

**D**isorders of the red blood cells (RBCs), which in large part consist of **the anemias**, are important to the dentist and to the health of the patient for several reasons. First, the dentist serves an important role in detecting patients with anemia through history, clinical examination, and results of screening laboratory tests. These screening procedures should lead to early referral to a physician and finalization of the diagnosis.

Anemia is an independent risk factor for the development of:

- 1- Adverse cardiovascular outcomes (i.e., acute myocardial infarction and death)
- 2- In a variety of patient populations (e.g., chronic kidney disease, acute coronary syndrome, old age)

Therefore, preventive measures should be considered when one is performing stressful dental procedures.

### ***Anaemia***

Reduction in the oxygen carrying capacity of the blood, is usually associated with a decreased number of circulating RBCs or an abnormality in the Hb contained within the RBCs, this is defined as anaemia. Anemia, defined as Hb values below 13 g/dL for men and below 12 g/dL for women. Iron deficiency anemia is the most common type.

Anaemia is result from one of three underlying causes:

- (1)- Decreased production of RBCs (iron deficiency, pernicious anemia, folate deficiency)
- (2)- Blood loss
- (3)- Increased rate of destruction of circulating RBCs (hypersplenism, autoimmune destruction).

The normal red cell is about 33% hemoglobin by volume. Hemoglobin (Hb), the oxygen-carrying molecule of erythrocytes.

The kidney serves as the primary sensor for determining the level of oxygenation. If the level is low, the kidney releases erythropoietin, a hormone that stimulates the bone marrow to release RBCs. Erythropoietin is produced by:

- 1- 95% Cortical cells in the kidney
- 2- 5% Liver

### ***Types of Anemia***

There are many types of anemia, like;

- 1- Iron Deficiency Anemia,
- 2- Folate Deficiency Anemia and Pernicious Anemia,
- 3- Hemolytic Anemia,

- 4- Hemolytic Anemia: Glucose-6-Phosphate Dehydrogenase Deficiency,
- 5- Sickle Cell Anemia
- 6- Renal Disease; The kidney produces the hormone erythropoietin, which stimulates RBC production by the bone marrow. If significant renal damage occurs, lack of production of this hormone results in anemia. Patients who have chronic renal failure and are on dialysis often have anemia and low erythropoietin levels. Erythropoietic drug therapy is offered to these patients.

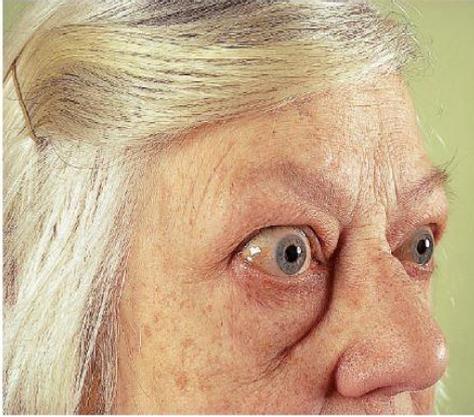
All types explained in the following table

TABLE 23-1 Types of Anemia	
Classification by Size and Shape of RBC	Cause
MICROCYTIC (MCV < 80 $\mu\text{m}^3$ )	
Iron deficiency anemia	Decreased production of RBCs
Thalassemias	Defective hemoglobin synthesis
Lead poisoning	Inhibition of hemoglobin synthesis
NORMOCYTIC (MCV 80-100 $\mu\text{m}^3$ )	
Hemolytic anemia	Increased destruction of RBCs
Sickle cell anemia	
Glucose-6-phosphate dehydrogenase deficiency	
Aplastic anemia	Decreased production of RBCs
Renal failure	Decreased production of RBCs
Anemia of chronic disease	Decreased production of RBCs
MACROCYTIC (MCV > 100 $\mu\text{m}^3$ )	
Pernicious anemia	Decreased production of RBCs
Folate deficiency	Decreased production of RBCs
Hypothyroidism	Decreased production of RBCs

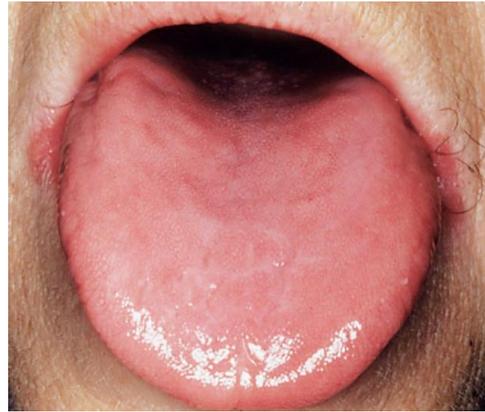
RBC, Red blood cell; MCV, mean corpuscular volume.

