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Eponyms in Hematology: A Tabulation Overview

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Abstract:

Hematology is the science or study of blood, blood-forming organs and blood diseases. There are several eponyms in hematology literature.

The aim in this review is to shed some lights on the hematological eponyms.

Key words: Diseases, Eponyms, Hematology

Hematology is the science or study of blood, blood-forming organs and blood diseases. Four major areas of study within hematology include hemoglobinopathy, hematological malignancies, anemia and coagulopathy.

There are several eponyms in the hematology literature [1-62]. We have listed some of them in the following table.

As in other medical specialties, the usage of some eponyms remains constant over the years, whereas the others were replaced by other names.

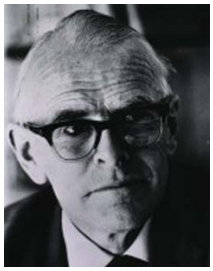
It is to be noted that some major contributions in hematology were not eponymously credited.

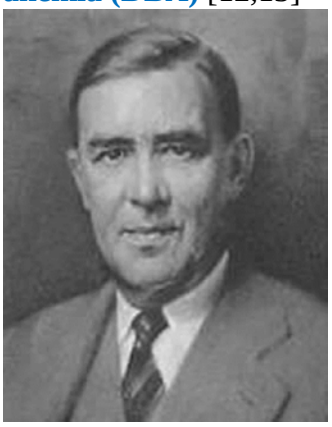
For examples; Platelets were discovered by Giulio Bizzozero; sickle cell was first described by Ernest Edward Irons; and pure red cell aplasia by Paul Kaznelson, but none were named eponymously.

Selected eponyms in hematology

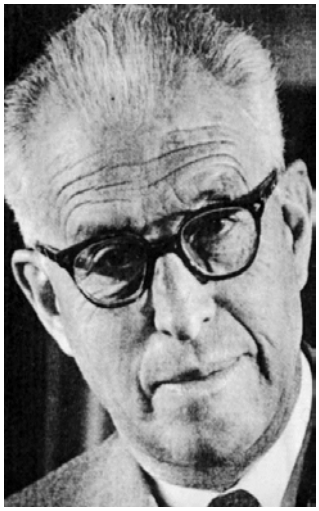
Eponyms in hematology	Remarks
Alder-Reilly anomaly [1]	Alder's anomaly was first described by Alder in 1939 and by Reilly in 1941. It is an autosomal recessive inherited disorder characterized by the presence of large azurophilic and basophilic granules in cells of the myeloid and lymphocytic series. The Alder-Reilly anomaly is seen in the mucopolysaccharidoses. The most characteristic finding is the metachromatic granules surrounded by a clear zone seen in lymphocytes. Dense granules, resembling toxic granulation in neutrophils, are seen in all leukocytes. Named for Albert von Alder (1888-1951), who was a Swiss haematologist.William Anthony Reilly was an American paediatrician, born 1901.
Auer rods [2,3] John Auer (1875 – 1948) 	Auer rods are clumps of azurophilic granular material that form elongated needles seen in the cytoplasm of leukemic blasts. First described by John Auer, who was an American physiologist and pharmacologist. Faggot cell is a term used for cells normally found in the hypergranular form of acute promyelocytic leukemia (FAB - M3). The accumulation of the Auer rods inside these cells gives the appearance of a bundle of sticks, from which the cells are given their name.

