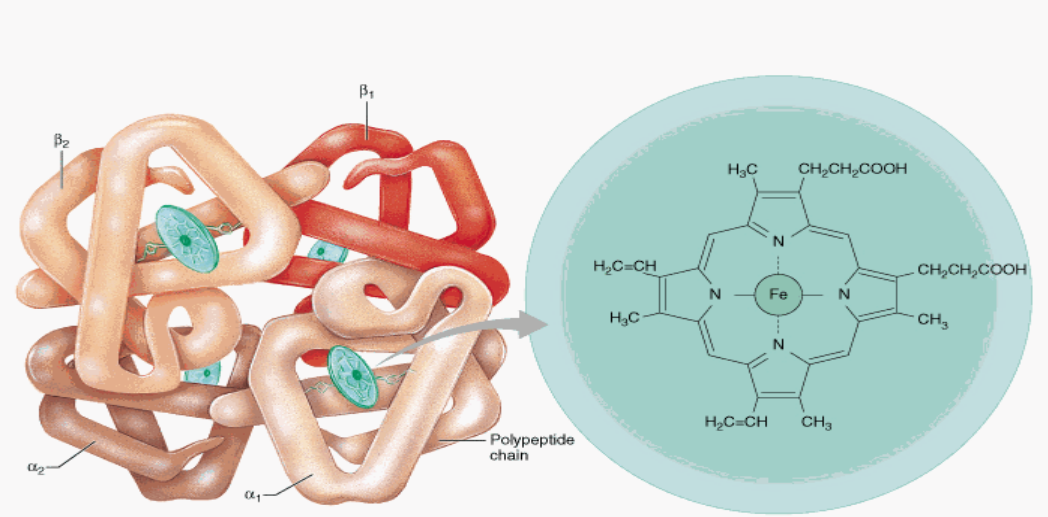
Hemogiobin and anemia and hematocrit(HCT) and erythrocyte sedimentation rate (ESR)



Hemoglubin synthesis

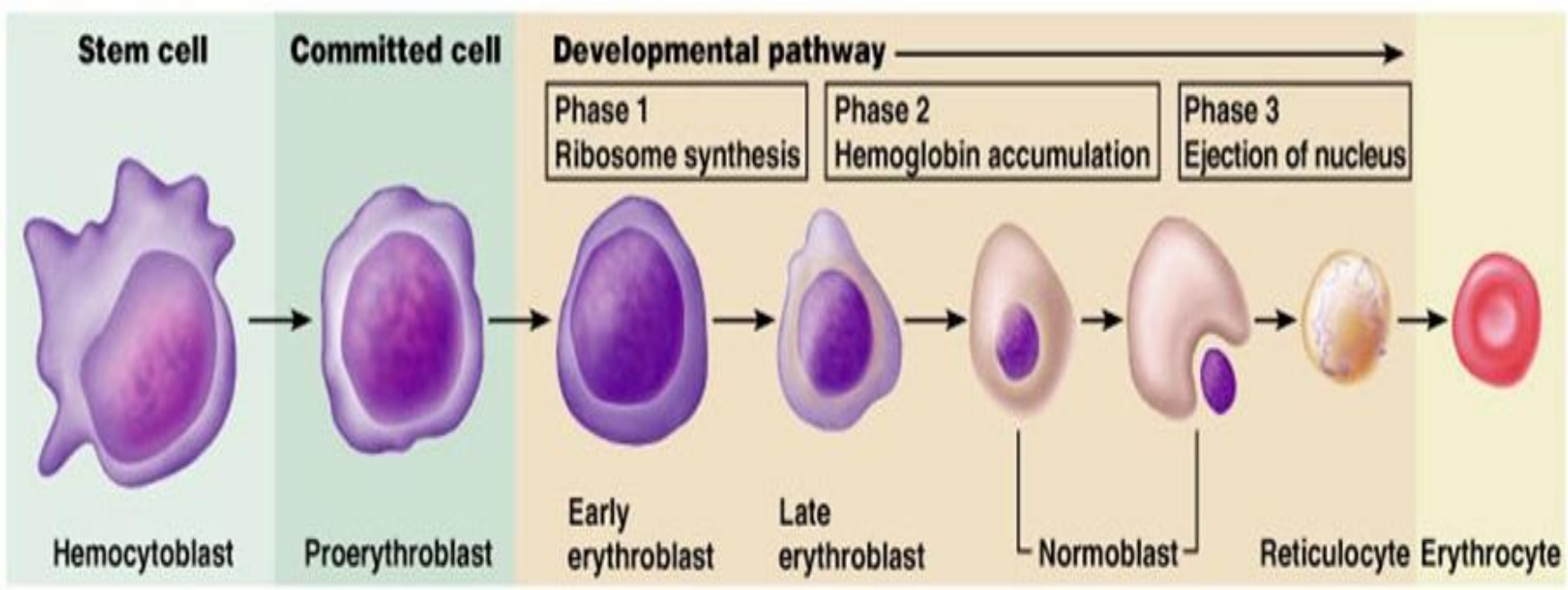
➢The circulation blood of normal adult contain about 750 mg of hemoglobin and of this about 7 – 8 gm are degraded daily.

