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HEMOGLOBIN

ABNORMAL HEMOGLOBIN IN ANEMIAS. *A. Gajdos.* From the Hôtel Dieu, Paris, France. Sang *24:* 185–201, 1953.

It is a very careful review of the literature dealing with abnormal hemoglobins in different types of anemia.—*J.P.S.*

FILTER PAPER ELECTROPHORESIS OF HUMAN HEMOGLOBINS WITH SPECIAL REFERENCE TO THE INCIDENCE AND CLINICAL SIGNIFICANCE OF HEMOGLOBIN C. *E. W. Smith and C. L. Conley.* From the Department of Medicine, The Johns Hopkins University and Hospital, Baltimore, Md. Bull. Johns Hopkins Hosp. *93:* 94–106, 1953.

A simple and inexpensive apparatus for the separation of various types of hemoglobin by paper electrophoresis is described. The apparatus is a modification of the glass plate method of Kunkel and Tiselius.

In a mass survey of five-hundred white persons, no instance of S, C, or D hemoglobin was discovered. A similar survey of five hundred Negroes revealed an incidence of 8.4 per cent hemoglobin S and 2 per cent hemoglobin C. Of those showing the C trait, one individual was S-C and nine were C-A. No instances of homozygous hemoglobin C (C-C) were found.

C trait when combined with normal hemoglobin appeared to be benign though some of the individuals had as many as 10 per cent target cells in the peripheral smear.

In addition to the one person found on the survey to have the C-S hemoglobin combination, six others with this combination were studied. In general, those with C-S hemoglobin had a disorder much milder than sickle cell anemia. However two such patients had hemolytic episodes with jaundice and profound anemia occurring during pregnancy. Episodes of gross unilateral hematuria were noted in two patients with this disorder. Six of the seven C-S patients had palpable spleens.—*C.E.R.*

IRON METABOLISM

HISTOLOGICAL APPEARANCES FOLLOWING INTRAVENOUS SACCHARATED IRON OXIDE, AND THE FACTORS GOVERNING PRECIPITATION OF THE COMPOUND IN VIVO. *J. A. Nissim.* From the Department of Pharmacology, Guy's Hospital Medical School, London, England, Guy's Hosp. Rep *102:* 164–179, 1953.

In experiments with mice, rabbits, rats, and guinea pigs it is shown that intravenously injected saccharated iron oxide in doses up to about 45 mg. Fe/Kg. remains in solution in the blood and is rapidly taken up and stored by the cells of the reticuloendothelial system lining the vascular channels. The liver is the chief organ concerned in removing the iron from the blood.

When given in large doses, intravascular precipitation occurs and this is proportional to the size of the dose. Embolic phenomena result. The precipitation is partly gradual and partly immediate.

Colloidal ferric hydroxide in aqueous solution (pH about 7.2) precipitates in vitro at pH 2.0, but when mixed with plasma it remains in solution only if the pH exceeds 8.0.

All samples of saccharated iron oxide, irrespective of their aqueous precipitation points, began to precipitate, when mixed with an equal volume of plasma, at gout pH 7.2, and reached their maximum at about pH 4.5. After that the precipitate gradually redissolves.—*R.H.G.*

THE RELATIONSHIP BETWEEN FERRITIN AND HEMOSIDERIN IN RABBITS AND MAN. *A. Shoden, B. W. Gabrio, and C. A. Finch.* From the Department of Medicine, University of Washington School of Medicine, Seattle, Wash. J. Biol. Chem. *204:* 823–830, 1953.

By employing methods previously described (J. Biol. Chem. *204:* 815, 1953) human and rabbit tissue iron content was studied. Radioiron was demonstrated as stored in both ferritin and hemosiderin. The results indicate that these two types of storage iron are functionally indistinguishable and, as has been considered previously, the compounds differ only in physical form.—*P.F.W.*

IMMUNOHEMATOLOGY

HAEMOLYTIC DISEASE OF NEW-BORN PIGS CAUSED BY ISO-IMMUNIZATION OF PREGNANCY. *J. C. Buxton and N. H. Brooksbank.* From the Veterinary Investigation Laboratory, University of Nottingham, Loughborough, England. Nature, London *172:* 355, 1953.

