ABSTRACTS

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BONE MARROW


Author surveyed the literature for data on growth of pre- and post-natal body and organ weight and volume and number of cells. Determinations of DNA phosphorus of fetuses were done. Growth data could be fitted best to a straight line on log-log paper. This method of graphic analysis may be applied to growth of hemopoietic tissues as well as to total growth of fetus and child. Consistent changes in growth rate between the ages of 7 and 8 years were noted. Interesting findings were the constant values for lymphocytes during most of childhood; the approximately equal numbers of non-nucleated vs. nucleated cells in the fetus and the constancy of the total hemoglobin in postnatal life at 1 per cent of body weight. It is suggested that this method should be used for growth analysis of normal children as well as of tumors and other biologic systems. Extensive graphs and charts are given.—A. G. M.


The author describes a familial and probably hereditary form of agranulocytosis occurring in a small geographic isolate in the northernmost province of Sweden. Fourteen cases (boys and girls) belonging to nine families are described in more or less detail. The onset of the disease occurred in early infancy with fever and skin infections. A complete or almost complete lack of granulocytes in the peripheral blood could be demonstrated. The bone marrow showed maturation arrest in the myeloid series. Without treatment, the disease ran a short course with lethal outcome. If the infections were treated with antibiotics, the affected infants might survive for several months or even years. Most of the affected infants could be referred to a common pedigree by genealogic methods. Close consanguinity between the parents was established in five of the nine families. Genetic analysis supports the conclusion that the disease is caused by a single recessive autosomal gene difference (a simple recessive mutation).—M. S.
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On the basis of some unpublished personal cases the authors attempted to isolate two new syndromes: (1) Congenital thrombopenic purpura with megakaryocytopenia; (2) Congenital neutropenia with myelocytopenia. By comparing these new syndromes with Blackfan-Diamond congenital hypoplastic anemia they were led to identify 3 types of congenital hypoplastic cytopenia affecting selectively each of the three blood lineages. They discuss on the nosology of such syndromes.—J. D.

EXPERIMENTAL APLASTIC ANEMIA. Naioe Kumagai, From the Department of Internal Medicine, School of Medicine, Kyoto University, Kyoto, Japan. Symposium on Hematology Japan, 7: 285-311, 1954.

Maekawa and his coworkers have reported many works on experimental allergy which was produced selectively in some organ, such as heart, liver, peritoneum, pleura, stomach and white and gray substances of brain by the method of sensitizing animals with a combined antigen, composed of cell phosphatide of the organ and foreign protein. In this paper similar studies on hematopoietic organs were presented. A combined antigen of rabbit’s red cell phosphatide, especially cephalin, which was extracted by their own method, using some organic solvents, and cow serum was injected into rabbits intravenously for 4 days. Three weeks later, the same antigen was reinjected, a distinct anemia developed in all cases after 3 to 5 days, and it continued to 9th-13th day. The anemia was hypoplastic. There were no signs of hemolysis. The anemia occurred alone without any disturbances of leucopoietic and other related systems. The bone marrow was also hypoplastic at the critical time, showing maturation arrest of basophilic erythroblasts. This anemia was also observed in rabbits previously splenec tomized. The author thought that the erythron of animal was damaged selectively by using antigen containing a specific red-cell cephalin, and he called this condition “Erythron-Allergy”. In the same way he also produced Leucok-Allergy with phosphatide from fresh pus, Myelon-Allergy with that of calf bone marrow, Lymphon-Allergy with that of peritoneal lymphnodes of calf or human tonsil, and if the phosphatide from cow spleen was used, a combined allergy of Erythron, Leucon and Myelon was produced. These allergic disturbances were, however, transitory and they suggested that some additional factors which suppress the regeneration process of hematopoiesis may be necessary to cause idiopathic aplastic anemia in man.—K. M.

