Haematology and Dentistry 1: Red Blood Cells (Anaemia)

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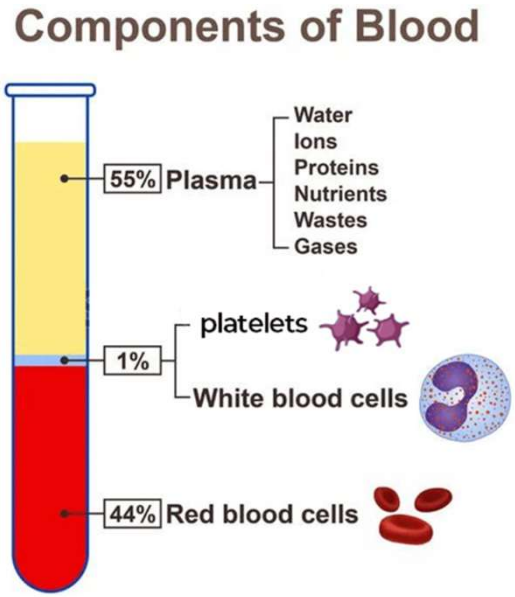
Lecture Outcomes

- 1. Review the role of RBCs, systemic disorders and treatments which affect their structure and function, and general management
- 2. Review the oral manifestations of disorders of RBCs
- 3. Risk assess the implications of RBC disorders, and the treatment modifications required, when planning dental procedures

Blood

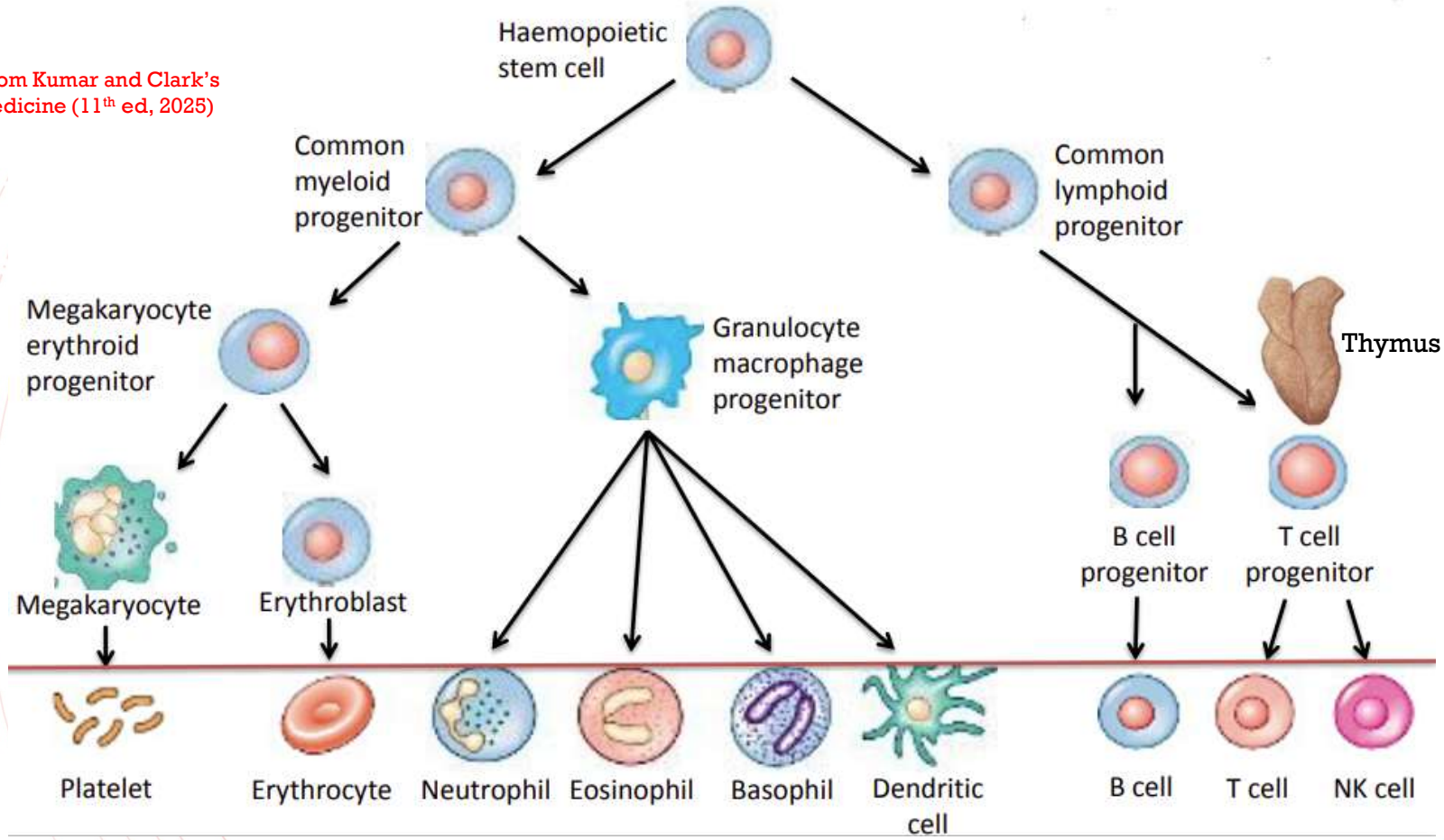
4 main components:

1. Plasma
2. Red blood cells
3. White blood cells
4. Platelets



Formation of Blood Cells

Adapted from Kumar and Clark's Clinical Medicine (11th ed, 2025)



Haemopoietic system

Bone marrow

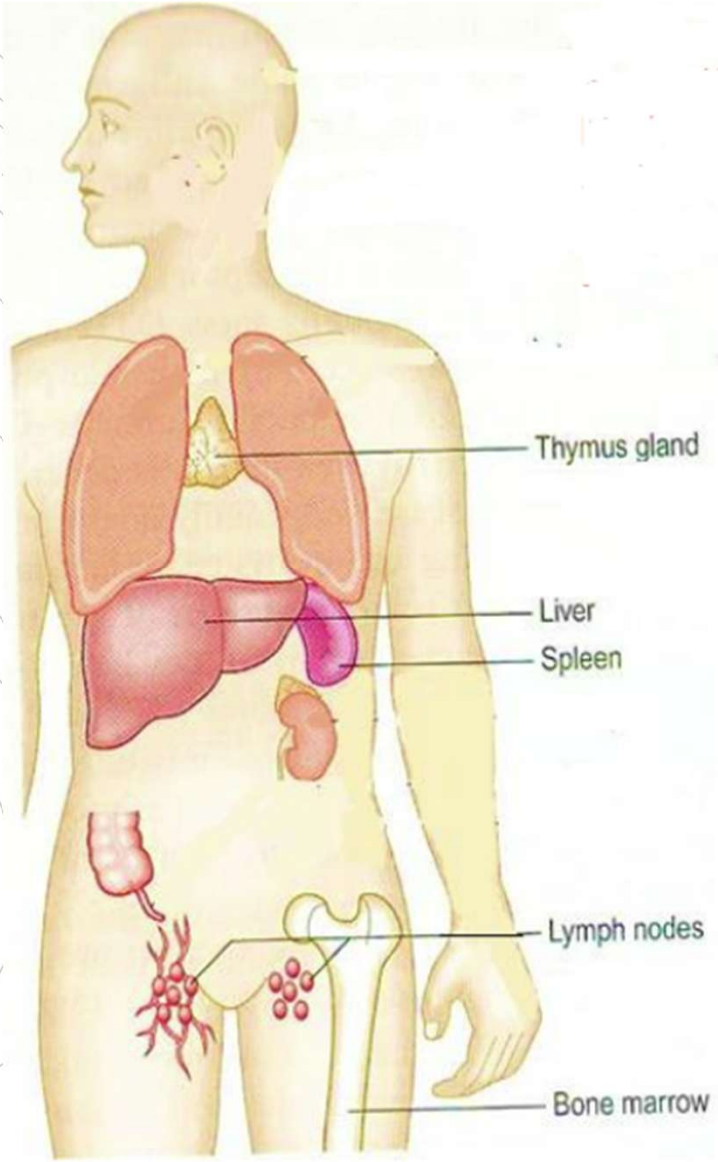
Liver

Lymph nodes

Spleen

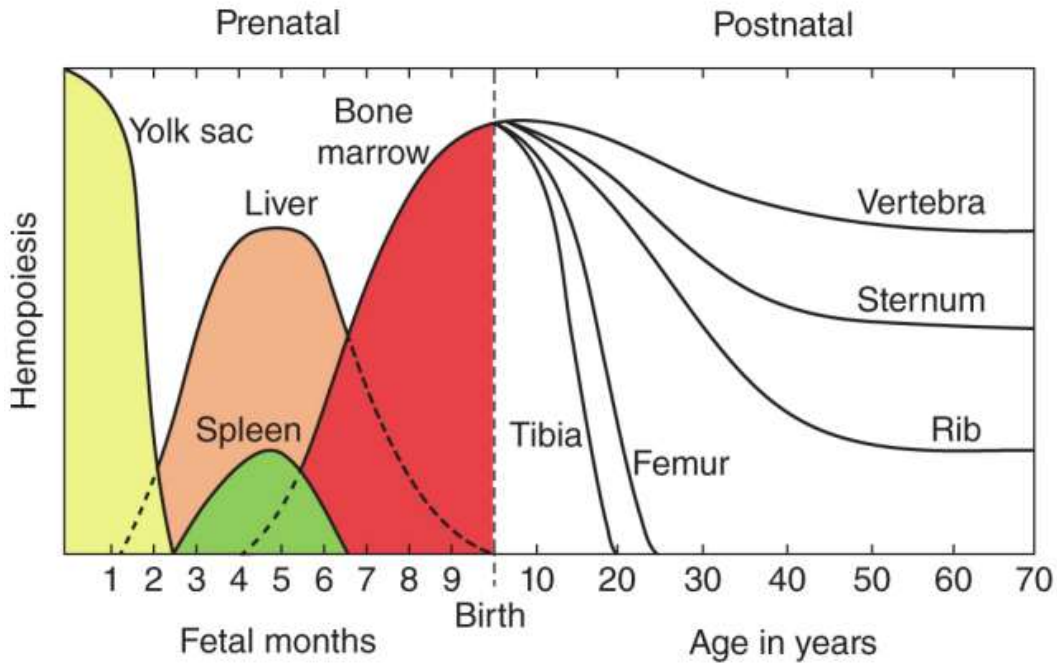
Thymus

Adapted from Scully (2014) – Medical Problems in Dentistry 7th ed



Haemopoiesis

- Blood cell formation
- Occurs in haemopoietic system



Blood tests reference values

Blood parameter	Normal range	Units
White cell count	3.0 – 10.0	x 10^9/L
Neutrophils	2.0 – 7.5	x 10^9/L
Haemoglobin	115 – 155 (female)	g/L
	130 – 170 (male)	g/L
Platelets	150 - 400	x 10^9/L

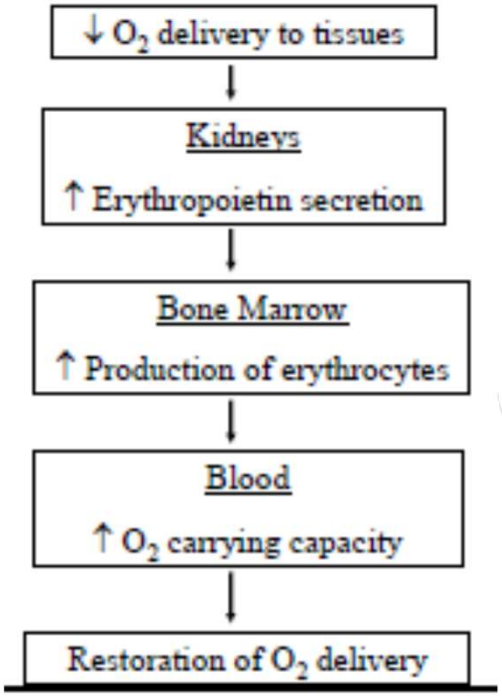
Red blood cells

Erythrocytes

Red Blood Cells (Erythrocytes)

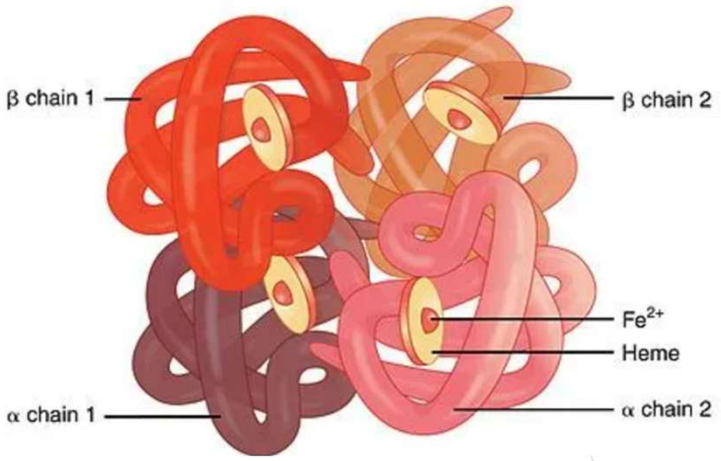
- **Erythrocytes**: responsible for oxygen carriage from lungs to tissues
  - 120 day lifespan
  - Broken down in spleen

- **Erythropoiesis**: red blood cell production



Haemoglobin (Hb)

- **Haemoglobin:**
  - Protein contained by mature erythrocytes
  - 4 globin chains (2 alpha, 2 beta)
  - Degradation:
    - Hb → haem → bilirubin in the liver



Anaemia - Overview

Anaemia

- **Anaemia:** Disorder of red blood cells, defined as a reduction in the oxygen carrying capacity of the blood.

- **Classification:** on the basis of
  - **Cause**:
    - blood loss anaemia
    - deficiency anaemia (reduced erythropoiesis)
    - haemolytic anaemia (increased rate of RBC destruction)
  - **Red-cell size:**
    - microcytic (MCV <78fl)
    - macrocytic (MCV >99fl)
    - normocytic (79 < MCV < 98fl)

Anaemia

Microcytic anaemia (MCV <78fl)	Macrocytic anaemia (MCV >99fl)	Normocytic anaemeia (79 < MCV < 98fl)
Iron deficiency Thalassemia Chronic disease	Vit B12 deficiency Folate deficiency Drugs (e.g. methotrexate, azathioprine) Chronic alcohol intake Liver disease Hypothyroidism Reticulocytosis Pregnancy Aplastic anaemia	Chronic diseases e.g. leukaemia Liver disorders Chronic inflammatory disease Renal failure Infection Malignancy Sickle cell disease

