ABSTRACTS

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ERYTHROCYTES AND HEMOGLOBIN


Many factors seem to play a part in the production of anemia after burns. Two of these, heat and anhydremia, have been investigated in healthy adult male rabbits. Acute anhydremia was produced by the subcutaneous injection of 60 per cent glucose. Some anhydremic animals were subsequently bled to study the bone marrow response. Blood was subjected to mechanical trauma and heated to determine its susceptibility to alteration. Some animals received blood which had been withdrawn, heated and then reintroduced into the body. The results show that anhydremia produces a temporary rise in hemoglobin, erythrocytes and the volume of packed cells. Hemorrhage does not interfere with the response of anhydremic animals. Hemolysis is not increased following mechanical trauma. Heated blood returned to the animals caused a fall in the hemoglobin and erythrocyte values, as well as changes in shape and increased fragmentation. Acute anhydremia does not produce corpuscular changes but heat and the duration of exposure to heat do.—O.P.J.

HEMOGLOBIN IRON AS A STIMULUS FOR THE PRODUCTION OF FERRITIN BY THE KIDNEY. J. K. HAMPTON, Jr. and H. S. MAYERSON. From the Department of Physiology, Tulane University School of Medicine, New Orleans, La. Am. J. Physiol. 156: 1-8, 1950.

This investigation was designed to study the role of ferritin in the handling of iron by renal tissue, especially the iron of hemoglobin. Ferritin was isolated from the kidney (and the spleen) of the mouse and of the rabbit after intraperitoneal injections of hemoglobin solutions. The amount of ferritin formed was proportional to the amount of hemoglobin solution injected. (Crystallizable amounts of ferritin were also located in the bone marrow of rabbits after intraperitoneal injections of hemoglobin solutions.) The authors 'suggest' that when large amounts of hemoglobin iron is available, iron is present in the kidneys in the form of ferritin. At first the iron is split from the ferritin, removed from the kidney, and deposited in other organs. When the preferred organs of storage become saturated, removal of iron from the kidneys is slowed. When the capacity of the ferritin iron-handling mechanism of the kidney is exceeded, some ferric hydroxide will likely form at the intracellular pH. This is suggested as a possible explanation of the tubular necrosis observed in renal damage from severe hemoglobinuria.—R.C.C.

COMPARISON OF PERIPHERAL BLOOD WITH HEART BLOOD IN GUINEA PIGS. P. G. ROYES, H. B. LATIMER, M. MADISON, M. MAFFET, and P. WILKINSON. From the Department of Anatomy, University of Kansas School of Medicine, Lawrence, Kan. Science 111: 337, 1950.

Blood was removed from the ear of normal guinea pigs without anesthesia. After Nembutal anesthesia, blood was also removed from the heart. Fifty-six guinea pigs were used in the experiment. Peripheral blood exhibited an average total erythrocyte count of 6.3 million cells per cu. mm. and an average total white cell count of 7.5 thousand cells per cu. mm. Heart blood exhibited 5.5 million
cells for the average erythrocyte count and 5.8 thousand cells for the average white cell count. The authors conclude that peripheral blood shows more red and more white cells than does heart blood.

—R.C.C.


The data in this paper indicate that hemin synthesis can be carried out by rabbit spleen homogenates. These homogenates utilize the methylene carbon atom of glycine as a precursor. The authors state that experiments are now in progress to estimate the biologic significance of these findings.— R.C.C.

Physiopathology and Course of Polycythemia Vera as Related to Therapy. W. Dameshek. From the Ziskind Research Laboratories (Hematology Section), J. H. Pratt and New England Centre Hospitals, and Department of Medicine, Tufts College Medical School, Boston, Mass. J.A.M.A. 142: 790-797, 1950.

The author reviews the theories of pathogenesis of polycythemia vera, and discusses in some detail both the course of the disease and the approaches to therapy. He presents the thesis that the true nature of the disorder is unknown, and that, although it resembles in many ways a neoplastic proliferation, it is perhaps better considered a relatively benign hyperplastic process, not truly neoplastic, and not malignant. Therapy, therefore, is aimed at a patient who is "relatively normal," rather than one who has a malignant proliferative process.