➢**This amount has to be newly synthesized each day because:**

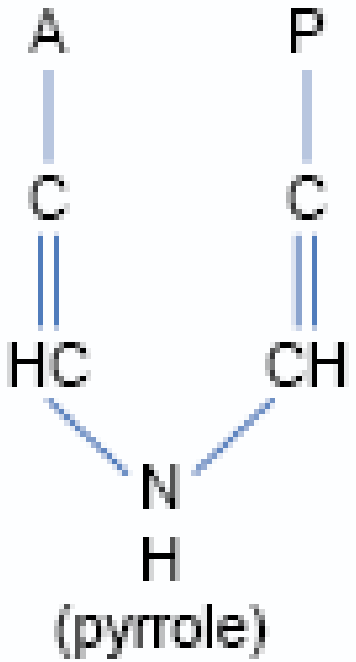
1) The globin part of Hemoglobin can be reutilized only after catabolism into its constituent amino acid
2) The free heam is broken down into bile pigment which is excreted.
3) Iron alone is reutilized in the synthesis of hemoglobin.

➢ **The rate at which haemoglobin is synthesized and which red cell are formed are related to :**

1) Oxygen content of the blood
2) Capacity of the blood to carry oxygen ,which in turn depend on the amount of circulating hemoglobin



immature RBC Reticulocytes develop and mature in the red Bone marrow and then circulate for about a day in the blood stream before developing into mature red blood cells. Like mature red blood cells, reticulocytes do not have a cell nucleouse They are called reticulocytes because of a reticular network of ribosomal RNA that becomes visible under a microscope with certain stains such as new methylene blue

I. 2 succinyl-CoA + 2 glycine → 

(pyrrole)

II. 4 pyrrole → protoporphyrin IX

III. protoporphyrin IX + Fe^{++} → heme

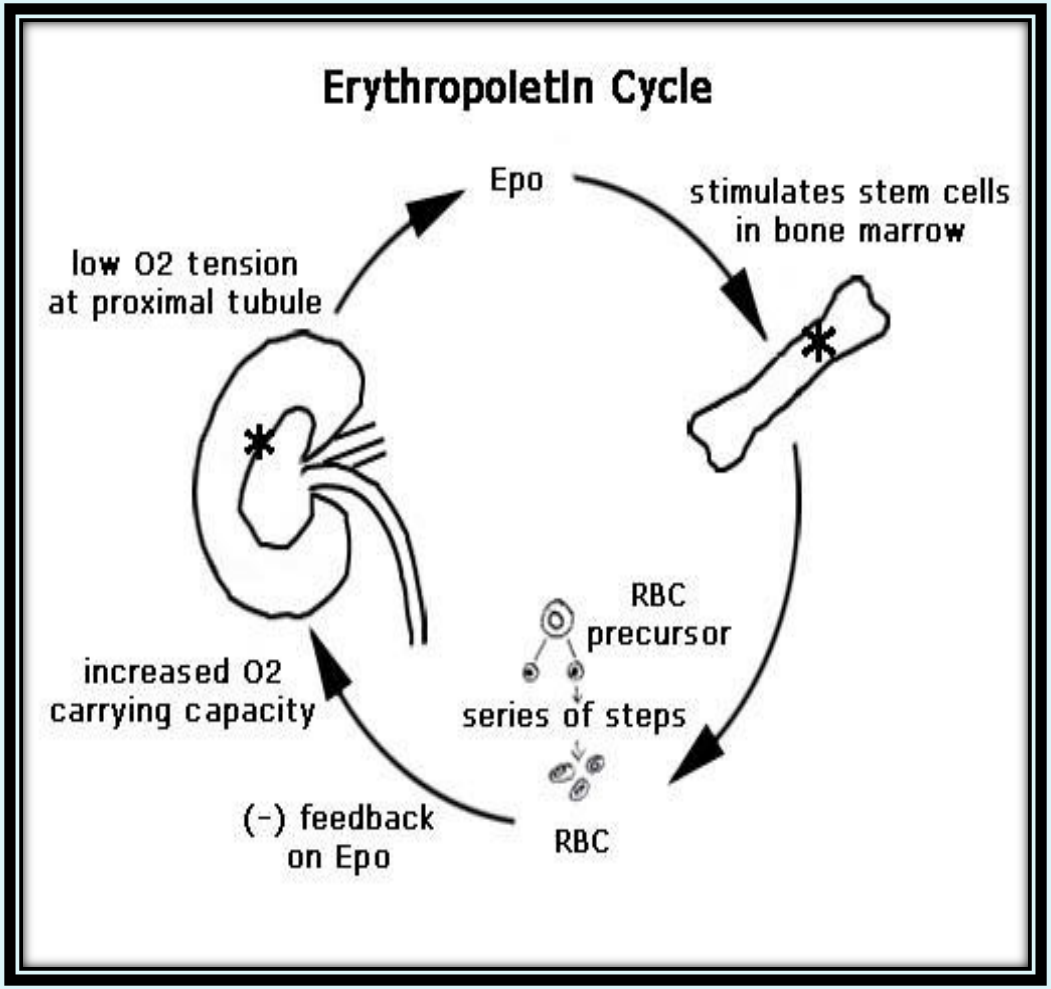
IV. heme + polypeptide → hemoglobin chain (α or β)

