wash hands with antibacterial soap and warm water before and after handling food, and especially after handling raw meat, poultry, and fish. Wash hands after sneezing or coughing.
- Use separate cutting boards, platters, trays, and utensils for cooked and uncooked meat, poultry, and fish. Always wash contact surfaces and utensils with a dilute bleach solution immediately after preparing these products.
- Scrub all fresh fruits and vegetables in hot, soapy water before eating.
- Avoid sushi, sashimi, seviche, raw oysters and clams, Caesar salads, homemade salad dressings and mayonnaise, homemade ice cream, homemade eggnog, homemade cheeses, and cookie dough or cake batter. These foods contain raw seafood, meat, poultry, or eggs and therefore may contain harmful pathogens (bacteria).

Cooking

- Cook ground beef to a minimum internal temperature of 160°F. Cook poultry to a minimum internal temperature of 165°F, and cook fish to a minimum internal temperature of 145°F. Steaks and roasts should be cooked to a minimum internal temperature of 145°F. Meeting these temperature requirements does not necessarily mean that the food is well done.
- Use an instant-read thermometer for all types of meat, including pork and fish, to accurately determine the internal temperature. An instant-read thermometer is different from a meat thermometer in that it does not stay in the meat while cooking. It has a small, round temperature display and .
- Avoid cooking meats at a very low oven temperature (less than 300°F) or overnight, as this may encourage bacterial growth before cooking is complete.
- Cook all ground meats until they are brown all the way through. This is especially important in a restaurant where you may not be able to check the internal temperature.
- Cook stuffing separately from meat.
- To marinate foods, keep them in the refrigerator in covered containers.
- Cook eggs until well done. Eggs that are cooked over easy or undercooked increase the risk of salmonella infection. Egg dishes should be cooked to a minimum temperature of 160°F.
- Cook raw seafood within 24 hours of purchase.

Microwave Oven Cooking

- Do not cook meat that contains bones in the microwave. Bones can shield the surrounding meat from the microwaves and therefore leave some meat undercooked.
- Because microwaves do not always cook foods evenly, try using a turntable to help increase the consistency.
- Check meat in several places to ensure that is cooked to the proper temperature throughout.

Water Safety

- Bacteria that may contaminate a water supply include *Giardia, Cryptosporidium, Microsporidia,* and *Mycobacterium avium-intracellulare.*
- For drinking water, boil tap water for 1 to 5 minutes.
- Only bottled water that has been purified by distillation, reverse osmosis, or absolute 1-μm filtration can be considered safe.
- Home water filters should use reverse osmosis or absolute 1-µm filtration. Safe water filters will be labeled "NSF Standard 53for Cyst Removal" on the box.
- Ice is safe only if it is made from water that has been boiled or properly distilled or filtered.

Eating Away From Home

- Make sure meat, fish, and poultry are cooked thoroughly. Check to see that burgers are no longer pink in the middle and the juices run clear. Send back undercooked meats for further cooking.
- When eating from a buffet, make sure cold foods are cold (at or below 41°F) and hot foods are hot (at or above 140°F). Avoid buffets when possible.
- Order fried eggs cooked on both sides. Send back scrambled eggs that look runny.
 - Look for cleanliness at salad bars and at meat and deli counters. Immune-compromised individuals are at risk when they eat from salad bars. Often fruits and vegetables are not washed thoroughly, and prepared foods are not kept at proper temperatures to prevent bacteria from thriving.
 - Confirm that soap and towels are available in the restroom. If not, it may mean that the wait staff and cooks cannot wash their hands properly after using the restroom.
- Consumption of foods purchased from street vendors is risky and should be discouraged.

Foreign Travel

- Do not purchase foods from street vendors.
- Choose cooked foods over salads, fruits, and raw vegetables.
- Bring drinking water with you, or drink only boiled water that has been cooled. Beverages made with boiled water, such as coffee or tea, are safer to drink, as are canned or bottled carbonated beverages, beer, and wine. Do not drink beverages containing ice cubes.
- Note that the United States has one of the safest food supplies in the world. Be aware that not all countries have the same high standards for sanitation and food safety.

HIV Infection and AIDS

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Position of the American Dietetic Association: nutrition intervention in human immunodeficiency virus infection. *J Am Diet Assoc.* 2010; 110:1105-1119.
- 2. Joint United National Programme on HIV/AIDS. *Report on the Global AIDS Epidemic. Executive Summary.* Available at: http://data.unaids.org/pu/GlobalReport/2008/JC1511_GR08_ExecutiveSummary_en.pdf. Posted July 2008. Accessed October 1, 2010.
- Centers for Disease Control and Prevention. HIV/AIDS data through December 2006. *HIV/AIDS Surveillance Supplemental Report*. 2009;14 (No. 1). Available at: www.cdc.gov/hiv/topics/surveillance/resources/reports/2009supp_vol14no1/pdf/HIVAIDS_SSR_Vol14_No1.pdf. Accessed July 23, 2009.
- 4. Hall HI, Song R, Rhodes P, Prejean J, An Q, Lee LM, Karon J, Brookmeyer R, Kaplan EH, McKenna MT, Janseen RS. Estimation of HIV incidence in the United States. *JAMA*. 2008;300:520-529.
- 5. Centers for Disease Control and Prevention. *CDC HIV/AIDS Facts: New Estimates of U.S. HIV Prevalence, 2006.* Available at: www.cdc.gov/hiv/topics/surveillance/resources/factsheets/pdf/prevalence.pdf. Posted October 2008. Accessed July 23, 2009.
- 6. Centers for Disease Control and Prevention. 1993 revised HIV classification and expanded AIDS surveillance definition for AIDS among adolescents and adults. *MMWR Morb Mortal Wkly Rep.* 1992;41:1-19.
- Cohen PT, Sande MA, Volberding PA, eds. The AIDS Knowledge Base: A Textbook on HIV Disease From the University of California, San Francisco, and the San Francisco General Hospital. 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999. Available at: hivinsite.ucsf.edu. [University of California, San Francisco website]. Accessed March 30, 2005.
- World Health Organization. WHO Case Definitions of HIV for Surveillance and Revised Clinical Staging and Immunological Classification of HIV-Related Disease in Adults and Children. World Health Organization; 2007. Available at: www.who.int/hiv/pub/guidelines/HIVstaging150307.pdf. Accessed October 28, 2010.
- HIV/AIDS. In: *Nutrition Care Manual*. Updated annually. Academy of Nutrition and Dietetics. Available at: www.nutritioncaremanual.org. Accessed November 1, 2010.
- 10. Mangili A, Murman DH, Zampini AM, Wanke CA. Nutrition and HIV infection: review of weight loss and wasting in the era of highly active antiretroviral therapy from the nutrition for healthy living cohort. *Clin Infect Dis.* 2006;42:836-842.
- 11. Lo J, You SM, Wei J, Canavan B, Grinspoon S. Relationship of peak growth hormone to cardiovascular parameters, waist circumference, lipids and glucose in HIV-infected patients and healthy adults. *Clin Endocrinol (Oxf)*. 2009;71:815-822.
- 12. Slama L, LeCamus C, Serfaty L, Pialoux G, Capeau J, Gharakhanian S. Metabolic disorders and chronic viral disease: the case of HIV and HCV. *Diabetes Metab.* 2009;35:1-11.
- 13. Llibre JM, Flaco V, Tural C, Negredo E, Pineda JA, Munoz J, Ortega E, Videla S, Sirera G, Martinez E, Miralles C, Iribarren J, Galindo MJ, Domingo P, d'Arminio-Monforte A, Miro JM, Clotet B. The changing face of HIV/AIDS in treated patients. *Curr HIV Res.* 2009;7:365-377.
- 14. Faintuch J, Soeters PB, Osmo HG. Nutritional and metabolic abnormalities in pre-AIDS HIV infection. *Nutrition*. 2006;22:683-690.
- 15. Kruzich LA, Marquis GS, Carriquiry AL, Wilson CM, Stephensen CB. US youths in the early stages of HIV disease have low intakes of some micronutrients for optimal immune function. *J Am Diet Assoc.* 2004;104:1095-1101.
- 16. Butensky E, Harmatz P, Lee M, Kennedy C, Petru A, Wara D, Miaskowski C. Altered iron metabolism in children with human immunodeficiency virus disease. *Pediatr Hematol Oncol.* 2009;26:69-84.
- 17. Schaible UE, Kaufmann SH. Malnutrition and infection: complex mechanisms and global impacts. *PLoS Med.* 2007 May;4:e115.
- Srasuebkul P, Lim PL, Lee MP, Kumarasamy N, Zhou J, Sirisanthana T, Li PC, Kamarulzaman A, Oka S, Phanuphak P, Vonthanak S, Merati TP, Chen YM, Sung-kanuparph S, Tau G, Zhang F, Lee CK, Ditangeo R, Pujari S, Choi JY, Smith J, Law MG. Short-term clinical disease progression in HIV-infected patients receiving combination antiretroviral therapy: results from the TREAT Asia HIV observational database. *Clin Infect Dis.* 2009;48:940-950.
- 19. Siddiqui J, Phillips AL, Freedland ES, Sklar AR, Darkow T, Harley CR. Prevalence and cost of HIV-associated weight loss in a managed care population. *Curr Med Res Opin*. 2009;25:1307-1317.
- Kotler DP, Tierney AR, Wang J, Pierson RN Jr. Magnitude of body-cell-mass depletion and the timing of death from wasting in AIDS. Am J Clin Nutr. 1989;50:444-447.
- 21. HIV/AIDS Evidence Based Nutrition Practice Guidelines. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; December 2010. Available at: http://www.andevidencelibrary.com/topic.cfm?cat=4248. Accessed February 7, 2013.
- 13th annual HIV drug guide. *Positively Aware*. March/April 2009;20:28-55. Available at: positivelyaware.com/2009/09_02/09_02.pdf. Accessed April 21, 2009.
- Fichtenbaum CJ. Metabolic abnormalities associated with HIV infection and antiretroviral therapy. *Curr Infect Dis Rep.* 2009;11:84-92.
- Visnegarwala F, Shlay JC, Barry V, Gibert CL, Xiang Y, Wang J, Kotler D, Raghavan S, El-Sadar WM; for Terry Beirn Community Programs for Clinical Research on AIDS (CPCRA). Effects of HIV infection on body composition changes among men of different racial/ethnic orgins. *HIV Clin Trial*. 2007;8:145-154.
- Lundgren JD, Battegay M, Behrens G, De Wit S, Guaraldi G, Katlama C, Martinez E, Nair D, Powderly WG, Reiss P, Sutinen J, Vigano A; EACS Executive Committee. European AIDS Clinical Society (EACS) guidelines on the prevention and management of metabolic diseases in HIV. *HIV Med.* 2008;9:72-81.
- 26. Lagathu C, Kim M, Maachi M, Vigouroux C, Cervera P, Capeau J, Caron M, Bastard JP. HIV antiretroviral treatment alters adipokine expression and insulin sensitivity of adipose tissue in vitro and in vivo. *Biochimie*. 2005;87:65-71.
- Diehl LA, Fabris BA, Barbosa DS, De Faria EC, Wiechmann SL, Carrilho AJ. Metformin increases HDL3-cholesterol and decreases subcutaneous truncal fat in nondiabetic patients with HIV-associated lipodystrophy. *AIDS Patient Care STDS*. 2008;22:779-786.
- Schindler K, Rieger A, Tura A, Gmeinhardt B, Touzeau-Romer V, Haider D, Pacini G, Ludvik B. The effect of rosiglitazone on insulin sensitivity, beta cell function, bone mineral density, and body composition in HIV-positive patients on highly-active antiretroviral therapy (HAART). Horm Metab Res. 2009;41:573-579.
- 29. Mulligan K, Yang Y, Wininger DA, Koletar SL, Parker RA, Alston-Smith BL, Schouten JT, Fielding RA, Basar MT, Grinspoon S. Effects of Metformin and rosiglitazone in HIV-infected patients with hyperinsulinemia and elevated waist/hip ratio. *AIDS*. 2007;21:47-57.
- 30. Rawat R, Stoltzfus RJ, Ntozini R, Mutasa K, Iliff PJ, Humphrey JH. Influence of inflammation as measured by alpha-1-acid glycoprotein on iron status indicators among HIV-positive postpartum Zimbabwean women. *Eur J Clin Nutr.* 2009;63:787-793.

- 31. Mburu AS, Thurnhm DI, Mwankiki DL, Muniu EM, Alumasa F, deWagt A. The influence and benefits of controlling for inflammation on plasma ferritin and hemoglobin responses following a multi-micronutrient supplement in apparently healthy, HIV+ Kenyan adults. *J Nutr.* 2008;138:613-619.
- 32. Feldman JG, Goldwasser P, Holman S, DeHovitz J, Minkoff H. C-reactive protein is an independent predictor of mortality in women with HIV-1 infection. J Acquir Immune Defic Syndr. 2003;32:210-214.
- 33. Salomon J, de Truchis P, Melcior JC. Body composition and nutritional parameters in HIV and AIDS patients. *Clin Chem Lab Med.* 2002;40:1329-1333.
- 34. Northrop-Clewes CA. Interpreting indicators of iron status during an acute phase response--lessons from malaria and human immunodeficiency virus. *Ann Clin Biochem.* 2008;45:18-32.
- 35. Batterham MJ. Investigating heterogeneity in studies of resting energy expenditure in persons with HIV/AIDS: a meta-analysis. *Am J Clin Nutr*. 2005;81:702-713.
- 36. Fitch KV, Guggina LM, Keough HM, Donal Looby SE, Hadigan C, Anderson EJ, Hubbard J, Liebau JG, Johnsen S, Wei J, Makimura H, Stanley TL, Lo J, Grinspoon SK. Decreased respiratory quotient in relation to resting energy expenditure in HIV-infected and noninfected subjects. *Metabolism.* 2009;58:608-615.
- 37. Kulstad R, Schoeller DA. The energetics of wasting disease. Curr Opin Clin Nutr Metab Care. 2007;10:488-493.
- 38. Mwamburi DM, Wilson IB, Jacobson DI, Spiegelman D, Gerbach SL, Knox TA, Wanke CA. Understanding the role of HIV load in determining weight change in the era of highly active antiretroviral therapy. *Clin Infect Dis.* 2005;40:167-173.
- Campa A, Yang Z, Lai S, Xue L, Phillips JC, Sales S, Page JB, Baum MK. HIV-related wasting in HIV-infected drug users in the era of highly active antiretroviral therapy. *Clin Infect Dis.* 2005;41:1179-1185.
- 40. Williams SB, Bartsch G, Muurahainen N, Collins G, Raghavan SS, Wheeler D. Protein intake is positively associated with body cell mass in weight-stable HIV infected men. *J Nutr.* 2003;133:1143-1146.
- 41. Shah M, Tierney K, Adams-Huet B, Boonyavarakul A, Jacob K, Quittner C, Dinges W, Peterson D, Garg A. The role of diet, exercise and smoking in dyslipidaemia in HIV-infected patients with lipodystrophy. *HIV Med.* 2005;6:291-298.
- 42. Woods MN, Speigleman D, Knox TA, Forrester JE, Connors JL, Skinner SC, Silva M, Kim JH, Gorbach SL. Nutrient intake and body weight in a large HIV cohort that includes women and minorities. *J Am Diet Assoc.* 2002;102: 203-211.
- 43. Mondy K, Tebas P. Emerging bone problems in patients infected with human immunodeficiency virus. *Clin Infect Dis.* 2003;36(suppl 2): S101-S105.
- 44. Project Inform. Herbs, recreational drugs and HIV medications. January 2010. Available at: http://www.projectinform.org/info/herbs/index.shtml. Accessed December 31, 2010.
- 45. Ferrando SJ, Rabkin JG, Lin SH, McElhiney M. Increase in body cell mass and decrease in wasting are associated with increasing potency of antiretroviral therapy for HIV infection. *AIDS Patient Care STDS*. 2005;19: 216-223.
- 46. Duvivier C, Kolta S, Assoumou L, Ghosn J, Rozenberg S, Murphy RL, Katlama C, Costagliola D, ANRS 121 Hippocampe study group. Greater decrease in bone mineral density with protease inhibitor regimens compared with nonnucleoside reverse transcriptase inhibitor regimens in HIV-1 infected naïve patients. *AIDS*. 2009;27:817-824.
- 47. Boescke C, Cooper DA. Toxicity of HIV protease inhibitors: clinical considerations. Curr Opin HIV AIDS. 2008;3:653-659.
- Lopez JC, Moreno S, Jimenez-Onate F, Clotet B, Rubio R, Hernandez-Quero J. A cohort study of the food effect on virological failure and treatment discontinuation in patients on HARRT containing didanosine enteric-coated capsules (FOODDIe Study). *HIV Clin Trials*. 2006;7:155-162.
- 49. Hengge UR, Stocks K, Wiehler H, Faulkner S, Esser S, Lorenz C, Jentzen W, Hengge D, Goos M, Dudley RE, Ringham G. Double-blind, randomized, placebo-controlled phase III trial of oxymetholone for the treatment of HIV wasting. *AIDS*. 2003; 17:699-710.
- 50. Cofrancesco J Jr, Freedland E, McComsey G. Treatment options for HIV-associated central fat accumulation. *AIDS Patient Care STDS*. 2009;23:5-18.
- 51. Matthews SJ, McCoy C. Thalidomide: a review of approved and investigational uses. Clin Ther. 2003;25:342-395.
- World Health Organization. Rapid advice: revised WHO principles and recommendations on infant feeding in the context of HIV November 2009. World Health Organization; 2009. Available at: whqlibdoc.who.int/publications/2009/9789241598873_eng.pdf. Accessed January 18, 2010.
- 53. US Dept of Agriculture, Food Safety and Inspection Service. Food Safety for People With HIV/AIDS: A Need-to-Know Guide for Those Who Have Been Diagnosed With HIV/AIDS. US Dept of Agriculture; 2006. Available at: www.fsis.usda.gov/PDF/Food_Safety_for_People_with_HIV.pdf. Accessed November 16, 2010.

HYPERTENSION

Discussion

At least 50 million adults in the United States have hypertension (1). According to the *Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* (referred to as the JNC 7), 30% of these adults are unaware that they have hypertension, and more than 40% of adults with hypertension are not receiving treatment (2). The prevalence of hypertension increases with advancing age. More than half of people aged 60 to 69 years and approximately three fourths of people aged 70 years and older have hypertension (1). Based on data from the Framingham Heart Study, people who are normotensive at 55 years of age have a 90% lifetime risk of developing hypertension (3). In African Americans, hypertension is more common and more severe. It develops at an earlier age and leads to more clinical complications (2).

Hypertension is an increasingly important medical and public health issue (2). For every 20 mm Hg systolic or 10 mm Hg diastolic increase in blood pressure, the mortality from ischemic heart disease and stroke doubles. Data from the Framingham Heart Study indicate that blood pressure values in the range of 130 to 139 mm Hg systolic and 85 to 89 mm Hg diastolic are associated with a more than 2-fold increase in the relative risk of cardiovascular disease when compared with blood pressure levels below 120/80 mm Hg (4.5). Based on this emerging data on the lifetime risk of hypertension and the impressive increase in the risk of cardiovascular complications associated with levels of blood pressure previously considered normal (140/90 mm Hg), the JNC 7 has established a blood pressure classification system that contains a prehypertension category and ranges for staging hypertension (2). (Refer to Table III-13.)

Tuble III 10, jile 7 clussification of blood i ressure for fluits figer 10 rears and ofder (2)			
Category	Systolic (mm Hg)	Diastolic (mm Hg)	
Optimal ^a	<120	and <80	
Prehypertension ^b	120-139	or 80-89	
Stage 1 hypertension ^c	140-159	or 90-99	
Stage 2 hypertension ^c	<u>≥</u> 160	or ≥ 100	

^aOptimal blood pressure with respect to cardiovascular risk is less than 120/80 mm Hg. Unusually low readings should be evaluated.
 ^bPrehypertension is not a disease category, rather it is a designation that identifies individuals at high risk of developing hypertension. Lifestyle modifications are recommended to reduce the risk for developing hypertension. For patients who have diabetes mellitus or kidney disease, drug therapy should be considered if a trial of lifestyle modifications fails to reduce blood pressure to 130/80 mm Hg or less.

^cBased on the average of two or more properly measured blood pressure readings taken while seated during two or more visits

Classification and Approaches to Treatment

The classification system introduced by the JNC 7 includes a prehypertension category for individuals who have blood pressure readings of 120 to 139 mm Hg systolic or 80 to 89 mm Hg diastolic. Prehypertension is not a disease category, rather it is a designation that identifies individuals at high risk of developing hypertension (2). Individuals in the prehypertension category are advised to adopt lifestyle modifications, including dietary modifications and physical activity, to reduce their risk of developing hypertension. For patients who have prehypertension and diabetes mellitus or kidney disease, drug therapy should be considered if lifestyle modifications fail to reduce blood pressure to 130/80 mm Hg (2).

The JNC 7 does not stratify individuals with hypertension by the presence or absence of risk factors or target organ damage. The JNC 7 suggests lifestyle modifications as an adjunctive therapy to pharmacotherapy for all persons who have stage 1 or stage 2 hypertension (2). The treatment goal for individuals who have hypertension and no other medical conditions is a blood pressure of 140/90 mm Hg or less (2). For individuals who have hypertension and diabetes mellitus or kidney disease, the treatment goal is 130/80 mm Hg (2). The JNC 7 indicates that greater attention should be provided to the monitoring and evaluation of systolic blood pressure as a major risk factor for cardiovascular disease (2). The increase in systolic blood pressure continues throughout life, in contrast to diastolic blood pressure, which increases until 50 years of age and then tends to level-off during the next decade. Systolic hypertension is the most common form of hypertension in people older than 50 years. The diastolic blood pressure is a more potent cardiovascular risk factor than systolic blood pressure prior to 50 years of age; thereafter, systolic blood pressure is more important (2). (Refer to Table III-14.)

Blood Pressure Classification	Systolic ^a (mm Hg)	Diastolicª (mm Hg)	Lifestyle Modification	Drug Therapy With No Medical Conditions	Drug Therapy With Existing Medical Conditions ^b
Normal	<120	and <80	Encourage	Not indicated	Not indicated
Prehypertension	120-139	or 80-89	Yes	Not indicated	Indicated in cases of diabetes or kidney disease ^c
Stage 1 hypertension	140-159	or 90-99	Yes	Thiazide-type diuretics for most patients Consider ACEI, ARB, BB, CCB, or combination	Drugs specific for medical conditions Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed
Stage 2 hypertension	>160	or >100	Yes	Two-drug combination for most ^d patients (thiazide-type diuretic and ACEI, ARB, BB, or CCB)	Drugs specific for medical conditions Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed

Table III-14: JNC 7 Classification and Management of Blood Pressure for Adults (6)

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta blocker; CCB, calcium channel blocker ^aTreatment is determined by highest blood pressure category.

^bMedical conditions include: heart failure, postmyocardial infarction, high risk of coronary disease, chronic kidney disease, and recurrent stroke prevention.

^c Treat patients who have chronic kidney disease or diabetes to achieve the blood pressure goal of <130/80 mm Hg.

^d Initial combined therapy should be used cautiously in patients at risk for orthostatic hypotension.

Evaluation

According to the JNC 7, the evaluation of patients with documented hypertension has three objectives (2): 1) assess lifestyle and identify other cardiovascular risk factors or concomitant disorders that affect prognosis and guide treatment, 2) reveal identifiable causes of high blood pressure (refer to Table III-15), and 3) assess the presence or absence of target organ damage and cardiovascular disease.

Table III-15: Causes of Hypertension (2)

Sleep apnea

Drugs such as cyclosporine, nonsteroidal anti-inflammatory drugs, sympathomimetics (decongestants, anorectics), oral contraceptive hormones, adrenal steroid hormones, and erythropoietin

Herbal supplements such as ephedra (ma huang) and bitter orange

Chronic kidney disease Primary aldosteronism Renovascular disease Chronic steroid therapy and Cushing's syndrome Pheochromocytoma Coarctation of the aorta Thyroid or parathyroid disease

Causal factors for hypertension include excess body weight; excess dietary sodium intake; reduced physical activity; inadequate intake of fruits, vegetables, and potassium; and excess alcohol intake (1,7,8). According to the JNC 7, the prevalence of these characteristics is high. Recent data indicate that 64% of American adults are either overweight or obese (9). Mean sodium intake is 4,100 mg/day for men and 2,750 mg/day for women. The consumption of processed foods accounts for 75% of the daily sodium intake (2). Fewer than 20% of Americans engage in physical activity (10), and fewer than 25% consume five or more servings of fruits and vegetables daily (11).

Nutritional Assessment and Evaluation

Routine nutritional assessment should include blood pressure evaluation (Grade IV)* (12); assessment for signs of edema; and review of laboratory tests that assess blood glucose, hematocrit, serum potassium, calcium, creatinine or glomerular filtration rate, and lipid profiles (2). The patient's height and weight should be measured, and the patient's body mass index should be evaluated to assess the need for weight management

Hypertension

as an adjunct to treatment. These parameters should be assessed to estimate risk for disease and to identify treatment options (Grade IV) (12). The food and nutrition history should assess the patient's intake of sodium, potassium, and calcium and the frequency at which the patient consumes fruits, vegetables, low-fat dairy products, and processed food items. The patient should be advised not to use herbal supplements, such as ephedra (ma huang) and bitter orange,that increase blood pressure (2). Interactions between antihypertensive medications and nutrients or foods should be examined (Grade IV) (12). Patients who take monoamine oxidase inhibitors should be advised that consumption of licorice and tyramine-containing foods will increase their blood pressure (2). Based on the nutritional assessment and the patient's concomitant conditions, refer to the following sections for nutritional management:

Section C: "Medical Nutrition Therapy for Diabetes Mellitus" Section C: "Calorie-Controlled Diet for Weight Management" Section C: "Medical Nutrition Therapy for Disorders of Lipid Metabolism" Section G: "Medical Nutrition Therapy for Chronic Kidney Disease"

A treatment goal of less than 140/90 mm Hg is recommended for individuals who do not have comorbidities (Grade IV) (12). This blood pressure level is associated with preventing target organ damage and decreasing cardiovascular risk factors and complications (Grade IV) (12). For individuals who have hypertension and diabetes or renal disease, a treatment goal of less than 130/90 mm Hg is recommended (Grade IV) (12). These individuals are at an increased risk for cardiovascular and renal morbidity and mortality (Grade IV) (12).

Accurate Blood Pressure Measurement

The ausculatory method of blood pressure measurement with a properly calibrated and validated instrument is recommended (6,7). The patient should be seated quietly for at least 5 minutes in a chair (rather than on an exam table), with feet on the floor and an arm supported at heart level. Caffeine, exercise, and smoking should be avoided for at least 30 minutes prior to blood pressure measurement (2). An appropriate-sized cuff (cuff bladder encircling at least 80% of the arm) should be used to ensure accuracy. At least two measurements should be made. The systolic blood pressure is the point at which the first two or more sounds are heard (phase 1), and the diastolic blood pressure is the point before the disappearance of sounds (phase 5) (6).

Hypertension in Children and Adolescents

In children and adolescents, hypertension is defined as elevated blood pressure that persists on repeated measurement at the 95th percentile or greater for age, height, and sex (2). Chronic hypertension is becoming increasingly common in adolescence and is associated with obesity, a sedentary lifestyle, and a family history of hypertension or other cardiovascular diseases (2). Lifestyle interventions should be recommended for all children and adolescents with hypertension (2). Pharmacological therapy is recommended for children and adolescents who have higher levels of blood pressure or who do not sufficiently respond to lifestyle modifications (2).

Benefits of Lowering Blood Pressure and Lifestyle Modification Intervention

Major lifestyle modifications that lower blood pressure include limiting sodium intake to no more than 2,300 mg/day (Grade I) (12); weight reduction for individuals who are overweight or obese (Grade IV) (12); adoption of the Dietary Approaches to Stop Hypertension (DASH) eating plan, which is a diet rich in potassium and calcium and lower in sodium, dietary cholesterol, saturated fat, and total fat (<27% of total energy) (Grade IV) (12,13); physical activity (Grade IV) (12); and moderation of alcohol consumption (Grade IV) (2,12). (See Table III-16) Lifestyle modifications reduce blood pressure, prevent or delay the incidence of hypertension, enhance antihypertensive drug efficacy, and decrease cardiovascular risk (Grade IV) (2,6,12). The effects of a DASH eating plan that limits daily sodium intake to 1,500 mg are similar to the effects of single-drug therapy (13). Combinations of two or more lifestyle modifications can achieve even better results (2). Modifiable lifestyle factors have significant blood pressure–lowering effects, and the adoption of a healthy lifestyle is an indispensable part of the management of hypertension (2,8).

