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## **CLASSIFICATION OF ANEMIAS IRON-DEFICIENCY ANEMIA**

**CHISINAU – 2020**

**Anemia is functionally best characterized by a hemoglobin concentration and red cell count below normal. Anemia is a disorder in which the patient suffers from tissue hypoxia, the consequence of a low oxygen-carrying capacity of the blood.**

### **CLASSIFICATION OF ANEMIAS:**

- I. Anemias due to the decreased red cell production,**
- II. Anemias due to the increased red cell destruction (hemolytic),**
- III. Anemias due to the acute blood loss.**

### **ANEMIAS DUE TO THE DECREASED RED CELL PRODUCTION:**

**iron deficiency anemia,  
megaloblastic anemias (vitamin B<sub>12</sub> deficiency, folate deficiency),  
aplastic anemias (including pure red cell aplasia),  
anemias of chronic disorders,  
renal anemia,  
anemia due to neoplastic infiltration of the bone marrow,  
myelodysplastic anemia.**

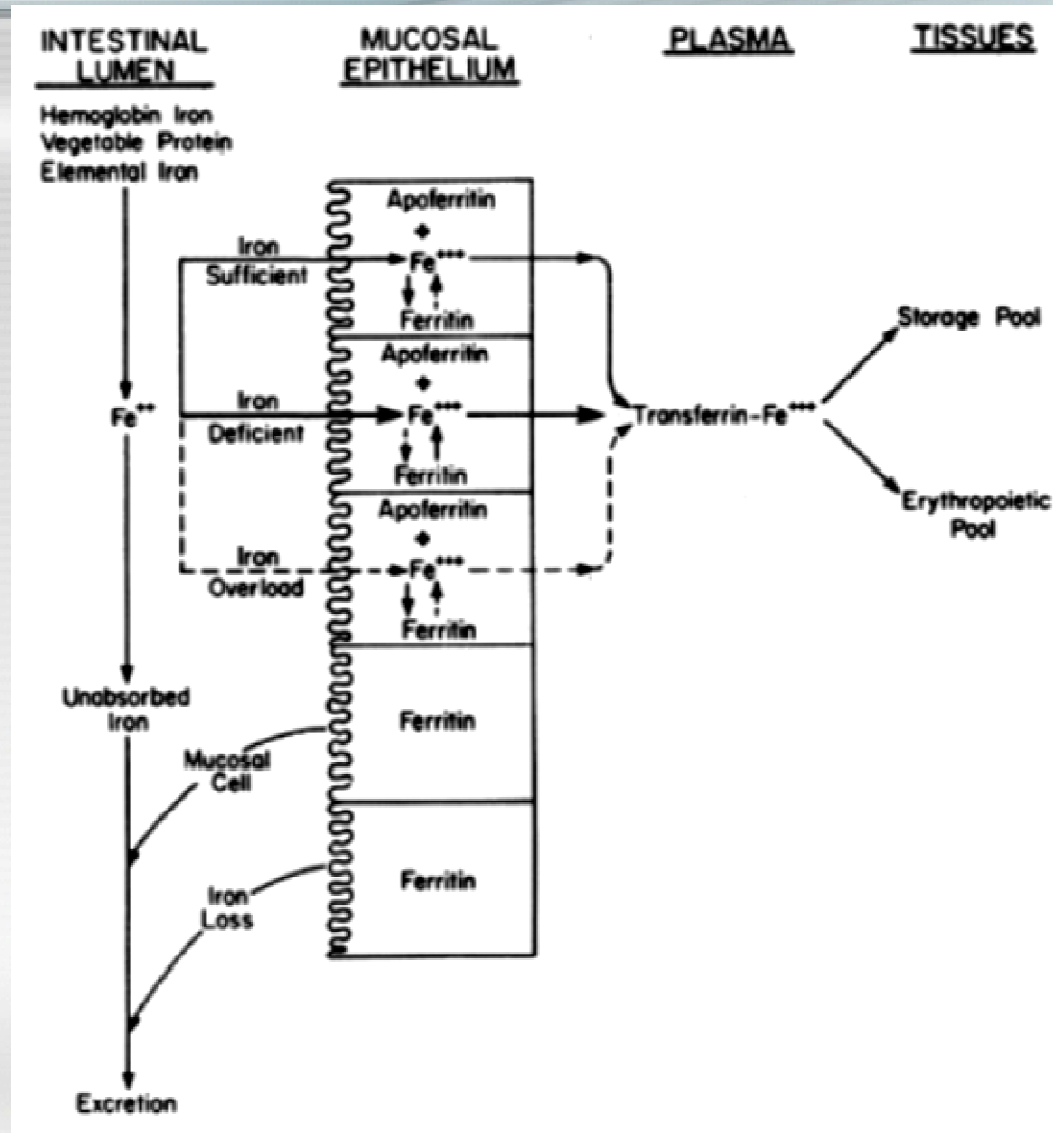
## ***DEGREE OF ANEMISATION***

| <b><i>Degree of anemia</i></b> | <b><i>Hemoglobin (g/l)</i></b> |
|--------------------------------|--------------------------------|
| <b>I</b>                       | <b>91—110</b>                  |
| <b>II</b>                      | <b>71—90</b>                   |
| <b>III</b>                     | <b>51—70</b>                   |

**Iron deficiency is the state in which the content of iron in the body is less than normal. It occurs in varying degrees of severity that merge imperceptibly into one another, and accounts 80 – 85% of all anemia cases. Iron deficiency anemia is the most advanced stage of iron deficiency, characterized by decreased or absent iron stores, low serum iron concentration, low transferrin saturation, and low hemoglobin concentration or hematocrit value.**

**In 2001 WHO/UNICEF estimated the number of anemic people worldwide to be a staggering two billion and that approximately 50% of all anemia could be attributed to iron deficiency. Data of WHO (1993) showed that approximately 44.5 % of preschoolers (3 – 6 years old ), 8 – 15 % of fertile women, 45 – 50 % of pregnant women, and 37 % of school-age children were anemic. Data from Russian Federation (1998) demonstrated that anemia had been diagnosed in 30% of children of up 2 years old, in 60% of pregnant women, and in 30% of fertile women.**

