
A young woman with Hodgkin's disease who developed profound marrow aplasia during treatment with aminochlorambucil, was given an intravenous infusion of her sister's marrow, containing $1.1 \times 10^9$ nucleated cells. Clinical and hematologic improvement began at once, and the blood count was within normal limits 16 weeks after the marrow transfusion. The patient remains well at the time of writing, 9 months after marrow transfusion. Red cells bearing the donor's Rhesus (CDE) and MN antigens were repeatedly demonstrated in the recipient's circulation by various technics and comprised 24 per cent of the circulating red cells 8 months after the marrow transfusions, and 40 per cent after 10 months. No anti-D or anti-E could be demonstrated in the patient's serum at any time. Skin grafts from donor to patient and patient to donor were unsuccessful.—R. M. H.

The enlargement of this month's abstract section was made possible by a kind contribution from Geigy Pharmaceuticals, Ardsley, N. Y.
Intravenous injection of living homologous thoracic duct lymphocytes into the newborn rat caused runt disease in 25 of 30 recipients, while injection of isologous lymphocytes regularly failed to cause runt disease. Thoracic duct lymph was used as a convenient method of separating lymphocytes from granulocytes and monocytes. These studies suggest that some peripheral blood lymphocytes are immunologically competent cells, capable of de novo antibody synthesis.—T. E. B.


Lethally irradiated mice were injected intravenously with concentrated leukocytes from homologous mice having a distinctive hemoglobin and also having a marked granulocytic leukemoid reaction produced by growth of a particular squamous cell carcinoma in the donor mouse. The leukemoid blood did not appear to contain megakaryocytes or nucleated red cells, though it frequently had an elevated reticulocyte count. A small percentage of the leukemoid leukocytes were shown to take up tritiated thymidine in vitro, indicating that they were probably capable of DNA synthesis; the precise cell type of these few leukocytes was uncertain. By the seventy-seventh post-irradiation day the recipient mice showed 100 per cent donor-type hemoglobin. A possible interpretation of this striking result is that certain peripheral blood leukocytes are capable of transplanting and giving rise to mature erythrocytes of donor type in lethally irradiated mice.—T. E. B.


Heterologous isolated splenic cell nuclei were given intramuscularly to rats. In normal animals a leukocytosis was produced, and with subsequent irradiation there was less than the expected leukopenia and a more rapid restoration of the leukocyte count. Acetone extracts were prepared from splenic tissue, and these were found to contain methanolcholesterin, several steroids and alxo-glycerol butyl alcohol. These extracts, whether homologous or heterologous, were protective against acute or chronic radiation injury in mice, rabbits and suckling pigs.—L. D.


When whole blood was passed through a column of siliconized glass wool, the granulocytes were almost quantitatively retained on the column (by a mechanism which remains unknown), and apparently uninjured lymphocytes in 30 to 89 per cent yield were recovered in the effluent fluid. The fate of the monocytes was not mentioned.—T. E. B.


Phosphatidylethanolamine was prepared from egg yolk. Although these preparations contained only the diesters of glycerophosphorylcholine, the fatty acid composition was heterogeneous. Single phosphatidylethanolamine preparations could be separated into subfractions of decreasing unsaturation. The coagulation activity (platelet-like) of these fractions were tested in a thrombin generating system consisting of purified plasma protein fractions to supply prothrombin, PTC, Stuart factor, AHF and AcG, and Ca++ and Mg++. The effect on the coagulation activity of variation in concentration of the lipid, temperature, pH, ionic environment and oxidation were investigated. Although active phospholipid suspension contained no particles visible by phase microscopy at 1000× magnification, all the phospholipid and clot-promoting activity was sedimented by centrifugation for 15 minutes at 105,000 × g. From their findings, the authors suggest that the clot-promoting activity of the phospholipids studied is a property of particles in colloidal suspension. Conditions which alter the colloidal behavior of the particles alter the clot-promoting activity. The colloidal behavior is altered by the degree of unsaturation of the fatty acid in the phospholipid, the temperature, pH and ionic composition. They believe that the active micelles are bimolecular leaflets of limited size, thickness and surface configuration. Once the critical micelle concentration is attained, further addition of lipid acts to form larger micelles, a phenomenon which may explain the inhibitory effect of high concentration of phosphatidylethanolamine. The forces of aggregation between the saturated phospholipid molecules is thought to be higher than those between the...
unsaturated analogues; as a consequence the saturated compounds tend to produce larger aggregates and have less clot-promoting activity.

R. G.


Previous clinical reports have frequently associated disseminated intravascular coagulation, fibrinolysis and hypofibrinogenemia. Phillips, Soulier and Albrechtsen, respectively, have recently proposed a causal concurrent relationship between these isolated events. The experimental approach of the present authors, however, has done much to relate the quantitative levels of fibrinogen to these phenomenon. The generalized Schwartzman reaction was induced in rabbits by the injection of bacterial endotoxin. The resultant disseminated intravascular coagulation caused a moderate depression in the level of circulating fibrinogen. A similar, but smaller depression of fibrinogen occurred when fibrinolysis was produced in rabbits with intravenously injected streptokinase. When both endotoxin and streptokinase were injected in vivo, however, a severe fibrinopenemia or afibrinogenemia developed. Furthermore, when these conditions were simulated by in vitro experiments with streptokinase and small amounts of thrombin, a similar afibrinogenemia developed. The authors suggest that the fibrinolysis acts on the fibrin monomer, or partially polymerized fibrin as soon as it forms, thus accelerating the clotting reaction by removing one of the products of the reaction. The alternative explanation that fibrin lysis in native plasma or serum is more effective than fibrinogenolysis cannot be ruled out, however.—A. J. J.


A "cobamide co-enzyme" was isolated which appeared to differ from vitamin B₁₂ in only one respect: it had an adenine moiety instead of a cyano group. This cobamide co-enzyme was active in catalyzing the interconversion of glutamate and B-methylaspartate. This was the first demonstration of the involvement of vitamin B₁₂ in an isomerase reaction. The authors previously presented evidence suggesting that cobamide co-enzymes may be the major liver storage form of vitamin B₁₂ as well as its metabolically active form (Proc. Nat. Acad. Sci. U. S. 45:521, 1959). The great photolability of the cobamide co-enzymes may be a handicap in studies of these agents in humans.—V. H.


In these three papers, a cobamide co-enzyme was demonstrated to participate in the interconversion of succinate and methylmalonate. This is the second isomerase reaction in which a vitamin B₁₂ co-enzyme has been demonstrated to be involved.—V. H.

ERYTHROCYTES


Agar electrophoresis was carried out on blood specimens with a variety of hemoglobin combinations. The technic of electrophoresis and its several pitfalls are well described. Varying the pH, size of the agar film, concentration of the hemoglobin and percentages of the individual components all influence the patterns obtained. A number of minor components in normal hemoglobin solutions are detected by the agar electrophoretic method. As yet, these are unidentified but may be related to the minor components seen in starch electrophoresis. Agar electrophoresis has proven to be particularly useful in distinguishing Hgb D from Hgb S. Four of 400 patients with the pattern A + S on paper electrophoresis...
were found to have A + D when checked by the agar technic. The authors calculate the prevalence of Hgb D to be 0.085 per cent in the Negro population in Baltimore.—A. I. C.