The author’s preferred mode of therapy in most cases is repeated phlebotomies, as indicated by symptoms and by blood values, until normal blood values are reached. The use of an iron-poor diet thereafter, combined with the repetition of phlebotomy as necessary, allows remissions lasting from months to many years. The author prefers to reserve radioactive phosphorus for stubborn and selected cases (notably those with very high platelet counts), lest irradiation result in potential undesirable effects, especially acute leukemia.

The emphasis is on polycythemia vera as a panmyelopathy—not merely as an increase in red cells alone—and on the relatively benign course of this disease. The discussion following this article includes several contrary points of view, in which the relative advantages and dangers of radioactive phosphorus are dealt with.—S.E.


A case is reported of a 17 year old Negro pregnant female who died rather suddenly before delivery of an approximately full term baby. Pertinent pathologic findings were profound sickling of the red cells, cerebral vascular occlusions with focal hemorrhages, other embolic and thrombotic phenomena, and an atrophic fibrotic spleen.

Twenty-three other reported cases of pregnancy associated with sickle cell anemia are mentioned and the clinical and pathologic findings in 5 of these are summarized briefly.

It is generally supposed that pregnancy, when it does occur in cases of sickle cell anemia, exerts its unfavorable effect by aggravating the anemic state which in turn predisposes to anoxemia and an exaggeration of the sickling process. It is the author’s contention that sudden death in sickle cell anemia can often, as in this reported case, be attributed largely to the cerebral vascular lesions secondary to the sickling of the red cells.—H.W.B.


Exposure of normal subjects to a low oxygen gas producing arterial saturation of 70 to 80 per cent
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led to a mean drop in cosinophils of 40 per cent. It was felt this reflected an increase of adrenal cortical activity following anoxemia.—W.N.V.


In studying the glycogen metabolism of leukocytes of the horse, dog and man, it was found that glycogen breakdown in white blood cells follows a pattern similar to that in liver and muscle. It is not the result of diastatic activity but rather of the presence of a phosphorylase. With increasing concentration of phosphate ions in the incubation mixture, the rate of glycogen degradation increases. Adenosine-5-phosphoric acid constitutes an essential activator of the phosphorylase in the reaction between glycogen and phosphate ions in white blood cells.—W.N.V.


After it was observed that globule leukocytes were found in the transitional epithelium of the urinary system of albino rats infested with the nematode, Trichosomoides crassicauda, a painstaking and exhaustive series of studies was undertaken to determine their nature. The article contains a table of 80 differential characteristics of "Schollenleukocyten" of the urinary tract and of certain possibly related cells. There is no doubt about the connective tissue origin of globule cells, but their relationship to plasma cells with acidophilic inclusions and Russell body cells remains uncertain.—O.P.J.


A case report is presented of a 7 month old girl with multiple skeletal lesions which on biopsy showed histiocytic overgrowth with eosinophil reaction. The peripheral blood showed granulocytosis, lymphocytosis, and eosinophilia. A bone marrow puncture gave normal results. There was rapid progression of the disease, and death followed within two months. Autopsy revealed splenomegaly and multiple bone tumors, with reticulum cell proliferations in various organs; a diagnosis of reticulo-endothelial granulomatosis or Letterer-Siwe disease was made. The authors discuss the relationship of this diagnosis to other similar histologic and clinical disorders, notably Hand-Schüller-Christian's disease and eosinophilic granuloma.—S.E.

HEMOLYTIC DISEASE


The first section of this comprehensive review of erythroblastosis fetalis concerns data on 157 children with hemolytic disease of the newborn, born to 149 mothers in the years 1937 to 1947 ("the pre-Rh era") in Edinburgh. In 118 of these mothers in whom complete histories of previous pregnancies could be obtained, 60 of 178 pregnancies resulted in stillbirths or abortions, i.e., 11 per cent, higher than that seen in unselected pregnancies. The usual Rh pattern was present in those patients in whom immunologic studies could be done (i.e., mother Rh negative, child and father Rh positive). It is of especial interest that 10 of the 149 women were pregnant for the first time, yet no history of previous transfusion or injection of blood could be obtained in them.