Clinical Manifestations

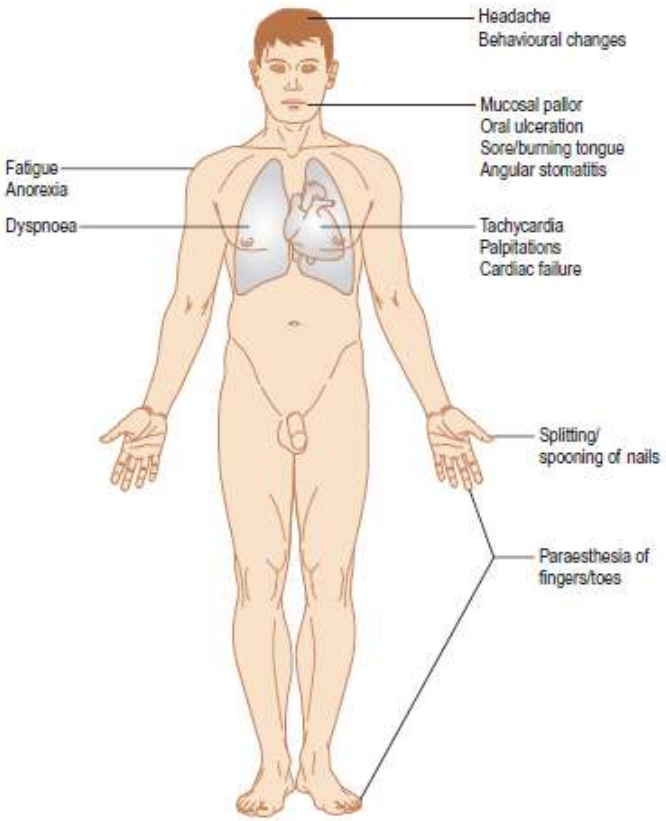
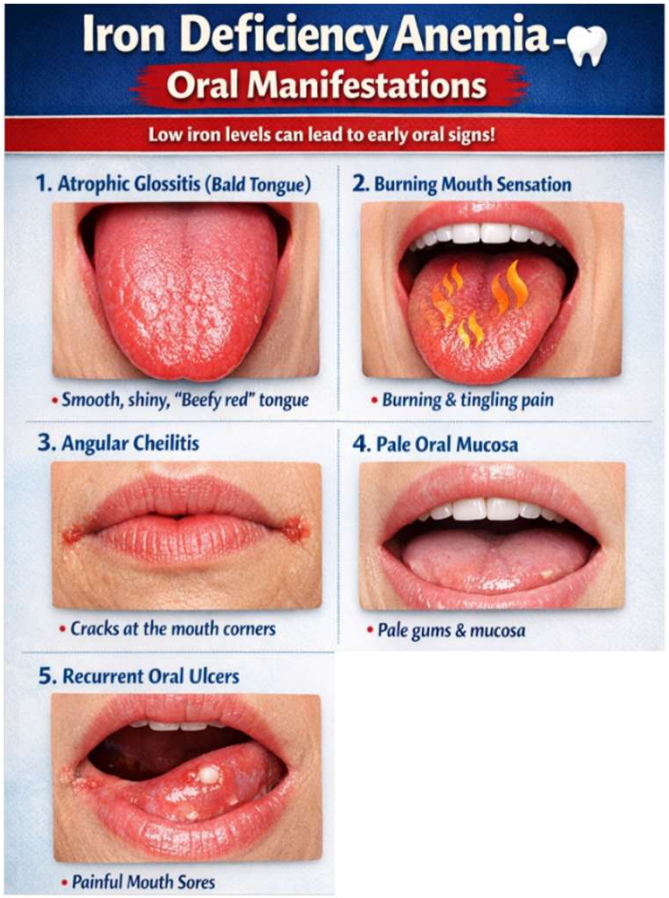


Fig. 8.22 Anaemia – features.

Box 8.9 *Clinical features of anaemia*

- Typically none in the early stages
- General lassitude
- Cardiorespiratory
  - Dyspnoea
  - Tachycardia
  - Congestive cardiac failure
  - Murmurs
  - Palpitations
  - Angina pectoris
- Cutaneous
  - Pallor
  - Brittle nails
  - Koilonychia (spoon-shaped nails – iron deficiency)
- Oral
  - Sore mouth
  - Ulceration
  - Angular stomatitis
  - Glossitis
  - Burning mouth symptoms

Oral Manifestations



Cause:
↓ haemoglobin → ↓oxygen supply → ↓ epithelial regeneration and immunity

1 Atrophic Glossitis

Smooth, shiny, "beefy red" tongue due to loss of filiform papillae.
🔬 Caused by impaired epithelial turnover.
⚠️ May cause altered taste (dysgeusia).

