CHAPTER 15

Nutrition and Metabolic Stress

> *One of the first body functions affected by impaired nutritional statue is the immune system.*

Role in Wellness

In its never-ending quest to maintain homeostasis, the human body responds to stress, physiologic or psychologic, with a chain reaction that involves the central nervous system and hormones that affect the entire body. Magnitude and duration of the stress determine just how the body will react. It is important for nurses to understand metabolic changes that take place in reaction to stress, both in uncomplicated stress that is present when patients are at nutritional risk, and in more multifarious variations that result from severe stress brought about by trauma or disease.

Immune System

One of the first body functions affected by impaired nutritional status is the immune system. When metabolic stress develops, hormonal and metabolic changes subdue the immune system's ability to protect the body. This activity is further depressed if impaired nutritional status accompanies the metabolic stress. A deadly cycle often develops: impaired immunity leads to increased risk of disease, disease impairs nutritional status, and compromised nutritional status further impairs immunity. Recovery requires that this cycle be broken.

Role of Nutrition

For the immune system to function optimally, adequate nutrients must be available. A well-nourished body will not be ravaged by infections the way a poorly nourished body. (See the *Cultural Considerations* box, The Process of Balance, for a multicultural perspective on balanced eating for good health.) To prove this point, think of the leading causes of death in industrialized countries such as the United States. The majority are chronic diseases associated with lifestyle. In developing countries, however, infections lead to high morbidity and mortality rates, especially in children, largely because of the high rate of protein-energy malnutrition (PEM). The majority of people in the United States who have serious problems with malnutrition and infections are (1) those with severe medical problems, (2) those who suffer from major metabolic stress, (3) those who suffer from a diseased state that causes metabolic stress and/or decreased nutrient intake and/or nutrient malabsorption, and (4) those who have poor nutritional intakes as a result of socioeconomic conditions (e.g., poverty, homelessness).

Compromised nutritional status creates a vulnerable immune system by making it difficult to mount both a stress response and an immune response when confronted with a metabolic stress. A number of nutrients are known to affect immune system functioning. It is difficult to determine which specific nutrient factor results in symptoms when a patient is malnourished because of overlapping nutrient deficiencies combined with illness and accompanied by weakness, anorexia, and infection.[1]

Immune system components affected by malnutrition include mucous membrane, skin, gastrointestinal tract, T-lymphocytes, macrophages, granulocytes, and antibodies.

CULTURAL CONSIDERATIONS

The Process of Balance

What is a balanced way of eating for good health? To most Americans, the response is to eat foods from each of the food groups, with particular emphasis on fruits and vegetables. Among other cultures, foods consumed to achieve balance and good health do not follow the American food categories. The Chinese system of *yin-yang* sorts foods into *yin* (bean curd or tofu, bean sprouts, bland and boiled foods, broccoli, carrots, duck, milk, potatoes, spinach, and water) and *yang* (bamboo, beef, broiled meat, chicken, eggs, fried foods, garlic, gingerroot, green peppers, and tomatoes). Foods should be selected from each group to achieve balance. Which foods belong in each group may vary by region, but some foods such as rice and noodles are considered neutral. The overall goal is to maintain the harmony of the body with adjustments for climate variations and physiologic factors.

Balance is also the focus of the *hot-cold classification* of foods practiced in the Middle East, Latin American, India, and the Philippines. This concept is derived from the Greek humoral medicine based on the four natural world characteristics of air-cold, fire-hot, water-moist, and earth-dry related to the body humors of hot and moist (blood), cold and moist (phlegm), hot and dry (yellow/green bile), and cold and dry (black bile). Although this concept is related to the development of disease and their remedies, it also applies to foods. The hot and cold aspects of specific foods are emphasized. This does not relate to the actual temperature of the foods but to their innate characteristics. To achieve balance, eating cold foods offsets hot foods. The list of foods in each category varies among subgroups within each culture. Often, younger generations follow this concept but without knowing that it is based on the hot-cold theory of balance.

Application to nursing: Each of the cultures, subscribing to the yin-yang concept and the hot-cold theory, has sizable populations in the United States. When treating Americans of Chinese, Indian, Latino, Middle Eastern, and Filipino descent, these concepts of food selection to achieve health and harmony may affect client food choices. Although healthy selections are often selected, subtle effects may occur. For example, within the hot-cold theory, pregnancy may be considered "hot" as are vitamins. Consequently, vitamins are not taken during pregnancy because to do so would not restore balance. If a client seems unwilling to follow dietary and supplement recommendations, discussion of these classifications and ways to remedy the situation can be created.

Data from Kittler PG, Sucher KP: *Food and culture in America: A nutrition handbook*, ed 4, Belmont, Calif, 2004, Brooks/Cole.

The effects on the mucous membrane are that the microvilli become flat, which reduces nutrient absorption and decreases antibody secretions. Integrity of the skin may be compromised as it loses density and wound healing is slowed. Injury to the gastrointestinal tract because of malnutrition may increase risk of infection-causing bacteria spreading from inside the tract to outside the intestinal system. T-lymphocytes are affected as the distribution of T cells is depressed. The effect on macrophages and granulocytes requires that more time be needed for phagocytosis kill time and lymphocyte activation to occur. Antibodies may be less available because of damage to the antibody response. Table 15-1 outlines how specific nutrient deficiencies affect immune system functions; note that fat and water-soluble vitamins, fatty acids, minerals, and protein are important for adequate functioning of most immune system components.[1]

The Stress Response

The body's response to metabolic stress depends on the magnitude and duration of the stress. Stress sets up a chain reaction that involves hormones and the central nervous system that affects the entire body. Whether stress is uncomplicated (altered food intake or activity level) or multifarious (trauma or disease), metabolic changes take place throughout the body.

According to Gould,[2] the body's constant response to minor changes brought about by needs or environment was first noted in 1946 by Hans Selye when he described the "fight or flight" response, or general adaptation syndrome (GAS). The body constantly responds to minor changes to maintain homeostasis. Research following Selye's work has identified that the stress response involves an integrated series of actions that include the hypothalamus and hypophysis, sympathetic nervous system, adrenal medulla, and adrenal cortex.[2] Significant effects of this response to stress are outlined in Table 15-2. These responses to stress produce multiple changes in metabolic processes throughout the body. The effect of different levels of stress on metabolic rate is illustrated in Figure 15-1.

TABLE 15-1 Role of Nutrients and Nutritional Status on Immune System Components

Immune System Component	Effects of Malnutrition	Vital Nutrients
Mucus	Decreased antibody secretions	Vitamin B_{12}, biotin, vitamins B_6 and C
Gastrointestinal tract	Flat microvilli, increased risk of bacterial spread to outside GI tract	Arginine, omega-3 fatty acids
Skin	Integrity compromised, density reduced, wound healing slowed	Protein, vitamins A and C, niacin, zinc, copper, linoleic acid, vitamin B_{12}
T-lymphocytes	Depressed T-cell distribution	Protein, arginine, omega-3 fatty acids, vitamins A, B_{12}, B_6, folic acid, thiamine, riboflavin, niacin, pantothenic acid, zinc, iron
Macrophages and granulocytes	Longer time for phagocytosis kill time and lymphocyte activation	Protein, vitamins A, C, B_{12}, B_6, folic acid, thiamine, riboflavin, niacin, zinc, iron
Antibodies	Reduced antibody response	Protein, vitamins A, C, B_{12}, B_6, folic acid, thiamine, biotin, riboflavin, niacin

TABLE 15-2 Effects of the Stress Response*

Target Organ	Hormonal Response	Physiologic Response	Signs/Symptoms
Sympathetic nervous system and adrenal medulla	Norepinephrine	Vasoconstriction	Pallor, decreased glomerular filtration rate, nausea, elevated blood pressure
Adrenal medulla	Epinephrine	Vasoconstriction Increased heart rate Vasodilation Central nervous system (CNS) stimulation Bronchodilation Glycogenolysis, lipolysis, gluconeogenesis	See above Elevated blood pressure Increased skeletal muscle function More alert, increased muscle tone Increased O_2 Increased blood glucose
Adrenal pituitary and cortex	Cortisol (glucocorticoids)	CNS stimulation Protein catabolism, gluconeogenesis	Increased blood glucose, increased serum amino acids, delayed wound healing
		Stabilize cardiovascular system Gastric secretion Inflammatory response decreased Allergic response decreased Immune response decreased	Enhance catecholamine action Ulcers Decreased white blood cells (WBCs)
	Aldosterone (mineralocorticoid)		Retain sodium and water, increased blood volume, increased blood pressure
Posterior pituitary	Antidiuretic hormone	Water reabsorbed, increased blood volume, increased blood pressure	
Other feedback mechanisms	Aldosterone and antidiuretic hormone	See above	See above

*Possible complications include hypertension, tension headaches, insomnia, diabetes mellitus, infection, heart failure, peptic ulcer, and fatigue.
Data from Gould BE: *Pathophysiology for the health-related professions,* ed 3, Philadelphia, 2006, Saunders.

