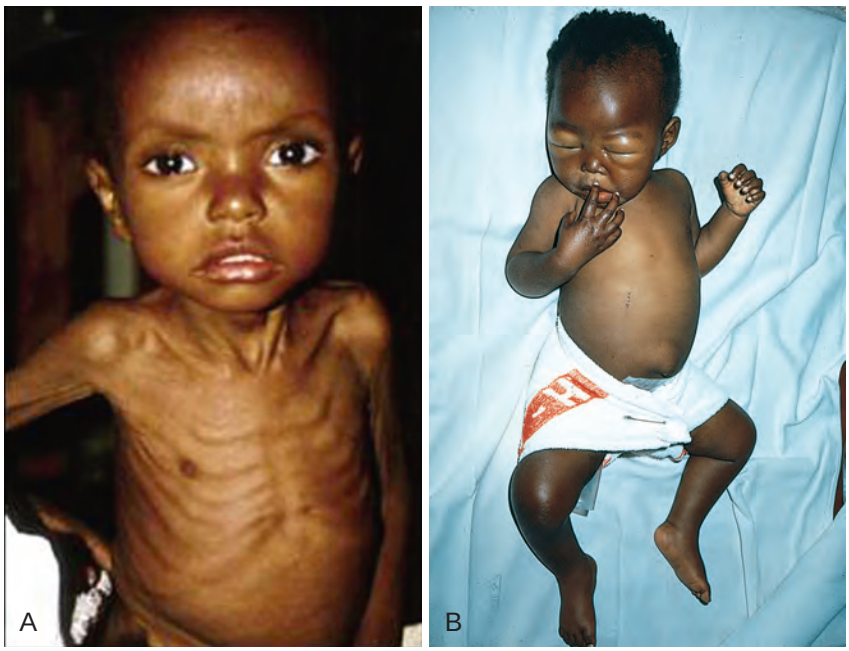

Figure 9-22 Childhood malnutrition. **A,** Marasmus. Note the loss of muscle mass and subcutaneous fat; the head appears to be too large for the emaciated body. **B,** Kwashiorkor. The infant shows generalized edema, seen as ascites and puffiness of the face, hands, and legs. (**A,** From Clinic Barak, Reisebericht Kenya.)

generalized or *dependent edema* (Fig. 9-22*B*). The loss of weight in these patients is masked by the increased fluid retention. In further contrast to marasmus, there is relative sparing of subcutaneous fat and muscle mass. Children with kwashiorkor have characteristic *skin lesions*, with alternating zones of hyperpigmentation, areas of desquamation, and hypopigmentation, giving a "flaky paint" appearance. *Hair changes* include overall loss of color or alternating bands of pale and darker hair. Other features that differentiate kwashiorkor from marasmus include an enlarged, *fatty liver* (resulting from reduced synthesis of the carrier protein component of lipoproteins), and the development of apathy, listlessness, and loss of appetite. Vitamin deficiencies are likely to be present, as are *defects in immunity* and *secondary infections*. As already stated, marasmus and kwashiorkor are two ends of a spectrum, and considerable overlap exists between these conditions.

PEM in the developed world. In the United States, secondary PEM often develops in chronically ill, older, and bedridden patients. An 18-item questionnaire known as the *Mininutritional Assessment* (MNA) is often used to measure the nutritional status of older persons. It is estimated that more than 50% of older residents in nursing homes in the United States are malnourished. Weight loss of more than 5% associated with PEM increases the risk of mortality in nursing home patients by almost five-fold. The most obvious signs of secondary PEM include: (1) depletion of subcutaneous fat in the arms, chest wall, shoulders, or metacarpal regions; (2) wasting of the quadriceps and deltoid muscles; and (3) ankle or sacral edema. Bedridden or hospitalized malnourished patients have an increased risk of infection, sepsis, impaired wound healing, and death after surgery.

MORPHOLOGY

The main anatomic changes in PEM are (1) growth failure, (2) peripheral edema in kwashiorkor, and (3) loss of body fat and atrophy of muscle, more marked in marasmus.

The **liver** in kwashiorkor, but not in marasmus, is enlarged and fatty; superimposed cirrhosis is rare. In kwashiorkor (rarely in marasmus) the **small bowel** shows a decrease in the mitotic index in the crypts of the glands, associated with mucosal atrophy and loss of villi and microvilli. In such cases concurrent loss of small intestinal enzymes occurs, most often manifested as disaccharidase deficiency. Hence, infants with kwashiorkor initially may not respond well to full-strength, milk-based diets. With treatment, the mucosal changes are reversible.

The **bone marrow** in both kwashiorkor and marasmus may be hypoplastic, mainly as a result of decreased numbers of red cell precursors. The peripheral blood commonly reveals mild to moderate anemia, which is often multifactorial in origin; nutritional deficiencies of iron, folate, and protein, as well as the suppressive effects of infection (anemia of chronic disease) may all contribute. Depending on the predominant factor, the red cells may be microcytic, normocytic, or macrocytic.

The **brain** in infants who are born to malnourished mothers and who suffer PEM during the first 1 or 2 years of life has been reported by some to show cerebral atrophy, a reduced number of neurons, and impaired myelinization of white matter.

Many other changes may be present, including (1) thymic and lymphoid atrophy (more marked in kwashiorkor than in marasmus), (2) anatomic alterations induced by intercurrent infections, particularly with all manner of endemic worms and other parasites, and (3) deficiencies of other required nutrients such as iodine and vitamins.