COLOR ATLAS OF PATHOLOGY

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Hematopoietic System • Reticulo-Endothelial System
Respiratory Tract • Cardiovascular System • Liver
Alimentary Tract • Kidney and Urinary Tract
Musculoskeletal System

☆

Illustrated with 1053 figures in color on 365 plates

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Foreword

Somewhere in their medical careers most physicians have heard the dictum from a teacher that "As a doctor's pathology so is he." This observation does not sound particularly sage, but its cogency is both pat and poignant. The doctor who undertakes to cope with the maladies to which the living organism is heir without possessing a knowledge of the fundamental pathologic principles involved merely guesses at what he is doing.

In this exceedingly important field the profession of medicine has for long lacked a comprehensive, concise and realistic source of reference with reproductions in full color which would bring to the student, the clinician, the laboratory diagnostician a readily utilizable and intelligible standard of comparison as a guide in the study and interpretation of both gross and microscopic findings. The kind of treatise needed has been such as would seem most aptly designated by the title, COLOR ATLAS OF PATHOLOGY. The knowledge that this need existed is not new. It has been well recognized moreover that to realize such a goal a number of contingencies would have to be met and a combination of requirements satisfied. I shall not attempt to enumerate the requisites, each a sine qua non, in order of their importance. A prime essential was access to a large store of clinical material. Another foremost necessity was the availability of highly qualified technicians, including artists of a rarely specialized type, capable of processing the material. Add to these elementary requirements the acquisition of funds, the procurement of a publisher interested in the undertaking, and finally, and most essential of all perhaps, a guiding spirit to give a vital spark to the endeavor and to breathe life into it.

At least the potentiality of all these elements existed at the U. S. Naval Medical School in 1944. The highly skilled technicians came to the Navy by reason of the war. The availability of this rare talent at no more expense per capita than is represented by an enlisted man's pay was an important factor.

The possibility of utilizing material in the Pathology Department of the Navy's Medical School, the Army's Institute of Pathology, Johns Hopkins Hospital and Georgetown University Medical School was important. One of the most important advantages offered by these rich stores of available pathologic material was the opportunity to correlate the clinical histories and findings with the pathology. This has added immeasurably to the value of the presentations.

Most important were the services of an individual who had free access to all of these sources and who at the same time possessed an ability to evaluate, correlate and assimilate this rich legacy. World War II provided the Navy with such a man in the person of Doctor Charles F. Geschickter, Commander, Medical Corps, U. S. Naval Reserve, then Chief of Pathology at the Naval Medical School.

The initial definitive move toward the creation of COLOR ATLAS OF PATHOLOGY was made by Doctor Geschickter and Captain Paul Wilson, Medical Corps, U. S. Navy, Medical Officer in Command of the Naval Medical School, when they negotiated in 1944 with the J. B. Lippincott Company for publication of this work. It was Doctor Geschickter who constituted the quarterback of the Naval Medical School's COLOR ATLAS OF PATHOLOGY team when the undersigned assumed command of that institution in 1944, and it was this redoubtable individual who, during the remainder of the war and throughout the

early postwar years bore the brunt of the endeavor and kept the project on schedule. His was the responsibility of supervising the color lithographic work of the entire volume and of writing the text.

Following the release of Doctor Geschickter from active duty it was necessary to find a capable pathologist to share with him the burden of the undertaking. The Navy was fortunate in having available Commander W. W. Ayres, Medical Corps, U. S. Navy, presently Chief of Pathology at the Naval Medical School. His close cooperation with those in charge of the project and his special ability in microphotography, together with his high qualifications as a pathologist, have, along with his untiring efforts, proved invaluable in bringing this work to completion.

Particular and abundant credit must go to Vice Admiral Ross T. McIntire, the wartime Surgeon General of the Navy, with whose blessing and enthusiastic backing the undertaking was launched. It was he who immeasurably strengthened the efforts of Doctor Geschickter and the Naval Medical School's Chief Administrator in obtaining funds for the continuation of the enterprise by employing civilian technicians at the close of the war.

Of paramount importance has been the spirit of the incumbent Surgeon General, Rear Admiral Clifford A. Swanson, under whose dynamic urge the effort of six years has come to its fruition. Second in importance to none, albeit in the early history of this Atlas was Captain Otis Wildman, then Director of Laboratories at the Naval Medical School.

The unqualified support given this undertaking by Rear Admirals William Chambers, T. C. Anderson and M. D. Willcutts, as successive Commanding Officers of the National Naval Medical Center, is hereby accorded grateful recognition.