Arterial oxygen unsaturation is not infrequent in S-S anemia. The authors show that one explanation for this unsaturation may be that the oxygen dissociation curve is shifted to the right. Patients with A-S, A-C and S-A hemoglobin had normal curves. It is suggested that this displacement may be related to an intracranial reduction in pH. This paper, along with the paper by Sproule et al. (J.Clin.Invest. 37:486, 1958), in which an intrapulmonary shunting of blood was demonstrated, appears to give good explanations for the unsaturation of arterial blood and possibly for the cardiomegaly so often observed in S-S disease.—A. J. E.


Tryptic and chymotryptic digests of hemoglobin E were compared with those of hemoglobin A by paper electrophoresis and chromatographic “fingerprinting.” In hemoglobin E, all the peptides were found to occupy the same positions as in hemoglobin A, except that at position 26, which was replaced by two new ones, 26a and 26b. The amino acid composition of these two peptides was determined and compared with that of peptide 26 of hemoglobin A. The changes explain the electrophoretic mobility of hemoglobin E at pH 8.6, but not at pH 6.5. The amino acid changes in hemoglobins S and C affect peptide 4 in the same β peptide chain. This suggests that the genes for these two hemoglobins are allelic, and that the one controlling hemoglobin E is also closely linked, as it affects another peptide in the same chain.—R. M. H.


Tryptic digests of hemoglobin ‘Bart’s’ were compared with those of hemoglobin F, by one-dimensional paper electrophoresis and by chromatographic “fingerprinting.” It was found that all the α-chain peptides were missing from hemoglobin ‘Bart’s.’ Hemoglobin ‘Bart’s’ therefore consists solely of γ-chains, as opposed to hemoglobin F, which contains both α- and γ-chains; it is thus the exact counterpart in the fetal system of hemoglobin H in the adult system, as both these hemoglobins lack α-chains. Alternative explanations for the production of an all α-chain hemoglobin are discussed.—R. M. H.


This is the first recorded instance of a hemoglobinopathy other than thalassemia in a Jewish person. It is of particular interest in that both hemoglobin H and hemoglobin “Bart’s” are deficient in the α-polypeptide chain. The findings suggest that the α-chain deficiency in this mother and daughter represents a single genetic defect.—R. M. H.


Two hundred samples of cord blood were analyzed for their Hgb F content. Correlation of the amount of Hgb F with either prematurity or postmaturity, as well as with birth weight, was attempted. Because of the marked overlap in values between the groups, no significance could be assigned to any single determination. As a group, however, both premature and postmature infants had significantly different average Hgb F concentrations from those found in full term infants. Similar differences were demonstrable using birth weight as the determinant.—A. I. C.


No changes (swelling of the cells, increased osmotic fragility and elevation of plasma potassium) were noted in the red cells with sickle trait after storage for 28 days. Twenty-one day old stored blood of a sickle trait patient survived normally when transfused into a normal individual. There was no evidence of increased splenic sequestration of destroyed cells.—W. J. M.

The obstetric histories of 42 adult Negro women with electromorphically proved hemoglobin abnormalities are reviewed. Of this group, 31 women (with sickle cell anemia, sickle cell-Hgb C disease, thalassemia-Hgb S disease and pure Hgb C disease) experienced a total of 77 pregnancies. Fetal survival exceeded 65 per cent except for patients with sickle cell anemia, of whom only 52 per cent survived. Maternal morbidity was highest in those with sickle cell-Hgb C disease. All four maternal deaths occurred in this same group. The onset of active disease, increasing anemia, bone pain or pulmonary infarction occurred in the last trimester of many women with sickle cell-Hgb C disease. The authors conclude that pregnancy is usually of greater potential hazard to women with sickle cell-Hgb C disease than to those with sickle cell anemia, Hgb C disease or other variants.—A. I. C.


The author discusses different pathogenetic mechanisms in the development of femoral head damage. Breakdown of the hip joint in patients with sickle cell anemia is secondary to local damage to extremely small vessels brought about by several mechanisms. Complete interruption of the blood supply to the head results in the classic picture of destruction. Focal hemorrhages, depending on size and location, may separate off the articular cartilage or leave a persistent, structurally weakening, infarct. Chronic synovitis due to focal hemorrhage may lead to destruction of the cartilage from above. Roentgenographic findings of increased and decreased density are interpreted in the light of the above phenomena.

—A. I. C.


The case of a 30 year old Negro with sickle cell anemia is reported. The disease was complicated by an extracorpuscular erythrocytic defect, proved by the rapid disappearance of tagged normal cells in the patient's circulation, and by megaloblastic changes in the bone marrow which respond to B12 administration. No defect in B12 metabolism was demonstrable and no intrinsic gastric or intestinal disease could be found. The authors suggest that the combination of hemolytic processes may have contributed to the megaloblastic state. However, no mention of a possible aplastic crisis is made.—A. I. C.


The use of two different carbonic anhydrase inhibitors in 4 adult patients with sickle cell anemia was without significant effect on the course of the disease. Observations ranged over a period of up to 7 months, without consistent or beneficial effects being noted.—A. I. C.

Clinical and Laboratory Features of Two Variants of Methemoglobin M Disease. A. V. Pisciotta, S. N. Ebbe and J. E. Hinz. From the Department of Internal Medicine, Marquette University School of Medicine, Milwaukee, Wis. J.Lab.& Clin.Med. 54:73-87, 1959.

Methemoglobin M disease, a hereditary anomaly of globin first described by Horlein and Weber, has now been described in several additional families. Two new families are presented by the author with findings suggestive of at least two different varieties of Hgb M. Both methemoglobins showed an abnormal spectrophotometric curve but revealed differences in their reaction with potassium cyanide. Methemoglobin reductase activity was normal in the erythrocytes of the affected members. One of the families had the unusual combination of an abnormal hemoglobin, possibly Hgb C, in combination with the Hgb M anomaly. Since the evidence presented suggests several variants of Hgb M, subscripts B (Boston type) M (Milwaukee type) and S (Saskatoon type) are suggested to identify these varieties.—A. I. C.


A colored student from Ghana developed acute intravascular hemolysis and methemoglobinemia following the ingestion during 5 days of 21 compound aspirin tablets, containing a total of 3.5 Gm. phenacetin. Tests of in vitro Heinz body production in the presence of acetylphenyldrazine, and of glutathione stability,
showed that the patient's red cells behaved similarly to those of primaquine-sensitive patients. Cr\[^{51}\] labeled red cells from the patient were injected into the same normal recipient on two occasions with 1 month's interval. The survival was normal on the first occasion, but during the second test the recipient took phenacetin by mouth and the survival of the transfused patient's cells was grossly reduced.—R. M. H.