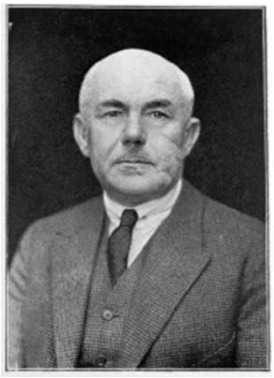
Bernard–Soulier syndrome (BSS) [4,5]  Jean Bernard (1907 – 2006)  Jean Pierre Soulier (1915 –2003)	Also called hemorrhagiparous thrombocytic dystrophy. It is a rare autosomal recessive coagulopathy (bleeding disorder) that causes a deficiency of glycoprotein Ib (GpIb), the receptor for von Willebrand factor, an important glycoprotein involved in hemostasis. BSS is a giant platelet disorder, meaning that it is characterized by abnormally large platelets. The syndrome is named after Dr. Jean Bernard and Dr. Jean Pierre Soulier Jean Bernard (1907 – 2006), was a French physician and haematologist. He was professor of hematology and director of the Institute for Leukaemia at the University of Paris. Dr. Jean Pierre Soulier (1915 – 2003), was a French physician and haematologist. He was the General Director of Centre National de Transfusion Sanguine (CNTS) Paris and professor of hematology at the University of Paris, at the Necker Hospital for Sick Children.
Birbeck bodies [6]	These are Tennis-racquet-shaped cytoplasmic bodies seen by electron microscopy in Langerhans cells. They were discovered by Michael Stanley Clive Birbeck (1925-2005), a British scientist and electron microscopist. Langerhans cells are dendritic cells (antigen-presenting immune cells) of the skin and mucosa. It is named for Paul Langerhans (1847-1888), who was a German pathologist. Langerhans cell histiocytosis (LCH) which is a rare disease involving clonal proliferation of Langerhans cells , is also named after him.

Burkitt's lymphoma [7]  Denis Parsons Burkitt (1911-1993)	Burkitt lymphoma is an aggressive non-Hodgkin lymphoma which can be classified into endemic, sporadic, and immunodeficiency variants. Although each variant frequently involves extranodal sites, cutaneous involvement with Burkitt lymphoma is very rare. This lymphoma is named after, Denis Parsons Burkitt British surgeon (1911-1993), who first described the disease in 1956 while working in equatorial Africa.
Chédiak–Higashi syndrome [8]	A rare autosomal recessive disorder caused by a qualitative defect in leukocyte function, characterized clinically by partial oculocutaneous albinism, recurrent bacterial infections, photophobia, and peripheral neuropathy. It is named for the Cuban physician and serologist Alejandro Moisés Chédiak (1903–1993) and the Japanese pediatrician Otokata Higashi (1883–1981).
Cooley's anaemia [9]  Thomas Benton Cooley (1871-1945)	Is another name for Beta Thalassemia is an inherited disorder that affects the production of normal hemoglobin (a type of protein in red blood cells that carries oxygen to the tissues of the body). Thalassemia includes a number of different forms of anemia. Beta thalassemia is caused by mutations in the beta chain of the hemoglobin molecule. Named for Thomas Benton Cooley (1871 –1945), who was an American pediatrician and hematologist. William Dameshek was an American physician and hematologist, 1900-1969.In 1946 William Dameshek was founding editor of the journal Blood, published by PA Saunders in Philadelphia. The Dameshek Prize from American Society of Hematology is named in his honour.

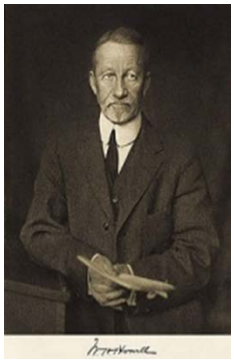
Di Guglielmo's disease [10,11]  Giovanni Di Guglielmo (1886-1961)	A syndrome of unknown origin characterized by enormous numbers of nucleated red cells appearing in the bone marrow and blood. Named for Giovanni Di Guglielmo (1886-1961), who was an Italian hematologist.
Diamond–Blackfan anemia (DBA) [12,13]  Figure.8:Kenneth Blackfan (1883 - 1941)	DBA also known as Blackfan-Diamond anemia, inherited pure red cell aplasia and as inherited erythroblastopeniais .It is a congenital pure red cell aplasia often associated with skeletal malformations. Mutations in ribosomal protein coding genes, mainly in RPS19, account for the majority of DBA cases. It is first noted by Joseph in 1936. However, it is named for Diamond and Blackfan, who described congenital hypoplastic anemia in 1938.Louis Klein Diamond (1902 –1999) was an American pediatrician, known as the "father of pediatric hematology"Kenneth Blackfan (1883 - 1941), was an American pediatrician. He took particular interest in nutrition and hematology. Early in his career, Blackfan did work that identified the origin of cerebrospinal fluid.
Döhle bodies [14]	Döhle bodies are light blue-gray, oval, basophilic, leukocyte inclusions located in the peripheral cytoplasm of neutrophils. They are named after German pathologist, Karl Gottfried Paul Döhle (1855-1928). They are often present in conjunction with toxic granulation.