Aside from Doctor Geschickter's signal and sustained influence and effort from start to finish and listed approximately in the order of their period of participation in the genesis and development of the COLOR ATLAS OF PATHOLOGY, liberal credit is accorded and genuine appreciation is expressed in recognition of the contributions made by the following individuals: First, during the incipient stage of this endeavor a most vital motivating force in its inauguration was Captain Paul Wilson. Following a two-year tenure as Captain Wilson's successor, the writer of this Foreword was succeeded by Captain Otis Wildman, Captain Robert Parsons, Captain Melville Aston, and Captain Bartholomew Hogan as Medical Officers in Command of the Naval Medical School. Without the unflagging support and patient nurturing on the part of these several officers during their respective incumbencies the "baby" could have died a-borning.

In the Department of Pathology at the Naval Medical School, in addition to the great contribution of Captain Wildman, Captain W. M. Silliphant, currently Director of Laboratories, has made notable contributions to this volume. The Chiefs of the several Professional Services at the U. S. Naval Hospital, Bethesda, and the several Executive Officers of the Naval Medical School during the past six years, also cooperated in a wholehearted manner.

Not only was a vast quantity and variety of pathologic material necessary to a satisfactory pursuit of this enterprise, but carefully selected and properly prepared sections were essential. The rejection of many sections and the restaining of new ones was a common occurrence. Alterations and new equipment were required for proper photography and the closest possible teamwork between the photographic department and the pathology laboratory was of prime importance. The pathology department was fortunate in having at the outset and dur-

ing the first year of progress the co-operation of Lieutenant H. F. A. Long (HC), USN. Following his retirement as Chief of the photographic department of the Naval Medical School, Lieutenant Raymond Borland, H(S), USNR, histopathologist extraordinary, and Miss Nell Starkey made outstanding contributions.

In the Department of Hematology Miss Doris Cranmore, Lieutenant, MSC(W), USN, was a noteworthy contributor, and the superb drawings by Miss Annette Conry, of microscopic views typical of various blood dyscrasias, comprise a notable contribution.

When we come to the actual technical field, however, credit for the ultimate success of this COLOR ATLAS OF PATHOLOGY really belongs to a group of civilian artists, lithographers and an assortment of technicians, headed by Mr. Ernest H. Gramatte, who stuck by the project through its most troublous periods with a loyalty and devotion that could not have been exceeded by the most devout service personnel.

In special recognition of the debt of gratitude the Navy and the medical profession owe this group of men, their names and specialties are listed below in everlasting token of appreciation:

Ernest H. Gramatte
Chief, Lithographic Reproduction Division
Glen A. Lee
Chief, Photographic Section
John N. Fournier
Chief, Press Room
Hamilton H. Poole
Foreman, Negative Engraver
Peter A. Spatford
Color Verification Specialist
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Thomas E. Penrod
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Anton Pankau
Color Verification Specialist
Walter O. Harders
Color Verification Specialist
Albert F. Barry
Color Verification Specialist
Arthur L. Mallory
Color Verification Specialist

An expression of appreciation is due Mrs. Gertrude M. Kessel, whose technical contribution was highly valuable, and to Mrs. Clara Gene B. Young for compiling and documenting the clinical histories used and who also bore the brunt of typing and editing the final text. Among others who contributed significantly to the documentation necessary for this treatise in a secretarial or statistical capacity, were Misses Elaine Wilcox, Dorothy Lord and Marjorie Johnson.

Pointed recognition and an expression of genuine gratitude is certainly due representatives of the J. B. Lippincott Company, notably Mr. Ellis W. Bacon and Mr. Walter Kahoe, without whose sympathetic understanding, enduring interest and patience, the consummation of this volume would never have been possible.

Lastly, the writer of this Foreword comes to pay tribute to the memory and to acknowledge the great inspiration which accrued to him during his early days as Medical Officer in Command of the Naval Medical School from one of its illustrious former commanding officers. Reference is, of course, made to the late Rear Admiral Edward Rhodes Stitt. The support and encouragement realized by those in whose hands the fate and destiny of the Atlas rested, from the interest of this scholarly, enthusiastic and profoundly sincere man, was a significant factor in the furtherance of this worthy endeavor in its early stages.

LAMONT PUGH
Rear Admiral (MC), USN,
Deputy Surgeon General

Contents

Diseases of the Hematopoietic System

In the embryo the blood cells are formed from the mesenchyme of the blood islands. Later this mesenchyme forms the endothelium of blood capillaries of bone marrow, lymph nodes, spleen and liver but retains its erythropoietic functions. The mesenchyme associated with this endothelium forms reticulum and gives rise to granulocytes, monocytes and lymphoid elements. In the bone marrow the red blood cells arise within the capillaries from endothelium, while the granulocytes are formed extravascularly from reticulum. The stromal elements about embryonal lymphatics are the antecedents of lymphoid tissue. Reticulum cells in this stroma form large lymphoblasts which develop into small lymphocytes. Large phagocytic cells or histiocytes can arise either from this reticulum, from undifferentiated connective tissue or from the endothelial lining of lymphatic or venous sinuses, in the liver, spleen, lymph nodes, etc. Collectively these tissues which furnish histiocytes are referred to as the reticulo-endothelial system.

