Pathophysiology of blood disorders 2nd ed

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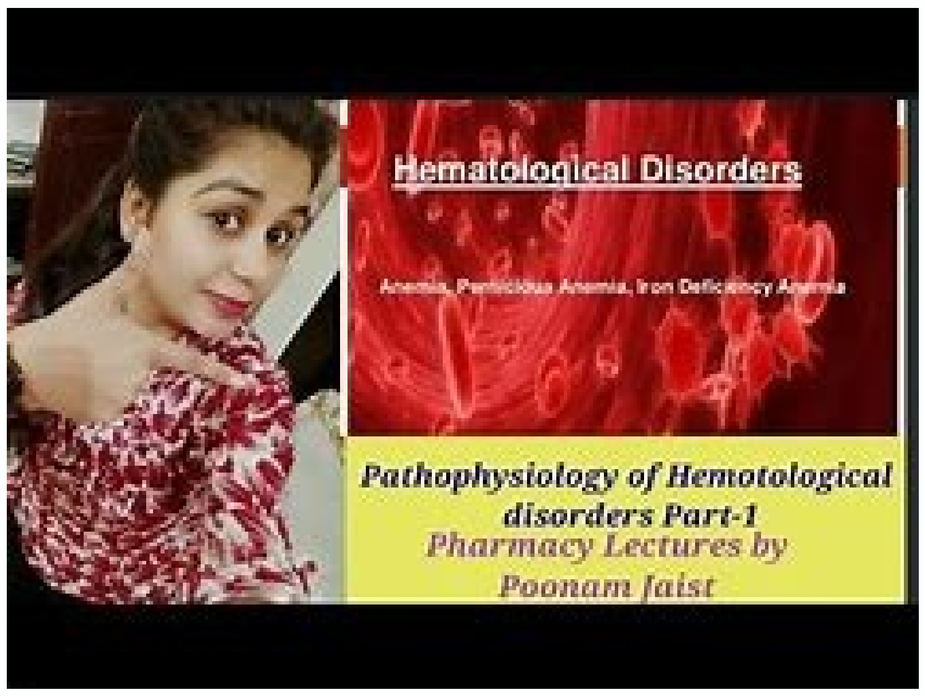