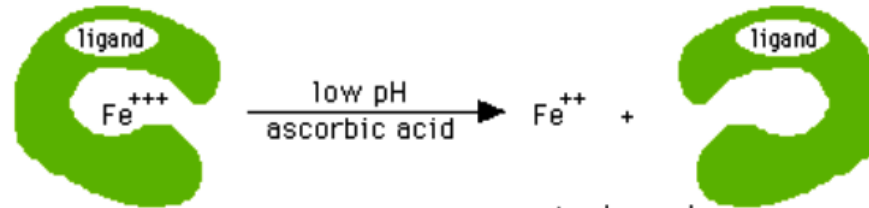
# SCHEME OF IRON ABSORPTION



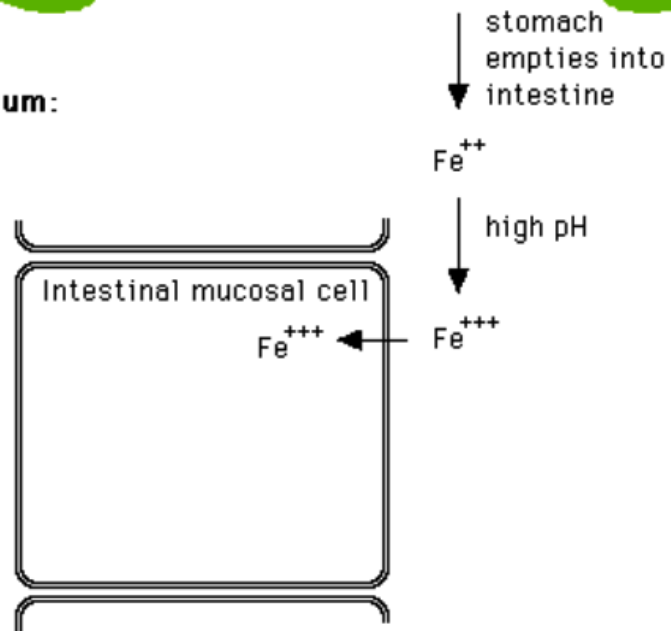
# SCHEME OF IRON ABSORPTION

## Iron Absorption -- Events in Stomach and Duodenum

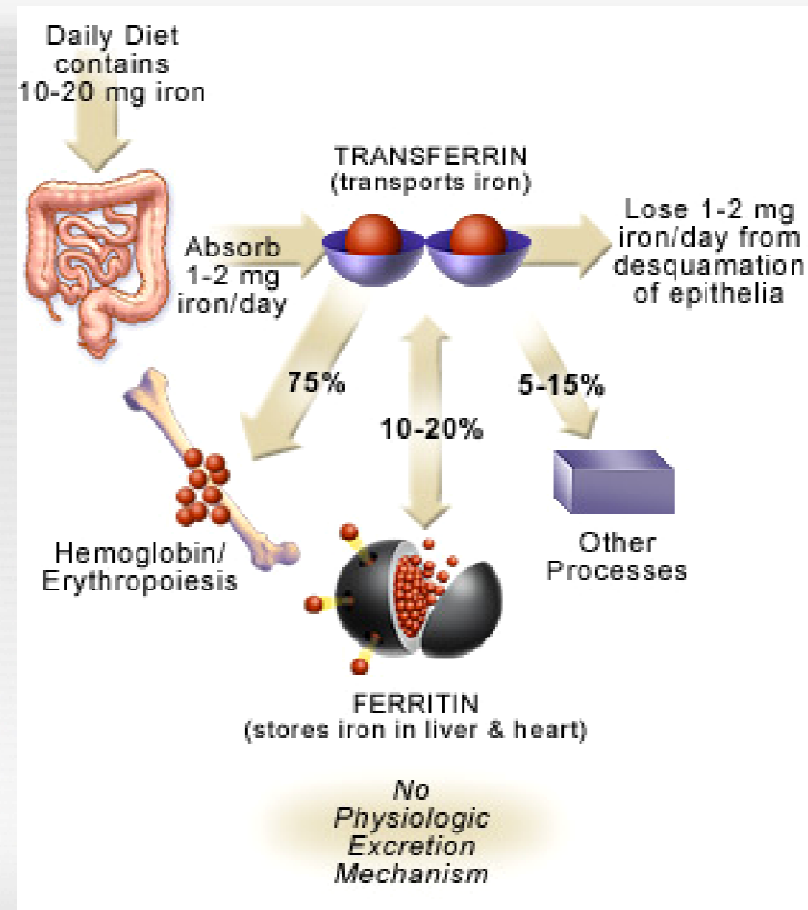
In the stomach:



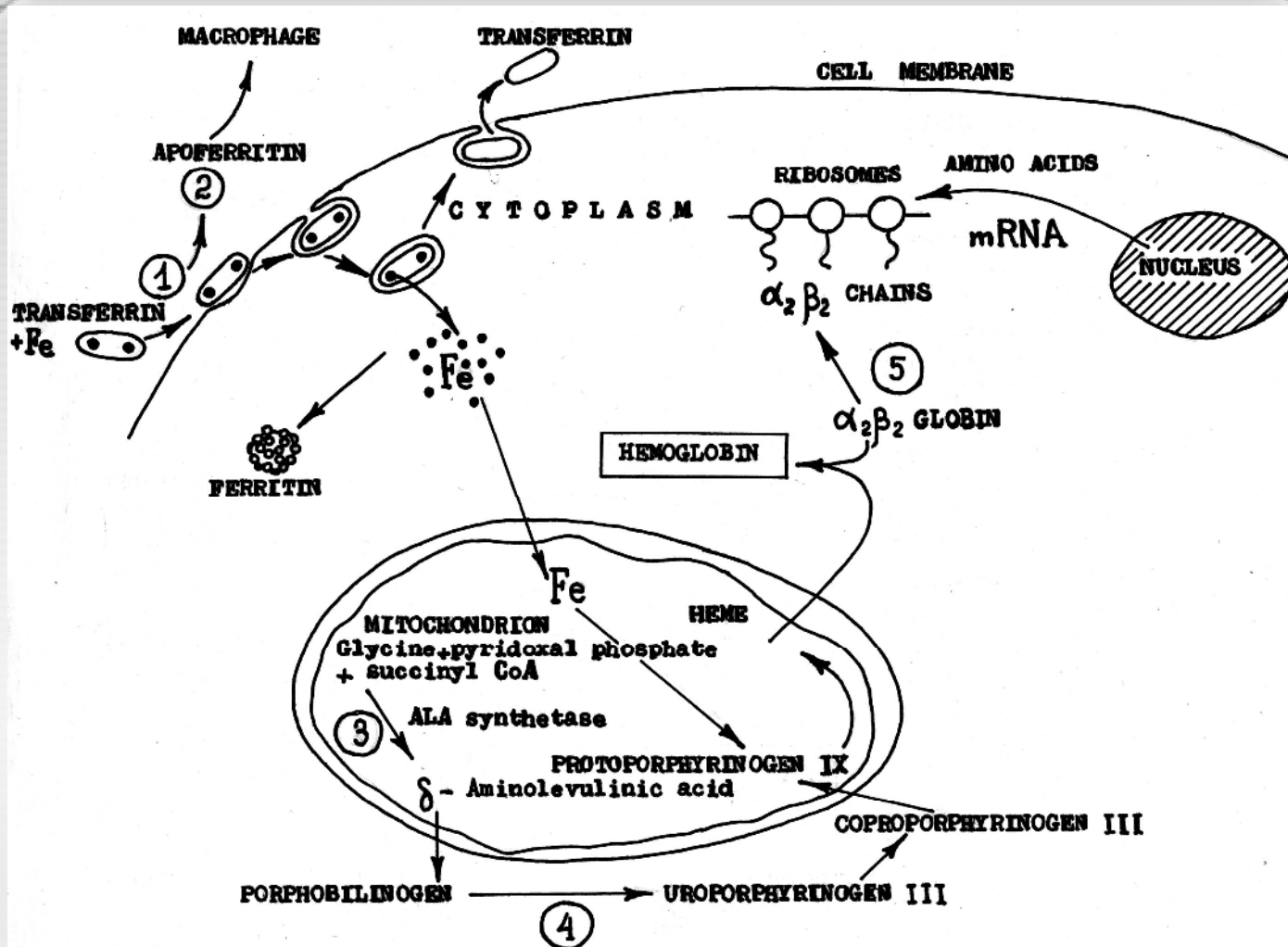
In the duodenum:



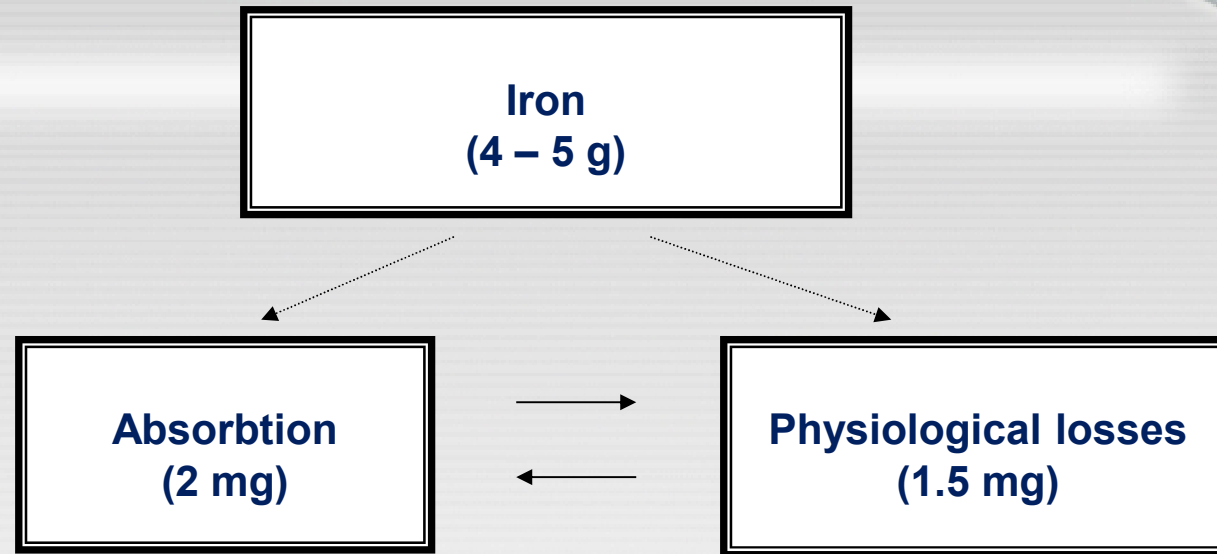
## SCHEME OF IRON METABOLISM



# SCHEME OF HEMOGLOBIN SYNTHESIS IN ERITROBLAST







### **Mechanisms of the development of iron deficiency**

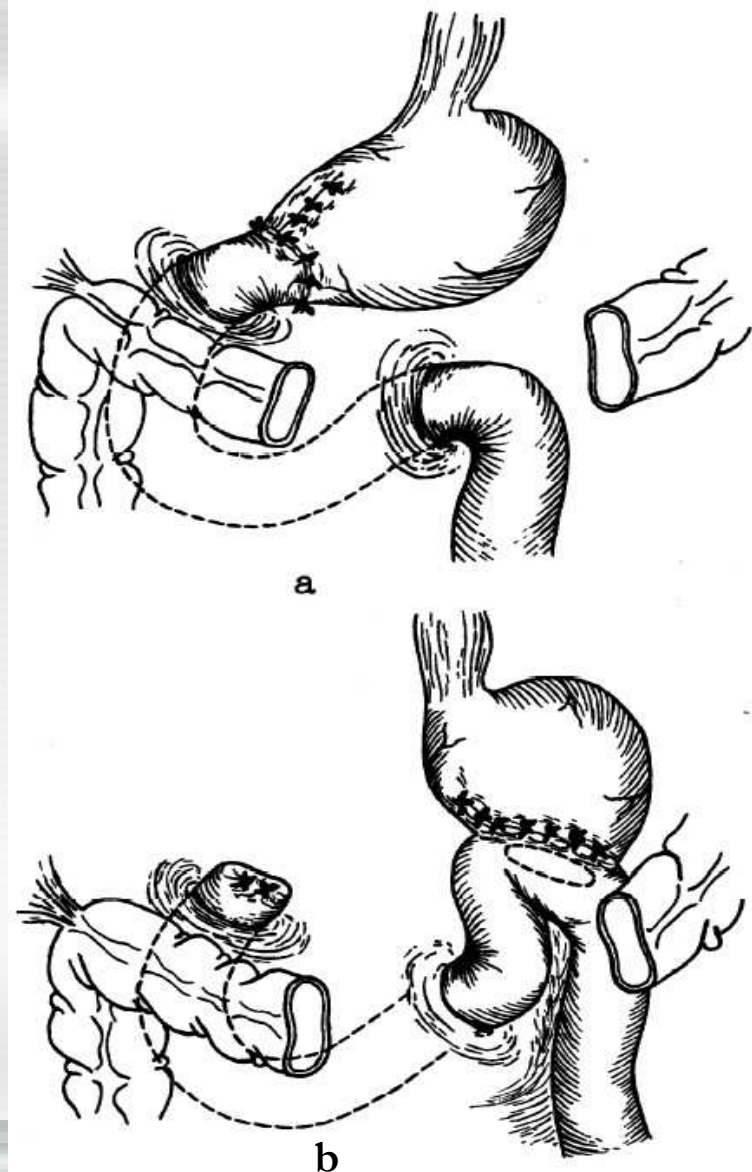
- **Dietary iron deficiency**
- **Additional iron requirements**
- **Increased iron losses**
- **Malabsorption of iron**