The hematopoietic organs and their associated structures are divided into two classes: (1) the erythropoietic tissues and (2) the leukopoietic tissues.

The diseases of the erythropoietic tissues include polycythemia, the various forms of anemia, the purpuras and megakaryocytosis. Primary neoplasia of the erythropoietic system is not definitely established.

The diseases of the leukopoietic tissue include a variety of chronic infections and neoplastic diseases which involve myeloid and lymphoid elements.

DISEASES OF THE ERYTHROPOIETIC SYSTEM

Anemias

The anemias may be divided into three major groups: those resulting from a specific deficiency (deficiency anemias), those resulting from lowered hematopoietic function (hypopoietic anemias), and those caused by increased blood destruction (hemolytic anemias).

Deficiency Anemias

Acute Blood Loss. Severe anemia may result from a deficiency of all the circulating elements, following acute blood loss. This occurs with post-traumatic hemorrhage, ruptured duodenal ulcer, ectopic pregnancy and in hemophilia. The etiology of the condition is exsanguination and the symptomatology is that of shock. The blood volume is first replaced by plasma, and there is a lowering of the hemoglobin, marked increase in the platelet count and a leukocytosis. Rapid loss of one third of the blood volume (1500-2000 cc.) is usually fatal, but 50 per cent may be lost more slowly, over a period of more than 24 hours, without death. The administration of plasma or transfusions may be life saving.

Iron Deficiency. Patients of either sex with chronic iron deficiency usually give a history of chronic blood loss (melena, hemoptysis, epistaxis or metrorrhagia). Iron deficiency may also develop during periods of maximum growth in childhood, puberty and pregnancy because of its increased utilization. The blood picture is of the hypochromic, microcytic type. Because of the iron deficiency the red cells contain less hemoglobin and there is a low mean corpuscular hemoglobin concentration. Fatigability, headache, weakness or dyspnea on exertion are outstanding symptoms. The finger nails may be brittle and spoon shaped. This type of anemia responds to iron therapy.

Deficiency of Anti-Anemic Principle (B_{12}) in Pernicious Anemia. The liver

under normal conditions stores an anti-anemic factor which is absorbed from food of high protein content, such as liver, yeast and eggs. Absorption of this *extrinsic factor* is aided by a factor in gastric secretion, formerly called the *intrinsic factor*. The intrinsic factor is apparently absent in patients suffering achylia in pernicious anemia and in certain stomach disorders. The essential extrinsic factor is vitamin B_{12}. The interaction of these two factors and their absorption from the gastro-intestinal tract provides the anti-anemic principle stored in the liver. Its formation and absorption may be interfered with in cases of diarrhea or steatorrhea (sprue or celiac disease), or its storage in the liver may be reduced in hepatic diseases, such as cirrhosis, with resultant macrocytic anemia. This anti-anemic principle, which is necessary for the maturation of red blood cells, has been designated vitamin B_{12}.

In pernicious anemia caused by vitamin B_{12} deficiency the patients have a characteristic lemon-color tint. Neurologic symptoms, such as parathesias and difficulty in walking, may be prominent, and the anemic triad, pallor, weakness and dyspnea, is present. In sprue, celiac disease and pellagra, there are diarrhea and loss of weight. All patients with macrocytic anemia may suffer with sore tongue and a variety of gastro-intestinal disturbances. The blood findings in this type of anemia are characterized by macrocytosis, hyperchromia and marked variation in the size and shape of the red blood cells. Following specific therapy, signs of blood regeneration (reticulocytosis) are seen. There is leukocytosis in which large multisegmented neutrophilic leukocytes are conspicuous. The platelet count may be lowered. In relapse, the icteric index may be raised to 20 or more units (normal, from 5 to 7) and circulating normoblasts are numerous. There is a marked hyperplasia of the bone marrow in which the count of nucleated red blood cells may reach 50 per cent or more with megaloblasts predominating. This group of anemias responds specifically to liver therapy or vitamin B_{12}.

Hypopoietic Anemias

Aplastic Anemias. The aplastic anemias follow marrow poisoning with benzol, arsenicals, gold or radiant energy (x-ray or radium). Idiopathic aplastic anemia may develop rapidly without known cause. There is a characteristic triad in these cases, consisting of progressively severe anemia, thrombocytopenia and leukopenia. The absence of a palpable spleen helps to distinguish this condition from aleukemic leukemia. The red corpuscles are normal in appearance. Nucleated and stippled forms and other signs of regeneration are absent. The bone marrow is hypoplastic. The white count usually shows 70 to 90 per cent lymphocytes. Repeated transfusions are usually the only remedy.