Of the 157 infants studied, 31 were stillborn (20 per cent), and an additional 85 died soon after birth (54 per cent of total). Details of hematology, pathology, and postmortem findings are given. In the entire series, active treatment consisted only of blood transfusions.

The second section of this report discusses kernicterus, which was found in one-third of the infants with icterus gravis (i.e., 37 of 110 such infants). Of these 37 infants, 33 died within two weeks after birth, largely within the first seven days. In those who survived, the usual evidences of involvement of the pyramidal and extrapyramidal structures were present (choreoathetosis, incoordination, spasticity).

The pathology and pathogenesis of kernicterus are discussed in detail.—S.E.
BOOK REVIEW


The author isolated a hemolytic substance of high activity from plasma and other organs of horses. The substance was found to be a mono-unsaturated, mono-carboxylic fatty acid with the chain length C8. The acid has been identified as cis-11-12-octadecenoic acid and this has been confirmed by synthesis. It is found in various parts of the body and in various concentrations in parts of the same system, i.e., nervous system. In erythrocytes, the substance is bound to and inactivated by the stromata. Optimal activity of the substance in vitro is dependent on the presence of phosphate. The body contains many substances which can inhibit the hemolytic activity of this substance, such as proteins, cholesterol, lecithin, and calcium. The presence of the hemolytic substance in the erythrocytes themselves is assumed to be related to their life span.—R.C.C.

BOOK REVIEW


A concise and able presentation of the essential features of arteriosclerosis in the lower extremities with the development of thrombosis and its serious sequelae has been made available in this monograph. The first portion of the book deals with a discussion of the etiology and pathology of thrombosis in arteriosclerosis with numerous illustrations of the induction of thrombosis, the later changes in the vessel and the effects of ischemia. Symptoms and signs, clinical course, complications and prognosis and diagnosis are briefly discussed. The author endorses the proposition that excessive cholesterol is causative in the disease and believes that prophylaxis may be encouraged by limitation of animal fats.

Because increase of the circulation has proved clinically to give considerable protection against thrombosis, lumbar sympathectomy is favored although criteria for the choice of the operation for arteriosclerosis in general are not discussed. The author believes that immediate lumbar sympathectomy in the treatment of established thrombosis is the most significant single item available today. Anticoagulants and Buerger's drugs have been disappointing.

The second half of the book is devoted to case reports illustrating thrombosis due to various causes in many of the major arteries of the lower extremity. These are well chosen examples, demonstrating as they do the complications of the disorder, the difficulties of treatment and the correlation of the clinical picture with the pathology.

The author makes the point that no special instruments or laboratory procedures are required to make the diagnosis, that history and physical examination alone are sufficient, and expresses the hope that a greater awareness of the frequency and seriousness of thrombosis will bear fruit in the prevention of gangrene and of seriously impaired function.—Allan D. Callow.

ERRATA: Frommeyer, Epstein and Taylor: Refractoriness in hemophilia to coagulation-promoting agents. Whole blood and plasma derivatives. Blood 5: 401-420 (May), 1950. The authors wish to note that the following changes should have appeared in the article:

1. Page 402, first line: "Lawrence and Johnson, 1941, " reported" (instead of "Lawrence and Johnson, in 1946, reported".)
2. Page 402, second sentence of same paragraph: "In this instance the patient had received whole blood and plasma in therapeutic management prior to the appearance of resistance to therapy." (instead of " . . . had received chemically prepared plasma fractions in the form of Fraction I of Cohn in addition to whole blood and plasma in therapeutic management prior to . . . ").
3. Page 410, reference 8. To this reference should be added: "(Presented before the American Clinical and Climatological Society, October 1941.)"
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