ABSTRACTS

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ERYTHROCYTES


Electron microscopic studies of the effect of trauma on the sickling phenomenon were undertaken using SCA erythrocytes exposed to metabisulphite and then crushed in a tissue homogenizer. Sickled red cells were more susceptible to mechanical trauma than the control erythrocytes. The conversion of the Hgb S into parallel bundles of rods oriented in the long axis of cellular distortion has been previously described. When more than a mild degree of cell injury was incurred, a clear area was evident at the cell periphery, and instead of the highly organized bundles of parallel rods, a large mat of irregular fibers occupied the central cytoplasm. The filaments and rods were shredded at the periphery and appeared as amorphous debris below the cell surface. The most severely damaged cells looked like swollen ghosts. The author suggests that the substructural organization of the hemoglobin in sickled red cells, while associated with increased susceptibility to mechanical trauma, delays the transformation of the cells into ghosts. White reiterates his conclusion that sickling is basically a sol-gel transformation, in which protein polymers of Hgb S are formed, which undergo molecular stacking into filaments which then aggregate into parallel rods. During hemolysis the series of events appears to occur in reversed order.—J. B. S.


Administration of unconjugated bilirubin to pregnant rats during the last 5 days of pregnancy, caused the appearance of significant amounts of glucuronyl transferase activity in the liver of their offsprings at birth. A single day of treatment did not cause appearance of the enzyme at birth, but led to activation of the enzyme earlier than in normal controls.—J. B. S.

Blood, Vol. 34, No. 6 (December), 1969

811

A mild “waring blender” syndrome is described in an adolescent with congenital aortic coarctation. Following surgical correction, there was a return to normal red cell morphology, normal reticulocyte levels, and appearance of circulating haptoglobin.

—J. B. S.


Twelve of 232 erythroblastic infants demonstrated repeated blood glucose levels below 30 mg. per 100 ml. Eight of these infants had cord hemoglobin levels below 10 Gm. per cent, and hypoglycemia was seen in almost one fifth of the infants within this hemoglobin range. The hypoglycemia was usually first noted before the infant was 12 hours old, and it was asymptomatic. Plasma insulin levels varied inversely with blood glucose level, and there was also a negative correlation between cord hemoglobin and plasma insulin levels. Plasma free fatty acid (FFA) concentrations were decreased in infants with high insulin levels.

—J. B. S.


A toddler with severe plumbism presented with a severe, mostly normochromic anemia, without evidence of erythrocyte stippling. Reticulocytes were below 1 per cent, and bone marrow revealed erythroid hypoplasia. Treatment with Ca EDTA was undertaken, and several transfusions were necessary during the first month. After a second 5-day course of Versene treatment, 5 weeks after admission, mild reticulocytosis appeared. During the 8th week the bone marrow revealed normoblastic hyperplasia, and stippled red cells were apparently seen in significant numbers for the first time. Normal hemoglobin levels were observed shortly thereafter.—J. B. S.


Among 150 children three to 18 years of age enuresis was reported in 36 per cent of patients with HGB SS, 14 per cent of control children and 8 per cent with sickle trait. Among 45 adults nocturia was present in two of every three with sickle cell anemia, one-fourth of individuals with sickle trait, and one-third of the controls. Polydipsia was reported present among enuretic and non-enuretic youngsters to an equal degree. Males predominated among the sicklemic children with enuresis (16:8), but females predominated among the nocturic adults (15:4). Although no data are presented, the authors state that nocturia becomes progressively more prevalent as age increases.—J. B. S.


Two children in whom hemolytic anemia was seen as a complication of IM, are described. In both, the hemolytic anemia was the cause for the presenting complaint. In both patients the hemoglobin fell below 7 Gm. per cent, while the reticulocyte count exceeded 16 per cent. Neither youngster demonstrated a positive antglobulin test, although in one child, crossmatch problems were encountered. This youngster also had petechiae and a platelet count of 3000/cu.mm. on admission. Without therapy, the hemolytic anemia remitted in each child within two weeks.—J. B. S.