Repressive Anemia. This anemia is the simple chronic type, and occurs symptomatically in chronic infections, renal disease, malignancy, endocrine disorders, pregnancy and vitamin deficiencies other than B_{12}. Erythropoiesis is inhibited by circulatory toxins or other causes. The red cell count rarely falls below 3.5 million. The number of cells, their size and the quantity of hemoglobin are proportionately decreased, so that the anemia is normocytic. Evidence of regeneration or increased blood destruction is usually absent.

Myelophthisic Anemias. This form of anemia is produced by replacement of the marrow by nonfunctioning elements. Metastatic carcinoma to bone, multiple myeloma, myelosclerosis and lipoid storage diseases affecting bones are etiologic factors. Pain referred to the skeleton and splenomegaly are often present. The anemia is

variable in degree and most often normocytic. Primitive red blood cells or leukocytes may be found in the peripheral circulation, together with reticulocytes and stippled cells. Roentgenography of the skeleton aids in diagnosis. Extramedullary hematopoiesis may be present.

Hemolytic Anemias

The hemolytic anemias are characterized by excessive blood destruction which results in such symptoms as jaundice, formation of gallstones, and increased amounts of urobilinogen and urobilin in the stools and urine. Phagocytosis of red blood cells undergoing destruction with deposition of hemosiderin results in splenomegaly. The acute forms result from blood-stream infections or hemolytic poisons. The chronic forms may be congenital disorders characterized by abnormal forms of hemaglobin and include familial hemolytic icterus, Mediterranean anemia and sickle cell anemia.

Acute Hemolytic Anemia. Acute hemolytic anemia may result from mismatched transfusions, hemolytic toxins, such as snake venoms, bacterial toxins, phenyl hydrazine or phenol, and from malaria or bartonellosis. The symptoms include chills and rigor, vomiting and diarrhea, pain in the back and legs, hemoglobinuria, albuminuria and anuria. The red blood cells show marked variation in size and staining reaction. There is striking evidence of regeneration, including reticulocytosis and circulating normoblasts. Treatment consists of removal of the cause.

Acquired and Congenital Hemolytic Icterus. In congenital hemolytic icterus repeated crises of blood destruction occur, with fever, abdominal pain and nausea. Once severe anemia and jaundice develop, they tend to persist. The spleen is enlarged, gallstones are found in 68 per cent of the cases, skeletal anomalies such as "tower" skull occur, and chronic leg ulcers are common. Reticulocytes, small dense red cells (spherocytes) and increased fragility of red cells to hypotonic salt solution are the chief blood findings. The acquired form is similar to the congenital, but the increased fragility of the red blood cells may be absent. Both forms show an increased icteric index, a hyperplastic bone marrow and are benefited by splenectomy.

Sickle Cell Anemia. This is a hereditary disease in Negroes. It is characterized by the appearance of sickle-shaped red corpuscles in blood which has been standing away from oxygen two to six hours after it has been withdrawn and protected from drying. The cells show increased resistance to hypotonic salt solution. The patients suffer from jaundice, "rheumatic" pains in the extremities, leg ulcers, and irregular thickenings of the cortex of the bones. There is no satisfactory treatment.

Mediterranean Anemia (Thalassanemia). This is a familial disease in individuals of Mediterranean stock. Those afflicted are often short in stature and have a large head. The bones about the hands and wrists may have increased density on roentgen examination. Examination of the blood shows variations in the size and staining reactions of the red blood cells. Nucleated forms are common. The hemoglobin is concentrated at the periphery of the red corpuscle or as a central dot forming so-called target corpuscles. Uniformly colored cells are rare. There is often a leukocytosis. The icteric index is moderately increased. The spleen is enlarged and infarcts are common. Later the organ shrinks and is fibrosed. The bone marrow is hyperplastic. Severe forms are present in children; milder forms in adults. Transfusion is of temporary value but there is no specific treatment.

Erythroblastosis Fetalis. This is a hemo-

lytic anemia, occuring late in fetal life or in the newborn, in a child whose blood agglutinogens differ from its mother's. As a regenerative response to blood destruction, the hematopoietic centers in the bone marrow, liver and spleen become crowded with erythroblasts, hemosiderin and foci of hematopoiesis. Nucleated red blood cells appear in the peripheral blood. The infant in severe cases is edematous and jaundiced, and hence the terms "fetal hydrops" and "familial icterus gravis." The basal ganglia of the brain may be deeply icteric, so-called kernicterus.

