Anemia is the commonest manifestation of malnutrition, which is so rampant in India and other economically under-developed countries. Of the various types of anemias met with in infants and children, iron-deficiency anemia is the most frequent. It may be mild enough to go unnoticed, or so severe as to kill.

Although iron-deficiency anemia is seen frequently in other age groups as well, its occurrence predominantly in children is due to several factors peculiar to the young. Rapid acceleration of growth makes large demands on iron. Minor disturbances, such as mild infections, diarrhoea and vomiting affect the delicate and labile functional balance of iron metabolism, so as to result in deficiency. In the very young infants, factors of maternal nutrition, and the acquisition of iron stores from the mother are important. Loss of foetal blood at the time of delivery or ante-partum, and premature birth results in deficient iron stores.

The most important single causative factor is reduced intake of iron. Milk is notoriously deficient in this element, and being the chief and often the sole source of nutrition for the infant, it produces anemia during the latter half of the first year and the second year. Recurrent infections, especially of the gastrointestinal tract, with resultant anorexia, faulty absorption and increased needs are additive factors. Blood loss from any site (due to trauma, bleeding disorder or scurvy) reduces the body haemoglobin content. Hookworm infestation is the most frequent etiologic reason in this latter group.

The clinical features of iron-deficiency anemia depend on the rapidity of production, the basic cause of the deficiency, effect of iron loss on epithelial tissues and any complicating factors. In children with mild anemia, vague symptoms of failure to thrive, irritability, asthenia and anorexia may be complained of. In more severe cases, hemodynamic changes occur, and palpitation, exertional dyspnea and oedema may be seen. In the severely anemic children, the heart may be enlarged, there are loud precordial murmurs which may be conducted to the axilla and the back, and decompensation of the myocardium.
may occur. Anginal features are sometimes seen. Cardio-respiratory distress may be serious enough to be fatal.

Besides marked pallor, changes in the nails indicate iron-deficiency anemia. Flattening and later on spooning (platynychia and koilonychia), brittleness and thinning of the nails takes place. The epithelium of mouth, tongue and esophagus is affected, producing superficial stomatitis, glossitis and dysphagia. The hair loses lustre, and becomes dry and fragile. Hepatosplenomegaly is commonly encountered, and is due to several factors—infections, extra-medullary hematopoiesis, congestive cardiac failure, etc. Since the infants’ bone marrow is almost exclusively red, there is little opportunity of using any reserves in the face of a stress. The spleen and liver, therefore, take over the burden of producing more cellular elements for blood. Gastric achlorhydria, impaired absorption of xylose and vitamin A, steatorrhoea, chronic duodenitis and muscle atrophy have been shown in such children. The presence of milk precipitins probably reflects absorption of whole milk proteins across the altered gastrointestinal epithelium. Gastric atrophy has been demonstrated historically.

Examination of the peripheral blood film reveals the characteristic microcytic hypochromic picture. The red cells are small, pale and deformed. Depending upon the degree of anemia, the M.C.V. ranges from 60-80 C. microns, and M.C.H.C. 15-30 per cent. Marked variation in size, shape, content and distribution of haemoglobin may be observed. Many target cells, and a few erythrocytes exhibiting basophilic stippling may be encountered. An occasional normoblast in the peripheral blood indicates rapid turn over of cells from the marrow. Reticulocyte count is normal, but may be increased or reduced. Osmotic fragility is slightly reduced. The bone-marrow shows erythroid hyperplasia, with asynchronous maturation of the nucleus and haemoglobin. A paucity of siderocytes can be demonstrated with appropriate stains.

Biochemical investigations show a reduction in serum iron level to 15-50 microgram/100 ml, and increase in total iron binding capacity to 400-450 microgram/100 ml. There is often a hypercupremia and increased protoporphyrin content of the red cells.

While considering the iron-deficiency anemias, it is useful to keep in mind other conditions producing hypochromia of red cells. These are—thalassemia and other haemoglobinopathies, pyridoxin (vitamin B6) deficiency, anemias of infection, toxic agents like lead, and deficiency of rare metals such as copper.

The platitude ‘prevention is better than cure’ is most appropriately suited for iron-deficiency. Prophylactic administration of iron to infants beyond 3 months, and to those under the stress of infection, rapid growth and blood loss should be given. Depending upon the age and built, 10-50 mg. of elemental iron, besides dietary articles known to contain a large amount of iron, would suffice.