**The Reduction of Methemoglobin in Human Erythrocytes Incubated with Purine Nucleosides. Ernst R. Jaffe. From the Department of Medicine, Albert Einstein College of Medicine and the Bronx Municipal Hospital Center, New York, N. Y. J.Clin.Invest. 38:1555-1563, 1959.**

The capacity of various compounds to serve as substrates for the reduction of methemoglobin in intact human erythrocytes has been investigated. Effective compounds included the following: glucose, adenosine, guanosine, inosine, 2,6-diaminopurine ribose, xanthosine, deoxyguanosine, deoxyadenosine, galactose, fructose, fumaric acid ribose and lactate. A variety of pyrimidine ribosides, pyrimidine deoxyribosides, nucleotides, phosphorylated intermediates of carbohydrate metabolism and other compounds were found to be lacking in ability to promote reduction of methemoglobin in erythrocytes. Studies were also carried out using the red cells from a patient with congenital methemoglobinemia. Incubation of these erythrocytes with the purine nucleosides and sugars that promoted methemoglobin reduction in normal erythrocytes did not result in significant reduction of methemoglobin to hemoglobin. Lactic acid production by erythrocytes of congenital methemoglobinemic patient was found to be slightly increased when compared with normal control erythrocytes. These findings are consistent with Gibson's concept of a defect in electron transport from DPNH to methemoglobin in this disease.—E. B.


Only proerythroblasts are diploid; their reproduction is a homoplastic one. The other erythroblasts are hypoploid.—H. M.

**The Hemolytic Effect of Various Sulfonamides on Subjects with a Deficiency of Glucose-6-Phosphate Dehydrogenase of Erythrocytes. A. Széneberg, M. Pras, Ch. Sheba, A. Adam and B. Ramot. From Tel-Hashomer Government Hospital, Israel. Israel M.J. 18:176, 1959.**

Sulfapyridine produced acute hemolysis in three erythrocyte glucose-6-phosphate dehydrogenase-deficient subjects on the fourth day of drug administration. No such reactions were observed in the control group. Sulfadiazine did not cause any demonstrable blood destruction, but one patient developed mild hemolysis after trisulfa.—B. R.


After oral administration of 15 Gm. l-histidine daily for 2 to 3 days, only patients with folic acid deficiency excreted in urine amounts of formiminoglutamic acid (FCA) exceeding 30 \(\mu g./\)per milliliter. Other workers have not felt that the presence of FCA in the urine is an entirely satisfactory test for folic acid deficiency at present (C. S. Davidson and J. H. Jandl, Am. J.Clin.Nutrition. 7:711, 1959). Further studies are required to demonstrate that this test will not lead to a false diagnosis of primary folic acid deficiency in some patients whose supply of folic acid is adequate but who may be unable to utilize it adequately due to vitamin B\(_12\) deficiency (M. Silverman and A. J. Pitney, J.Biol. Chem. 233:1179, 1958; V. Herbert, The Megaloblastic Anemias, New York, Grune & Stratton, 1959). In any event, it is quite probable that the large metabolic load with l-histidine suggested by the authors will invariably demonstrate the existence of inadequate folic acid metabolism, though it may not indicate such defect is due to inadequate supply of folic acid in the body. —V. H.

**Megaloblastic Anaemia during Antiepileptic Therapy. T. Wilkinaon. Royal Newcastle Hospital, Newcastle, N.S.W., Australia. Med.J. Australia. 2:894, 1959.**

This is a case report of a patient who developed megaloblastic anemia during antiepileptic therapy with phenytoin and phenobarbitone. His blood picture returned to normal following treatment with folic acid, and has remained normal with a maintenance dose of folic acid, despite the continued administration of phenytoin and phenobarbitone.—G. C. de G.

**Hemolytic Disease of the New-Born. R. J. Walsh and H. K. Ward. From the New South...**
This paper analyzes the fate of the infants of 395 immunized Rh-negative women. The severity of the disease in the first affected child frequently sets the pattern for subsequent children. If the first affected child is mildly affected, the subsequent children are more likely to survive than to die; and first-born severely affected children are likely to be followed by stillbirths or fatally affected children. The parity of the mother when the first affected child is born is not related to the chances of survival of the infant. The severity of the disease increases as the mean titer of the incomplete antibody in the maternal serum rises, but there are numerous exceptions to the general rule. The effects of immunization by a blood transfusion are similar to those when immunization follows a pregnancy. The frequency of stillbirths is not significantly greater when immunization follows transfusions.—G. C. de G.


A newborn infant presented the clinical signs of hemorrhagic shock at delivery but responded to immediate blood transfusion. Although the mother was Rh negative, it was the first pregnancy and there were no signs of immunization. By the Ashby technic fetal cells were demonstrated in the maternal blood. These cells disappeared by the fiftieth postpartum day with subsequent development of Rh antibodies. The data are interpreted as indicating fetal bleeding into the maternal circulation.—M. J.


Reticular cells, during the course of digesting erythrocytes, contain molecules of ferritin among the phagocytosed stroma. Ferritin often has a crystalline arrangement. Sometimes ferritin and apoferritin are arranged alternately in these crystals, and sometimes hemosiderin contains crystals which seem to be pure apoferritin. Animal experiments have shown that after the injection of iron salts, ferritin appears in reticular cells after an interval of 3 days. An intermediate aspect between that of the injected iron and that of the ferritin has been found. In the case of injected saccharated iron fine needles are formed, whereas the injection of lactated iron produces fibrous masses.—O. P. J.


Electron microscope studies of iron transport to erythroblasts and its subsequent incorporation within them were made on human bone marrow from patients with hemochromatosis, hypersiderocytic hypochromic anemia, acute rheumatoid arthritis, thalassemia major, and thalassemia minor associated or not with other hemoglobinopathies. In addition to these, bone marrow, spleen, liver and lymph nodes preparations were studied after numerous rats had been subjected to diverse experimental procedures. In erythroblastic islands, iron is obtained from reticular cells by a process similar to pinocytosis, which the authors have designated rhophagocytosis. Iron may be acquired by the reticular cells either by direct destruction of erythrocytes or by way of transferrin. Perhaps some iron is carried directly to erythroblasts by transferrin. Mitochondria rarely contain iron in the normal state, but it may be present either as ferritin or ferruginous micelles. In the thalassemias and other disorders accompanied by a disturbance in hemoglobinogenesis, a large quantity of iron is found in the erythroblasts as well as in the mitochondria. It seems that a disturbance in hemoglobin synthesis causes the unused iron to accumulate in hypochromic erythroblasts. Apparently iron is normally metabolized in mitochondria, but this mechanism is blocked in the severe anemias.—O. P. J.


Relative marrow insufficiency was produced in rats by repeated bleeding over a period of 5 months. This was not produced in animals in which iron and protein depletion was avoided by reinfusion of lyophilized plasma.—G. M.