Serious loss of life from hemolytic disease occurred amongst baby pigs on a farm in England. Isoimmunization was shown by applying the direct antiglobulin sensitization test to the piglets' blood, using anti-pig globulin serum prepared in rabbits. There was a well-marked reaction between serum from affected sows and the red cells of the boar.—*R.H.G.*

SOME INTERRELATIONSHIPS OF THE ERYTHROCYTES OF VARIOUS SPECIES WITH PLANT AGGLUTININS. *G. W. G. Bird.* From the Blood Transfusion Department, Armed Forces Medical College, Poona, India. Nature, London *172:* 401–402, 1953.

Extracts of *Ricinis communis, Dolichos lablab, Vicia faba,* and *Phaseolus lunatus* were each put up against cell suspensions obtained from the blood of the following species: sheep, goat, water buffalo, cow, horse, dog, chicken, pigeon, rabbit, guinea pig, and man (Groups A, B, and O). Agglutination was then looked for after ten minutes. There was evidence of differences in the erythrocyte antigen pattern of various species.

The pattern of agglutination was the same in each member of the following four groups: (1) Sheep-goat-buffalo-cow-horse-dog; (2) Chicken-pigeon; (3) Rabbit-guinea-pig-man (Groups B and O); (4) Man (Group A).—*R.H.G.*

A NEW RARE HUMAN BLOOD GROUP ANTIGEN (Wr^a). *C. A. Holman.* From the Group Laboratory, Lewisham Hospital, London, England. Lancet *2:* 119–120, 1953.

A new antigen responsible for hemolytic disease of the newborn. The name of the family was Wright, and the gene and the antigen have been named Wr^a. In the family of Mr. Wright the antigen was found in five members of the three generations examined.—*R.H.G.*

SEROLOGICAL FINDINGS IN A CASE OF HAEMOLYTIC ANAEMIA. *W. Weiner, D. A. Battey, T. E. Cleghorn, F. G. W. Marson, and M. J. Meynell.* From the Blood Transfusion Service, Birmingham, England. Brit. M. J. *2:* 125–128, 1953.

A man, aged 32, with features of idiopathic acquired hemolytic anemia did not respond to splenectomy but showed temporary improvement with cortisone therapy. At all times the direct Coombs test was positive. The peculiar feature of the case was that an antibody present in the serum and in an eluate from the patient's cells was specific against all cells containing the anti-e antigen, but nevertheless the genotype of the patient was CDe/CDe. It appeared that an anti-e had developed in the presence of the homologous red cell antigen without stimulation by transfusion or blood injection.—*R.H.G.*

HEMOLYTIC TRANSFUSION REACTION CAUSED BY ANTI-c ANTIBODY. *P. G. Frick.* From the Department of Medicine, University of Minnesota Medical School, Minneapolis, Minn. Am. J. M. Sc. *225:* 630–635, 1953.

A case report is presented of a 43 year old woman who was genotype A, B CDe/CDe MN; and whose husband was A_2O CDe/cde MN. She had had thirteen pregnancies of which the third, fourth, and fifth resulted in spontaneous abortion and the twelfth in a stillbirth. She was admitted for thyroidectomy during which she received two units of citrated blood, one of which was group A_2B type cde/cde and the other A_2B CDe/cde. Twenty years previously she received one unit of blood which was found to be cde/cde. There was a hemolytic reaction to the second transfusion and possibly to the one twenty years before.

The patient's serum did not contain any albumin active antibodies before the transfusion, nor did the "serum method" of cross match reveal incompatibility, but the anti-c was detectable by the indirect Coombs test. Albumin active antibody appeared on the fifth day after transfusion and reached a maximum on the ninth day.

In spite of normal urinary volume, the blood urea nitrogen rose to 96 mg. per 100 cc. on the sixth day and gradually returned to normal by the forty-fourth day.

It is pointed out that the antigens Kell, c, and E are the most frequent cause of sensitization of Rh-positive mothers. Since none of the patient's children had evidence of erythroblastosis, the suggestion is made that the anti-c did not pass the placenta.

The author concludes that all patients who have a suspicious history should receive the benefits of an indirect Coombs test. It is emphasized that anti-Kell, anti-Kidd, and anti-Duffy cannot be detected by any other technic.