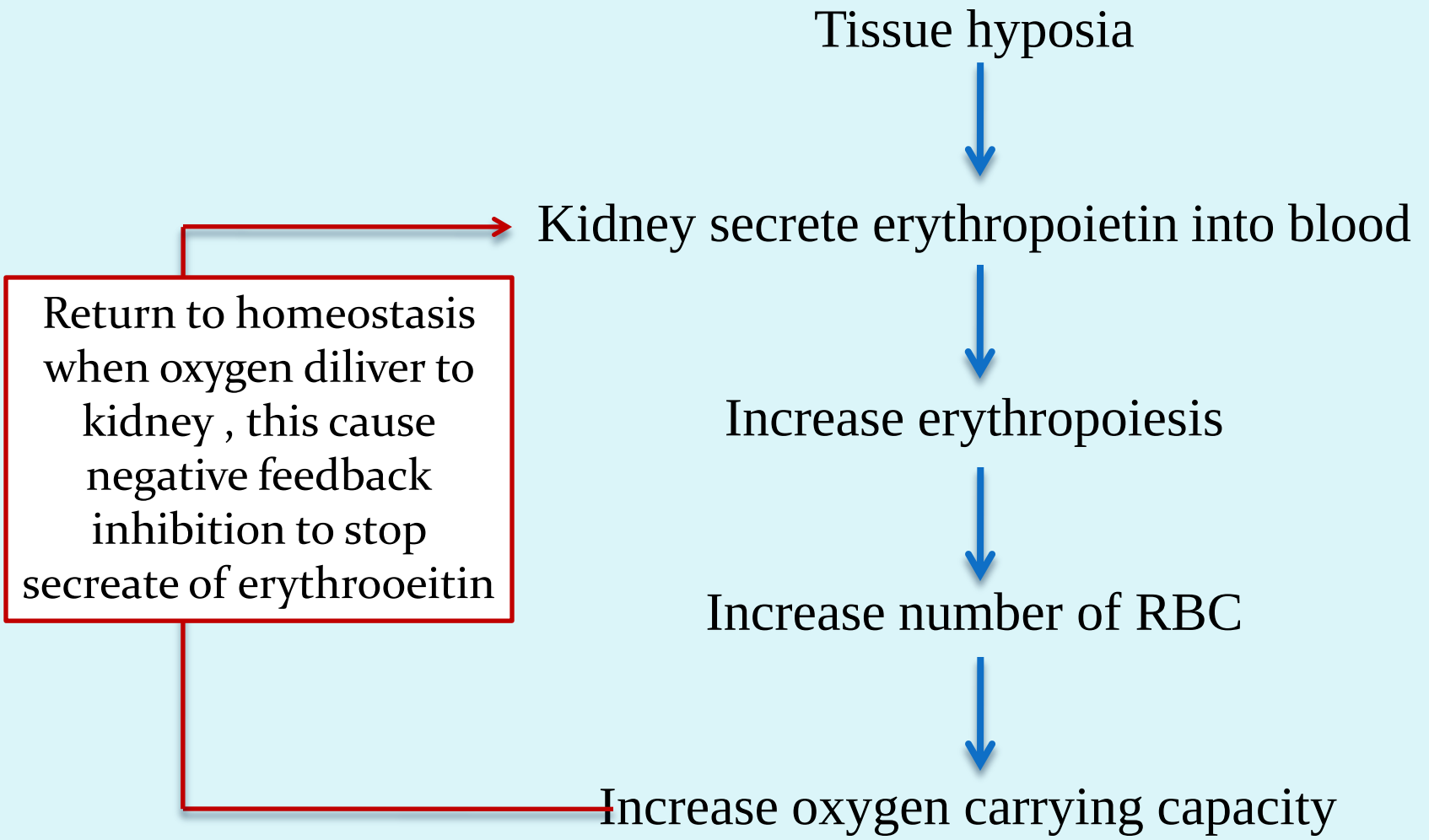
V. 2 α chains + 2 β chains → hemoglobin A