### ***Signs & Symptoms of anemia***

It may include jaundice, pallor, cracking, splitting and spooning of the fingernails, increased size of the liver and spleen, lymphadenopathy, and blood in the stool. Premature graying of hair (Figure 1) and yellowing of the skin (due to jaundice) have been reported with pernicious anemia. Patients with anemia may also describe a sore or painful tongue (glossitis) (Figure 2), a smooth tongue, or redness of the tongue or cheilosis. Some patients may complain of loss of taste sensation.



**Figure 1:** Pernicious anemia. This 38-year-old woman has blue eyes and vitiligo and shows premature graying of the hair—three features that are more common in patients with pernicious anemia.



**Figure 2:** Smooth red tongue and cheilitis in a patient with iron deficiency anemia.



**Figure 3:** Sickle cell anemia, growth deformation of the middle finger from vaso-occlusive attack and dactylitis of the growth plate.

## Screening Laboratory Tests

If the dentist identifies a patient with signs or symptoms suggestive of anemia, this patient should be sent to a commercial laboratory for a complete blood count CBC and differential, or referred to a physician for evaluation.

Hb level, hematocrit, and RBC indices (mean corpuscular volume [MCV], mean corpuscular hemoglobin [MCH], and mean corpuscular hemoglobin concentration [MCHC]) are tests that are used to screen the patient.

In addition, total white blood cell (WBC) count and platelet count should be obtained to determine whether a generalized bone marrow defect has occurred and to inspect for hypersegmented neutrophils.

In accordance with the size of RBCs, anemia is classified as:

- 1- microcytic (MCV < 80  $\mu\text{m}^3$ ),
- 2- macrocytic (MCV > 96  $\mu\text{m}^3$ ),
- 3- normocytic (MCV, 80  $\mu\text{m}^3$  to 96  $\mu\text{m}^3$ )

To further distinguish the various types of anemias, key laboratory tests, are shown in Table 23-2:

TABLE 23-2

Laboratory Assessments to Aid in the Diagnosis of Anemia\*

	Type	Tests to Discriminate Types of Anemia
Microcytic anemia	Iron deficiency	Serum iron, ferritin, total iron binding capacity (TIBC), transferrin saturation, bone marrow aspirate. Also, stool examination for occult blood
Macrocytic anemia	Folate deficiency	CBC, serum folate level
Macrocytic anemia	Pernicious anemia	CBC, serum vitamin B <sub>12</sub> (cobalamin) assay levels, Schilling's test, serum antiparietal cell, and intrinsic factor antibodies
Normocytic anemia	G-6-PD	Staining peripheral blood smear with methyl or crystal violet, cyanide-ascorbate assay, qualitative (fluorescent spot) test and quantitative test for G-6-PD, reticulocyte count, indirect bilirubin levels
Normocytic anemia	Sickle cell anemia	Sickledex, high-performance liquid chromatography, hemoglobin electrophoresis, reticulocyte count, indirect bilirubin levels
Normocytic anemia	Aplastic anemia	Erythropoietin levels, bone marrow aspirate

CBC, Complete blood count; G-6-PD, glucose-6-phosphate dehydrogenase.

MCV (mean corpuscular volume), MCH (mean corpuscular hemoglobin), and MCHC (mean corpuscular hemoglobin concentration) have been assessed, and values indicate that anemia is present.

\*These tests are ordered after the initial CBC and differential, including red cell indices.

## Medical Management

The goal of treatment is to eliminate the underlying cause. Management protocols for several types of anemia are discussed in the following paragraphs.

In microcytic anemia (iron deficiency), the physician should look for a source of bleeding. Iron deficiency associated with pregnancy often resolves after childbirth.

In children, iron supplements (ferrous sulfate, 2 to 6 mg/kg/day) are recommended to arrest motor and cognitive impairment brought on by iron deficiency.

