Iron

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Gastrointestinal Bleeding
Anemia
Failure to Thrive

Iron, the most abundant trace mineral, is used in the synthesis of hemoglobin, myoglobin, and enzyme iron. Body iron content is regulated primarily through modulation of iron absorption, which depends on the state of body iron stores, the form and amount of iron in foods, and the mixture of foods in the diet. There are two categories of iron in food. The first is *heme iron,* present in hemoglobin and myoglobin, which is supplied by meat and rarely accounts for more than one fourth of the iron ingested by infants. The absorption of heme iron is relatively efficient and is not influenced by other constituents of the diet. The second category is **nonheme iron,** which represents the preponderance of iron intake consumed by infants and exists in the form of iron salts. The absorption of nonheme iron is influenced by the composition of consumed foods. Enhancers of nonheme iron absorption are ascorbic acid, meat, fish, and poultry. Inhibitors are bran, polyphenols (including the tannates in tea), and phytic acid, a compound found in legumes and whole grains. The percent intestinal absorption of the small amount of iron in human milk is 10%; 4% is absorbed from iron-fortified cow's milk formula and from iron-fortified infant dry cereals.

In a normal term infant, there is little change in total body iron and little need for exogenous iron before 4 months of age. Iron deficiency is rare in term infants during the first 4 months, unless there has been substantial blood loss (see Chapter 62). After about 4 months of age, iron reserves become marginal, and, unless exogenous sources of iron are provided, the infant becomes progressively at risk for anemia as the iron requirement to support erythropoiesis and growth increases (see Chapter 150). Premature or low birth weight infants have a lower amount of stored iron because significant amounts of iron are transferred from the mother in the third trimester. In addition, their postnatal iron needs are greater because of rapid rates of growth and when frequent phlebotomy occurs. Iron needs can be met by supplementation (ferrous sulfate) or by iron-containing complementary foods. Under normal circumstances, iron-fortified formula should be the only alternative to breast milk in infants younger than 1 year of age. Premature infants fed human milk may develop iron deficiency anemia earlier unless they receive iron supplements. Formula-fed preterm infants should receive iron-fortified formula.

In older children, iron deficiency may result from inadequate iron intake with excessive cow's milk intake or from intake of foods with poor iron bioavailability. Iron deficiency also can result from blood loss from such sources as menses or gastric ulceration. Iron deficiency affects many tissues (muscle and central nervous system) in addition to producing anemia. **Iron deficiency** and **anemia** have been associated with lethargy and decreased work capacity and **impaired neurocognitive development,** the deficits of which may be irreversible when onset is in the first 2 years of life.

The diagnosis of iron deficiency anemia is established by the presence of a microcytic hypochromic anemia, low serum ferritin levels, low serum iron levels, reduced transferrin saturation, normal to elevated red blood cell width distribution, and enhanced iron-binding capacity. The mean corpuscular volume and red blood cell indices are reduced, and the reticulocyte count is low. Iron deficiency may be present without anemia. Clinical manifestations are noted in Table 31-4.

Treatment of iron deficiency anemia includes changes in the diet to provide adequate iron and the administration of 2 to 6 mg iron/kg/24 hr (as ferrous sulfate) divided bid or tid. Reticulocytosis is noted within 3 to 7 days of starting treatment. Oral treatment should be continued for 5 months. Rarely, intramuscular or intravenous iron therapy is needed if oral iron cannot be given. Parenteral therapy carries the risk of anaphylaxis and should be administered according to a strict protocol, including a test dose.

Zinc

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Diarrhea
Vesicles and Bullae

Zinc is the second most abundant trace mineral and is important in protein metabolism and synthesis, in nucleic acid metabolism, and in stabilization of cell membranes. Zinc functions as a cofactor for more than 200 enzymes and is essential to numerous cellular metabolic functions. Adequate zinc status is especially crucial during periods of growth and for tissue proliferation (immune system, wound healing, skin and gastrointestinal tract integrity); physiologic functions for which zinc is essential include normal growth, sexual maturation, and immune function.

Dietary zinc is absorbed (20% to 40%) in the duodenum and proximal small intestine. The best dietary sources of zinc are animal products, including human milk, from which it is readily absorbed. Whole grains and legumes also contain moderate amounts of zinc, but phytic acid inhibits absorption from these sources. On a global basis, poor bioavailability secondary to phytic acid is thought to be a more important factor than low intake in the widespread occurrence of zinc deficiency. Excretion of zinc occurs from the gastrointestinal tract. In the presence of ongoing losses, such as in chronic diarrhea, requirements can drastically increase.

Zinc deficiency dwarfism syndrome was first described in a group of children in the Middle East with low levels of zinc in their hair, poor appetite, diminished taste acuity, hypogonadism, and short stature. Zinc supplementation reduces morbidity and mortality from **diarrhea** and **pneumonia** and enhances growth in developing countries. Mild to moderate zinc deficiency is considered to be highly prevalent in developing countries, particularly in populations with high rates of **stunting**. Mild zinc deficiency occurs in older breastfed infants without adequate zinc intake from complementary foods or in young children with poor total or bioavailable zinc intake in the general diet. A high infectious burden also may increase the risk of zinc deficiency in developing countries. Acute, acquired severe zinc deficiency occurs in patients receiving