ABSTRACTS

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ERYTHROCYTES

COMPARATIVE STUDIES OF THE STIPPLING OF ERYTHROCYTES WITH BRIGHT AND DARK FIELD MICROSCOPY. *K. Humperdink and E. Landolt.* From the Arbeitsmedizin. Institut Stuttgart, Germany. Ztschr. ärztliche Fortbildung *43:* 518–522, 1949.

The authors examined 55 patients exposed to lead, showing more or less marked anemia. The dark field method allows recognition of as much as one hundred per cent more stippled cells than the ordinary smear stained with Wright's method. Maximal values have been found in mild cases, whereas patients with severe intoxications have shown lower values. This is because of the aregenerative damage of the bone marrow in severe cases. The number of stippled cells does not have a direct relation to the reticulocytes, an observation made previously by Whitby and others.—*C.M.*

FAMILIAL SPHEROCYTOSIS AS A PROBLEM OF MEMBRANE STRUCTURE. *B. Lindemann.* From the X ray Institute Allg. Krankenhaus Hamburg, Germany. Arch. f. exper. Path. u. Pharmakol. *207:* 569–575, 1949.

Normal erythrocytes hemolyzing in hypotonic solution and those from patients with familial spherocytosis showed the same pattern in the electron-optic picture. These observations are considered to prove the existence of a plasma hemolysin in familial spherocytosis.—*C.M.*

RELATIONSHIP BETWEEN SERUM PROTEIN AND REGENERATION AND DESTRUCTION OF ERYTHROCYTES IN CHILDREN. *K. Betke and R. Thuran.* From the Pediatric Clinic, University of Erlangen, Germany. Monatschr. Kinderheilkunde *97:* 331–334, 1949.

In previous publications the authors have described a reduction of the serum globulin fraction in cases with marked blood regeneration. Observation on two more patients (one with malaria, one with familial spherocytosis) revealed that the cause of the phenomenon is not the elevated level of reticulocytes but blood destruction which is greater than regeneration.—*C.M.*

X-RAY HEMOLYSIS AS A PROBLEM OF MEMBRANE STRUCTURE. *B. Lindemann.* From the X Ray Institute Allg. Krankenhaus. Hamburg, Germany. Fortschritte auf dem Gebiet der Röntgenstrahlen vereinigt mit Röntgenpraxis *72:* 365–370, 1950.

Photographs of hemolyzing erythrocytes taken by the electron microscope showed exactely the same morphologic alterations (1) in hypotonic saline solution, (2) in 1 per cent saline after aging of the cells, (3) in 1 per cent saline after x-ray treatment and (4) in protein denaturating solutions. The author concludes that x-ray hemolysis therefore has to be regarded as a hemolysis of the colloid-osmotic type.—*C.M.*

DETERMINATION OF CIRCULATING RED CELL VOLUME BY RADIOACTIVE CHROMIUM. *S. J. Gray and K. Sterling.* From the Biophysical Laboratory and Department of Medicine, Harvard Medical School, and Medical Clinic, Peter Bent Brigham Hospital, Boston, Mass. Science *112:* 179–180, 1950.

The authors present a method of determining circulating red cell volume by means of tagging the patient's own cells with radioactive chromium. The authors point out that the determinations with radioactive chromium share with the radioactive phosphorus method the advantage that a small sample of the subject's own blood may be tagged rapidly in vitro and then reinjected. In contrast to the radioactive phosphorus method, however, the chromium tagged cells retained their activity without loss to the plasma for periods of one day or longer after injection. This fact should prove advantageous for clinical studies. After three days the red cell activity decreased by approximately 15 per cent. The authors point out that a similar fall occurs within three hours in the radioactive phosphorus method. A detailed account of this method utilizing radioactive chromium is presented.—*R.C.C.*

BLOOD FINDINGS IN MEN ON A DIET DEVOID OF MEAT AND LOW IN ANIMAL PROTEIN. *L. Mirone.* From the Nutrition Department, University of Georgia, Athens, Georgia. Science *111:* 673–674, 1950.