2 Burning Mouth Sensation

Burning or tingling pain without obvious lesions.
🔬 Due to mucosal atrophy + neural sensitivity.

3 Angular Cheilitis

Cracks at mouth corners.
🔬 Epithelial thinning + secondary Candida/Staph infection.
⚠️ Often linked with iron ± B-complex deficiency.

4 Pale Oral Mucosa

Pale gums and inner lips.
🔬 Reduced hemoglobin → decreased tissue oxygenation.

5 Recurrent Oral Ulcers

Painful, slow-healing ulcers.
🔬 Impaired oxygen delivery delays mucosal repair.

Blood loss anaemia

Blood Loss Anaemia

Definition: occurs after sustaining a wound in which there is acute blood loss

Compensatory mechanism:

- immediate threat at this stage is hypovolomic shock (cf. anaemia) and surviving is the primary objective.
- Kidneys retain salt and water to increase the blood volume.
- Once stabilised, haemodilution begins and achieves its full effect within 2-3 days, masking the extent of the red cell loss.
- Healthy patient with adequate iron stores: full recovery from this transient anaemia by utilising iron stores to increase erythropoiesis → complete cell replacement and correction of the anaemia

Deficiency anaemia

Iron, folate, Vit B12

Deficiency Anaemia

Definition: Caused by inadequate supply to the bone marrow of some substances necessary for haemopoiesis (Hb synthesis).

Haematinics: nutrients required to make red blood cells

- Vitamin B12 = 3 year store
- Iron = 12 month store
- Folate = 3 month store

Iron Deficiency Anaemia

Causes:

- Malabsorption (e.g. Coeliac disease, Crohn's disease)
- Nutritional deficiency (low iron intake – malnutrition, reduced meat diet)
- Excess demand (e.g. pregnancy, infancy)
- Chronic blood loss (e.g. especially GIT – peptic ulcers, haemorrhoids, colon cancer; menstruation)

Features

- Weakness
- Pallor
- Koilonychia (nail spooning)
- Hair loss



Iron Deficiency Anaemia



General Management:

- Dietary iron
  - Heme iron: animal tissues - higher absorption rate
  - Non-heme iron: plants, fortified foods – lower absorption rate
  - Enhancers of non-heme absorption: Vitamin C
  - Inhibitors of non-heme absorption: phytates (in grains/beans), oxalates, phosphates present in plant foods ; tannin in tea/coffee/wine; calcium in diary, oxalates

- Iron supplements:
  - Oral intake, may take few months to correct, constipation

- Iron infusions (IV)

Vitamin B12 Deficiency Anaemia

Vit B12: role in synthesising and breaking down amino acids, and synthesising DNA/RNA.

- Required to build new cells, especially blood, skin and mucosal cells.

Cause:

- Usually defect in intrinsic factor (as a result of pernicious anaemia or gastrectomy)
- occasionally ileum disease or resection
- rarely congenital ileum absorption defect.

Pernicious Anaemia: autoimmune condition where the body cannot absorb vitamin B12 due to autoantibodies against gastric parietal cells, and/or intrinsic factor. Without intrinsic factor, Vit B12 is unable to form the complex to absorb in the terminal ileum.

Vitamin B12 Deficiency Anaemia

Additional Features:

- ** Burning tongue
- Neurological symptoms (paraesthesia of extremities) e.g. Loss of toe positional sense and diminished perception of tuning fork vibration, unsteady movement, muscle weakness
- shortness of breath
- slower healing
- premature greying of hair
- increased homocysteine

Treatment: parenteral or high-dose oral replacement

Folic Acid Deficiency Anaemia

Folic Acid:

- needed by body to synthesise and break down amino acids, and synthesise DNA/RNA to build new cells, especially blood, skin and mucosal cells.
- Plays a role in: myelination (liposynthesis to maintain healthy CNS), RBC production, DNA/RNA synthesis → joint metabolism with Vit B12
- Found in fresh leafy vegetables (spinach, kale, Brussel sprouts, asparagus).
- Absorbed from proximal small intestine

Cause:

- Usually dietary deficiency
- Some caused by disease of small intestine (e.g. Coeliac)
- Cancer patients treated with methotrexate

Folic Acid Deficiency Anaemia

Features

- Similar to B12 deficiency
- In pregnancy, predisposes foetus to neural tube defects and cleft lip/palate
- Predisposes to IHD in adults

Treatment

- Folic acid 5mg daily for at least 4 months (rapidly restores)

Haemolytic anaemia

Sickle Cell, Thalassaemia

Haemolytic Anaemias

- **Haemoglobinopathies (inherited abnormalities of Hb formation)**
  - *Sickle cell anaemia / sickle cell trait (globin structure affected)
  - *Thalassaemia (globin chain number diminished)
- **Inherited abnormalities of erythrocyte structure or function**
  - Hereditary spherocytosis (cell shape change)
  - Glucose-6-phosphate dehydrogenase deficiency (cell membrane enzyme defect)
- **Damage to erythrocytes**
  - *Aplastic anaemia
  - Malaria
  - Anaemia of chronic disease
- **Others**
  - Pure red-cell aplasia
  - Paroxysmal nocturnal haemoglobinuria