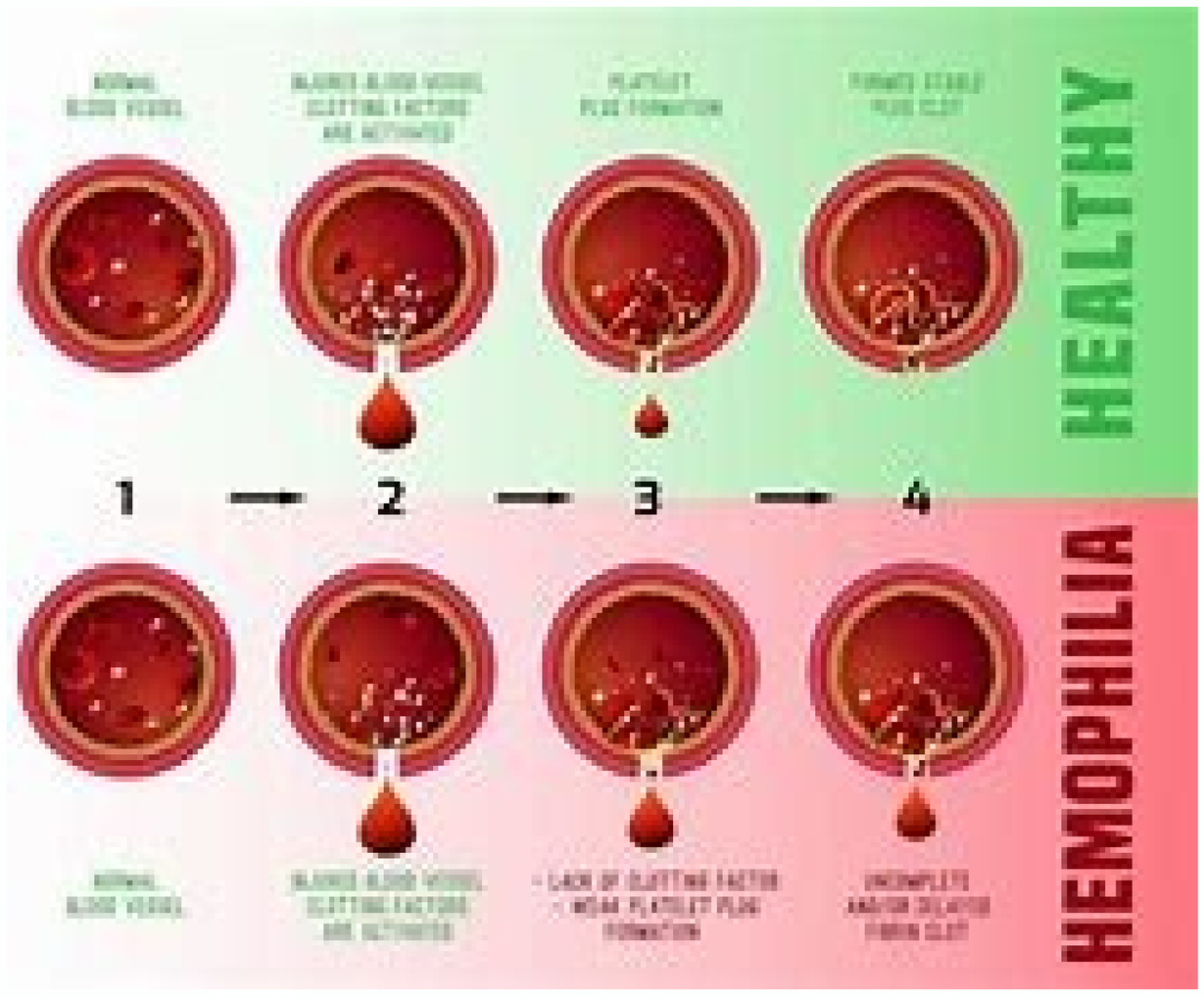

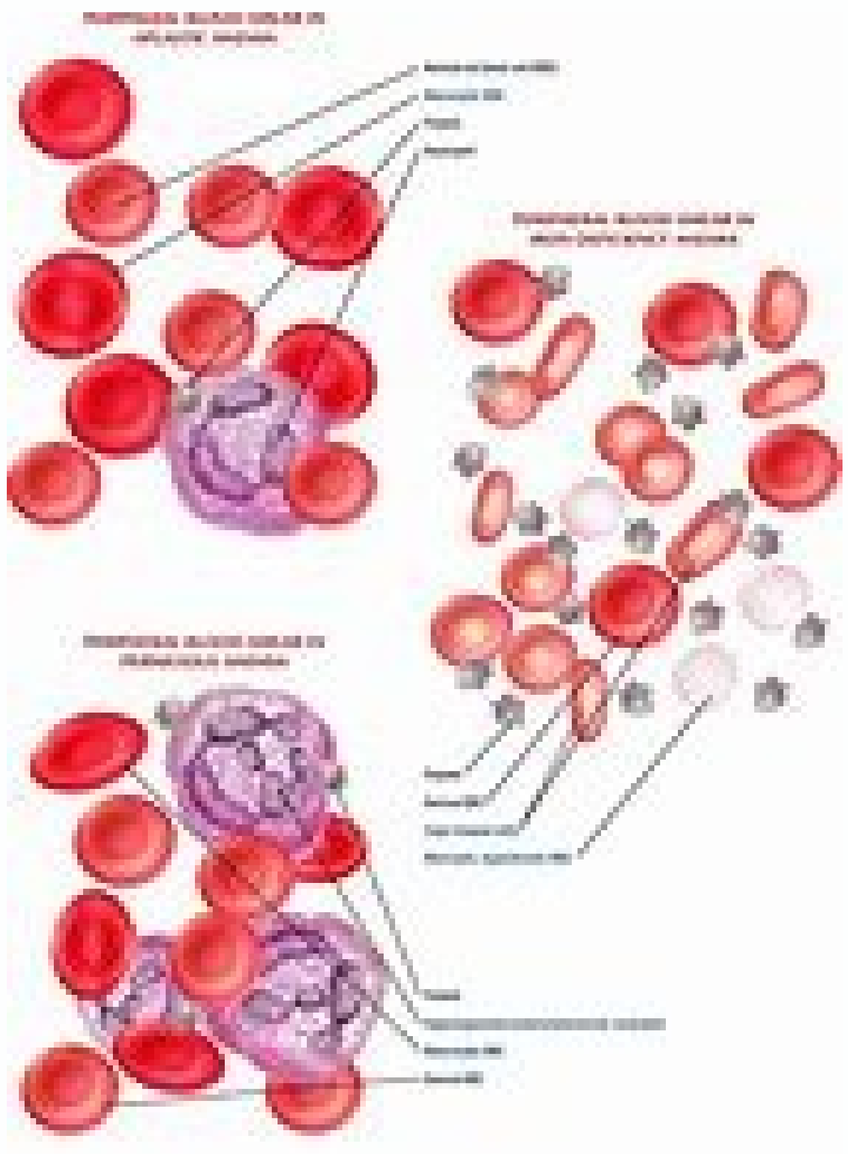

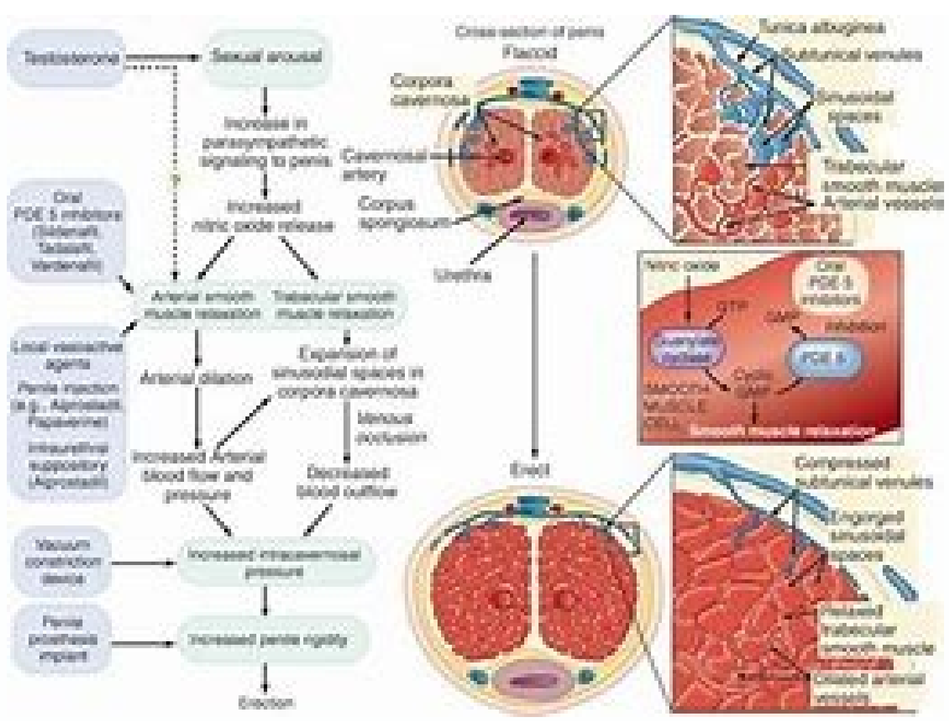

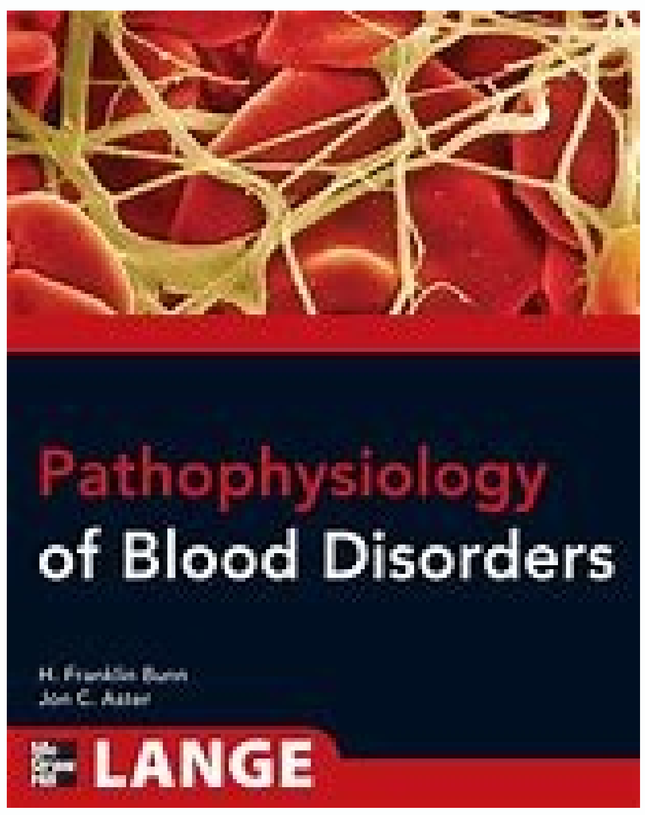

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Medical conditionThalassemiaOther namesThalassaemia, Mediterranean anemiaPeripheral blood film from a person with Delta Beta thalassemiaPronunciation/θæl'si:miə/ SpecialtyHematologySymptomsFeeling tired, pale skin, enlarged spleen, yellowish skin, dark urine[1]CausesGenetic (autosomal recessive)[2]Diagnostic methodBlood tests, genetic tests[3]TreatmentBlood transfusions, iron chelation, folic acid[4]Frequency280 million (2015)[5]Deaths16,800 (2015)[6] Thalassemias are inherited blood disorders characterized by decreased hemoglobin production.[7] Symptoms depend on the type and can vary from none to severe.[1] Often there is mild to severe anemia (low red blood cells or hemoglobin).[1] Anemia can result in feeling tired and pale skin.[1] There may also be bone problems, an enlarged spleen, yellowish skin, and dark urine.[1] Slow growth may occur in children.[1] Thalassemias are genetic disorders inherited from a person's parents.[2] There are two main types, alpha thalassemia and beta thalassemia.[7] The severity of alpha and beta thalassemia depends on how many of the four genes for alpha globin or two genes for beta globin are missing.[2] Diagnosis is typically by blood tests including a complete blood count, special hemoglobin tests, and genetic tests.[3] Diagnosis may occur before birth through prenatal testing.[8] Treatment depends on the type and severity.[4] Treatment for those with more severe disease often includes regular blood transfusions, iron chelation, and folic acid.[4] Iron chelation may be done with deferoxamine, deferasirox or deferiprone.[4][9] Occasionally, a bone marrow transplant may be an option.[4] Complications may include iron overload from the transfusions with resulting heart or liver disease, infections, and osteoporosis.[1] If the spleen becomes overly enlarged, surgical removal may be required.