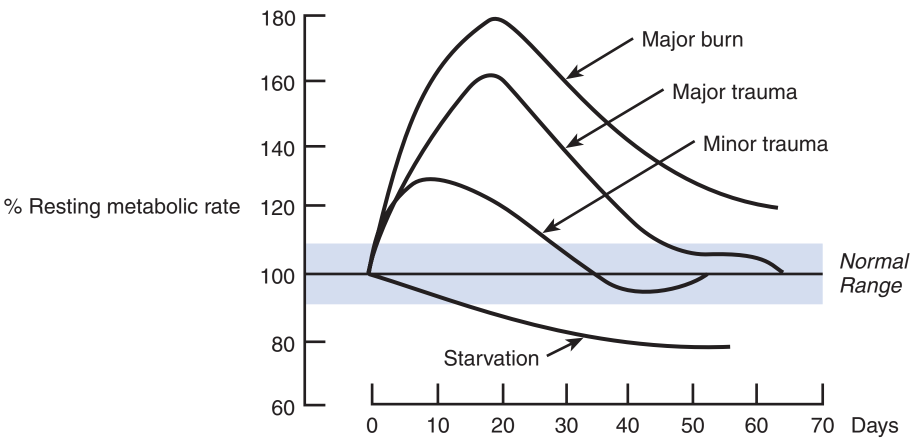

Figure 15-1 Percent resting metabolic rate. *(From Kinney JM et al:* Nutrition and metabolism in patient care, *Philadelphia, 1988, Saunders.)*

Starvation

If someone must involuntarily go without food, that can be defined as *starvation*. If we withhold food from ourselves, such as when we try to lose weight, that act can be defined as *dieting* or *fasting*. Whatever the cause of inadequate food intake and nourishment, results are the same. After a brief period of going without food (fasting) or an interval of nutrient intake below metabolic needs, the body is able to extract stored carbohydrate, fat, and protein (from muscles and organs) to meet energy demands.

Liver glycogen is used to maintain normal blood glucose levels to provide energy for cells. Although readily available, this source of energy is limited, and glycogen stores are usually depleted after 8 to 12 hours of fasting. Unlike glycogen stores, lipid (triglyceride) stores may be substantial, and the body also begins to mobilize this energy source. As the amount of liver glycogen decreases, mobilization of free fatty acids from adipose tissue increases to provide needed energy by the nervous system. After approximately 24 hours without energy intake (especially carbohydrates), the prime source of glucose is from gluconeogenesis.[3]

Some body cells, brain cells in particular, use mainly glucose for energy. During early starvation (about 2 to 3 days of starvation), the brain uses glucose produced from muscle protein. As muscle protein is broken down for energy, the level of **branched-chain amino acids (BCAA)** in circulation increases although they are primarily metabolized directly inside muscle.[3] The body does not store any amino acids as it does glucose and triglycerides; therefore, the only sources of amino acids are lean body mass (muscle tissue), vital organs including heart muscle, or other protein-based body constituents such as enzymes, hormones, immune system components, or blood proteins. By the second or third day of starvation, approximately 75 g of muscle protein can be catabolized daily, a level inadequate to supply full energy needs of the brain.[3] At this point, other sources of energy become more available. Fatty acids are hydrolyzed from the glycerol backbone and both free fatty acids and glycerol are released into the bloodstream. Free fatty acids are used as indicated earlier and glycerol can be used by the liver to generate glucose via the process of gluconeogenesis.

As starvation is prolonged, the body preserves proteins by mobilizing more and more fat for energy (Figure 15-2). Ketone body production from fatty acids is accelerated, and the body's requirement for glucose decreases. Although some glucose is still vital for brain cells and red blood corpuscles, these and other body tissues obtain the major proportion of their energy from ketone bodies. Muscle protein is still being catabolized but at a much lower rate, which prolongs survival. During this period of starvation, approximately 60% of the body's energy is provided by metabolism of fat to carbon dioxide, 10% from metabolism of free fatty acids to ketone bodies, and 25% from metabolism of ketone bodies.[4]

An additional defense mechanism of the body to conserve energy is to slow its metabolic rate, thereby decreasing energy needs. As a result of declining metabolic rate, body temperature drops, activity level decreases, and sleep periods increase—all to allow the body to preserve energy sources. If starvation continues, intercostal muscles necessary for respiration are lost, which may lead to pneumonia and respiratory failure.[4] Starvation will continue until adipose stores are exhausted.

Severe Stress

Whether stress is accidental (e.g., from broken bones or burns) or necessary (e.g., from surgery), the body reacts to these stresses much as it does to the stress of starvation—with a *major* difference. During starvation, the body's metabolic rate slows, becoming hypometabolic. During severe stress, the body's metabolic rate rises profoundly, thus becoming hypermetabolic.

The body's response to stress can be summarized by two phases: ebb phase and flow phase (Figure 15-3). The *ebb phase*, or *early phase* (Table 15-3), begins immediately after the injury and is identified by decreased oxygen consumption, hypothermia (lowered body temperature), and lethargy. The major medical concern during this time is to maintain cardiovascular effectiveness and tissue perfusion.

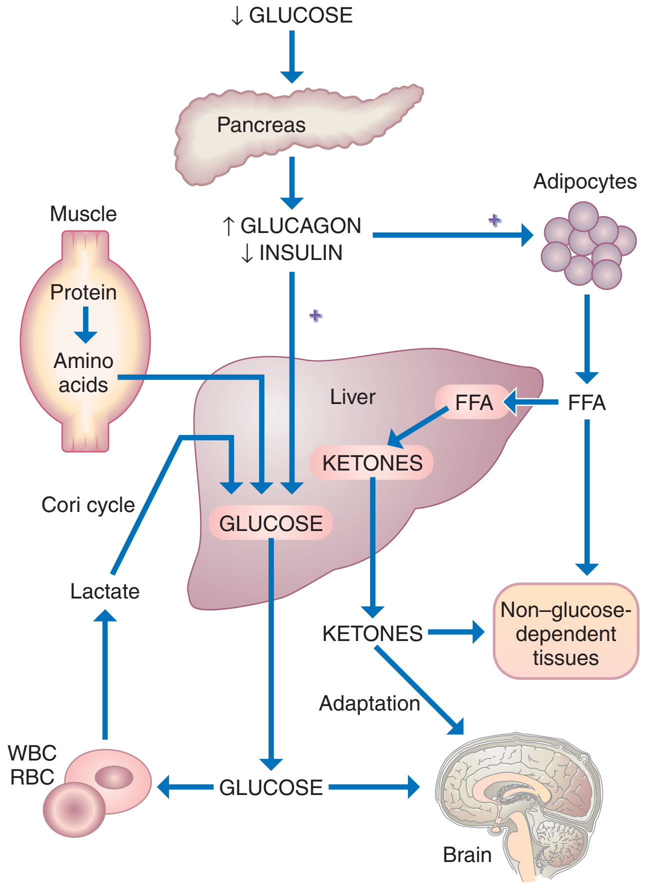

Figure 15-2 Metabolic changes in starvation. *FFA,* Free fatty acids; *RBC,* red blood cells; *WBC,* white blood cells. *(From Simmons RL, Steed DL:* Basic science review for surgeons, *Philadelphia, 1992, Saunders.)*