Lifestyle Modification	Rationale
Tobacco avoidance	Although not directly related to hypertension, tobacco use may impair the protective effect of antihypertensive medications on coronary heart disease (2).
Weight reduction(if heavier than ideal weight)	Research has shown a direct positive correlation between body weight or body mass index and blood pressure (2,14,15). Weight reduction by energy restriction may result in a substantial decrease in blood pressure. As little as 4.5 kg of weight loss is associated with reductions of 4 to 5 mm Hg systolic and 2 to 4 mm Hg diastolic pressure (16). Reductions of 5 to 20 mm Hg systolic blood pressure occur with every 10 kg of weight loss (Grade IV) (12-15).
Moderate alcohol intake	Moderate consumption of alcohol (<30 g of ethanol per day or two drinks per day) is not associated with blood pressure increases (2). Larger amounts of alcohol ingestion have a dose-related effect on blood pressure in both hypertensive and normotensive subjects (8). Consumption of more than 2 oz of ethanol per day may cause elevated blood pressure and resistance to antihypertensive treatment (2). Hypertensive patients should limit their alcohol consumption. Hypertensive men should consume no more than 1 oz of ethanol per day (equivalent to two drinks) (Grade IV) (2,12). Hypertensive women should consume no more than 0.5 oz of ethanol (one drink) per day (Grade IV) (2,12). A reduction in alcohol consumption may reduce systolic blood pressure by approximately 2 to 4 mm Hg (Grade IV) (12). One drink is equivalent to 12 oz of beer, 5 oz of wine, or 1.5 oz of 80-proof liquor (2). Rebound hypertension frequently occurs during alcohol withdrawal, but it generally reverses within a few days to 6 weeks (17).
Physical activity	Regular aerobic activities, such as walking, jogging, or swimming, may aid in the prevention and treatment of hypertension (2). Regular physical activity can enhance weight loss, reduce the risk of coronary heart disease (17), and prevent the increase in blood pressure that is associated with aging (17). Regular exercise can reduce systolic blood pressure by approximately 4 to 9 mm Hg (Grade IV) (12,17,18). Since exercise can initially increase blood pressure, patients should consult their physician before beginning an exercise program (1,17). The JNC 7 guidelines are consistent with other national guidelines and recommend at least 30 minutes of aerobic activity on most days of the week (Grade IV) (2,12).
Moderate sodium intake	Dietary sodium intake should be limited to 2,300 mg/day (100 mmol/day) or less (Grade I) (2,12). African Americans, older people, and patients who have hypertension or diabetes tend to be more sensitive to dietary changes in sodium intake (2,13,19,20). The <i>Dietary Guidelines for Americans</i> suggests that persons who are 51 and older and those of any age who are African Americans or have hypertension, diabetes or chronic kidney disease should not consume more than 1,500 mg/day of sodium (21). Dietary sodium reduction is associated with a 2 to 8 mm Hg reduction in systolic blood pressure (Grade I) (12,13,19,20). According the Academy, if a patient demonstrates adherence to a 2,300 mg/day sodium diet but has not achieved the treatment goal, then the DASH dietary pattern or a reduction in sodium to 1,600 mg/day can further reduce blood pressure (Grade I) (12). (See "Sodium-Controlled Diet" in Section IF.)
Adequate calcium intake	Population studies have shown an inverse association between blood pressure and calcium intake (2,13,22). However, no evidence suggests that the calcium intake should be increased beyond the Dietary Reference Intake (DRI) (2,12,20). Epidemiological studies have found that dietary patterns that do not meet the DRI for calcium are associated with increased blood pressure (Grade II) (12). The DASH eating plan, which significantly reduces blood pressure, provides 1,240 mg/day of calcium based on a 2,000 kcal combination diet (23).

 Table III-16: Evidence Supporting Lifestyle Modifications to Manage Hypertension

_

Table III-16: Evidence S	apporting Lifestyle Modifications to Manage Hypertension
Lifestyle Modification	Rationale

Factor	
Adequate potassium intake	Observational studies suggest that increased consumption of potassium is associated with a lower incidence of stroke (17). High potassium intake may also be protective against hypertension (2,13,22). The diet should emphasize the consumption of foods rich in potassium, except when contraindicated (eg, patients who receive angiotensin-converting enzyme inhibitors or who have renal insufficiency). The JNC 7 does not specify a potassium intake level. However, the JNC 6 recommended a potassium intake of 3,510 mg/day (90 mmol/day) from food sources such as fresh fruits and vegetables. The latest DRIs for potassium have been increased to 4,500 mg/day for adults to provide a protective effect against hypertension (23). Potassium intakes that do not meet the DRIs are associated with increased blood pressure (Grade II) (12). The DASH eating plan, which significantly reduces blood pressure, provides 4,700 mg/day of potassium based on a 2,000 kcal combination diet (13,19,20,24). The <i>Dietary Guidelines for Americans</i> suggests that individuals who have hypertension, African Americans, and middle-aged and older adults should meet the potassium recommendation of 4,700 mg/day from food sources (21). The best sources of potassium are fruits from vines, leafy green vegetables, and root vegetables (21). Although meat, milk, and cereal products contain potassium, the form of potassium in these foods is not as readily absorbed (21).
Adequate magnesium intake	Because of its vasodilative properties, magnesium may have beneficial effects on hypertension (17). However, no evidence suggests that patients should increase their magnesium intake beyond the DRI (Grade II) (2,12,22). The DASH eating plan, which significantly reduces blood pressure, provides 500 mg/day of magnesium based on a 2,000 kcal combination diet (24). Dietary patterns that do not meet the DRI for magnesium may be associated with increased blood pressure (Grade II) (12).
DASH eating plan	The DASH clinical study demonstrated that a diet (referred to as a combination diet) that is rich in fruits and vegetables (five to ten servings) and low-fat dairy food and reduces the intake of saturated fat (6%), total fats (<27% energy), and sodium (<2,400 mg/day and 1,500 mg/day) significantly lowers blood pressure (Grade I) (12,13,19-25). The DASH eating plan that limits sodium intake to 1,500 mg/day provides the greatest blood pressure reductions (13,19). Following the DASH eating plan is associated with an 8 to 14 mm Hg reduction in systolic blood pressure (13,19). The DASH 2,000 kcal combination diet also provides 31 g/day of fiber (21). The JNC 7 and the Academy of Nutrition and Dietetics recommend the DASH eating plan for the treatment of hypertension (Grade IV) (2,12). The serving sizes from the <i>Dietary Guidelines for Americans</i> are used as the reference guide in the DASH eating plan (21). The DASH eating plan provides 2,000 kcal/day, however it can be modified to meet lower or higher energy needs (26). (Refer to Tables III-17 and III-18 for details regarding the DASH eating plan (23, 26).

Table III-17: DASH Eating Plan (26)				
Food Group	Daily Servings	Serving Sizes	Examples and Notes	Significance to the DASH Eating Plan
Grains and grain products	Seven to eight	One slice of bread 1 oz dry cerealª ½ cup cooked rice, pasta, or cereal	Whole wheat bread, English muffin, pita bread, bagel, cereals, grits, oatmeal, crackers, unsalted pretzels and popcorn	Major source of energy and fiber Select unsalted or lower sodium products

Food Group	Daily Servings	Serving Sizes	Examples and Notes	Significance to the DASH Eating Plan
Vegetables	Four to five	1 cup raw leafy vegetables ½ cup cooked vegetables 6 oz vegetable juice	Tomatoes, potatoes, carrots, green peas, squash, broccoli, turnip greens, collard, kale, spinach, artichokes, green beans, lima beans, sweet potatoes	Rich sources of potassium, magnesium, and fiber Select lower salt canned vegetables or tomato juice.
Fruits	Four to five	6 oz fruit juice One medium fruit ¼ cup dried fruit ½ cup fresh, frozen, or canned fruit	Apricots, bananas, dates, grapes, oranges, orange juice, grapefruit, grapefruit juice, mangoes, melons, peaches, pineapples, prunes, raisins, strawberries, tangerines	Important sources of potassium, magnesium, and fiber
Low-fat or fat- free dairy	Two to three	8 oz milk 1 cup yogurt 1½ oz cheese	Fat-free or low-fat (1%) milk, fat-free or low-fat buttermilk, fat-free or low-fat regular or frozen yogurt, fat-free or low-fat cheese	Major sources of calcium and protein Monitor sodium content of processed cheeses (600 mg of sodium) and natural cheeses (110-450 mg of sodium)
Meats, poultry, and fish	Two or less	3 oz cooked meat, poultry, or fish	Select only lean meats; trim away visible fats; broil, roast, or boil, instead of frying; remove skin from poultry	Rich sources of protein and magnesium Limit ham and processed meats that contain sodium
Nuts, seeds, and dry beans	Four to five per week	 1/3 cup or 1½ oz nuts 2 tbsp or ½ oz seeds ½ cup cooked dry beans or peas 	Almonds, filberts, mixed nuts, peanuts, walnuts, sunflower seeds, kidney beans, lentils	Rich sources of energy, magnesium, potassium, protein, and fiber Select unsalted versions
Fats and oils ^b	Two to three	1 tsp soft margarine 1 tbsp low-fat mayonnaise 2 tbsp light salad dressing 1 tsp vegetable oil	Soft margarine, low-fat mayonnaise, light salad dressing, vegetable oil (such as olive, corn, canola, or safflower oil)	DASH has 27% of total energy as fat, including fat in or added to foods Select low-salt versions of dressing
Sweets	Five per week	1 tbsp sugar 1 tbsp jelly or jam ½ oz jelly beans 8 oz lemonade	Maple syrup, sugar, jelly, jam, fruit-flavored gelatin, jelly beans, hard candy, fruit punch, sorbet, ices	Sweets should be low in fat

^aOne ounce equals ½ to 1¼ cups, depending on the type of cereal. Check the product's Nutrition Facts label.

^bFat content changes serving counts for fats and oils. For example, 1 tbsp of regular salad dressing equals one serving; 1 tbsp of low-fat dressing equals one half of a serving; and 1 tbsp of fat-free dressing equals zero servings.

Food Group	Servings per day for the 1.600 kcal DASH Eating Plan	Servings per day for the 3.100 kcal DASH Eating Plan
Grains and grain products	Six	12 to 13
Vegetables	Three to four	Six
Fruits	Four	Six
Low-fat or fat-free dairy foods	Two to three	Three to four
Meats, poultry, and fish	One to two	Two to three
Nuts, seeds, and dry beans	Three per week	One
Fats and oils	Two	Four
Sweets	One	Two

Table III-18: DASH Eating Plan-Number of Servings for Other Energy Levels

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988-2000. *JAMA*. 2003;290:199-206.
- 2. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ, the National High Blood Pressure Education Program Coordinating Committee. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42:1206-1252.
- 3. Vasan RS, Beiser A, Seshadri S, Larson MG, Kannel WB, D'Agostino RB, Levy D, **[**for the Framingham Heart Study. Residual lifetime risk for developing hypertension in middle-aged women and men: the Framingham Heart Study. *JAMA*. 2002;287:1003-1010.
- Lewington S, Clarke R, Qizilbash N, Peto R, Collins R, for the Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet.* 2002; 360:1903-1913.
- 5. Vasan RS, Larson MG, Leip EP, Evans JC, O'Donnell CJ, Kannel WB, Levy D. Impact of high-normal blood pressure on the risk of cardiovascular disease. *N Engl J Med*. 2001;345:1291-1297.
- 6. JNC 7 Express: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Bethesda, Md: US Dept of Health and Human Services; 2003. NIH Publication No. 03-5233.
- 7. World Hypertension League. Measuring your blood pressure. Available at: http://www.mco.edu/org/whl/bloodpre.html. Accessed April 1, 2003.
- 8. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, Roccella EJ, Stout R, Vallbona C, Winston MC, Karimbakas J, for the National High Blood Pressure Education Program Coordinating Committee. Primary prevention of hypertension: clinical and public health advisory from the National High Blood Pressure Education Program. *JAMA*. 2002;288:1882-1888.
- 9. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among U.S. adults, 1999-2000. JAMA. 2002;288:1723-1727.
- 10. US Dept of Health and Human Services. *Physical Activity and Health: A Report of the Surgeon General.* Atlanta, Ga: US Dept of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996. Available at: http://www.cdc.gov/nccdphp/sgr/contents.htm.
- 11. Division of Adult and Community Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention. Five a day surveillance: behavioral risk factor surveillance system online prevalence data, 1995-2000. Available at: http://apps.nccd.cdc.gov/5ADaySurveillance/. Accessed November 2003.
- 12. *Hypertension Evidence-Based Nutrition Practice Guideline*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.andevidencelibrary.com. Accessed February 2013.
- 13. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER 3rd, Simons-Morton DG, Karanja N, Lin PH, for the DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med.* 2001; 344:3-10.
- 14. Trials of Hypertension Prevention Collaborative Research Group. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The Trials of Hypertension Prevention, phase II. *Arch Intern Med.* 1997;157:657-667.
- 15. He J, Whelton PK, Appel LJ, Charleston J, Klag MJ. Long-term effects of weight loss and dietary sodium reduction on incidence of hypertension. *Hypertension*. 2000;35:544-549.
- 16. Ernst N, Obarzanek E, Clark MB, Briefel R, Brown C, Donato K. Cardiovascular health risks related to overweight. *J Am Diet Assoc.* 1997;97:S47-S51.
- 17. Alderman MH. Non-pharmacological treatment of hypertension. Lancet. 1994;344:307-311.
- 18. Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med.* 2002;136:493-503.
- 19. Vollmer WM, Sacks FM, Ard J, Appel LJ, Bray GA, Simons-Morton DG, Conlin PR, Svetkey LP, Erlinger TP, Moore TJ, Karanja N, for the DASH-Sodium Trial Collaborative Research Group. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-sodium trial. *Ann Intern Med*. 2001;135:1019-1028.
- 20. Chobanian AV, Hill M. National Heart, Lung, and Blood Institute Workshop on Sodium and Blood Pressure: a critical review of current scientific evidence. *Hypertension*. 2000;35:858-863.
- Dietary Guidelines for Americans 2010. Available at: http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf. Accessed Jan 31, 2011.
- 22. Sacks F, Willett WC, Smith A, Brown L, Rosner B, Moore T. Effect on blood pressure of potassium, calcium, and magnesium in women with low habitual intake. *Hypertension*. 1998;31:131-138.

- 23. Svetkey LP, Simons-Morton D, Vollmer WM, Appel LJ, Conlin PR, Ryan DH, Ard J, Kennedy BM, for the DASH Research Group. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomized clinical trial. *Arch Intern Med.* 1999;159:285-293.
- 24. Institute of Medicine. *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate.* Washington, DC: National Academy Press; 2004.
- 25. Appel L, Moore TJ, Obarzanek E, Vollmer W, Svetkey L, Sacks FM, Bray AG, Vogt TM, Cutler JA, Windhauser MM, Pao-Hwa L, Karanja N, for the DASH Collaborative Research Group. A clinical trial of the effects of dietary patterns on blood pressure. N Engl J Med. 1997;336:1117-1123.
- 26. DASH Eating Plan. Bethesda, Md: US Dept of Health and Human Services; revised April 2006. NIH Publication No. 06-4082.

HYPERTRIGLYCERIDEMIA

Discussion

Elevated serum triglyceride levels are positively correlated with the risk for coronary heart disease (1). This relationship is complex and may be explained by the association between high triglyceride levels, low levels of high-density lipoprotein (HDL) cholesterol, and unusually atherogenic forms of low-density lipoprotein (LDL) cholesterol. A high triglyceride level may also reflect an increased level of triglyceride-rich lipoproteins, known as very low-density lipoproteins, which have atherogenic effects (1).

The National Cholesterol Education Program Adult Treatment Panel III (ATP III) developed a classification system for triglyceride levels to facilitate cholesterol management. The classification system and guidelines for cholesterol management are presented in "Medical Nutrition Therapy for Disorders of Lipid Metabolism" in Section IC (1).

Triglyceride Category	Serum Triglycerides (mg/dL)
Normal	<150
Borderline-high	150-199
High	200-499
Very high	<u>></u> 500

Causes of hypertriglyceridemia include:

- obesity
- excessive alcohol intake
- uncontrolled type 1 diabetes mellitus
- type 2 diabetes mellitus (degree often parallels obesity)
- nephrotic syndrome
- thiazide diuretics
- beta adrenergic blocking agents
- hypothyroidism
- chronic renal disease
- obstructive liver disease

Nutrition Assessment and Diagnosis

Medical nutrition therapy is indicated in patients who have triglyceride levels greater than 150 mg/dL (Grade III)* (1,2). As part of the nutrition assessment, the patient's lipid panel, which includes total cholesterol, LDL cholesterol, and HDL cholesterol, should be comprehensively evaluated. Individuals who have hyperlipidemia secondary to familial disorders or other diseases such as diabetes often have borderline-high or high triglyceride levels (1). Causes of hypertriglyceridemia should be evaluated and nutrition intervention targeted to promote optimal nutrition and metabolic outcomes. Complementary drug therapy that includes LDL-lowering medications, fibrates, or nicotinic acid is indicated to prevent acute pancreatitis or abdominal pain when triglyceride levels are very high ($\geq 500 \text{ mg/dL}$) (1).

Nutrition Intervention (1-2)

An energy-controlled, cardioprotective dietary pattern that avoids extremes in carbohydrate and fat intake, limits alcohol and refined sugar, increases the intake of complex carbohydrates, and includes physical activity should be used for patients who have elevated triglycerides ($\geq 150 \text{ mg/dL}$) (Grade III) (2). Weight loss of 7% to 10% of body weight should be encouraged, if indicated. Lifestyle changes that lower triglyceride levels include (Grade III) (2):

- control of body weight
- diet low in saturated fat and cholesterol
- regular exercise
- limiting alcohol intake
- restriction or dietary modification of simple and refined sugars

The ideal macronutrient composition for lowering triglyceride levels in patients who have hypertriglyceridemia is unclear (Grade III) (2). The dietitian should refer to the ATP III guidelines for the suggested nutrition and lifestyle interventions based on the triglyceride level and therapy goals (Grade II) (1.2). A very low-fat diet (<15% of energy) in addition to lifestyle changes reduces triglyceride levles and the risk of pancreatitis in persons who have very high triglycerides ($\geq 500 \text{ mg/dL}$) (Grade II) (2).

Triglyceride Level	ATP III Recommendations (1)
Borderline-high (150-199 mg/dL)	weight reductionincreased physical activity
High (200-499 mg/dL)	 weight reduction increased physical activity drug therapy (LDL-lowering drugs and/or nicotinic acid or fibrates)
Very high (≥500 mg/dL)	 very low-fat diet (<15% of energy intake) to prevent acute pancreatitis weight reduction increased physical activity drug therapy (LDL lowering-drugs and/or nicotinic acid or fibrates)

Table III-19: Recommendations for Elevated Triglyceride Levels

Elevated Triglycerides and Eicosapentaenoic Acid/Docosahexaenoic Acid (EPA/DHA) Supplements

High doses of supplemental EPA/DHA lower triglyceride levels in patients who have elevated triglycerides. If a patient has a high triglyceride level (>200 mg/dL) that is refractory to other lifestyle therapies, physicianprescribed supplemental EPA/DHA capsules (2 to 4 g) can be considered (Grade II) (2). Refer to the discussion of omega-3 fatty acids in Section C: "Medical Nutrition Therapy for Disorders of Lipid Metabolism".

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, 1. Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001; 285: 2486-2497.
- Disorders in Lipid Metabolism Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis 2 Library. Academy of Nutrition and Dietetics; 2011. Available at: http://www.andevidencelibrary.com. Accessed January 15, 2013.
HYPOGLYCEMIA

Discussion

There are two primary categories of hypoglycemia: fasting and postprandial (reactive) hypoglycemia. True hypoglycemia (nondiabetic origin) is a clinical syndrome with diverse causes in which low levels of plasma glucose eventually lead to neuroglycopenia (1). Hypoglycemia is defined as the presence of the following three features, known as Whipple's triad (1):

- a low level of plasma glucose
- symptoms of hypoglycemia, such as sweating, shaking, weakness, hunger, headaches, and irritability, that occur at the same time as the low–blood glucose values
- amelioration of the symptoms by the ingestion of carbohydrate

Maintaining blood glucose levels within a normal range is important because the brain and central nervous system must have a steady supply of glucose to function properly. Symptoms can be recognized when the blood glucose level is 60 mg/dL, and impaired brain function can occur at a level of approximately 50 mg/dL (1). When blood glucose levels fall below normal limits within 2 to 5 hours after eating, this condition is referred to as reactive or postprandial hypoglycemia (1). Postprandial hypoglycemia most frequently occurs as alimentary hypoglycemia (dumping syndrome) in adults who have undergone gastric surgery, such as a Billroth gastrectomy. The episode of hypoglycemia usually occurs $1\frac{1}{2}$ to 5 hours after meals, especially carbohydrate-rich meals. Refer to the "Dumping Syndrome Diet" in Section IB.

Fasting hypoglycemia may occur in response to not eating for 8 hours or longer. Other less common causes are pancreatic tumors (insulinoma), pancreatic islet cell disease, severe heart failure, and critical organ failure. Certain medications, such as exogenous insulin, sulfonylureas, ethanol, salicylates, pentamidine, and quinine, may also cause hypoglycemia in some patients. Diet therapy is the primary treatment, and, in some cases, adjustments in medications are also needed. Surgery may be required for some conditions, such as insulinoma. The most frequent cause of fasting hypoglycemia results from the use of insulin or oral glucose-lowering medications in the treatment of diabetes mellitus. Refer to "Medical Nutrition Therapy for Diabetes Mellitus" in Section IC.

There are currently no consensus guidelines for the diagnosis of reactive hypoglycemia (1). The diagnostic techniques range from confirming that the blood glucose level is low during a hypoglycemic reaction after an ordinary meal to performing an oral glucose tolerance test. However, 10% of asymptomatic healthy persons respond to the oral glucose tolerance test with a lower-than-normal glucose level.

Nutrition Intervention

The goal of treatment is for the patient to adopt eating habits that will keep blood glucose levels as consistent as possible (1). These eating habits include consistent meal times and consistent carbohydrate intake (1). As part of meal planning, determine the frequency and symptoms of hypoglycemia, as well as activity levels and exercise frequency, and schedule appropriate times for meals and snacks. The treatment of reactive hypoglycemia depends on the specific cause. The treatment of alimentary hypoglycemia following gastric surgery is discussed in Section IB: "Dumping Syndrome Diet"). Provided below are guidelines for avoiding symptoms of hypoglycemia (1):

- Allow five or six small meals or feedings per day. Eat consistent amounts of carbohydrate at meals and snacks from day to day and avoid skipping meals.
- Spread carbohydrate intake throughout the day. Most individuals need three to four carbohydrate servings at meals and one to two carbohydrate servings for snacks (one serving = 15 g carbohydrate). Include protein foods and vegetables at each meal for satiety and extra energy.
- Avoid foods that contain a large amount of carbohydrates, because reactions can occur as a result of a high carbohydrate load. Examples of these foods are regular soft drinks, syrups, candy, regular fruited yogurts, cookies, pies, and cakes.
- Avoid beverages and foods containing caffeine. Caffeine can cause the same symptoms as hypoglycemia.
- Limit alcohol consumption because it inhibits gluconeogenesis. If an individual chooses to drink alcohol, it should be limited to one drink per day for women and two drinks per day for men. Drinking alcohol on an empty stomach and without food can cause hypoglycemia. A carbohydrate food should always be consumed along with the alcoholic beverage.

Glucagon injections are used to treat severe hypoglycemia. The primary effect of glucagon is to increase blood glucose levels by accelerating hepatic glycogenolysis and stimulating hepatic gluconeogenesis (2). The most common adverse effect of glucagon is nausea or vomiting (2).

Educate the patient regarding fast-acting carbohydrate foods that should be consumed or avoided depending upon the patient's blood glucose level. Refer to "Treatment of Hypoglycemia" in Section IC: "Medical Nutrition Therapy for Diabetes Mellitus".

References

- 1. Reactive hypoglycemia. In: *Nutrition Care Manual*. Chicago, Ill: Academy of Nutrition and Dietetics; Updated annually. Available at: nutritioncaremanual.org. Accessed January 16, 2008.
- 2. Stuart N, ed. Medications and diabetes: new helps and old friends. *On The Cutting Edge* [Diabetes Care and Education newsletter]. 2006; 27:1-32.

Bibliography

International Diabetes Center. *Reactive and Fasting Hypoglycemia*. Minneapolis, Minn: International Diabetes Center, Park Nicollet Institute; 2004.

INBORN ERRORS OF METABOLISM

Discussion

Inborn errors of metabolism are inherited disorders in which there is an absence or reduced activity of a specific enzyme or cofactor. Impaired metabolism and disease result.

A rational approach to the nutrition intervention of an inborn error of metabolism requires some understanding of the pathogenic mechanisms and resulting consequences. One or both of the following mechanisms may be present, depending on the type of disorder:

- 1. The accumulated substrate or its metabolites may have toxic effects.
- 2. A harmful deficiency may result from the decreased synthesis of a necessary end product.

The rationale for nutrition intervention depends on which of these mechanisms is thought to be important. For example, in galactosemia, the goal is to reduce the accumulation of substrate (galactose and galactose-1-phosphate). In type I glycogen storage disease, the aim is to supply the deficient product (glucose). In phenylketonuria both reduction of the substrate (phenylalanine) and provisions of adequate amounts of the product (tyrosine) must be accomplished.

Successful nutrition intervention for patients with inborn errors requires a keen appreciation of the tremendous variability among individuals and a willingness to tailor the therapeutic approaches to the specific needs of each patient.

Approaches

The following table lists genetic disorders in which nutrition intervention has been employed.

Disorder	Enzyme Defect	Nutrition Intervention	Special Formulas ^a	Possible Outcomes Without Effective Medical Treatment
Phenylketonuria (PKU)	Phenylalanine	Diet low in phenylalanine. Increase tyrosine in diet.	Lofenalac® (MJ), Phenyl-free® (MJ), Phenex® (RL), Maxamaid® (SHS)	Growth delay, mental impairment, seizures
Tyrosinemia	Cytosol tyrosine aminotransferase	Diet low in phenylalanine and tyrosine	Product 3200 AB® (MJ), XYPHEN® (SHS), Tyromex® (RL)	Death, growth delay, mental impairment, renal disease, liver dysfunction, seizures, hypoglycemia, metabolic acidosis, hyperammonia
Maple syrup urine disease (MSUD)	Ketoacid decarboxylase	Diet low in leucine, isoleucine, and valine	MSUD Diet Powder® (MJ), MSUD Maxamaid® (SHS), Ketonex® (RL)	Death, growth delay, mental impairment, seizures, hypoglycemia, metabolic acidosis, hyperammonia

Table III-20: Metabolic Disorders That Respond to Dietary Treatment

Disorder	Enzyme Defect	Nutrition Intervention	Special Formulas ^a	Possible Outcomes Without Effective Medical Treatment
Urea cycle defects	Depends on defect. Enzymes that may be defective include carb- amoylphosphate synthetase, ornithine trans- carbamoylase, angininosuccinic acid synthetase, argininosuccinic acid lyase, arginase	Diet low in protein, +/- L-amino acid(s) missing or inactive with defect. All conditions require a diet low in sodium benzoate, except in the arginase defect.	UCD® (MJ), Cyclinex® (RL)	Death, growth delay, mental impairment, seizures, hyperammonia
Organic acidemia	Depends on defect. Enzymes that may be defective include methlmalonyl coenzyme A (CoA) mutase or coenzyme B ₁₂ or propionyl CoA carboxylase	Diet high in energy and low in protein, +/- L-amino acid(s) missing or inactive with defect. For methlmalonyl CoA mutase or coenzyme B ₁₂ , supplement with B ₁₂ . In acute state, give IV fluids and bicarbonate	OS® (MJ), XMTVI Maxamaid® (SHS), Propimex® (RL)	Death, growth delay, mental impairment, renal disease, seizures, hypoglycemia, metabolic acidosis, hyperammonia
Galactosemia	Galactose-1- phosphate uridyl transferase	Diet free of galactose and lactose		Death, growth delay, mental impairment, liver dysfunction, hypoglycemia
Fructose intolerance	Fructose-1- phosphate aldolase	Diet free of fructose, sucrose, and sorbitol		Death, growth delay, renal disease, liver dysfunction, hypoglycemia, metabolic acidosis
Glycogen storage diseases	Type O: glycogen synthetase	Diet high in protein, frequent carbohydrate feedings; high protein feeding at night		Death, growth delay, renal disease, liver dysfunction, seizures, hypoglycemia, metabolic acidosis
	Type I: glucose 6- phosphatase	Normal diet with high carbohydrate feedings between meals; nasogastric drip at night		
	Type III: debrancher enzyme	Diet with normal energy and high protein intake; night feeding high in protein		
	Type VI: hepatic phosphorylase	High protein diet and frequent feedings		

^aMedical products are made by the following manufacturers: (MJ) Mead Johnson Nutritionals, Evansville, Ind; 800/755-4805 (RL) Ross Laboratories, Columbus, Ohio; 800/986-8755 (SHS) Scientific Hospital Supplies, Gaithersburg, Md; 800/365-7354

Inborn Errors of Metabolism

Bibliography

Trahms CM. Medical nutrition therapy for metabolic disorders. In: Mahan KL, Escott-Stump S, eds. Krause's Food, Nutrition and Diet *Therapy.* 10th ed. Philadelphia, Pa: WB Saunders; 2000: 987-1009. Nutrition management of inborn errors of metabolism. *Pediatric Manual of Clinical Dietetics*.2nd ed. Chicago, Ill: American Dietetic

Association; 2003.

IRON DEFICIENCY ANEMIA

Discussion

For a discussion of assessment, see "Classification of Some Anemias" in Section II.