In rare instances the incompatibility of the fetal and maternal bloods is because of the ABO grouping. However, the cause of the disease nearly always is the immunization of the Rh negative mother by previous transfusions with Rh positive blood cells or by Rh positive red cells of the fetus. The mother's Rh antibodies pass into the fetal circulation and damage the child's erythrocytes. The offspring of an Rh positive father and Rh negative mother always will be Rh positive if the male is homozygous (all dominant genes for Rh). If the Rh positive father is heterozygous (one or two of the genes recessive) 50 per cent or less of the offspring will be Rh positive. Levine found that 87 per cent of the white population is Rh positive. The mothers of infants developing erythroblastosis fetalis belong to the 13 per cent who are Rh negative. The treatment consists of an adequate number of transfusions or exchange transfusions from a compatible Rh-negative donor.

Polycythemia

The term polycythemia is applied to conditions in which the number of circulating red corpuscles is increased. Erythrocytosis indicates a symptomatic response to anoxia, which may occur in cardiac or pulmonary disease, or at high altitudes. Erythrocytosis is also produced by hypertrophy or tumors of the adrenal cortex, by certain poisons, such as aniline and its derivatives, phosphorus, and occasionally by some metals.

Erythremia or polycythemia vera is a chronic disease of obscure origin, with an increase in the total blood volume and the number of red blood cells. The patients have a plethoric complexion, splenic and hepatic enlargement and a variety of neurologic and vasomotor disturbances. Venous thromboses, bleeding tendency, varicosities and phlebitis, or arterial occlusion are common, due to alteration in the blood. Thrombi in the cerebral vessels may lead to hemiplegia or paresis. The red cell count varies from 7 to 10 million, as a rule. The hemoglobin may reach as high as 25 Gm. (140%). The individual red corpuscles appear normal. Leukocytosis is frequently present, and the platelet count may reach 4 million. Some patients develop leukemia.

Purpura

Essential Thrombocytopenia (Purpura Hemorrhagic, Idiopathic Purpura). This disease is characterized by diminution of the platelets with prolonged bleeding time and a nonretractile blood clot. The coagulation time in glass is not markedly affected. Clinically, there are spontaneous hemorrhages—epistaxis, menorrhagia and petechiae in the skin or mucous membranes. Children and young adults are most frequently affected. Capillary resistance is diminished and the so-called tourniquet test is positive. In order to make a diagnosis of primary purpura it is necessary to rule out acute leukemia or aplastic anemia. There is no enlarged spleen, such as occurs in Banti's disease, Gaucher's disease or hemolytic jaundice. The blood shows a reduction of platelets, fewer than 60,000 per cu. mm. and the platelets vary in size and

staining characteristics. There may be an anemia due to blood loss, and a posthemorrhagic leukocytosis with relative lymphocytosis. The marrow shows increased numbers of megakaryocytes but inhibited platelet production. Small, repeated transfusions of whole blood are temporarily beneficial. Splenectomy may be curative.

Anaphylactoid Purpura (Schonlein and Henoch's Purpura). This condition is due to increased permeability of the capillary endothelium which permits extravasation of blood into the tissue spaces. Henoch's variety is characterized by colic and other gastro-intestinal symptoms, which often precede the purpuric eruptions so that needless operations may be performed. Schonlein's purpura is characterized by effusion into the joints and periarticular structures, which may precede the purpuric manifestations so that an erroneous diagnosis of rheumatic fever may be made.

Myeloid Metaplasia (Megakaryocytic Myelosis). A number of cases have been reported which have in common megakaryocytic infiltrations of the bone marrow, liver and spleen. At times there is a marked leukocytosis with increased number of megakaryocytes in the peripheral blood. The constant features of the disease are the splenomegaly, the infiltration of the hematopoietic organs with megakaryocytes, the tendency for the bone marrow to undergo progressive fibrosis and frequent fatal termination. Bacilli which take the acid-fast stain have been found in the lungs of some of these patients. Because of the involvement of the bone marrow by fibrosis, and the compensatory hematopoiesis in the spleen, splenectomy is contraindicated.

DISEASES OF THE LEUKOPOIETIC SYSTEM

Infectious Mononucleosis, Infectious Lymphocytosis, and Tropical Eosinophilia. Infectious mononucleosis is a febrile disease of probable viral origin, affecting the reticulo-endothelial system and producing lymphadenopathy, splenomegaly and increase of mononuclear cells in the peripheral blood. The leukocyte count may be as high as 40,000 and contain 50 to 90 per cent of large lymphocytes, many of which are atypical and known as Downey cells. A positive test for heterophile antibodies distinguishes this disease from a similar one in children known as infectious lymphocytosis, in which this test is negative.

In tropical eosinophila the white count may be as high as 15,000 with 60 per cent or more eosinophils of the mature type. Many of the patients give a past history of malarial infection and persistent respiratory complaints and at times a low-grade fever. There is no demonstrable cause. Recovery occurs without specific treatment.