This paper reports the results of experiments in rats which were designed to investigate the value of adding ascorbic acid to therapeutic inorganic iron compounds. A solution of ferrous sulfate labeled with Fe^{59} was administered to rats by stomach tube, and the amount absorbed was determined by radioassay of the blood, liver and spleen 3 weeks later. The amount absorbed was significantly increased when the iron was administered together with ascorbic acid. The addition of milk to the administered iron solution did not reduce the amount absorbed.—G. C. de G.


Among 1100 women examined immediately following delivery, 312 (28 per cent) were found to be anemic, i.e., the hemoglobin content was found to be below 10 Gm. per cent. Hematologic examinations carried out in 70 anemic women included blood counts, serum iron and unsaturated iron binding capacity, and serum vitamin B_{12} determinations. On the basis of the morphologic findings as well as the biochemical investigations, the following results were obtained: 34 per cent of the anemic women were found to be suffering from iron deficiency anemia with low serum iron and normal serum vitamin B_{12} levels. In 9 per cent a pure macrocytic anemia was found with low serum vitamin B_{12} and normal serum iron levels. In the remaining 57 per cent the anemia was of a dimorphic type with both serum iron and vitamin B_{12} levels much below normal, indicating a double deficiency. Of 49 women low folic acid levels were found in 20.—B. R.


Secondary sideroachrestic anemias are the anemias accompanying infectious diseases, the anemia in chronic lead poisoning (in the latter case there seem to be 3 blocks: (1) in the synthesis of delta-aminolevulinic acid, (2) in the uptake of iron into the protoporphyrin and (3) in the synthesis of protoporphyrin), the anemias in certain vitamin deficiency (pyridoxine, ascorbic acid) and thalassemia. Essential types are the "anemia hypochromica siderochrestica hereditaria" and the "anemia refractaria sideroblastica." The first begins in childhood, and has a chronic, benign course. The life span of the red cells is normal, osmotic resistance is increased, there are no signs of hyperhemolysis. The marrow shows increased erythroblasts. The serum iron is normal, but plasma clearance and iron turnover are accelerated. Red cell protoporphyrin is decreased, and coproporphyrin is increased. There are no hemoglobin anomalies; leukocytes and platelets are normal. Anemia refractaria sideroblastica becomes manifest in older patients, and conversion into leukemia is common. Leukopenia and occasionally thrombocytopenia occur. The marrow is hyperplastic, and there are some megaloblastoid erythroblasts. Protoporphyrin in the red cells is normal or increased, and the lack of alkaline phosphatase in the leukocytes is a frequent finding. Differentiation between the congenital and acquired types is possible by investigation of porphyrin metabolism, since there is lack of protoporphyrin in the congenital type. In 4 of 5 investigated cases of both types siderosis of the liver was found, and it is not impossible that there is a relation to hemachromatosis. Although neither variety shows hyperhemolysis, the output of urobinol compounds may be increased; there seems to be occult hemolysis, perhaps occurring in the marrow.—H. M.

RESEARCH "IN VIVO" AND "IN VITRO" ON THE BIOLOGICAL ACTIVITIES OF ERYTHROBLASTS IN CASES OF ERYTHROPATHY DUE TO BENZENE. E. C. Rondanelli, P. Gorini, D. Pecorari and R. Colombi. From the Università, Pavia, Italy. Lavoro Umano 11:1–13, 1959.

In 4 cases of chronic benzene poisoning, maturation of erythrocytes was studied in the bone marrow, and by means of the statocytometric test. In all cases the maturation of the erythrocytes was slower than normal in the basophilic phase. Furthermore, the proliferative activity of both basophilic and polychromatophilic erythroblasts was reduced. The duration of mitosis in the same cells was prolonged, owing to a prolongation of the metaphase and, to a lesser extent, of the anaphase.—P. d. N.


Erythropoietin obtained by ultracentrifugation of urine from a patient with PNH was bioassayed exposing normal rats to
simulated altitudes of 20,000 feet. The 17 hour radioactive iron utilization was measured in normal, fasted, and hypophysectomized rats after 2 days' administration of erythropoietin. A significant correlation was found between the red cell incorporation of radioiron and the dose of erythropoietin. Furthermore, there was a high correlation between the increase in total circulating hemoglobin and in Fe\(^{59}\) incorporation into red cells. These studies establish convincingly that material obtained from urine may have an erythropoietic activity. However, the correlation found between the increase in circulating hemoglobin and the 17 hour Fe\(^{59}\) utilization is remarkable, in view of the many factors which are involved in the early appearance of labeled red cells in the circulation. The per cent of injected Fe\(^{59}\) which can be recovered from circulating red cells after 17 hours is primarily determined by the rate of release of labeled cells from the bone marrow, by uptake in circulating reticulocytes and by competitive uptake in tissues other than the bone marrow. Despite the statistical data, it may still be wise to consider the Fe\(^{59}\) utilization a useful qualitative test rather than an exact quantitative test of changes in red cell production.—A. J. E.


Rat bone marrow suspension was grown in a mixture of \(\frac{3}{4}\) Eagle medium and \(\frac{1}{4}\) serum in a sealed culture chamber for 12 and 24 hours. Careful differential counts and total cell counts were carried out, and the effect on these counts of normal rat serum, serum from anemic rats, serum from polycythemic rats, serum from rats given cobalt and serum with cobalt added was evaluated. A significant increase in nucleated red cells was demonstrated only in bone marrow exposed to anemic serum or to serum from cobalt-treated rats. These interesting results show that it may be possible to develop an in vitro technic for the study of erythropoietins, although it must be hoped that it can be based on a less vulnerable measurement than the cell count of bone marrow suspensions.—A. J. E.


The method for the demonstration of an impure erythropoietic active substance from the plasma is given. This factor cannot be precipitated by heat, alcohol, trichloracetic acid or perchloric acid. The substance is nondialyzable and is stable between —20 C. and +4 C. By electrophoresis (pH 8.6, ionic strength 0.075–0.1), it has the mobility of \(\alpha\)-globulin. By high voltage electrophoresis, there are 2 fractions: at pH 3.6 one of them shows no migration, but the other migrates towards the anode. The content of carbohydrates and neuraminic acid (sialic acid) in the first fraction is high, indicating a glycoprotein or a mucopolysaccharide. This agent is found in increased amounts in the plasma of patients with symptomatic or idiopathic polycythemia, and there was no difference in untreated patients and those who received P\(^{32}\).—H. M.

**MECHANISM OF HEMATOPOIESIS. HEMATOPOIETIC EFFECTS OF SERUM ALBUMIN; HEMATOPOIETIC REGULATORS IN SERUM ALBUMIN. B. Steinberg, A. A. Dietz and M. A. Atamer.** From Toledo Hospital Institute of Medical Research, Toledo, Ohio. Arch.Path. 67:489–495; 496–504, 1959.