## CAUSES OF IRON DEFICIENCY AND IRON DEFICIENCY ANEMIA

(Iron Deficiency - Investigation and Management. Guidelines and Protocols Advisory Committee, British Columbia, Canada, 2010)

| Increased Requirements  | Decreased Intake  |
|---|---|
| <ul style="list-style-type: none"> <li>•Growing infants and children</li> <li>•Menstruating women</li> <li>•Pregnancy</li> <li>•Lactation</li> <li>•Multiparity</li> <li>•Parturition</li> </ul>  | <ul style="list-style-type: none"> <li>•Low socioeconomic status</li> <li>•Vegetarian diet</li> <li>•Lack of balanced diet or poor intake</li> <li>•Alcoholism</li> <li>•Elderly</li> <li>•High risk ethnic groups (<i>First Nations, Indo-Canadians</i> *)</li> </ul>  |
| Increased Loss  | Decreased Absorption  |
| <ul style="list-style-type: none"> <li>•Menorrhagia</li> <li>•GI bleeding</li> <li>•Regular blood donors</li> <li>•Post-operative patients with significant blood loss</li> <li>•Hematuria</li> <li>•Intestinal parasites (travel or immigration from an endemic area)</li> <li>•Intravascular hemolysis: hemoglobinuria</li> <li>•Extreme physical exercise (endurance athletes)</li> <li>•Pathological (hemolytic anemias)</li> </ul> | <ul style="list-style-type: none"> <li>•Dietary factors (<i>tannins, phytates in fibre, calcium in milk, tea, coffee, carbonated drinks</i>)</li> <li>•Upper GI Pathology:               <ul style="list-style-type: none"> <li>• Chronic gastritis</li> <li>• Gastric lymphoma</li> <li>• Celiac disease</li> <li>• Crohn's disease</li> </ul> </li> <li>•Medications that decrease gastric acidity or bind iron</li> <li>•Gastrectomy or intestinal bypass</li> <li>•Duodenal pathology</li> <li>•Chronic renal failure patients</li> </ul> |