Treatment should focus primarily on the underlying disease or situation leading to the anemia. The chief treatment of iron deficiency anemia is oral administration of inorganic iron in the ferrous iron form. The most widely used preparation is ferrous sulfite, and the dose is calculated in terms of the amount of elemental iron provided. Depending on the severity of the anemia, daily dosage of elemental iron should be 50 to 200 mg for adults and 6 mg/kg for children. Ascorbic acid greatly increases iron absorption. It takes 4 to 30 days to note improvement with iron therapy, especially the hemoglobin level. Iron therapy should be continued for several months, even after the hemoglobin level is restored, so that the body iron reserves are replete.

In addition to medication, attention should be given to the amount of absorbable iron in food. Dietary modification can be adjunctive to iron administration or can be prophylactic in the individual who is at risk for iron deficiency anemia. The diet can be modified to increase the iron intake for any individual.

Dietary strategies involve:

- 1. providing foods that have a higher iron density
- 2. increasing the iron absorption from food

Iron Density

The normal mixed diet has been said to have an iron density of around 6 mg/1000 kcal. Beef, legumes, dried fruit, and fortified cereals are foods that rank the highest in iron content.

In general, foods that obtain most of their calories from sugar, fat, and unenriched flour have a low iron density. Foods made from whole grain and enriched flour, as well as unrefined foods (fruit, vegetables, and meats), have a higher iron density. Dairy products have a low iron density.

Iron Absorption

The iron content of the body is highly conserved and in the absence of bleeding, little is lost each day. For men and postmenopausal women, for whom the RDA is 8 mg/day of iron, 1 mg of absorbable iron per day will meet this requirement (1).

Dietary iron is provided in the diet in two forms: heme and nonheme. Heme iron constitutes 40% of the iron present in meat, fish, and poultry. Nonheme iron constitutes the balance of the iron in meat, fish, poultry and all the iron present in plant food, eggs, milk, and cheese. Heme iron is better absorbed than nonheme iron. The absorption of nonheme iron is influenced by several dietary enhancing factors, particularly ascorbic acid and meat, fish, and poultry. Ascorbic acid binds iron to form a readily absorbed complex. Good sources of ascorbic acid include, but are not limited to, citrus fruit and juices, tomatoes and tomato juice, greens, broccoli, strawberries, and sweet potatoes.

Iron absorption is also influenced by other factors, such as:

- Nutritional status with respect to iron: Individuals with an iron deficiency will have greater iron absorption.
- The presence of substances that decrease iron absorption: Phytates, tannic acid, carbonates, oxalates, phosphates, ethylenediaminetetraacetic acid (EDTA), phosvitin. Phytates found in unleavened bread, unrefined cereals, and soybeans inhibit iron absorption. Tannic acid found in tea and coffee and phosvitin found in egg yolk have been shown to decrease iron absorption. Calcium phosphate salts and EDTA, a food preservative, can also reduce iron absorption.
- Cooking utensils: Cooking with an iron skillet may contribute minute amounts of iron to the diet.
- Gastric acidity: Subnormal acidity of the gastric juices, fairly common in older persons, can cause them to absorb less dietary iron.

Iron Deficiency Anemia

Approaches

Guidelines to increase iron intake and absorption are as follows:

- Increase ascorbic acid at every meal.
- Include meat, fish, and poultry at each meal, if possible.
- Avoid drinking tea or coffee with meals.
- Avoid foods with high quantities of EDTA by checking food labels.
- Increase food selections that have a high iron density.

Food	Amount	Iron (mg)
Sources of heme iron:		
Beef, cooked, lean	1 oz	0.7
Chicken, cooked	1 oz	0.4
Cod, cooked	1 oz	0.14
Egg	1 large	0.6
Liver, beef, cooked	1 oz	1.9
Liver, chicken, cooked	1 oz	2.4
Oysters, cooked	6 medium	5.0
Pork, cooked, lean	1 oz	0.33
Sources of nonheme iron:		
Apricots, dried	4	0.6
Bread, enriched	1 slice	0.7
Bread, whole wheat	1 slice	0.8
Cereal, dry, fortified	1 cup	4.5 - 18.0
Cream of wheat, cooked	³ ⁄4 cup	9.0
Farina, cooked, enriched	½ c	7.4
Green beans	½ cup	0.9
Greens, turnip, cooked	½ cup	1.0
Kale, cooked	½ cup	0.6
Kidney beans, cooked	½ cup	2.6
Lentils, cooked	½ cup	3.3
Molasses, blackstrap	1 tbsp	3.5
Pasta, cooked, enriched	½ cup	1.25
Peanut butter	2 tbsp	0.6
Prunes	5	1.0
Prune juice	½ cup	1.5
Raisins	1/3 cup	1.0
Spinach, cooked	½ cup	1.4

Table III-21: Iron Content of Common Foods

Source: Pennington JAT. Bowes & Church's Food Values of Portions Commonly Used. Philadelphia, Pa: Lippincott; 1998.

Reference

 Institute of Medicine. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Food and Nutrition Board, Washington, DC: National Academies Press,2002:290-292; at http://www.nap.edu.books/0309072794/html/index.html, 2002 (accessed 1/28/03).

Bibliography

Food and Nutrition Board, National Research Council: Recommended Dietary Allowances. 10th ed. Washington, DC: National Academy of Sciences; 1989.

Kasdan TS. Medical nutrition therapy for anemia. In: Mahan KL, Escott-Stump S, eds. *Krause's Food, Nutrition and Diet Therapy.* 10th ed. Philadelphia, Pa: WB Saunders; 2000: 781-800.

NEPHROTIC SYNDROME

Discussion

Nephrotic syndrome is characterized by urinary losses of albumin and other plasma proteins, resulting in hypoalbuminemia. Nephrotic syndrome is caused by the failure of the glomerular capillary wall to act as an impermeable barrier to plasma proteins. A 24-hour urinary protein excretion of 3.0 g or greater is indicative of nephrotic syndrome (1,2). Nephrotic syndrome is associated with other metabolic disturbances including hyperlipidemia caused by increased lipid synthesis and decreased lipid catabolism (2,3). Edema is caused by sodium retention or imbalance, fluid retention, hypoalbuminemia, and underlying diseases such as renal, cardiac, or liver disease (3). Causes of nephrotic syndrome include diabetes mellitus, infections, certain medications, neoplasms, preeclampsia, and chronic allograft nephropathy (2).

Approaches (2,3)	Rationale
 Protein: Provide 0.8 to 1.0 g/kg of ideal body weight. High-biological value protein should contribute at least 50% of protein intake. 	In contrast to the treatment of protein-energy malnutrition, the treatment of nephrotic syndrome does not include a high-protein diet; a high-protein diet would not replenish plasma protein levels and could cause further renal damage in patients who have nephrotic syndrome (2,3). Mild protein restriction and provision of an angiotensin-converting enzyme inhibitor diminish urinary protein losses and increase serum albumin levels (2,3). Soy protein decreases urinary protein excretion and blood lipid levels (2,4). Some studies suggest that a low or very-low protein diet with essential amino acid supplementation reduces proteinuria and improves protein nutriture (5). The recommended protein intake for children who have nephrotic syndrome is the Dietary Reference Intake for age plus the amount of urinary protein loss. Children who have persistent proteinuria may require 2.0 to 2.5 g/kg of protein per day (3).
 Sodium: See "Sodium-Controlled Diet" in Section IF. 	The level of sodium prescribed is based on the severity of edema and hypertension. Sodium is usually restricted to 1 to 2 g/day, depending on the severity of the patient's signs and symptoms (3). Fluid restriction is often necessary and should be based on the patient's symptoms. Diuretics can help maintain fluid and sodium balance (3).
Energy:Calculate according to individual needs.	The energy intake requirement is determined by the nutritional evaluation and can be as high as35 kcal/kg (3). Complex carbohydrates should be the primary source of energy intake (2). Weight loss may be recommended for obese patients, because they have an increased risk of comorbid diseases and complications.
 Fat: Use the National Cholesterol Education Program Adult Treatment Panel III guidelines (2,3). Refer to Section C: "Medical Nutrition Therapy for Disorders of Lipid Metabolism" for the Therapeutic Lifestyle Changes (TLC) diet. Target <30% of energy from fat, saturated fat <7% of total fat, and cholesterol <200 mg/dL per day (2). 	Hypercholesterolemia or hypertriglyceridemia) results from increased lipid synthesis and decreased lipid catabolism (3). This disturbance in lipid metabolism increases the risk for cardiovascular disease, stroke, and progressive renal failure (2). A combination of statin therapy and the Therapeutic Lifestyle Changes diet lowers serum lipid levels (2). Fish oil supplementation (12 g/day) may be beneficial for patients who have IgA nephropathy, which is a caused by the deposition of immunoglobin A in the kidneys (3).

Approaches (2,3)

Rationale

Vitamins and minerals:

Base intake on food and nutrition assessment and biochemical levels:

- Provide iron, based on the individual patient's need (2).
- Ensure patient is meeting Dietary Reference Intakes for B-complex vitamins (niacin, riboflavin, and thiamin) and vitamin C. Supplement as needed (2).
- Supplement 1 to 1.5 g of calcium, not to exceed 2,000 mg (2).
- Limit phosphorus to <12 mg/kg per day (6).

Abnormalities in iron, copper, zinc, and calcium levels are directly related to the urinary loss of proteins that are involved in their metabolism (2,3). For example, the increased loss of transferrin causes decreased plasma iron levels. Iron supplementation is important for patients who have nephrotic syndrome (3). Copper is also bound to protein, and serum copper levels are often compromised. However, clinical manifestations do not occur as a result of the low copper levels; therefore, supplementation is not necessary (3). Supplemental zinc may be needed, as zinc is bound to albumin (3). In addition, decreased levels of calcium and serum 1,25-dihydroxycholecalciferol may occur as a result of being bound to albumin (3). Supplemental calcium, vitamin D, and iron may be needed to normalize serum levels.

References

- 1. Madaio M, Harrington J. The diagnosis of glomerular diseases: acute glomerulonephritis and nephrotic syndrome. Arch Intern Med. 2001;161:25-34.
- 2. Nephrotic syndrome. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed January 12, 2013.
- 3. Kopple JD, Massry SG, eds. Nutrition Management of Renal Disease. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2004.
- 4. D'Amico G, Gentile MG, Manna G, Fellin G, Ciceri R, Cofano F, Petrini C, Lavarda F, Perolini S, Porrini M. Effect of vegetarian soy diet on hyperlipidaemia in nephrotic syndrome. *Lancet.* 1992;339:1131-1134.
- 5. Giordano M, De Feo P, Lucidi P, DePascale E, Giordano D, Cirillo D, Dardo G, Signorelli SS, Castellino P. Effects of dietary protein restriction on fibrinogen and albumin metabolism in nephrotic patients. *Kidney Int.* 2001;60:235-242.
- 6. McCann L, ed. Pocket Guide to Nutrition Assessment of the Patient with Chronic Kidney Disease. 3rd ed. New York, NY: National Kidney Foundation Council on Renal Nutrition; 2002.

OBESITY AND WEIGHT MANAGEMENT

Discussion

Recent data indicate that 64% of American adults are either overweight (body mass index [BMI] of 25.0 to 29.9 kg/m²) or obese (BMI >30 kg/m²) (1). This figure has sharply increased since 1994 when 55% of American adults were overweight or obese. The rate of obesity has doubled from 15% in 1980 to 30% in 1999 (1,2). The trend is similar in American children and adolescents. The 1999 to 2000 National Health and Nutrition Examination Survey (NHANES) identified the prevalence of overweight as 15.5% among 12-through 19-year-olds; 15.3% among 6- through 11-year-olds; and 10.4% among 2- through 5-year-olds. These percentages are greater than the corresponding values of 10.5%, 11.3%, and 7.2% in 1988 to 1994 (NHANES III) (2). Overweight children have a greater risk for becoming overweight adults (3). Obesity contributes to many adverse health outcomes, including type 2 diabetes, cardiovascular disease, hypertension, stroke, osteoarthritis, gallbladder disease, sleep apnea, respiratory problems, and cancers of the endometrium, breast, prostate, and colon (1,4). The total estimated cost of obesity in 1995 was \$99.2 billion, which includes \$51.6 billion spent on direct medical costs (5).

Obesity is a complex multifactorial disease that results from the positive energy balance that occurs when energy intake exceeds energy expenditure. Lifestyle and environmental factors, including excessive energy intake, high fat intake, and physical inactivity, are associated with the pathophysiology of obesity. Growing evidence suggests a strong link between genetic factors and the pathogenesis of obesity. Genes involved in energy regulation such as leptin, a signal protein for satiety produced in the adipose tissue, and other hormones or peptides, such as neuropeptide Y, may have important implications for understanding the causes of obesity (6). Ongoing research is required to determine the role of genetic factors in obesity treatment.

Adults

The Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (4) provide guidelines for classifying the degree of overweight and obesity and the associated health risk as well as guidelines for developing treatment strategies. The BMI is used to classify the degree of overweight or obesity in adults because it is highly correlated with body fatness (4). The BMI is calculated by dividing weight in kilograms by height in meters squared. Studies have identified a relationship between an elevated BMI (>25 kg/m²) and an increased incidence of morbidity and mortality (1,4). The BMI and waist circumference should be used to classify overweight and obesity, estimate risk for disease, and identify treatment options (Grade II) *(4,7). (Refer to Table III-22.) The BMI and waist circumference are highly correlated to obesity or fat mass and risk of other diseases (Grade II) (4,7). Waist circumference is also used as an assessment parameter because excess fat in the abdomen is an independent predictor of increased risk and morbidity, even for individuals with a normal weight (4). Evidence from epidemiologic studies shows waist circumference to be a better marker of abdominal fat than the waist-to-hip ratio. Waist circumference also is the most practical anthropometric measurement for assessing a patient's abdominal fat content before and during weight loss treatment (4). A high waist circumference is associated with increased risk for type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease in patients whose BMI is between 25.0 and 34.9 kg/m². However, for individuals whose BMI is greater than 35.0 kg/m^2 , waist circumference adds little to the predictive power of the disease risk classification of BMI (4). (Refer to Table III-23)

	cumerence			
	BMI (kg/m²)	Obesity Class	Disease Ris (Relative to Normal Weight and	kª Waist Circumference)
			Men <u><</u> 40 inches (<u><</u> 102 cm)	>40 inches (>102 cm)
			Women <35 inches (<88 cm)	>35 inches (>88 cm)
Underweight	<18.5			
Normal ^b	18.5-24.9			
Overweight	25.0-29.9		Increased	High
Obesity	30.0-34.9	Ι	High	Very high
	35.0-39.9	II	Very high	Very high
Extreme Obesity	<u>></u> 40.0	III	Extremely high	Extremely high

Table III-22: Classification of Overweight and Obesity and Associated Disease Risk^a by BMI and Waist Circumference

^aDisease risk for type 2 diabetes, hypertension, and cardiovascular disease

^bIncreased waist circumference can also be a marker for increased risk in persons of normal weight.

Table III-23: A Guide to Selecting Treatment for Obesity and Overweight

]	BMI (kg/m²)			
	<24.9	25.0-26.9	27.0-29.9	30.0-34.9	35.0-39.9	>40.0
Treatment						
Diet, exercise, and behavior therapy		With comorbidities	With comorbidities	+	+	+
Pharmacotherapy			With comorbidities	+	+	+
Surgery					With comorbidities	+

+ Options for treatment (Note that when the BMI is >29.9 kg/m², adjunctive treatment options should be considered.)

- Prevention of weight gain with lifestyle therapy is indicated in any patient with a BMI >25 kg/m², even without comorbidities. However, weight loss is not necessarily recommended for patients with a BMI of 25.0 to 29.9 kg/m² or a high waist circumference, unless they have two or more comorbidities.
- Combined therapy with a low-energy diet, increased physical activity, and behavior therapy provides the most successful intervention for weight loss and weight management.
- Consider pharmacotherapy only if a patient has not lost 1 lb/week after 6 months of combined lifestyle therapy.

Source for Table III-22 and Table III-23: *The Practical Guide to the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults.* The National Institutes of Health, National Heart, Lung, and Blood Institute, and the North American Association for the Study of Obesity; October 2000. NIH publication No. 00-4084.

Children and Adolescents

Overweight children have a greater risk for becoming overweight adults (3). Whether or not the child is obese, obesity of at least one parent more than doubles the risk of a child being obese as an adult (8). The latest statistics indicate that obesity is more prevalent among non-Hispanic black and Mexican-American adolescents (2). Weight gain among children and adolescents is attributed to a combination of poor dietary habits, family lifestyle, physical inactivity, ethnicity, socioeconomic status, and genetic makeup (9,10). Early intervention is recommended to prevent overweight and obesity from continuing later in life (9).

Recommendations have been established for the intervention and treatment of overweight and obesity in children and adolescents (9). The Expert Committee recommends evaluation and possible treatment for children with a BMI greater than or equal to the 85th percentile with complications of obesity (9). The classification of overweight for children is determined by calculating the BMI and plotting it on the appropriate BMI-for-age chart developed by the Centers for Disease Control and Prevention (11). The Centers for Disease Control and Prevention recommends that the BMI-for-age charts be used for all children and adolescents aged 2 to 20 years, instead of the weight-for-stature charts previously developed by the National Center for Health Statistics (11). Complications of obesity include hypertension, dyslipidemia, orthopedic disorders, sleep disorders, gallbladder disease, and insulin resistance (9). The use of weight maintenance vs weight loss to

achieve weight goals depends on the patient's age, baseline BMI percentile, and the presence of medical complications (9).

The Expert Committee recommends that the first step in the assessment of an overweight child includes evaluation for underlying syndromes, including genetic causes (eg, Prader-Willi syndrome) and endocrinologic causes (eg, hypothyroidism and Cushing syndrome). In addition, a complete medical and psychosocial history should be gathered, and a physical exam should be performed to identify complications. These complications include sleep apnea, pseudotumor cerebri, orthopedic complications, and acanthosis nigricans (the coarse, hyperpigmented areas in the neck folds or axilla that are associated with insulin resistance and type 2 diabetes). Children with eating disorders or symptoms of depression require psychological treatment and should not participate in a weight-control program without the concurrence of a therapist or specialist (9).

Approaches

Children and Adolescents

The primary goal of a program to manage uncomplicated obesity in children and adolescents is healthy eating and activity, not achievement of ideal body weight (9). For children with a secondary complication of obesity (eg, hypertension or dyslipidemia), improvement or resolution of the complication is an important medical goal. The first step in weight control for all overweight children (85th to 94th percentile) older than 2 years is the maintenance of baseline weight. Prolonged weight maintenance, which allows a gradual decline in BMI as children grow in height, is a sufficient goal for many children (9). Weight maintenance is also the goal for children aged 2 to 7 years who have a BMI greater than the 95th percentile and no complications (9). However, if complications (eg, hypertension, dyslipidemia, or insulin resistance) are identified, weight loss is indicated in this group. For children 7 years and older who are overweight (85th to 94th percentile), weight maintenance is the goal if no complications are present, and weight loss is indicated if complications are present (9). Weight loss is indicated independent of complications for children aged 7 years and older who are at or above the 95th percentile (9).

Weight loss achieved by lifestyle approaches, including a low-energy diet and increased physical activity, is recommended (Grade I) (12). Energy intake levels need to be individualized to meet the patient's growth and development needs. Approaches to weight loss should be based on family readiness and involvement (9). The dietary goals for patients and families should include well-balanced, healthy meals and a healthy approach to eating. A low-energy diet (900 to 1,200 kcal/day) as part of a clinically supervised, multicomponent weight loss program is associated with both short-term and longer-term reductions in adiposity among children ages 6 to 12 years (Grade I) (12). A reduced energy diet < 1,200 kcal/day) in the acute treatment phase of adolescent overweight is generally effective for short-term improvement in weight status. However, without continuing intervention weight is regained (Grade I) (12). Counting calories can be tedious and inaccurate (9). As an alternative, targeting high-energy and high-fat foods and beverages in the existing diet and focusing on one or two small dietary changes at a time is suggested (9). Behavior modification approaches, such as the traffic light diet, are very effective (Grade I) (9,12). Children and adolescents must receive adequate vitamins, minerals, protein, and energy to maintain healthy growth. Linear growth may slow during weight loss (9). However, most overweight children are tall and impact on adult stature appears to be minimal (13). Although pharmacological means of treatment are being investigated (9), the only treatment option currently available is for adolescents (12 years or older) who may be treated with Orlistat for up to one year. See Weight Contol Information Network information at http://www.win.niddk.nih.gov. Bariatric surgery (eg, laparoscopic adjustable gastric band (LAGB) and Roux-en-Y gastric bypass (RYGB)) may be a treatment option for children and adolescents who are obese (> 95th percentile) for weight based on age in specialized centers when a severe comorbidity is present (refer to discussion on Surgery in this section).

Like adults, children and adolescents who are obese have an increased risk for vascular disease (9,10). The characteristic pattern consists of elevated serum low-density lipoprotein cholesterol and triglycerides levels and lowered high-density lipoprotein cholesterol levels (14). The American Heart Association (AHA) dietary guidelines for primary prevention of atherosclerotic heart disease recommend that children older than 2 years gradually begin to adopt a diet that contains no more than 30% of energy from fat. A diet low in fat (<30% of total energy, but no less than 20% of total energy), saturated fat (<10% of energy per day), cholesterol (less than 300 mg/day), and low in *trans* fatty acids is encouraged (15,16). Similar to adult guidelines, the AHA suggests daily consumption of five or more servings of fruits and vegetables and six to eleven servings of whole grain foods. In addition, low-fat dairy products, fish, legumes, poultry, and lean

Obesity and Weight Management

meats are recommended (15,16). The AHA recommends that children consume adequate amounts of dietary fiber (equal to the child's age plus 5 g) each day (16,17).

Adequate physical activity also is encouraged (9,15,16,18). The US Surgeon General's Report on Physical Activity and Health recommends 30 minutes per day of moderate to vigorous physical activity for children and adults (18).

Adults

Weight management is defined as the adoption of healthful and sustainable eating and physical activity behaviors indicated for a reduced disease risk and improved feelings of energy and well-being (6). Weight loss therapy should be based on a comprehensive weight management program including diet, physical activity, and behavior therapy. The combination therapy is more successful than any one intervention alone (Grade I) (7). A nonrestrictive approach to eating based on internal regulation of food (hunger and satiety), physical activity, and healthful eating habits should be emphasized (6). Data on lifestyle weight loss interventions indicate that they produce low levels of sustained loss. Typically reported weight losses remaining after 4 to 5 years are about 3% to 5% of initial body weight (6). Based on data from the National Weight Control Registry, long-term maintenance of weight loss and goals is increased in persons who have specific health habits and behaviors. These habits include eating a lower energy diet (average = 1,381 kcal/day) that is low in fat and high in carbohydrates, regular self-monitoring of food intake and activity level, and participating in regular physical activity comparable to 1 hour per day of moderate-intensity physical activity, such as brisk walking (4,19). The National Weight Control Registry registrants who have documented these behaviors have demonstrated sustained weight loss of 10% of initial body weight for at least 1 year (19). Weight loss of 5% to 10% of initial body weight can lead to a substantial improvement in risk factors for diabetes and heart disease and can lead to reductions in or discontinuations of medications for these conditions (4). The results of the Diabetes Prevention Program have provided the most definitive evidence of the health benefits of modest weight loss (20). In this study, the lifestyle intervention group had a 58% reduced risk of developing type 2 diabetes when compared to the placebo group and a 39% reduced risk when compared to the pharmacotherapy intervention group that used metformin (20). Individualized goals of weight loss therapy should be to reduce body weight at an optimal rate of 1 to 2 lb/week for the first 6 months and to achieve an initial weight loss goal of up to 10% from baseline. These goals are realistic, achievable, and sustainable (Grade I) (4,7).

Energy: Energy requirements should be based on individual needs to promote gradual and safe weight loss. Estimated energy requirements should be based on resting metabolic rate (RMR) (7). If possible, RMR should be measured (eg, indirect calorimetry). If RMR cannot be measured, then the Mifflin–St. Jeor equation using actual body weight is the most accurate for estimating RMR for overweight and obese individuals (Grade I) (7). The prescribed energy level should promote a weight loss of 0.5 to 1.5 lb/week (Grade I) (4,7). The recommended minimum energy levels are 1,200 kcal/day for women and 1,400 to 1,500 kcal/day for men (4, 21). Evidence suggests that moderation in total energy is the key variable in promoting weight loss, rather than modification of the diet's macronutrient composition (21). Consideration of a realistic energy goal is important for successful patient compliance with a weight-management program (4). Total energy intake should be distributed throughout the day, with the consumption of four to five meals/snacks per day including breakfast (Grade II) (6,7). Consumption of greater energy intake during the day may be preferable to evening consumption (Grade II) (6,7). (See the "Calorie-Controlled Diet for Weight Management" in Section IC.)

Protein: To preserve lean body mass, daily protein intake should be in the range of 0.8 to 1.2 g/kg of body weight (22). During energy restriction, it is generally suggested that 72 to 80 g of high-quality protein be consumed per day (23). Patients who ingest too little protein (<40 g/day) are at risk for ventricular arrhythmias (23).

Fat: Fat should account for 20% to 30% of total energy. Saturated fats should be limited to less than 6% to 8% of total fat energy. The US Department of Agriculture has found that diets with low to moderate fat intake (15% to 30% of total energy) tend be lower in total energy and highest in diet quality when compared to low-carbohydrate diets (21).

Carbohydrates: Carbohydrates should account for 50% to 60% of total energy. Carbohydrates can help prevent the loss of lean tissue (23). It has been suggested that at least 100 g of carbohydrates should be consumed per day to minimize ketosis (23). Hyperuricemia can result from low-carbohydrate diets (23). Maintaining a minimum level of carbohydrate intake (\geq 100 g/day) reduces the risk of increased uric acid levels that may predispose the patient to gout (23). High-fiber carbohydrate sources, such as fruits, vegetables,

whole grain breads, cereals, and legumes, are recommended. The daily consumption of 20 to 35 g of fiber reduces the energy density of foods consumed and promotes satiety by delaying gastric emptying (17). The US Department of Agriculture has found that diets high in carbohydrate (>55%) and low to moderate in fat (15%) to 30%) tend to be lower in total energy and higher in diet quality when compared to low-carbohydrate diets (<30%) (19). High-carbohydrates diets have been scrutinized based on outcomes and personal testimony of individuals who follow popular low-carbohydrate diets. A randomized controlled trial published in 2003 investigated weight loss outcomes using a low-carbohydrate diet compared to a low-fat, low-energy high carbohydrate (conventional) diet. Although the initial weight loss outcome was significantly greater in the low-carbohydrate group, the difference between the two groups was not statistically significant at 1 year (24). The difference in weight loss between the two groups in the first 6 months was attributed to an overall greater energy deficit in the low-carbohydrate group (24). The low-carbohydrate diet was associated with greater improvement in some risk factors for coronary heart disease. Adherence was poor and attrition was high in both groups (24). Results of this study should be interpreted with caution, given the study's relatively small sample size and the 1-year duration (24). Longer and larger studies are required to determine the longterm safety and efficacy of low-carbohydrate diets that are high in protein and fat (24). The low glycemic index diet is not recommended for weight loss or weight maintenance, since studies have not shown it to be effective in these areas (Grade I) (7).

Calcium: A review of evidence suggest calcium intake lower than the recommended level is associated with increased body weight (Grade II) (6,7). However, the effect of calcium at or above recommended levels on weight management is not clear (Grade II) (6,7). Incorporating 3 to 4 servings of low-fat dairy foods a day as part of the diet component of a comprehensive weight management program is suggested (6).

Physical activity: The US Surgeon General's Report on Physical Activity and Health recommends 30 minutes of moderate to vigorous physical activity per day for children and adults (18). Physical activity contributes to weight loss, may decrease abdominal fat, and may help with maintenance of weight loss (Grade I) (7). Increased physical activity should be a key component of a weight-loss program (4,6,7). A combination of weight resistance or strength training and aerobic exercise is recommended to preserve lean body mass and promote the loss of adipose tissue (4). Federal Physical Activity Guidelines for Americans make recommendations in weekly versus daily doses (6,25). These guidelines suggest that many people may need more than the equivalent of 150 minutes/week of moderate-intensity physical activity to maintain their weight and more than 300 minutes/week (or 42 minutes/day) to meet weight-control goals (25). Long-term goals should be to accumulate at least 30 minutes of moderate intensity physical activity on most, preferably all, days (unless medically contraindicated) (Grade I) (7).

Behavior modification: Behavior modification is an integral component of weight loss and weight management and, in addition to diet and physical activity, leads to additional weight loss (Grade I) (4,6,7). Behavior modification is based on the premise that eating is a conditioned response. A goal of behavior modification is to help the patient realize and eliminate the associations that control eating behavior. Portion control, one method of behavior modification, at meals and snacks results in reduced energy intake and weight loss (Grade II) (6,7). A comprehensive weight management program should make maximum use of multiple strategies for behavior therapy (eg, self-monitoring, portion control, stress management, stimulus control, problem solving, contingency management, cognitive restructuring, and social support) (7). Continued behavior interventions may be necessary to prevent a return to baseline weight (Grade I) (7).

