IRON deficiency anemia in adults, when not caused by obvious blood loss, is frequently called "idiopathic hypochromic anemia." This term erroneously implies that the cause of the iron deficiency is unknown and obscure. While it is certainly true that many aspects of iron metabolism are still poorly understood, the pathogenesis of iron deficiency has become clear enough that the "idiopathic" designation no longer applies. Objection to the term "idiopathic hypochromic anemia" is not prompted by an academic interest in nomenclature, but by the observation that many physicians are led to believe that there is something mysterious about the development of iron deficiency in some adults and fail, therefore, to search sufficiently for the discernible causes. "Chronic hypochromic anemia" is a simpler, more accurate term.

According to all available evidence, the human organism has very little ability to rid itself of iron through ordinary excretory channels: its major losses of iron are through hemorrhage and pregnancies. Hence, it follows that once the body has achieved adult size without showing iron deficiency it will not become iron-deficient unless it is drained of its iron by repeated pregnancies or chronic hemorrhage. Poor diet or poor absorption from the intestinal tract for any reason may, to be sure, accelerate development of the deficiency. When achlorhydria is present, for instance, there is almost certainly less ionization of the iron in food and more prompt precipitation of iron in the small intestine. Rapid intestinal motility and steatorrhea probably also cause defective assimilation. But while an inadequate diet and poor absorption frequently contribute to the pathogenesis of iron deficiency, they do not seem able to precipitate its development unless iron is also lost from the body. The loss may come from repeated pregnancies, from chronic hemorrhage, or even from normal menstrual blood flow. No carefully studied patient has ever been shown to provide an exception to these statements. In our own experience we certainly have seen none, even though it has sometimes been necessary to continue observations for months in order to detect intermittent bleeding from the genito-urinary or gastro-intestinal tracts.

While never proved, it is conceivable that exceptions may be found in regions where the diet is so poor in iron that the population exists in precarious iron balance, because even though the daily loss of iron is small, some loss does occur. The fecal excretion of iron may be as great as 0.3 to 0.5 mg. per day, perspiration and urine contain measurable quantities, the cells desquamated from epithelial surfaces and leukocytes discharged from body orifices have small amounts of the metal, and there is loss in hair growth. It is conceivable that diets may be so deficient or intestinal absorption so poor that compensation is not made for this small excretion. Even so, if iron deficiency is brought about in this way, there is no justification for the term "idiopathic."

If other exceptions do exist, if there are patients who excrete unusually large
amounts of iron, or if there are other obscure examples of primary disturbances of iron metabolism per se, it is to these instances that the designation "idiopathic" should be applied. Such abnormalities have never been recognized; if they are observed, it would be highly important to define them as precisely as possible. Technics are now sufficiently good to make this possible; the resultant information would constitute a significant contribution to the understanding of iron metabolism.

But for the designation of iron deficiency anemia as it is now known to occur in adults, the term "idiopathic hypochromic anemia" is confusing and should be abandoned.

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EDITORIAL: "IDIOPATHIC" HYPOCHROMIC ANEMIA—AN OBITUARY

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