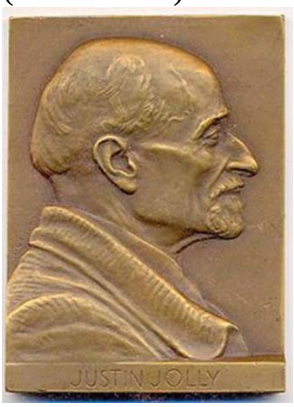
Dresbach's anemia	Elliptocytosis, first described by Dresbach1 in 1904A hematological disorder characterized by the presence of elliptical erythrocytes in the blood. Melvin Dresbach American Physician, 1874-1946. Philadelphia.
Dutcher Bodies [6]  William Russell (1852-1940).	Dutcher bodies are PAS-positive, diastase-resistant nuclear pseudoinclusions of eosinophilic cytoplasm found in plasma cells described by Dutcher and Fahey in Waldenstrom macroglobulinemia. Dutcher bodies are a feature of clinically indolent, mucosa-associated lymphoid tissue (MALT) lymphomas. There are no essential differences between Dutcher bodies, single or multiple Russell bodies, and the inclusions of Mott cells. They are all aspects of the same phenomenon, representing spherical cytoplasmic inclusions that are either clearly within the cytoplasm or are overlying the nucleus or invaginated into it. Russell bodies, is named after William Russell (1852-1940), Scottish pathologist and physician. Mott cell is named after Mott, who described it in 1905. Dutcher bodies may rarely occur in a benign reactive condition, such as synovitis. While Dutcher bodies may be a clue to the presence of low-grade lymphoma, they are not a definitive feature, particularly in unusual contexts.

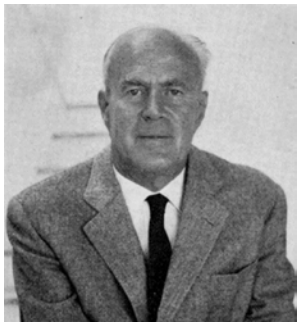
Evans syndrome [15]	Evans syndrome is a rare autoimmune disorder characterized by simultaneous or sequential presence of a positive anti-globulin test, autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP). Evans syndrome was first described in 19512 and it is recognized as a poor prognostic factor in autoimmune cytopenias. Its etiology and cause are unknown, but alterations in immune regulation mechanisms are documented. The syndrome was first described in 1951 by R. S. Evans and colleagues
Factor V Leiden thrombophilia [16]	Factor V Leiden is the most common hereditary hypercoagulability disorder amongst ethnic Europeans. It is named after the city Leiden (Netherlands), where it was first identified in 1994 by Prof R. Bertina et al.
Fanconi's anemia (FA) [17]  Guido Fanconi (1892-1979)	It is a very rare genetic disease which result from a genetic defect in a cluster of proteins responsible for DNA repair. As a result, the majority of FA patients develop cancer, most often acute myelogenous leukemia, and 90% develop bone marrow failure by age 40. In FA, there are genetic defects of DNA repair mechanisms, which share many clinical features such as growth retardation, neurological disorders, premature ageing, skin alterations including abnormal pigmentation, telangiectasia, xerosis cutis, pathological wound healing as well as an increased risk of developing different types of cancer. It is named for, Guido Fanconi (1892-1979), a Swiss pediatrician. His name is also linked to Fanconi syndrome (osteomalacia, aminoaciduria, hyperphosphaturia, glycosuria and aciduria). Fanconi is regarded as one of the founders of modern pediatrics.