During a course of a nutritional survey, this author became interested in a community of men who did not consume meat and whose animal protein intake was low. Eleven men were selected for the study. These subjects were never allowed meat, poultry or fish. The daily animal protein ingested was the milk added to coffee. In addition, five months of the year, three times a week, extra animal protein was consumed in the form of fresh skim milk and American cheddar cheese. During the remaining seven months, the extra animal protein was consumed once a week for seventeen weeks. During the year, on twenty-eight occasions, the men were served two ounces of butter at breakfast and five ounces of plain cake, prepared with eggs and milk, at the noon meal. Hematocrit, hemoglobin, red cell count, white cell count, iron, sugar and non-protein nitrogen determinations were made on oxalated venous blood. Total protein, albumin and globulin determinations were made on the blood serum; differential counts were done on blood smears. Despite the fact that the subjects had been on a diet devoid of meat and low in animal protein for at least twelve years, and in one case for forty-seven years, the blood findings fell within the normal range.—*R.C.C.*

IRON METABOLISM

IRON INTAKE AND HAEMOCHROMATOSIS IN THE BANTU. *A. R. P. Walker and U. B. Arvidsson.* From the Nutrition Unit, Council for Scientific and Industrial Research, South African Institute for Medical Research, Johannesburg, South Africa. Nature, London *166:* 438–439, 1950.

Dietary analyses of food taken by the Bantus showed that a typical diet, cooked in the traditional manner in iron cooking pots, might contain as much as 100–150 mg. of iron per day. Confirmation of this high intake was found by stool analyses. It is suggested that high intake is an etiologic factor in the hemochromatosis which develops commonly in the Bantu.—*S.C.*

ANEMIA

ABSORPTION OF VITAMIN B_{12} IN PERNICIOUS ANAEMIA. I. Oral Administration without a Source of Intrinsic Factor. *C. C. Ungley.* From the Royal Victoria Infirmary, Newcastle-upon-Tyne, England. Brit. M. J. *2:* 905–908, 1950.

Vitamin B_{12} was given orally in varying dosage to patients with untreated pernicious anemia. In one case a daily dose of 5 μg. was ineffective. In another 80 μg. per day produced a response equivalent to that expected from 2.5 μg. given parenterally. The response in 5 patients given a single dose of 3,000 μg suggested an absorption of 80 to 160 μg. or more.

It is suggested that B_{12} in these patients must have been absorbed directly and cannot have combined first with intrinsic factor.

ABSORPTION OF VITAMIN B_{12} IN PERNICIOUS ANAEMIA. II. Oral Administration with Normal Gastric Juice. *C. C. Ungley*. From the Royal Victoria Infirmary, Newcastle-upon-Tyne, England. Brit. M. J. *2:* 908–911, 1950.

The effect of vitamin B_{12} given orally plus normal gastric juice was studied in 8 patients with pernicious anemia. Seitz filtration was shown in 1 patient to lead to loss of activity of the gastric juice. The doses were mostly 50 μg B_{12} plus 500 ml. gastric juice given singly or in divided doses. The results were variable. In 4 patients the response was equivalent to the expected response to a similar parenteral injection of vitamin B_{12}. In the others the response was nil or considerably less than with parenteral B_{12}. The necessary ratio of gastric juice to vitamin B_{12} varied considerably. One hundred ml. of gastric juice appeared to promote absorption of from 0 to 10 μg. of the vitamin.

ABSORPTION OF VITAMIN B_{12} IN PERNICIOUS ANAEMIA. III. Failure of Fresh Milk or Concentrated Whey to Function as Castle's Intrinsic Factor or to Potentiate the Action of Orally Administered Vitamin B_{12}. *C. C. Ungley and G. A. Childs*. From the Royal Victoria Infirmary, Newcastle-upon-Tyne and Glaxo Laboratories, Greenford, Middlesex, England. Brit. M. J. *2:* 911–915, 1950.

Five patients with pernicious anemia were given fresh milk or concentrated whey together with vitamin B_{12}. Although some response was obtained in some of the cases it was concluded that this was no more than might have been expected from the B_{12} alone and that neither fresh milk nor whey showed intrinsic factor activity comparable with that of normal gastric juice.

