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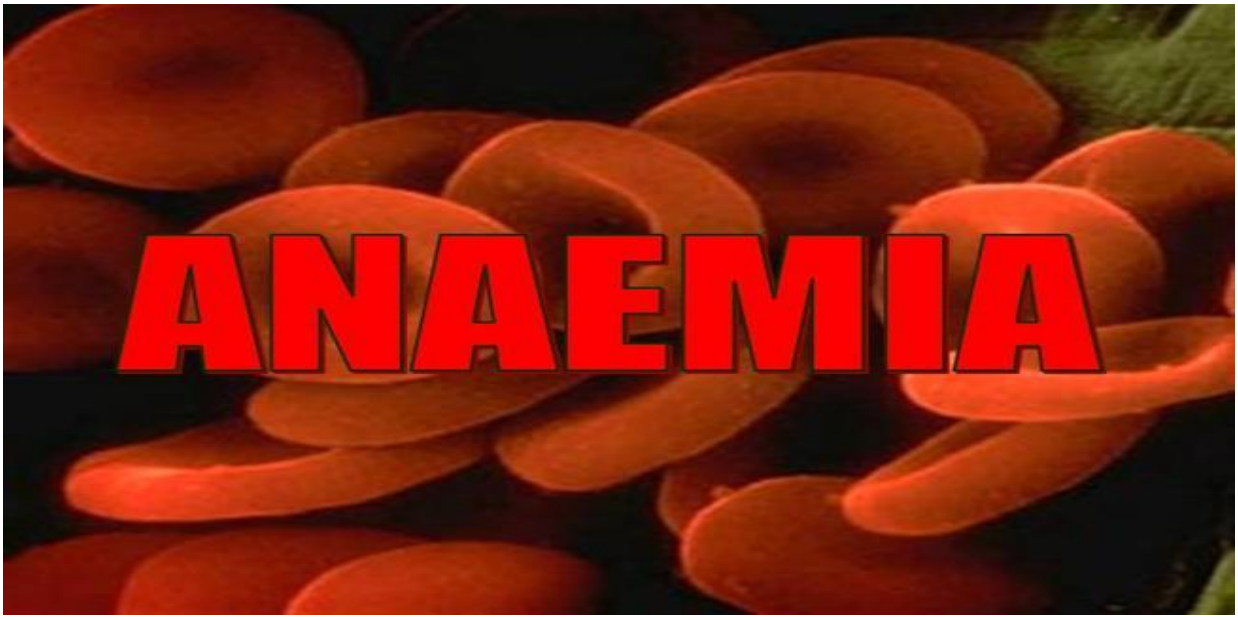
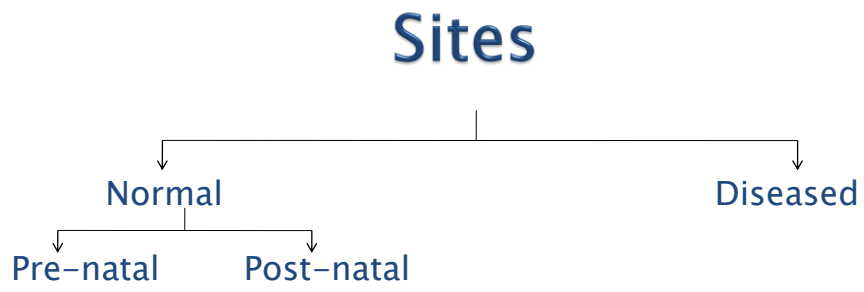


Prof. Dr. SHEREEN MOHAMED EL-HOSEINY
Professor of Clinical and Chemical Pathology

Haematopoiesis

Def:

It's formation of blood cells from earlier stem cells & progenitor cells



Sites:

Normal:

Pre-natal: (Intra-uterine)

- **Mesoblastic** (yolk sac) :3weeks-3 months
- **Hepatic** (liver,spleen,thymus):6 wks-6 ms
- **Myeloid** (BM): 5ms-cont. after birth

Post-natal:(BM only):

At birth:

All marrow cavity r red marrow

Adults:

Flat bones,upper,lower ends of long bones

Old age:

Replaced by yellow marrow

Diseased:

1-Premature release

its activity 10 times 2-BM↑↑

red marrow 3- yellow marrow→

4-Extra-medullary haematopoiesis (liver, spleen)