The reviewer would like to add, however, that other blood banks have found the trypsinized red blood cell test an extremely useful routine method to use on all recipients, utilizing the Coombs test in addition where indicated.—*T.R.T.*

A STUDY OF TWO UNUSUAL BLOOD-GROUP ANTIGENS IN WEST AFRICANS. *J. N. M. Chalmers, E. W. Ikin, and A. E. Mourant.* From St. George's Hospital Medical School, and the M.R.C. Blood Group Reference Laboratory, London, England. Brit. M. J. *2:* 175–177, 1953.

A detailed account of the distribution in West Africa and the genetics of the Hunter antigen and of a newly discovered antigen to which the name Henshaw has been given. The genes for these antigens appear to be closely linked to those of the MNS_s blood group system.—*R.H.G.*

HIGH R^z FREQUENCY IN THE BLOOD OF AUSTRALIAN ABORIGINES. *R. T. Simmons, J. J. Graydon, and J. B. Birdsell.* From the Commonwealth Serum Laboratories, Melbourne, Australia and the University of California, Los Angeles, Calif. Nature, London *172:* 500, 1953.

Three examples of the genotype R^zR^z were found in three hundred and ninety-five unmixed aborigines in Western Australia. There was a high R^z frequency averaging 6 per cent but approaching 20 per cent in certain tribes.—*R.H.G.*

A RABBIT SERUM CONTAINING A SPECIFIC AGGLUTININ FOR THE RED CELLS OF THE NEWBORN. *E. W. Ikin, H. Lehmann, and A. E. Mourant.* From the Blood Group Reference Laboratory and St. Bartholomew's Hospital, London, England. Brit. M. J. *2:* 602–604, 1953.

Human cord red cells were injected into rabbits and an antiserum was produced which agglutinated specifically the red cells of cord blood and of newborn infants.—*R.H.G.*

THE SYNDROME OF HIGH-TITRE COLD HAEMAGGLUTINATION. *M. G. Nelson and R. J. Marshall.* From the Royal Victoria Hospital, Belfast, North Ireland. Brit. M. J. *2:* 314–317, 1953.

A man, aged 47, developed attacks of Raynaud's phenomenon with associated hemoglobinuria and hemoglobinemia. The serum contained a cold agglutinin active against the

patient's own and homologous erythrocytes to a very high titer. The cold agglutinin was absorbed on to the surface of erythrocytes from the patient's blood which was allowed to clot at room or refrigerator temperature. It could be eluted from these cells by washing with warm saline, and a Coombs' test on the washed cells gave a positive result, indicating the presence of an adsorbed cold immune body.

A 40 year old man developed Raynaud's phenomenon and dry gangrene. There was anemia and hemoglobinemia.

As in the other case a cold agglutinin and incomplete cold antibody could be demonstrated.—*R.H.G.*

HAEMOLYTIC ANAEMIA IN TYPHOID FEVER. *A. J. S. McFadzean and G. H. Choa.* From the Department of Medicine, University of Hong Kong, China. Brit. M. J. *2:* 360–366, 1953.

In one hundred and twenty-nine consecutive cases of *Salm. typhi* infection, hemolytic anemia occurred in six. No cases were seen amongst forty-seven patients infected with *Salm. paratyphi* A, B, or C. Excess of urobilinogen was present in the urine of five patients and the sixth had hemoglobinuria and methemoglobinuria. The mean corpuscular volume was raised in all six and the direct Coombs' test was positive in the four patients examined.

Two patients were treated with ACTH by slow intravenous drip in a dosage of 20 mg. in twenty-four hours. There was rapid improvement, not only in the hematologic findings, but also in the typhoid state.

The anemia is considered to be a symptomatic acquired hemolytic anemia related to the typhoid infection.—*R.H.G.*

EFFECT OF SOME SERUMS ON TITER OF Rh ANTIBODIES. A METHOD FOR DETECTION OF Rh SENSITIZATION. *E. P. Le Roy and W. Spurrier.* From the Department of Pathology, Mount Sinai Hospital, Chicago, Ill. J. Lab. & Clin. Med. *42:* 85–91, 1953.