TABLE 15-3 Metabolic Responses to Severe Stress

Ebb Phase	Flow Phase
↓ Oxygen consumption	↑ Oxygen consumption
↓ Cardiac output	↑ Cardiac output
↓ Plasma volume	↑ Plasma volume
Hypothermia	Hyperthermia ↑ Nitrogen excretion
↓ Insulin levels	Normal or elevated insulin levels
Hyperglycemia	Hyperglycemia
Hypovolemia	
Hypotension	
↑ Lactate	Normal lactate
↑ Free fatty acids	↑ Free fatty acids
↑ Catecholamines, glucagon, cortisol	↑ Catecholamines, glucagon, cortisol
Insulin resistance	↑ Insulin resistance

key term

branched-chain amino acids (BCAA) leucine, isoleucine, and valine

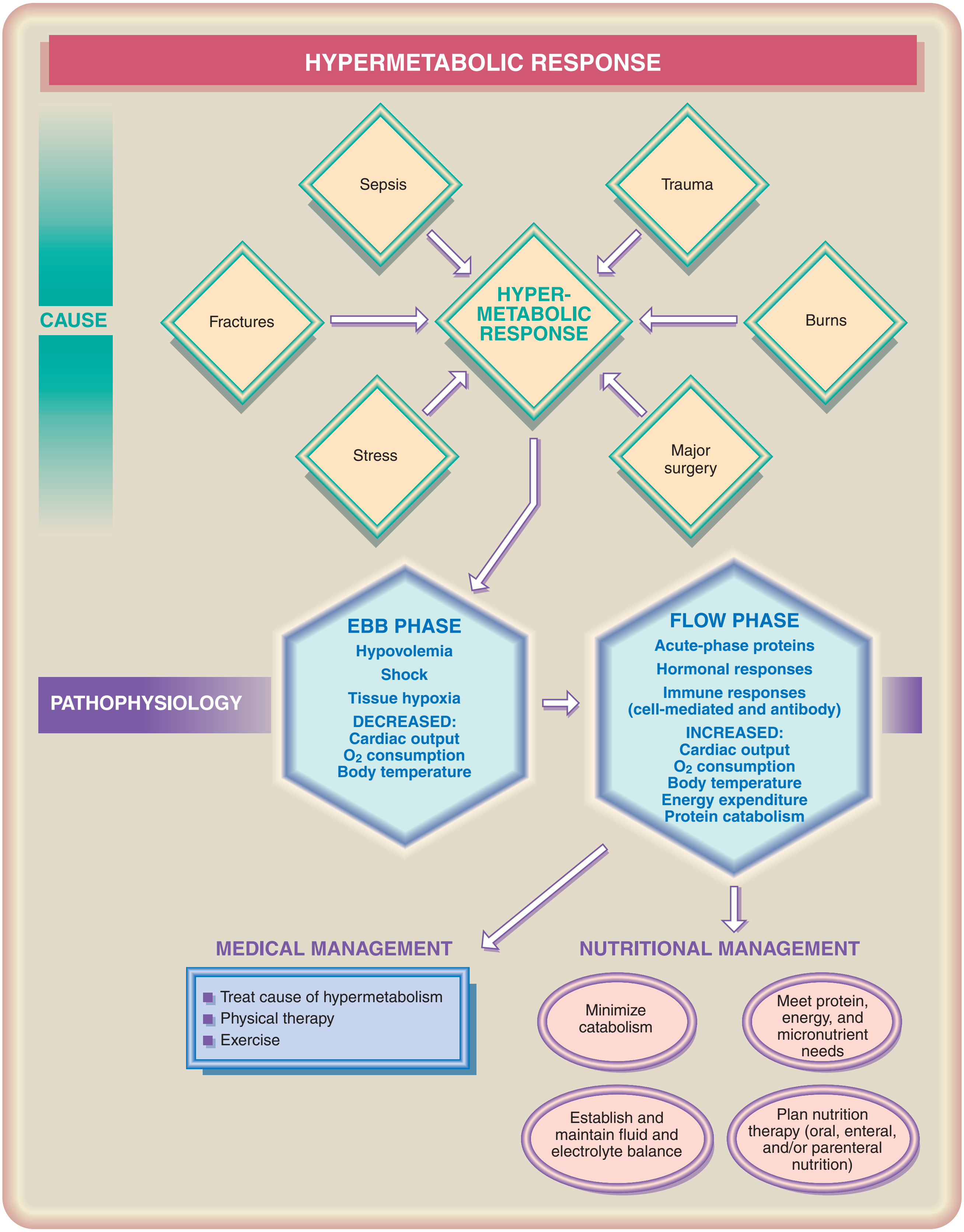

Figure 15-3 Hypermetabolic response to stress pathophysiology algorithm. *(From Mahan LK, Escott-Stump S:* Krause's food, nutrition, & diet therapy, *ed 11, Philadelphia, 2004, Saunders. Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Marion F. Winkler, MS, RD, LDN, CNSD and Ainsley Malone, MS, RD, CNSD, 2002.)*

As the body responds to injury, the ebb phase evolves into the flow phase, usually about 36 to 48 hours after injury.[5] The *flow phase* is characterized by increased oxygen consumption, hyperthermia (increased body temperature), and increased nitrogen excretion, as well as expedited catabolism of carbohydrate, protein, and triglycerides to meet the increased metabolic demands.[5] The flow stage will last for days, weeks, or months until the injury is healed.

Multiple stresses result in increased catabolism and even greater loss of body proteins. Unfortunately, some stresses that patients are obliged to endure are iatrogenic. Think, for example, of the series of stresses a patient admitted for elective surgery might experience. Preoperatively, most surgical patients receive only clear liquids or nothing by mouth (NPO). After surgery, they may remain NPO until the return of bowel sounds, then progress through clear liquid and full-liquid diets until they can tolerate food.

If the patient is in poor nutritional status before the stress of surgery, he or she is at greater risk for developing pneumonia or a wound infection accompanied by fever as a result of decreased protein synthesis. As in starvation, energy requirements will be met from **endogenous** sources if **exogenous** sources are not available or adequate.[4] Thus intercostal muscles may be depleted, leading to pneumonia, or inadequate amino acids may be available to synthesize antibodies, leading to impaired immune response to infection. Either complication has a negative impact on metabolic demands.

Nutrients affected by hypermetabolic stress include protein, vitamins, and minerals, as well as related nutritional concerns for total energy and fluid intake. During moderate metabolic stress, protein requirements have been reported to increase from 0.8 g/kg body weight (amount recommended for an average healthy adult) to 1 to 1.5 g/kg body weight[6,7] and for severe stress (e.g., thermal injuries exceeding 20% total body surface area) can rise to 1.5 to 2 g/kg body weight.[7] These levels are based on sufficient energy consumption to allow for protein synthesis. Requirements of vitamins and minerals all increase during stress. Tissue repair especially depends on adequate intakes of vitamin C, zinc, calcium, magnesium, manganese, and copper. At the least, dietary reference intake (DRI) levels of nutrients should be consumed, preferably from foods rather than from vitamin or mineral supplements. Achieving requirements through food intake also supports provision of sufficient kcal to meet increased energy demands during critical illness.

Several formulas have been used to determine energy needs of patients experiencing hypermetabolic stress. One formula (Harris-Benedict) takes into account basal energy expenditure (BEE), activity level, and severity of injury.[8] Activity level considers energy required if the patient is confined to bed or is ambulatory. Severity of injury is a factor based on whether the injury is caused by major or minor surgery, mild to severe infection, skeletal or blunt trauma, or burns (based on percentage of body surface area affected) (Box 15-1).[9]

Registered dietitians, in collaboration with the medical team, use these formulas to determine energy requirements. As factor assessments change, nurses can alert either the registered dietitian or other members of the medical team to ensure adequate energy provision.

Fluid need during hypermetabolic stress is based on age, reflecting age-related modifications of body composition. For adults younger than 55 years, fluid needs are calculated at 35 to 40 mL/kg body weight. Adults between the ages of 55 and 75 years require a lower amount, 30 mL/kg body weight; and for adults older than age 75, 25 mL/kg body weight is recommended.[7]

Effects of Stress on Nutrient Metabolism

Protein Metabolism

Even if adequate carbohydrate and fat are available, protein (skeletal muscle) is mobilized for energy (amino acids are converted to glucose in the liver). There is decreased uptake of amino acids by muscle tissue, and increased urinary excretion of nitrogen[9,10] (Figure 15-4). Some nonessential amino acids may become conditionally essential during episodes of metabolic stress. During stress, glutamine is mobilized in large quantities from skeletal muscle and lung to be used directly as a fuel source by intestinal cells.[11] Glutamine also plays a significant role in maintaining intestinal immune function and enhancing wound repair by supporting lymphocyte and macrophage proliferation, hepatic gluconeogenesis, and fibroblast function (Figure 15-5).[11]

Carbohydrate Metabolism

Hepatic glucose production is increased and disseminated to peripheral tissues although proteins and fats are being used for energy. Insulin levels and glucose use are in fact increased, but hyperglycemia that is not necessarily resolved by the use of exogenous insulin[9,10] is present. This appears, to some extent, to be driven by an elevated glucagon-to-insulin ratio.[10]

Fat Metabolism

To support hypermetabolism and increased gluconeogenesis, fat is mobilized from adipose stores to provide energy (lipolysis)[9] as the result of elevated levels of catecholamines along with concurrent decrease in insulin production.[10] If hypermetabolic patients are not fed during this period, fat stores and proteins are rapidly depleted. This malnutrition increases susceptibility to infection and may contribute to multiple organ dysfunction syndrome (MODS), sepsis, and death.[9]

key terms

endogenous originating from within the body or produced internally

exogenous originating outside the body or produced from external sources

BOX 15-1 Medical Nutrition Therapy for Metabolically Stressed Patients

Energy requirements are highly individual and may vary widely from person to person. Total kcal requirements are dependent on the basal energy expenditure (BEE) plus the presence of trauma, surgery, infection, sepsis, and other factors. Additionally, age, height, and weight are often taken into consideration.[2]