These two papers are part of a series devoted to the exploration of the nature of the regulatory mechanism of hematopoiesis, including initiation of blood cell production, selective maturation, delivery of specific numbers of cells into the blood stream, maintenance of cell ratios, cell aging, and time-spaced disintegration. The concept is advanced that the serum albumin is concerned with hematopoiesis. This protein constituent of serum contains regulators of hemocytopenin type, which control the degree of maturation and proliferation of cells in the marrow. Fractions of serum albumin from normal people, obtained by the action of sodium tetrametaphosphate and continuous-flow electrophoresis, produced several changes in the marrow and peripheral blood of rabbits. Perhaps a pair of regulators exists for each cell type, one concerned with cell proliferation and maturation, and the other regulator holding these functions to a physiologic level.—O. P. J.


The cells were incubated up to 15 minutes with \(\alpha\)-naphthylacetate, 20 to 30 minutes with naphthol-AS-acetate. There were occasional cells with a relative high enzyme activity. As was demonstrated by Pappenheim and with toluidine-blue stain—these cells were blood mast cells and monocytes.—H. M.
Abstracts


Treatment with salicylate significantly reduces the potassium content and markedly increases the sodium and water content of erythrocytes both in vivo and in vitro. Since the maintenance of the electrolyte balance of cells requires energy, it is suggested that salicylate may interfere with the production or adequate utilization of energy in the erythrocytes.—S. R. H.


In a thorough study of the dynamics of hemorrhagic shock the authors have shown the importance of considering both oxygen-carrying capacity and cardiac output in the symptomatology of anemias and polycythemias. Dogs were rendered anemic or polycythemic and examined after having been made hypotensive. The flow of oxygen-containing blood to the tissues was calculated by multiplying cardiac output with arterial oxygen content. When plotted against hematocrit the flow of oxygen was found to reach a distinct maximum at 42 per cent hematocrit. The reason for this shape of the curve is that flow decreases as hematocrit and viscosity increase and oxygen-carrying capacity increases as hematocrit increases. Oxygen consumption was determined, plotted against the hematocrit and found to have a similar optimum at a hematocrit of 42 per cent. Consequently this was the value at which most oxygen was consumed and at which the oxygen deficit was smallest. In addition, it was found that the maximum resistance to hemorrhagic shock coincided with this optimum hematocrit.

In dogs it was shown that a reduction in the oxygen saturation of blood perfusing a hind leg was accompanied by vasodilation and increased blood flow. This compensatory mechanism was exceedingly efficient and would minimize tissue hypoxemia. It was found that the increase in blood flow was not caused by an increased pCO2, and it was thought possible that the lack of oxygen supply directly to the muscle fibers of the blood vessels would cause these to relax.—A. J. E.

Hemostasis


Adsorbed plasma, serum, a cephalin reagent and calcium were used to produce thromboplastin activity. By high speed centrifugation the thromboplastin was sedimented on the crude cephalin. Activity was lost if washing of the sediment was undertaken in the absence of calcium ion. The thromboplastic activity of the resuspended sediment was more stable than that of the usual thromboplastin generation mixture. Its activity was irreversibly destroyed following incubation with serum. It normally clotted plasma deficient in Stuart factor or factor V.—R. G.


The effect on hemostasis of lyophilized and fresh platelets was determined by their ability to decrease the output of red blood cells in the thoracic duct lymph of dogs made thrombocytopenic by irradiation. Eight dogs were used. Each experiment is reported in detail. In 3 dogs lyophilized platelets alone were given; in 4, lyophilized platelets were followed by infusion of fresh platelets; in 1, fresh platelets were given first and were followed by an infusion of lyophilized platelets. Lyophilized dog platelets were infused in amounts calculated to increase the recipient's platelet level by approximately 200,000 per cubic millimeter. The circulating platelet levels did not increase following these infusions, and the output of red cells in the lymph did not decrease significantly. Those
animals receiving fresh platelet concentrates all showed a rise in circulating platelets, although usually considerably below the calculated one, and there was a significant decrease in the output of red cells in the lymph. This report, in agreement with others, indicates that viable platelets in addition to platelet thromboplastic activity, are necessary for maintenance of hemostasis.—R. G.


Continuous flow electrophoresis fractionation was applied to plasmas deficient in PTC or AHF and to one plasma from a patient with AHF deficiency and an acquired inhibitor. Fractions from nonresistant hemophilic plasmas showed no AHF activity and no anti-AHF activity. In plasma from one "mild" hemophilic, no anti-AHF was found, and one fraction showed AHF activity. In the plasma from the resistant hemophilic, anti-AHF activity was found in the gamma globulin area, and there was no AHF activity in any fractions. Fractions from PTC-deficient plasmas showed no PTC or anti-PTC activity. The findings with the AHF-deficient plasmas is further evidence against the theory that classic hemophilia is due to the binding of AHF by a circulating inhibitor.—R. G.


The effect of heparin on thromboplastin formation was studied in the thromboplastin generation test. It markedly inhibits the formation of thromboplastin, and when added to generating mixture after the point of optimum generation of thromboplastin, it causes a marked and rapid deterioration of activity. The effect on the inhibitory action of heparin of varying the concentration of platelets or cephalin, adsorbed plasma and serum is described. The authors believe that heparin interferes with thromboplastin generation by interacting with or antagonizing whatever is responsible for PTC activity; they suggest that it may interfere with the formation of a thrombin-PTC complex necessary for PTC activity.—R. G.


Dogs were irradiated, and when they developed severe thrombocytopenia and purpura, their thoracic duct was cannulated and the output of red cells per minute in the lymph determined. The effectiveness in correcting bleeding by fresh canine platelets disintegrated by a sonic oscillator and by fresh intact platelets was determined by their ability to decrease the number of cells in the lymph. Simultaneously, studies were done on the ability of the two platelet preparations to correct the prothrombin consumption of the recipient's blood. Disintegrated platelets corrected prothrombin consumption but did not decrease the red cells in the lymph, whereas fresh whole platelets causing the same degree of correction of prothrombin consumption also produced a marked decrease of red cells in the lymph. The authors conclude that a normal clotting system is not enough for normal hemostasis, but that the mechanical effect of intact platelets is also needed.—R. G.


The sera of the majority of patients with liver disease show an abnormality in the thromboplastin generation when tested with normal plasma. According to the authors, this defect is not due to PTC or Stuart factor deficiency, but rather to the same deficiency as seen with stored plasma. This was originally referred to as factor X by Duckert et al. (It should be noted that factor X is now referred to by some authors as Stuart-Prower factor.) The authors believe that this test may be positive when other liver function tests are normal and suggest that this test, which they refer to as the serum thromboplastin generation test (STGT), may be of value as a liver function test.—R. G.