STUDIES WITH ANTI-BONE MARROW SERA. S. Kumar. From the Department of Pathology and Bacteriology, Lucknow University, Lucknow, India. Indian J. M. Res. 44: 71-90, 1956.

The effect of antibone-marrow sera on the blood and bone marrow was studied in rabbits, guinea-pigs and swine. In rabbits a prompt and profound pancytopenia occurred after the injection of swine and guinea-pig antisera. Continuous intravenous administration of antibone-marrow serum in rabbits produced profound hemolytic anemia with erythroid hyperplasia in bone marrow.—J. B. C.


Eighteen cases of myelophthesis anemia secondary to metastatic cancer of the breast were examined. Besides the usual finding of leukoerythroblastic anemia, tumor cells in the bone marrow were found in eight out of fourteen patients examined. One case with clinical evidence of increased hemolysis was observed. Myeloid metaplasia was invariably present at autopsy. However, splenic enlargement in several patients was caused by metastases rather than by myeloid metaplasia. Myelophthesis anemia in cancer of the breast is an ominous prognostic sign indicating limited life expectancy. Temporary remissions were
Tuberculous Miliary Necrosis with Pancytopenia. W. E. Medd and F. G. J. Hayhoe.
From St. Thomas’s Hospital, London and the Department of Medicine, University of Cambridge. Quart. J. Med. 24: 351-363, 1955.

The hematologic complications of tuberculosis fall loosely into three groups: leukemoid reactions, myelosclerosis with leuko-erythroblastic anemia and peripheral pancytopenia with normally cellular or hypoplastic marrow but no marrow fibrosis. The groups are not sharply differentiated, intermediate and transitional forms occurring. An account is given of four cases of peripheral pancytopenia associated with concealed disseminated tuberculosis. In each case the histology was atypical in that the tuberculous lesions consisted of miliary necrotic foci with but little cellular reaction.

In the first patient, a male aged 49, there was fever and splenic enlargement. The marrow showed maturation arrest. The patient died soon after splenectomy: tubercles were found in most of the organs. In a male aged 50 the marrow was moderately cellular with maturation arrest. Post mortem there was caseous tuberculosis in retroperitoneal and mediastinal glands and in the liver. A man aged 49 had anemia with splenic enlargement, with an erythematous rash. The marrow showed normoblastic hyperplasia. An aspiration liver biopsy showed acute miliary tubercles; and following treatment with streptomycin, PAS and isoniazid the patient was able to return to work. A 56 year old female patient had transient purple, painless, subcutaneous nodules on the legs and then an upper respiratory infection. These improved, but the patient became weak, tired and anemic. The marrow was poorly cellular and a diagnosis of aplastic anemia was made. Later a sample of marrow gave a picture of acute leukemia with 65 per cent of primitive cells, probably myeloblasts. Treatment with cortisone did not prevent death. Post mortem there was microscopic evidence of tuberculosis in visceral and parietal pleura and in the cervical and tracheobronchial lymph nodes, but not elsewhere. There was no evidence of leukemia.

In difficult cases of this type, aspiration liver biopsy should be made, and it is possible that in cases of aplastic anemia of unknown origin, treatment with streptomycin and isoniazid should be used in case concealed disseminated tuberculosis is being missed.

The hematologic disorders due to tuberculosis may share a similar common pathogenetic mechanism, with the product of the tubercle bacillus acting on over-sensitive “allergic” hemopoietic tissues. —R. H. G.


Eighty cases of visceral Leishmaniasis were separated in two groups according the presence or not of an associated hepatosplenic schistosomiasis. The bone marrow pictures of the groups were different. Reticulum cells hyperplasia and eosinophilic depression were seen in the Kala-azar group in contrast to the schistosomiasis group. Bone marrow erythroid hyperplasia and plasmacytosis were present in Kala-azar. In this condition the granulocytic series exhibited inhibition of maturation at metamyelocyte and stab cell level, while in schistosomiasis the inhibition was usually at promyelocyte level, with eosinophilia. —M. A. J.