ABSORPTION OF VITAMIN B_{12} IN PERNICIOUS ANAEMIA. IV. Administration into Buccal Cavity, into Washed Segment of Intestine, or after Partial Sterilization of Bowel. *C. C. Ungley*. From the Royal Victoria Infirmary, Newcastle-upon-Tyne, England. Brit. M. J. *2:* 915–919, 1950.

In 1 patient application of 5 μg. of vitamin B_{12} to the buccal mucosa failed to produce a response. No response was obtained in 2 patients in whom vitamin B_{12} was instilled into washed segments of intestine with and without the addition of normal gastric juice. One patient, first given phthalysulfathiazole and aureomycin to partially sterilize the gut, failed to show improvement after 80 μg. of vitamin B_{12} orally. (This is in contrast to the more protracted observations of Lichtman et al. [Proc. Soc. Exp. Biol. Med., *74*, 884, 1950], who showed improvement in 5 cases of megaloblastic anemia given intestinal antiseptics with and without B_{12}).—*S.C.*

DYSPIGMENTATION OF THE SKIN IN PERNICIOUS ANEMIA. *D. Henke*. From the I Medical Clinic University of Berlin, Germany. Ztschr. f. innere Medizin *5:* 78–85, 1950.

The well known pigmentation in pernicious anemia is recognized merely as a dyspigmentation with areas of both reduced and increased cutaneous pigment. Based on the observation of 7 patients the author finds a relation between the pigment alterations and the distribution of autonomic nerves of the skin. He therefore considers the cause of the pigmentation to be situated in the autonomic ganglion centers of the medulla.—*C.M.*

STATE OF HAEMOGLOBIN IN SICKLE-CELL ANAEMIA. *M. F. Perutz and J. M. Mitchison*. From the Cavendish Laboratory and Molteno Institute and Department of Zoology, University of Cambridge, England. Nature, London. *166:* 677–679, 1950.

Sickle cells were suspended in 0.1 M phosphate buffer at pH 7, saturated with hydrogen sulfide, sealed on a slide and examined under a polarizing microscope. Between crossed

nicols, without compensation, they lit up in a deep blue color and were birefringent. Introduction of an elliptical compensator produced color changes from deep blue through violet to green. When the compensator was turned the other way the color changed to pale pink and white. Normal reduced hemoglobin crystals examined in the same way showed exactly the same characteristics, indicating that sickled cells contain crystalline reduced hemoglobin with the needle axis parallel to the length of the cells. The heme groups appeared to be orientated roughly normal to the needle axis.

The anomalous color effects could be well explained by the dispersion curve.

The solubility of the sickle reduced hemoglobin was found to be no more than one-hundredth of the oxyhemoglobin, in contrast to the normal difference of about a half between normal reduced and oxyhemoglobin.—*S.C.*

AMINO ACID COMPOSITION OF HEMOGLOBINS OF NORMAL NEGROES AND SICKLE-CELL ANEMICS. *W. A. Schroeder, L. M. Kay and I. C. Wells.* From the Gates and Crellin Laboratories of Chemistry, California Institute of Technology, Pasadena, California. J. Biol. Chem. *187:* 221–240, 1950.

Hemoglobins of normal Negroes and sickle-cell anemics were quantitatively analyzed for 17 amino-acids and ammonia. The analyses accounted for approximately 98 per cent of the weight and nitrogen. Sickle-cell hemoglobin apparently contains less lucine and valine and more serine and threonine than normal hemoglobin. This difference would not change the net charge of hemoglobins directly but might alter the configuration of the polypeptide chains and thereby account for the previously described differences in electrophoretic properties.—*P.F.W.*

ANEMIES TARDIVES DU NOUVEAU-NÉ. RÔLE DE L'ALLAITEMENT MATERNEL. (Delayed hemolytic disease of the new born, role of breast feeding.) *M. Goudemant et L. Bayart.* Centre Régional de Transfusion Sanguine, Institut Pasteur de Lill. Semaine d. hôp. Paris. *26:* 2804–2807, 1950.