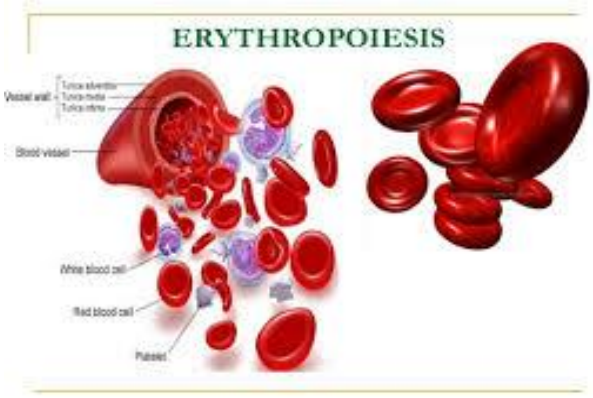
Erythropoiesis

1st stage:

Pluripotent stem cell ⟹ CFU-GEMM IL3,6 ⟹ BFU-E
EPO ⟹ CFU-E

2nd stage:

Pro-erythroblast → Early normoblast → Intermediate normoblast → Late normoblast → Reticulocyte → RBC



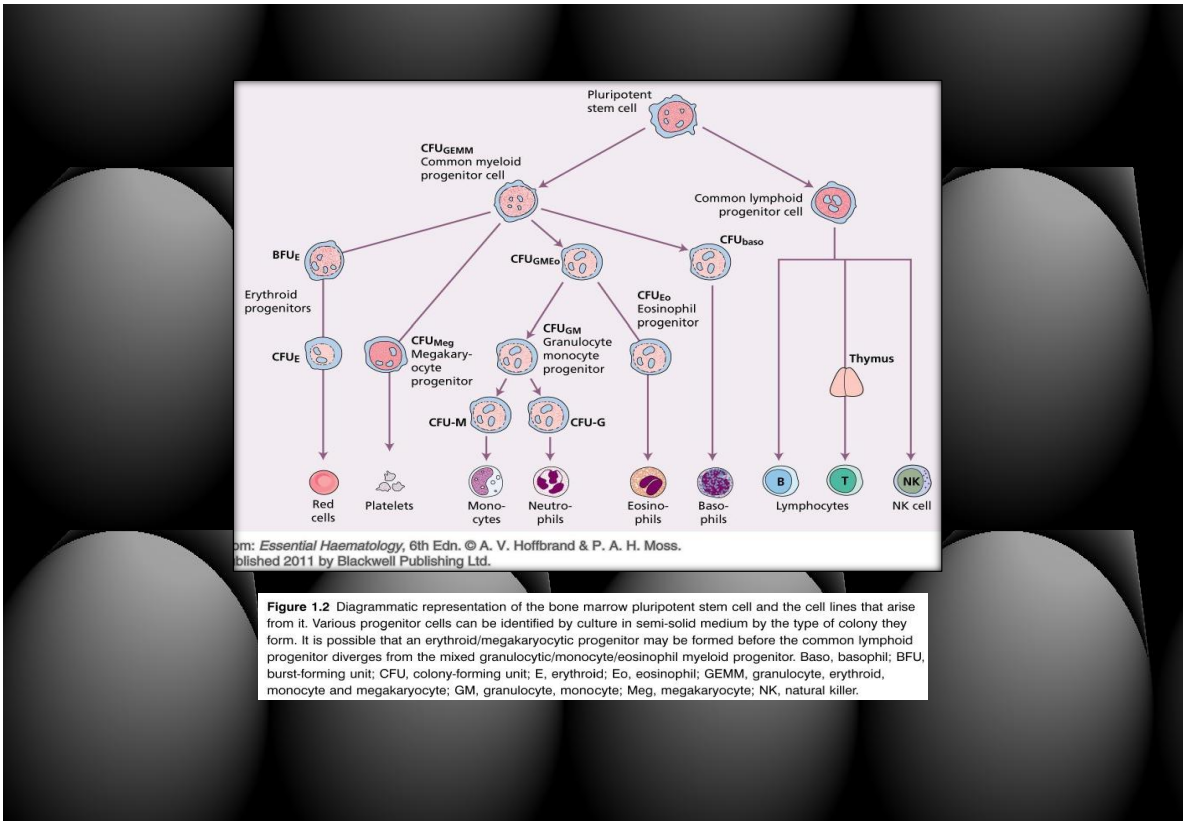


Figure 1.2 Diagrammatic representation of the bone marrow pluripotent stem cell and the cell lines that arise from it. Various progenitor cells can be identified by culture in semi-solid medium by the type of colony they form. It is possible that an erythroid/megakaryocytic progenitor may be formed before the common lymphoid progenitor diverges from the mixed granulocytic/monocyte/eosinophil myeloid progenitor. Baso, basophil; BFU, burst-forming unit; CFU, colony-forming unit; E, erythroid; Eo, eosinophil; GEMM, granulocyte, erythroid, monocyte and megakaryocyte; GM, granulocyte, monocyte; Meg, megakaryocyte; NK, natural killer.