The technics for detection of isoimmunization caused by Rh or other blood group agglutinogens may be listed according to increasing sensitivity, as follows: (1) blocking test of Wiener; (2) use of protein (or other macromolecular substances) as a diluent in place of saline; (3) the antiglobulin test; (4) enzyme-treated test cells. Although it is rare for these tests to miss a case of Rh sensitization, occasional cases occur in which mild symptoms of erythroblastosis occur in the absence of positive tests. Eight such cases are the subject of this report.

"Serums from eight Rh-negative women in whom sensitization to Rh agglutinogen by pregnancy was suspected were tested for an enhancing effect on the titer of Rh antibodies. Although they contained no Rh antibodies detectable by the usual techniques, they caused an increase in titer when they were used as diluent in the titration of known anti-Rh serums. This effect was abolished by absorption with O Rh-positive cells, but not with O Rh-negative cells. Thus it appears to be caused by the presence of Rh antibodies in the serums. Evidence is presented suggesting that this specific enhancement of Rh antibodies is a more sensitive test for Rh sensitization than the techniques available to date."—*T.R.T.*

STUDIES ON THE NORMAL SERUM PANAGGLUTININ ACTIVE AGAINST TRYPSINATED HUMAN ERYTHROCYTES II. RELATIONSHIP TO COLD AGGLUTINATION. *T. H. Spaet and B. G. Kinsell.* From the Department of Medicine, Stanford University School of Medicine, San Francisco, Calif. J. Lab. & Clin. Med. *42:* 205–211, 1953.

Normal human serum contains an autoagglutinin active against trypsinated erythrocytes, differing from abnormal agglutinins in that incubation of the test cells with serum at room temperature or at 37 C. causes reversal of the agglutination, usually within one hour. The agglutinin can be absorbed out of serum by repeated treatment with trypsinated cells and can then be eluted from the cells in pure form. The present report concerns the study of the relationship between this normal autoagglutinin (NAA) active only against trypsinated erythrocytes and normal cold agglutinins of the complete and incomplete varieties.

The results of the author's studies indicate a clear distinction between the NAA and extended thermal activity of cold agglutinin. This is shown by the following observations: a) a lack of correlation exists between NAA and cold agglutinin activity, whether the latter be tested against untreated cells, trypsinated cells, or by its ability to render cells Coombs' test positive; (b) the activity of eluded NAA is not affected by cold; (c) absorption of serum with trypsinated cells at 20 C. removes NAA activity but does not significantly affect cold agglutinin; (d) cold agglutinin is found in the gamma globulin fraction of the serum proteins exclusively whereas the panagglutinin appears in fractions I nad III which represent mainly alpha and beta globulins.

The authors speculate concerning the possibility that NAA is not an antibody, but that the agglutination associated with it may be due to a nonspecific protein reaction.

It is also of interest that Dacie's report stating that about one fourth of all normal sera, when incubated at refrigerator temperature with cells from the same donor, are capable of producing a positive Coombs' test. Furthermore, the cells did not become Coombs' negative even after warming to 37 C.—*T.R.T.*

LEUKOCYTES

THE RELATIONSHIP OF THE TISSE MAST CELLS TO THE BLOOD VESSELS IN THE RAT. *J. F. Riley.* From the Radiotherapy Department, Royal Infirmary, Dundee, Scotland. J. Path. & Bact. *65:* 461–469, 1953.

The primary purpose of this paper was to determine the origin and distribution of the mast cell in the rat. The author neglected an excellent review of the mast cell problem by Michels (Downey's Handbook of Hematology, Vol. 1, p. 235, 1938). Two types of mast cell have been described. Type I in which the cells stain uniformly in a blue, orthochromatic tint and so densely as to obscure the nucleus. In type II the homogeneous staining is gradually resolved into orthochromatic granules set in a clear cytoplasm and, as the granules become further dispersed, they stain purple to red (metachromasia). It should be pointed out that Wislocki and Fawcett (J. Nat. Cancer Inst. *12:* 258, 1951) also described two types of mast cells, but on the basis of granule size and staining with periodic acid-Schiff reagents. —*O.P.J.*

THE EFFECTS OF HISTAMINE-LIBERATORS ON THE MAST CELLS OF THE RAT. *J. F. Riley.* From the Radiotherapy Department, Royal Infirmary, Dundee, Scotland. J. Path. & Bact. *65:* 471–479, 1953.