Three cases of hemolytic disease are reported in infants four to five weeks old, whose mothers were Rh negative, with anti-Rh agglutinins in their blood. In the first 2 cases infants were fed from the breast. In the third case the infant was fed from birth with cow milk.—*J.P.S.*

SLUDGED BLOOD FOLLOWING SEVERE THERMAL BURNS. *J. Brooks, L. R. Dragstedt, L. Warner and M. H. Knisely.* From the Department of Surgery and the Hull Laboratory of Anatomy, University of Chicago, Chicago, Ill. and the Department of Anatomy, Medical College of the State of South Carolina, Charleston, S. C. Arch. Surg. *61:* 387–418, 1950.

Observations were made on animals in whom burns were experimentally produced under anesthesia and in one severely burned human subject as to the physical alterations in the circulating blood in vivo. Vessels in the bulbar conjunctiva and in various transilluminated tissues and internal organs of animals were selected for direct observation by in vivo microscopy.

Following burns, intravascular agglutination of erythrocytes into visible, hard masses resisting passage through smaller vessels was noted shortly after the injury. Leukocytes stuck to the endothelium of vessel walls soon after the burn. Plasma viscosity increased as evidenced by loss of laminar flow and lack of immediate mixing of joining venous strains. Rate of flow was reduced to as little as one-third the pre-burn rate. Leakage of fluid through vessel walls, extravasation of erythrocytes without direct evidence of vessel rupture and small hemorrhages were also observed; and red cells were seen in the phagocytes of spleen, liver and bone marrow. It is suggested that these physical changes in the circulating blood may account for much of the "toxic effect" of burns, and that extravasation loss, thrombus formation and phagocytic loss of erythrocytes may contribute to the postburn anemia.—*W.N.V.*

THE DISTRIBUTION OF COBALT IN POLYCYTHEMIC RATS. *N. I. Berlin*. From the Division of Medical Physics, Donner Laboratory, University of California, Berkeley, Calif. J. Biol. Chem. *187:* 41–45, 1950.

Following a single intraperitoneal injection in rats cobalt is taken up rapidly by the liver, kidney, spleen, lung and blood. However, most of it leaves the tissues quickly and is excreted. On repeated injections there is no marked progressive storage. The amount in the bone marrow may increase because the hematopoietic tissue replaces the fat as polycythemia develops.—*P.F.W.*

IMMUNOHEMATOLOGY

OCCURRENCES IN NORMAL HUMAN SERA OF "INCOMPLETE" FORMS OF "COLD" AUTOANTIBODIES. *J. V. Dacie*. From the Department of Pathology, Postgraduate Medical School of London, England. Nature, London *166:* 36, 1950.

Incomplete "cold" antibodies have been demonstrated by chilling defibrinated blood at 2 to 5 C. for one to two hours, and then washing them repeatedly in saline warmed to 37 C. and exposing them to the action of antiglobulin rabbit serum. The cold hemagglutinins are rapidly eluted into the saline but incomplete antibodies appear to remain adsorbed on the red cells. There is evidence that the presence of fresh serum is necessary for the adsorption of the antibody. Heparin, sodium citrate and Wintrobe's oxalate mixture all inhibit sensitization to some extent.—*S.C.*

A PROBABLE DELETION IN A HUMAN RH CHROMOSOME. *R. R. Race, Ruth Sanger and J. G. Selwyn*. From the Medical Research Council, Blood Group Research Unit, Lister Institute, London, England, and the Pathological Department, General Infirmary, Salisbury, England. Nature, London *166:* 520, 1950.

A blood is described in which no trace of the antigens of the C or E locus was found after testing for C, c, C^w, c^v, C^u, E, e and E^u in saline and albumin, by indirect antiglobulin tests and by trypsin and absorption tests. The serum of the same blood contained anti-e, anti-C and anti-c. It is thought that the most likely genetic interpretation is deletion of a portion of the Rh-chromosome. The fact that C and E are involved supports Fisher's suggestion that the order of the genes on the chromosome would be found to be DCE.—*S.C.*

THE P FACTOR AND ITS VARIANTS IN CAUCASIANS, NEGROES AND CHINESE. *E. B. Miller, H. D. Tannor and C. F. Hsu*. From the Laboratory of the American Red Cross Blood Donor Service, New York, N. Y. J. Lab. & Clin. Med. *36:* 230–233, 1950.