In patients who have undergone a gastrectomy, iron supplements (ferrous sulfate or ferrous gluconate) are provided on a long-term basis. In men, management often

involves treatment of the underlying cause (e.g., peptic ulcer disease, gastrointestinal malignancy).

Folate deficiency is managed by administering folic acid supplements and by increasing the intake of green, leafy vegetables and citrus fruits.

In the case of poor intestinal absorption, replacement therapy with folic acid may be lifelong. Cyanocobalamin injections are used to treat patients with pernicious anemia. Injections generally are given daily for the first week, then are tapered eventually to once a month, as needed. Anemic patients may require hospitalization if the **hematocrit value** is less than 20%.

## ***Dental Management***

### ***Medical Considerations***

The dentist should obtain a careful history to identify conditions associated with anemia.

This assessment should include questions concerning dietary intake, malnutrition, alcohol or drug use, use of non-steroidal anti-inflammatory drugs, menstrual blood loss, pregnancies, hypothyroidism, jaundice, gallstones, splenectomy, bleeding disorders and abnormal Hb, and organ transplantation. Historical information concerning family members also is important for identifying hereditary risk for hemolytic anemias.

The dentist should be keen to identify signs and symptoms of anemia in patients who are seen for dental treatment. A patient with classic signs or symptoms of anemia should be referred directly to a physician and screened by appropriate laboratory tests.

**Patients with anemia, particularly men, may have a serious underlying disease such as peptic ulcer or carcinoma**, for which early detection may be lifesaving. Patients with sickle cell anemia may be in grave danger if the disease is not detected before dental treatment is started. Thus, it is important for the dentist to attempt to identify these patients through history and clinical examination before starting any treatment.

Assessment of the severity of a patient's anemia is important for preventing complications. First and foremost, the dentist should ensure that the patient's underlying condition is under good medical control before proceeding with routine dental treatment.

In many cases, anemia is associated with chronic illness; thus, treatment may be provided in the presence of anemia. To minimize the risk of medical complications, Hb levels should be above 11 g/dL, and the patient should be free from symptoms. Patients who are short of breath and in whom Hb levels are less than 11 g/dL, an abnormal heart rate, or oxygen saturation less than 91% (as determined by pulse oximetry) are considered medically unstable, and routine treatment should be deferred until their health status improves.

**Patients with G-6-PD deficiency** exhibit an increased incidence of drug sensitivity, with sulfonamides (sulfamethoxazole), aspirin, and chloramphenicol being the . Penicillin, streptomycin, and isoniazid also have been linked to hemolysis in these patients. Dental infection may accelerate the rate of hemolysis in patients with this type of anemia. Thus, dental infections should be avoided, and if they occur, they must be dealt with effectively. The astute clinician will recognize that febrile illness

and elevated bilirubin are features of this condition. The drugs listed previously should not be used in these patients.

Dental management considerations for the patient with sickle cell anemia are summarized in Box 1.

Dental Management of the Patient With Sickle Cell Anemia
1. Confirm with patient's physician that the condition is stable.
2. Arrange short appointments.
3. Avoid long and complicated procedures.
4. Maintain good dental repair.
5. Institute aggressive preventive dental care. a. Oral hygiene instruction b. Diet control c. Toothbrushing and flossing d. Fluoride gel application
6. Avoid oral infection; treat aggressively when present.
7. Use pulse oximeter, maintain O <sub>2</sub> saturation above 95%.
8. Use local anesthetic without epinephrine for routine dental care. For surgical procedures, use 1:100,000 epinephrine in local anesthetic.
9. Avoid barbiturates and strong narcotics; sedation may be attained with diazepam (Valium).
10. Use prophylactic antibiotics for major surgical procedures.
11. Avoid liberal use of salicylates; control pain with acetaminophen and codeine.
12. Use nitrous oxide–oxygen with greater than 50% oxygen, high flow rate, and good ventilation.

**Persons with aplastic anemia** are susceptible to infection and bleeding, so clinical recognition of such patients before invasive dental procedures are performed is important.

Patients with signs and symptoms of anemia, petechiae, ecchymoses, and gingival bleeding should be referred to a physician for evaluation, diagnosis, and treatment as indicated.