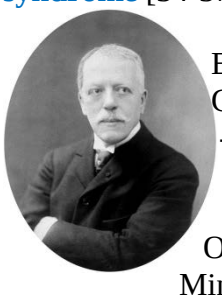
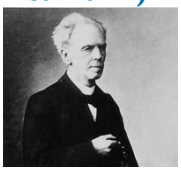
Giemsa stain [18]  Gustav Giemsa (1867-1948).	It is a classic blood film stain for peripheral blood smears and bone marrow specimens. Stains myeloid and mast cell granules purple (it is the heparin in the mast cells that is staining). Also good for many types of organisms, including bacteria, Leishmania, malaria and Histoplasma. Gustav Giemsa (1867-1948), was a German chemist and bacteriologist. Giemsa improved the Romanowsky stain (Eosin Yand Methylene Blue) by stabilizing this dye solution with glycerol. This allowed for reproducible staining of cells for microscopy purposes.
Glanzmann's thrombasthenia [19,20] Eduard Glanzmann (1887-1959) 	Thrombasthenia Glanzmann, named after the Swiss pediatrician Eduard Glanzmann (1887-1959), is a rare disease of platelet dysfunction. This disease is characterized by a deficiency or defect of the fibrinogen receptor (GPIIb-IIIa) on the platelet surface. The GPIIb-IIIa receptor has an essential function in the adhesion and aggregation of the platelets. The platelets of these patients cannot bind fibrinogen and aggregation does not occur. Patients have a severe lifelong risk of bleeding, especially during surgical procedures.
Griscelli syndrome [21] Claude Griscelli, 	It is a rare autosomal recessive disorder characterized by albinism (hypopigmentation) with Immunodeficiency, that usually causes death by early childhood. It is caused by mutations in either the myosin VA (GS1), RAB27A (GS2) or melanophilin (GS3) genes. The three GS subtypes are commonly characterized by pigment dilution of the skin and hair, due to defects involving melanosome transport in melanocytes. It is named after Claude Griscelli, born in 1936, professor of pediatry at Hôpital des Enfants-Malades in Paris.

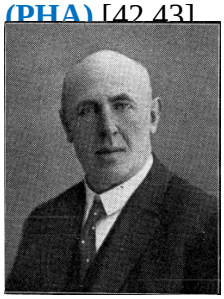
Heinz bodies [22] Paul Ehrlich (1854-1915) 	Also known as Heinz-Ehrlich bodies. These are inclusions with in red blood cells composed of denatured hemoglobin. They are named after Robert Heinz (1865–1924), a German physician who in 1890 described these inclusions in connection with cases of hemolytic anemia. Paul Ehrlich (1854-1915), was a German physician and scientist. In 1908, he received the Nobel Prize in Physiology or Medicine for his contributions to immunology. Heinz bodies are found in glucose-6-phosphate dehydrogenase deficiencies but also found in congenital hemolytic anaemias and in premature infants.They appear as small round inclusions within the red cell body, though they are not visible when stained with Romanowsky dyes. They appear more clearly when supravitally stained (e.g., with new methylene blue or bromocresol green). Damaged cells are cleared by macrophages in the spleen, where the precipitate and damaged membrane are removed, leading to characteristic "bite cells". The denaturing process is irreversible and the continual elimination of damaged cells leads to Heinz body anemia. They are best seen when blood films are stained with dyes such as crystal violet or methylene blue.
Hermansky–Pudlak syndrome [23] Frantisek Hermansky (1916-1980)  Pavel Pudlak (1927 – 1993) 	It is a rare multisystemic, disorder characterized by oculocutaneous albinism, and a bleeding diathesis, sometimes accompanied by immunodeficiency and other features. Named for 2 Czech internists; Frantisek Hermansky (1916-1980), and Pavel Pudlak (1927-1993), . Pudlak served as chairman of the Czechoslovak Society of Hematology in the years 1982-1986. Along with F. Heřmanským, he is awarded State Prize for the discovery and Hermansky Pudlak syndrome.

