ABSTRACTS

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HEMORRHAGIC DISEASES


Case report of purpura fulminans in a 9-year-old girl. The initial disease was a streptococcal angina. Anatomic findings and coagulation studies are discussed. Degenerative signs were observed above all in the lymph nodes, spleen and tonsils. Large histiocytic cells with signs of phagocytosis were detected in the spleen, lymph nodes and bone marrow. The plasma cells were increased in the spleen and in the lymph nodes. Hyalin thrombi were observed in the glomerular capillaries of the kidney. The bone marrow also exhibited megakaryocytic degeneration. There was a complete absence of fibrinogen, factor V, marked decrease of factor VIII (AHG) and pronounced thrombocytopenia.—P. d. N.


In a 22-month-old infant boy, signs of malignant osteopetrosis were associated with marked hemorrhagic diathesis. A complete absence of prothrombin, factor V, factor VII and fibrinogen was demonstrated in addition to a marked thrombocytopenia. The pathogenesis of such alterations is interpreted on the basis of lesions in the liver, the bone marrow and the mesenchymal tissue as a whole.—P. d. N.


The initials of abstracters who are not listed in the above masthead refer to those abstracters listed in the masthead of the December 1958 issue of Blood, p. 1206.
A 25-year-old male with classical osteogenesis imperfecta gave an additional history of repeated and severe epistaxes and easy bruising since childhood. Although platelets were normal in number, the bleeding time was persistently prolonged to over 20 minutes, and the Rumpel-Leede test was strongly positive. The patient presented impaired prothrombin consumption which was corrected by normal platelets, but not by normal plasma; and similarly, the patient’s platelets failed to improve prothrombin consumption in thrombocytopenic blood. In the thromboplastin generation test the patient’s platelets appeared to be inert. Other coagulation factors were normal. The suggestion is made that the platelet and bone defects might be causally related, although the patient’s sister had osteogenesis imperfecta without hemorrhagic manifestations or abnormal laboratory findings.—T. H. S.


A case of Moschowitz’s disease is reported as presenting the essential features of this condition: acute and febrile hemolytic anemia, thrombocytopenia, extreme severe status, thrombosis of multiple minor vessels due to fibrinoid necrosis of the arteriolar and capillary walls.

The case is remarkable on account of the subject’s age (10 months), the discretion of the purpura as compared with the thrombocytopenia, the significance of the hemolytic anemia with presence of an acid hemolysis and absence of other antibodies, the single renal location of the thromboses causing death by renal insufficiency and lastly the absence of neurologic signs and any other visceral sign.—J. D.


Previous reports of a sex-linked recessive disease manifest as infantile eczema with thrombocytopenic purpura are dealt with in detail by the authors who report five additional patients.

The disease had its onset in the first few months of life with infantile eczema and chronic draining otitis media. Bleeding from the ears, gastrointestinal tract or oral cavity appeared early in the course of each patient. In some instances the thrombocytopenia was intermittent, but clearly defined and severe in each patient most of the time. Splenectomy produced only temporary improvement in the four patients in which it was carried out.—N. J. S.

Leukocytes


This truly outstanding contribution deals with the authors’ observations and experiments involving a 15-year-old female who has suffered from cyclic neutropenia since early infancy. She was observed for 11 years at the University of Minnesota Hospital and studied intensively in that institution for a period of 1 year. It is not possible here even to list the large number of elaborate and thought-provoking experiments which were pursued in an attempt to define the etiology and immunologic consequences of this disease. It is concluded that cyclic neutropenia appears to be a periodic failure of neutrophilic maturation.

No factor injurious or destructive to neutrophils could be found in the patients plasma. Specific studies of gonadal, pituitary and adrenal function revealed no evidence of a
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hormonal factor responsible for the disturbance. The most extensive studies of neutrophil function in the patient are reported. The world literature on this rare disease is reviewed and the results of therapy with splenectomy, adrenal steroids, staphylococcal vaccine, and injection of normal leukocytes are reported. This study and report will stand as the classic discourse on this fascinating disease for years to come.—N. J. S.

ROLE OF SPLEEN IN MAINTENANCE OF CIRCULATING LEUCOCYTES. PART IV. EFFECT OF SPLENECTOMY ON MYELOID ELEMENTS OF BONE MARROW IN ALBINO RATS. S. Kumar, V. S. Mangalik and N. N. Sen. From the Division of Hematology, Department of Pathology and Bacteriology, Lucknow University, Lucknow, India. Indian J. M. Res. 45:485–492, 1957.