Two brothers with triphalangeal thumbs and radial hypoplasia, demonstrated a se-
ABSTRACTS

were normochromic normocytic anemia at, or shortly after birth. Karyotyping was normal and the only other laboratory abnormality was an increased fetal hemoglobin concentration. The anemia seemed to remit somewhat with increasing age so that transfusion need was minimal. In addition, one patient may have had a response to steroid therapy.—J. B. S.


A new hemoglobin variant is described. The variant was found in six members of one family; it does not seem to cause anemia nor does it interfere with β thalassemia, present concomitantly in two members of the pedigree. The hemoglobin was discovered in a young girl suffering from chronic, nonhemolytic anemia, which could not be associated with the hemoglobin variant.—H.-J. H.


Case reports, biochemical, microspectrophotometric and ultrastructural findings of two patients with unstable hemoglobin disease are presented. Characteristics of the inclusion bodies brought about by the intrinsic instability of the hemoglobin molecule are compared with those of the inclusion bodies resulting from the extrinsic effect of exposure to phenylhydrazine. In the splenectomized patient very high percentage of Heinz-body containing reticulocytes and erythrocytes could be detected even in the Romanovsky stained blood films. In the blood smears treated with the acid elution technique much less inclusion bodies could be detected in the fetal cells. The big inclusion bodies could not be eluted from the adult red cell ghosts due to the firm adherence of the denatured unstable hemoglobin to red cell membrane. Non-electrolyte permeability was decreased, the methemoglobin level and the rate of methemoglobin formation were elevated in both patients. The unstable hemoglobin fraction could be separated from Hb A by heat precipitation in both patients. The unstable Hb fraction of Case 2 could also be detected by starch gel electrophoresis as an abnormal fraction just preceding Hb A2. The results of the microspectrophotometric analyses proved that inclusions contain only a hemoglobin-like substance and no nucleotides or nucleic acids. According to our ultramicroscopic results the most conspicuous ultrastructural changes in unstable hemoglobin disease are: 1. The presence of inclusion bodies in the majority of the erythroblasts and reticulocytes; 2. the close association of inclusion bodies with clusters of iron-laden mitochondria; 3. Heinz-body formation within the nuclei of erythroblasts. The intranuclear inclusion bodies have essentially the same ultrastructure as the cytoplasmic precipitation of denatured hemoglobin compounds and 4. demarcation and autolysis of inclusion body containing cytoplasmic regions. Our ultrastructural findings suggest that inclusion body containing nuclei and digested Heinz-body containing cytoplasmic particles are extruded in the same way. It is assumed that the abnormal site and environment of hemoglobin breakdown are responsible for the abnormal metabolic pathway resulting in the appearance of pigmented dipyrrols which are excreted in the urine.—S. R. H.

CHANGES IN ENZYME ACTIVITY DURING RETICULOCYTE MATURATION AND RED CELL AGING. Ch. Bishop and C. Van Gastel. From School of Medicine, State University of New York at Buffalo, N.Y. Haematologia 3:29-41, 1969.

Activities of glucose-6-phosphate dehydrogenase (G-6-PD), 6-phosphogluconate dehydrogenase (6-PGD), hexokinase (HK), isocitrate dehydrogenase (ICD) and glutamic-oxalacetic transaminase (GOT) were studied in rabbit and human red cells, separated according to density equilibrium by ultracentrifugation on bovine albumin.
gradients. The rabbits were studied before, during and after bleeding, and during arrest of erythropoiesis induced by actinomycin-D. Human cells from normal individuals were also studied, both by this technic and by ultracentrifugation with dense solutions of either albumin or phthalate esters to separate only the most dense (oldest) red cells. The observed changes in enzyme activity in relation to increasing density can be most easily explained by dividing the process of red cell aging into two phases, reticulocyte maturation and true erythrocyte aging or senescence. Thus, it appeared that G-6-PD activity falls markedly with reticulocyte maturation and decreases also, but to a lesser extent, during subsequent cell aging. ICD, GOT and probably also HK decrease mainly during reticulocyte maturation and remain nearly constant during subsequent cell aging. 6-PGD activity is uninfluenced by either phase of the life period of the red cell.—S. R. H.