In bone marrow and splenic smears, plasma cells in some cases of kala-azar contained characteristic intracellular, mostly intracytoplasmic, globular bodies similar to those described in multiple myeloma. —J. B. C.
ABSTRACTS


This is a clinical, hematologic and histologic discussion of myelofibrosis as typified by study of 23 patients in whom the diagnosis was established by clinical investigation plus biopsy or autopsy verification. Splenectomy was performed in 2 cases in whom hemolysis was a complicating feature: the results were good.—S. E.


The effect of 2,4-diamino 5-P-Chlorphenyl-6-ethyl-pyrimidin (Daraprim) on the hematopoietic system is studied. Daraprim, used in the treatment of malaria, is also used in polycythemia vera. In animal experiments (15 rats) decrease of erythro- and granulopoiesis is observed. The effect on the thrombopoiesis is less striking. The effecting mechanism does not seem quite clear. A standstill of the maturing process, an inhibition of forming elements, and a hemolytic factor are regarded as causes of the anemia.—M. H. H.


During 8 weeks, over 12 Gm. of chlorpromazine HCl were given to a 59-year-old man with an anxiety state. He developed agranulocytosis with a white count of 800 (lymphocytes 100%). The marrow showed nine per cent myeloblasts, one per cent promyelocytes, and no further neutrophils. He recovered on cortisone and antibiotics.

A 61 year old woman developed leukopenia (white cells 1,200, polymorphs 34%) after 41.4 Gm. of chlorpromazine, and recovered on penicillin.—R. H. G.

RED CELLS AND RED CELL DISEASES


This is a discussion of the mathematical possibilities involved in the interpretation of results obtained from tagging the red cells of cases of familial hemolytic anemia isotopically as with glycine-N14. The birth rate and death rate of the cells have both to be considered, and this involves consideration of other evidence derived from the extent of hypertrophy of the bone marrow.—R. H. G.


Severe anemia is known to be accompanied by an increased output of blood from the heart, an increase in cardiac weight and external dimensions. Since piglets reared on concrete are very liable to have a severe hypochromic anemia at 3 to 5 weeks, this material offered an excellent opportunity to study the nature of the cardiac enlargement. Seven anemic and seven normal piglets were used for this work. In piglet anemia the cardiac output increases considerably and the heart grows proportionately faster than the body. This "hypertrophy" is thought to be due to an increase in the number of normal muscle cells because no change has been found in (a) the quantity of water, of protein or of mineral salts in unit weight of heart muscle; (b) the deoxyribonucleic acid-protein-water relationships within the heart.—O. P. J.

The possible etiologic and pathogenetic factors in anemia of chronic renal disease are discussed. Red cell survival was studied by differential agglutination and was found to be reduced. The reduced erythrocyte life span is thought to be due partly to intracorpuscular factors, partly to extracorpuscular hemolytic agents. Erythropoiesis may be increased (in milder cases) or somewhat depressed (in more severe cases).—M. S.


The authors point to the frequency of the occurrence of anemia in myxedema and to the possibility of a mistake with pernicious anemia. Out of 26 patients suffering from myxedema, there were 17 cases of anemia; 14 patients had macrocytic, normochromic anemia, 3 had normocytic, normochromic anemia. Seven patients with myxedema and anemia were under treatment for several years for pernicious anemia. Treatment with liver extracts had no effect. In all patients a therapeutic effect was registered after administration of thyreoidin.—M. N.


In a series of 96 cases of ankylostomiasis, 74 were anemic. The severity of anemia could not be correlated with the load of the parasite as judged by the egg count. In the majority of cases the red cells were normocytic or microcytic and hypochromic. Bone marrow was normoblastic in 54 and megaloblastic in 22. The 22 cases with megaloblastic marrow were considered to be cases of nutritional megaloblastic anemia complicated by hookworm infestation.—J. B. C.