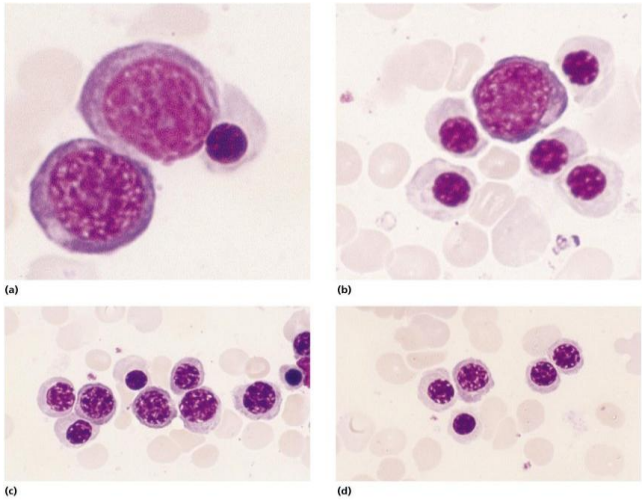


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Figure 2.1 Erythroblasts (normoblasts) at varying stages of development. The earlier cells are larger, with more basophilic cytoplasm and a more open nuclear chromatin pattern. The cytoplasm of the later cells is more eosinophilic as a result of haemoglobin formation.

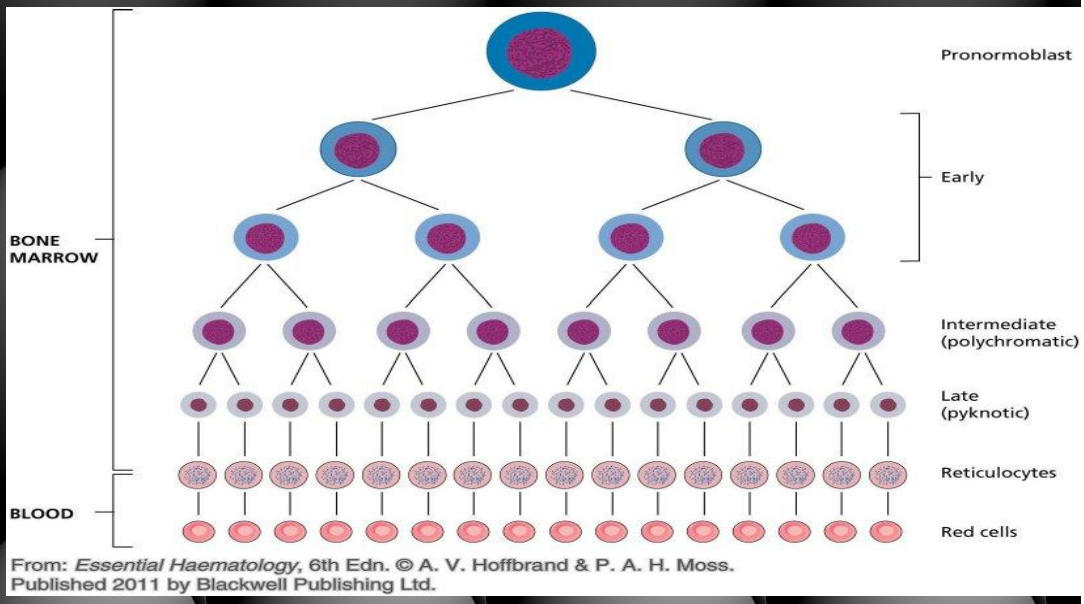

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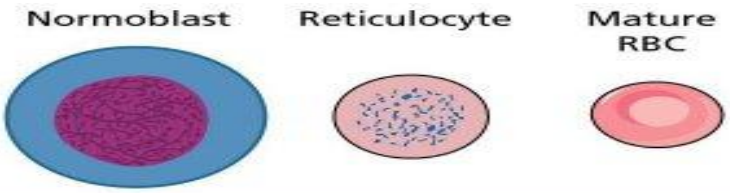
Figure 2.2 The amplification and maturation sequence in the development of mature red cells from the pronormoblast.



Nuclear DNA	Yes	No	No
RNA in cytoplasm	Yes	Yes	No
In marrow	Yes	Yes	Yes
In blood	No	Yes	Yes

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Figure 2.3 Comparison of the DNA and RNA content, and marrow and peripheral blood distribution, of the erythroblast (normoblast), reticulocyte and mature red blood cell (RBC).