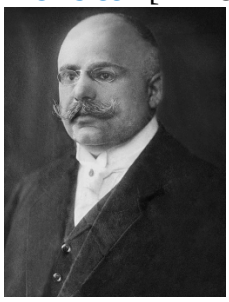
Herrick's syndrome [24,25]  James Bryan Herrick (1861-1954)	Another name for sickle-cell anemia .Named for James Bryan Herrick (1861 - 1954) , who was an American physician .He is credited with the description of sickle-cell disease and was one of the first physicians to describe the symptoms of myocardial infarction. Research showed that Soviet physicians V.P. Obraztsov and N.D. Strazhesko; describe the symptoms of myocardial infarction before James B. Herrick.
Hodgkin lymphoma [7]  Thomas Hodgkin (1798-1866) Dorothy Reed (1874-1964) 	Cutaneous Hodgkin's disease is a rare condition that usually occurs late in the course of Hodgkin's lymphoma. Hodgkin lymphoma was named after Thomas Hodgkin, who first described abnormalities in the lymph system in 1832. Thomas Hodgkin (1798-1866) was an English physician and pathologist. The multinucleated Reed–Sternberg cells (RS cells) are the characteristic histopathologic finding of this disease. This type of cells are named after Dorothy Reed (1874-1964) an American pathologist, and Carl Sternberg (1872-1935), an Austrian pathologist.
Howell–Jolly bodies [26,27] 	These are histopathological findings of basophilic nuclear remnants (clusters of DNA) in circulating erythrocytes. During maturation in the bone marrow late erythroblasts normally expel their nuclei, but in some cases a small portion of DNA remains. Its presence usually signifies a damaged or absent spleen. Understanding the process by which red cell precursors lose their nuclei developed in the late 19th and early 20th centuries led to the identification of nuclear remnants in circulating red cells in certain pathological states, particularly absence or decreased function of the spleen.

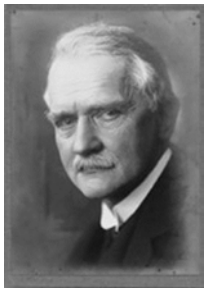
Howell–Jolly bodies [26,27] William Henry Howell (1860 – 1945)  	William Howell, an American, and Justin Jolly, a Frenchman, were among a number of early contributors to this field. Early on, their names were applied, singly or in tandem, to these red cell inclusions, and the eponym, Howell-Jolly bodies, has stuck. It was, however, not until after the mid-20th century that Howell-Jolly bodies were clearly differentiated from basophilic stippling and that the mechanisms of their formation and removal from red cells were understood. William Henry Howell (1860 – 1945), was an American physiologist. He pioneered the use of heparin as a blood anti-coagulant. Justin Marie Jolly (1870 – 1953), was a French hematologist and histologist .
Jenner's Stain [28,29]	Jenner's Stain (methylene blue eosinate) is used in microscopy for staining blood smears. Methylene blue was synthesized by Caro in 1876 at BASF, a chemical company. Six years later, Koch employed methylene blue when he discovered the tubercle bacillus. In 1880, Ehrlich described what he termed "neutral" dyes: mixtures of acidic and basic dyes for the differentiation of cells in peripheral blood smears. Today, the Malachowski-Wright-Giemsa stain continues to be regarded as the world's standard diagnostic technique for malaria.
Leishman stain [28,29]  William Boog Leishman (1865 –1926)	Leishman's staining method for thin and thick smears is a good alternative to Giemsa's stain for identifying Plasmodium parasites. The Leishman method is superior for visualization of red and white blood cell morphology. It is based on a methanolic mixture of "polychromed" methylene blue (i.e. demethylated into various azures) and eosin. Leishman stain is named after its inventor, the Scottish pathologist William Boog Leishman (1865-1926),

May-Grünwald stain [28-33]	May-Grünwald-Giemsa (MGG) stain is a Romanowsky-type, polychromatic stain as those of Giemsa, Leishman and Wright. Apart being the reference method of hematology, it has become a routine stain of diagnostic cytopathology. Named for German physician Richard May (1863 – 1936) and Ludwig Grünwald (1863-1927) who was A German otolaryngologist. Grünwald worked in Richard May's laboratory in Munich when they developed the solution now known as May-Grünwald stain. He was probably the first to describe the Haller cells in the nasal mucosa, first found by Albrecht von Haller (1708-1777).
May-Hegglin anomaly [30-33] Robert Hegglin (1907–1969) 	MHA is an autosomal dominant disorder, characterized by a variable degree of thrombocytopenia, large platelets and inclusion bodies in white blood cells. Mutations in the gene encoding for nonmuscle myosin heavy chain IIA (MYH9) were identified. Bleeding manifestations are generally mild, but severe bleeding episodes have been reported.MHA is named for German physician Richard May (1863 – 1936) and Swiss physician Robert Hegglin (1907–1969), The disorder was first described by May in 1909 and was subsequently described by Hegglin in 1945. Interestingly, MYH9 is also found to be responsible for several related disorders with macrothrombocytopenia and leukocytes inclusion, including Sebastian, Fechtner, and Epstein syndromes, which feature deafness, nephritis, and/or cataract. MHA also known as Dohle leukocyte inclusions with giant platelets and macrothrombocytopenia with leukocyte inclusions.Karl Gottfried Paul Döhle (1855-1928), was a German pathologist.