Regulation of hemoglobin synthesis:

Erythropoietin : is formed in kidney in response to decrease oxygen carrying capacity (hypoxia or anemia), in order to stimulate the erythropoiesis or hematopoiesis

*anoxia: means a total depletion in the level of oxygen an extreme form of hypoxia or "low oxygen




Tissue hyposia
Kidney secrete erythropoietin into blood
Increase erythropoiesis
Increase number of RBC
Increase oxygen carrying capacity
Return to homeostasis when oxygen diliver to kidney , this cause negative feedback inhibition to stop secreate of erythrooeitin

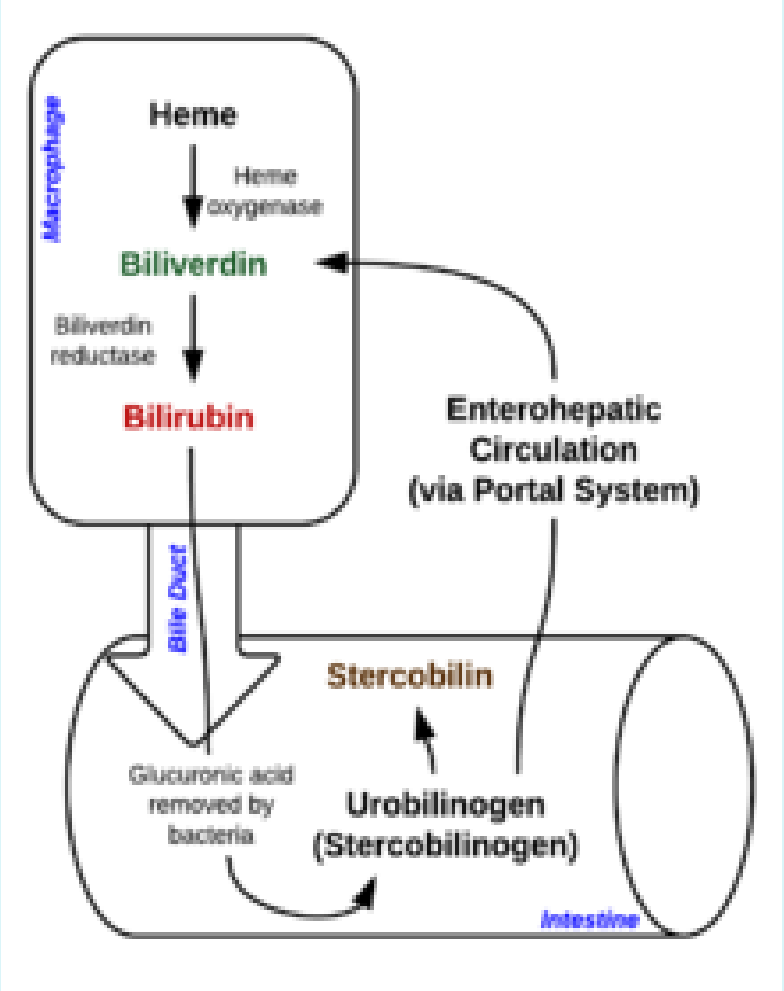
Haemoglobin catabolism:

❖ In the reticuloendothelial system erythrocytes are destroyed and haemoglobin is released.