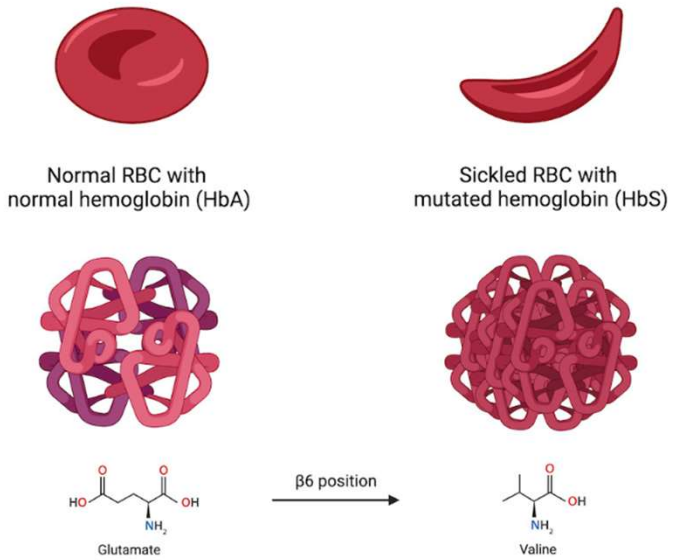
Sickle Cell Anaemia/Trait - *infarcts*

- **Sickling disorders:** hereditary disorder characterised by the presence of structurally abnormal Hb (point mutation in gene encoding for b-chain; Hb → HbS)
  - **Sickle cell anaemia or disease** (HbSS): homozygous, usually a serious disease with widespread complications, frequently apparent in 3rd month of life. Sickle Cell Anaemia is most severe type of the group of sickle cell diseases.
  - **Sickle cell trait** (HbSA): heterozygous and 10 times more common. Frequently asymptomatic, but sickle cell crises can be caused by low oxygen tension (GA, high altitudes, unpressurised aircraft).

- **Epidemiology**
  - Sickle cell trait carriage: Afro-Carribean (1/10), then India / Middle East / Mediterranean (1/100)

Sickle Cell Anaemia/Trait - *infarcts*

- **Cause:**
  - On deoxygenation, HbS molecules undergo polymerisataion (crystallisation).
  - The change in physical shape distorts the RBC, which assumes a sickle shape.
  - Initially this shape deformation is reversible with reoxygenation, but damage occurs with every episode of sickling (e.g. loss of water and potassium, and accumulation with calcium)
  - Eventually the sickle shape becomes permanent



Sickle Cell Anaemia/Trait - *infarcts*

- **Consequence of sickle cells**
  - *Membrane damage and dehydration of RBC:* renders them rigid and irreversibly sicked → mean lifespan reduced from 120 to 20 days as mononuclear phagocytes remove damaged RBCs → chronic extravascular haemolytic anaemia
  - *Widespread microvascular obstructions:* occurs in kidney & spleen due to congestion of red pulp with masses of sickled RBC packed together → splenic erythrostasis → hypoxia → shrunken/fibrous spleen

Sickle Cell Anaemia/Trait - *infarcts*

- **Features**
  - Chronic anaemia
  - Chronic hyperbilirubinaemia
  - *Painful crisis (ie. vaso-occlusive crisis)
    - Infarcts as a result of sickling: spleen, lungs, kidneys, eyes, cerebrovascular stroke
    - Brought on by infection, dehydration, hypoxia, acidosis, cold.
  - Haematological crises: caused by parovirus infection
  - *Infections:
    - Due to immune defect resulting from splenic infarction and dysfunction.
  - Sequestration syndromes:
    - sequestration of sickle cells in: lungs (impeding gas exchange → chest syndrome), spleen (→ septicaemia), liver (→ bowel dilatation)



Fig. 8.35 Dactylitis showing painful red swellings.

Box 8.13 *Features of sickle cell anaemia*

- Anaemia
- Crises
  - Painful crises
  - Aplastic crises
  - Dactylitis – painful osteitis in the hands (Fig. 8.35)
  - Infarcts of central nervous system, lungs, kidney, spleen, bone
  - Skin ulcers
- Haemolysis
  - Jaundice
  - Gallstones
  - Reticulocytosis
- Impaired growth
- Skeletal deformities
- Susceptibility to infections