The galactose exchange transport in erythrocytes of 13 patients suffering from autoimmune hemolytic disease and of 9 patients with paroxysmal nocturnal hemoglobinuria was investigated. As compared to healthy persons, patients with autoimmune hemolytic anemia showed increased “apparent steady state” erythrocyte galactose values. The increase was statistically significant in 9 patients in a hyperhemolytic state and in 4 patients in a state of remission, showing, however, positive Coombs test. In erythrocytes of patients with paroxysmal nocturnal hemoglobinuria the galactose exchange transport was also increased, especially in patients with hyperhemolysis, and slightly increased in two patients in a state of remission. Increased erythrocyte sugar exchange transport in both hemolytic diseases has been discussed and assumed to be due to an increase in the percentage of young erythrocytes or to a functional alteration in the membrane of these erythrocytes. —S. R. H.

IRON STORAGE. VI. MOBILIZATION OF IRON BY BLEEDING. A. Shoden and Ph. Stur-geon. From the Institute of Biological Research and U.C.L.A. School of Medicine, Department of Pediatrics, Los Angeles, California. Haematologia 2:267-278, 1968.

Both hemosiderin and ferritin deposits in the rabbit liver are available for blood formation. Kupffer cell hemosiderin iron tends to diminish spontaneously with redistribution to ferritin and to parenchymal cell hemosiderin. Under the added influence of bleeding, this rate of disappearance is increased. Parenchymal cell hemosiderin granules also decrease with bleeding, but a few remain as large portal aggregates; under the experimental conditions, they cannot be mobilized. In the heavily laden animal parenchymal cell hemosiderin is more available but, at lower levels, ferritin stores diminish more rapidly. This relationship corresponds to that observed during iron loading; at low levels ferritin deposits increase but at higher levels hemosiderin functions as the reservoir. The ultimate iron deposit drawn upon by blood loss, therefore, is related to the degree of iron load at the time.—S. R. H.


Previously reported studies (Ann. Int. Med. 67:1201, 1967) have described the presence of ileal malabsorption, reversible following treatment with vitamin B12, in persons with pernicious anemia. Three additional subjects are presented who had malabsorption of vitamin B12 both alone and with added intrinsic factor, in whom the presence of serum autoantibodies to intrinsic factor or parietal cells or both indicated the presence of pernicious anemia. Absorption of vitamin B12 in the presence of intrinsic factor was restored to normal in two by therapy with cyanocobalamin.—F. A. K.

Therapeutic studies in women with pregnancy-induced megaloblastic anemia due to folate deficiency, showed that a daily dosage of 50 μg. of folic acid was effective in inducing a hematologic remission during the postpartum period; 500 μg. was necessary to induce a comparable hematologic response in the presence of a fetus and placenta; and 1 mg was required when pregnancy requirements were augmented further by twin fetuses or chronic hemolysis.—F. A. K.


The absorption of synthetically prepared isotopically labeled folate polyglutamate was compared with that of free pteroylglutamic acid in 5 subjects. Pteroylpolyglutamates were prepared by the solid phase method permitting placement of carbon-14 labels in selected glutamate units of the gamma peptide chain. The results indicated that ingested folate polyglutamates are cleaved to the monoglutamate form in the process of absorption. Net retention of the ingested folate polyglutamate ranged from 37-67 per cent. Fecal losses tended to be greater with increasing length of the poly-y-glutamyl chain.—F. A. K.