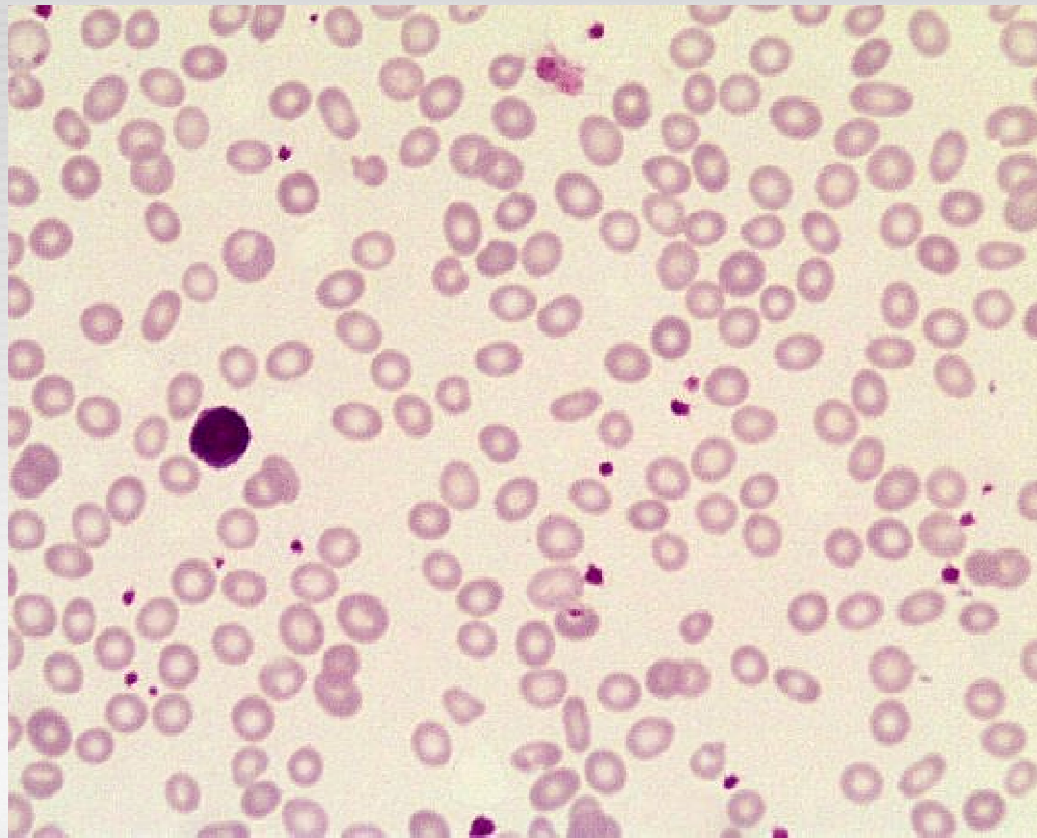
***DISTURBANCE OF IRON ABSORPTION  
AFTER BILLROTH II GASTRIC RESECTION***



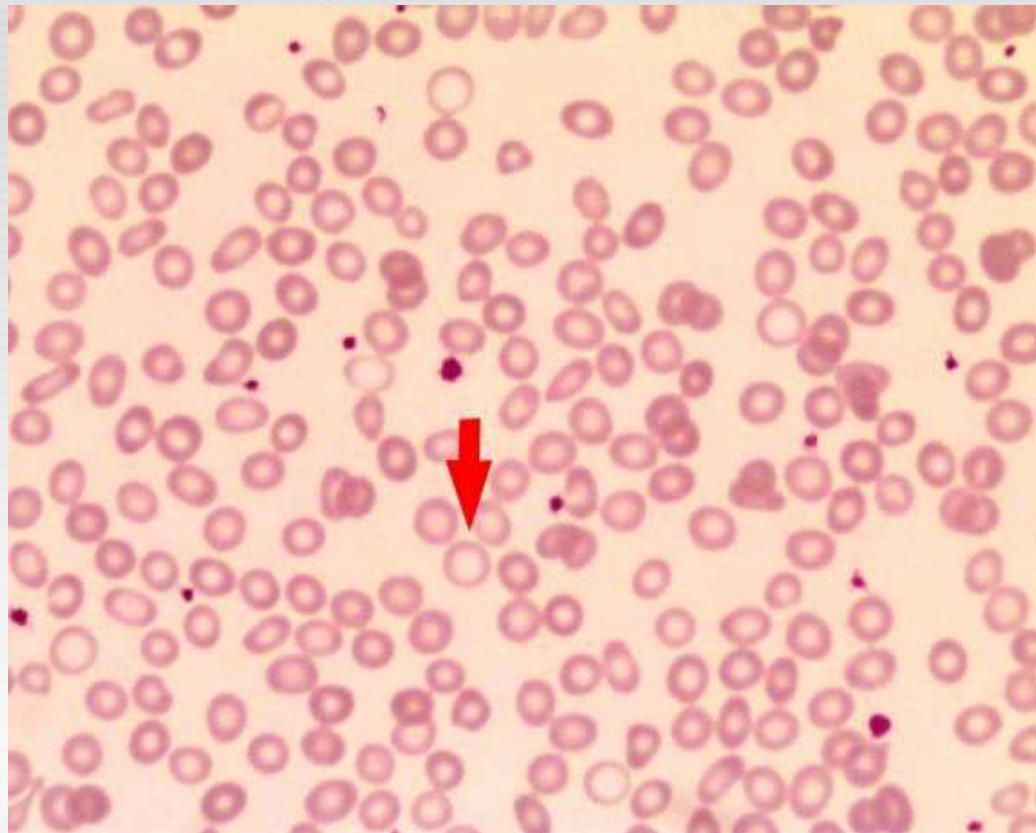
## ***EVOLUTIONAL STAGES OF IRON DEFICIENCY***

| <b><i>Stages of iron deficiency</i></b> | <b><i>Serum ferritin</i></b>         | <b><i>Plasma iron concentration</i></b> | <b><i>Hemoglobin</i></b>         |
|---|--------------------------------------|---|----------------------------------|
| <b><i>Prelatent deficiency</i></b>      | <b><i>Decreased</i></b>              | <b><i>Normal</i></b>                    | <b><i>Normal</i></b>             |
| <b><i>Latent deficiency</i></b>         | <b><i>Considerably decreased</i></b> | <b><i>Decreased</i></b>                 | <b><i>Normal</i></b>             |
| <b><i>Iron deficiency anemia</i></b>    | <b><i>Markedly decreased</i></b>     | <b><i>Decreased</i></b>                 | <b><i>Decreased (anemia)</i></b> |