Persistent reticulocytosis, hyperbilirubinemia and positive Coombs test in a young female, age 20, suffering from chronic Kala-azar with severe anemia indicated the presence of a hemolytic component developing on an immunologic basis. Specific anti-kala-azar therapy controlled the hemolysis.—J. B. C.


The study was based largely upon the examination of case records between 1948 and 1951 inclusive; only those patients with sufficient data for assessment were included. The survey covered three hospitals for natives and two for Europeans and was mainly of primary blood disorders. Altogether 412 patients had blood dyscrasias. The conditions found particularly in natives were sickle cell anemia, nutritional megaloblastic anemia, onyalai, hemorrhagic disease of the newborn and multiple myeloma. The conditions found particularly in Europeans were pernicious anemia, idiopathic hypochromic anemia, erythroblastosis fetalis, leukemia, Hodgkin's disease, infectious mononucleosis, and essential thrombocytopenia.—R. H. G.


Plasma amino acids were measured by paper chromatography in 50 cases of hypochromic anemia. Compared to normal mean value (1.91 mg. per 100 ml.), the glycine content was significantly elevated (2.55 mg.)—J. B. C.

The content and behavior of TPN, FAD, CoA and TPP in hemolysates of red blood corpuscles of varying age have been investigated. FAD and CoA are stable while TPN and TPP undergo slow cleavage. Hemolysates are able to synthesize TPN from NSA and ATP. Reticulocytes contain approximately twice as much FAD as erythrocytes.—M. H. H.


Earlier reports deal with an inhibitor of succinoxydase in reticulocytes. This inhibitor acts on the functional specializing process of reticulocyte maturation. The inhibitor cannot be demonstrated in mature erythrocytes. The mechanism of its inactivation is investigated. The factor which inactivates the reticuloocyte inhibitor can be demonstrated in bone marrow and stroma of red blood corpuscles. The effect is only observed under aerobic conditions. Attention is directed to the biological significance of the system (respiration-respiration inhibitor-inactivator of inhibitor) for erythrocyte maturation.—M. H. H.


The appearance and disappearance of the reticuloocyte inhibitor of succinoxydase in the course of the development and during the regeneration phase of anemia produced by bleeding or by injection of phenylhydrazine were investigated in rabbit experiments. The appearance and disappearance of the inhibitor is a further characteristic of the maturation of the blood corpuscles and is coordinated with other characteristics such as reticulocytosis and respiration.—M. H. H.

PIGMENT METABOLISM—HEME PIGMENTS


A positive correlation was demonstrated between the severity of the erythroblastosis fetalis and the amount of heme pigments present in the plasma. The latter were estimated by their strong absorption in the Soret band. The level of plasma heme pigments present is a more reliable guide for the management of erythroblastosis than the level of bilirubin. Particularly in the early period after delivery, heme pigments may already be high while the serum bilirubin is still relatively low.—R. S.


Methemalbumin may be present in small amounts in cord serum of healthy newborn infants. In hemolytic disease of the newborn the methemalbumin level may be markedly elevated, but may also be normal in cord serum. A long-standing and intense hemolytic process combined with hepatic dysfunction was found to be a predisposing factor to the accumulation of methemalbumin in the plasma. The concentration of this pigment in
patients with hemolytic disease of the newborn varies greatly and cannot be used as a guide to exchange transfusion therapy.—M. S.


The levels of methemoglobin varied from the mean of 0.11 Gm. per 100 ml. for children, adults and the cord blood of mature newborn infants to higher values of from 0.43 to 0.52 Gm. per 100 ml. the first 48 hours of postnatal life in the premature infant. During the period from one month to one year of age, in the premature, the concentration of methemoglobin decreased, but was still higher than that found in older children or adults. The degree of methemoglobinemia was insufficient to produce cyanosis. Various explanations are offered as to the cause of the methemoglobinemia.—W. N. J.


