The Mechanism of Iron Absorption

Iron deficiency anaemia is commonest of all anaemias all over the world affecting all of both sexes. Excessive loss of blood almost always results in this type of anaemia. Iron common natural element forming as much as 4 percent of the earth's crust. Small intestine plays to prevent the body from adequate absorption of iron whenever there is excessive loss of iron due to any cause. Treatment of iron deficiency can be more effective if the practitioner is aware of the knowledge of the mechanism of iron absorption.

Normal adult male body contains 4 to 5 gm. and adult female 3.5 to 4.0 gm of iron. The test amount of iron in the body is present in the form of haemoglobin which accounts to about 70 percent of the body iron. Most of the remainder is present as haemosiderin, globin, ferritin, and haem containing enzymes such as catalase, cytochrome peroxidase, thione oxidase etc. However, a small amount of metabolically most active form of iron is present in the plasma which constitutes about 0.1 percent of the total body iron. An adult male requires about 1.0 mg of iron daily. Requirement are highest during pregnancy. menstruating females, infants, children, and adolescents requires higher amount of iron. Normally, loss of iron from the body occurs in stool from shedding intestinal cells, bile and le-faden macrophages, urine, nail, hair and desquamated epithelial cells.

An average adult diet contains 10 to 20 mg of iron daily. About 10 percent of this dietary iron is absorbed normally in an average adult. In general the proportion of iron absorption decreases as the total amount of ingested iron increases and vice versa. A subject in the negative state of iron balance absorbs greater amount of ingested iron than a normal subject when both are provided with identical diet. Absorption of dietary iron also depends upon the type of diet. About 25 percent of the iron present in the liver or meat are absorbed whereas it is only about 5 percent from egg, wheat, or soya bean. Gastric acid and proteolytic enzymes act on dietary iron in the stomach and intestine resulting release of iron. During
this process greater proportion of dietary iron which is mainly... in the ferrous form is reduced to the ferric form. It is only this ferrous form that the body is able to absorb. Maximum absorption of iron is absorbed in the duodenum probably because the duodenal pH and redox potential are favourable. Iron is less well absorbed in the jejunum. Only a tiny amount of iron is absorbed from the stomach and the ileum. Because of this reason enteric coated tablets and sustained release preparations should not be used in the treatment of the iron deficiency anaemia. Since much of the iron is released past the duodenum where the body is able to absorb only a minimum amount of iron. The less undesirable side effects due to these preparations could be achieved by using ordinary iron preparations at a reduced dose.

Inorganic iron is better absorbed than dietary iron. The ferrous form is more soluble than the ferric form. The low pH and the acidity in the stomach helps to keep ingested in the ferrous state. Absorption of iron is enhanced by any factors that help to keep it in a soluble ferrous form. Likewise, chelating agents such as glucose, amino acids etc., chelates and these iron chelates are highly soluble and facilitate the attachment of iron to the intestinal mucosa. Factors favouring increased iron absorption includes iron deficiency in pregnancy, anoxia increased erythropoiesis, and pyridoxine deficiency state. Absorption of dietary iron is inhibited by the presence of phosphates and phytates in the diet, and by pancreatic secretions. Ingestion of alkalis converts the iron into insoluble ferric form and retards its absorption. Achlorhydria and hypochlorhydria also results in reduction of dietary absorption. Similarly iron absorption is usually decreased when the body is overloaded with iron, in acute and in chronic infections, and in conditions causing depressed erythropoiesis. Some conditions such as primary haemochromatosis, thalassemia, sideroblastic anaemia and iron-loading states absorption may be normal or even increased despite the presence of increased body iron stores.

Intestinal mucosa act to block iron absorption. This block is only partial and can be overcome when large doses of iron is presented to the intestine or when the body is in a state of iron balance. Transfer of iron across the mucosal cells normally occurs by an energy requiring process though this transfer may occur by the process of passive diffusion when higher doses of iron is available. The exact mechanism by which intestinal mucosa regulate the amount of iron absorption is still uncertain. Some iron passes from the mucosal cell to bind to transferrin in the plasma while the remainder is thought to combine with ferritin in the mucosal cell to form ferritin. It is thought that the mucosal cell has two roles. One is to favour absorption and the other to block further absorption once apoferritin is saturated. Recently it has been suggested that the sole function of the apoferritin-Fe

11 JNMA July-Sept. (1977)
Mechanism is to prevent iron absorption. According to this later suggestion little ferritin is present in the mucosal cell when the body is in the state of negative iron balance and the basal cells take up and transfer iron to the plasma as iron could not be trapped due to the chance of apoferritin. On the other hand the iron over loaded cells take up much less iron than iron deficient cells and iron is trapped as ferritin and lost when the cell is shed at the end of its life-span.

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