**TABLE 97-1 COMPARISON OF MARASMUS/CACHEXIA AND KWASHIORKOR/ACUTE MALNUTRITION**

| Feature | Marasmus (Starvation–Related Malnutrition) and Cachexia (Chronic Disease–Related Malnutrition) | Kwashiorkor (Acute Disease– or Injury–Related Malnutrition)[a] |
|---|---|---|
| Clinical setting | Prolonged ↓ energy and protein intake with or without systemic inflammation | Acute ↓ energy and protein intake with substantial systemic inflammation |
| Time course to develop | Months or years | Weeks |
| Clinical features | Starved appearance (body mass index <18.5) | Normal body mass index (although loss of body mass may be masked by edema) |
| | Reduced triceps skinfold | Easy hair pluckability[b] |
| | Reduced midarm muscle circumference | Edema |
| Laboratory findings | Serum albumin normal (marasmus, no inflammation) or decreased (cachexia, with inflammation) | Serum albumin <2.8 g/dL |
| | | Total iron-binding capacity <200 µg/dL |
| | | Lymphocytes <1500/µL |
| | | Anergy |
| Clinical course | Reasonably preserved responsiveness to short-term stress | Infections |
| | | Poor wound healing, decubitus ulcers, skin breakdown |
| Mortality risk | Low unless related to underlying disease | High |
| Diagnostic criteria | Triceps skinfold <3 mm | Serum albumin <2.8 g/dL |
| | Midarm muscle circumference <15 cm | At least one of the following: |
| | | Poor wound healing, decubitus ulcers, or skin breakdown |
| | | Easy hair pluckability[b] |
| | | Edema |

[a]The findings used to diagnose kwashiorkor/acute malnutrition must be unexplained by other causes. [b]Tested by *firmly* pulling a lock of hair from the top (not the sides or back), grasping with the thumb and forefinger. An average of three or more hairs removed easily and painlessly is considered abnormal hair pluckability.

appearance. The diagnosis is based on fat and muscle wastage resulting from prolonged calorie deficiency and/or inflammation. Diminished skinfold thickness reflects the loss of fat reserves; reduced arm muscle circumference with temporal and interosseous muscle wasting reflects the catabolism of protein throughout the body, including in vital organs such as the heart, liver, and kidneys.

Routine laboratory findings in cachexia/marasmus are relatively unremarkable. The creatinine-height index (24-h urinary creatinine excretion compared with normal values based on height) is low, reflecting the loss of muscle mass. Occasionally, the serum albumin level is reduced, but it remains above 2.8 g/dL when systemic inflammation is absent. Despite a morbid appearance, immunocompetence, wound healing, and the ability to handle short-term stress are reasonably well preserved in most patients.

Pure starvation–related malnutrition is a chronic, fairly well adapted form of starvation rather than an acute illness; it should be treated cautiously in an attempt to reverse the downward trend gradually. Although nutritional support is necessary, overly aggressive repletion can result in severe, even life-threatening metabolic imbalances such as hypophosphatemia and cardiorespiratory failure (*refeeding syndrome*). When possible, oral or enteral nutritional support is preferred; treatment started slowly allows readaptation of metabolic and intestinal functions (Chap. 98e).

## KWASHIORKOR (ACUTE DISEASE– OR INJURY–RELATED MALNUTRITION)

By contrast, *kwashiorkor* (acute disease– or injury–related malnutrition) in developed countries occurs mainly in connection with acute, life-threatening conditions such as trauma and sepsis. The physiologic stress produced by these illnesses increases protein and energy requirements at a time when intake is often limited. A classic scenario is an acutely stressed patient who receives only 5% dextrose solutions for periods as brief as 2 weeks. Although the etiologic mechanisms are not fully known, the protein-sparing response normally seen in starvation is blocked by the stressed state and by carbohydrate infusion.

In its early stages, the physical findings of kwashiorkor/acute malnutrition are few and subtle. Initially unaffected fat reserves and muscle mass give the deceptive appearance of adequate nutrition. Signs that support the diagnosis include easy hair pluckability, edema, skin breakdown, and poor wound healing. The major sine qua non is severe reduction of levels of serum proteins such as albumin (<2.8 g/dL) and transferrin (<150 mg/dL) or of iron-binding capacity (<200 µg/dL). Cellular immune function is depressed, as reflected by lymphopenia (<1500 lymphocytes/µL in adults and older children) and lack of response to skin test antigens (*anergy*).

The prognosis of adult patients with full-blown kwashiorkor/acute malnutrition is not good even with aggressive nutritional support. Surgical wounds often dehisce (fail to heal), pressure sores develop, gastroparesis and diarrhea can occur with enteral feeding, the risk of gastrointestinal bleeding from stress ulcers is increased, host defenses are compromised, and death from overwhelming infection may occur despite antibiotic therapy. Unlike treatment of marasmus, therapy for kwashiorkor entails aggressive nutritional support to restore better metabolic balance rapidly (Chap. 98e).

## PHYSIOLOGIC CHARACTERISTICS OF HYPOMETABOLIC AND HYPERMETABOLIC STATES

The metabolic characteristics and nutritional needs of hypermetabolic patients who are stressed from injury, infection, or chronic inflammatory illness differ from those of hypometabolic patients who are unstressed but chronically starved. In both cases, nutritional support is important, but misjudgments in selecting the appropriate approach may have serious adverse consequences.

The *hypometabolic* patient is typified by the relatively less stressed but mildly catabolic and chronically starved individual who, with time, will develop cachexia/marasmus. The *hypermetabolic* patient stressed from injury or infection is catabolic (experiencing rapid breakdown of body mass) and is at high risk for developing acute malnutrition/kwashiorkor if nutritional needs are not met and/or the illness does not resolve quickly. As summarized in Table 97-2, the two states are distinguished by differing perturbations of metabolic rate, rates of protein breakdown (*proteolysis*), and rates of gluconeogenesis. These differences are mediated by proinflammatory cytokines and counterregulatory hormones—tumor necrosis factor, interleukins 1 and 6, C-reactive protein, catecholamines (epinephrine and norepinephrine), glucagon, and cortisol—whose levels are relatively reduced in hypometabolic patients and increased in hypermetabolic patients. Although insulin levels are also elevated in stressed patients, insulin resistance in the target tissues blocks insulin-mediated anabolic effects. Physiologic

**TABLE 97-2 PHYSIOLOGIC CHARACTERISTICS OF HYPOMETABOLIC AND HYPERMETABOLIC STATES**

| Physiologic Characteristics | Hypometabolic, Nonstressed Patient (Risk for Starvation/Marasmus) | Hypermetabolic, Stressed Patient (Risk for Kwashiorkor/Acute Malnutrition) |
|---|---|---|
| Cytokines, catecholamines, glucagon, cortisol, insulin | ↓ | ↑ |
| Metabolic rate, $O_2$ consumption | ↓ | ↑ |
| Proteolysis, gluconeogenesis | ↓ | ↑ |
| Ureagenesis, urea excretion | ↓ | ↑ |
| Fat catabolism, fatty acid utilization | Relative ↑ | Absolute ↑ |
| Adaptation to starvation | Normal | Abnormal |