Splenectomy in albino rats was followed by hyperplasia of erythroid, myeloid and megakaryocytic elements in the bone marrow. There was no change in M:E ratio. Maturation curve was normal for the granular, but delayed for the erythrocytic series—J. B. C.


Acetylated lipopolysaccharide No. 1083 II, isolated from E. coli, has, as previously shown, produced marked granulocytosis in normal subjects following i.v. injection of 20 gamma dissolved in 0.2 ml. of water. 150 patients suffering from various diseases, including idiopathic pancytopenia, myelosclerosis, bone marrow metastases, cirrhosis of the liver, with and without splenomegaly, chronic lymphocytic and myelocytic and acute leukemia, lympho-granulomatosis, and various acute infectious diseases, were tested with the lipopolysaccharide. The bone marrow response in these cases, manifested by a significant rise of the granulocytes in the peripheral blood, proved to be a valuable means in the differential diagnosis of true bone marrow damage and “dys-regulative leukopenia.” It is hoped that future work will confirm the preliminary findings, and will further help to elucidate the problem of splenogenic bone marrow suppression.—M. H. H.


Are the cytochemical changes which take place in macrophages and giant cells in human inflammatory lesions the same as those produced in tissue culture of human blood cells? Experiments designed to test these questions are reported in the present article. Cells from both sources show an increase in acid phosphatase, lipid, polysaccharide and ribose nucleic acid. These cytochemical reactions indicate that the transformation of monocytes of the blood during inflammation is not a degenerative process.—O. P. J.


There is some understanding of virus localization and the means of release of virus from cells, but visual study of the virus during the initial stages of cell infection has not been possible so far. Three strains of Newcastle virus were studied in these sections of normal chicken macrophages grown in roller-tube cultures. The virus, which was previously placed on the surface of the cells, was found in the ingested fluid within the cell. Destruction of the entire cell substance was apparent at 5 hours. No specific lesions were found, nor was the process of virus multiplication elucidated.—O. P. J.
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By investigating the insulin inhibition in vitro, the authors have demonstrated a decreasing activity from the leukocytes, to the erythrocytes, the plasma and the platelets, both in normal and in diabetic subjects. In cases of insulin resistance, the plasma inhibitory activity is greater than in other blood constituents.—P. d. N.


The many advantages presented by the ascitic forms of transmissible animal tumors as a source of cellular material have led to their increasing use, in recent years, in various fields of investigation. The ascites form of mouse Sarcoma 37 produces basophilic tumor cells and many acidophilic peritoneal cells. The fine structure of the leukocytes examined was similar in all respects to the descriptions of such cells. However, it was found that the characteristic granules of eosinophil granulocytes are bounded by two fine limiting membranes about 7 μ apart and that the cell membrane of such cells appears to be composed of more than one layer. The two fine limiting membranes around the granules of eosinophil granulocytes are of much smaller dimensions and are much closer together than the limiting membranes of mitochondria. They thus do not suggest, morphologically, that these two types of organelles are related as has been claimed. The significance of the membranes is not known.—O. P. J.


In 3 cases a diagnosis of glandular fever was made, but the Paul-Bunnell test was found to be negative and serologic tests for toxoplasmosis were positive. The features were:

Case 1. Male, aged 16: sore throat, cough, headache, generalized pains, fever and petechial rash. White cells 6,200; monocytes 28%.

Case 2. Female, aged 31: pregnant; sore throat, blotchy rash, vomiting, aching; fever. White count 7900; monocytes 10%.

Case 3. Female, aged 36: enlarged glands and spleen; felt well and had normal blood findings.—R. H. G.

LYMPHOCYTES


For many years hematologists have believed that reticulum cells could perform only one of their functions at a time. In other words, phagocytic cells could not differentiate toward hematopoietic cells. The interesting tissue culture experiments reported in this brief note now cast some doubt upon this basic concept. Trowell believes it is possible for reticulum cells not only to re-utilize lymphocytic degradation products, but also to differentiate along lymphocytic lines. He has interpreted this to mean that there is a self-regulating "feed back" mechanism governing the total lymphopoiesis of the body. It would be interesting to study the reconstruction of nuclear pattern of these tissue culture cells in trypsin-treated smears stained with May-Grunwald-Giemsa according to the technic of Berman.—O. P. J.