Sickle Cell Anaemia/Trait - *infarcts*

- **General Management**

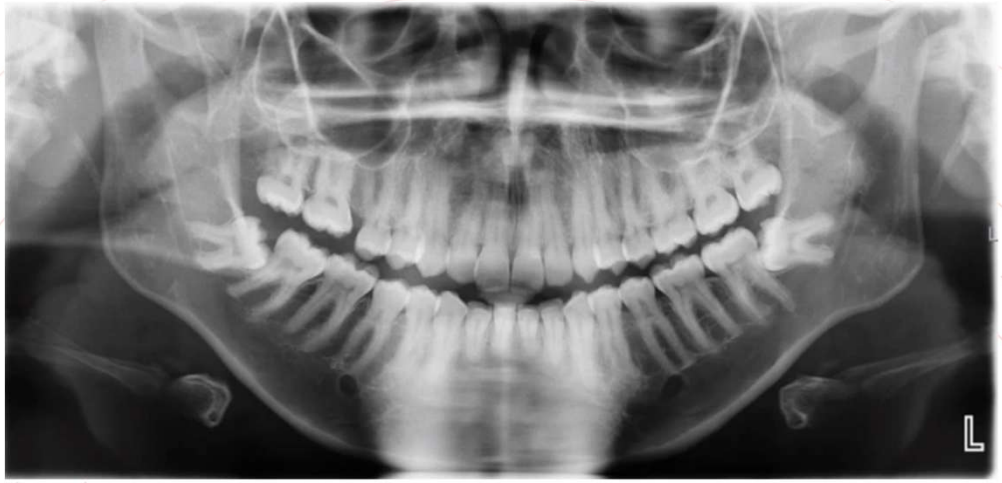
  Main principles in treatment are to prevent trauma, infection, hypoxia, acidosis or dehydration

  - regular blood pictures
  - comprehensive care programmes
  - regular folic acid
  - exchange transfusions (remove sickled cells and replace defective cells) - heparinised
  - fluids
  - keep warm (clothes)
  - long term antibiotics

- most pts on prophylactic penicillin too

Oral Findings – Sickle Cell Anaemia

Increased radiolucency in posterior body of mandible



Increased radiolucency of the bone in the inferior body of the mandible
(Grossman S, Bailey E, Makdissi J. *Oral aspects of sickle cell disease, Br Dent J. 2019; 226: 818)*

due to micro-emboliams in sickle cell anemia

Enlarged haemopoietic maxilla with excessive OJ and OB (due to bone marrow hyperplasia)
Trigeminal neuropathy (due to osteomyelitis)
Painful infarcts in jaws (can be mistaken for toothache)
Enamel hypomineralisation and calcified pulp canals
Pulp symptoms and pulp necrosis in absence of obvious dental disease
Delayed skeletal maturation

Radiological features:

- Hypercementosis (dense lamina dura)
- Sparse mandibular trabecular bone pattern (due to bone marrow hyperplasia)
- Thickening/expansion of skull diploe with "hair-on-end" appearance
- Dense radio-opacities with "stepladder" trabeculae pattern (due to previous infarcts)

Thalassemia – *iron deposition*

- **Definition:** group of heterogenous autosomal dominant genetic disorders of Hb synthesis, characterized by a lack or decreased synthesis of globin chains.
  - May be severe (major; homozygous) or mild (minor;heterozygous)
  - α-thalassemia: α-globin chain synthesis is ↓
  - β-thalassemia: β-globin synthesis is ↓ or non-existent

- **Epidemiology:**
  - Alpha: south-east Asian, southern Chinese, sub-Saharan Africa, Middle East
  - Beta: Mediterranean

Thalassemia – *iron deposition*

- **Cause – Beta thalassemia:** β-globin synthesis is ↓ or non-existent
  - Excess of unpaired α-chains → chains aggregate within the RBC and form insoluble precipitates → damages RBC → sequestration
  - Systemic iron overload due to mass destruction of RBCs
  - Can be life threatening in homozygous form

Thalassemia – *iron deposition*

- **Clinical Presentation –** homozygotes
  - **Iron Overload from repeated transfusions**:
    - Thyroid & parathyroid damage
    - pituitary (delayed puberty, short stature)
    - liver & kidney damage
    - cardiac failure
    - pancreas (secondary diabetes)
    - skin (green/bronze pigmentation due to iron deposition)
    - low levels estrogen or testosterone
  - **Impact of Treatment**:
    - Regular hospital admissions for transfusions
    - disruption to family/school/social life
    - psychological implication of side effects
    - *compliance with chelation (particularly adolescence)

Thalassemia – *iron deposition*

- **General Management**
  - Regular blood transfusion (to treat anaemia)
  - Folic acid supplements *(*builds new blood cells*)*
  - *iron-chelating agents, ascorbic acid.
  - Splenectomy (if hypersplenism causing greater blood destruction → accumulation of iron elsewhere) → daily antibiotic prophylaxis

Other organs affected, e.g.