Hypoxia:

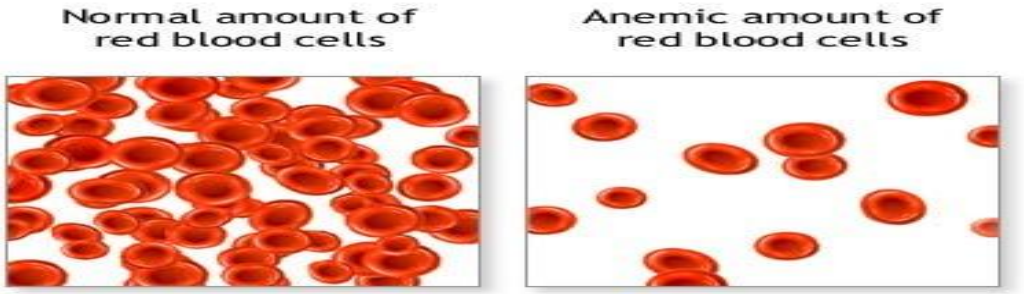
It’s the main factor controlling erythropoiesis (stimulation F. for EPO)

May be due to:

- Hge, haemolysis
- ↓O2 (shift of O2 diss curve to left) e.g. high affinity Hb
- Failure of Hb synthesis e.g. Thalasamia

ANAEMIA

Def: ↓ RBC, Hb, Hct — taking in considerations: -age, -sex, -high altitudes



Values:

RBC's: → M: 5.5±1 million/ul (cmm)
→ F: 4.8±1 million/ul (cmm)

Hb: → M: 13–18 g/dl (g%)
→ F: 12–16 g/dl (g%)

Hct (PCV): → M: 39–54 %
→ F: 36–48 %

Indices:

MCV = $\frac{\text{Ht}}{\text{RBC}}$ X 10 = 76-96 Fl

MCH = $\frac{\text{Hb}}{\text{RBC}}$ X 10 = 27-32 pg

MCHC = $\frac{\text{Hb}}{\text{Ht}}$ X 100 =31-37%

ANAEMIA

Def: ↓ RBC, Hb, Hct taking in considerations - age, - sex, -high altitude

Exceptions : -Hb sea level low at high altitude

-Ht : Hge ↓ RBC mass = ↓vol.

→N Hct

Clinical Picture:

Rate of onset

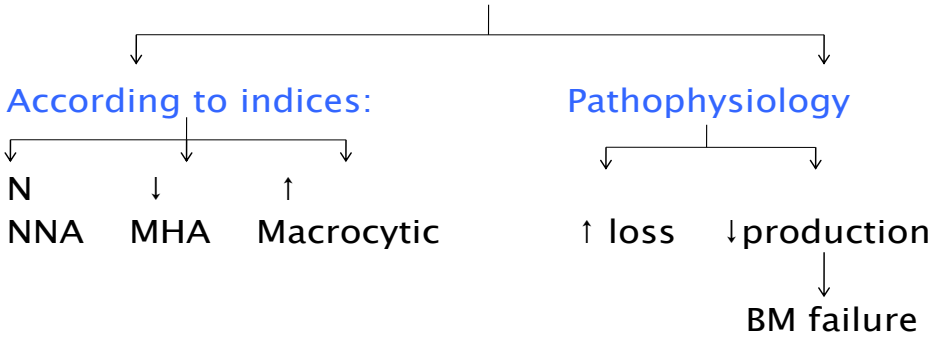
Sudden : Hge ↓ 30% RBC→ cir collapse
Slow: Adaptation mechanisms (few symptoms)

Adaptation mechanisms:
↑ O2 delivery to tissues in spite of ↓ Hb
- ↑ HR, ↑ RR, ↑ CO
- ↑ 2-3 DPG → shift of O2 diss. Curve to Rt → low affinity Hb →deliver O2 more to tissues
- (tolerate up to 50% ↓ RBC mass)

Symptoms & Signs:

1- Underlying cause
2- Anemia :
  -↓ O2 transport: dysnea,fatigue,syncope,anginia, organ dysfunction e.g. GIT
  - ↓ blood vol.: pallor, postural hypotension
  - ↑ CO : palptation, haemic murmurs, CHF
3- Special type of anemia

Classification:



Approach to patients:

History:

- Drug intake, toxic , lead
- Family H. Genetic dis.
- Past H. Anemia
- Racial/ Geographic
- Metabolic disease

Physical examination:

- S&S of inflammation
- Evidance of bleeding tendency
- ↑ spleen ↑ LNs
- Chronic leg ulcers
- Epithelial changes

Lab. Investigations:

- Hb, Hct
- Indices
- CBC
- Retics
- Special tests