Very-low-calorie diets (VLCD): These specialized feeding regimens provide 800 kcal/day (or 6 to 10 kcal/kg) or less per day, is enriched with high biological value protein and provides at least 100% of the Daily Value of essential vitamins and minerals (6). These diets consist of a premixed liquid or meat, fish, or poultry (6). This type of diet is recommended *only* to patients who are at a very high health risk related to obesity. Criteria for these regimens generally are a BMI of at least 30 kg/m² and previous failures from other treatment approaches (6). Individuals on a VLCD should be supervised by a physician and receive supplemental vitamins and minerals (26). The typical treatment duration is 4 to 6 months. Because patients who consume less than 800 kcal/day are at risk for protein, vitamin, and mineral deficiencies, they should be metabolically monitored. High-quality protein (0.8 to 1.5 g/kg of ideal body weight) and a minimum of 100 g of carbohydrate should be provided each day (23). People with a history of gallbladder disease, cardiac abnormality, cancer, renal or liver disease, type 1 diabetes, or HIV should use these regimens with caution. VLCDs produce weight losses of 15% to 25% in 8 to 16 weeks (27). Adherence to VLCD's results in lower calorie intakes and therefore significant intial weight loss than reduced-calorie diets (Grade 1) (6,7). Despite the short-term success of achieving significant weight losses, there is poor long-term maintenance of the weight loss (Grade 1) (6,7,26). Several

randomized trials found VLCDs to be no more effective than low-energy diets 1 year after treatment, leading the National Institutes of Health Expert Committee to not recommend VLCDs (4).

Meal replacements: Meal replacements are growing in popularity as another category of energy-controlled diets (6). A meal is replaced with a liquid drink that contains approximately 200 kcal per serving and approximately 50% to 60% carbohydrate, 30% protein, and 10% fat, or a pre-measured frozen meal of a set energy value (6). The meal replacement helps the patient control energy intake and reduces sensory stimulation and the need to make decisions about portion size (6). Weight loss outcomes using a meal replacement are dependent on the patient's adherence to the meal plan. Studies demonstrate sustained weight loss of 3.2% to 8.4% over 4 years using meal replacements (28,29). For people who have difficulty with self-selection or portion control, meal replacements (eg, liquid meals, meal bars, energy-controlled packaged meals) may be used as part of the diet component of a comprehensive weight management program. Substituting one or two daily meals or snacks with meal replacements is a successful weight loss and weight maintenance strategy (Grade I) (6.7).

Pharmacotherapy: Pharmacologic agents for obesity intervention contribute to energy deficit through a variety of mechanisms (27). There are currently few pharmacotherapy options available for long-term use (6). Medications approved by the Food and Drug Administration for the treatment of significant clinical obesity include sibutramine (Meridia) and orlistat (Xenical) (6). The greatest benefit of pharmacotherapy may be the facilitation of weight loss maintenance, rather than the induction of weight loss (29,30). Two-year studies of sibutramine and orlistat showed that participants who remained on medication at the end of this time maintained weight losses that were nearly twice as great as those of participants who received placebo (30). It may be recommended that these medications be used long-term in the same manner as agents for hypertension, hyperlipidemia, and diabetes mellitus (31). Criteria for pharmacotherapy intervention include a BMI of at least 30 kg/m² with no comorbid conditions, or a BMI of at least 27 kg/m² with comorbid conditions or a very high health risk (4). Research indicates that pharmacotherapy may enhance weight loss in some overweight and obese adults (Grade I) (6.7).

Sibutramine is a combined centrally acting serotonin-norepinephrine reuptake inhibitor that is associated with increased satiation. Sibutramine seems to reduce body weight by modifying intake through increased satiety, and animal data suggest that it may also increase energy expenditure by stimulating thermogenesis (31). When combined with a low-energy diet, 10 to 15 mg/day of sibutramine produces a clinically significant greater loss of initial weight (7%) when compared to a low-energy diet plus placebo (32). Weight loss of 10% to 15% has been observed in studies that combine sibutramine with intensive lifestyle modification (33,34). Sibutramine is not recommended for patients with uncontrolled hypertension or a history of coronary artery disease, arrhythmias, heart failure, or stroke. In addition, some antidepressants, such as monoamine oxidase inhibitors or selective serotonin reuptake inhibitors, may counteract with the mechanism of sibutramine; therefore, it is not recommended that these medications be taken concurrently (31). Complications of sibutramine include dry mouth, nausea, headache, insomnia, increased blood pressure, and increased heart rate (4,6,29).

Amphetamine-like derivatives, such as phentermine and phentermine resin (Adipex-P, Fastin, and others), mazindol, benzphetamine, and phendimetrazine, which are frequently approved and prescribed for short-term use (\leq 3 months), have a different mechanism of action other pharmological medications. They decrease appetite and food intake by increasing the availability of norepinephrine in the brain. The complications of these drugs are similar to those of sibutramine. Fenfluramine (Pondimin) and dexfenfluramine (Redux) were voluntarily withdrawn from the market because of reports of their association with valvular heart disease. They are serotoninergic agents that act primarily by increasing serotonin levels in the brain, leading to a decrease in appetite (6). Herbal preparations for weight loss do not have standardized amounts of active ingredients and have been reported to have harmful effects (6). Certain overthe-counter preparations containing phenylpropanolamine (eg, Dexatrim) and related compounds have no proven efficacy and have been recalled because of reported incidences of hemorrhagic stroke (6).

Orlistat, a pancreatic lipase inhibitor, is the first obesity medication that does not act systemically. Orlistat inhibits the absorption of up to 30% of dietary fat contained in a meal, leading to a loss of 150 to 180 kcal/day (35). Patients should take this medication with meals, as it takes effect within 2 hours of ingestion. Patients receiving orlistat should follow a moderately low-fat diet (less than 30% of total energy from fat), with fat distributed evenly at each meal. Consumption of more than 20 g of fat per meal or 70 g of fat per day can induce adverse gastrointestinal events that include flatus, oily leakage or oily stools, and diarrhea (6). Supplementation with fat-soluble vitamins may be needed (6). In randomized trials, participants who received placebo plus diet lost 6% of their weight in 1 year, compared with a 10% weight loss for those

treated with orlistat plus diet (36). Because long-term safety has been demonstrated, orlistat has been approved by the Food and Drug Administration for over-the-counter sales at a reduced dosage (6).

Medical nutrition therapy, exercise, and behavior modification should be provided in adjunct to pharmacotherapy (Grade I) (4,6,7,29). With pharmacotherapy, weight loss plateaus by 6 months, and weight regain occurs after medication therapy stops (31). A limited number of studies have evaluated the safety and efficacy of anorexiant medications for more than 2 to 3 years. The physician must continually assess drug therapy for efficacy and safety (4).

Surgery: Weight loss surgery is one option for weight reduction in adult patients with severe obesity, defined as a BMI of at least 40 kg/m² or a BMI of at least 35 kg/m² with comorbid conditions (Grade I) (4,6,37). Roux-en-Y gastric bypass and laparoscopic adjustable gastric band are the most common and widely accepted surgical procedures for weight loss (6). More recent guidelines suggest weight loss sugery may be an option for children and adolescents over the 95th percentile for weight based on age who present with a severe comorbidity (36). Children and adolescents should be managed in specialized centers and currently only the laparoscopic adjustable gastric band (LAGB) and Roux-en-Y gastric bypass (RYGB) should be offered as surgical options in this population group (37). The primary benefit of surgical therapy is durable weight loss and maintenance of weight loss (4,6). More than 90% of patients experience significant (>20% to 25%) weight loss, and between 50% and 80% of patients maintain the weight loss for more than 5 years (38). Most weight loss occurs in the first 6 months and continues for up to 18 to 24 months. The initial 6-month period is marked by the most rapid weight reduction and improvements in comorbid conditions (1). Prospective studies show that the average weight 10 years after surgery is approximately 55% of excess body weight, with a weight regain of 10% to 15% of the initial weight lost (39). A preoperative behavior change program is highly recommended. Complications of weight loss surgery include gallstones, nutritional deficiencies requiring supplementation, dumping syndrome, protein-calorie malnutrition, and the complications that are associated with any surgery. (Refer to "Nutrition Management of Bariatric Surgery" in Section B.)

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

- 1. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among U.S. adults, 1999-2000. JAMA. 2002;288:1723-1727.
- Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among U.S. children and adolescents, 1999-2000. JAMA. 2002;288:1728-1732.
- 3. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med.* 1993;22:167-177.
- 4. National Heart, Lung, and Blood Institute Obesity Education Initiative Expert Panel. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.* National Institutes of Health; 1998. NIH publication No. 98-4083. Available at: http://nhlbi.nih.gov/nhlbi/htm.
- 5. Wolf A, Colditz GA. Current estimates of the economic cost of obesity in the United States. Obesity Res. 1998;6:97-106.
- 6. Position of the American Dietetic Association: weight management. *J Am Diet Assoc*. 2009;109:330-346.
- 7. Adult Weight Management Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2006. Available at: http://www.andevidencelibrary.com. Accessed February 10, 2009.
- 8. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parenteral obesity. N Engl J Med. 1997;337:869-873.
- 9. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee recommendations. Pediatrics. 1998; 102:1-11
- 10. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics*. 1998;101:518-525.
- 11. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, Mei Z, Curtin LR, et al. *CDC Growth Charts: United States. Advance Data From Vital and Health Statistics*. Hyattsville, Md: National Center for Health Statistics; 2000. Publication No. 314.
- 12. Pediatric Weight Management Evidence Analysis Project. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2006. Available at: http://www.andevidencelibrary.com. Accessed August 1, 2006.
- 13. Epstein LH, Valoski A, McCurley J. Effect of weight loss by obese children on long-term growth. *Am J Dis Child*. 1993;147:1076-1080.
- 14. Caprio S, Hyman LD, McCarthy S, Lange R, Bronson M, Tamborlane WV. Fat distribution and cardiovascular risk factors in obese adolescent girls: importance of the intraabdominal fat depot. *Am J Clin Nutr.* 1996;64:12-17.
- 15. Kavey RE, Daniels SR, Lauer RM, Atkins DL, Hayman LL, Taubert K. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. *Circulation*. 2003;107:1562-1566.
- 16. Williams CL, Hayman LL, Daniels SR, Robinson TN, Steinberger J, Paridon S, Bazzarre T. Cardiovascular health in childhood: a statement for health professionals from the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation*. 2002;106:143-160.
- 17. Slavin, JL. Position of the American Dietetic Association: health implications of dietary fiber. J Am Diet Assoc. 2008;108:1716-1731.
- 18. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American
- College of Sports Medicine. *JAMA*. 1995;273:402-407. 19. Wing RR, Hill JO. Successful weight loss maintenance. *Annu Rev Nutr*. 2001;21:323-341.

Obesity and Weight Management

- 20. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.* 2002;346:393-403.
- 21. Kennedy ET, Bowman SA, Spence JT, Freedman M, King J. Popular diets: correlation to health, nutrition, and obesity. J Am Diet Assoc. 2001;101:411-420.
- 22. Wadden T, Van Itallie T, Blackburn G. Responsible and irresponsible use of very-low-calorie diets in the treatment of obesity. *JAMA*. 1990;263:83-85.
- 23. Nonas C. A model for chronic care of obesity through dietary treatment. J Am Diet Assoc. 1998;98(suppl 2):S16-S22.
- 24. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med.* 2003;348:2082-2090.
- 25. Physical activity guidelines advisory committee report to the Secretary of Health and Human Services, 2008. US Department of Health and Human Services Web site. http://www.health.gov/PAGuidelines/committeereport.aspx. Accessed February 10, 2009.
- 26. National Task Force on the Prevention and Treatment of Obesity, National Institutes of Health. Very low-calorie diets. *JAMA*. 1993;270:967-974.
- 27. Wadden TA, Osei S. The treatment of obesity: an overview. In: Wadden TA, Stunkard AJ, eds. *Handbook of Obesity Treatment*. New York: Guilford Press; 2002:229-248.
- 28. Flechtner-Mors M, Ditschuneit HH, Johnson TD, Suchard MA, Adler G. Metabolic and weight loss effects of long-term dietary intervention in obese patients: four-year results. *Obes Res.* 2000;8:399-402.
- 29. National Task Force on the Prevention and Treatment of Obesity. Long-term pharmacotherapy in the management of obesity. *JAMA*. 1996;276:1907-1915.
- 30. Fabricatore AN, Wadden TA. Treatment of obesity: an overview. Clin Diabetes. 2003;21:67-72.
- 31. Fernstrom JD, Atkinson RL. Antiobesity agents: pharmacology and pharmacotherapy. In: *Obesity as a Chronic Disease: New Implications for Management.* Clinical Management Conference Proceedings. Minneapolis, Minn: University of Minnesota Office of Continuing Education; 1998:18-27.
- 32. Lean MEJ. Sibutramine: a review of clinical efficacy. Int J Obes. 1997;21:30S-36S.
- 33. James WP, Astrup A, Finer N, Hilsted J, Kopelman P, Rossner S, Saris WH, Van Gaal LF. Effect of sibutramine on weight maintenance after weight loss: a randomized trial. *Lancet.* 2000;356:2119-2125.
- 34. Wadden TA, Berkowitz RI, Sarwer DB, Prus-Wisniewski R, Steinberg C. Benefits of lifestyle modification in the pharmacologic treatment of obesity: a randomized trial. *Arch Intern Med.* 2001;161:218-227.
- 35. Sjostrom L, Rissanen A, Andersen T, Boldrin M, Golay A, Koppeschaar HP, Krempf M. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. *Lancet.* 1998;352:167-172.
- Davidson MH, Hauptman J, DiGiorlamo M, Foreyt JP, Halstead CH, Heber D, Heimburger DC, Lucas CP, Robbins DC, Chung J, Heymsfeld SB. Weight control and risk factor reduction in obese subjects treated for 2 years with orlistat: a randomized controlled trial. *JAMA*. 1999;281:235-242.
- 37. Mechanick JI, Surgerman HJ, Collazo-Clavell ML, Spitz AF, Livingston EH, Anderson WA. Executive Summary of The Recommendations of The American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery Medical Guidelines For Clinical Practice for The Perioperative, Nutritional, Metabolic, and Nonsurgical Support of The Bariatric Surgery Patient. *Endocrine Practice*. 2008;3:318-336.
- 38. MacLean LD, Rhode BM, Nohr CW. Late outcome of isolated gastric bypass. Ann Surg. 2000;231:524-528.
- 39. Pories WJ, Swanson MS, MacDonald KG, Long SV, Morris PG, Brown BM, Barakat HA, deRamon RA, Israel G, Delezal JM, et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes. *Ann Surg.* 1995;222:339-352.

PANCREATITIS

Discussion

The pancreas is a major organ involved in the digestion and absorption of food. The pancreas is comprised of two major glands each providing a unique function. The endocrine gland known as the Islets of Langerhans is responsible for producing insulin, glucagon, and somatostatin. The exocrine portion of the gland (referred as the ductal system) is responsible for secretion of multiple digestive enzymes including amylase, lipase, carboxypeptidase, phospholipase-alpha, chymotrypsin, aminopeptidase, trypsinogen, and cholesterase (1).

Acute pancreatitis involves a systematic immunoinflammatory response to a localized process of autodigestion of the pancreatic gland and can include the involvement of other tissues and organ systems (1-3). The onset of pain usually occurs 24 to 36 hours after the peak of cytokine production which leads to the inflammatory response (4). Distant organ failure can occur in 1 to 3 days (4).

The etiology of acute pancreatitis in the U.S. involves alcohol abuse in 75% of cases (1,5). Approximately 15% of case are either idiopathic or caused by biliary tract diseases (passage of common bile duct stone), while 10% is accounted for by a variety of disorders such as pancreas divisum, trauma, hypoparathyroidism, hyperclacemia, hyperlipidemia, post-endoscopic retrograde cholangiopancreatography (ERCP), medications, and biliary dyskinesia (1,5). Treatment often depends on severity which is identified by objective scoring systems such as Acute Physiology and Chronic Health Evaluation (APACHE) II score and Ranson Criteria (1,6-9), and the presence of necrosis on CT scan (1,6). In addition, disease severity is determined by the adequacy of fluid resuscitation, the presence and extent of necrosis within the gland, the presence of obesity, infection within the gland, failure of at leaset one organ system, and the route of nutrition support (1). For severe cases the anticipated hospital length of stay is approximately 1 month with a high percentage of patients developing complications such as infection, sepsis, and organ failure (1). For mild to moderate cases, there is a low risk for complications (< 5%), with the chance for 80% to advance to an oral diet successfully within 1 week (1, 6-9).

Nutritional Assessment and Diagnosis

Acute pancreatitis produces a hypermetabolic response that alters carbohydrate protein, fat, and energy metabolism leading to rapid deterioration of nutritional status (1). Reduced oral intake can occur from abdominal pain, food aversion, nausea, vomiting, gastric atony, paralytic ileus, or partial obstruction of the duodenum from enlargement of the pancreatic gland (1). Pancreatitis can also produce a hemodynamic response, which is similar to that of sepsis (1). This response includes increased cardiac output, decreased peripheral resistance, and increased oxygen consumption, currently known as the systemic inflammatory response syndrome (SIRS) (1). Energy expenditure reportedly increases by 139% of that predicted by the Harris-Benedict equation and can further be increased by 15% if the pancreatitis is complicated by sepsis (1,10). Patients with acute pancreatitis are more hypermetabolic when compared to those with chronic pancreatitis (10).

Nutrient losses are often increased because of maldigestion from reduced enzyme output, malabsorption of luminal nutrients, or excessive protein loss caused by diarrhea or pancreatic fistulas (1). Protein catabolic rate and urea production rates are significantly increased. Errors in carbohydrate metabolism leading to hyperglycemia and insulin resistance occur in 40% to 90% of patients (1,11). Errors in fat metabolism with hypertriglyceridemia occur in 12% to 15% of cases (1,12,13). Hypocalcemia has been shown to be prevalent (13) and occurs as a result of reduced calcium levels related to decreased parathyroid hormone release, increased calcitonin, decreased magnesium levels, hypoalbuminemia, and saponification of calcium with unabsorbed fatty acids (1). Since long-term alcohol use is a primary cause of acute pancreatitis, decreases in zinc, magnesium, thiamin, and folate levels are often observed and should be evaluated (1,11-13). Table III-24 provides common assessment parameters that may be altered as a result of pancreatitis and can be used in diagnosing nutrition related problems.

Table III-24: Common Assessment Parameters for Pancreatitis

Data	Reference Range	Discussion
Serum amylase	30 to 95 IU	Elevated with pancreatic dysfunction, due to liberation of
		digestive enzymes from pancreas into neighboring tissues and bloodstream

Pancreatitis

Data	Reference Range	Discussion
Serum bilirubin	0.2 to 0.9 mg/dL	Elevated values may be due to compression of the distal common duct within the pancreas, biliary stones, or inflammation of the liver and bile ducts
Serum lipase	<1.5 IU/mL	Elevated with liberation of lipase from the pancreas into the bloodstream
Blood glucose	70 to 110 mg/dL	Elevated with impaired secretion of insulin in response to glucose load because of inflammatory destruction of Islets of Langerhans Glucagon released from alpha cells may contribute to the elevation of blood glucose
Fecal fat	5 to 6 g/100 g of stool	Elevated with fat malabsorption secondary to impaired digestion of fat due to impaired secretion of pancreatic lipase Value may reach 50 g/24 hours
Serum carotene	90 to 280 μg/dL 100 to 300 IU/dL	Decreased secondary to fat malabsorption associated with steatorrhea This test is rarely done.
Serum Total calcium	9 to 11 mg/dL	Decreased due to saponification of calcium with unabsorbed fatty acids, or as result of increased calcitonin, decreased magnesium levels, hypoalbuminemia, and/or decreased parathyroid hormone release.
Ionized calcium	4.5-5.6 mg/dL	Recommended for critically ill patients or patients with hypoalbuminemia. Ionized calcium levels are unaffected by changes in serum albumin levels providing a more accurate assessment of calcium status in critically ill and patients receiving specialized nutrition support (14).
Hematocrit	42 to 52% (males) 37 to 47% (females)	Elevated due to hemoconcentration when serum exudes into the abdomen Decreased in severe hemorrhagic pancreatitis
CT scan		Identifies level of necrosis of pancreas
Physical examination	Subjective	Hypoactive bowel sounds Abdominal pain, Nausea, vomiting Diarrhea or bulky foul-smelling stools and flatulence indicate fat malabsorption.

Nutrition Intervention in Acute Pancreatitis

Treatment of acute pancreatitis has drastically changed over the past decade (1). Parenteral nutrition (PN) and bowel rest has traditionally been the primary management approach based on the hypothetical reasons that enteral feeding may stimulate the synthesis of pancreatic enzymes and worsen the severity of disease. However, current evidence suggests that the provision of enteral nutrition (EN) has a dramatic impact on patient outcome compared to the provision of parenteral nutrition (PN) (15-16). Two landmark meta-analyses demonstrated that use of EN reduces infection by as much as 52%, hospital length of stay by as much as 4 days, need for surgical intervention by as much as 52%, and trend toward reduced organ failure by as much as 41% when compared to use of PN (15-16). Experience from the literature suggests that efforts to promote pancreatic rest as the sole management strategy to treat pancreatitis is ineffective and does not have impact on patient outcome (1,17).

The choice of nutrition therapy should be determined by disease severity, anticipated length of intervention, and tolerance to the intervention (1,17). Current standards of care indicate that patients with mild to moderate pancreatitis should initially be prescribed NPO with intravenous hydration support and

then, as symptoms subside, progress to an oral diet (17,18,19). A recent prospective, randomized controlled double-blind clinical trial demonstrated no difference between symptom relapse in patients with mild pancreatitis who progressed to a solid food diet as opposed to clear liquids or a reduced-energy solid food diet (20). The need for gradual diet advancement or reducing fat intake (eg, < 50 g/day) to reduce pancreatic stimulation appears not to be supported by the evidence (1,17). For patients with severe pancreatitis current guidelines suggest improved patients outcomes when early enteral nutrition (EN) is initiated over both continued NPO and parenteral nutrition (17-19). The timing of enteral nutrition support is critical for improving patient outcomes in severe pancreatitis. EN should be initiated within 48 to 72 hours of admission after fluid resuscitation and when patients are hemodynamically stable. Enteral nutrition should also be considered for patients who are malnourished, or have not been able to tolerate oral feedings within 5 to 7 days (17,18). Placement on PN should be reserved for only those patients with severe acute pancreatitis in whom poor tolerance has been documented with EN intervention or in cases where EN is not feasible due to access or other medical issues (1,17).

Several factors have been identified that influence tolerance to enteral feedings in patients with acute pancreatitis (1,17,18). Complications of pancreatitis such as presence of pseudocyst, abscess, or ascites are not a contraindication to EN (1). EN may be provided as long as tolerance is demonstrated. Evidence of intolerance to EN (eg, increase in abdominal pain, fever, or WBC count in association with increases in amylase, lipase) should be routinely evaluated. If intolerance is documented, a change in enteral formula may be indicated (1). The following Table III-25 provides guidelines for initiating EN in severe acute pancreatitis.

Table III-25: Guidennes	for Using Enteral Nutrition in Severe Acute Pancreatitis (17,18)
Tube placement:	Nasogastric, nasoduodenal or nasojejunal route are currently recommended
	options (17,18). Positioning feeding tube tip just below the Ligament of Treitz
	may further improve tolerance (1,16).
Feeding rate:	Use continuous infusion over 24 hours per day (17,18)
Formula selection:	Use small peptide based medium chain triglyceride formula such as
	Peptamen [™] or Subdue [™] (18,21). Small peptide best tolerated in those with
	diarrhea or steatorrhea.

 Table III-25:
 Guidelines for Using Enteral Nutrition in Severe Acute Pancreatitis (17,18)

If intolerance to semi-elemental formula documented consider (1):

 Use elemental formula. Try an elemental formula that provides < 2 to 3% of total calories from fat such as Vivonex[™], Criticare[™], Vital HN[™]; or switch to semi-elemental formula with small peptides and mediumchain triglycerides such as Peptamen[™] or Subdue[™].

Implications of Parenteral Nutrition (PN)

Patients who are placed on PN should be managed using hospital PN protocols and/or guidelines. If pancreatitis is caused by hypertriglyceridemia (> 1,000 mg/dL) or the patient has a history of hyperlipidemia, infusion of IV fat emulsion (IVFE) should be used with caution (1). Pancreatitis due to IVFE-induced hyperlipidemia is rare unless serum triglycerides exceed 1000 mg/dL (1). IVFE is considered safe for use in patients with pancreatitis without hypertriglyceridemia (17,18). However, IVFE should be withheld from the PN regimen if serum triglyceride concentrations exceed 400 mg/dL (1). Consider using glutamine at 0.30 g/kg Ala-Gln dipeptides (17, 18).

Nutrition Approaches and Intervention in Chronic Pancreatitis

Chronic pancreatitis is a chronic, persistent inflammatory state resulting in progressive, irreversible fibrosis and destruction of the endocrine and exocrine tissue. What differentiates chronic pancreatitis from acute is evidence of permanent damage to the anatomy or function of the gland (1). The etiologies that can lead to chronic pancreatitis are nearly identical to those for acute pancreatitis (1). A flare up of chronic pancreatitis is identical to acute pancreatitis (1), however, after the acute episode patients may go on to have recurrent abdominal pain complicated by diarrhea, steatorrhea, and weight loss. Chronic pancreatitis causes many digestive and metabolic disturbances and can compromise the patient's nutritional status over time. Malnutrition occurs late in the disesase course and is a result of a reduction in nutrient absorption, and an increase in metabolic activity (1). Nutrition intervention and management should focus on maintaining the patient's weight, nutritional status and controlling abdominal pain through symptom management (1).

Most chronic pancreatitis patients can be managed with dietary recommendations and pancreatic enzyme supplementation (1,17). Enzyme replacement therapy is used to control malabsorption and to relieve pain, and should be given with meals and snacks (1,17). There is no evidence to support the contention that a fat-Manual of Clinical Nutrition Management III-91 Copyright © 2013 Compass Group, Inc. All rights reserved.

Pancreatitis

restricted diet will influence the recurrence rate of pancreatitis, except in those patients with severe hypertriglyceridemia. Pancreatic lipase reserves are large; as much as 80% of pancreatic lipase secretion can be lost without interfering with fat digestion. A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1). Vegetable fats have been shown to be better tolerated than animal fats (1). Substituting medium-chain triglyceride oil for long-chain fat has been shown to decrease cholecystokinin (CCK) levels and pancreatic stimulation and improve persistent pain in patients with chronic pancreatitis (21). One study found that approximately 5% of those with chronic pancreatitis have severe and ongoing maldigestion and may enteral feeding support. Providing enteral nutrition using a route that provides feedings beyond the Ligament of Treiz is recommended over parenteral nutrition (22).

The following table provides a summary of nutrition intervention strategies for patients with acute and chronic pancreatitis.

First oral feedingNPO with intravenous hydration to support and correct fluid, electrolyte and acide-base disturbances (17).Advance to liquids or solids as symptoms subside and laboratory values normalize (18). Initiating the first oral feeding as clear liquids does not improve tolerance when compared to advancing to solid feedings. Current evidence suggests a liberalized approach with diet progression should be considered based on patient tolerance and response to therapy (17.18.20).Total energyRefer to Section II: "Estimation of Energy Expenditures" using predictive equations relevant to patient medical condition 25 kcal/kg/day for chronic pancreatitis (17) 35 kcal/kg/day for chronic pancreatitis (11) 1.0 to 1.5 g/kg/day in chronic phase (1)Protein1.2 to 1.5 g/kg/day with acute pancreatitis (11) 1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1.17). A low fat diet is not routinely required, unless symptoms supplementation and narcotic analgesia (1.17).Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplements may need to be increased to 3 to 4 tablets	Approach	Rationale
Advance to liquids or solids as symptoms subside and laboratory values normalize (18). Initiating the first oral feeding as clear liquids does not improve tolerance when compared to advancing to solid feedings. Current evidence suggests a liberalized approach with diet progression should be considered based on patient tolerance and response to therapy (17.18.20).Total energyRefer to Section II: "Estimation of Energy Expenditures" using predictive equations relevant to patient medical condition 25 kcal/kg day for chronic pancreatitis (17) 35 kcal/kg/day for chronic pancreatitis (11) 1.0 to 1.5 g/kg/day with acute pancreatitis (11) 1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1.17). A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1.17).GarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	First oral feeding	NPO with intravenous hydration to support and correct fluid, electrolyte and acide-base disturbances (17).
Total energyRefer to Section II: "Estimation of Energy Expenditures" using predictive equations relevant to patient medical condition 25 kcal/kg to 35 kcal/kg/day for acute pancreatitis (17) 35 kcal/kg/day for chronic pancreatitis (1)Protein1.2 to 1.5 g/kg/day with acute pancreatitis (1) 1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1,17). A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17). Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		Advance to liquids or solids as symptoms subside and laboratory values normalize (18). Initiating the first oral feeding as clear liquids does not improve tolerance when compared to advancing to solid feedings. Current evidence suggests a liberalized approach with diet progression should be considered based on patient tolerance and response to therapy (17,18,20).
25 kcal/kg to 35 kcal/kg/day for acute pancreatitis (17) 35 kcal/kg/day for chronic pancreatitis (1)Protein1.2 to 1.5 g/kg/day with acute pancreatitis (1) 1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1,17). A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17). Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Total energy	Refer to Section II: "Estimation of Energy Expenditures" using predictive equations relevant to patient medical condition
35 kcal/kg/day for chronic pancreatitis (1)Protein1.2 to 1.5 g/kg/day with acute pancreatitis (1)1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1,17).A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17).Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		25 kcal/kg to 35 kcal/kg/day for acute pancreatitis (17)
Protein1.2 to 1.5 g/kg/day with acute pancreatitis (1) 1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1,17). A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17). Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		35 kcal/kg/day for chronic pancreatitis (1)
1.0 to 1.5 g/kg/day in chronic phase (1)FatPatients with exocrine insufficiency should receive pancreatic enzymes (1,17). A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17). Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Protein	1.2 to 1.5 g/kg/day with acute pancreatitis (1)
FatPatients with exocrine insufficiency should receive pancreatic enzymes (1,17).A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17).Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		1.0 to 1.5 g/kg/day in chronic phase (1)
A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17).Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Fat	Patients with exocrine insufficiency should receive pancreatic enzymes (1,17).
Modifications in fat may be indicated with diabetes, obesity Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1,17).
Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		Modifications in fat may be indicated with diabetes, obesity
CarbohydratesNormal percentage of carbohydrates.FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).		Note: During measurement of 72-hour (quantitative) fecal fat, patients need to be on a 100-g fat diet and have actual fat intake calculated.
FluidsPatients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Carbohydrates	Normal percentage of carbohydrates.
FiberAvoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17). Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Fluids	Patients may have increased fluid, chloride, sodium, potassium, and calcium needs secondary to nasogastric suction, diarrhea, or emesis.
VitaminsFor patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17).Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Fiber	Avoid high-fiber diets in patients with exocrine insufficiency; large amounts of fiber can increase steatorrhea.
Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below. If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Vitamins	For patients with alcoholism or history of alcoholism supplement: Thiamin 100 mg, folate 1 mg and a general multivitamin by mouth once per day (17).
MonitoringIf abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below.If delayed gastric emptying, consider prokinetic medication if appropriate (1).		Patients with fat malabsorption may need supplementation of fat-soluble vitamins (17).
If delayed gastric emptying, consider prokinetic medication if appropriate (1).	Monitoring	If abdominal pain persists pancreatic enzyme supplements may need to be increased to 3 to 4 tablets with meals and at bedtime (1). See typical dose below.
		If delayed gastric emptying, consider prokinetic medication if appropriate (1).