Minkowski–Chauffard syndrome [34-37]  Emile Chauffa--rd(1855 -1932) Oscar Minkowski (1858 – 1931) 	Is another name for Hereditary spherocytosis.It is an autosomal dominant abnormality of erythrocytes. Named after, Anatole Marie Émile Chauffard (1855-1932), who was a French internist and Oskar Minkowski (1858-1931), who was a German internist and physiologist. Minkowski was a physician of many talents. He contributed to our understanding of the pathogenesis of diabetes mellitus after removal of the pancreas (experimental pancreatectomy).Hereditary spherocytosis and elliptocytosis are the two most common inherited red cell membrane disorders resulting from mutations in genes encoding various red cell membrane and skeletal proteins.
Neumann's cells (Franz Ernst Christian Neumann)  FEC Neumann (1834-1918)	This term is not in common usage but refers to nucleated cells in the bone marrow developing into red blood cells. Named after Franz Ernst Christian Neumann (1834 - 1918), who was, a German histologist, anatomist, and pathologist.
Osler-Vaquez disease [38,39]  William Osler (1849-1919) Louis Henri Vaquez (1860-1936) 	It is another name for Polycythemia Vera (PV), which is 1 of the 3 Philadelphia-negative myeloproliferative neoplasms. First described In 1892 Clinically, PV is an indolent disease, but its course can be complicated by arterial and venous vascular incidents, evolution to myelofibrosis, or leukemic transformation. The disease is named after physician William Osler (1849–1919), who was a Canadian physician and one of the four founding professors of Johns Hopkins Hospital. Louis Henri Vaquez (1860 – 1936), was a French internist . He is known for his work in the field of hematology and his research of heart disease.

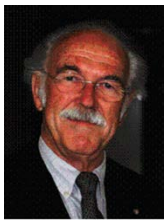
Pappenheimer bodies [40,41]	In 1945, Dr A. M. Pappenheimer described intraerythrocytic collections of iron, or siderotic granules, as they appear on Wright-stained blood smears of certain patients after splenectomy. Confirmation of non-heme iron in the granules is made with a Perls' Prussian blue stain. Pappenheimer bodies are more evident in asplenic individuals; however, they may also be found in patients with alcohol excess, sideroblastic anemia, and lead poisoning. Other red cell inclusions, such as Howell Jolly bodies and Heinz bodies, which are also seen more frequently in asplenic individuals, are negative with iron stains. They can interfere with platelet counts when the analysis is performed by electro-optical counters.
Pelger–Huët anomaly (PHA) [42,43]  Karl Pelger (1885-1931),  Gauthier Jean Huët (1879-1970)	PHA is a recognized morphologic variant affecting all granulocytes but is most evident in polymorphonuclear neutrophils (PMNs). PHA is caused by a decreased amount of the lamin B receptor (LBR). It is a genetic disorder genetic disorder with an autosomal dominant inheritance pattern. It is characterized by a white blood cell type known as a neutrophil whose nucleus is hyposegmented. Heterozygotes are clinically normal, although their neutrophils may be mistaken for immature cells which may cause mistreatment in a clinical setting. Homozygotes tend to have neutrophils with rounded nuclei that do have some functional problems. The characteristic leukocyte appearance was first reported in 1928 by Karl Pelger (1885-1931), a Dutch hematologist. In 1931 Gauthier Jean Huët (1879-1970), a pediatrician, identified it as an inherited disorder.