Harris-Benedict Formula

The Harris-Benedict formula is one of the most useful and accurate for calculating basal energy requirements,[2] although it generally overestimates BEE by 5% to 15%. It is important to remember this formula uses *current (actual)* weight in the calculation.

$$\text{Wt in pounds} \div 2.2\ \text{kg} = \text{Wt in kg}$$
$$\text{Ht in inches} \times 2.54\ \text{cm} = \text{Ht in cm}$$
$$\text{Men} = 66.5 + (13.8 \times \text{Wt in kg}) + (5 \times \text{Ht in cm}) - (6.8 \times \text{Age})$$
$$\text{Women} = 65.1 + (9.6 \times \text{Wt in kg}) + (1.8 \times \text{Ht in cm}) - (4.7 \times \text{Age})$$

Once BEE has been calculated, additional kcal for activity and injury are added:

$$\text{BEE} \times \text{Activity factor (AF)} \times \text{Injury factor (IF)}$$

Protein Requirements

Additional protein is required to synthesize the proteins necessary for defense and recovery, to spare lean body mass, and to reduce the amount of endogenous protein catabolism for gluconeogenesis.

Vitamin/Mineral Needs

Needs for most vitamins and minerals increase in metabolic stress; however, no specific guidelines exist for provision of vitamins, minerals, and trace elements. It is usually believed that if the increased kcal requirements are met, adequate amounts of most vitamins and minerals are usually provided. In spite of this, vitamin C, vitamin A or beta carotene, and zinc may need special attention.

Fluid Needs

Fluid status can affect interpretation of biochemical measurements as well as anthropometry and physical examination. Fluid requirements can be estimated using several different methods.

Micronutrient Supplementation

Vitamin C: 500 to 1000 mg/daily in divided dose
Vitamin A: one multivitamin tablet containing vitamin A, one to four times daily
Zinc sulfate: 220 mg, one to three times daily

Activity	Activity Factor	Clinical Status	Energy Stress Factor	g Protein/kg Body Wt/Day
Bed rest	1.2	Elective surgery	1-1.2	1-1.5
Ambulatory	1.3	Multiple trauma	1.2-1.6	1.3-1.7
		Severe infection	1.2-1.6	
		Peritonitis	1.05-1.25	
		Multiple/long bone fractures	1.1-1.3	
		Infection with trauma	1.3-1.5	
		Sepsis	1.2-1.4	1.2-1.5
		Closed head injury	1.3	
		Cancer	1.1-1.45	
		Burns (% BSA)	1.8-2.5	
		0%-20%	1-1.5	
		20%-40%	1.5-1.85	
		40%-100%	1.85-2.05	
		Fever	1.2 per 1° C >37° C	

BSA, Body surface area.

Fluid Requirements Based On:		Water (mL)
Weight		100 mL/kg for first 10 kg
		50 mL/kg for next 10 kg
		20 mL/kg for each kg above 20 kg
Age and weight	16-30 yr (active)	40 mL/kg/day
	20-55 yr	35 mL/kg/day
	55-75 yr	30 mL/kg/day
	>75 yr	25 mL/kg/day
Energy	1 mL/kcal	
Fluid balance	Urine output + 500 mL/day	

Data from American Dietetic Association: *Manual of clinical dietetics,* ed 6, Chicago, 2000, American Dietetic Association; Heimburger DC, Ard J: *Handbook of clinical nutrition,* ed 4, St Louis, 2006, Mosby; Moore MC: *Mosby's pocket guide to nutritional care,* ed 5, St Louis, 2005, Mosby; and Winkler MF, Malone AM: Medical nutrition therapy for metabolic stress: sepsis, trauma, burns and surgery. In Mahan LK, Escott-Stump S, eds: *Krause's food, nutrition, & diet therapy,* ed 11, Philadelphia, 2004, Saunders.

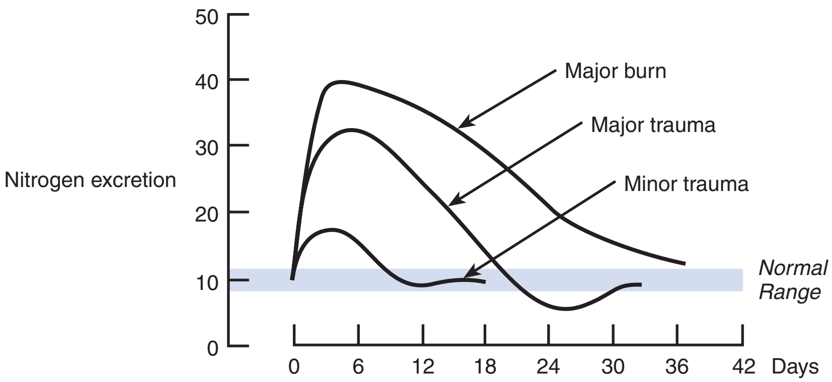

Figure 15-4 Nitrogen excretion. *(From Kinney JM et al:* Nutrition and metabolism in patient care, *Philadelphia, 1988, Saunders.)*

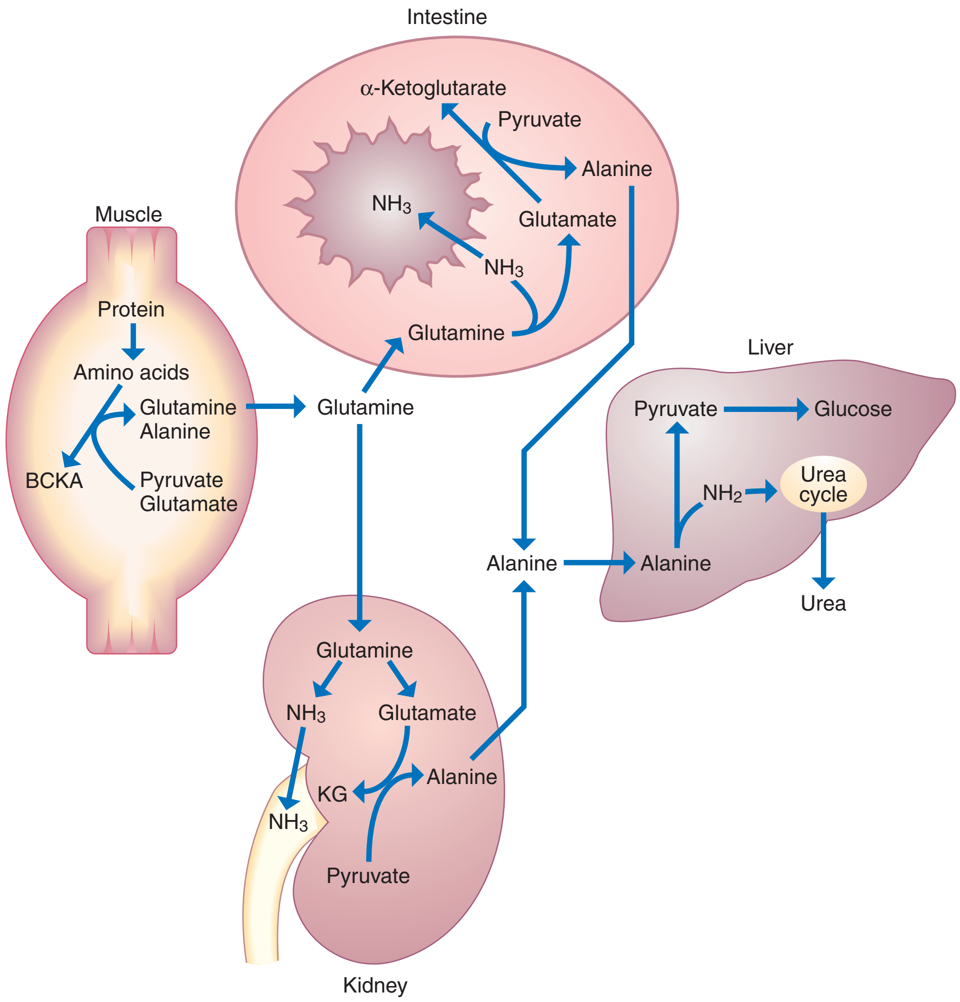

Figure 15-5 Glutamine metabolism. Glutamine is generated by skeletal muscle from glutamate by transamination. Glutamine is taken up by the intestine and kidney, where deamination and ammonia elimination occur. The glutamate formed is transaminated with pyruvate to form alanine, which goes to the liver for gluconeogenesis, and alpha-ketoglutarate (KD), which can be used for energy production by the muscle or kidney. *NH_2*, Amine; *NH_3*, ammonia. *(From Simmons RL, Steed DL:* Basic science review for surgeons, *Philadelphia, 1992, Saunders.)*