Table III-26: Nutrition Intervention Strategies for Pancreatitis

Approach	Rationale
Self-management	Avoid alcohol.
training	Small feedings.
	Pancreatic enzymes must be taken at the same time as food, at all meals and snacks (no more than 30 minutes prior to eating to maximize effectivenesss) (1).
	A low fat diet is not routinely required, unless symptoms are poorly controlled on enzyme therapy or if pain persists on both enzyme supplementation and narcotic analgesia (1).
Pancreatic enzymes	Typical dose is 2 tablets taken orally (30,000 IU of lipase) per meal and/or snacks (1,21,22) ^a .
	Must be taken while eating (no more than 30 minutes prior to eating to maximize effectivenesss) ₍₁₎
	Do not crush enteric coated enzyme capsules. The use of H2 receptor antagonists (eg, cimetidine, ranitidine, famotidine) or proton-pump inhibitors (omeprazole) increases the efficiency of non-enteric coated pancreatic enzyme supplements by decreasing gastric acid production (1).
oml 1 C	

^aThe dose of enzymes usually required to treat steatorrhea should contain the concentration of enzymes that approximates 10% of what the pancreas normally produce (1)

References

- 1. Parrish CR, Krenitsky J, McClave SA. Pancreatitis. In: Mueller CM ed. *The A. S. P. E. N. Adult Nutrition Support Core Curriculum*. Silver Spring, MD: American Society of Enteral and Parenteral Nutrition; 2012:472-490.
- 2. Bradley EL 3rd. A clinically based classification system for acute pancreatitis. *Arch Surg.* 1993;128:586-590.
- 3. Saluja AK, Steer MLP. Pathophysiology of pancreatitis. Role of cytokines and other mediators of inflammation. *Digestion*. 1999;60(suppl 1):27-33.
- 4. Norman JG. New approaches to acute pancreatitis: role of inflammatory mediators. Digestion. 1999;60(suppl 1):57-60.
- 5. Holt S. Chronic pancreatitis. South Med J. 1993;86:201-207.
- 6. Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol.* 2006; 101:2379-2400.
- 7. Larvin M, McMahon MJ. APACHE-II score for assessment and monitoring of acute pancreatitis. Lancet. 1989;2:201-205.
- 8. Sax H, Warner B, Talawini M. Early total parenteral nutrition in acute pancreatitis: lack of beneficial effects. *Am J. Surg.* 1987;153:117-124.
- 9. Wilson C, Heath DI, Imrie CW. Prediction of outcome in acute pancreatitis: a comparative study of APACHE II, clinical assessment and multiple factor scoring systems. *Br J Surg.* 1990;77:1260-1264.
- 10. Dickerson RN, Vehe KL, Mullen JL, Feurer ID. Resting energy expenditure in patients with pancreatitis. *Crit Care Med.* 1991;19:484-490.
- 11. Marulendra S, Kirby D. Nutrition support in pancreatitis. Nutr Clin Pract. 1995;10:45-53.
- 12. Havala T, Shronts E, Cerra F. Nutritional support in acute pancreatitis. Gastroenterol Clin North Am. 1989;18:525-542.
- 13. Kohn CL, Brozenec S, Foster PF. Nutritional support for the patient with pancreatobiliary disease. *Crit Care Nurs Clin North Am.* 1993;5:37-45.
- 14. Dickerson RN, Alexander KH, Minard G, Croce MA, Brown RO. Accuracy of methods to estimate ionized and corrected serum calcium concentrations in critically ill multiple trauma patients receiving specialized nutrition support. *J Parenter Enteral Nutr.* 2004;28:133-141.
- 15. McClave SA, Chang WK, Dhaliwal R, Heyland DK. Nutrition support in acute pancreatitis: a systematic review of the literature. *J Parenter Enteral Nutr.* 2006;30:143-156.
- 16. Marik PE, Zaloga GPL. Meta-analysis of parenteral nutrition versus enteral nutrition in patients with acute pancreatitis. *BMJ*. 2004;328:1407-1412.
- 17. Pancreatitis. In: *Nutrition Care Manual*. Chicago, II: The Academy of Nutrition and Dietetics; Updated annually. Available at <u>www.nutritioncaremanual</u>. org. Accessed January 16, 2013.
- 18. Mirtallo JM, Forbes A, McClave SA, Jensen GL, Waitzberg DL, Davies AR; International Consensus Guideline Committee Pancreatitis Task Force. International consensus guidelines for nutrition therapy in pancreatitis. *J Parenter Enteral Nutr.* 2012; 36:284-291.
- 19. Anand N, Park JH, Wu Bu. Modern management of acute pancreatitis. Gastroenterol Clin North Am. 2012; 41:1-8.
- Moraes, JM, Felga, GE, Chebli LA, Franco MB, Gomes CA, Gaburri PD, Zanin A, Chebli JM. A full solid diet as the initial meal in mild acute pancreatitis is safe and results in shorter length of hospitalization: results from a prospective, randomized, controlled, double-blind clinical trial. *J Clin Gastroenterol.* 2010;44:517-522.
- 21. Shea JC, Bishop MD, Parker EM, Gelrud A, Freedman SD. An enteral therapy containing medium-chain triglycerides and hydrolyzed peptides reduces postprandial pain associated with chronic pancreatitis. *Pancreatology*. 2003;3:36-40.
- 22. Stanga Z, Giger U, Marx A, DeLegge MH. Effect of jejunal long-term feeding in chronic pancreatitis. *J Parenter Enteral Nutr.* 2005;29:12-20.

Complication	Causes	Symptoms	Treatments
Hyperglycemia (1-4)	Trauma Infection Diabetes mellitus Excessive dextrose administration Corticosteroids or immunosuppressive therapy	Elevated blood glucose level	 With persistent hyperglycemia, provide insulin when necessary to maintain blood glucose levels of 100 to 150 mg/dL in critically ill patients (4,5). In diabetic patients, the plasma goal is 140 to 180 mg/dL for the majority of critically ill patients (2,5) and <140 mg/dL for non-critically ill patients with random blood glucose targeting levels <180 mg/dL (2,5). More stringent targets may be appropriate in stable patients with previously tight glycemic control. Less stringent targets may be appropriate in patients with severe comorbidities (5). The rate of dextrose infusion in parenteral nutrition (PN) should not exceed 4 to 5 mg/kg per min (1,4).
Hypoglycemia	Sudden cessation of PN Excessive insulin administration	Low blood glucose level (≤70 mg/dL) (5,6) Headache Sweating Thirst Disorientation Convulsions Coma	If managed on insulin, decrease insulin administration. Give intravenous dextrose. Avoid the abrupt cessation of PN. Studies show that hypoglycemia is equally prevalent in nondiabetic patients due to stress. Taper PN solution for 1 to 2 hours. If PN must be discontinued quickly, 10% dextrose should be infused for 1 or 2 hours following PN discontinuation (1). Check the capillary blood glucose concentration 30 min to 1 hour after the discontinuation of PN to identify rebound hypoglycemia (1).
Hyperglycemic hyperosmotic syndrome	Dehydration from osmotic diuresis (type 1 diabetes mellitus) Poor intake of water (occurs in elderly patients with type 2 diabetes mellitus)	Lethargy Stupor Convulsions Blood glucose level >600 mg/dL (6-7) Serum osmolality >350 mOsm/L	Discontinue PN. Initiate rate of half of the estimated needs or approximately 150 to 200 g (or 100 g/day if severe hyperglycemia) for the first 24 hours until tolerance is documented (1). Provide insulin to correct the blood glucose level. Carbohydrate administration should not exceed a rate of 4 to 5 mg/kg per min (1).

PARENTERAL NUTRITION (PN): METABOLIC COMPLICATIONS (1-4)

Complication	Causes	Symptoms	Treatments
Azotemia	Dehydration	High blood urea nitrogen	Increase administration of free fluid.
	Renal insufficiency	level	Reduce amino acid dose.
	Excessive protein		Patients with impaired renal or
	administration or		hepatic disease may require dialysis.
	inadequate		
Urmonhoanhotomia	Alashalism	Anonovia	Increase phosphomys in DN solution
Hypophosphatemia	AICONONSIN Intro stable requiting	Anorexia	hased on individual medical needs
	Intractable vomiting	Muscle weakness	
	Refeeding syndrome	Long hone pain	If refeeding syndrome occurs, modify
	Vitamin D deficiency		the amount of energy provided by
	Huporparathyroidism	Pospiratory distross	carbohydrates (15 to 20 kcal/kg per
	Tryperparatityroluisin	Respiratory distress	day) until electrolytes are stable (1).
Cholestasis	Disuse of	Elevated total bilirubin	Reduce total energy provided.
	gastrointestinal	level >2 mg/dL (1)	Decrease dextrose to <5 mg/kg per
	tract	Elevated alkaline	min.
	Overfeeding	phosphatase level and	Decrease IVFE to ≤ 1 g/kg per day (1).
	Long-term use of PN	transpentidase level	Consider cyclic PN infusion.
	Excessive use of	er unspeptituse rever	
	emulsions (IVFE)		
Steatosis	Overfeeding	Elevated	Consider cyclic PN infusion.
(hepatic	Essential fatty acid	aminotransferase level	Decrease total energy provided.
accumulation)	deficiency		Balance dextrose energy with energy from fat.
			Decrease dextrose to <5 mg/kg per min.
			If essential fatty acid deficiency occurs, treat with lipid infusions.
Hypomagnesemia	Malabsorption	Muscle weakness	Increase Mg in PN.
	Massive small bowel	Depression	Provide additional intravenous
	resection	Apathy	supplementation (4,7).
	Acute pancreatitis	Nausea	If refeeding syndrome occurs, modify
	Prolonged nasogastric	Vomiting	the delivery of energy from
	suction	Irritability	day) until electrolytes are stable (1)
	Intestinal fistula	Vertigo	
	Vomiting Defeeding symdrome	Ataxia	
	Refeeding synulonie	Muscle tremor	
		Hypocalcemia	
		Hypoparathyroidism	
Hypermagnesemia	Excessive Mg	Drowsiness	Decrease Mg in PN.
	auministration	Weakness	
	Renai insumciency	Nausea	
		Vomiting	
		Cardiac arrhythmia	
		Hypotension	

Complication	Causes	Symptoms	Treatments
Hyponatremia	Fluid overload Excessive gastrointestinal loss Excessive urinary loss Adrenal insufficiency Congestive heart failure Syndrome of inappropriate antidiuretic hormone secretion	Decreased serum Na levels and osmolality Irritability Confusion Seizures	Evaluate free-water intake and total volume status considering disease state and underlying causes. If the volume is excessive (eg, free water, intravenous fluids), decrease fluid intake and/or adjust free-water volume. If the volume is deficient, increase water and Na. Adjust Na intake as condition dictates.
Hypernatremia	Dehydration Excessive Na intake Osmotic diuresis Hypoglycemia Hypocalcemia Head trauma Antidiuretic hormone deficiency	Increased serum Na level Convulsions Irritability Restlessness Coma	Evaluate for dehydration or deficit of water or total volume. Increase free- water or fluid intake as appropriate. Evaluate intravenous sources and consider decreasing Na if excessive.
Hyperphosphatemia	Renal insufficiency Excessive phosphorous administration	Elevated serum phosphorous level	Decrease phosphorous in PN.
Hypokalemia	Inadequate K ⁺ intake Diarrhea Intestinal fistula Anabolism Metabolic alkalosis K ⁺ -wasting medications Vomiting Refeeding syndrome	lleus Cardiac arrhythmia	Correct K ⁺ prior to starting PN or adjust PN formula. Consider additional intravenous supplementation (8). If refeeding syndrome occurs, modify the delivery of energy from carbohydrates (15 to 20 kcal/kg per day) until electrolytes are stable (1).
Hyperkalemia	Renal insufficiency Excessive K ⁺ administration Medication (spironolactone)	Cardiac arrhythmia Paresthesias	Decrease K ⁺ in PN (also consider decreasing K ⁺ from other intravenous sources). Provide K ⁺ binders and antagonists. K ⁺ ≥5.5 mEq may warrant an electrocardiogram (8).

Parenteral Nutrition

Complication	Causes	Symptoms	Treatments
Hypocalcemia	Hypoalbuminemia Inadequate vitamin D intake Hypoparathyroidism Inadequate Ca intake Increased gastrointestinal losses Inadequate phosphorus intake High protein dose Metabolic acidosis	Paresthesias Tetany Muscular cramping/ spasms	 Increase Ca²⁺ in PN. (Use caution and follow protocols to avoid Ca²⁺– phosphorus precipitation. Evaluate ionized calcium level or adjusted total serum Ca²⁺ if hypoalbuminemic prior to increasing Ca²⁺ in PN.) Ensure adequate phosphorus (20 to 40 mmol) in PN (1,3). Evaluate protein (>2 g/kg per day associated with increased urinary excretion of calcium) (1). Ensure adequate acetate and Mg in PN(1). For critically ill patients or patients with hypoalbuminemia it is preferable to evaluate ionized calcium levels. Ionized calcium is unaffected by changes in serum albumin levels and provides a more accurate assessment of calcium status in critically ill and patients receiving specialized nutrition support (9).
Hypercalcemia	Excessive vitamin D administration Prolonged immobilization Stress Hyperparathyroidism Malignancy	Thirst Polyuria Decreased appetite Nausea Vomiting Itching Muscle weakness	Evaluate Ca ²⁺ in PN. Ensure adequate hydration. Provide intravenous hydration using 0.9% sodium chloride at 200 to 300 mL/hour when calcium >13 mg/dL (8). After adequate hydration, furosemide can be used to increase renal calcium excretion (8).
Hypertriglyceridemia	Overfeeding with dextrose Rapid administration of IVFE >110 mg/kg per hour (1) Intravenous infusion of propofol (Diprivan), which has the same lipid content as IVFE providing 1.1 kcal/mL (11)	Triglyceride level >400 mg/dL (1,3,10) Impaired immune response Pancreatitis (risk occurs when the triglyceride level exceeds 1000 mg/dL) (1)	Reduce dose or lengthen the IVFE infusion time (1). Provide <30% of energy from IVFE, or provide 1 g/kg per day and infuse slowly (no less than 8 to 10 hours) (1,4). Avoid excessive dextrose administration.

References

- 1. Kumpf VJ, Gervasio J. Complications of parenteral nutrition. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 284-297.
- Critical Illness Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: www.andevidencelibrary.com. Accessed January 16, 2013.
- 3. Task Force for the Revision of Safe Practices for Parenteral Nutrition. Safe practices for parenteral nutrition. *J Parenter Enteral Nutr.* 2004;28(6 suppl):S39-S70.
- 4. McClave SA, Martindale RG, Vanek VW, McCarthy M, Roberts P, Taylor B, Ochoa JB, Napolitano L, Cresci G; A.S.P.E.N. Board of Directors; American College of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A. S. P. E. N.). J Parenter Enteral Nutr. 2009;33:277-316.

5. Glycemic targets in hospitalized patients. Standards of medical care in diabetes—2013. Diabetes Care. 2013;36 (suppl 1): S46-S48.

- Newton L, Garvey T. Nutritional and Medical Management of Diabetes Mellitus in Hospitalized Patients. In: Mueller CM, ed. *The A.S.P E.N. Adult Nutrition Support Core Curriculum. 2nd ed.* Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012: 580-602.
- 7. Garber AJ, Moghissi ES, Bransome ED Jr, Clark NG, Clement S, Cobin RH, Furnary AP, Hirsch IB, Levy P, Roberts R, Van den Berghe G,

Parenteral Nutrition

Zamudio V. American College of Endocrinology Task Force on Inpatient Diabetes Metabolic Control. American College of Endocrinology position statement on inpatient diabetes and metabolic control. *Endocr Pract.* 2004;10(suppl 2):4-9.

- 8. Langley G, Tajchman S. Fluid, electrolytes, and acid-base disorders. In: Mueller CM, ed. *The A.S.P.E.N. Adult Nutrition Support Core Curriculum.* 2nd ed. Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:104-108,112.
- 9. Dickerson RN, Alexander KH, Minard G, Croce MA, Brown RO. Accuracy of methods to estimate ionized and corrected serum calcium concentrations in critically ill multiple trauma patients receiving specialized nutrition support. *J Parenter Enteral Nutr.* 2004;28:133-141.
- 10. ASPEN Board of Directors and the Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *J Parenter Enteral Nutr.* 2002;26(1 suppl):1SA-138SA.
- 11. Hise ME, Brown JC. Lipids. In: Mueller CM, ed. *The A. S. P. E. N. Adult Nutrition Support Core Curriculum 2nd ed.* Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2012:71-72.

CALCULATING TOTAL PARENTERAL NUTRITION

I. Steps for Calculating TPN.

Assess calorie, protein, and fluid requirements.

- 1. Determine calories to be provided as fat.
- 2. Convert fat calories to volume of lipid emulsion or lipid concentration in 3-in-1 solution (or total nutrient admixture TNA)
- 3. Determine calories provided from protein
- 4. Determine amino acid concentration.
- 5. Determine calories provided from carbohydrate.
- 6. Determine carbohydrate (dextrose) concentration.
- 7. Determine if final solution can be compounded.
- These steps will be outlined in detail below.
- **II. Determine amount of each substrate.** Guidelines for usual and maximum substrate amounts are provided in the Table. After these determinations have been made substrate concentrations are easily calculated based on TPN volume. End concentrations reflect grams of substrate in the final volume of TPN solution (see Table).

Substrates in TPN

SUBSTRATE	USUSAL AMOUNT	MAXIMUM UNITS OF SUBSTRATE
Carbohydrate (cho)	40-60% of total Kcal	<5 mg/kg/minute
Protein (pro)	1.0-2.0 gm/kg/day	2.0-2.5 gm/kg/day
Fat	20-40% daily total Kcal	2 gm/kg/day
		<1 gm/kg/day in high stress

Note: Sample calculations are based on total calorie needs rather than nonprotein calorie needs. Either method may be used; however, the former yields approximately 250 to 350 calories more.

Estimated Needs: 1,700 calories (kcal), 70 gm protein.

III. Calculating macronutrient concentrations.

A. 2-in1 (traditional) solution calculations:

Calculate 2-in-1 solution using 2,000 mL fluid (this volume will include lipid emulsion)

- 1. Determine kcal to be provided as fat. Estimated kcal x desired percent of fat = kcal as fat 1,700 kcal x 30% = <u>510 kcal from fat</u>
- **2.** Convert fat kcal to volume of lipid emulsion.
 Fat kcal ÷ kcal/mL lipid emulsion = volume of lipid emulsion

510 kcal \div 1.1 (10% emulsion) kcal/mL = <u>463 mL</u> of 10% lipid

or

510 kcal ÷ 2.0 (20% emulsion) kcal/mL = <u>255 mL</u> of 20% lipid

Some institutions prefer lipid to be ordered in volumes as packaged by the supplier (250 mL or 500 mL).

For this example, <u>500 mL of 10% lipid</u> will be used leaving 1,500 ML for the remaining TPN solution.

3. Determine kcal to be provided from protein.

Estimated protein needs x kcal/gm protein = 70 gm protein x 4 kcal/gm = <u>280 kcal</u> protein

4. Determine amino acid concentration (AA).

 $\begin{array}{ll} \underline{gm \ protein} & x \ 100 = \% \ amino \ acids \\ \hline TPN \ volume & \\ \hline \underline{70 \ gm} & x \ 100 = \underline{4.7\% \ amino \ acids} \\ \hline 1,500 \ mL & \end{array}$

5. Determine kcal to be provided from carbohydrate.

Estimated kcal needs – (kcal as fat + kcal as protein) = carbohydrate kcal 1,700 kcal – (510 kcal + 280 kcal) = <u>910 kcal carbohydrate</u>

6. Determine carbohydrate concentration.

- a. Carbohydrate kcal ÷ kcal/gm dextrose* = gm dextrose
 910 kcal ÷ 3.4 kcal/gm = 268 gm dextrose
 *Dextrose solutions are 3.4 kcal/gm rather than 4 kcal/gm
- b. <u>gm dextrose</u> x 100 = % dextrose volume TPN <u>268 gm</u> x 100 = <u>17.9% dextrose</u>
 - 1,500 mL

B.

C.

	Calculate a 2-in-1 solution with a fluid restriction of 1,250 mL.
1.	Determine kcal to be provided as fat.
	Estimated kcal x desired percent = kcal as fat
	1,700 kcal x 30% = 510 kcal fat
2.	Convert fat kcal to volume of lipid emulsion.
	A 20% lipid solution is preferred for fluid-restricted patients.
	k_{cal} as fat $\pm k_{cal}/mL$ linid emulsion = volume linid
	$510 \text{ kcal} \div 2 \text{ kcal/mL} (2006 \text{ amulsion}) = 255 \text{ mL}$
	510 Kcal + 2 Kcal/IIIL (2070 elliuisioII) - 253 IIIL This can be reunded to 250 mL leaving 1 000 mL for the remaining TDN solution
2	This can be founded to <u>250 mL</u> , leaving 1,000 mL for the remaining TFN solution.
3.	Determine kcal to be provided from protein.
	Estimated protein needs x kcal/gm protein = kcal protein
	$70 \text{ gm x } 4 \text{ kcal/gm} = \frac{280 \text{ kcal protein}}{280 \text{ kcal protein}}$
4.	Determine amino acid concentration (AA).
	$\underline{gm \ protein}$ x 100 = % AA
	TPN volume
	<u>70 gm</u> x $100 = 7.0\%$ AA
	1,000 mL
5.	Determine kcal to be provided from carbohydrate.
	Estimated kcal needs – (kcal as fat + kcal as protein) = carbohydrate kcal
	1,700 kcal – (510 kcal + 280 kcal) = 910 kcal carbohydrate
6.	Determine carbohydrate concentration.
-	a. Carbohydrate kcal \div kcal/gm dextrose = gm dextrose
	910 kcal \div 3.4 kcal/gm = 268 gm dextrose
	h gm devtrose x $100 = \%$ devtrose
	volume TPN
	268 gm v 100 - 26.8% deverse
	$\frac{200 \text{ gm}}{100 \text{ m}}$ x 100 - $\frac{20.070 \text{ dext10se}}{100 \text{ m}}$
7	I,000 IIIL Final order:
7.	Final order:
	1 liter 7% AA, 26.8% dextrose, and 250 lite 20% lipid. This solution will not
	compound, so the dextrose will have to be reduced to 17.5% until fluid restrictions
	are lifted.*
	(See compounding guidelines for specific calculations Section IV.)
	*If your pharmacy uses a 15% amino acid stock solution, this will compound.
4	Calculate a 3-In-1 Solution with 2,000 mL liulu.
1.	Determine kcal to be provided as fat.
	Estimated kcal x desired percent = kcal as fat
	1,700 kcal x 30% = 510 kcal fat
2.	Convert fat kcal to volume of lipid emulsion.
	a. kcal fat ÷ kcal/gm = gm fat
	$510 \text{ kcal} \div 9 \text{ kcal/gm}^* = 56.7 \text{ gm fat}$
	b. <u>gm dextrose</u> x 100 = % dextrose
	volume TPN
	<u>268 gm</u> x 100 = <u>26.8% dextrose</u>
	1,000 mL
	*9 kcal/gm is used as an estimate; however, lipid emulsions are actually 10 kcal/gm because of
	components other than fat within the emulsion.
3.	Determine kcal to be provided from protein.
	gm protein x kcal/gm = kcal as protein
	70 gm x 4 kcal/gm = <u>280 kcal protein</u>
4.	Determine amino acid concentration (AA).
	gm protein x 100 = %AA
	TPN volume
	70 gm x $100 = 3.5% AA$
	2,000 mL
5.	Determine kcal to be provided from carbohydrate.
	Estimated kcal needs – (kcal as fat + kcal as protein) = carbohydrate kcal
	1.700 kcal - (510 kcal + 280 kcal) = 910 kcal carbohydrate
6	Determine carbohydrate concentration
0.	a kcal deverses \pm kcal/gm deverses $-$ gm deverses
	a. Real uctions $-$ real/gill uctions $-$ gill uctions
	710 Ktal + 3.4 Ktal/gill – 200 gill uexu 05e

Manual of Clinical Nutrition Management

b. $\underline{\text{gm dextrose}}_{\text{TPN volume}}$ x 100 = %dextrose $\underline{268 \text{ gm}}_{2,000 \text{ mL}}$ x 100 = $\underline{13.4\% \text{ dextrose}}_{2,000 \text{ mL}}$

- 7. Final order:
 - 2 liters 3.5% AA, 13.4% dextrose, and 2.8% lipid

D. Calculate a 3-in-1 solution with 1,250 mL fluid restriction.

1. Determine kcal to be provided as fat.

Estimated kcal x desired percent = kcal as fat 1,700 kcal x 30% = <u>510 kcal fat</u>

2. Convert fat kcal to volume of lipid emulsion.

- a. kcal fat ÷ kcal/gm = gm fat 510 kcal ÷ 9 kcal/gm = 56.7 gm fat
- b. $\underline{gm fat}$ x 100% = % lipid TPN volume $\underline{56.7 gm}$ x 100% = $\underline{4.5\% lipid}$ 1,250 mL
- **3.** Determine kcal to be provided from protein. gm protein x kcal/gm = kcal as protein 70 gm x 4 kcal/gm = <u>280 kcal protein</u>
- 4. Determine amino acid concentration (AA).

<u>gm protein</u>	x 100 = % AA
TPN volume	
<u>70 gm</u>	x 100 = 5.6% AA
1 250 I	

1,250 mL

5. Determine kcal to be provided from carbohydrate. Estimated kcal needs - (kcal as fat + kcal as protein) = carbohydrate kcal 1,700 kcal - (510 kcal + 280 kcal) = 910 kcal carbohydrate

6. Determine carbohydrate concentration.

- a. kcal dextrose ÷ kcal/gm dextrose = gm dextrose 910 kcal ÷ 3.4 kcal/gm = 268 gm dextrose
- b. $\underline{\text{gm dextrose}}$ x 100 = % dextrose TPN volume $\underline{268 \text{ gm}}$ x 100 = $\underline{21.4\% \text{ dextrose}}$ 1250 mL
- 7. Final order:

1.25 liters 5.6% AA, 21.4% dextrose, and 4.5% lipid. This solution will not compound, therefore the dextrose will have to be reduced to $\underline{12.3\%}$ dextrose until fluid restrictions are lifted.*

(See compounding guidelines for specific calculations. Section IV) *If your pharmacy uses a 15% amino acid stock solution, this will compound.

IV. Compounding Parenteral Nutrition

Assume stock solutions of 70% dextrose, 10% amino acids, and 20% lipids.