In a previous report from Puerto Rico (Blood 18:623, 1961), treatment with physiologic doses of crystalline folic acid of 25 μg. or more produced a hematologic response in 7 of 26 patients with untreated tropical sprue. In the present study, treatment with 100 μg. daily resulted in a hematologic response in 3 patients who were maintained on a low folate diet while no hematologic response was observed in 19 other subjects who received doses ranging from 25 to 220 μg. for 10 days while on a regular diet. Abstractor’s comment: As the authors point out, the hematologic response to physiologic doses of folic acid in this condition is probably related to the severity of the absorptive defect, the presence of concomitant vitamin B12 deficiency and the duration of treatment. Eleven of 16 patients treated in Malaya (Brit. Med. J. 2:1573, 1963) responded to a daily parenteral dose of 200 μg. daily but the response in some was delayed for up to 3 weeks and deficiency of vitamin B12 was infrequent. In contrast, 17 of the 22 Puerto Ricans in the present series were B12 deficient.—F. A. K.


Pyrimethamine appeared to act similarly to the folate antagonist methotrexate in short term in vitro culture studies of megaloblastic bone marrow obtained from a subject receiving pyrimethamine. Both drugs produced defective conversion of deoxyuridi-
dine to thymidylate, a defect that was corrected poorly by oxidized and well by reduced folate. Increased deoxyuridine did not correct the defect induced by either of these two drugs but did correct that produced by 5-flourouracil. These observations indicate that pyrimethamine acts biochemically in man as a folate antagonist.—F. A. K.

Studies on Folate and Vitamin B₁₂ Metabolism in Primates. I. Blood and Bone Marrow Morphology, Folate and Vitamin B₁₂ Levels. H. J. Huser and M. E. J. Beard. From the Yerkes Regional Primate Research Center, Emory University, Atlanta, Ga. and the Blood Research Laboratory, New England Medical Center Hospitals, Boston, Mass. Folia Primat. (Basel) 10:172-180, 1969.

Comparative studies on morphology, folate content of serum and erythrocytes and serum vitamin B₁₂ concentrations revealed significant, species specific characteristics, suggesting differences in the metabolism of these vitamins. The biochemical parameters studied indicate that of the species examined, the rhesus monkey most closely resembles man and may, therefore, be the most suitable species to use for experimental studies in this field.—H.-J. H.


Starting with death probability, and the relative number of cells lost at a certain age, and the probability to reach this age could be calculated. Age-dependent and age-independent cell death mechanisms were simulated. Some data were compatible with both mechanisms, but “the life span frequency function was quite well determined with respect to the mean cell life span.”—P. G. R.


Alpha-amino-nitrogen of free and bound amino acids was weekly estimated for 28 days in plasma and in erythrocytes of 7 different samples of the blood diluted with a preservative solution. A continuous increment of alpha-amino-nitrogen of the free plasma amino acids (average 1.96 mg. per cent) during 28 days was found. The alpha-amino-nitrogen of free amino acids in erythrocytes was rising proportionally to the time and during 28 days the average increment was 0.90 mg. per cent. The concentration of alpha-amino-nitrogen of the bound plasma amino acids as well as of the erythrocytes was stable during the preservation time and was found to be on an average 0.74 mg. per cent in the plasma and 1.52 mg. per cent in the erythrocytes. The sources of origin of new amino acids in the preserved blood and the facts indicating that new proteins or polypeptides were formed during the preservation time and their possible role in producing post-transfusional reactions were discussed.—S. R. H.


The author has studied 340 patients with metastatic cancers of the bone marrow. Approximately in half of the patients, white and red blood cells remained essentially normal. Hemoglobin content was below 7 Gm. per cent in 13.2 per cent of the patients, and erythrocyte counts were below 3,000,000 per cu. mm. of blood in 21.5 per cent. With exception of one case, anemia was hypochromic or normochromic in all cases. Normoblasts were found in the peripheral blood of 14.4 per cent of patients. In most of the patients, the total number of leukocytes was found to be within normal limits. A leukocytosis of over 10,000 was observed in 12 per cent of the patients, and leukocyte numbers less than 4,000 per cu. mm. of blood in 4.7 per cent. On rare occasions, a sharply marked leukopenia in combination with anemia and thrombocytopenia (panhemocytopenia) was associated with a diffuse lesion of the almost entire
active bone marrow, with the disease taking on a severe course and rapidly resulting in the death of the patient. Most of the patients showed lymphopenia, neutrophilia with a shift to the left. In 3.8 per cent of the patients, the shift to the left was due to metamyelocytes, in two patients (0.6 per cent) to myelocytes, and in one female patient, to myeloblasts.—S. R. H.