Hydration/Fluid Status

Increased fluid losses can result from fever (increased perspiration), increased urine output, diarrhea, draining wounds, or diuretic therapy.[10]

Vitamins and Minerals

Just as kcal needs increase during hypermetabolic conditions, so, too, do needs for most vitamins and minerals. And if kcal needs are met, the patient will most likely receive adequate amounts of most vitamins and minerals. Special attention, however, should be given to vitamin C (ascorbic acid), vitamin A or beta-carotene, and zinc.[12] Vitamin C is crucial for the collagen formation necessary for optimal wound healing. Supplements of 500 to 1000 mg/day are recommended.[12] Vitamin A and beta-carotene (vitamin A's precursor) play an important role in the healing process in addition to their role as antioxidants. Zinc increases the tensile strength (force required to separate the edges) of a healing wound. Supplements of 220 mg/day zinc sulfate (orally) when stable are commonly used.[12] Additional zinc may be necessary if there are unusually large intestinal losses (small bowel drainage or ileostomy drainage).[12]

Protein-Energy Malnutrition

Inadequate intake of energy, particularly from protein, can result in acute or chronic protein deficiency, or protein-energy malnutrition (PEM). PEM can be primary or secondary. Primary PEM is the result of inadequate intake of nutrients. Secondary PEM results from inadequate nutrient consumption caused by some disease state that impairs food consumption, interferes with nutrient absorption, or increases nutritional requirements. PEM, kwashiorkor, and marasmus are presented in detail in Chapter 6 and only briefly reviewed here.

Kwashiorkor

The clinical syndrome kwashiorkor is diagnosed largely on the basis of results of laboratory tests on patients in the acute state of poor protein intake and stress. Although etiologic mechanisms are not understood, it appears that normal adaptive response of protein sparing seen in fasting fails. Kwashiorkor may develop in as little as 2 weeks.[6]

Patients with kwashiorkor appear to be adequately nourished, tending to have normal fat reserves and muscle mass (or even above normal). However, findings such as easily pluckable hair, edema, skin breakdown, and delayed

Figure 15-6 Clinical findings in kwashiorkor include **(A),** easy, painless hair pluckability; **(B),** pitting edema; **(C),** skin breakdown; and **(D),** delayed wound healing. *(From Morgan S, Weinsier R:* Fundamentals of clinical nutrition, *ed 2, St Louis, 1998, Mosby.)*

wound healing are telltale signs of kwashiorkor (Figure 15-6). Characteristic laboratory changes include severely depressed **visceral proteins:** serum albumin (<2.8 g/dL), transferrin (<150 mg/dL), or reduced iron-binding capacity (<200 ug/dL) and depressed immune function (<1500 lymphocytes/mm^3).[6]

Marasmus

Another form of PEM—marasmus—is manifested by severe loss of fat and muscle tissue as a result of chronic energy deficiency. Unlike kwashiorkor, an individual with marasmus will appear thin and is weak and listless. Visceral protein stores are preserved at the expense of **somatic proteins:** skeletal muscle is severely reduced, but laboratory values are relatively unremarkable (serum albumin is usually within normal range).[6] Immunocompetence and wound healing are fairly well preserved in patients with marasmus. Marasmus is a chronic rather than acute condition. Treatment is directed toward gradual reversal of the downward trend. And although medical nutrition therapy or support is necessary, overly aggressive repletion of nutrients can lead to a life-threatening condition called **refeeding syndrome** (Box 15-2).[6]

Marasmus-Kwashiorkor Mix

This combined form of PEM develops when acute stress (surgery or trauma) is experienced by someone who has been chronically malnourished.[13] The condition becomes life threatening because of the high risk of infection and other complications. It is important to determine whether marasmus or kwashiorkor is predominant so appropriate medical nutrition therapy can be initiated. The undernourished, unstressed (hypometabolic) patient is at risk of complications such as those observed in refeeding

key terms

visceral proteins proteins other than muscle tissue; for example, internal organs and blood

somatic proteins skeletal muscle proteins

key term

refeeding syndrome physiologic and metabolic complications associated with reintroducing nutrition (refeeding) too rapidly to a person with PEM; these complications can include malabsorption, cardiac insufficiency, congestive heart failure, respiratory distress, convulsions, coma, and perhaps death

BOX 15-2 Refeeding Syndrome

Refeeding a patient with protein-energy malnutrition can result in many complications if not initiated correctly. In fact, refeeding can be fatal if done too rapidly. The introduction of excess protein and kcal can overload various enzymatic and physiologic functions that may have adapted during malnutrition. As refeeding is initiated, rapid changes occur in thyroid and endocrine function, causing increased oxygen consumption cardiac output, insulin secretion, and energy expenditure. Refeeding syndromes are associated more with parenteral (feeding via circulatory system; see Chapter 14) nutrition than enteral (feeding via GI tract; see Chapter 14), but discretion and common sense are of key importance in refeeding semistarved and chronically ill patients. The pathogenesis of refeeding syndrome is described in the following sections.

Phosphorus

During starvation, total phosphorus is greatly reduced. During refeeding there is an increase in cellular influx of phosphorus, leading to severe extracellular **hypophosphatemia.** This will occur in enteral and parenteral feeding but can be prevented by a slower rate of nutrient infusion. Hypophosphatemia can also cause **cardiac decompensation.** (Sodium shifts are thought to play a separate, additional role in cardiac overload.) In addition, hypophosphatemia can lead to tissue **hypoxia** and subsequent altered tissue function.

Potassium

Because potassium is greatly reduced from tissue, and under anabolic conditions, extracellular fluid levels fall (hypomagnesemia), which in turn can lead to cardiac depression, arrhythmias, neuromuscular weakness, irritability, and **hyporeflexia.**

Magnesium

Magnesium is also greatly reduced from tissue, and under anabolic conditions, extracellular fluid levels fall (hypomagnesemia), which in turn can lead to cardiac depression, arrhythmias, neuromuscular weakness, irritability, and hyporeflexia.

Glucose Metabolism

When glucose is reintroduced via high-glucose or high-volume enteral or parenteral feedings, the starved patient loses the stimulus for gluconeogenesis (an important adaptive mechanism during nutritionally depletion). Suppression of gluconeogenic glucose production leads to a corresponding decrease in amino acid use and negative nitrogen balance. Additionally, hyperglycemia can precipitate osmotic diuresis, dehydration, hypotension, hyperosmolar nonketotic coma, ketoacidosis, and metabolic acidosis. Hyperosmolar nonketotic coma and ketoacidosis are discussed in Chapter 19.

Fluid Intolerance

Refeeding with carbohydrate results in sodium and water excretion. With concurrent sodium ingestion, this can lead to a rapid expansion of extracellular fluid volume, which will result in fluid retention and subsequent weight gain. This enhanced fluid retention seen with carbohydrate refeeding may in turn be exacerbated because of the loss of tissue mass resulting from starvation.

Preventing Refeeding Syndrome

Nutrients should be reintroduced slowly to the malnourished patient while medical and metabolic status is closely monitored. Careful estimation of energy requirements should be made through a complete nutritional assessment (see Chapter 14). Care should also be taken to minimize fluid retention (weight gain >1 kg/wk can be assumed to be fluid retention and should be avoided) and provide adequate repletion of phosphorus, potassium, and magnesium on a daily basis. Weight and fluid balance should be monitored daily to assess the rate of weight regain. Refeeding formulas (whether enteral or parenteral) must also contain adequate amounts of other essential nutrients such as vitamins and minerals. Greater than routine amounts are not necessary, but their absence may be lethal. After 1 week, intake of kcal, fluid, and sodium can be liberalized without fear of consequences because the various metabolic equilibrations should have taken place.

Data from Marinella MA: The refeeding syndrome and hypophosphatemia, *Nutr Rev* 61(9):320, 2003; and Parrish CR: Much ado about refeeding, *Pract Gastroenterol* 29(1):26, 2005.

syndrome, and the stressed patient at risk for kwashiorkor is more likely to suffer from underfeeding.[6]

Nurses can be key players in the recognition and prevention of any of the different forms of PEM. By being alert to clinical signs and laboratory values seen in kwashiorkor and marasmus, further deterioration of the patient's nutritional status can be prevented.

