Disorders Characterized by Microcytosis.



This can result from a lack of iron (deficiency or inflammation)

defects in heme synthesis (sideroblastic anemias), or defects in the production of hemoglobin protein (thalassemia).

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Features of the Thalassemias.

Table 1. Features of the Thalassemias.*				
Туре	Mean Corpuscular Volume	Hemoglobin	Findings on Electrophoresis	Other Features
	fl	g/dl		
eta-Thalassemia				
Major	50-75	<7	Increased hemoglobin A_2	Severe anemia
Intermedia	50-75	<9	Increased hemoglobin A_2	Target cells on smear
Minor	65–75	9–10	Increased hemoglobin A_2	Target cells on smear
lpha-Thalassemia				
Trait 1 ($\alpha \alpha / \alpha$ –)	80-85	12–14	Normal	
Trait 2 (α –/ α –) or ($\alpha \alpha$ /–)	65-75	12-13	Normal	
Hemoglobin H disease (α –/–)	60–69	9–8	Hemoglobin H	Hemolysis, splenomegaly
Hemoglobin Bart's (/-)			Hemoglobin H, hemoglobin Bart's	Hydrops fetalis
Hemoglobin E disease				
Heterozygous	80-85	12	Hemoglobin E present	Rare target cells on smear
Homozygous	70–79	11–12	Hemoglobin E predominant	Target cells on smear

* The normal range for mean corpuscular volume is 80 to 100 fl. The normal range for hemoglobin level is 13.5 to 17.5 g per deciliter in men and 12 to 16 g per deciliter in women.

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Mechanism of Anemia of Inflammation.



Figure 2. Mechanism of Anemia of Inflammation.

Normally, iron is absorbed in the gastrointestinal tract and is delivered to transferrin for transport to the developing red cells, with any excess stored in hepatocytes. In inflammatory states, decreased absorption of iron leads to reduced saturation of transferrin and impaired release of iron from storage, resulting in a lack of iron delivery to the developing red cells. These changes are mediated by hepcidin, which binds and inhibits ferroportin, the main iron-export protein. DMT1 denotes divalent metal transporter 1.







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