In a study of 34 patients with hypoplastic anemia including radio-chromium red cell survival tests, the authors found a considerable increase in red cell leakage or destruction stimulating regeneration with an increased proportion of macrocytes in the peripheral blood. Further, the blood of patients with chronic hypoplastic anemia contains a factor reducing the electrical charge of both patient and donor red cells. It is suggested that this might lead to accelerated elimination of red cells from the circulation and ultimately to exhaustion of the compensatory capacity of the bone marrow.—J. V.

Leukocytes


Suspension cultures of established cell lines originated from peripheral leukemic blood, stopped growing when the populations became crowded. The arrest of division was not due to depletion of nutrients in the culture medium since medium taken from inhibited cultures was able to stimulate growth of cells in small inocula. This indicated that cells might secrete growth-stimulating substances. In order to check this possibility cells were incubated with serum, with synthetic medium and with both, at a density where division did not take place. Twenty-four hours later the cells were centrifuged, the supernatants were passed through millipore filters 1:1 with fresh medium and this was used to support cell growth in different inocula. Cells grown in the supernatants that had been in contact with cells for 24 hours, supported growth less than the control medium.—G. M.


Earlier studies showed presence of non-DNA-synthesizing, non-dividing but RNA- and protein-accumulating peripheral cells in lymphoblastic leukemia. Present studies showed that 90 per cent mononuclear cells had 2 c. DNA, "a minor proportion" (less than 10%) had 4 c. Variation coefficient for dry mass and total nucleotides was 2 to 4 times normal in mononucleosis cells. Authors suggest small dividing and large non-dividing population of peripheral mononuclear cells, as in leukemia. Similarly, the first population is suggested to be RNA-synthesizing and protein-accumulating McKinlay-cell. Later abstract in Fed. Proc. (28:617, 1969) by same authors suggests increased acridine orange binding as sign of weakened DNA-protein bond, a prerequisite for RNA synthesis.—P. G. R.


A child with ALL in relapse, while undergoing therapy for recurrent meningeal leukemia, demonstrated group D streptococci in his CSF. At the time the bacteria were cultured his temperature was ranging between 103° and 104° F. CSF smear at that time showed a predominance of blasts, with only an occasional poly. The next day, however, CSF differential showed 73 per cent neutrophils. Therapy with intrathecal Mtx was continued, and systemic ampicillin and kanamycin were administered with clearing of the meningitis.—J. B. S.

Serum and urine samples of 82 patients with multiple myeloma and macroglobulinemia were tested with different methods for the identification of BJP. The classic heat test was found to give erratic and contradictory results and should be replaced by more accurate methods. Zone electrophoresis of the urine was found to be an adequate screening test. Immunoelectrophoresis with an anti-light chain antiserum which was absorbed with normal human serum as well as with type K or type L of BJP allows for differentiation between "microglobulins" and BJP on the basis of their different origins: polyclonal "microglobulins" react with anti-K as well as with anti-L serum; monoclonal BJP react with only one of the test sera. The methods described should prove to be useful in future investigations on disorders of immunoglobulins.—H.-J. H.


Formation of clover-leaf like nuclei converging in the centrosomal region is enhanced by oxalate more than by EDTA or heparin.—P. G. R.


Platelet-sized cytoplasmic fragments in needle biopsies from lymph nodes, spleen, etc., are described. They permit distinction of malignant lymphoma from other tumors and may be regarded as analogs to other corpuscles. They are seen in sections and smears and are not considered to be artefacts.—P. G. R.