Multiple Organ Dysfunction Syndrome

Multiple organ dysfunction syndrome (MODS) involves the progressive failure of two or more organ systems at the same time (e.g., the renal, hepatic, cardiac, or respiratory systems).[14,15] It may occur following trauma, severe burns, infection, or shock and usually results from an uncontrolled inflammatory response and can progress to organ failure and death.[14,16] MODS commonly begins with lung failure followed by failure of the liver, intestine, and kidney.[16] Myocardial failure generally manifests later, but central nervous system changes can occur at any time.[16] The pathogenesis of MODS is complex but usually results in the initiation of the stress response and release of catecholamines,[14] producing a hypermetabolic state in the patient.[16] Higher levels of kcal and protein are necessary to meet increased metabolic demands. How patients are

key terms

hypophosphatemia low serum phosphorus levels
cardiac decompensation impaired cardiac output (reasons not entirely understood)
hypoxia lack of oxygen to the cells
hyporeflexia a neurologic condition characterized by weakened reflex reactions
multiple organ dysfunction syndrome (MODS) the progressive failure of two or more organ systems at the same time (e.g., the renal, hepatic, cardiac, or respiratory systems)

TABLE 15-4 Nutritional Concerns in Multiple Organ Dysfunction Syndrome

System	Effects	System	Effects
Pulmonary	Acute respiratory distress syndrome (ARDS): patient requiring ventilator support may need higher lipid content in their diet (even with cardiac failure)	**Central nervous system**	Lethargy Altered level of consciousness Fever: increased energy needs Hepatic encephalopathy
Gastrointestinal	Abdominal distention and ascites Intolerance to internal feedings Paralytic ileus Diarrhea Ischemic colitis Mucosal ulceration Bacterial overgrowth in stool	**Immune**	Infection: increased energy needs Decreased lymphocyte count Anergy
Liver	Increased serum ammonia level		
Hypermetabolism	Decreased lean body mass Muscle wasting Severe weight loss Negative nitrogen balance Hyperglycemia	**Gallbladder**	Abdominal distention Unexplained fever: increased kcal needs Decreased bowel sounds

Data from Baldwin KM, Cheek DJ, Morris SE: Shock, multiple organ dysfunction syndrome, and burns in adults. In McCance KL, Huether SE eds: *Pathophysiology: The biologic basis of disease in adults and children,* ed 5, St. Louis, 2006, Mosby; and Escott-Stump S: *Nutrition and diagnosis-related care,* ed 5, Baltimore, 2004, Lippincott Williams & Wilkins.

fed is also important. Early enteral feedings (Chapter 14) appear to maintain gut mucosal mass and barrier function and promote normal enterocytic growth in the gut.[13,17] This is not possible with parenteral feedings (Table 15-4).

Surgery

In a perfect world, all patients undergoing surgery would be at optimal nutritional status to help them tolerate the physiologic stress of the surgery and temporary starvation that follows. But all too often, surgical patients may be malnourished secondary to the medical condition causing the need for surgery. Additionally, they may experience anorexia, nausea, or vomiting, which decrease their ability to eat. Fever may increase their metabolic rate. Or nutritional needs may not be met because of malabsorption. For surgery to be successful, patients who are malnourished or in danger of malnutrition must be identified so corrective action may be arranged. Before surgery, patients are typically limited to NPO to prevent aspiration. Oral intake is generally resumed when bowel sounds return, usually 24 to 48 hours after surgery. The postoperative diet usually progresses from clear liquid to solid foods as tolerated.

Burns (Thermal Injury)

Burns are customarily defined as electrical, thermal, chemical, or radioactive. They produce tissue destruction that results in circulatory and metabolic alterations that require the compensatory response to injury (Table 15-5).[18] Actual cause of burns may be thermal or nonthermal, such as chemical, electrical, or radioactive sources. Thermal burns are usually characterized as contact (hot solid object), flame (direct contact with flames), or scald injuries (heated liquid).[14] These events have significant effects on nutritional status.

Burns are generally classified by physical appearance and symptoms associated with the affected skin[14] and are often described in terms of percent of body surface burned (Figure 15-7). First-degree burns (or partial-thickness injury) involve only the epidermis, resulting in simple reddening of the area with no injury to underlying dermal or subcutaneous tissue.[14,15] Sunburns are an example of first-degree burns caused by ultraviolet radiation damage to skin.

TABLE 15-5 Nutritional Goals for Burned Patients

Goal	Action
Minimize metabolic response	Control environmental temperature Monitor fluid and electrolyte balance Control pain and anxiety Cover wounds early
Meet nutritional needs	Provide adequate kcal to prevent weight loss >10% of usual body weight Provide adequate protein for positive nitrogen balance and maintenance or repletion of visceral protein stores
Prevent Curling's ulcer	Provide antacids or continuous enteral feedings

Modified from Winkler MF, Malone AM: Medical nutrition therapy for metabolic stress: Sepsis, trauma, burns and surgery. In Mahan LK, Escott-Stump S, eds: *Krause's food, nutrition, & diet therapy,* ed 11, Philadelphia, 2004, Saunders.

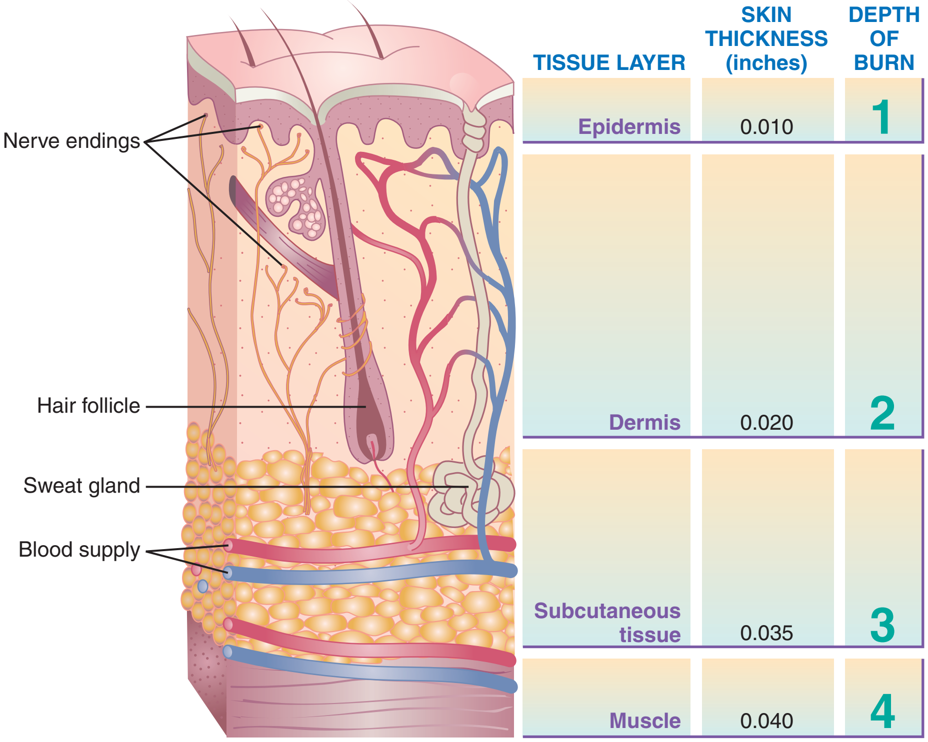

Figure 15-7 Interpretation of burn classification based on damage to the integument. *(From Mahan LK, Escott-Stump S:* Krause's food, nutrition, & diet therapy, *ed 11, Philadelphia, 2004, Saunders.)*

First-degree burns heal within 3 to 5 days without scarring.[14] Second-degree burns (superficial partial-thickness injury and deep partial-thickness injury) involve two categories of burn depth with distinctly different characteristics.[14] Superficial partial-thickness burns are characterized by redness and blistering that affects epidermis and some dermis.[14,15] Deep partial-thickness burns are characterized by destruction of epidermis and dermis (resulting in a waxy, white, mottled appearance), leaving only skin appendages such as hair follicles and sweat glands.[14] Second-degree burns take weeks to months to heal. Third-degree burns (full-thickness injury) are characterized by destruction of the entire epidermis, dermis, and frequently the underlying subcutaneous tissue. Occasionally, muscle or bone tissue may be destroyed.[14] Third-degree burns do not heal and require skin grafts[14] (see the *Personal Perspectives* box, Love, Greg & Lauren, for one couple's struggle with the aftermath of severe burns).