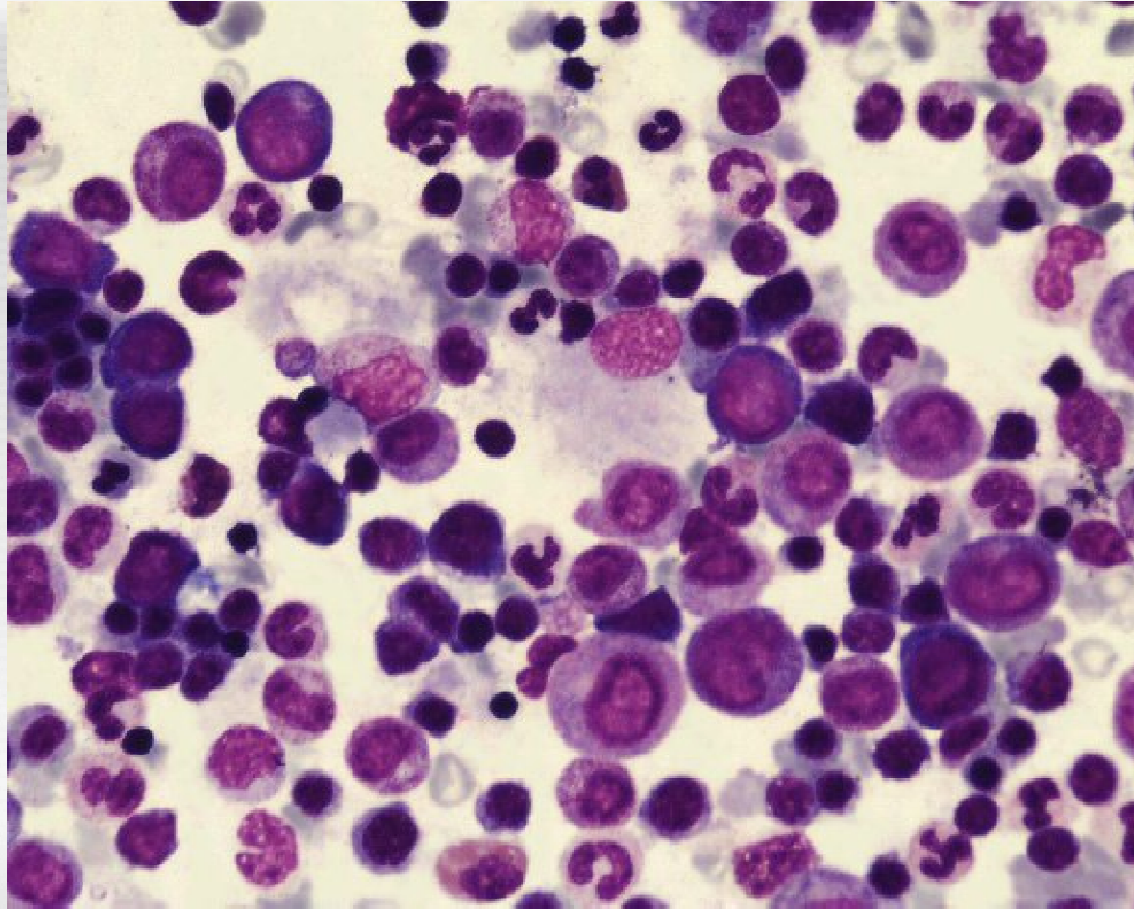
***BLOOD SMEAR IN IRON DEFICIENCY ANEMIA***



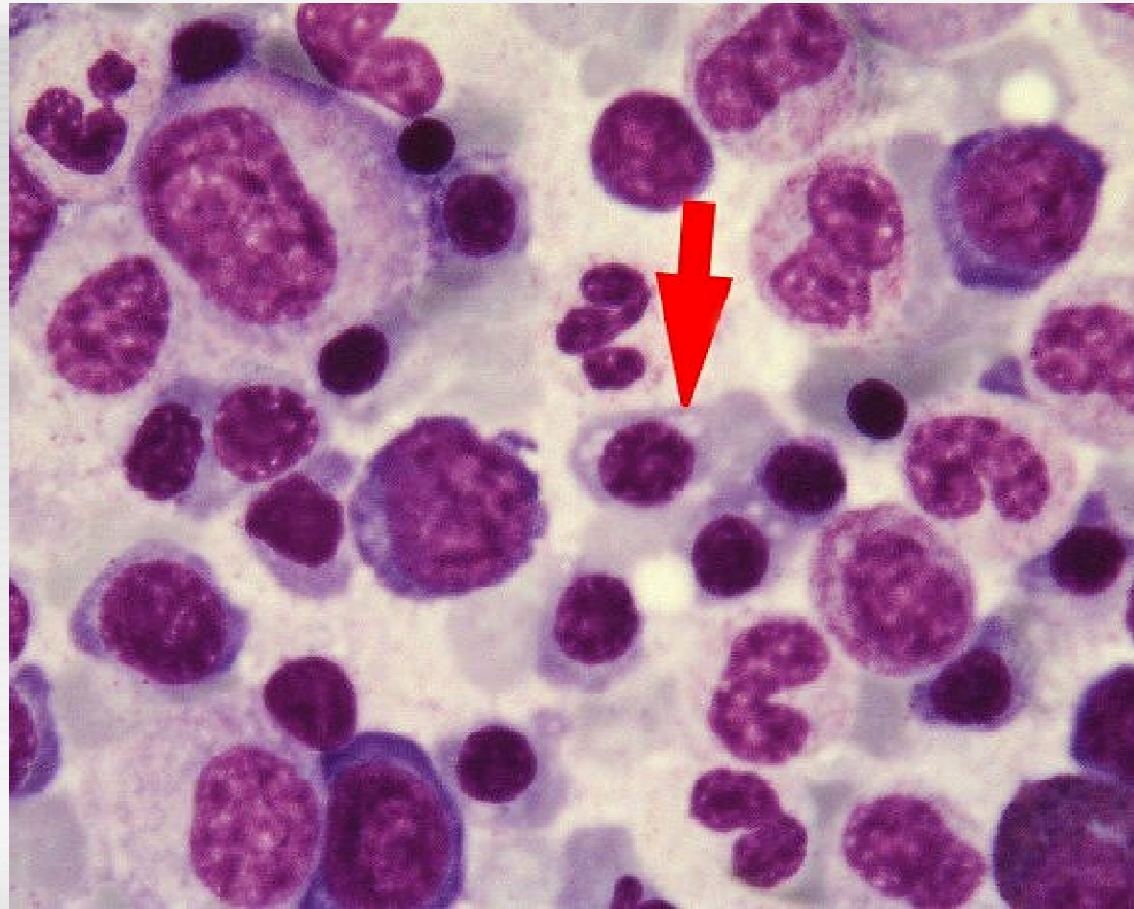
***BLOOD SMEAR IN IRON DEFICIENCY ANEMIA***



***BONE MARROW SMEAR IN IRON DEFICIENCY ANEMIA:  
NUMEROUS ERYTHROBLASTS***

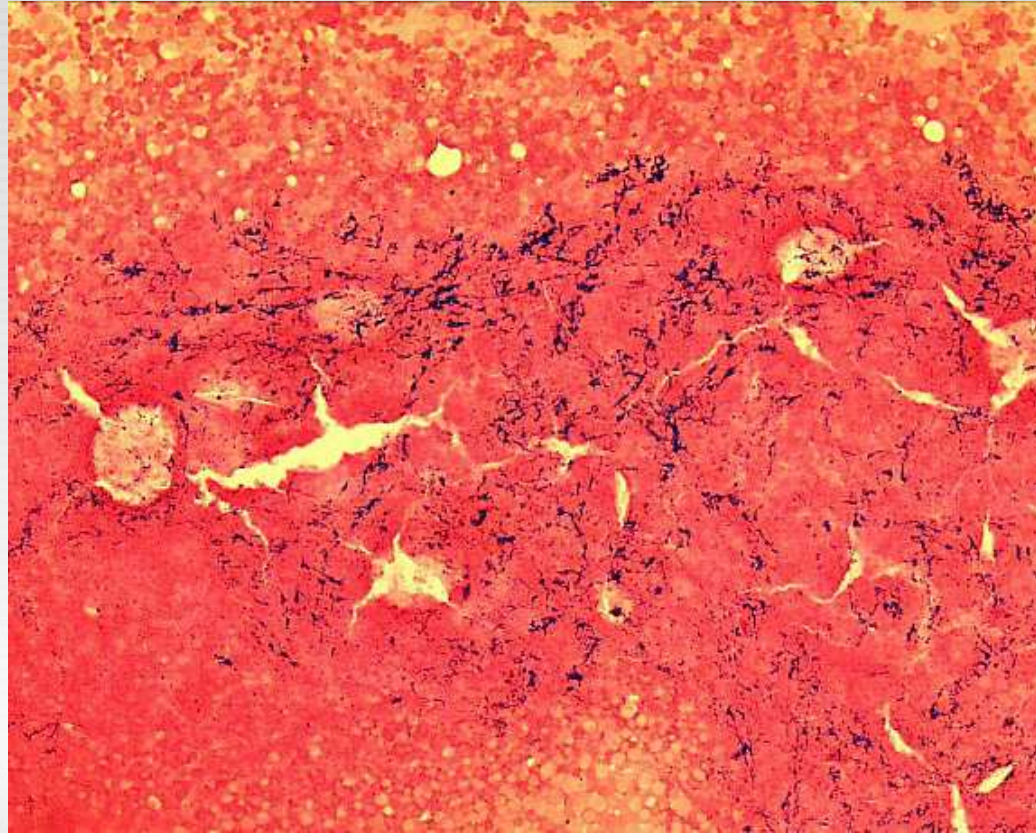


***BONE MARROW SMEAR IN IRON DEFICIENCY ANEMIA:  
DEFECTS OF HEMOGLOBINIZATION IN ERYTHROBLASTS***

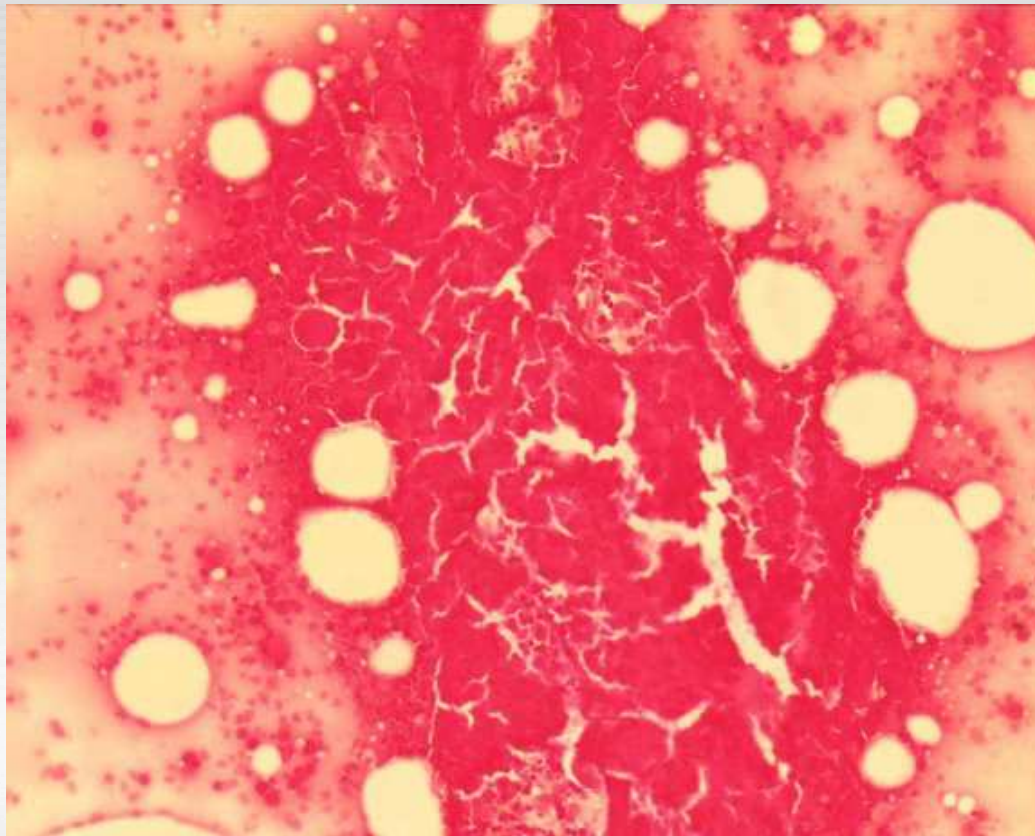




***DETECTION OF FREE IRON IN THE BONE MARROW (PERLS REACTION)***

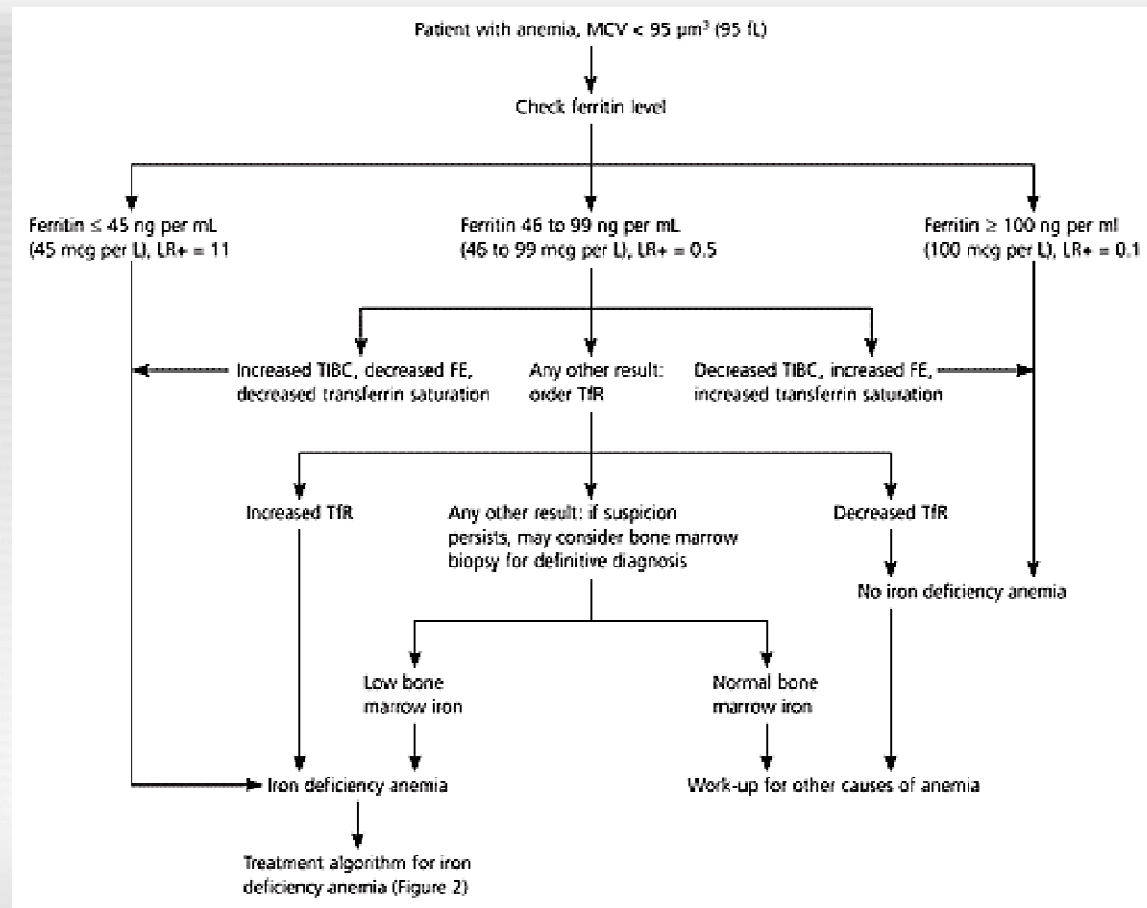


***PERLS REACTION IN IRON DEFICIENCY ANEMIA***



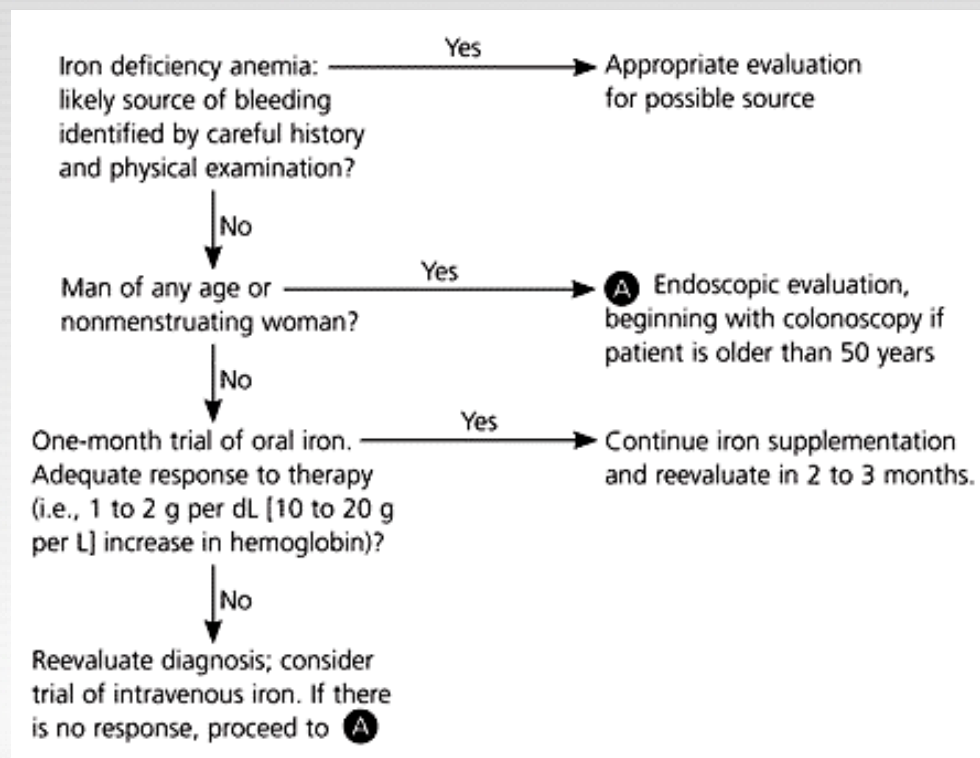
# DIAGNOSIS OF IRON DEFICIENCY ANEMIA

(Am Fam Physician, 2007 Mar 1;75 (5): 671-678)



# EVALUATION AND TREATMENT OF IRON DEFICIENCY ANEMIA

(Am Fam Physician, 2007 Mar 1;75 (5): 671-678)



## **LABORATORY DIFFERENTIATION OF IRON DEFICIENCY ANEMIA (IDA) VERSUS ANEMIA OF CHRONIC DISEASE (ACD)**

(Iron Deficiency - Investigation and Management. Guidelines and Protocols Advisory Committee; British Columbia, Canada, 2010)

| <b>Investigation</b>                                | <b>Results In</b> |             |                  |
|---|-------------------|-------------|------------------|
|   | <b>IDA</b>        | <b>ACD</b>  | <b>ACD + IDA</b> |
| <b>Serum Ferritin</b>                               | ↓                 | ↑           | ↓ or normal      |
| <b>Serum Iron</b>                                   | ↓                 | ↓           | ↓                |
| <b>Iron Binding Capacity</b>                        | ↑                 | ↓           | ↓ or low normal  |
| <b>Transferrin Saturation / Fraction Saturation</b> | ↓                 | ↓ or normal | ↓                |

## ***PRINCIPLES OF TREATMENT AND MONITORING OF IRON DEFICIENCY ANEMIA:***

(Iron Deficiency - Investigation and Management. Guidelines and Protocols Advisory Committee;  
British Columbia, Canada, 2010)