**VASCULAR ANOMALY ASSOCIATED WITH PLASMA THROMBOPLASTIN ANTECEDENT DEFICIENCY.** P. G. Frick, F. Bachmann and F. Duckert. From the Department of Internal Medicine, University of Zurich, Zurich, Switzerland. J. Lab. & Clin. Med. 54:680–684, 1959.
ABSTRACTS

This paper is essentially a case report of a 21 year old female who began having purpura at the age of 15. At the time of study the following abnormalities were found: slightly prolonged bleeding time, a greatly increased capillary fragility, abnormal appearing capillaries in the nail beds, and thromboplastin antecedent approximately 12 per cent of normal. There was no family history of any hemorrhagic tendencies. There was no evidence of any platelet abnormality in the studies done.—R. G.

LEUKOCYTES

LYMPHOCYTOPHORESIS IN THE BURSA OF FABRICIUS.

The histogenesis of the lymphoid follicles in the bursa of Fabricius, a lympho-epithelial organ peculiar to birds, was studied in 160 white Leghorn chicks and embryos. The morphologic and histochemical evidence indicates the epithelial origin of the lymphocyte during the early stages of development in the bursa of Fabricius of the chick. A continuous line of transitional stages from undifferentiated epithelial cells to mature small lymphocytes in the medullary portion of the follicle was easily discernible. Such changes relate to the size and contour of the cells, their chromatin pattern, nucleoli, degree of cytoplasmic basophilia and size and number of mitochondria. Minor changes were also apparent in sudanophilic and PAS-positive granules. Lymphocytes may develop, at least in this organ, from sources other than mesodermal derivatives.—O. P. J.


During a series of studies on plasma cells, it was found that lymphoid tissues usually contain variable numbers of small lymphocytes with nuclei. These are characteristic among themselves but are atypical on several scores from the nuclei of the other small lymphocytes about them. The nuclei contain larger amounts of chromatin and this is intermingled with other substances that have solubilities of specific lipids, but which fail to stain as lipids under the given experimental conditions. The process involved in the development of these lymphocytes begins in prophase and progresses into distorted stages of metanaphase. There seems to be a combination of the lipid-soluble substances with other nuclear components.—O. P. J.


Leukocytes derived from fresh horse serum were cultured for 24 to 64 hours. The neutrophils and the eosinophils did not undergo any change, but after 48 hours the lymphocytes and the monocytes transformed first into polyblasts and then macrophages. The change was that of general cell enlargement and of the acquisition of the properties of ameboid movement and phagocytosis. The cells were subsequently incubated for 1 hour with substrates such as ATP-ic acid, RNA or Na-β-glycerophosphate and stained for alkaline phosphatase activity. The black cytoplasmic coloration was absent in lymphocytes and monocytes but strongly present in polyblasts and macrophages. As the cells became finally transformed into histoid cells, the color faded. The nuclei did not stain at any stage, but nucleoli of polyblasts did to a mild degree. There may exist a relationship between alkaline phosphatase activity and phagocytosis, as far as the dealing with ingested material is concerned.—J. J. B.


Other workers have shown that Charcot-Leyden crystals can develop in vitro in eosinophil leukocytes within a few minutes after the application of surface-active wetting agents. The present investigation is the first report of electron microscope examination of developing crystals. Blood was obtained from patients suffering from a variety of allergic disorders, and the best preparations were made from a patient with severe bronchial asthma with a 75 per cent eosinophilia. Aerosol OT, di-octyl sodium sulfosuccinate, was used as the wetting agent. The results of these studies on ultra-thin sections indicate that a portion of the eosinophil granule acts as a nidus in the genesis of the crystal. Apparently the inner substance of the eosinophil granules served as the basic protein building material for the crystal.—O. P. J.

FLUORESCENT ANTIGLOBULIN STUDIES IN LEUKOPENIA AND RELATED DISORDERS. P. Calabresi, E. A. Edwards and R. F. Schilling. From the
ABSTRACTS

- T. E. B.

AGRAINULOCYTOSIS FOLLOWING plasia, usually following prolonged exposure to systemic lupus erythematosus and of all patients fore any therapy had been instituted. One of described. Globulins active against leukocyte nuclei by the fluorescent antiglobulin technic is de-

jaundice developed, as well as circulatory renal in a disease spectrum involving simple rheu-
gest that Felty's syndrome may be a connecting

ACQUIRED PSEUDO-PELGER ANOMALY OF GisNuLo-

ACQUIRED PSEUDO-PELGER ANOMALY OF GRANULO-

The patient presented with pneumonia, and of special interest was the finding of many neutrophils in the sputum when there were virtually none in the peripheral blood.—T. E. B.


Immuno-electrophoretic analysis with 2 anti-
sera to normal human serum and 1 anti-gamma globulin serum was performed on 60 patients with acute leukemia, 30 with chronic myeloid leukemia and 20 with chronic lymphatic leukemia. In patients with chronic lymphatic leukemia, marked changes were often found in the $\beta_2 A$, $\beta_2 M$ globulins and in the gamma globulins; a considerable reduction in the gamma and $\beta_2$ globulins, or only in the $\beta_2$ globulins occurred, although rarely these were increased; and in one case macroglobulinemia was found. In chronic myeloid leukemia gamma globulins, $\beta_A$ globulins and $\alpha_2$ macroglobulin were frequently increased. In acute leukemia the changes were inconstant and variable. In 1 case of acute lymphoblastic leukemia, "agammaglobulinemia" was found.—G. M.

PREDNISONE IN CHILDHOOD LEUKEMIA. COMPARISON OF INTERRUPTED WITH CONTINUOUS THERAPY. Carol B. Hyman, Eduardo Borda C, Charles Brubaker, Denman Hammond and Phillip Sturgeon. From the University of Southern California School of Medicine, Los Angeles, Calif. Pediatrics 24:1005—1008, 1959.

The authors compare continuous with inter-
rupted steroid therapy in the treatment of child-
hood leukemia. All patients in the study had initially been treated with 6-mercaptopurine either alone or in combination with azaserine or DON (6-diazo-5-ox-L-norleucine). Following relapse from their initial course of therapy, all patients were given a 28 day course of prednisone in a dose of 2.2 mg. per kilogram per day. At the end of this period, steroid therapy was tapered over the next week and stopped in one group of patients and was continued in a dose of 0.55 mg. per kilogram per day in the second group. The groups did not differ significantly in the number of remissions induced, their duration or the dura-
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tion of clinical control, and therefore the study did not indicate any advantage for continuous over interrupted therapy with prednisone. More-
over, the continuous therapy had the added disadvantages of increased side effects, higher cost and the possibility of permanent refractoriness to the drug.—J. S.