[1] Thalassemia patients who do not respond well to blood transfusions can take hydroxyurea or thalidomide, and sometimes a combination of both.[10] Hydroxyurea is the only FDA approved drug for thalassemia. Patients who took 10 mg/kg of hydroxyurea every day for a year had significantly higher hemoglobin levels, and it was a well-tolerated treatment for patients who did not respond well to blood transfusions.[11] Another hemoglobin-inducer includes thalidomide, although it has not been tested in a clinical setting. The combination of thalidomide and hydroxyurea resulted in hemoglobin levels increasing significantly in transfusion-dependent and non-transfusion dependent patients [12] As of 2015, thalassemia occurs in about 280 million people, with about 439,000 having severe disease.[13] It is most common among people of Italian, Greek, Turkish, Middle Eastern, South Asian, and African descent.[7] Males and females have similar rates of disease.[14] It resulted in 16,800 deaths in 2015, down from 36,000 deaths in 1990.[6][15] Those who have minor degrees of thalassemia, similar to those with sickle-cell trait, have some protection against malaria, explaining why they are more common in regions of the world where malaria exists.[16] Signs and symptoms Left: Hand of a person with severe anemia. Right: Hand of a person without anemia. Iron overload: People with thalassemia can get an overload of iron in their bodies, either from the disease itself or from frequent blood transfusions. Too much iron can result in damage to the heart, liver, and endocrine system, which includes glands that produce hormones that regulate processes throughout the body. The damage is characterized by excessive deposits of iron. Without adequate iron chelation therapy, almost all patients with beta-thalassemia accumulate potentially fatal iron levels.[17] Infection: People with thalassemia have an increased risk of infection. This is especially true if the spleen has been removed.[18] Bone deformities: Thalassemia can make the bone marrow expand, which causes bones to widen. This can result in abnormal bone structure, especially in the face and skull. Bone marrow expansion also makes bones thin and brittle, increasing the risk of broken bones.