- 1. Determine TPN volume and grams of carbohydrate, protein, and fat.
- 2. Determine volume of 70% dextrose stock solution.
- 3. Determine volume of 10% amino acid stock solution.
- 4. Determine volume of 20% lipid.
- 5. Determine volume of sterile water and additives.
- 6. Adjust dextrose or other substrates if solution cannot be compounded at the desired volume. Sample calculations:
- A. 2-in-1 solution with fluid restriction 1,250 mL
 - 1. Determine TPN volume and grams of carbohydrate, protein, and fat (see III.B.)
 - 1,250 mL volume
 - 250 mL 20% lipid 268 gm carbohydrate
 - 70 gm protein
 - 2. Determine volume of 70% dextrose solution.

gm dextrose ÷ gm/100 mL stock solution = volume of stock solution 268 gm ÷ 70 gm/100 mL = <u>383 mL 70% dextrose solution</u>

3. Determine volume of 10% AA solution. gm protein \div gm/100 mL stock solution = volume of stock solution $70 \text{ gm} \div 10 \text{ gm}/100 \text{ mL} = 700 \text{ mL} 10\% \text{ AA solution}.$ 4. Determine volume of 20% lipid. 250 mL (see III.B.2.) In a 2-in-1 solution, this is separate from the TPN solution. This will leave 1.000 mL for AA and dextrose. 5. Determine volume of sterile water and additives. Desired volume TPN solution – (volume of dextrose + volume AA) = volume for water and additives. 1,000 mL - (383 mL + 700 mL) = 83 mL These volumes of stock solution do not fit into the desired TPN volume. 6. Adjust solution. a. Determine the volume available for dextrose. Desired volume – (volume of AA solution + volume for additives*) = volume available for dextrose. 1,000 mL - (700 mL + 50 mL) = 250 mL*Usually, 50-100 mL is needed for additives. b. Determine new grams of dextrose. Volume of dextrose x solution concentration = gm dextrose stock solution 250 mL x 70 gm/100mL = <u>175 gm dextrose</u> c. Determine dextrose concentration. <u>gm dextrose</u> x 100 = % dextrose TPN volume <u>175 gm</u> x 100 = <u>17.5% dextrose</u> 1,000 mL B. 3-in-1 solution with fluid restriction of 1,250 mL. 1. Determine TPN volume and grams of carbohydrate, protein and fat (see III.D.). 1,250 mL 268 gm carbohydrate 70 gm protein 56 gm fat 2. Determine volume of 70% dextrose solution. gm dextrose \div gm/100 mL stock solution = volume of stock solution 268 gm ÷ 70 gm/100 mL = 383 mL 70% dextrose solution 3. Determine volume of 10% AA solution. gm protein \div gm/100 mL stock solution = volume of stock solution $70 \text{ gm} \div 10 \text{ gm}/100 \text{ mL} = 700 \text{ mL} 10\% \text{ AA solution}$ 4. Determine volume of 20% lipid. gm fat ÷ gm/100 mL stock solution = volume of stock solution 56 ÷ 20 gm/100 mL = <u>280 mL 20% lipid</u> 5. Determine volume of sterile water and additives. Desired TPN volume – (volume dextrose solution + volume AA solution + volume lipids = volume of water and additives 1,250 mL - (383 mL + 700 mL + 280 mL) = -113 mLThis solution will not compound. 6. Adjust solution. Determine the volume available for dextrose. а Desired volume – (volume of AA solution + volume lipid + volume additives) = volume available for dextrose 1,250 mL - (700 + 280 + 50) = 220 mLb. Determine new grams of dextrose. Volume of dextrose x solution concentration = gm dextrose stock solution 220 mL x 70 gm / 100 mL = 154 gm dextrosec. Determine new dextrose concentration. <u>gm dextrose</u> x 100 = % dextrose TPN solution x 100 = <u>12.3% dextrose</u> <u>154 gm</u> 1.250 mL

Reprinted by permission: from Fish J. Worksheet for calculating total parenteral nutrition. *Support Line*. 1995: 17(6): 10-13.

PEPTIC ULCER

Discussion

Peptic ulcer disease includes esophageal, gastric and duodenual ulcers. Research identifies the Helicobacter (H.) pylori bacteria as the primary cause in 95% of gastric and duodenal ulcers (1-3). The remaining 5% is caused by non-steroidal anti-inflammatory medication usage (eg, aspirin and ibuprofen) and excessive production of stomach acid. The treatment for individuals infected with the H. pylori bacteria includes healing the ulcer with acid suppressing medication and curing the infection by using antibiotics (3-6).

There is no evidence that food, beverages, or spices cause or reactivate ulcers (3,4).

Table III-27: Medical Approaches for Peptic Ulcer Disease

Medical Approaches	Rationale
Avoid foods not tolerated. (See Section ID: "Gastrointestional Soft Diet")	Eliminate foods that cause pain or discomfort to the patient during the acute phases
Antisecretory medication (histamine H ₂ antagonist blocker)	Reduces gastric acid and pepsin secretion
Cimetidine (Tagamet), Ranitidine (Zantac)	
Famotidine (Pepcid), Nizatidine (Axid)	
Antibiotics	Inhibits growth and destroys microorganisms, ie, H. pylori bacteria
Antacids	Buffers acidity
Sucralfate (carafate)	Forms protective coating over ulcer

References

1. National Institutes of Health Consensus Statement. Helicobacter Pylori in Peptic Ulcer Disease. Bethesda, Md: NIH Office of Medical Applications of Research. 1994;12(1):1-15.

2. Marshall B, Warren J. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet.* 1984;1:1311-1315.

3. A breakthrough in ulcer treatment. Tufts University Diet and Nutrition Letter. 1996;13(11):4-6.

4. Rubin, R, Cascade, E, Peura D, et al. Diagnosis and eradication of helicobacter pylori in the management of peptic ulcer disease: a decision analysis model. *Am J Man Care.* 1996; 2(4):375-383.

5. Rubin R, Cascade E, Barker R, et al. Management of dyspepsia: a decision analysis model. *Am J Man Care*. 1996;2(6):145-153.

6. Sung J, Chung S, Ling T, et al. Antibacterial treatment of gastric ulcers associated with helicobacter pylori. *N Engl J Med.* 1995;332:139-142.

PNEUMONIA

Discussion

Pneumonia is defined as inflammation and consolidation of lung tissue in response to an infectious agent (1). Several organisms and disease conditions have been identified to infect or inflame the lungs. The epidemiology of the disease has changed due to changes in the microorganisms and modalities used to treat the condition. The incidence of pneumonia requiring hospitalization is highest among the elderly (2). Subgroups at risk for pneumonia include individuals with chronic obstructive pulmonary disease (COPD), diabetes mellitus, asthma, alcoholism, and heart failure and diseases that affect the immune system (eg, HIV disease/AIDS and cancer). Mechanically ventilated patients are at most risk for developing hospital-acquired pneumonia.

Approximately 50% of pneumonia cases are caused by viruses and tend to be less severe than those of bacterial origin (1-3). Pneumococcus (*Streptococcus pneumoniae*) is the most common cause of bacterial pneumonia (4). Aspiration pneumonia results when solid or liquid food passes into the lungs, causing infection. Aspiration pneumonia results in approximately 50,000 deaths per year, mostly in the elderly (1). Nosocomial pneumonia is the leading cause of death from hospital-acquired infection in the United States (2).

Prevention of pneumonia primarily includes maintenance of immune status and pneumococcal vaccination. Treatment of pneumonia involves a combination of pharmacologic therapy (eg, antibiotics), pulmonary rehabilitation, and maintenance of nutritional status. Protein energy malnutrition (PEM) is associated with involuntary weight loss, functional impairment and impaired immunity (3). It has been demonstrated that nutritional status plays a critical role in the modulation of immune function. In a study of 277 patients admitted to the hospital for treatment of community-acquired pneumonia, the most important factor independently associated with fatal disease was a low serum albumin level (4). In the same study, a serum albumin level under 3.0 g/dL during treatment of the pneumonia was also associated with death due to pneumonia after discharge. Craven and colleagues identified malnutrition as a risk factor for nosocomial pneumonia in hospitalized patients (5).

Approaches

The primary goal of medical nutrition therapy in the management of pneumonia is to preserve lean body mass and immune function, prevent unintentional weight loss, and maintain nutritional status. For detailed intervention strategies, refer to the Pneumonia Medical Nutrition Therapy Protocol in *Medical Nutrition Therapy Across the Continuum of Care* (6).

- **Energy:** Provide enough energy to maintain reasonable body weight. Increased energy may be needed for patients with infection, fever, or weight loss.
- **Protein:** Provide enough protein to maintain visceral protein status and meet the demands of infection.
- **Fluid:** Fluids are encouraged, unless contraindicated. From 3 to 3.5 L of fluid per day has been recommended to liquefy secretions and help lower temperature in febrile patients (7).

Nutrients and the immune system: Several nutrients have been linked to the preservation and maintenance of immune function. Nutrients that have been identified include vitamins A, E, and B₆, zinc, copper, selenium, the amino acids glutamine and arginine, and omega-3 fatty acids. These nutrients all seem to modulate specific aspects of human immune function (8). Current studies do not demonstrate a direct cause and effect relationship with the incidence of pneumonia. The current thought is that these nutrients may play a key role in the immune function, leading to less of a risk of developing pneumonia (9). Currently, supplementation with these identified nutrients is not warranted. However, it is recommended to increase the consumption of foods that provide these nutrients as good sources, such as fruit, vegetables, grains, meats, and fish.

Aspiration risk reduction: Instituting feeding techniques that prevent risk for aspiration may be indicated in patients who demonstrate symptoms of aspiration, such as coughing before, during, or after consumption of solids, liquids or medications; drooling; pocketing food in the mouth; and repetitive movement of the tongue from front to back of the mouth. To reduce the risk of aspiration, consider the following strategies (10):

- Position patient at a 90° angle during meals.
- Serve food at appropriate temperatures.
- Limit mealtime distractions.

- Encourage small bites.
- Avoid using straws since liquids will be rushed to the back of the mouth before swallowing is safe.
- Avoid serving thin liquids, as they can be easily aspirated. Thickened liquids will slow transit time.

References

- 1. Marrie TJ. Bacterial pneumonia. In: Conn RB, Borer WZ, Snyder JW, eds. *Current Diagnosis 9*. Philadelphia, Pa: WB Saunders; 1997: 307-311.
- 2. Pneumonia. American Lung Association; 1996. Fact sheet.
- 3. White J, ed. The Role of Nutrition in Chronic Disease Care. Washington, DC: Nutrition Screening Initiative; 1997:22-35...
- 4. Hedlund JU, Hansson LO, Ortqvist AB. Hypoalbuminemia in hospitalized patients with community-acquired pneumonia. Arch Intern Med. 1995;155:1438-1442.
- 5. Craven DE, Steger KA, Barat LM, Duncan RA. Nosocomial pneumonia: epidemiology and infection control. *Intensive Care Med.* 1992; 18(suppl 1):S3-9.
- 6. Inman-Felton A, Smith K, Johnson E, eds. *Medical Nutrition Therapy Across the Continuum of Care*. 2nd ed. Chicago, Ill: American Dietetic Association; 1998.
- 7. Escott-Stump S. Nutrition and Diagnosis-Related Care. 5th ed. Baltimore, Md: Lippincott Williams & Wilkins; 2002:199.
- 8. Romore MM. Vitamin A as an immunomodulating agent. *Clin Pharm*. 1993;12:506-514.
- 9. Chandra RK. Effect of vitamin and trace element supplementation on immune responses in elderly subjects. *Lancet.* 1992;340:1124-1127.
- 10. Neidert KC, ed. Nutrition Care of the Older Adult. Chicago, Ill: American Dietetic Association; 1998:213.

PRESSURE ULCERS

Discussion

A pressure ulcer is an area of localized injury to the skin and/or underlying tissue, usually over a bony prominence, as a result of pressure alone or pressure combined with shear or friction (1). Pressure ulcers can develop within 2 to 6 hours when normal capillary blood flow is obstructed, leading to tissue necrosis (1). Persons who have comorbidities or who are severely ill are more vulnerable to developing pressure ulcers (1).

The National Pressure Ulcer Advisory Panel (NPUAP) developed a staging system to classify pressure ulcers in 1989 and revised the staging system in 2007. The new staging system consists of six categories: stages I to IV, unstageable, and suspected deep tissue injury (1,2).

Stage I: Intact skin with non-blanchable redness of localized area usually over a bony prominence. Darkly pigmented skin may have visible blanching; its color may differ from the surrounding area. (The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Stage I may be difficult to detect in individuals with dark skin tones.)

Stage II: Partial thickness loss of dermis presenting as a shallow, open ulcer with a red-pink wound bed, without slough. May also present as an intact or open/ruptured, serum-filled blister. (Presents as a shiny or dry, shallow ulcer without slough or bruising, which indicates suspected deep tissue injury. This stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration, or excoriation.)

Stage III: Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling. (The depth of a stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue and stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep stage III pressure ulcers. Bone/tendon is not visible or directly palpable.)

Stage IV: Full thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling. (The depth of a stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or supporting structures (eg, fascia, tendon, or joint capsule), making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable.)

Unstageable: Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. (Until enough slough and/or eschar is removed to expose the base of the wound, the true depth and, therefore, stage cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as "the body's natural biological cover" and should not be removed.

Suspected deep tissue injury: Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.

Nutrition Assessment and Diagnosis

A number of contributing or confounding factors are associated with pressure ulcers, although the significance of these factors remains unknown (1). Surveys report a high prevalence of pressure ulcers in hospitalized patients (3% to 4%) and in residents of nursing homes (20% to 33%) that coincides with a high prevalence of malnutrition (30% to 50% and 19% to 59%, respectively) (3). Although poor nutrition is commonly cited as a risk factor for the development of pressure ulcers, the precise role of nutritional status remains controversial (3). Common causes of pressure ulcers include restricted mobility and limited physical activity, a compromised level of consciousness, incontinence, peripheral vascular disease that causes poor circulation and lack of oxygen to the tissues, and conditions that cause impaired sensory perception. Other

conditions that place a patient at risk for the development of pressure ulcers include: diabetes mellitus; obesity; chronic obstructive pulmonary disease; sepsis; chronic or end-stage renal, liver, or heart disease; diseases related to immunosuppression; hip fractures; and spinal cord injury. Medical treatments and medications that may contribute to the risk of pressure ulcer development include antikinetic drugs (eg, antidepressants and sleeping pills), immunosuppressive drugs, steroids, radiation, chemotherapy, and renal dialysis. Malnutrition, dehydration, or unintentional weight loss (greater than 5% in 1 month, 7.5% in 3 months, or 10% in 6 months), whether secondary to poor appetite or another disease process, places the client at risk of tissue breakdown and poor healing (4).

Patients with risk factors for pressure ulcer development should receive a complete nutrition assessment and care plan designed to address each nutrition problem area that is identified (2). It is important to identify and assess patients who are at risk of developing pressure ulcers. Assessment of pressure ulcer risk should be documented with a validated tool, such as the Braden scale (2-5). Patients who are identified to be at risk should be monitored at regular intervals in a preventive program when appropriate. Monitoring may include a systematic skin inspection at least daily (paying particular attention to the bony prominences), daily physical activity or a mobility program (1), and routine evaluation of nutritional and hydration status according to the organization's protocols. National guidelines have been established to address the issue of pressure ulcers and the best practice guidelines for care (2).

Nutrition Intervention

Patients who require treatment of pressure ulcers should receive adequate nutrition, including energy, protein, fluids, and vitamins and minerals (1,2). The assessment should include a review of the exudate losses from wounds, in consideration of fluid and protein losses. The following guidelines will usually meet the patient's needs (2):

- Provide a well-balanced diet adequate in energy, high-biological value protein, and fluid as well as vitamins and minerals to meet the estimated requirements. The goal is to maintain or regain lost weight (1). In general, the patient should be on the least restricted diet possible (1).
- The energy requirement for wound healing is not known. Recommendations must be individualized with the goal to provide adequate energy for anabolism and collagen synthesis (6). Adequate energy intake should be determined by using the appropriate prediction equation as outlined in Section II: "Estimation of Energy Expenditures". The general recommendation for people with pressure ulcers is 30 to 35 kcal/kg (1,6-8). In the National Pressure Ulcer Long-Term Care Study, adequate nutrition support at 30 kcal/kg of actual body_weight per day was a strong predictor of stage III and IV pressure ulcer healing (9). The NPUAP recommends increasing the energy level to 35 to 40 kcal/kg per day for people who are underweight or losing weight (1,6). A recent meta-analysis suggests using the Harris-Benedict equation multiplying with a correction factor of 1.1 to accurately assess energy needs in patients with pressure ulcers (10). The analysis found that energy intake of 30 kcal/kg/day is appropriate to cover the daily requirements of patients with pressure ulcers (10). When available, indirect calorimetry is recommended to more closely identify the individual energy needs of patients who fail to achieve anabolism; exhibit delayed wound healing; require more energy to assist in healing larger or multiple wounds; or need a more aggressive approach to the nutrition care plan (3).
- Provide adequate protein for a positive nitrogen balance. Adequate protein is essential in all stages of wound healing; but, without adequate energy intake, the protein will be used as an energy source (6). Daily protein intake of 1.25 to 1.5 g/kg of actual body weight from food sources of high-biological value protein (1) is the most commonly cited recommendation in the literature (1.6) The European Pressure Ulcer Advisory Panel recommends 1.0 to 1.5 g/kg per day (6.7). Excess dietary protein in amounts greater than 1.5 to 2.0 g/kg per day can be a risk factor for dehydration, especially in the elderly (6.8,11).
- Provide adequate fluid intake each day to keep the patient well hydrated and prevent dehydration (6). The optimal fluid intake is 30 to 35 mL/kg of actual body weight or a minimum of 1,500 mL/day (2,6). It is often difficult to meet fluid needs when fluids are only provided with meals. Ensure supplementary fluids are provided to meet fluid needs. Patients who are on thickened liquid meal plans should be carefully monitored and supplemented with fluids as needed (6). In addition, patients who are medically managed on air-fluidized beds may be at greater risk for dehydration and should be evaluated for greater fluid needs. Patients who require air-fluidized beds set at a high temperature will need additional fluids, estimated to be approximately 10 to 15 mL/kg because of an increase in insensible water loss (6,11,12). Refer to "Nutrition Management of Fluid Intake and Hydration" in Section IA for guidelines on fluid requirements for patients being treated on an air-fluidized bed.
Pressure Ulcers

• A daily multiple vitamin and mineral supplement meeting 100% of the Recommended Daily Allowances (RDAs) (Dietary Reference Intakes [DRIs]) is given if energy intake is substantially below the required level, or if vitamin or mineral deficiencies are confirmed by laboratory assessment (2,6). Supplementation should not be greater than 10 times the RDAs (DRIs) for water-soluble vitamins (2). However, this recommendation should be reevaluated in light of the most recent DRIs, in which the Tolerable Upper Intake Levels for vitamins are often much less than 10 times the RDA (6). For patients with renal failure, current guidelines recommend giving no more than 60 to 100 mg of vitamin C per day due to the risk of renal oxalate stone formation (3).

Special Considerations

Vitamins: All vitamins are essential in wound healing; however, vitamins A, E, C, and K (especially vitamins C and A) have been given the most attention (6). Limited research is available to validate the roles of specific vitamins in wound healing. Although the research may show that <u>increased vitamin intake improves blood</u> assay results, there is no evidence that vitamins directly impact pressure ulcer healing rates (13). Studies demonstrate that a lack of adequate energy and protein intake places the patient at the greatest risk of pressure ulcer development (6).

Trace minerals: The trace elements present in the body, zinc, copper, and iron, have the closest relationship to wound healing (6,13). However, studies have not demonstrated a significant improvement in pressure ulcer healing with routine zinc supplementation (14,15). No studies have shown improvement in wound healing after the administration of zinc to patients who are not zinc deficient (6). The European Pressure Ulcer Advisory Panel has also concluded that there is no evidence to recommend zinc supplementation for patients with pressure ulcers (7). If a zinc deficiency is confirmed or suspected, zinc supplementation is warranted (6). The Tolerable Upper Intake Level of zinc for healthy adults is 40 mg/day (6). Clinicians should be aware of adverse side effects of zinc supplementation, including an adverse effect on copper and calcium status, compromised immune responses, and gastrointestinal effects (eg, nausea, vomiting, and diarrhea) (3,6). To minimize the risk of adverse effects, zinc supplementation should not be given longer than 2 to 3 weeks (3,6). Although low serum zinc concentrations have been associated with impaired healing and alterations in immune function (13,14), serum zinc levels decrease with inflammation and may not accurately represent the total level of zinc in the body (6,16).

Although vitamin C and zinc may play a role in wound healing, the adequacy of total energy, protein, and fluid to maintain nutritional status remains the most relevant dietary approach to the prevention and healing of pressure ulcers (3,6). Thompson and Fuhrman's in-depth review of the literature on nutrients and wound healing is recommended for further information (17). Table III-28 lists guidelines for supplementation if a deficiency is suspected or confirmed.

Stage	vitamin C"	ZINC
Stages I and II	100 to 200 mg/day	15 mg elemental zinc per day (or RDA); if deficiency is suspected, supplement with up to 50 mg/day for no longer than 10 to 14 days.
Stages III and IV	Up to 1,000 to 2,000 mg/day in divided doses for stressed patients or patients at risk for deficiency; reassess after 10 to 14 days.	25 to 30 mg elemental zinc per day for patients at risk for marginal zinc status; reassess after 10 to 14 days; ongoing losses may warrant longer supplementation.

Table III-28: Guide	lines for Vitamin C an	d Zinc Supplementation (3)
Data a survey III a sur	Vitamin Ca	77 ah

^aSome references suggest high-dose vitamin C supplementation is warranted in conditions such as acute stress, smoking, and malnutrition to ensure adequate tissue stores for wound healing. These data have not been validated. Note that the Tolerable Upper Intake Level is 2,000 mg/day. There is an increased risk of oxalate stone formation when excessive vitamin C is given to patients with chronic renal failure. Therefore, doses > 500 mg/day should be reviewed by a nephrologist (3).

^bA 220-mg dose of zinc sulfate is equivalent to 50 mg of elemental zinc (a common oral supplement dosage). Continued zinc supplementation may be warranted with chronic losses (eg, high-volume fistula, ileostomy, or diarrhea losses). In some cases, the recommended supplementation may exceed the Tolerable Upper Intake Limit of 40 mg/day (3). Parenteral requirements are significantly less than oral or enteral requirements because of the different absorption rates (3).

Medical nutrition supplements and enteral feedings: If the patient consistently consumes less than 50% of the estimated energy and protein needs, the need for oral supplementation or enteral nutrition support should be evaluated (3,6). The use of food alone has shown to be ineffective in improving pressure ulcers (6).

For patients who are underweight (body mass index < 20) and have challenges meeting nutritional requirements, oral intake of medical nutrition supplements is very effective in promoting intake and weight gain and improving functional status (6). Medical nutritional supplements provide a better source of energy, protein, and micronutrients than food snacks and do not inhibit meal intake (6,18). If the patient is tube fed, the feeding should provide a minimum of 100% of the patient's energy, protein, and RDAs (DRIs) for vitamins and minerals (2). Because the effectiveness of specialized enteral formulas for wound healing has not been validated, further study is needed before the routine use of wound healing formulas can be recommended (3).

Amino acids: The amino acids arginine and glutamine have been studied for their possible role in enhancing wound healing. However, the literature does not provide evidence to support the supplementation of the diet with these amino acids (3).

Evaluation and Monitoring

Treatment of pressure ulcers should always be provided by a multidisciplinary team (2). Nutrition intervention is part of a multidisciplinary approach that also includes nursing intervention (reduction of pressure and shearing, skin and wound care, feeding assistance, and a bowel and bladder care program), medical intervention (treatment of infection and other medical conditions), and physical and occupational therapy (promotion of increased activity and feeding ability).

References

- 1. National Pressure Ulcer Advisory Panel. Updated Staging System. 2007. Available at: www.npuap.org/pr2.htm. Accessed November 5, 2007.
- 2. *Pressure Ulcer Treatment Clinical Practice Guidelines*. Rockville, Md: US Dept of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; 1994. Quick Reference No. 15, AHCPR publication 95-0650.
- 3. Stechmiller JK, Cowan L, Johns P. Wound healing. In: Gottschlich MM, ed. *The A.S.P.E.N. Nutrition Support Core Curriculum: A Case-Based Approach—The Adult Patient.* Silver Spring, Md: American Society of Enteral and Parenteral Nutrition; 2007:405-423.
- 4. Skin integrity. In: Neidert KC, ed. *Nutrition Care of the Older Adult.* Chicago, Ill: Consulting Dietitians in Health Care Facilities, A Practice Group of the Academy of Nutrition and Dietetics; 1998.
- 5. Braden BJ, Bergstrom N. Predictive validity of the Braden Scale for pressure sore risk in a nursing home population. *Res Nurs Health*. 1994; 17:459-470.
- 6. Pressure ulcers. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; 2007. Available at: nutritioncaremanual.org. Accessed November 5, 2007.
- 7. Clark M, Schols JM, Benati G, Jackson P, Engfer M, Langer G, Kerry B, Colin D. Pressure ulcers and nutrition: a new European guideline. J Wound Care. 2004;13:267-272.
- 8. Thomas DR. Improving outcome of pressure ulcers with nutrition interventions: a review of the evidence. *Nutrition*. 2001;17:121-125.
- 9. Bergstrom N, Horn SD, Smout RJ, Bender SA, Ferguson ML, Taler G, Sauier AC, Sharkey SS, Voss AC. The National Pressure Ulcer Long-term Care Study: outcomes of pressure ulcer treatments in long-term care. J Am Geriatr Soc. 2005;53:1721-1729.
- 10. Cereda E, Klersy C, Rondanelli M, Caccialanza R. Energy balance in patients with pressure ulcers: A systematic review and metaanalysis of observational studies. J Am Diet Assoc. 2011;111:1868-1876.
- 11. Ayello EA, Thomas DR, Litchford MA. Nutrition aspects of wound healing. Home Healthc Nurse. 1999;17:719-729.
- 12. Breslow RA. Nutrition and air-fluidized beds: a literature review. Adv Wound Care. 1994;7:57-62.
- 13. Williams JZ, Barbul A. Nutrition and wound healing. *Surg Clin North Am.* 2003;83:571-596.
- 14. Gray M. Does oral zinc supplementation promote healing of chronic wounds? J Wound Ostomy Continence Nurs. 2003;30:295-299.
- 15. Haggard J, Houston MS, Williford JH, Meserve LA, Shewokis P. Retrospective study of the effects of zinc supplementation in an elderly institutionalized population with decubitus ulcers. *J Am Diet Assoc.* 1999;99:A-11. Abstract.
- 16. Fuhrman MP. Wound healing and nutrition. Top Clin Nutr. 2003;18:100-110.
- 17. Thompson C, Fuhrman MP. Nutrients and wound healing: still searching for the magic bullet. Nutr Clin Pract. 2005;20:331-347.
- 18. Stratton RJ, Boywer G, Elia M. Greater total energy and protein intakes with liquid supplements than food snacks in patients at risk of malnutrition. Poster presented at: European Society of Parenteral and Enteral Nutrition; October 21, 2006; Istanbul, Turkey.

MANAGEMENT OF ACUTE KIDNEY INJURY AND CHRONIC KIDNEY DISEASE (Stage V)

Acute Kidney Injury

An international network of kidney and critical care specialists, the Acute Kidney Injury Network, developed consensus recommendations for the terminology, diagnostic criteria, and staging of acute kidney injury (AKI). AKI replaces the term acute renal failure, as the condition does not always result in renal failure (1). The diagnostic criterion for AKI is an abrupt (within 48 hours) reduction in kidney function defined as an absolute increase in the serum creatinine level of at least 0.3 mg/dL, a 50% increase in the serum creatinine level, or a documented urine output of less than 0.5 mL/kg per hour for more than 6 hours (1). (Refer to Table III-29)

Table l	III-29: Classification/Staging System for AKI (1)	
Stage	Creatinine Clearance	Urine Output
1	Serum creatinine increase of at least 0.3 mg/dL, or a 150% to 200% increase	<0.5 mL/kg per hour for more than 6 hours
2	Increase in serum creatinine level to greater than 200% to 300% of baseline	<0.5 mL/kg per hour for more than 12 hours
3	Increase in serum creatinine level to greater than 300% of baseline, or serum creatinine level of 4.0 mg/dL with an acute increase of at least 0.5 mg/dL	<0.3 mL/kg per hour for 24 hours or anuria for 12 hr

Causes of AKI include:

- systemic shock due to a sudden loss of blood supply to the kidneys from trauma, surgical complications, or sepsis
- exposure to a nephrotoxic chemical or drug (eg, radiologic dyes, cleaning solvents, pesticides, and gentamicin)
- streptococcal infection

AKI is often complicated by sepsis, trauma, and multiple organ failure (2). Acute kidney injury (AKI) occurs in approximately 20% of hospitalized patients and is associated with a 40% to 80% mortality rate (2-4). Continuous renal replacement therapy (CRRT) is a type of dialysis used for hemodynamically unstable patients who have AKI, as it is better tolerated than conventional intermittent hemodialysis (2). CRRT removes fluids and solutes slowly, corrects electrolyte and metabolic abnormalities, and maintains fluid balance until renal function returns or until the patient can tolerate hemodialysis (3). The primary types of CRRT include continuous hemofiltration, continuous hemodialysis, continuous hemodiafiltration, and slow continuous ultrafiltration. Peritoneal dialysis is another option, but it is often contraindicated in critically ill patients (2).