In addition to pain management, wound care, and infection control, nutrition support is recognized as one of the most significant considerations of patient care.[14,16] The first 24 to 48 hours of treatment for burn patients are dedicated to replacement of fluid and electrolytes. Fluid needs are based on the patient's age, weight, and extent of the burn.[19] Total body surface area (TBSA), used to estimate the extent of the burn, can be estimated using the "rule of nines" (Figure 15-8). Thermal injury wounds will heal only if the patient is in an anabolic state. Therefore, feeding should be initiated as soon as the patient has been hydrated.[19] Very early enteral feeding (within 4 to 12 hours of hospitalization) has been shown to be successful in decreasing the hypercatabolic response, decreasing the release of catecholamines and glucagon, reducing weight loss, and shortening the length of the hospital stay.[20]

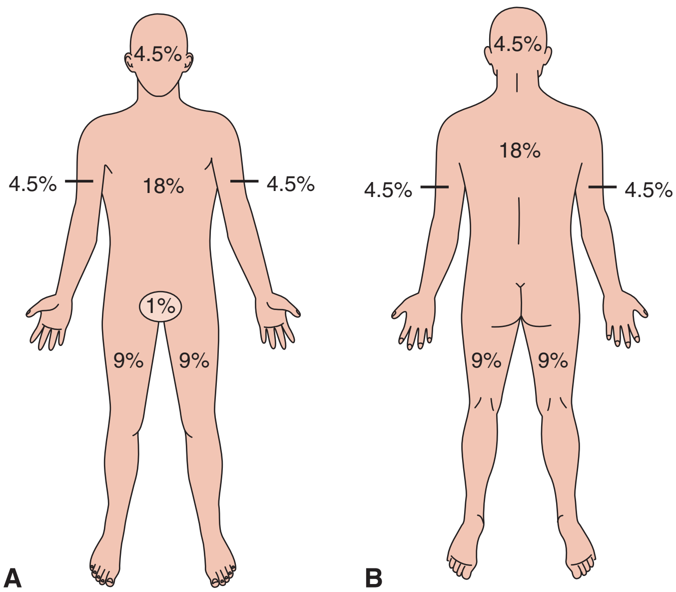

Figure 15-8 Rule of nines—a commonly used assessment tool with estimates of the percentages (in multiples of nine) of the total body surface area burned. **A,** Adults (anterior view). **B,** Adults (posterior view). *(From Thompson JM et al:* Mosby's clinical nursing, *ed 5, St Louis, 2002, Mosby.)*

Nutritional goals for patients with burns are outlined in Table 15-5. Several methods may be used to estimate energy and protein needs in burn patients. Energy needs vary according to the size of the burn.[16] One of the simplest

PERSONAL PERSPECTIVES

Love, Greg & Lauren

On September 11, 2001, at 8:48 AM, Lauren Manning, a senior vice president, partner, and director of global data sales for Cantor Fitzgerald, was entering the lobby of 1 World Trade Center in New York City. As the first of two planes drove into the World Trade Center buildings, an explosive fireball ran through the lobby. Lauren was burned on more than 82.5% of her body. Below is an excerpt from her husband's day-by-day e-mail account in the months following the tragedy of Lauren's struggle to heal and survive for her son, Tyler, and her husband. Consider the effect of serious injuries on patients, their families, and the medical personnel who assist in the healing process.

From: Greg
To: Everyone
Date: Saturday, September 29, 2001, 12:40 AM
Subject: Lauren Update for September 28 (Friday)

Today was a stable day. Lauren still has the septic infection, which they are fighting with antibiotics, but her lungs are functioning well, as is her stomach, two very important factors. The oxygen and the protein intake she is receiving through a feeding tube are needed to build new tissue and for her skin to heal.

I have a better understanding now of something the doctor told me about doing Lauren's grafts. He said he would "mesh 3-1" when doing autografts. Basically, a special machine is used to create a mesh pattern in the donor skin—her own skin—that permits it to cover an area three times as large as the site from which it was taken. The homograft, or skin-bank skin, is then placed over this mesh, creating a layer that enables the autograft beneath to heal better. The goal is for the mesh to take and for healing to occur in the open spots. More than one graft is often necessary to finish each site.

The grafts already done look good, which means the majority have probably taken. Unfortunately, the infection does have an adverse effect on the healing process, both of the grafts, and of the donor sites. That is why Lauren's time in the burn ICU is such a balancing act. Negative factors have to be controlled so that positive factors can win out. The good aspect for Lauren is that she was strong and healthy going in, so she has managed to keep herself mostly stable, a word that has become very important for the families of all the burn patients.

Her nurse explained to me tonight how Lauren's various systems were adjusting on their own to maintain stability. For example, her heart was pumping faster to maintain her blood pressure despite a slight dilation of blood vessels due to infection. A glass-half-full type of sign.

I put two pictures—of Lauren and of Lauren, Tyler, and my dad—up on her wall. The pictures are an important way for the nursing staff to make a connection to her. They are all looking forward to meeting her when she is more awake, later in her treatment course.

That alone should tell you how difficult the work is that these nurses do; the patients arrive gravely injured, frequently unable to communicate, and highly critical. The medical and nursing staffs often fight for weeks to keep the patient improving; this is well before they have a chance to encounter the patient's personality. The staff first gets to know the patient through the family visitors, and the photographs help the staff connect with the life they are trying to help the patient return to.

The WTC disaster families have been there for seventeen days now and we know each other well. This bonding between families is due to the utter stress of the situation; we have all spent days, now weeks, and hopefully will spend months, worrying minute to minute about a loved one's condition. It is the same as if a surgical procedure were to last for weeks on end. We learn to read the facial expressions and voices of doctors and nurses.

So we, the waiting, speak to each other, and to the staff psychologists and chaplain and the Red Cross volunteers who wander through, and we are visited by Good Samaritans of all types, who provide food . . . And in the end, we alone understand what we are going through: we are the loved ones of critically injured patients from a massive tragedy in which most victims either died or walked out under their own power.

We, the waiting, are therefore at somewhat of a disconnect from the world at large, which is pursuing closure (not my favorite word), whether coping with loss of a family member; coming to terms with having one's life saved by something so trivial as arriving late for work; or honoring the heroism of lost firefighters and police.

Most of the world is already viewing the attacks from a distance, but we are pretty much still there at Time Zero, with the outcome unknown. However, we are all making it through, with the help of the huge support networks that have sprung up all around us. Including y'all . . . It really does help us, me and Lauren, to know how many people care.

Love,
Greg & Lauren

Update: *Lauren Manning left home for work on September 11, 2001, and returned home on March 15, 2002. She continues to regain the life she had including running, biking, getting back to work, and just being there with and for her son as he grows up.*

From Manning G: *Love, Greg and Lauren,* New York, 2002, Bantam Books.

and easiest to use is the Curreri formula (adults), as follows[21]:

$$\text{kcal needed per day} \times [25\ \text{kcal} \times \text{kg } \textit{usual} \text{ body weight (kg)}] + [40\ \text{kcal} \times \%\ \text{TBSA burned}]$$

Estimates using the Curreri formula may exceed actual energy needs,[15,19] but it is not uncommon for a patient to need 4000 to 5000 kcal.[15] Another method is to calculate BEE (Harris-Benedict) and multiply by a factor of 1.5 to 2.[15]

Protein lost through urine and wounds, increased protein use for gluconeogenesis, and wound healing increase protein needs in burned patients.[16] It is therefore important that kcal from protein are not calculated into total energy needs.[5] Carbohydrates and fats are good for protein sparing (nonprotein energy sources).[5,16] Whether a patient receives adequate amounts of energy or protein is best evaluated by wound healing, graft take, and basic nutritional assessment parameters.[16]

In conjunction with increased energy demands, vitamin and mineral needs are generally increased in burn patients, but exact requirements are not known.[16] Most patients will receive vitamins in excess of the recommended intake because of their high kcal intakes, but special consideration should be given to vitamin C (collagen synthesis, immune function) and vitamin A (immune function and epithelialization). Supplements are commonly recommended.[16]

Summary

The stress response of the body also affects nutritional status. Whether the stress response is caused by physiologic or psychologic determinants, the entire body is affected. Metabolic changes take place in reaction to stress. This includes changes caused by uncomplicated stress that is present when patients are at nutritional risk and severe stress caused by trauma or disease. The functioning of the immune system is also affected by the hormonal and metabolic changes that occur when metabolic stress develops. The immune system's ability to protect the body is further depressed if impaired nutritional status accompanies the metabolic stress.

The Nursing Approach

Case Study: Nutritional Needs With Physical Stress

Daniel, age 65, developed pneumonia 4 days after his left hip replacement. He is receiving physical therapy, IV antibiotics, supplemental oxygen, and a high-kcal, high-protein diet. The hospital dietitian met with Daniel to individualize his diet, based on his Orthodox Jewish religion.

ASSESSMENT

Subjective

- "The muscles in my chest ache from coughing and my hip hurts."
- Pain rating: 3 of 10
- "I don't feel like eating, but if I have to eat, I want to observe dietary laws for an Orthodox Jew."
- "I feel tired and sometimes short of breath."