Pick's cell [44-48]  Ludwig Pick (1868-1944)  Otto Lubarsch (1860 – 1933)	Histiocyte found in the spleen and bone marrow in Niemann-Pick disease, a genetic disorder characterized by the accumulation of sphingomyelin which usually results in progressive enlargement of the liver and spleen (hepatosplenomegaly), along with lymphadenopathy, anemia and mental and physical deterioration. Niemann-Pick disease is named after Professor Ludwig Pick (1868 –1944), , was a German pathologist. Albert Niemann (1880 –1921) was a German physician. Pick's cell is similar in appearance to "Gaucher's cell"; however the cytoplasm of the cell appears foamy. Pick bodies sometimes homogenous, round or ovoid structures found in histiocytes of Pick's cell are seen. They are strongly argyrophilic .These are called Pick bodies. Lubarsch–Pick syndrome is a rare combination of macroglossia with systematized amyloidosis of the skin and skeletal muscles. Otto Lubarsch (1860 –1933), was a German pathologist
Plummer–Vinson syndrome (PVS), also called Paterson–Brown–Kelly syndrome [49] Henry Stanley Plummer (1874–1936), 	The syndrome presents as a classical triad of dysphagia, iron deficiency Anemia and esophageal webs. The syndrome eponym has been frequently discussed. The most used name is Plummer-Vinson syndrome, named after two Americans, the physician, Henry Stanley Plummer (1874–1936), and surgeon, Porter Paisley Vinson (1890–1959) who were physicians on the staff of the Mayo Clinic. Is occasionally known as Kelly-Paterson syndrome in the UK, Another term is Paterson-Kelly syndrome, named after, Donald Ross Paterson (1863–1939) and Adam Brown-Kelly (1865–1941), both British laryngologists, who published their findings independently in 1919. They were the first to describe the characteristic clinical features of the syndrome

Richter syndrome [7] Maurice Nathaniel Richter 	Richter syndrome (RS) is large-cell transformation of chronic lymphocytic leukemia (CLL). It commonly involves lymph nodes and bone marrow, but may rarely manifest in skin. Certain triggering factors, such as Epstein-Barr virus infection and p53 overexpression, have been implicated in the pathogenesis of RS.It is named for the American pathologist Maurice Nathaniel Richter born in 1897.
Romanowsky staining [28,29] Dmitri Leonidovich Romanowsky (1861–1921), 	Romanowsky staining is a prototypical staining technique that was the forerunner of several distinct but similar methods, including Giemsa, Jenner, Wright, Field, and Leishman stains, which are used to differentiate cells in pathologic specimens. It was named after the Russian physician Dmitri Leonidovich Romanowsky (1861–1921), who invented it in 1891. Paul Ehrlich was the first in developing several methods for staining tissue which made it possible to distinguish between different types of blood cells, which led to the capability to diagnose numerous blood diseases. He invented the precursor technique to Gram staining bacteria.
Schüffner's dots [50] Wilhelm August Paul Schüffner (1867 -1949), 	Schüffner's dots refer to a hematological finding that is associated with malaria, exclusively found in Plasmodium ovale and Plasmodium vivax. They are visible by light microscopy in Romanovsky-stained blood smears as multiple brick-red dots. These morphologic changes, referred to as Schüffner's dots, are important in the identification of this species of malarial parasite and have been associated by electron microscopy with caveolavesicle complexes along the erythrocyte plasmalemma.

Schüffner's dots [50]	They are named for Wilhelm Schüffner, who described them in 1904 Wilhelm August Paul Schüffner (1867 -1949), was a Professor of microbiology and immunology.
Sézary syndrome or Sézary disease [7]  Albert Sézary (1880-1956)	In a series of papers from 1938 to 1949, Albert Sézary (1880-1956) a French dermatologist and syphilologist, described erythroderma with cellules monstrueuses (monster cells) in the skin and blood, which is now known as Sézary syndrome or Sézary disease.
Upshaw–Schulman syndrome (USS) [51] Upshaw 	USS is an extremely rare hereditary deficiency of ADAMTS13 activity, termed congenital thrombotic thrombocytopenic purpuraTTP. The clinical signs are usually mild during childhood, often with isolated thrombocytopenia. But their symptoms become more evident when patients have infections or get pregnant. TTP was first recognized as a disease in 1947. Schulman reported a case of TTP in 1960, and Upshaw published a paper in 1978 about relapsing TTP in a patient whom he had followed for 11 years. In his report Upshaw noted the similarities with the reported case by Schulman and hypothesized that the two cases had similar causes – a missing plasma factor. One year later the disease was named Upshaw-Schulmann Syndrome.
Virchow's triad [52]  Rudolf Virchow (1821-1902),	Virchow's triad describes three factors that contribute to the development of venous thrombosis: hypercoagulability, stasis and endothelial injury. However, the elements comprising Virchow's triad were neither proposed by Virchow, nor did he ever suggest a triad to describe the pathogenesis of venous thrombosis Interestingly, Virchow only began to be routinely credited with this triad one hundred years after publication of his work on venous thrombosis.