Agranulocytosis. This disease is characterized by an absence or a marked decrease in the number of neutrophils in the differential count. Most cases have been reported in patients who have acquired a sensitivity to certain drugs, among them amidopyrine, organic arsenicals, sulfonamides, dinitrophenol, thiouracil, and gold salts. There is a low white count but no anemia. The platelet count and the bleeding and coagulation times are normal. The sedimentation is increased. The patient suffers from fever, weakness and usually from gangrenous ulcerations and infection of the throat (agranulocytic angina). Other severe infections and septicemia may occur unless penicillin is administered during the leukopenic phase of the disease.

Leukemoid Reaction. This is characterized by an elevated leukocyte count that closely resembles leukemia. Such leukocytic reactions have been found occasionally in various infections, chemical and drug poi-

sonings, severe burns, severe hemorrhages or sudden hemolysis of blood and in metastases to bone. The clinical history, subsequent course, or bone marrow biopsy may be necessary for differential diagnosis.

Leukemia. The various forms of leukemia are fatal diseases characterized by uncontrolled proliferation of leukopoietic tissue. The cell may be myelocytic, lymphocytic or monocytic. The diseases may be acute, subacute, or chronic. In the acute form, myeloblasts, lymphoblasts or monoblastic cells are seen in the peripheral circulation or are found in increased numbers in the bone marrow. In the chronic varieties immature cells more highly differentiated than the "blast" stage are found either in the circulation or in predominant numbers in the bone marrow. Anemia is present and is more severe in the acute or terminal phases; it is associated with thrombocytopenia and a tendency to hemorrhage. Not all leukemias have a high white count, even though the leukopoietic tissues are hyperplastic, because the marrow may be unable to mobilize the cells. In such cases the white count will be low or normal with the presence of a few immature leukocytes. Such leukemias are called aleukemic, leukopenic or subleukemic.

In nearly all forms of leukemia the liver, spleen and lymph nodes are infiltrated, in addition to the bone marrow. The liver is often conspicuously enlarged. In myelogenous leukemia, the spleen may weigh as much as 5,000 Gm. and a diagnosis of this form of leukemia is favored clinically when increased numbers of atypical leukocytes are found in the circulating blood in the presence of pronounced splenomegaly. In lymphocytic and monocytic leukemia, the lymph nodes are usually enlarged. Inflamed or bleeding gums are often important clinical manifestations. Secondary infiltrations may be found in any organ in all varieties of leukemia. Involvement of the skin is known as leukemia cutis. Massive growths may occur in the osseous or periosseous structures. Terminal infections or hemorrhages are usually associated with the profound anemia or thrombocytopenia that results from leukemic replacement of the normal marrow.