- Pancreas → diabetes testing
- Hypothyroidism → thyroid hormone
- Osteoporosis → bisphosphonates, calcium
- Liver → MRI monitoring
- Hormone replacement
- Gallbladder → gallbladder removal if gallstones

Oral Findings – Thalassaemia

- Enlargement of maxilla ("chipmunk face") due to extramedullary haemopoiesis → OJ and OB
- Migration and spacing of upper anterior teeth
- Painful swelling of parotids and hyposalivation (due to iron deposits)
- Delayed pneumatisation of maxillary sinuses
- Teeth discoloured with short crowns/roots

- Alveolar bone with "chicken wire" radiographical appearance (extramedullary haematopoiesis)

- Sore/burning tongue (folate deficiency)
- Pale gingiva and mucosa (anaemia)







Aplastic Anaemia

- **Aplastic anaemia:** Macrocytic pancytopenia with a non-functioning bone marrow. A rare disease, characterised by suppression of multipotent myeloid stem cells causing leukopenia, thrombocytopenia and refractory anaemia.
- **Fanconi anaemia**: inherited type of aplastic anaemia associated with progressive deficiency of all bone marrow production blood cells and increased risk of AML
- **Causes:**
  - Idiopathic: poor prognosis
  - Secondary: after whole body irradiation/chemo, chemicals, drugs, hepatitis viruses, GVHD

Box 8.14 *Causes of aplastic anaemia*

- Genetic
  - Fanconi anaemia
  - Dyskeratosis congenita
- Immunological
  - Autoimmune
  - Graft-versus-host disease
- Drugs
  - Allopurinol
  - Anticonvulsants
  - Antithyroids
  - Chloramphenicol
  - Cytotoxic agents
  - Gold
  - Non-steroidal anti-inflammatory drugs (NSAIDs)
  - Penicillamine
  - Phenylbutazone
  - Sulphonamides
- Chemicals
  - Benzene
  - Glue-sniffing
  - Heavy metals
  - Toluene
- Viruses
  - Hepatitis
- Irradiation

Aplastic Anaemia

- **Features:** those of anaemia + abnormal susceptibility to infection/bleeding
  - Purpura, petechiae, ecchymoses
  - Predisposition to infection
  - Weakness, pallor, dyspnoea

Aplastic Anaemia

- **General Management:**
  - Remove cause
  - Isolation and antibiotics to control infection
  - immunosuppression
  - Blood transfusion
  - Stem cell transplant

- **Prognosis** - if treated
  - 90% for 19-39yo
  - 70% for 40-59yo
  - 38% for >60yo
  - Can evolve into AML

Aplastic Anaemia

- **Dental management**
  - Oral features:
    - similar to leukaemia (ulceration, infection susceptibility, haemorrhaging tendencies)
    - Oral lichenoid lesions
    - Sjogren-like syndrome of GVHD
    - Gingiva enlargement (ciclosporin, or AML transformation)

Dental Implications

Risk Assessment

Red blood cell count (>80-100 g/L)

Questions for patient

- Are you transfusion-dependent?
- How many times per week/month?
- Do you experience SOB? (if so, when)

Risks specific to thalassaemia (especially beta-thalassaemia major)

- Splenectomy = infection risks
- Osteoporosis (possible MRONJ risk)
- Cardiomyopathy and cardiac failure
- Liver cirrhosis
- Kidney
- Diabetes (secondary to pancreas iron deposition)
- Thyroid/parathyroid damage

Risks specific to sickle cell anaemia

- Timing around transfusions
- Oxygen support
- Infection risks
- Carrying out treatment during phase when haemolysis is minimal
- *Depends on other organs affected
- Heparinised for exchange transfusions

Dental Management

Access:

- Short appointments in morning to ensure patient not too fatigued.
- If patient receives regular exchange/blood transfusions, avoid treatment on same day (will also be heparinised)

Communication/Consent:

- Nil significant

Education/Prevention:

- Ensure strict OH regime and prevention education to avoid need for dental treatment
- Removeable prosthetics may be unable to be tolerated when oral ulcers present

Dental Management

Surgery – Pre-operative

- Haemoglobin assay/complete blood count performed
- Ideally correct anaemia prior to dental treatment and check Hb level raised
  - if necessary, by transfusion in emergency
- Patient stabilised at least 24hrs pre-operatively.
  - Hb estimations are unreliable for 12hrs post-transfusion or after acute blood loss

- Caution advised if <80g/L: ideally only emergency dental treatment to stabilise dentition with peri-operative nasal oxygen/pulse oximeter.

Dental Management

Surgery – Peri-operative

- Consider supplemental nasal oxygen (3L/min) if Hb <80-100g/L
- [SCA] Avoid prilocaine (risk: methemoglobinemia)
- Nitrous oxide contraindicated in Vit B12 deficiency
  - interferes with B12 metabolism
- Deeper levels of sedation – risk of hypoxia
  - Reconsider elective IV/GA's if Hb <80-100g/L
- [SCA/Th] Consider prophylactic/surgical antibiotics in post-splenectomy patients

Dental Management

Spread

- Standard infection control: risk of BBVs in older patients receiving recurrent transfusions prior to current blood product screening standards
- Consider antibiotic prophylaxis if:
  - Other cell lines affected
  - Splenectomy
  - Sickle cell disease

Questions?

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