Virchow's triad [52]	It is named after the eminent German physician Rudolf Virchow (1821-1902), anthropologist, pathologist, prehistorian, biologist, writer, editor, and politician, known for his advancement of public health.
Von Willebrand disease (vWD) [53]  Erik Adolf von Willebrand (1870-1949)	vWD is the most common inherited bleeding disorder. It is characterized by a deficiency in the clotting protein called von Willebrand's Factor, a protein that is required for platelet adhesion. The most common symptom of the disease is prolonged bleeding time. vWD is named after Erik Adolf von Willebrand (1870-1949) who was a Finnish internist.
Waldenström's macroglobulinemia (WM) [54]  Jan Gösta Waldenström (1906- 1996),	WM, also known as lymphoplasmacytic lymphoma) is cancer affecting B cells, a type of white blood cell. The main attributing antibody is immunoglobulin M (IgM). WM is an "indolent lymphoma," (i.e., one that tends to grow and spread slowly). It is a type of lymphoproliferative disease, which shares clinical characteristics with the indolent non-Hodgkin lymphomas. The disease, first identified in 1944. named after the Swedish oncologist , Jan Gösta Waldenström (1906- 1996), who was a Swedish doctor of internal medicine.
Weibel-Palade bodies (WPB)[6]	WPBs are elongated secretory organelles specific to endothelial cells that contain von Willebrand factor (VWF) and a variety of other proteins that contribute to inflammation, angiogenesis, and tissue repair. They are associated with von Willebrand's disease.

Weibel-Palade bodies (WPB) [6] Ewald Rudolf Weibel  George Emil Palade (1912- 2008), 	Weibel–Palade bodies were initially described, by the Swiss anatomist and biologist, Ewald Rudolf Weibel, born 1929, and the Romanian physiologist George Emil Palade (1912- 2008), Palade was described as „the most influential cell biologist ever". In 1974 he was awarded the Nobel Prize in Physiology and Medicine, for his work on the function of organelles in cells together with Albert Claude and Christian de Duve.
Wright's stain [55,56] James Homer Wright (1869 –1928) 	It is a histologic stain that facilitates the differentiation of blood cell types. It is classically a mixture of eosin (red) and methylene blue dyes. It is used primarily to stain peripheral blood smears and bone marrow aspirates which are examined under a light microscope. It is named for James Homer Wright, who devised the stain, a modification of the Romanowsky stain, in 1902. The May-Grünwald stain, which produces a more intense coloration, also takes a longer time to perform. James Homer Wright (1869 –1928), was an early and influential American pathologist.
Zinsser-Cole-Engman syndrome [57-62]	This is another name for dyskeratosis Congenita (DKC) It is a rare inherited bone marrow failure (BMF) syndrome characterized by mucocutaneous abnormalities and an increased predisposition to cancer. Named for Ferdinand Zinsser (1865-1952), who was a German dermatologist. Martin Feeney Engman, (1869- 1953), was an American dermatologist. Harold Newton Cole (1884-1968), was an American dermatologist.

Zinsser-Cole-Engman syndrome [57-62]	There are also also few eponyms related to this entity such as ;Hoyeraal-Hreidarsson syndrome (HHS) which is considered to be a severe form of DKC. Which have been described, for the first time, in two brothers by Hoyeraal et al. in 1970 and in one boy by Hreidarsson et al. in 1988. Another one is Revesz syndrome is a fatal disease which is characterized by retinopathy, aplastic anemia, nail dystrophy, and cerebellar hypoplasia .The syndrome is named after the author of the original case published in 1992.

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