❖Globin is separated from haem amd haematin is formed (the iron oxidized to feeric iron Fe+3)

❖The porphyrin ring is then opened and the iron is removed with formation of straight chain compund biliverdin which is converted to bilirubine by reduction

❖The iron and amino acid of the globin are retained but pyrrole ring are excreted as bilirubin.



The role of some factor affecting on the native of haemoglobin:

1) Vitamins ,trace metals and cofactors Deficincies : such as biotin , coenzyme A ans pyrodixal phosphate are essential for haem synthesis . The folic acid deficiencies can cause megaloblastic anemia . Of the trace metals only copper and cobalt are known to play a role .(copper is playing a role in the absorption of iron while cobalt is essential constituent of vitamine B12(Cobalamin))

**** The causes which lead to deficiency of Vit B12?**

- Deficiency of cobalt or /and deficiency in intrinsic factor

2) Glucose -6-phsphatase dehydrogenase (G6PD) deficiency : G6PD is enzyme responsible for the conversion the glucose into pentose phosphate pathway to form 6-phosphogluconate , this pathway provide NADPH which use for produced reduced glutathion and for other reaction such as reduction methaemoglobin.

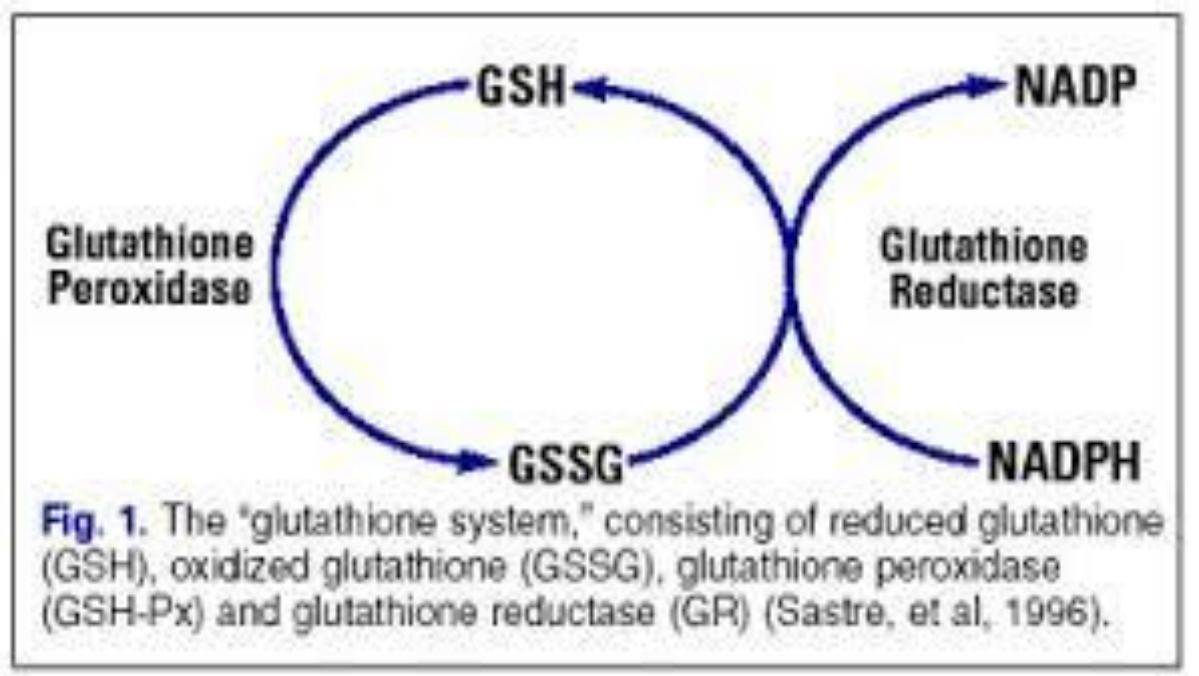


Fig. 1. The "glutathione system," consisting of reduced glutathione (GSH), oxidized glutathione (GSSG), glutathione peroxidase (GSH-Px) and glutathione reductase (GR) (Sastre, et al, 1996).