This paper reports a study of the type, age and sex incidence of leukemia in Adelaide public hospitals from 1933 to 1953, and of the numerical incidence of the disease from mortality rates in both South Australia and the Commonwealth of Australia from 1929 to 1956. The findings indicate that a considerable increase in mortality rates in both State and Commonwealth has occurred, and that this increase has been comparable to that reported from the U. S. A. In South Australia, the incidence of deaths from leukemia per 100,000 rose from 1.03 in 1929 to 5.18 in 1956. In the Commonwealth as a whole, the rise over the same period was from 1.88 to 5.21. A statistical analysis of the Commonwealth figures suggests a steady rate of increase from 1940 to 1945 and from 1947 to 1956, with a sudden increase in frequency of deaths at 1947. The rate of increase from 1933 to 1940 was nearly 10 per cent per annum. The mortality rates for 1933, 1940, 1947 and 1954 divided into quinquennial age periods also suggest that the increase after 1940 has been mainly in the older age groups. The type, age and sex incidence studied from 295 hospital cases in Adelaide show few unusual features. Chronic myeloid leukemia was the commonest type, but a greater increase in acute leukemia and the chronic lymphatic type had occurred, and that this increase has been comparable to that reported from the U. S. A. In South Australia, the incidence of deaths from leukemia per 100,000 rose from 1.03 in 1929 to 5.18 in 1956. In the Commonwealth as a whole, the rise over the same period was from 1.88 to 5.21. A statistical analysis of the Commonwealth figures suggests a steady rate of increase from 1940 to 1945 and from 1947 to 1956, with a sudden increase in frequency of deaths at 1947. The rate of increase from 1933 to 1940 was nearly 10 per cent per annum. The mortality rates for 1933, 1940, 1947 and 1954 divided into quinquennial age periods also suggest that the increase after 1940 has been mainly in the older age groups.


Secondary neoplastic involvement of the thyroid gland is generally considered uncommon, but relevant literature shows that this is not necessarily so. Of the 300 necropsies studied with generalized lymphoblastoma, 53 showed involvement of the thyroid gland. There were 188 examples of leukemic lymphoblastoma, and 34 of these showed involvement of the thyroid gland. Thyroid glands from the 112 patients with non-leukemic lymphoblastoma were involved in 19 instances. It is probable that this involvement is by way of the blood stream. Neoplastic transformation of the normal reticuloendothelial-cell component of the thyroid gland occurs, though much less frequently, and invasion by contiguity is rare. The fact that none of these patients experienced enlargement of the thyroid gland during life as a primary manifestation of the lymphoblastomatous process suggests that primary lymphoma of the thyroid gland is usually so characteristic in its clinical manifestations as to constitute a syndrome.—O. P. J.


The authors report a follow-up study of 26 apparently normal children who had splenectomy carried out for traumatic rupture. The ages at splenectomy ranged from 2 to 15 years, and the duration of the follow-up from 2 months to 10 years. At the time of follow-up, all 26 children were alive and well and had demonstrated no history of pneumonia, meningitis, pericarditis or septicemia.—I. S.


MISCELLANEOUS


The serum of a patient with systemic lupus erythematosus was fractionated by column chromatography, and the 10 resulting fractions were tested for their reaction with human liver nucleoprotein and with mouse liver cell nuclei and for their ability to produce L.E. cells. The L.E. cell activity was found entirely in the first fraction, consisting of 6.8(7) S gamma globulins. Most of the activity against human liver nucleoprotein, demonstrated by a tanned red cell
Technic, and against mouse liver cell nuclei, shown by means of fluorescein-labeled antihuman gamma globulin, was found in the seventh and eighth fractions. Ultracentrifuge studies showed that these last 2 activities resided in the human gamma globulin.


Forty serum samples from 19 patients with disseminated lupus erythematosus were studied. In almost half of these sera anti-thrombocytic antibodies were found by precipitin and complement fixation reactions. The presence of anti-leukocytic antibodies has been demonstrated by the same technics. It was possible to distinguish 3 varieties of anti-leukocytic antibodies which are present singly or in combination in most sera: (a) anti-DNA antibodies; (b) autoantibodies reacting with leukocytic constituents other than DNA; (c) post-transfusional isoantibodies. The substance in L.E. sera reacting with DNA has been demonstrated to be an antibody because it could be extracted and isolated from the specific precipitate and shown to have the antigenic properties of gamma globulins. This anti-DNA antibody was found in almost all the patients who had received no treatment. No positive reaction has up to now been found except in L.E. The anti-DNA antibodies react with a specific chemical group common to DNA from many sources (human, animal, bacterial). The gamma globulins extracted from the specific precipitate (L.E. serum + DNA) induce a typical L.E. phenomenon in the presence of normal leukocytes and normal fresh serum. These anti-DNA antibodies may play an important part in the formation of the diffuse nuclear lesions of L.E.—G. M.


Of the sera of 25 patients with S.L.E., 4 contained complement-fixing antibodies to rat liver nuclei, calf thymus nuclei and calf thymus DNA, rat liver mitochondria and rat liver soluble cellular protein. Five others reacted with the 2 cytoplasmic antigens and 6 with rat liver mitochondria only; 10 failed to react with any of the antigens. All those sera which reacted with nuclear antigens gave a positive L.E. cell test, as did 5 of 11 which reacted with cytoplasmic antigens only and 4 of 10 nonreacting sera. The sera of patients with lupoid hepatitis, primary biliary cirrhosis, macroglobulinemia and syphilis reacted with cytoplasmic but not with nuclear antigens. These findings are held to support the autoimmune concept of the pathogenesis of S.L.E.—R. M. H.


Hematoxylin bodies were found in two or more organs in 17 of 20 cases of systemic lupus erythematosus. Only 3 cases of S.L.E. did not show these changes. Of 70 control cases, 3 showed single hematoxylin bodies. These were scleroderma, cirrhosis and subacute nephritis and multiple myeloma.—W. J. M.


The absorption of ribonuclease by ascites tumor cells and bone marrow cells from young rats has been followed by measuring the enzymatic activity and radioactivity of cellular suspensions which were incubated in the presence of 113I-labeled enzyme. The experiments show that RNase penetrates into the cell; the rate of penetration depends on the concentration of the enzyme in the medium. RNase absorption in ascites tumor cells is not influenced by temperature unless the RNase concentration exceeds a certain value. This suggests that several mechanisms (diffusion, membrane activity, pinocytosis), differently affected by temperature, play a role in the absorption of RNase by the cell.—O. P. J.
splenic tissue of dogs transferred to nonpretreated control mice failed to "take."—S. R. H.

ADAPTATION TO TISSUE CULTURE AND HETERO-
TRANSPANTATION OF SPLENIC TISSUE. G. Gy.
Csaba and M. Iskum. From the Medical Uni-
versity, Budapest. Acta Morph. Hung. 8:70-76,
1958.

Heterologous splenic tissue was transplanted into the abdominal cavity of 60 albino rats and mice, respectively. Prior to implantation, the splenic tissue was grown in tissue culture, in the serum of the recipient. The method of this adaptation ensured the survival of the hetero-
transplants in 50 per cent of the cases.—S. R. H.

THE PATHOLOGY AND PATHOGENESIS OF HEPATO-
splenitic Disease Associated with Schistosomi-
asis. P. K. Hamilton, H. S. Hutchison, P.
W. Jamison and H. L. Jones. From the Ameri-
can Mission Hospital, Tauta, Egypt. Am. J. Clin.
Pathol. 32:18-33, 1959.