In shock states in the dog, histamine and heparin are released simultaneously from the liver, the heparin being thought to come from tissue mast cells. The present paper deals with an investigation of the relation of mast cells to histamine in the rat where mast cells can be readily observed in tissue spreads. Intravenous injection of chemical histamine-liberators or an anaphylatoxin produces not only edema but also a disruption of the tissue mast cells. Nonspecific edema, including the edema produced by the injection of histamine, also leads to mast-cell breakdown, but this is not believed to be the whole explanation for the changes in mast cells which follow the injection of a histamine-liberator. Apparently there is a direct effect by the histamine liberators on mast cells and these effects are prevented by premedication with an antihistamine drug. In addition to supplying heparin, the mast cells have a role in the production of histamine.—*O.P.J.*

MAST CELLS AND SUSCEPTIBILITY TO EXPERIMENTAL ATHEROSCLEROSIS. *P. Constantinides.* From the Anatomy Department, University of British Columbia, Vancouver, Canada. Science *117:* 505–506, 1953.

It has been known for many years that lipemia and atherosclerosis can be produced easily in the rabbit by cholesterol feeding, whereas the rat is refractory to cholesterol feeding. In recent years it has been found that heparin will prevent the development of atherosclerosis by cholesterol feeding. In addition, it has been shown that mast cells in all probability are the cells which produce heparin. Because of the above findings the present

author studied the mast cell content of rabbits and rats in an attempt to determine whether the presence of mast cells in the rat could account for the refractoriness of this animal to cholesterol feeding. Several methods of staining were utilized and all methods produced identical results. With the exception of the brain and spleen, which contained no mast cells, the connective tissue in all other organs of the rat was constantly supplied with mast cells. These cells were extremely abundant in the thymus and integument, numerous in the respiratory, circulatory, and urinary systems, and scarce in the digestive system. In contrast, with the exception of the skin, the skeletal muscle, the heart, and the intestine where a few cells were found, the author could find no mast cells in other organs of the rabbit. The author feels that the presence of numerous mast cells in the rat may account for the lack of susceptibility to the development of lipemia and atherosclerosis. Conversely, the author also feels that the lack of mast cells in the rabbit accounts for the susceptibility of this species. The author is now investigating the possibility that atherosclerotic or senile human subjects may present a mast cell deficiency similar to that of atherosclerosis susceptible rabbits.—*R.C.*

THE HEPARIN CONTENT OF MAST-CELLS. *H. Martin and L. Roka.* From the Second Medical Clinic, University of Frankfurt, Frankfurt, Germany. Acta haemat. *10:* 26–31, 1953.

The leukocytes from a patient with mast-cell leukemia (53 per cent mast cells of 284,000 leukocytes per cu. mm.) were isolated and dried in acetone. The substance obtained inhibited coagulation possibly by opposing thrombokinase. The biologic and physical properties were the same as those of heparin.—*C.M.*

MULTIPLE CARCINOMATA AND FOCAL MAST-CELL ACCUMULATIONS IN THE SKIN OF A FERRET (MUSTELA FURO L.), WITH A NOTE ON OTHER TUMOURS IN FERRETS. *W. St.C. Symmers and A. P. D. Thomson.* From the Department of Pathology, University of Birmingham, Birmingham, England. J. Path. & Bact. *65:* 481–493, 1953.

Multiple spontaneous basi-squamo-sebaceous tumors of the skin of a ferret are described. The most rapidly growing, least differentiated tumor was successfully transplanted by injecting tumor tissue into the subcutaneous tissue of the same animal. Attempts to transplant the tumor into other ferrets failed. Tumor-like collections of mast cells were found in the skin of the same animal. Histochemical investigations suggested that the mast cells contained hyaluronic acid and heparin monosulphate. Fibromatoid growths were present in both ovaries. The observation in a human patient of a basi-squamous-sebaceous tumor identical in structure with those in the ferret is reported.—*O.P.J.*

INVESTIGATION CONCERNING THE EXPERIMENTAL PRODUCTION OF L.E. CELLS. *H. Dietrich and F. Frühmann.* From the Evangelist Hospital for Medical Diseases, Vienna, Austria. Acta haemat. *10:* 239–246, 1953.