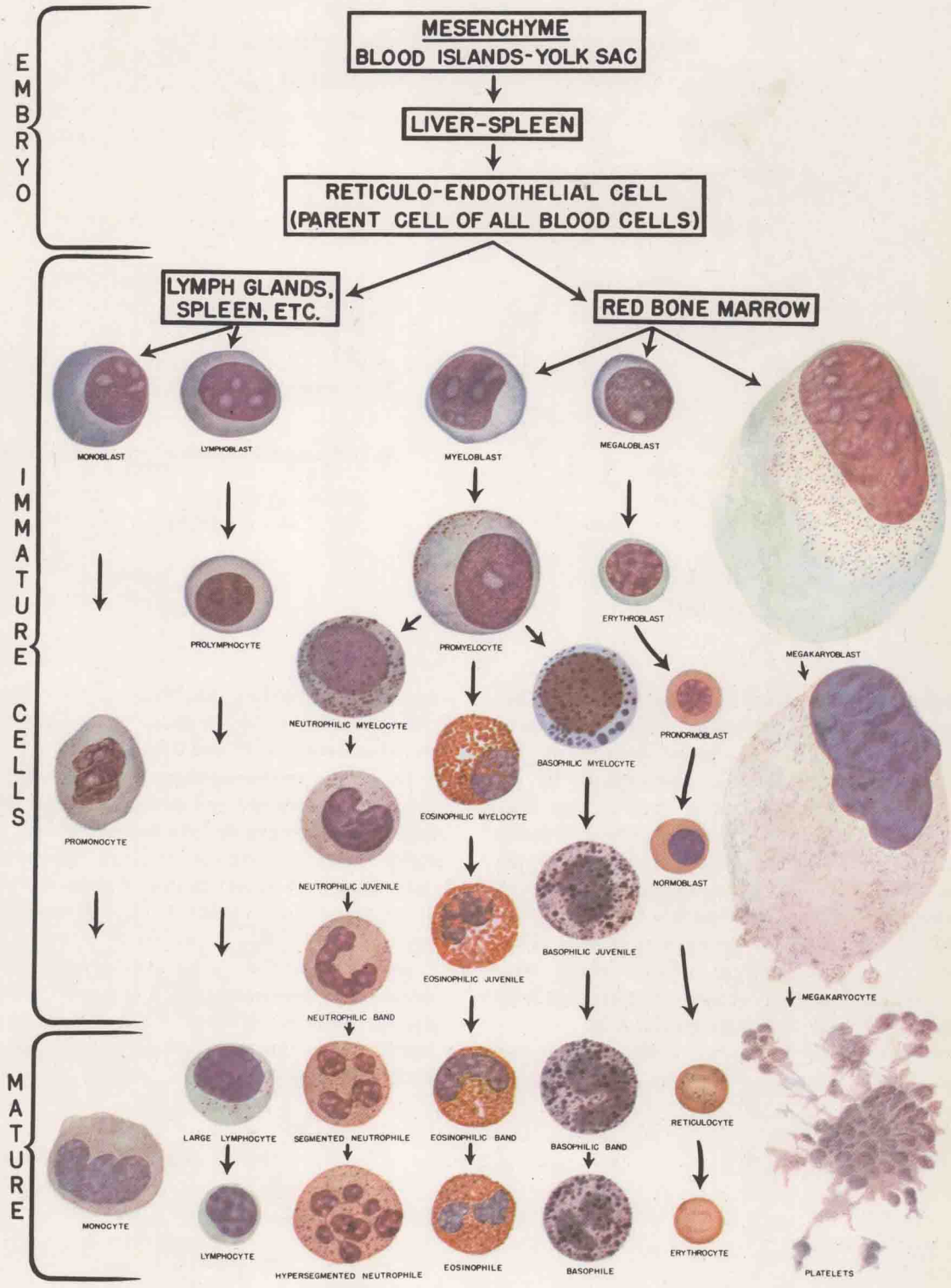
Eosinophilic Leukemia. Wintrobe states that about 20 cases of eosinophilic leukemia have been reported with a fatal course. Enlargement of the spleen, liver and lymph nodes occurs and symptomatic purpura may develop. A leukocyte count as high as 200,000, with over 90 per cent eosinophils, may occur.

Plasma Cell Leukemia. Plasma cell leukemia has not been reported in the absence of multiple myeloma of the bones or extramedullary plasmacytoma except in a few rare instances. The signs suggestive of leukemia are circulating plasma cells, a total leukocyte count as high as 60,000 or more and anemia.

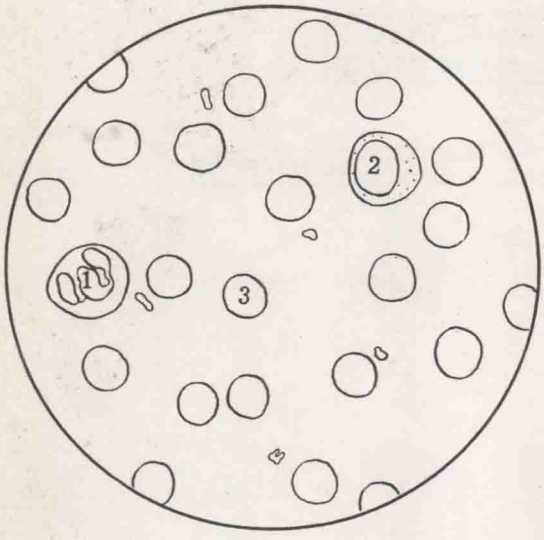
Chloroma. When tumor nodules of leukopoietic tissue (with a green color) are found in the bone marrow associated with a leukemic blood count, the condition is known as chloroma. The neoplastic cells may be either myelogenous or monocytic in such conditions. Fever, joint manifestations, and periorbital tumors are common. The disease is always fatal.

Multiple Myeloma. In this disease the marrow contains one or more tumor nodules or infiltrations composed of plasma cells. This entity is described in the section on Diseases of the Musculoskeletal System.

DEVELOPMENT OF BLOOD CELLS X 1500



ACUTE POSTHEMORRHAGIC ANEMIA
(Normocytic Normochromic Anemia)