**The goals of management of iron deficiency anemia are to treat its underlying cause and restore normal levels of red blood cells, hemoglobin, and iron.**

**Oral iron therapy is usually the first-line therapy for patients with iron deficiency anemia. Commonly used oral iron preparations include: ferrous gluconate, ferrous fumarate, and ferrous sulfate. One preparation is not preferred over another; patient tolerance should be the guide.**

**The usual adult dose is 180 mg of elemental iron/day in divided doses. Therapeutic doses can range from 100 to 200 mg of elemental iron/day, depending on severity of symptoms, ferritin levels, age of the patient, and gastrointestinal side effects.**

**Iron intolerance is very common; Oral iron preparations may cause nausea, vomiting, dyspepsia, constipation, diarrhea or dark stools. Strategies to minimize these effects include: start at a lower dose and increase gradually over 4 to 5 days; giving divided doses or the lowest effective dose, or taking supplements with meals (*note: iron absorption is enhanced if supplements are taken on an empty stomach; however, it may not be tolerated*). Although sustained release iron preparations tend towards less gastrointestinal side effects, they may not be as effective as standard film coated products due to reduced/poor iron absorption.**

**Iron absorption can be decreased by various medications and supplements; space administration apart by at least 2 hours.**

**Iron absorption from pharmaceutical preparations can be enhanced by taking them on an empty stomach (at least 1.5 to 2 hours after a meal), with acidic juices or vitamin C, and not with other multivitamin, calcium, or antacid tablets.**

**Iron replacement therapy may begin as soon as iron deficiency is detected; however, it is essential to determine and correct the underlying causes of iron.**

## ***PRINCIPLES OF TREATMENT AND MONITORING OF IRON DEFICIENCY ANEMIA:***

Oral iron therapy in iron deficient anemia will increase hemoglobin by 10-20 g/L in 2 to 4 weeks. Order a Hematology Profile initially at 2 to 4 weeks to monitor response to replacement regimen.