Nutrition Assessment and Nutrition Intervention in AKI

AKI causes nutritional imbalances including acidosis, hyperkalemia, hyperphosphatemia, fluid disturbances, impaired glucose utilization, protein catabolism, accumulation of metabolic waste, and a rapid decrease in urine output (4). Patients who have AKI should receive a comprehensive nutrition assessment to identify nutrition diagnoses and close monitoring to ensure that nutrition care outcomes and goals of therapy are achieved. The nutrition interventions should be based on the individualized patient assessment and identified nutrition diagnosis. Nutrition interventions should complement medical management strategies such as CRRT to optimize the patient's treatment response. Patients who have AKI are hypermetabolic and hypercatabolic as a result of the neurohumoral response associated with acute injury (3-6). The primary goals of medical nutrition therapy are to provide adequate protein, energy, and nutrients and to minimize malnutrition (2.6). The Academy of Nutrition and Dietetics, formerly American Dietetic Association, published *Guidelines for Nutrition Care of Renal Patients* in 2002; however, this publication has not been updated to reflect the changes in medical management strategies (5). Specific nutrition intervention strategies for acute care management of AKI are found in the Morrison Nutrition Practice Guideline – Acute Kidney Injury (AKI) (7). The following summary includes the most recent management strategies and guidelines for medical nutrition therapy in patients who have AKI (2,5,6,8).

Energy: Individualize based on level of care (eg, critically ill) or indirect calorimetry. Use 25 to 35 kcal/kg of actual body weight as an estimation of energy requirements. Energy expenditure and requirements will

depend on the patient's stress level, acuity level, and nutritional status and should include the energy obtained from CRRT (2). (Refer to Section II: "Estimation of Energy Expenditures".)

Protein: The optimal protein intake for patients who have AKI remains controversial and should be prescribed based on the degree of catabolism and type of renal replacement therapy (2,9). Without dialysis or catabolism, provide 0.8 to 1.2 g/kg of actual body weight. In the presence of catabolism, provide 1.2 to 1.5 g/kg of actual body weight. CRRT can remove amino acids and proteins; therefore, a minimal protein intake of 1.5 g/kg of recommended body weight per day is suggested (4,9). If CRRT is frequently used, the protein requirements may be higher.

Sodium: Sodium intake should be 2,000 to 3,000 mg/day based on the patient's blood pressure and the presence of edema. During the diuretic phase, replace sodium losses based on urinary output, edema, renal replacement therapy, and serum sodium levels.

Potassium: Provide 2,000 to 3,000 mg/day of potassium. During the diuretic phase, replace potassium losses based on urinary volume, serum and urinary potassium levels, renal replacement therapy, and drug therapy.

Phosphorus: Provide 8 to 15 mg/kg of phosphorus. Closely monitor the phosphorus levels of patients who receive renal replacement therapy.

Calcium: Maintain serum levels of calcium within normal ranges. Closely monitor the calcium levels of patients who receive renal replacement therapy.

Fluids: The daily fluid intake should be 500 mL plus the volume of urine output. The fluid intake also depends on the serum and urinary sodium level, total fluid output (including urine), and type of dialysis.

Vitamin and mineral supplementation: Individualize supplementation based on laboratory values, documented deficiencies, and the type of renal replacement therapy. Ensure that the Dietary Reference Intakes for vitamins and minerals are provided. Patients who receive CRRT must be carefully monitored because CRRT causes a significant loss of magnesium, calcium, phosphorus, and potassium (2,10). Patients who have AKI require supplementation of water-soluble vitamins to prevent deficiency caused by renal replacement therapy losses, inadequate intake, drug-nutrient interactions, and higher needs (2,6). Although the evidence is limited, the supplementation guidelines in Table III-30 have been proposed for patients who have AKI and receive CRRT (specifically continuous venovenous hemofiltration) (4). In addition, critically ill patients who stay in the intensive care unit for more than 10 days are often deficient in vitamin D and have increased bone turnover (11). Supplementation of vitamin D in AKI needs further research, because the mechanism of vitamin D metabolism" is not the same as in end-stage renal disease (11).

Table III-30: Vitamin and Mineral Supplementation in AKI Managed with CRRT (2,4)

Vitamin/Mineral	Dose
Vitamin K	4 mg/week
Vitamin E	10 IU/day
Niacin	20 mg/day
Thiamin	1.5 mg/day
Riboflavin	1.5-1.7 mg/day
Pantothenic acid (vitamin B ₅)	5-10 mg/day
Vitamin C	60-125 mg/day
Biotin	150-300 mcg/day
Folic acid	1 mg/day
Vitamin B ₁₂	4 mcg/day
Zinc	20 mg ?ª
Vitamin A	Avoid

^a The question mark is included in the reference article (4).

Also refer to Section G: "Medical Nutrition Therapy for Chronic Kidney Disease" for information and nutritional guidelines associated with CRRT.

Chronic Kidney Disease

Chronic kidney disease (CKD) is the result of the progressive deterioration of kidney tissue during several months or years as scar tissue is substituted for viable kidney tissue. Patients who have lost 85% or more of their kidney function have stage V CKD and require maintenance renal replacement therapy (dialysis) or renal transplantation (5,6).

Causes of CKD include:

- diseases of the glomeruli (glomerulonephritis)
- blood vessel damage in the kidney by nephrosclerosis from high blood pressure
- inherited diseases, such as polycystic kidney disease
- obstructive diseases, such as kidney stones
- congenital birth defects of the kidney and urinary tract
- systemic or metabolic diseases, such as diabetic nephropathy, systemic lupus erythematosus, and hyperuricemia, in which the kidneys and urinary tract are irreversibly damaged
- abuse of analgesic or illicit drugs

Nutrition Assessment and Nutrition Intervention in CKD

When evaluating the patient for nutrition intervention, the dietitian should use established practice guidelines (5) and the norms established at the institution-specific guidelines for a particular dialysis unit. Biochemical levels for dialysis patients will seldom be the same as for healthy individuals, since dialysis cannot completely replace kidney function. Various nephrologists may define normal levels differently, and the goal levels may be different based on the stage (1-5) of CKD and the management therapies. The following text outlines the biochemical parameters that are affected by CKD. These biochemical parameters should be routinely evaluated following institution-specific guidelines, especially guidelines that are certified by the Centers for Medicare and Medicaid Services.

Albumin: The presence of acute or chronic inflammation limits the specificity of albumin and other acutephase hepatic proteins as nutritional markers in CKD (12). The normalized protein catabolic rate may be a better marker of nutritional status in the CKD patient who receives renal replacement therapy. Uremia depresses albumin metabolism, which can affect the albumin concentration. Mean albumin levels are lower in CKD patients who have a glomerular filtration rate (GFR) that is less than 60 mL/min. Compared with normal levels, however, this decreased albumin level may be a reflection of decreased protein intake (5). Albumin losses are greater in patients who receive peritoneal dialysis. The National Kidney Foundation guidelines have not been updated to reflect the current evidence documenting the limitations for using albumin as an indicator for diagnosing nutrition related disorders or malnutrition in patients with chronic disease. The 2000 K/DOQI guidelines recommend for individuals with a GFR < 20 mL/min, protein-energy nutrition status be evaluated by serial measurements of a panel of markers including albumin and normalized protein nitrogen appearance (nPNA) (12). (See the discussion of protein requirements in "Medical Nutrition Therapy for Chronic Kidney Disease" in Section IG.)

Lipids: Increased lipid levels are associated with accelerated cardiovascular disease in patients who have renal failure. Cardiovascular disease is responsible for approximately 50% of all deaths in dialysis patients (13). The primary abnormality is a reduction in the catabolism of lipoproteins with unchanged or low hepatic synthesis. Dialysis patients commonly have increased levels of total cholesterol, very low-density lipid cholesterol, and triglycerides and decreased levels of high-density lipoprotein cholesterol (5). The National Kidney Foundation Task Force on Cardiovascular Disease has recommended the National Cholesterol Education Program Adult Treatment Panel III guidelines for patients who have chronic renal disease. Individuals who have low, low-normal, or decreasing serum cholesterol levels should be examined for nutritional deficits (12).

Blood (serum) urea nitrogen: A nitrogenous waste product of protein metabolism, the level of blood urea nitrogen increases with increased protein intake, catabolism, gastrointestinal bleeding, glucocorticoid use, or decreased dialysis efficiency. A low level of blood urea nitrogen may indicate decreased protein intake, loss of protein through emesis or diarrhea, frequent dialysis, protein anabolism, or overhydration. Values greater than 90 to 100 mg/dL may lead to azotemia.

Potassium: Hyperkalemia, which is a potassium level greater than 5.5 mEq/L, is potentially life-threatening and may precipitate cardiac arrest if not treated. When hyperkalemia occurs in patients who have chronic

Management of Acute Kidney Injury and Chronic Kidney Disease

renal failure, it is usually caused by oliguria (24-hour urine output <500 mL); excessive potassium intake; acidosis; the catabolic stress of infection, surgery, or trauma; inadequate dialysis or renal replacement therapy; or hypoaldosteronism. Excessive potassium intake is frequently related to the use of potassium-containing salt substitutes and dietary noncompliance. Hypokalemia, which is a potassium level less than 3.5 mEq/L, is caused by decreased dietary intake; vomiting; diarrhea; potassium-depleting diuretics; excessive use of sodium polystyrene sulfonate (Kayexalate), a potassium binder; or a low-potassium dialysate.

Sodium: The evaluation of serum sodium levels must always include the patient's hydration status. Hypernatremia can be caused by excessive water loss through diarrhea and vomiting (dehydration) and aggressive diuretic therapy without sodium restriction. Signs of hypernatremia include flushed skin, dry tongue and mucous membranes, and thirst. Hyponatremia can be caused by fluid overload and sodium depletion from sodium restriction along with sodium-losing nephropathy. Symptoms of hyponatremia include abdominal cramps, hypotension, and headaches.

Calcium: In CKD, calcium absorption decreases secondary to the abnormal metabolism of vitamin D. Hyperphosphatemia also leads to decreased serum calcium levels, which contribute to secondary hyperparathyroidism and renal osteodystrophy (14). Derangements in mineral and bone metabolism common to CKD are associated with increased morbidity and mortality (14). This finding prompted the development of Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease by the Kidney Disease Outcomes Quality Initiative (K/DOQI) (14). These guidelines provide recommendations for the evaluation of phosphorus, calcium, and plasma intact parathyroid hormone and management and treatment with vitamin D, phosphate binders, and the dialysate bath (14). Serum levels of phosphorus, ionized calcium or a corrected total calcium, and plasma parathyroid hormone should be monitored, and the appropriate guideline recommendations should be implemented when necessary (14). The goal of therapy is to achieve a normal range of serum calcium, with the optimum level of 8.4 to 9.5 mg/dL (5.14). The presence of calcium in the dialysate helps to normalize serum calcium levels in patients who receive hemodialysis, along with the use of an activated source of vitamin D (calcitriol), an oral calcium supplement, a vitamin D analog (doxercalciferol or paricalcitol), and a phosphorus binder. The most accurate method for assessing calcium abnormalities is to measure the ionized calcium directly (2). It is an accepted practice that total calcium levels need to be adjusted for the level of albumin to better reflect the jonized calcium (14). The corrected total calcium is considered when the albumin level—not the serum calcium—is low (14). Currently two formulas are suggested by K/DOQI guidelines for use (14). The first formula presented used a preferable study design in the validation and most closely approximates corrected total calcium in patients with CKD (14):

Corrected calcium (mg/dL) = Total Calcium (mg/dL) + 0.0704 x [34 – Serum albumin (g/L)*] (2,14) *uses g/L vs. g/dL

However, for the routine clinical interpretation of serum calcium needed for appropriate care of patients with kidney disease, a simple formula for adjutsting total serum calcium concentrations for changes in serum albumin concentration can by used by clinicians. This formula yields similar results to the formula described above (2,14).

Corrected total calcium: Total calcium (mg/dL) + 0.8 x [4.0 – Serum albumin (g/dL)] (2,14).

The most accurate method for assessing calcium abnormalities is to measure the ionized calcium directly. The second equation often overestimates the correted calcium concentration in critically ill patients receiving specialized nutrition support (2,15).

Phosphorus: Low levels of serum phosphate may lead to phosphorus depletion and osteomalacia. The goal of therapy is to maintain the phosphorus level between 2.7 to 5.0 mg/dL in patients who have stages I-IV CKD and between 3.5 to 5.5 mg/dL in patients who receive renal replacement therapy (5.6). Refer to the Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease by the Kidney Disease Outcomes Quality Initiative (14).

Calcium-phosphorus product: The calcium-phosphorus product, which is the result of multiplying the serum values of calcium and phosphorus, should be less than $55 \text{ mg}^2/dL^2$ to prevent soft-tissue calcification (16). Research regarding the calcium-phosphorus product is limited. Therefore, a target phosphorus level of less than 5.0 mg/dL is suggested for patients who have CKD.

Creatinine: Creatinine is a product of muscle metabolism that is used to assess renal function. A serum creatinine level that is double the normal level of 0.5 to 1.5 mg/dL suggests a greater than 50% nephron loss,

Management of Acute Kidney Injury and Chronic Kidney Disease

whereas a serum creatinine level of 10 mg/dL suggests a 90% nephron loss or end-stage renal disease. Serial serum creatinine levels can be used to determine the consistency of dialytic therapy. Eventually, a normal creatinine level can be established for each dialysis patient based on the patient's muscle mass and dialysis prescription. The predialysis or stabilized serum creatinine and the creatinine index reflect the sum of dietary intake of foods rich in creatine and endogenous creatinine production minus the urinary excretion, dialytic removal, and endogenous degradation of creatinine. Individuals who have low levels of predialysis or stabilized serum creatinine levels usually can be traced to changes in the dialysis regimen, such as skipped treatments, decreased dialysis time, or poor blood flow through an access. Increased blood urea nitrogen and serum potassium levels accompanied by a sudden increase in the serum creatinine level and a decrease in carbon dioxide level usually indicate decreased waste product removal.

Glucose: Normal glucose levels should be maintained in all dialysis patients to prevent the complications of hypoglycemia and hyperglycemia. Abnormal carbohydrate metabolism resulting in hyperglycemia occurs in individuals who are approaching end-stage renal disease. Although the cause of this abnormal carbohydrate metabolism is not known, the abnormality resolves after several weeks of dialysis therapy or after transplantation. High blood glucose levels can increase thirst, decrease serum sodium levels, and increase serum potassium levels. Acidosis, which is indicated by decreased carbon dioxide levels and an increased anion gap, increases protein catabolism and often accompanies increased blood glucose levels in patients who have chronic renal failure (17,18).

Glomerular Filtration Rate (Creatinine Clearance) (2,5,19)

Creatinine clearance is the most commonly used measurement of GFR. The normal GFR is 125 mL/min. Direct urinary clearance measurements are useful in determining the degree of renal dysfunction at lower levels of clearance (5). The estimated GFR provides a useful approximation value (ie, <25 mL/min) (12).

In principle, there is a reciprocal relationship between serum creatinine and creatinine clearance. To estimate creatinine clearance, factors such as body weight, age, and sex must be considered since creatinine increases with body weight and musculature and decreases with age. The relationship between serum creatinine and creatinine clearance is not valid for patients who receive dialysis, patients who have acute renal failure, or patients in a catabolic state in which muscle mass is being destroyed.

The most widely used method for estimating GFR is the Cockcroft-Gault equation (2,19):

$$GFR (Men) = \frac{Weight (kg) \times (140 - Age)}{72 \times Serum creatinine (mg/dL)}$$

$$GFR (Women) = \frac{Weight (kg) \times (140 - Age)}{72 \times Serum creatinine (mg/dL)} \times 0.85$$

For comprehensive guidance on the nutrition assessment, nutrition diagnosis, and nutrition intervention and the monitoring parameters in the acute care setting refer to Morrison Nutrition Practice Guideline – Chronic Kidney Disease (CKD) (20).

References

- 1. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A, for Acute Kidney Injury Network. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care*. 2007;11:R31. Available at: http://ccforum.com/content/11/2/R31. Accessed January 12, 2009.
- 2. Renal failure and Chronic Kidney Disease. In: *Nutrition Care Manual*. Academy of Nutrition and Dieteics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed January 31, 2013.
- 3. Paton M. Continuous renal replacement therapy: slow but steady. *Nursing.* 2003;33:48-50.
- 4. Marin A, Hardy G. Practical implications of nutritional support during continuous renal replacement therapy. *Curr Opin Clin Nutr Metab Care.* 2001;4:219-225.
- 5. Wiggins KL, ed. *Guidelines for Nutrition Care of Renal Patients*. Chicago, Ill: American Dietetic Association; 2002.
- 6. Kopple JD, Massry SG, eds. Nutrition Management of Renal Disease. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2004.
- Morrison Nutrition Practice Guideline Acute Kidney Injury. In: Inman-Felton A, Smith KG. Morrison Nutrition Practice Guidelines. Atlanta, Ga: Morrison Management Specialists Inc; 2012. Available at: www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition
- 8. Byham-Gray L, Wiesen K, eds. A Clinical Guide to Nutrition Care in Kidney Diseases. Chicago, Ill: American Dietetic Association; 2004.
- 9. Wooley JA, Btaiche IF, Good KL. Metabolic and nutritional aspects of acute renal failure in critically ill patients requiring continuous renal replacement therapy. *Nutr Clin Pract.* 2005;20:176-191.

^{10.} Klein CJ, Moser-Veillon PB, Schweitzer A, Douglass LW, Reynolds HN, Patterson KY, Veillon C. Magnesium, calcium, zinc, and nitrogen loss in trauma patients during continuous renal replacement therapy. *J Parenter Enteral Nutr.* 2002;26:77-92.

- 11. Van den Berghe G, Van Roosbroeck D, Vanhove P, Wouters P, De Pourcq L, Bouillon R. Bone turnover in prolonged critical illness: effect of vitamin D. *J Clin Endocrinol Metab.* 2003;88:4623-4632.
- 12. National Kidney Foundation K/DOQI. Clinical practice guidelines for nutrition in chronic renal failure. *Am J Kidney Dis.* 2000:35(suppl 2):S1-S104.
- 13. Block GA, Port FK. Re-evaluation of risks associated with hyperphosphatemia and hyperparathyroidism in dialysis patients: recommendations for a change in management. *Am J Kidney Dis.* 2000;35:1226-1237.
- National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. Am J Kidney Dis. 2003;42(suppl 3): S1-S201. Updates at www.kidney.org/professionals/kdoqi/guidelines_pedbone/guide7.htm. Accessed January 15, 2013.
- 15. Dickerson RN, Alexander KH, Minard G, Croce MA, Brown RO. Accuracy of methods to estimate ionized and corrected serum calcium concentrations in critically ill multiple trauma patients receiving specialized nutrition support. J Parenter Enteral Nutr. 2004;28:133-141.
- 16. Moe SA. Calcium and Phosphorus Balance in ESRD: Implications and Management. Cambridge, Mass: Genzyme Corp; 2001.
- 17. Weldy NJ. Body Fluids and Electrolytes. 7th ed. St. Louis, Mo: Mosby; 1996:104-106,108-112.
- 18. Mitch WE, Klahr S. Handbook of Nutrition and the Kidney. 3rd ed. Philadelphia, Pa: Lippincott-Raven; 1998:79-80,170-177.
- 19. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976; 16:31-41.
- 20. Morrison Nutrition Practice Guideline Chronic Kidney Disease (CKD). In: Inman-Felton A, Smith KG. *Morrison Nutrition Practice Guidelines*. Atlanta, Ga: Morrison Management Specialists Inc; 2012. Available at: www.morrisontoday.com/Documents/Nutrition/MHFS Nutrition

Bibliography

Centers for Medicare & Medicaid Services. End Stage Renal Disease (ESRD) Program Interpretive Guidance Version 1.1. October 2008 update. Baltimore, Md: Dept of Health and Human Services; 2008. Ref: S&C-09-01.

National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Am J Kidney Dis*. 2002;39:S1-S266.

NUTRITION CARE OUTCOMES and INTERVENTIONS IN CKD (Stage V) RENAL REPLACEMENT THERAPY BASED ON PATIENT ASSESSMENT PARAMETERS

I. Biochemical Parameters			
Parameter	Reference Range ^a	Goal for Dialysis ^b (1,2)	Nutrition Intervention(1-3)
Sodium	135-145 mEq/L	135-145 mEq/L	If high, assess sodium intake and hydration status. If low, assess fluid intake.
Potassium	3.5-5.5 mEq/L	3.5-5.5 mEq/L	If high, assess and limit potassium intake; modify potassium in dialysate and medications. If low, increase potassium intake and evaluate dialysis and medications.
Glucose	80-100 mg/dL	80-200 mg/dL, or <140 to 180 mg/dL in critically ill diabetic patients (3,4)	If high, avoid excess carbohydrate consumption. If low, assess total energy intake. Consider evidence-based guidelines on glycemic control in critical care patients and diabetic patients (3,4,5).
Total Calcium Ionized Calcium	8.5-10.5 mg/dL 4.5-5.6 mg/dL	8.4-9.5 mg/dL (If serum albumin is low, use ionized calcium or correct for low serum albumin)	If high, assess for the overuse of calcium supplements, vitamin D supplements, or other supplements that can increase calcium levels (6). Recommend avoiding high-calcium and calcium- fortified foods or the use of a calcium binder. If low, recommend that calcium binders be taken separately from meals, such as at bedtime (4,6).
Phosphorus	2.5-6.0 mg/dL	3.5-5.5 mg/dL	If high, limit total phosphorus intake and evaluate the use and timing of a phosphorus binder (6). If low, add one serving of high-phosphorus food per day or adjust binder.
Calcium- phosphorus product (7)	not available	<55 mg ² /dL ² (7)	Reduce serum phosphorus concentrations before increasing calcium levels. Utilize vitamin D or vitamin D analog per protocol. Refer to K/DOQI ^c guidelines as needed (6).
Blood (serum) urea nitrogen	4-22 mg/dL	<90-100 mg/dL	If high, assess adequacy of dialysis and medications (eg, steroids). If low, assess protein and energy intake and residual renal function.
Albumin	3.3-5.0 g/dL	>4.0 g/dL	If low, assess and increase protein intake. Consider impact of inflammatory metabolism. Low total cholesterol (<150 mg/dL) also indicates compromised nutritional status, especially in adults older than 60 years.
Plasma intact parathyroid hormone BioIntact	1-60 pg/mL Institution-specific	>100-300 pg/mL 75-150 pg/mL	If high, evaluate phosphorus and calcium. The goal is to establish limits in calcium range while maintaining phosphorus levels. Adjust dosage of calcitriol, paricalcitol, doxercalciferol, or Sensipar (calcimimetic agent).
parathyroid hormone	reference range		If low, evaluate dosage of calcitriol, doxercalciferol, or paricalcitol. It may need to be reduced. Refer to K/DOQI guidelines as needed (6).

I. Biochemical	Parameters		
Parameter	Reference Range ^a	Goal for Dialysis ^b (1,2)	Nutrition Intervention(1-3)
Hematocrit	Men: 38%-50% Women: 36%-45%	33%-36%	If high, check the dose of epoetin or other erythropoiesis-stimulating agents. If low, check ferritin stores, transferrin saturation, and iron
Hemoglobin	Men: 13-18 g/dL Women: 12-16 g/dL	11-12 g/dL	stores; increase epoetin or other erythropoiesis- stimulating agent. If no response, check folate and vitamin B_{12} levels (8).
Transferrin saturation	20%-50%	20%-50%	
Ferritin	12-800 ng/L	100-800 ng/L	If high, decrease iron supplementation; check for chronic inflammation.

II. Medications	
Drug	Approaches
Phosphate binders	Phosphate binders may decrease level of dietary phosphorus restriction.
Diuretics	If effective, diuretics may decrease level of sodium restriction. Diuretics may increase blood urea nitrogen levels and decrease potassium levels.

III. Other Parameters		
Parameter	Goal	Approaches
Interdialytic weight gain	1-2 lb/day	If high, limit fluid and sodium intake.
	2%-5% of body weight (9)	If low, modify fluid intake.
Dialysis adequacy assessed by urea kinetic modeling (1):	Kt/V \geq 1.2 or URR <65 in hemodialysis (1)	Evaluate adequacy of dialysis and protein metabolism.
Kt/V (UKK) ^a	Weekly Kt/V ≥2.0, creatinine clearance >60 L/week per 1.73 m ² in CAPD (1)	Refer to guidelines (1-3) for detailed discussion of urea kinetic modeling.
nPNA (1-3)	nPNA >1.2 (2,3)	
Glomerular filtration intake and creatinine clearance	Based on stage of CKD (1-5)	See discussion of glomerular filtration rate earlier in this section.
Urine Albumin-to-Creatinine Ratio (UACR)	< 30 mg/g	UACR estimates 24 -hour urine albumin excretion. Albuminuria is present when UACR is > 30 mg/g and is a marker for CKD. This parameter is used to diagnoise and monitor kidney disease. Change in albuminuria may reflect response to therapy and risk for progression. Refer to K/DOQI guidelines.
Hypertension	<130/80 mm Hg (Grade IV)* (10)	Restrict sodium.
Heart failure	Individualized based on symptoms	Restrict sodium and fluid as needed
		Restrict sodium and fluid as needed
Edema	Individualized based on symptoms	
Hypotension	>90/60	Assess sodium and fluid intake.
Urine output	Individualized based on	Adjust fluid intake based on urine output to
	symptoms	maintain stable dry weight.
Food intake	>80% estimated energy	Modify nutrition treatment plan and diet
	expenditure (1)	restrictions to increase food intake.

III. Other Parameters		
Parameter	Goal	Approaches
Altered taste sensation		Explore seasoning alternatives, as salt
		substitutes are contraindicated; problem may
		resolve as blood urea nitrogen normalizes.

^aBiochemical reference ranges vary based on the institution and the analytical methods used by the individual laboratory. ^bSuggested goals for dialysis are based on CKD stage 5(maintenance dialysis); goals may vary for stages 1-5 (1-3). ^cK/DOQI, kidney disease outcomes quality initiative

 d Kt/V, clearance of the dialyzer × time/volume; URR, urea reduction rate; CAPD, continuous ambulatory peritoneal dialysis; nPNA, normalized protein nitrogen appearance .

See Section 1G: "Medical Nutrition Therapy for Chronic Kidney Disease".

*The Academy of Nutrition and Dietetics has assigned grades, ranging from Grade I (good/strong) to Grade V (insufficient evidence), to evidence and conclusion statements. The grading system is described in Section III: Clinical Nutrition Management A Reference Guide, page III-1.

References

...

- 1. Wiggins KL, ed. *Guidelines for Nutrition Care of Renal Patients*. Chicago, Ill: American Dietetic Association; 2002.
- 2. Kopple JD, Massry SG, eds. *Nutrition Management of Renal Disease*. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2004.
- 3. Chronic kidney disease. In: *Nutrition Care Manual*. Academy of Nutrition and Dietetics; Updated annually. Available at: www.nutritioncaremanual.org. Accessed February 4, 2013.
- 4. Critical Illness Evidence-Based Nutrition Practice Guideline. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2012. Available at: http://www.andevidencelibrary.com. Accessed February 4, 2013.
- 5. Diabetes care in the hospital. In: Executive Summary: Standards of Medical Care in Diabetes 2013. *Diabetes Care*. 2013;36 (suppl 1): S45-S49.
- 6. National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. *Am J Kidney Dis.* 2003;42(suppl 3): S1-S201.
- 7. Moe SA. Calcium and Phosphorus Balance in ESRD: Implications and Management. Cambridge, Mass: Genzyme Corp; 2001.
- 8. NKF-DOQI Clinical Practice Guidelines for Treatment of Anemia of Chronic Renal Failure—Quick Reference Clinical Handbook. New York, NY: National Renal Foundation; 1998.
- 9. Sherman RA, Cody RP, Rogers ME, Solanchick JC. Interdialytic weight gain and nutritional parameters in chronic hemodialysis patients. *Am J Kidney Dis*. 1995;25:579-583.
- 10. *Hypertension Evidence-Based Nutrition Practice Guideline*. Academy of Nutrition and Dietetics Evidence Analysis Library. Academy of Nutrition and Dietetics; 2008. Available at: http://www.andevidencelibrary.com. Accessed January 5, 2009.

WILSON'S DISEASE

Discussion

Wilson's disease is an inherited disorder of copper metabolism that is characterized by the abnormal transport and storage of copper, resulting in hepatolenticular degeneration, neurologic damage, and damage to the kidney, brain, and cornea. Onset may occur from 5 to 40 years of age. Liver disease is always present when a patient with Wilson's disease presents with any symptoms. Wilson's disease is a highly treatable condition; with proper therapy, the disease progress can be halted, and often symptoms can be improved.

Copper promotes iron absorption for hemoglobin synthesis, formation of bone and myelin sheath. Most of copper in the copper-albumin complex is converted to ceruloplasmin in the hepatic tissues. In Wilson's disease, tissue deposition occurs instead of ceruloplasmin formation.

Approaches

Wilson's disease is treated with a copper chelating agent, such as penicillamine (Cuprimine, Depen) or trientine (Syprine), to keep the patient in a negative copper balance. The chelating agent bonds with copper to form stable complexes that are excreted in the urine. A patient initially treated with a chelating agent may receive maintenance therapy with zinc acetate (Galvin). This is particularly important for the patient who has experienced adverse reactions to chelating agents. Zinc acetate acts by blocking the absorption of copper in the intestinal tract, which results in both the depletion of accumulated copper and prevention of its reaccumulation.

Chelating agents should be taken orally before meals. A vitamin B_6 supplement is needed with this medication. Usually 25 mg/day of vitamin B_6 is adequate. Zinc may also be necessary. If the treatment with penicillamine alone does not achieve a negative copper balance, a low-copper diet (1 to 2 mg/day) may be an appropriate adjunct.