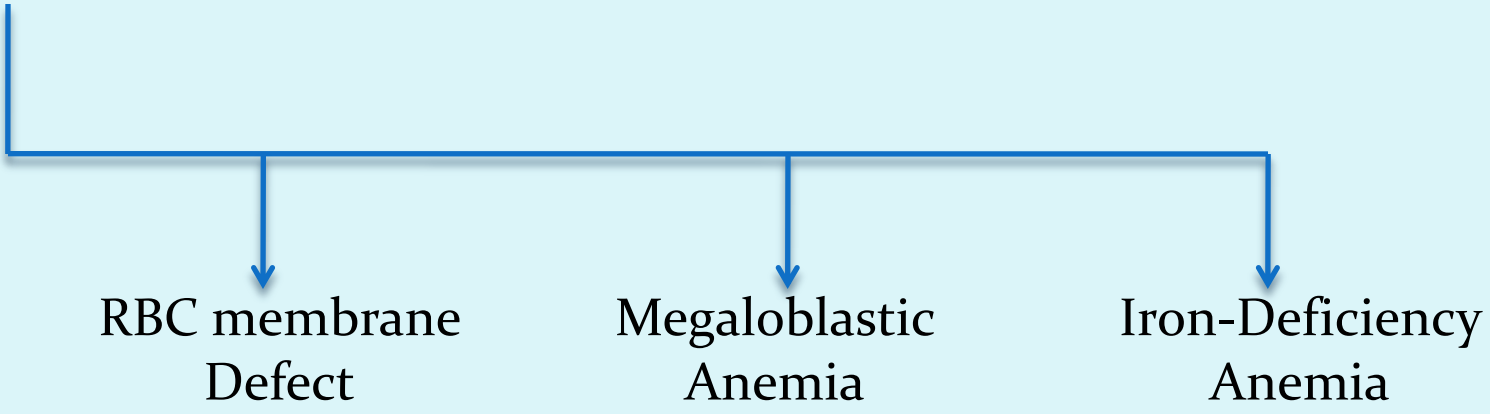
Deficiency of G6PD lead to Decrease level of NADPH , and this lead to broken of RBC membrane then cause hymolysis

Anemia :

Is in general decrease in number of RBC or less normal quantity of haemoglobin in blood leading to decrease oxygen –binding capacity .

Causes:

I. Genetics
II. Acquired



Iron-deficiency anemia:

Deficiency of iron is essentially due to blood loss with failure to replace the iron stores because of :

dietary deficiency or increase requrement or defective absorbtion all of this causes lead to decrease plasma iron .

Megaloblastic Anemia:

This may be due to deficiency of folic acid or cobaltamin (Vit. B12)

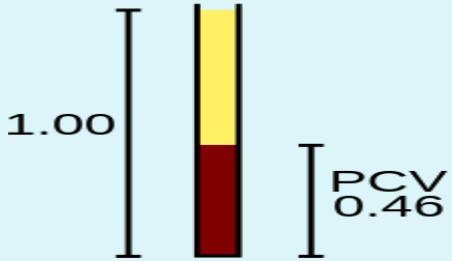
RBC membrane defects:

In this condition there is a defect of the erythrocyte membrane and an abnormally in the soduim pumps.

HCT & ESR and using in dignosis of anemia::

The hematocrit is the percentage of blood composed of red blood cells. People with a high volume of plasma (the liquid portion of blood) may be anemic even if their blood count is normal because the blood cells have become diluted. Like hemoglobin, (reduce in HCT indicate for Anemia) a normal hematocrit percentage depends on age and gender. Anemic ranges for hematocrit generally

Adult : Below 39%



ESR is the rate at which RBC sediment in a period of one hour. It is a common hematology test, and is a non-specific measure of inflamation . To perform the test, anticoagulated blood is placed in an upright tube, known as measured and a Westergren tube, and the rate at which the RBC fall is reported in mm/h. (milimeter per hr)

**nflammation or Anemia changes the proteins in red blood cells causing them to bind to one another in clumps, making them denser than normal red blood cells.

Estimation of blood haemoglobin:

Principle:

The ferrouse (Iron II) in each haem in RBC is oxidized by ferricyanide to Fe(III)-methaemoglobin . A cynide group is then attached to the iron atom (because it is positively charge) by reaction with KCN to give the brown cyanmethamoglobin (stable)which can be estimated quantitatively

Normal Haemoglobin conc. : 12 – 18 g/dl

- Pipette into clean dry test tubes

	test
Hemoglobin reagent	2 ml
sample	0.01 ml (10µl)
Mix, allow to stand at room temperature for 3 min and read the absorbance at 540 nm against hemoglobin reagent	

Calculation

Hemoglobin Conc. In test =Absorbance of test x 29.4

Hemoglobin Conc. In test = g/dl

Normal Haemoglobin conc. : 11 – 18 g/dl