Schistosomiasis is a major etiologic factor of the disease of the liver present in Egyptian splenomegaly. Intrahepatic thrombophilebitis and peripOLVEphilebitis associated with the pres-
ence of dead adult worms and live ova lead to cirrhosis. Dietary deficiency is a contributory factor. Portal hypertension causes splenomegaly.—W. J. M.

HUMAN CHIMERAS. TRANSIENT DOUBLE POPU-
LATION OF ERYTHROCYTES IN ONE OF A PAIR OF
NONIDENTICAL TWINS. R. Turpin, Ch. Salmon
et J. Crucéiller.

A PROPOS DES CHIMERES HUMAINES. PRESE-
NCE TEMPOURAIRE D'UNE DOUBLE POPULATION D'HÉM-
ATIES CHEZ L'UN DES JUMEAUX D'UN COUPLE
DIZIGOTE. From Chaire d'Hygiéne Clinique de
première Enfance, Hôpital Trousseau et Centre
départemental de Transfusion Sanguine, Paris.

The authors have observed the presence of red
cells of one of nonidentical twins in the circula-
tion of the other. On the one hundred twenty-
seventh day of life, these red cells constituted 5
per cent of the total red cell population of the
subject, but they disappeared after 6 months.

Reciprocal cutaneous grafts showed a tolerance of 16 days for the subject with the double red
cell population and 12 days for the other. These
facts seem to favor the hypothesis that the red
cells were eliminated through an immunologic
process. However, no isoantibodies could be found in the serum of the recipient against the red cell,
platelet and leukocyte antigens of the donor. In
man, it seems logical to assume that the passage
of blood cells from one twin to the other is not
necessarily followed by the graft of hemopoietic
tissue in the host, to give a chimera. The process
of grafting probably depends upon the time at
which fetal parabiosis begins, and upon the nature
of the transfused elements.—G. M.

USE OF FETAL HAEMOPOIETIC TISSUE TO PRE-
VENT LATE DEATHS IN RABBIT RADIATION
CHIMAERAS. K. A. Porter. From the Depart-
ment of Pathology, St. Mary's Hospital Medical

Male Chinchilla rabbits were exposed to 1600
r whole body irradiation and subsequently given
various types of homologous hemopoietic tissue
intravenously. The untreated control group all
died within 13 days of irradiation, but all treated
groups had a 3 week survival rate of 50 to 60
per cent. In rabbits receiving adult bone marrow
and neonatal liver, the survival rates at 8 weeks
had fallen to 25 per cent and 30 per cent, respec-
tively, while those receiving fetal liver showed a
45 to 55 per cent survival at this time. This
difference in late survival appeared to be due to the
elimination of "secondary disease" as a cause of
death in the group receiving fetal liver. Ani-
mals receiving 20 day fetal liver avoided "sec-
ondary disease" completely, but some receiving 27
day fetal liver developed early signs of this con-
dition with subsequent recovery. It is suggested
that the success of fetal as opposed to neonatal
or adult hemopoietic tissue in preventing late
deaths is due to the development of immunologic
tolerance to the host's antigens by the fetal cells
as they mature, with the consequent avoidance of
"secondary disease" due to a reaction of the
graft against the host. It appears from these ob-
servations that the use of early fetal hemopoietic
tissue might offer a greater chance of lasting
success in the treatment of marrow aplasia in
man than does adult bone marrow transplanta-
tion.—R. M. H.

TRANSFUSION HOMOGRAPHT OF BONE MARROW INTO
HUMANS EXPOSED TO ACCIDENTAL IRRADIATION.
G. Mathé, H. Jammet, B. Pendic, L. Schwarten-
berg, J. F. Duplan, B. Maupin, R. Latarjet, M.
J. Larrieu, D. Kalic and Z. Djukic. From
Hôpital Hérod et Hôpital Saint-Louis, Paris.

Six persons were accidentally exposed near an
atomic reactor to very high doses of neutrons
and gamma radiations (400 rem for the less
irradiated patient, between 700 and 1000 for the
others). The clinical and hematologic course con-
firmed the evaluation by physical methods of the
doses received. After a phase of initial shock, progressive blood pancytopenia occurred, with bone marrow aplasia. The 5 patients who received lethal doses were treated with intravenous administration of homologous marrow cells. A rapid clinical improvement followed and a concomitant correction of bone marrow and blood cytopenia occurred. Several facts suggest that the production of blood cells was assumed for about a month by the transfused bone marrow, the most convincing of these being the result of the study on red cell phenotypes produced respectively by the grafted marrow and the hemopoietic tissue of the graft (Ch. Salmon, Rev.Fr.Et.Clin.Biol. 4: 239–241, 1959).—G. M.


Although considerable work has been done on the pancytopenic sequelae following ionizing radiation exposure, most of the investigations are concerned mainly with condition of total body irradiation. During the course of an investigation on the effects of ionizing radiation on cells and tissues of regenerating amphibian forelimbs, changes were noted in the degree of leukocyte infiltration within comparable areas of control and x-rayed limb tissues. It was observed that in the x-rayed tissue areas the greatest majority of leukocytes was located along the periphery of the subepidermis (directly below the epithelium), and in pockets dispersed within the connective tissue. In the control areas, they were dispersed uniformly throughout all of the limb tissues. During the period of investigation, no apparent morphologic or histologic change could be noted in the vascular network supplying either the control or experimental areas. The major reduction observed in the leukocyte count in localized tissues exposed to ionizing radiation fails to support the contention that cell (leukocyte) infiltration does not normally occur and is affected by radio-illumination within localized areas.—O. P. J.


Hematologic values of 73 workers of the x-ray departments in 8 hospitals of Bombay are recorded. None of the workers was exposed to an annual dosage greater than 5 r. A significant finding was a variable degree of lymphocytosis observed in 34 persons; 15 persons showed bilobed or binucleated lymphocytes in their blood smear.—J. B. C.


Experiments with hepatectomized rabbits indicate that in vivo the bone marrow cells depend on the liver for their purine supply. It is known that in vitro they can utilize preformed adenine very efficiently. However, it is not known whether they depend entirely on preformed purines or whether they can complete a purine precursor provided to them by the liver. Therefore, experiments were designed to test whether bone marrow cells can utilize amino-imidazole carboxamide (AICA) or its riboside (one of the last steps in purine synthesis in the liver) for purine synthesis. The results indicate that AICA or its riboside are much less efficiently utilized than adenine for DNA and RNA purine synthesis in bone marrow cells in vitro.—O. P. J.


This paper describes a case of hemolytic anemia associated with a cold agglutinin and essential cryoglobulinemia. The patient did not respond to steroid therapy, but his condition was greatly improved by protection against the cold. Immunologic and electrophoretic studies showed that 2 abnormal proteins were present. One was a cold agglutinin immunologically identical with gamma globulin, and the other was a cryoglobulin which was unrelated immunologically to any normal serum protein.—G. C. de C.