Anemia will correct within 2 to 4 months if appropriate iron dosages are administered and underlying cause of iron deficiency is corrected.

Continue iron therapy an additional 4 to 6 months (adults) after the hemoglobin normalizes to replenish the iron stores. The frequency of subsequent monitoring depends upon the severity of the anemia, the underlying cause of the iron deficiency, and the clinical impact upon the patient.

If the patient's clinical status is compromised by moderate to severe anemia, consider admission to an acute care facility and blood transfusion. Once the patient is stable, iron replacement can be commenced.

Oral iron replacement is preferred to intravenous (IV) therapy. It is safer, more cost-effective, and convenient when compared to IV therapy. However, intravenous therapy ((iron dextran, sodium ferric gluconate, iron sucrose) may be substituted when there is: inadequate iron absorption, continued uncorrectable blood loss, noncompliance or intolerance to oral iron therapy, non-adherence, or a hemoglobin level less than 6 g per dL (60 g per L) with signs of poor perfusion in patients who would otherwise receive transfusion (e.g., those who have religious objections). Internal medicine/hematologist consultation is recommended.

Complete or partial failure of monitored iron therapy trial (in compliant patients) may be due to insufficient absorption or ongoing loss (e.g. hemorrhage) or both. It should be investigated appropriately. Intravenous iron preparations may be considered in these patients.

Intramuscular (IM) iron therapy is not recommended except in institutions with facility for treating anaphylactic reactions. Additional risks of IM iron therapy include unpredictable absorption and local complications (e.g. pain, staining of the skin, sarcoma formation).

*THANK YOU FOR YOUR ATTENTION!*