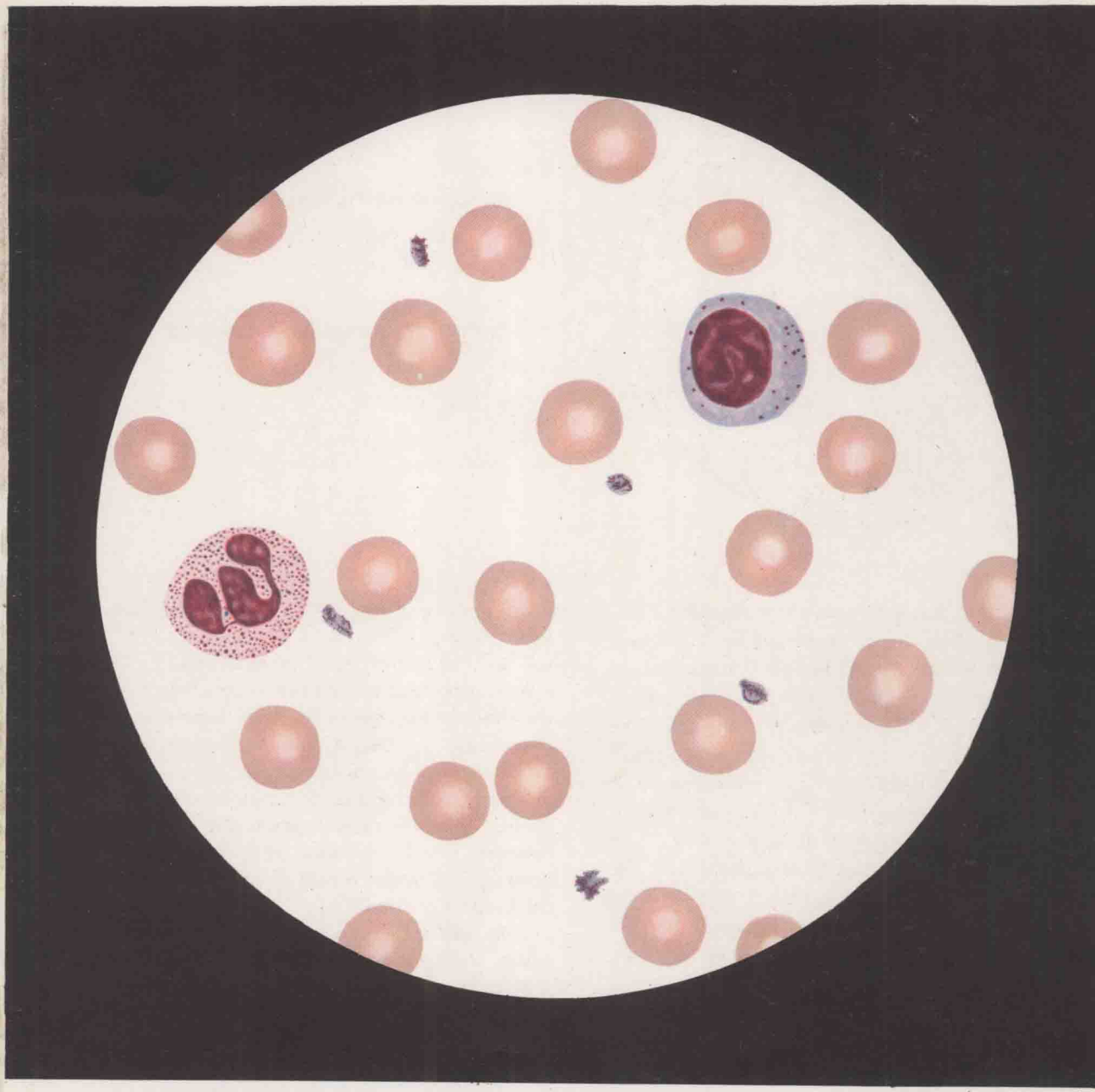
1. Segmented Neutrophil
2. Lymphocyte
3. Normocytic Normochromic Erythrocyte

The patient, a white male, age 36, gave a history of rapidly developing faintness, nausea, sweating and pallor. His blood findings at that time were: WBC 11,000, neutrophils 70 per cent; RBC 5,000,000, hemoglobin 96 per cent, reticulocytes 1 per cent. When admitted 36 hours later his RBC was 2,600,000, hemoglobin 50 per cent; WBC 13,500, neutrophils 76 per cent, reticulocytes 5 per cent. Platelets were increased. The red blood cells were normal in size, shape and hemoglobin content. Tarry stools were passed and a diagnosis of acute hemorrhage from a duodenal ulcer was later established.

In acute blood loss the red blood count and hemoglobin determination give misleading information regarding blood loss until the dilution of the blood by the tissue fluids has restored the blood volume 12 to 72 hours later.

In acute posthemorrhagic anemia the bone marrow restores the red blood count to normal in about five weeks and the hemoglobin in about eight weeks. During the first ten days reticulocytes increase 5-15 per cent. A transfusion of 500 cc. of blood will raise the hemoglobin about ten per cent.

In addition to acute posthemorrhagic anemia, normocytic normochromic anemia occurs in conditions causing destruction of blood, lack of blood formation and also in the physiologic hydremia of pregnancy.



Acute Posthemorrhagic Anemia (Normocytic Normochromic Anemia) X 1800