A rare, little-known diffuse variation of the eosinophilic osteogranuloma is described. It belongs to the group of blastomatic hyperplasias of the histiocyte-system. The characteristics of the case which distinguish it from the classical eosinophilic granuloma occurring in childhood are: the diffuse development of the process and the advanced age of the patient. The case studied can be differentiated from myelomatosis, for which it was mistaken at first, by the lack of the pathological changes in serum proteins characteristic of paraproteinemic reticulositis and by some cytological peculiarities, e.g. intense eosinophilia and "birdeye"-shaped azurophilic inclusion bodies which never occur in the plasmacytoid reticular cells in myelomatosis. The good effect of the combined X-ray and hormone (methylandrostendiol) therapy in eosinophilic osteogranuloma is worth mentioning (treatment with Sarcolysin was ineffective).—S. R. H.

HEMOSTASIS


Data on 13 cases of primary thrombocytopenia were reported. The patients included in the study had to fulfill the following criteria: platelet count above 1 million per mm.³, absence of any other condition known to induce high platelet counts and no tendency to spontaneous remission. In this group, thromboembolic complications were more frequent than hemorrhagic symptoms. In 8 patients, cytogenetic studies were performed. No Ph¹ chromosome was found. On the other hand, 6 of the 8 patients were reported to show an elongated short arm of the G-21 chromosome. This finding would be of great interest, since none of the previous reports has indicated such consistent anomaly. Unfortunately, the information on the chromosomal studies and the illustration presented in this paper do not allow for final conclusions and are in want of further confirmation.—H.-J. H.
The Possible Cause of the So-called "Heparin Rebound" Phenomenon. M. Boros. From First Surgical Clinic, University Medical School, Szeged, Hungary. Haematologia 2:331-335, 1968.

Natural fibrinolysis prolonged the thrombin time after two hours of incubation if the incubation mixture contained heparin-protamine sulfate complex. The above-mentioned prolongation of thrombin time was diminished by anti-fibrinolytics (EACA and Trasylol). A significant heparin rebound phenomenon was caused by enhanced fibrinolysis. The action of the released heparin caused an extra prolongation of the thrombin time. The breakdown products of protamine sulfate formed as a result of fibrinolysis had no role in the prolongation of the thrombin time. The complex of heparin-hexadimethrine bromide was found to be resistant to the fibrinolytic action. The in vitro heparin rebound is caused by plasmin alone and involves no other plasmatic components. It can be stated accordingly that the mechanism of heparin rebound known in clinical practice is similar after the neutralization of heparin with protamine sulfate.—J. V.

IMMUNOHEMATOLOGY


The present study confirms and extends the previous report of Brain and Grace on the hemagglutinating properties of saline extracts of the snail Achatina granulata. The snail hemagglutinin detects a blood factor designated Ac, shared by the red cells of all human beings, and also all mice tested to date, but absent from the red cells of non-human primates and from rats and rabbits. The saliva of some or all animals of every species tested to date has the capacity to neutralize this hemagglutinin to a greater or lesser degree, as demonstrated by the hemagglutination inhibition technique. In man, the saliva inhibition titers have a bimodal distribution, with the majority of saliva specimens giving little or no inhibition and the remainder giving inhibition only in relatively low titers. In the non-human primates, also, varying inhibition titers were obtained, but low inhibition titers were exceptional and many of the maximal inhibition titers obtained with human saliva. Though the presence of the Ac substance in saliva as well as on red cells suggests a possible relationship to the A-B-H substances, parallel inhibition titrations showed no relationship between the inhibition titers of saliva for the snail extract hemagglutinin anti-Ac and the inhibition titers for agglutinins of specificities A,B,H or Z.—S. R. H.