[19] Enlarged spleen: The spleen aids in fighting infection and filters unwanted material, such as old or damaged blood cells. Thalassemia is often accompanied by the destruction of a large number of red blood cells and the task of removing these cells causes the spleen to enlarge. Splenomegaly can make anemia worse, and it can reduce the life of transfused red blood cells. Severe enlargement of the spleen may necessitate its removal.[20] Slowed growth rates: anemia can cause the growth of a child to slow down. Puberty may also be delayed in children with thalassemia.[21] Heart problems: Diseases, such as congestive heart failure and abnormal heart rhythms, may be associated with severe thalassemia.[22] Hemoglobin structural biology Normal human hemoglobins are tetrameric proteins composed of two pairs of globin chains, each of which contains one alpha-like (α-like) chain and one beta-like (β-like) chain. Each globin chain is associated with an iron-containing heme moiety. Throughout life, the synthesis of the alpha-like and the beta-like (also called non-alpha-like) chains is balanced so that their ratio is relatively constant and there is no excess of either type.[23] The specific alpha and beta-like chains that are incorporated into Hb are highly regulated during development: Embryonic Hbs are expressed as early as four to six weeks of embryogenesis and disappear around the eighth week of gestation as they are replaced by fetal Hb.[24][25] Embryonic Hbs include: Hb Gower-1, composed of two ζ globins (zeta globins) and two ε globins (epsilon globins) (ζ2ε2) Hb Gower-2, composed of two alpha globins and two epsilon globins (α2ε2) Hb Portland, composed of two zeta globins and two gamma globins (ζ2γ2) Fetal Hb (Hb F) is produced from approximately eight weeks of gestation through birth and constitutes approximately 80 percent of Hb in the full-term neonate. It declines during the first few months of life and, in the normal state, constitutes

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