Objective

- Eats small amounts of food, then pushes the food tray away
- Drinks about 1200 mL of fluid per day (mostly water)
- Tympanic temperature 101.2° F
- Crackles in lungs bilaterally
- O_2 saturation 90% with oxygen at 6 liters via cannula
- White blood count 12,000/mm^3
- Productive cough, with thick yellow sputum
- Surgical wound on left hip intact without redness or drainage

DIAGNOSIS

Imbalanced Nutrition: Less Than Body Requirements related to inadequate food intake (secondary to shortness of breath and discomfort) and increased metabolic stresses (surgery, infection, and fever) as evidenced by "I don't feel like eating," eats small amounts then pushes meal tray away, drinks about 1200 mL of fluid per day

PLANNING

Outcomes

Short term (by discharge in 5 days):

- Eating moderate amounts of food, especially protein-rich and nutrient-dense foods
- Drinking at least 2000 mL per day
- No weight loss
- No shortness of breath, O_2 saturation 95% without supplemental oxygen
- Afebrile
- White blood cell (WBC) count below 10,000/ mm^3
- Decreased or absent crackles in lungs
- No signs of infection of surgical wound on left hip

Interventions

1. Provide high-kcal, high-protein diet.
2. Maintain dietary intake according to Orthodox Jewish Kosher dietary laws.

IMPLEMENTATION

1. Asked Daniel's family to bring in kosher ground meat and special serving dishes.
   If the hospital does not have a kosher kitchen, kosher meals may be ordered from special suppliers or family members may be able to obtain kosher food and reassure the patient that it is truly kosher. Meat (no pork or shellfish) must be properly slaughtered, blessed by a rabbi, and cooked in a kosher kitchen. Dishes reserved for meat must be separated from dishes for milk, and meat cannot be served in the same meal as milk. Ground meat provides high protein and requires little energy for chewing.
2. Asked Daniel and his family to inform nursing or dietary staff about special dietary needs, particularly for upcoming holidays and the Sabbath.
   Culturally sensitive staff will ask individuals and families how they can meet special dietary needs.
3. Provided rest periods and oral care before meals, and gave pain medicine as needed.
   Rest helps increase patient energy, and a fresh mouth promotes appetite. Patients eat more food when comfortable.
4. Pointed out the high-protein and nutrient-dense foods on meal trays and encouraged Daniel to eat them first.
   When patients can eat only small amounts of food, nourishment is better if they choose nutrient-dense foods.
5. Conferred with the dietitian and physician concerning high-kcal, high-protein snacks between meals.
   Increased kcal are needed to compensate for metabolic stresses, and additional protein is needed for healing and building up immunity. Frequent small meals are easier to consume than large meals when the patient is short of breath. Some doctors prefer to limit milk products because of potential phlegm. Milk-based supplements must be served several hours before or after meats, according to Jewish dietary laws.

6. Provided a vitamin/mineral supplement, as prescribed by the physician.
   Bone healing requires adequate calcium and vitamin D. Vitamins A and C help promote wound healing. B vitamins are needed in stressful conditions.
7. Offered water frequently between meals and encouraged drinking 2000 mL of fluid per day.
   Fluid intake is needed to replace fluids lost during fever. Additional fluids help thin sputum, making it easier for the patient to cough up the sputum. Liquids during meals should be minimized if patient feels full after eating little food.
8. Recorded intake and output and weighed the patient daily.
   Records can help show balance or imbalance of fluid intake and fluid output. Adequate nutrition is needed to prevent weight loss.
9. Encouraged Daniel to get a pneumonia shot this year and a flu shot every year.
   Serious respiratory infections may be prevented by immunizations. Health promotion is an important nursing responsibility.

EVALUATION

At discharge (after 5 days):

- Daniel was eating moderate amounts of food.
- He was drinking 2000 mL of fluids per day.
- No weight loss
- No shortness of breath, O_2 saturation 95% without supplemental oxygen
- Tympanic temperature 99° F
- WBC 9500/mm^3
- Decreased crackles in lungs
- No signs of infection of surgical wound on left hip
- Goals met

CRITICAL THINKING
Clinical Applications

Kristin, age 19, is a member of her college's cheerleading team and was involved in a serious motor vehicle accident when the team was returning from a game. She was admitted through the emergency department of your hospital suffering from multiple fractures and contusions. Kristin is 5 feet 5 inches tall and weighed 120 pounds before the accident. Because she is young, looked healthy, and is somewhat muscular from being a cheerleader, the physician did not request a consult for the dietitian to evaluate Kristin's nutritional status. After 2 weeks in intensive care, she developed pneumonia. The nurse learned that before the automobile accident, Kristin had been using a commercial weight loss product and was consuming approximately 400 kcal/day for 3 months before the accident in an attempt to "make weight" so that she could remain on the cheerleading team.

1. How did the very-low-calorie diet (VLCD) affect Kristin's nutritional status?
2. Why did Kristin develop pneumonia?
3. Describe the variety of stresses Kristin experienced.
4. Could the pneumonia have been prevented? How?

Want more practice? Visit http://evolve.elsevier.com/Grodner/

Websites of Interest

Burnsurgery.org

www.burnsurgery.org

This site provides up-to-date educational tools for health professionals who focus on burn care by providing dissemination of the most recent advances in burn treatment.

KidSource OnLine!

www.kidsource.com/kidsource/content2/ecoli/anna.1.html

This online community of parents presents support and resources on many topics related to parenting. The particular page cited above is a parent's personal account of her daughter's experience with MODS caused by an *Escherichia coli* infection.

References

1. Fernandes G et al: Nutrition and the immune system. In Shils ME et al, eds: *Modern nutrition in health and disease,* ed 10, Philadelphia, 2006, Williams & Wilkins.
2. Gould BE: *Pathophysiology for the health-related professions,* ed 3, Philadelphia, 2006, Saunders.
3. Cahill GF: Starvation: Some biological aspects. In Kinney JM et al, eds: *Nutrition and metabolism in patient care,* Philadelphia, 1988, Saunders.
4. Wolfe BM: Nutrition in hypermetabolic conditions. In Zeman FJ, ed: *Clinical nutrition and dietetics,* ed 2, New York, 1991, Macmillan.
5. Bessey PQ, Wilmore DW: The burned patient. In Kinney JM et al, eds: *Nutrition and metabolism in patient care,* Philadelphia, 1988, Saunders.
6. Morgan SL, Weinsier RL: *Fundamentals of clinical nutrition,* ed 2, St Louis, 1998, Mosby.
7. American Dietetic Association: *Manual of clinical dietetics,* ed 6, Chicago, 2000, Author.
8. Long CL: The energy and protein requirements of the critically ill patient. In Wright RA, Heymsfield SB, McManus CB III, eds: *Nutritional assessment,* Boston, 1984, Blackwell Scientific.
9. Kudsk KA, Sacks GS: Nutrition in the care of the patient with surgery, trauma and sepsis. In Shils ME et al, eds: *Modern nutrition in health and disease,* ed 10, Philadelphia, 2006, Williams & Wilkins.
10. Gottschlich MM: The burn patient. In Lysen LK, ed: *Quick reference to clinical dietetics,* Boston, 2006, Jones and Bartlett.
11. Heimburger DC, Ard J: *Handbook of clinical nutrition,* ed 4, St Louis, 2006, Mosby.
12. Moore MC: *Mosby's pocket guide to nutritional assessment and care,* ed 5, St Louis, 2005, Mosby.
13. Speath G et al: Food without fiber promotes bacterial translocation from the gut, *Surgery* 108(2):240, 1990.

14. Baldwin KM et al: Shock, multiple organ dysfunction syndrome, and burns in adults. In McCance KL, Huether SE, eds: *Pathophysiology: The biologic basis for diseases in adults and children,* ed 5, St Louis, 2006, Mosby.
15. Escott-Stump S: *Nutrition and diagnosis-related care,* ed 5, Baltimore, 2004, Williams & Wilkins.
16. Winkler MF, Malone AM: Medical nutrition therapy for metabolic stress: Sepsis, trauma, burns and surgery. In Mahan LK, Escott-Stump S, eds: *Krause's food, nutrition, & diet therapy,* ed 11, Philadelphia, 2004, Saunders.
17. Wilmore DW et al: The gut: A central organ after surgical stress, *Surgery* 104(5):917, 1988.
18. Gottschlick MM: Nutrition management for specific medical conditions. In Lysen LK, ed: *Quick references to clinical dietetics,* Gaithersburg, Md, 1997, Aspen.
19. Saffle JR, Larson CM, Sullivan J: A randomized trial of indirect calorimetry-based feedings in thermal injury, *J Trauma* 30:776, 1990.
20. Chiarelli A et al: Very early nutrition supplementation in burned patients, *Am J Clin Nutr* 51:1035, 1990.
21. Curreri PW: Supportive therapy in burn care. Nutritional replacement modalities, *J Trauma* 19(11 Suppl):906, 1979.