In the routine testing of about 30,000 serum specimens for their anti-A and anti-B agglutinins, five examples of anti-P agglutinin were encountered. Irregular agglutinins of group O sera could not be detected. Using the two most potent of these anti-P agglutinins, 600 Caucasian, 300 Negro, and 190 Chinese blood specimens were tested and the incidence of P-positives was determined. An unusual feature of one of the anti-P sera was its property of agglutinating certain P-positive blood at 37 C. P-positive blood specimens could be divided into at least two and possibly three classes based on the intensity of their reactions. —*G.E.C.*

HETEROPHILE AGGLUTINATION VARIABILITY OF ERYTHROCYTES FROM DIFFERENT SHEEP. *C. J. D. Zarafonetis and H. L. Oster*. From the Department of Internal Medicine, University of Michigan Medical School, Ann Arbor, Mich. J. Lab. & Clin. Med. *36:* 283–287, 1950.

Heterophile agglutination tests were performed on twenty-four sera patients with infectious mononucleosis. In tests on each serum specimen with erythrocytes from twenty-four different sheep, wide variations in agglutination titers were observed.—*G.E.C.*

THE ROLE OF IMMATURE PLASMA CELLS, LYMPHOBLASTS, AND LYMPHOCYTES IN THE FORMATION OF ANTIBODIES, AS ESTABLISHED IN TISSUE CULTURE EXPERIMENTS. *F. J. Keuning and L. B. Van Der Slikke.* From the Department of Histology, State University of Groningen, Groningen, the Netherlands. J. Lab. & Clin. Med. *36:* 167–182, 1950.

Rabbits were immunized by three intravenous injections of paratyphoid B vaccine. One to four days after the last immunizing injection, both white and red splenic pulp and splenic cell suspensions were found to produce agglutinins when explanted in vitro. At the height of antibody production in the animal, red pulp material was found to produce more agglutinin than white pulp. In splenic cell suspensions, both large, immature lymphoid cells and small lymphocytes may have contained agglutinins, but only the former ones were capable of synthesizing antibody, as deduced from experiments in which these cells were separated by sedimentation. The presence of large numbers of these cells in the red pulp, together with the high agglutinin production found in this latter material, supports the hypothesis that these antibodies are produced in the first instance by immature plasma cells of red pulp. The occurrence of immature lymphoid cells in white pulp where no plasma cells are found, together with an apparent production of agglutinin by this material in vitro, and a likewise apparent agglutinin content of mature lymphocytes, suggest that lymphoblastic cells of malpighian corpuscles are also involved in antibody production to some extent.—*G.E.C.*

USE OF BLOOD GROUPS IN HUMAN CLASSIFICATION. *W. C. Boyd.* From the Boston University School of Medicine, Boston, Mass. Science *112:* 187–196, 1950.

This article is too long to abstract but may be of interest to those working in this field. The author concludes his article as follows: "So, although we have found a method of race classification which gives promising results, we have found no indication of the existence of any inherited racial superiority or inferiority. In the present state of confusion about racial issues, this is a point which deserves to be underlined."—*R.C.C.*

KELL-CELLANO BLOOD GROUP SYSTEM IN PREGNANCY AND TRANSFUSION. *J. B. Cochrane, R. H. Malone and I. Dunsford.* From the Nuttingham City Hospital and Sheffield Blood Transfusion Centre, England. Brit. M. J. *2:* 1203–1204, 1950.

A saline agglutinating Kell antibody was found in the serum of a woman who had had transfusions following a post partum hemorrhage in her first pregnancy and two subsequent stillborn children. Later she had a normal child which was Kell negative. In spite of this the Kell antibody titer in the mother's serum rose during this pregnancy. Tests with Levine's Cellano serum supported the view that the Kell and Cellano groups are allelomorphic.—*S.C.*