A normal diet provides 2 to 5 mg/day of copper. To achieve 1 to 2 mg/day of copper, limit intake of the following foods:

- Organ meats, such as liver, kidney, and brain
- Shellfish, such as oysters, crab, and lobster
- Dried legumes, except peas
- Whole wheat and bran breads and cereals
- Baked potato with skin
- Sweet potato
- Dried fruits, such as raisins, dates, and prunes
- Mushrooms
- Chocolate
- Nuts and seeds
- Wild game, such as duck and goose
- Mineral water

No alcohol is permitted due to its hepatotoxic action.

Zinc acetate (Galvin) is given in 50-mg doses three times a day. Take separate from food and beverages (other than water) by 1 hour. Avoid liver and limit shellfish (the only restrictions). Check the copper content of drinking water. If copper is greater than 1 ppm, deionized water should be used.

Do not use copper or bronze cooking utensils.

Make sure supplements are copper free.

Bibliography

Escott-Stump S. Nutrition and Diagnosis-Related Care. 5th ed. Baltimore, Md: Lippincott, Williams & Wilkins; 2002.

Skipper A, ed. *Dietitian's Handbook of Enteral and Parenteral Nutrition*. 2nd ed. Gaithersburg, Md: Aspen Publishers; 1998. Pennington J. *Bowe's and Church's Food Values of Portions Commonly Used*. 17th ed. City: Philadephia, Pa: Lippincott-Raven; 1998. Wilson's Disease Association. Available at: http://www.medhelp.org/wda/lit Accessed April 28, 1998.

Hesse JM, Matarese LE. Medical nutrition therapy for liver, bilary system, and exocrine pancreas disorders. In: Mahan KL, Escott-Stump S, eds. *Krause's Food, Nutrition, and Diet Therapy*. 10th ed. Philadelphia, Pa: WB Saunders; 2000: 703.

IV. APPENDIX

Caffeine And Theobromine Content Of Selected Foods And Beverages	IV-1
Metric/English Conversions Of Weight And Measures	IV-2
Milligram/Milliequivalent Conversions	IV-2
Salicylate Content Of Selected Foods	IV-3

CAFFEINE AND THEOBROMINE CONTENT OF SELECTED FOODS AND BEVERAGES

	Caffeine	Theobromine
	mg per serving	mg per serving
Carbonated Beverages (12 oz.)	01.05	
Cola, regular and diet, unless caffeine free	31-37	
Dr. Pepper, regular and deit	37	
Fanta Orange, Patio Orange	0	
Fresca	0	
Hires	0	
Mountain Dew	55	
Mr. Pibb, regular and sugar-free	27	
Mellow Yellow	35	
Sprite, 7-Up	0	
Kick	58	
Coffee ^a (5 oz.)		
Brewed, Percolator	103	
Ground	59	
Instant	57	
Decaffeinated	2	
Tea ^a (6 oz.)		
American black. 3 min. brew	36	2
From instant (1 tsp)	30	2
Green, 5 min. brew	26-36	
Herb	none detected	
Chocolate Foods		
Chocolate haking (1 oz)	57	346
Chocolate candy milk (1 oz)	6	50
Chocolate sweet dark (1 oz)	27	137
Chocolate milk (1 c)	5 (2-7)	*
Chocolate nudding $(1/2 c)$	7	88
Chocolate syrun (1 oz)	5	68
Carob nowder	0	00
Chocolate ice cream (4 oz)	2	41
C_{0}	2 5 (2-8)	* 1
COLUA DEVELAGE (U UZ.)	3 (2-0)	

^a The amount of caffeine depends upon the ratio of coffee or tea to water, method of preparation, blend of coffee or tea, and the length of exposure of the coffee or tea to hot water.

* Data not available

Source

Pennington J. Bowes & Church's: Food Values of Portions Commonly Used. 17th ed. Philadelphia, Pa: 1998.

METRIC/ENGLISH CONVERSIONS OF WEIGHT AND MEASURES

METRIC EQUIVALENTS

1 centimeter (cm)	= 10 millimeters (mm)
1 kilogram (kg)	= 1000 grams (g)
1 gram (g)	= 1000 milligrams (mg)
1 milligram (mg)	= 1000 micrograms (µg)
1 liter (L)	= 1000 milliliters (mL)
1 mL liquid	= 1 gram
	= 1 cubic centimeter (cc)

ENGLISH EQUIVALENTS

1 bushel =	4 pecks
	= 8 quarts
1 gallon =	4 quarts
	= 2 pecks
1 peck	= 2 quarts
1 quart (qt)	= 2 pints
	= 4 cups
	= 32 fluid ounces
1 pint (pt)	= 2 cups
	= 16 fluid ounces
1 cup (c)	= 16 tablespoons
	= 8 fluid ounces
1 tablespoon (tbsp)	= 3 teaspoons
	= 0.5 fluid ounce
	= 15 milliliters
1 teaspoon (tsp)	= 1/6 fluid ounce
	= 5 milliliters

ENGLISH TO METRIC

1 inch (in)	= 2.54 centimeters
1 pound (lb)	= 0.45 kilograms
	= 454 grams (actual amount is 453.6 g)
1 quart (qt)	= 0.946 liter
	= 946 milliliters
1 pint (pt)	= 480 milliliters
1 fluid ounce (fl oz)	= 30 milliliters (actual 28.35 mL)

METRIC TO ENGLISH

1 centimeter	= 0.39 in
1 kilogram	= 2.2 lb
1 gram	= 0.035 ounce (oz)
1 milligram	= 0.015 grain
1 liter	= 1.057 quarts

MILLIGRAM/MILLIEQUIVALENT CONVERSIONS

<u>ELEMENT</u>	ATOMIC WEIGHT	VALENCE
Calcium	40.08	2
Chlorine	35.45	1
Magnesium	24.31	2
Phosphorus	30.97	3
Potassium	40 (39.10)	1
Sodium	23 (22.98)	1
Sulfur	32.06	2

Conversions:

mg to mEq: Divide the milligrams by the atomic weight; multiply by the valence.

Example: <u>200 mg Sodium</u> × Valence (1) = 87 mEq Atomic Weight (23)

mEq to mg: Multiply milliequivalents by the atomic weight; divide by the valence.

Example: 90 mEq Sodium \times 23 \div 1 = 2070 mg

SALICYLATE CONTENT OF SELECTED FOODS

The restriction of foods containing salicylates may be used to treat urticaria (hives). Berries and dried fruits are high in salicylates, as are most herbs and spices. Aspirin use or penicillin and food molds may also be restricted. Hives may appear within minutes or up to two hours after eating, depending on where the food is absorbed in the digestive tract.

Note: The most common foods that cause hives are chocolate, fish, tomatoes, eggs, fresh berries and milk (1). Of these foods, only fish, eggs, tomatoes and fresh berries contain salicylates Their salicylate content is <.1 mg/100 mg, <.1 mg/100 mg, <.5 mg/100 mg, and 1.0-4.99 mg/100 mg respectively.

FOOD GROUP	.50 – .99 mg salicylate/ 100	1.0 – 4.99 mg salicylate/ 100	5.0 – 10.0 mg salicylate/ 100
	mg	mg	mg
Fruits	mg apple, canned or granny smith avocado cherries, sweet figs, dried grapes, red grape juice, dark grapefruit mandarin orange peach tangelo	mg apricot berries, all except fresh raspberries (which is higher) cantaloupe cherries, canned cranberry sauce currants, black and red dates, fresh and dried grapes, sultana orange pineapple	mg raisins prunes, canned raspberries, fresh
		plum, dark red	
Vegetables	alfalfa broad beans broccoli chili peppers, green/yellow cucumber without peel eggplant with peel mushrooms, canned okra spinach, fresh squash sweet potato, white tomato, canned watercress	chicory chili peppers, red endive peppers, sweet green radishes tomato paste tomato sauce zucchini	
Nuts	macadamia nuts pine nuts pistachios	almonds peanuts waterchestnuts	
Other	sherry, sweet wine	all spices and herbs if used in high amounts	

Reference

1. American Academy of Dermatology. Urticaria-Hives. http://tray.dermatology.uiowa.edu (4/28/98).

Bibliography

Pennington J. Bowes and Church's Food Values of Portions Commonly Used. 17th ed. Philadelphia,Pa: Lippinott; 1998. Escott-Stump S. Nutrition and Diagnosis-Related Care. 4th ed. Baltimore: Williams & Wilkins; 1997:75-76.

INDEX

Α

Acute respiratory failure (ARF), III-17 Acidemia, organic, III-76 Acidophilus milk, H-14 Adolescents, diabetes mellitus, C-12 energy requirements, A-2, A-3 disorders of lipid metabolism, C-40 nutrition management, E-12 obesity and weight management, III-82 protein requirements, A-5 pregnancy, A-19 vegetarian diets, A-36 Albumin, II-18 Alcohol. hypertension, III-65 pregnancy, A-16 Allergy, H-16 Americans older, A-22 Amino acids, branched chain amino acids, B-39, B-52 formulas, B-39 parenteral, B-48-50, B-52-53 Amputees, IBW, II-10 Amylase, III-90 Anemia, II-18 Antacids, G-13, II-27, III-104 Anthropometric measurements, II-3, II-9 Anticoagulants, III-3 Antioxidants, C-44, III-11-12, III-18 Arm span determination, II-5 Aromatic amino acids, G-1 Ascites, F-1, G-2 Ascorbic acid, B-3, B-36 iron absorption and, III-78 Aspartame, III-32 AIDS, See HIV Infection

В

Bariatric Surgery B-17 Bilirubin, serum bilirubin, B-57, III-53, III-91 Body mass index, A-15, B-36, B-51, C-11, C-41, G-10, II-1 Bread & cereal group, A-12 Breast feeding, C-19, E-1 Breath hydrogen analysis test, H-12 BUN, A-34, G-17 Burns, II-17, III-6

С

Caffeine, A-16, IV-1 food and medication interactions, II-36 gastroesophageal reflux disease (GERD), III-41 heart failure, III-45 tyramine-restricted diets, H-10 Calcium, F-14, II-24 Calcium carbonate, F-14, II-27 Caloric requirements, (see Energy) Cancer, III-10 Cardiac cachexia. III-44 Celiac disease. H-1 Chemotherapy, III-10 Children, suggested food intake, E-9 Chloride, B-43, B-56, II-24 Cholesterol, C-6 Chromium, diabetes, C-8 major nutrients, functions and sources. II-25 parenteral nutrition for adults, B-55 Chronic obstructive pulmonary disease (COPD), III-16-18 Cirrhosis, F-1, G-1 Constipation, D-4 cancer, III-13 diabetes mellitus, gastrointestinal complications, III-27 food and medication interactions, II-31 Copper, major nutrients, functions and sources, II-25 wilson's disease, III-120 Coronary heart disease, diabetes mellitus, medical nutrition therapy for, C-6 disorders of lipid metabolism, medical nutrition therapy for, C-40 hypertension, III-66 hypertriglyceridemia, III-71 Corticosteroid therapy. III-20 Coumadin, III-45 Creatinine, end-stage renal disease, III-111 enteral nutrition support for adults, B-44 parenteral nutrition, support for adults, B-57

D

Cyclic feeding, B-43

Daily Food Guide, A-11 Dehydration, A-19, A-32 Dermatitis herpetiformis, H-1 Dextrose, parenteral nutrition, calculating central, III-99 parenteral nutrition, metabolic complications of CPN, III-94 parenteral nutrition support for adults. B-51 Diabetes mellitus. C-1 alternative sweeteners. III-32 calculated diet samples, C-34 complications, III-27 diagnosis, II-21 Copyright © 2013 Compass Group, Inc. All rights reserved.

Index

enteral nutrition, support for adults, B-40-41 exchange lists, C-21 exercise, C-5, III-23 fiber and. C-3-4 gestational pregnancy and, C-15 oral glucose-lowering medications and insulin, III-29 sample exchange patterns, C-34 vegetarian diets, A-36 Decubitus ulcer (see Pressure Ulcers) Diarrhea, bariatric surgery for obesity, B-19 cancer, III-13 diabetes mellitus, III-25, III-27 syndrome diet, B-15 enteral nutrition, management of complications, III-38 enteral nutrition, support for adults, B-38, 41, 45 fiber-restricted diets, D-1 fluid intake and hydration, nutrition management of, A-32 food and medication interactions, II-27 gluten-free diet, H-1 HIV infection and AIDS, III-46-61 high-fiber diet, D-5 lactose-controlled diet, H-12 pancreatitis, III-89 parenteral nutrition, metabolic complications, III-94 Dietary Guidelines for Americans, A-11 Diets. calcium enhanced, F-14 calcium restricted, F-15 calorie-controlled for weight management, C-36 clear liquid, B-1 full liquid, B-3 full liquid, blenderized, B-4 diabetic, C-1 dumping syndrome, B-15 dysphagia feeding plan, B-6 fat-controlled, C-55 fiber-restricted, D-1 gluten-free H-1 high-fiber, D-4 high-protein, high-calorie, A-13 kosher guidelines, A-40 lactose-controlled. H-12 mechanical soft, A-28 potassium, F-10 pureed, A-30 protein-controlled, G-1 regular, adult, A-11 regular, adolescents, E-12 regular, full-term infant, E-1 regular, school-age child, E-9 regular, toddler and preschool child, E-7 regular, pregnancy and lactation, A-14 renal disease, G-7 residue-restricted (see Fiber Restricted Diets) sodium-controlled, F-1 sugar in moderation, C-35

temporomandibular joint surgery and, A-28 therapeutic lifestyle change, C-40 tyramine-restricted, H-10 vegetarian, A-36 Disorders of Lipid Metabolism, C-40 high-fiber, D-4 nephrotic syndrome, III-80 Diverticulosis, D-1, D-5 Drugs, see Medications Dumping syndrome, B-15, B-19, III-12, III-73 Dysphagia, B-6, III-36

Ε

Edema, A-34 chronic kidney disease, G-9 heart failure, III-43 nephrotic syndrome, III-79 parenteral nutrition, B-52 sodium-controlled diet, F-1 Electrolytes, parenteral, B-50, C-16 Energy requirements, II-12, B-51 English/metric conversions, IV-2 Enteral nutrition, support for adults, B-35 management of complications, III-39 Esophageal varices, G-2 Exchange lists, C-1 ADA, C-21 protein-based, G-5

F

Fat-controlled diet, C-55 gastroesophageal reflux disease (GERD), III-41 medium-chain triglycerides (MCT), C-57 nephrotic syndrome, III-80 Fats, fat-soluble vitamins, G-1, G-13, II-23 fecal fat, III-91 Fiber. D-1-11 content of foods (chart), D-11 diabetes and, C-3 high-fiber diet, D-4 phytobezoar form, D-7 fiber-restricted diets, D-1 Fish oil, III-18, C-43 Fistulas, B-35, B-41, B-51 Fluoride, II-25 Fluid. calculation of needs, A-32-33 clear liquid diet, B-1 full liquid blenderized diet, B-3 pregnancy and lactation, nutrition management during, A-20 nutrition and the older adult, A-22 simplified renal diet. G-28 Fluid deficit, treatment/prevention, A-32

Fluid restriction, indications for, A-35 Folate, A-5, A-7, A-8, A-14, A-16, A-36, II-24 bariatric surgery for obesity, B-19 deficiencies, physical signs, II-24 food and medication interactions, II-27 gluten-free diet, H-2 ketogenic diet, E-14 protein-controlled diet, G-2 Food allergy, H-16 Food and medication interactions, II-27 Food Sensitivity, H-16 Frame size, determination, II-9 Fructose. diabetes mellitus, III-32 inborn errors in metabolism, III-76 Fruit group, A-12

G

Galactosemia, Inborn errors of metabolism, III-75 Gallbladder disease and diet, herb and medication interactions, II-38 obesity, III-82 Gastric banding, B-17 Gastric reduction surgery, B-17 Gastroparesis, III-27 high-fiber diet, D-7 Gastroesophageal reflux, III-41 gastrointestinal soft diet, D-14 Genetic disorders, III-75 Glomerular filtration rate, G-7, G-22, III-113 Glucose, II-21, III-21 calorie-controlled diet for weight management, C-36 corticosteroid therapy, III-20 diabetes mellitus, II-21, III-21 enteral nutrition, support for adults, B-41 gestational diabetes mellitus, C-15 herb and medication interactions, II-34 high-fiber diet, D-4 ketogenic diet, E-14 parenteral nutrition, support for adults, B-50 determination of absorption during CAPD, G-16 Gluten-free diet, H-1 Glycated hemoglobin, III-21 Glycogen storage diseases, III-75 Grain group, A-12

Η

Heart failure, F-1, III-43 fluid intake and hydration, nutrition management of, A-32 pneumonia, III-105 sodium-controlled diet, F-1 HDL cholesterol disorders of lipid metabolism, medical nutrition therapy for, C-40 hypertriglyceridemia, III-71 Height determination, II-5 Hemodialysis, G-7

Hepatic coma, G-3 Hepatic encephalopathy, G-1 medium-chain triglycerides (MCT), C-57 parenteral nutrition, support for adults, B-50 Hematocrit, II-19, III-49, III-64 classifications, anemias, II-20 laboratory indices of nutritional status, II-19 pregnancy and lactation, nutrition management during, A-18 pancreatitis, III-91 Hemoglobin, II-19, III-78 classifications, anemias, II-20 iron deficiency anemia, III-78 laboratory indices of nutritional status, II-19 pregnancy and lactation, nutrition management during, A-18 Hemoglobin A₁C, III-29 diabetes mellitus, C-1 HIV, III-47 high-protein, high-calorie diet, A-13 Hypercholesterolemia (see Disorders of Lipid Metabolism) Hyperphosphatemia, parenteral nutrition, metabolic complications, III-97 phosphorus intake, F-12 Hypertension, III-63 calcium and. III-66 calorie-controlled diet for weight management, C-36 corticosteroid therapy, III-20 diet F-1, III-67 heart failure, III-42 herb and medication interactions, II-34 lactose-controlled diet, H-12 nephrotic syndrome, III-790 pregnancy and lactation, nutrition management during, A-14 obesity and weight management, III-81 renal disease, G-7 sodium-controlled diet, F-1 vegetarian diets, A-36 Hypoglycemia, III-72 cancer, III-10 diabetes mellitus, C-8, III-23, III-72 dumping syndrome diet, B-15 inborn errors of metabolism, III-74 ketogenic diet, E-19 parenteral nutrition, metabolic complications, III-94 protein-controlled diet, hepatic

encephalopathy, G-1

I

Infant, full-term breast feeding, E-1 fiber and, D-4 comparison of various formulas, E-4-6 introduction of solid foods, E-3 nutrition guidelines, E-3 phenylketonuria (PKU), III-71 Immunodeficiency diseases, diet and, III-46 Inborn errors of metabolism, III-71 Insulin therapy, III-29 Irritable bowel syndrome, D-1, D-5 Iron. absorption, III-77 ascorbic acid and, III-77 deficiency anemia, II-20, III-77 full-liquid blenderized diet, B-4 food content, III-77 pregnancy, required in, A-15

К

Ketogenic diet, E-14 Knee height determination, II-5 Kosher dietary laws, A-40

vegetarian diets and, A-38

L

Laboratory indices, II-18 Lact-Aid®, H-13 Lactose deficiency, H-12 Lactose, D-1, H-12 controlled diet, H-12 intolerance test, H-12 yogurt, H-13-14 LDL Cholesterol, C-6, C-40 Lipase, serum, III-91 Liquid diets, B-1-3 Lithium carbonate, F-2 Lipids, parenteral, B-53 Liver disease, G-1 Lofenalac®, III-75

Μ

Magnesium, II-24 Malabsorption, fat, C-55 bariatric surgery for obesity, B-18 gluten-free diet, H-1 pancreatitis, III-89 protein-controlled diet, G-2 Manganese, II-25 parenteral nutrition, support for adults, B-55 Maple syrup urine disease (MSUD), III-74 MCH, II-20 MCHC, II-19, II-20 Meat group, A-12 Mechanical soft diet, A-28 dysphagia, nutritional management of, B-6 Medications, breast feeding and, E-1 enteral feedings and, B-46 food and medication interactions, II-27 Medium-chain triglycerides, C-57 dumping syndrome diet, B-15 enteral nutrition, support for adults, B-38. B-42 Metabolic syndrome, C-41 Metric/English conversions, IV-2 Milk, allergy, H-16 full-term infant, nutrition management of. E-1 lactose-controlled diet, H-12 nutrient comparison of, E-2 Milk group, A-12 Milligram/milliequivalent conversions, IV-Minerals, calcium (see Calcium) parenteral nutrition, and B-55 zinc, II-25 Modular components B-38 Monoamine oxidase inhibitors. tyramine-restricted diet, H-10 Monounsaturated fats, C-43

Ν

NHANES, II-8 Nephrotic syndrome, III-79 Nickel, II-25 Niacin, II-23 food and medication interactions, II-32 nephrotic syndrome, III-79 parenteral nutrition, support for adults, B-55 physical signs of deficiencies, II-26 protein-controlled diet, G-2 NutraSweet®, III-33 Nutrition Assessment, body mass index, calculation, II-6 calorie-controlled diet, C-36 deficiency, signs of, II-26 frame determination, II-9 height determination, II-5 laboratory indices, II-18 obesity and weight management, III-81 obesity and weight management, children and adolescents, III-82-83 weight evaluation, II-1, II-7 Nitrogen balance,

burns, III-7 corticosteroid therapy, III-20 estimation of protein requirements, II-17 Nutritional adequacy, statement, A-1

0

Oat bran. D-10 Obesity, III-81 basal energy expenditure, II-14 body mass index, II-6, III-82 calorie controlled diet, C-6 diabetes, type 2 and, C-11 pharmacotherapy, III-86 Older adults, A-22 Omega-3 fatty acids, C-43 chronic obstructive pulmonary disease, III-18 disorders of lipid metabolism, medical nutrition therapy for, C-43 enteral nutrition, support for adults, B-38 Osteoporosis, F-14 bariatric surgery for obesity, B-21 calcium intake, nutrition management of, F-14 chronic obstructive pulmonary disease, III-16 corticosteroid therapy, III-20 gluten-free diet, H-1 lactose-controlled diet, H-12 stature determination, II-5

Ρ

Pancreatic enzymes, III-93 Pancreatitis, III-89 enteral nutrition, support for adults, B-35, B-38 parenteral nutrition, metabolic complication of TPN, III-94-98 parenteral nutrition, support for adults, B-50 Pantothenic acid, II-24 parenteral nutrition, support for adults, B-50 protein-controlled diet, G-2 Parenteral nutrition, B-50 burns, III-8 calculating central, III-99 complications, III-94 contraindications, B-51 cyclic, B-57 indications, B-50 monitoring, B-57 pediatrics, neonates, B-53 protein-controlled diet, G-1-4 weaning from, B-57 Pediatrics, allergy, E-3 fluid intake and hydration, nutrition management of, A-33 weight management, III-82-83 Peptic ulcer disease, III-103

Pernicious anemia, II-19 Peptide base formulas, B-38 Peripheral nutrition, B-50 Peritoneal dialysis, G-8, G-10-11, G-12 Pharmocotherapy, for obesity, III-86 Phosphorus, II-24, III-111 content of food, F-13 nutrition management of, F-12 Phenylketonuria (PKU), III-74 diabetes mellitus, III-33 full-term infant, nutrition management of. E-2 inborn errors of metabolism. III-74 manufacturers of formulas, III-74 Phytobezoar form, D-7 Pneumonia, III-104 Polyunsaturated fats, C-43 Potassium, F-10, II-24 chronic obstructive pulmonary disease, III-18 content in food, F-11 food and medication interactions, II-27 heart failure, III-44 hypertension, III-66 major nutrients, functions and sources, II-24 metric/english conversions, IV-2 nutrition management of intake, F-10 parenteral nutrition, support for adults, B-50 renal disease, III-112 simplified renal diet, G-28 sodium-controlled diet, F-1 Prealbumin, II-18 body weight evaluation, II-1 enteral nutrition, support for adults, B-35 parenteral nutrition, support for adults, B-50 Pre-eclampsia, A-19 Pregnancy, A-14 alcohol and, A-16 caffeine and. A-16 diabetes and, C-12, C-15 diet in, A-14 energy and, A-15 lactation and, A-18 nausea and vomiting of, A-18 pre-eclampsia, A-19 weight gain and, A-15 Pressor amines, H-10 Pressure ulcers, III-106 Protein, adolescent, nutrition management of the, E-12 controlled diet, G-1

exchange lists, G-5

Index

dialysis and, G-10, G-15 diabetes mellitus, C-1 dumping syndrome diet, B-15 full liquid diet, B-3 high-protein, high-calorie diet, A-13 ketogenic diet, E-14 older adult, nutrition and the, A-22 pediatrics, full-term infant, nutrition management of, E-1 pregnancy and lactation, nutrition management of, A-20 renal disease, medical nutrition therapy of, G-7 school-age child, nutrition management of, E-9 toddler and preschool child, nutrition management of the , E-7 vegetarian diet and, A-37 Pulmonary disease, III-16 Pureed diet, A-30 Prothrombin time, II-19, III-4 parenteral nutrition, support for adults, B-50 protein-controlled diet, G-1 Pyridoxine, II-23

R

Radiation therapy, III-11 RDA (recommended dietary allowances), A-5 estimation of protein requirements, A-4, II-17 Renal disease, G-7, III-110 calcium and, G-13, III-111, III-113 diet in, G-9 energy and, G-10, G-16 enteral nutrition, support for adults, B-35 fluid balance in. G-11, G-15 glomerular filtration rate, III-114 glucose absorption during CAPD, G-16 hemodialysis, G-8, G-11, G-12 magnesium and, G-13 meal patterns, G-27 nephrotic syndrome, III-79 nutrient requirements, G-15 parenteral nutrition, B-53 peritoneal dialysis, G-8, G-10-12, G-15 phosphorus intake, nutrition management of, F-12, III-111 potassium intake, nutrition management of, F-10 protein and, G-10, G-15 renal choice system, dietary management with the, G-17 simplified diet. G-28 sodium-controlled diet F-1, G-11 trace minerals and, G-14 transplantation, G-9 vitamins and, G-14 Residue restricted diets, D-1 Red blood cells, II-19 Riboflavin, II-23 Roux-en-Y, B-18

S

Saccharin, III-33 Salicylate, IV-3 Salt substitutes. F-2 Saturated fats, C-42-43 Semisolid foods, B-4 Selenium, II-25 Sleeve gastrectomy, B-18 Sodium, F-1 diabetes mellitus and, C-7 dialysis and, G-11 food guides. 4000 mg sodium diet, F-4 3000 mg sodium diet, F-5 2000 mg sodium, low-fat diet, C-54 2000 mg sodium diet, F-6 1000 mg sodium diet, F-8 hypertension, F-1, III-65 levels of restriction, F-2 lithium carbonate and, F-2 no added salt diet, F-4 parenteral nutrition, support for adults, B-56 pregnancy and, A-16 protein-controlled diet, G-1 renal disease, medical nutrition therapy of, G-9 Splenda, III-33 Sorbitol, H-17, III-33 Steroid therapy, III-20 Swallowing disorders (see Dysphagia)

Т

Temporomandibular joint surgery, diet for, A-28 Theobromine food contents, IV-1 Thiamin, II-23 Tocopherol, II-23 Toddler and preschool child, nutrition management of, E-8 Trace elements. II-25 burns, III-8 parenteral nutrition and, B-55 Trans fat, C-43-44 Transitional feeding program, B-57 Transferrin, II-18 Triglycerides, diabetes mellitus, C-7 disorders of lipid metabolism, C-49-50 hypertriglyceridemia, III-70 Tube feeding, B-35 administration guidelines, B-43 burns, III-8 complications, III-38 energy density, B-42 enteral nutrition, support for adults,

Yogurt and lactose, H-13-14

Ζ

Zinc. II-25

B-35 formula selection, B-37 medications, B-46 monitoring, B-45 nutrient content, B-42 osmolality, B-42 protein-controlled diet, G-3 residuals, B-44 water/fluid requirements, B-41 vitamin K and, B-43 Tyramine-restricted diet, H-10 Tyrosine, III-74

U

Urea cycle disorders, III-75 Urea nitrogen, burns, III-7 enteral nutrition, support for adults, B-46 Urticaria, chronic, IV-3

V

Vegetable group, A-12 Vegetarian diets, A-36 classification of, A-36 vitamin supplements and, A-37 Vertical-banded gastroplasty, B-18 Vitamin(s), ascorbic acid, II-23 folate, II-24 niacin, II-23 parenteral nutrition and, B-55 supplements, A-10 vitamin A, II-23 vitamin B₁, II-23 vitamin B₂, II-23 vitamin B₆, (pyridoxine), II-23 vitamin B₁₂, A-37, II-24 vitamin D, A-38, II-23 vitamin E (tocopherol), II-23 vitamin K, II-23, III-4 anticoagulant, therapy, III-3

W

Water requirements, A-32 Water soluble vitamins, II-23-24 Weight, II-1, II-7-8, III-81 amputees, estimation of ideal body weight, II-10 body weight evaluation, II-1 reduction (see Obesity) surgery, B-17 weight for height calculation, 5' rule, II-7 Wheat bran, D-8 Wilson's disease, III-119 Wired jaw, B-4

Υ