Using organs from freshly killed experimental animals, homogenates were prepared and various subcellular fractions isolated by ultracentrifugation; these were used in blood coagulation studies. In the presence of microsomal and, to a lesser extent, mitochondrial and nuclear fractions of kidney and liver cells, coagulation systems showed a shortening of r and K intervals in the thromboelastogram and increased activity of thromboplastin and factors II, V and VII. Fibrin stabilizing factor activity was elevated in fractions from liver cells and kidney cells, and was higher in fractions from cortical than in those from medullary kidney cells. Fibrinolysis was delayed when mitochondrial fractions of liver cells were introduced into donor plasma or fibrinogen solution with plasminogen; in these solutions kidney medullary cell fractions increased the fibrinolytic activity.—J. V.


Immunologic studies in 29 patients with chronic myeloid leukemia revealed anti-
ERYTHROCYTE ANTIBODIES IN THREE, ANTI-LEUKOCYTE ANTIBODIES IN FOURTEEN, ANTI-PLATELET ANTIBODIES IN TWO AND ANTI-TISSUE CELL ANTIBODIES IN TWO. THE AUTHORS CONSIDER THAT IN CHRONIC MYELOID LEUKEMIA, PARTICULARLY IN PERIODS OF EXACERBATION, THERE MAY DEVELOP PHENOMENA OF "AUTO-AGGRESSION" AGAINST CELLS OF THE BLOOD AND TISSUES.—J. V.

MISCELLANEOUS


Transplants of autologous bone marrow, stored at −196° C for up to four months, were given to patients with inoperable lung cancer undergoing chemo-therapy with cyclophosphane. Given during the period of marrow depression (low leukocyte count and absence of any trend to recovery) marrow transplants were followed, in all cases, by hematologic improvement the peripheral blood and marrow appearing normal at 3-15 days. Transplants of allogeneic marrow, stored at −196° C for up to 3 years, were given to 30 patients with hypoplastic and aplastic anemia, some of them being in very poor condition with anoxia and hemorrhagic manifestations. Of 21 hypoplastic patients 11 experienced clinical and hematologic remissions of 7-30 months with almost normal blood pictures; some of the remainder showed some improvement while others were unchanged. The favorable reactions appeared by the end of the fourth week and continued for 2-4 months, one case cited in detail having been in remission for 7 months. Comments are also made on the differences between patient responses to fresh and preserved marrow transplants.—J. V.


From the cadaver 5-7 vertebral bodies (usually T10-L4) are separated, placed in a plastic bag with saline and antibiotic solution, and then subjected to a pressure of 25 Kg./sq.cm. in a press; the expressed bone marrow is filtered off. Marrow so obtained at up to 10 hours, at 10-20 hours and at more than 20 hours after death were studied. With the lapse of time after death the proportion of damaged nucleated cells rises but even so was less than that seen in marrow freshly aspirated from the sternum. It is recommended that marrow for transplantation be obtained by this method within 15 hours after death, the cadaver having been stored at 4° C; such a marrow specimen should have no more than 19.53 per cent damaged nucleated cells.—J. V.

PANMYELOPHTHISIS OF LESS KNOWN ETIOLOGY. R. Stieglitz and H. Stobbe. From Department of Hematology, First Clinic of Medicine, Humboldt University, Berlin, Germany. Haematologia 3:59-74, 1969.

The increasing incidence of panmyelophthisis makes it urgent to determine its causes. It may be assumed that in many cases the cause of panmyelophthisis is not discovered because no adequate efforts are made to find it or the potential role of the pathogenic factor is not well known by the physicians. A few observations are described showing how less known noxae, physical (thorium), toxic and/or allergic (contact with insecticides) and viral (infectious hepatitis) may be responsible for the affection of the bone marrow. The relationship between miliary tuberculosis and panmyelophthisis is also stressed. It is to be emphasized that the HCH insecticides in particular are not uncommon causes of the disease, as it is illustrated by the cases described in the literature. The results obtained concerning the nature of the changes and the role of special noxae in the pathogenesis of panmyelophthisis are dealt with. —S. R. H.
ABSTRACTS