### ***Treatment Planning Modifications***

Treatment planning modifications are directed primarily toward individuals who have severe anemia or sickle cell anemia. Elective surgical procedures are best avoided in patients with sickle cell anemia.

Routine dental care can be rendered for patients with sickle cell trait and for those whose disease is in a non-crisis state.

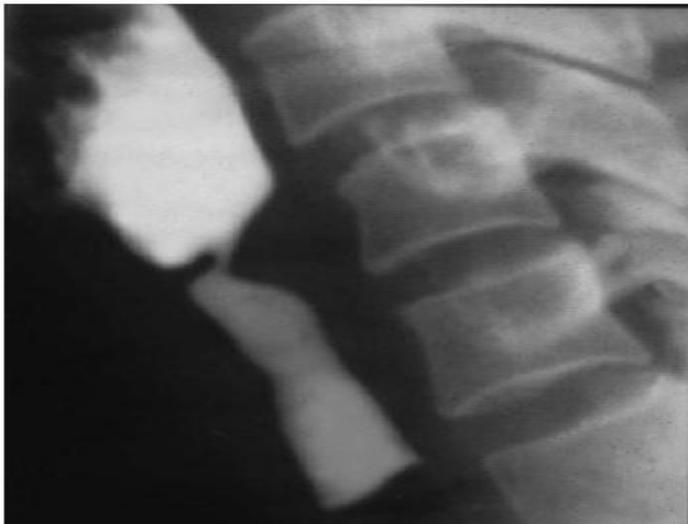
Special emphasis should be placed to avoid dental caries, gingival inflammation, and infection that can lead to osteomyelitis. Adequate oxygenation should be provided

during nitrous oxide inhalation procedures. Pulse oximetry monitoring is prudent during dental treatment of all patients with anemia.

### ***Oral Complications and Manifestations***

The oral mucosa often appears pale. Patients with nutritional causes of anemia (e.g., vitamin B12 or iron deficiency) may show loss of papillae from the tongue and atrophic changes in the oral mucosa (see Figure 2). Angular cheilitis, aphthae, burning or sore tongue may be found.

Some patients with iron deficiency anemia develop Plummer-Vinson syndrome (**Figure 4**), which is characterized by a sore mouth, dysphagia (resulting from muscular degeneration in the esophagus with esophageal stenosis or “webbing”), and an increased frequency of carcinoma of the oral cavity and pharynx.



**Figure 4:** Plummer-Vinson Syndrome. Barium contrast radiograph demonstrates esophageal webbing.

Patients with *hemolytic anemia* (e.g., *sickle cell anemia*) may show pallor and oral evidence of jaundice caused by hyperbilirubinemia caused by excessive erythrocyte destruction. The trabecular pattern of the bone on dental radiographs may be affected because of hyperplasia of marrow elements in response to increased destruction of RBCs.

Therefore, dental radiographs may show enlarged bone marrow (medullary) spaces associated with bone marrow hyperplasia, increased widening and decreased numbers of trabeculations, and generalized osteoporosis (thinning of the inferior border of the mandible).

The bone appears more radiolucent with prominent lamellar striations. The trabeculae between teeth may appear as horizontal rows or as a “stepladder” (**Figure 5**). This can also manifest as frontal bossing and/or “hair on end” in the cortical regions of a skull film (**Figure 6**).

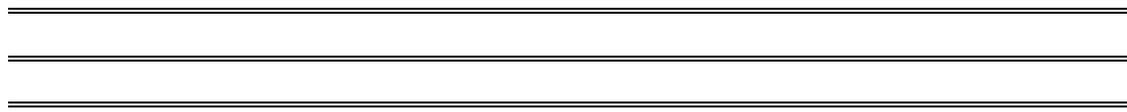
Vaso-occlusive events can promote asymptomatic pulpal necrosis, osteomyelitis, ischemic necrosis within the mandible, and peripheral neuropathy. Patients with sickle cell anemia often have delayed eruption of teeth and dental hypoplasia.



**Figure 5:** Prominent horizontal trabeculations and the dense lamina dura, in patient with sickle cell anemia



**Figure 6:** Skull Film shows new bone formation on the outer table, producing perpendicular radiations or "hair on end" appearance in patient with hemolytic anemia.



CD5 (Protein): Is a Cluster of Differentiation expressed on the surface of T cells

CD5 was used as a T-cell marker