The author found that the artificially produced L.E. cells by means of liquid (Inderbitzin) are due to phagocytosis of precipitated profibrin. The same results were obtained by mixing bone marrow with blood of a different group adding Russel's viper snake venom. It is concluded that phagocytosis of homologous cells is influenced by changes in the surface of cells, and by an opsonic factor acting on blood. The production of L.E. cells is regarded as the result of an antigen-antibody reaction by autoantibodies.—*C.M.*

ELECTROPHORETIC STUDY OF THE PLASMAPROTEINS. *P. de Nicola.* From the Clinic of the University of Pavia, Pavia, Italy. Prensa med. argent., *40:* 371–374, 1953.

The authors reviews the development of knowledge of plasmaproteins by electrophoretic methods, particularly in relation to hematologic processes and tumor-like conditions, e.g., myeloma and plasmacytoma. Taking into consideration the globulin fraction predominant in the plasma, the myelomas can be divided into the following types: alpha, $beta_1$, $beta_2$, and gamma myelomas. There is an important correlation among these types of globulins and the clinical evolution of the disease, i.e., alpha myelomas are characterized

by acute evolution and bone marrow infiltration by plasmablasts, whereas gamma myelomas have chronic and slow evolution and the bone marrow is infiltrated by plasmacytes. Cases of high globulinemia are usually associated with hemorrhagic tendency, so there is a possibility that the alteration of the globulins in these cases may have something to do with the coagulation phenomena.—*C.F.M.*

BLOOD COAGULATION and HEMORRHAGIC DISEASE

DIFFERENTIATION OF HEMOPHILIA INTO TWO GROUPS. A STUDY OF THIRTY-THREE CASES. *J. P. Soulier and M. J. Larrieu.* From the Centre National de Transfusion Sanguine, Paris, France. New England J. Med. *249:* 547–553, 1953.

It has been known for several years that the plasma of some hemophiliacs would apparently correct the clotting defect of the plasma of other hemophiliacs. This difference in the plasma of patients diagnosed clinically as having "hemophilia" is the subject of this very interesting paper. These authors categorize their cases as hemophilia A, hemophilia B, and possibly a hemophilia AB or mixed type. Hemophilia A plasma behaves like normal plasma with that of hemophilia B and vice versa. Presumably normal plasma contains antihemophilic factors A and B. Antihemophilic factor A disappears in oxalated stored plasma whereas factor B persists. Barium sulfate adsorbs factor B but not factor A. Fresh serum is rich in factor B and devoid of factor A. Both factors are thermolabile. The relative frequency of these types of hemophilia is unknown. In the current short accumulated series about 14 per cent are type B. It remains to be established whether there is any clinical difference between these types. Therapeutically it is important to note that fibrinogen or plasma fraction I is effective in hemophilia A but ineffective in the treatment of hemophilia B.—*P.F.W.*

HEMOPHILIA B. TWO CASES OF HEREDITARY HEMOPHILIA DUE TO A DEFICIENCY OF A NEW CLOTTING FACTOR. (CHRISTMAS FACTOR). *R. Cramer, P. Flückiger, C. Gasser, F. Koller, A. Loeliger, and M. Matter.* From the Department of Medicine and Pediatrics, University of Zurich, Zurich, Switzerland. Acta haemat. *10:* 65–76, 1953.

The authors describe a coagulation defect, independent of antihemophilic globulin, found in two boy patients with a recessive sex-linked heredity, suffering from severe hemophilia. Both cases showed the presence of antihemophilic globulin in normal amount. Tests made with plasma plasma and plasma serum mixtures showed that in both cases, the condition is due to the lack or deficiency of a coagulation factor present in normal and in hemophilic plasma. The coagulation defect of the two patients can therefore be corrected by the addition of either normal or hemophilic plasma. The described coagulation factor is not consumed during the coagulation process. It retains its activity in the serum in the same way as factor VII. Consequently the above mentioned coagulation defect can also be corrected by the addition of normal and hemophilic serum.

Hemophilia, a disease with an accurately defined diathesis, may therefore arise from two different pathogenic conditions. The prevalent form of the disease is due to a deficient formation and supply of antihemophilic globulin. The probably rarer form described, is due to the deficiency of another coagulation factor, also necessary for the formation of thromboplastin. The authors suggest that the particular bleeding disease described, should be named hemophilia B and the more common form referred to as hemophilia A.—*C.M.*