Two cases of familial, congenital methemoglobinemia are described in two sisters, 13 and 18 years old respectively. Cyanosis was present at birth and diagnosis of congenital heart disease was made in both cases. The most significant patterns were represented by the presence of 3.65 Gm. of methemoglobin in the blood (corresponding to 25.9 per cent of the total pigment) in one case, and 2.80 Gm. (19.55 per cent of the total pigment) in the second case. From the therapeutic point of view, the effectiveness of methylene blue (200 mg. i.v. or orally), and of vitamin C (1,000 mg. orally or parenterally) was observed. Side reactions occurred with methylene blue (headache, nausea, digestive troubles, etc.). Sixteen other families presenting the same syndrome were collected from the literature, including 101 cyanotic subjects, with slight predominance of the males.—P. d. N.


Almost immediately following electroshock therapy, a 31-year-old welder developed acute abdominal cramping pain, generalized muscle soreness and myoglobinuria. This was the first acute attack, although some degree of muscle soreness, reduced muscular strength and difficulty in relaxing his muscles after sustained contraction had been present for at least 10 years. Myoglobinuria or dark urine had not been observed before. The acute episode subsided spontaneously. While under observation muscle soreness and myoglobinuria could be precipitated by heavy physical exercise. Quinine and testosterone failed to prevent these symptoms. By carefully “calibrating” his physical activity the patient manages to get along well without medication. This case deserves particular interest because the presenting symptoms resembled more those of myotonia congenita than those of myoglobinuria.—R. S.

**Paroxysmal Myoglobinuria.** F. E. Schaar. From the Department of Pediatrics, University of Minnesota Medical School, Minneapolis, Minn. Am. J. Dis. of Child. 89: 23-30, 1955.

The author gives a review of the subject and summarizes 17 cases compiled from the literature. An additional case of severe but transient myoglobinuria in a 9 year old boy is reported in detail. The acute attack followed a slight upper respiratory infection and strenuous physical work. The urine gave a 4+ guaiac reaction, but contained no erythrocytes. Myoglobin was detected spectroscopically, but details of the absorption spectrum are not given. No evidence of increased hemolysis was found. The boy was in complete remission 20 days after the onset of his acute illness.—R. S.
ABSTRACTS


The author gives an excellent survey of the various forms of myoglobinuria. The following classification is suggested:


The author describes six of his own cases of myoglobinuria, three cases of familial paroxysmal myoglobinuria, two unclassifiable cases, and one case in a boy suffering from progressive muscular dystrophy. All patients were males.

Familial cases of paroxysmal myoglobinuria were for the first time reported by the author in 1947 in three brothers who all had repeated attacks from adolescence. The attacks were precipitated by muscular exertion, and could also be provoked by a low-carbohydrate diet. Pathologic glucose tolerance curves and pathologic creatine metabolism were found. These patients are feeling quite well when they are eating a normal diet and not doing unusually hard muscular exercise. One of the two cases of unclassifiable myoglobinuria ran a rapidly fatal course, hyperpotassemia being the probable cause of death.

The following points are stressed: 1. Attacks of myoglobinuria often cause renal damage. 2. During an attack there is risk of hyperpotassemia, particularly when there is coincident renal impairment. 3. Some degree of liver damage is often present. 4. The attacks are apt to be misinterpreted as glomerulonephritis.—M. S.


A case of myoglobinuria with a fulminant course and death presumably of hyperpotassemia is described. At autopsy, marked degenerative lesions were found in the muscles and kidney tubules. Serum potassium level was high, and ECG showed signs of hyperpotassemia.—M. S.


A 5 year old boy who was taking a tonic, containing “sulfur and molasses”, developed nausea, vomiting, constipation and slight fever. He was treated with a triple sulfonamide preparation, consisting of sulfadiazone, sulfamerazine and sulfamethazine in a dosage of 0.5 Gm. every six hours. On the fourth day of treatment, the patient developed progressive cyanosis without evidence of dyspnea. On spectroscopic examination of his blood, sulfhemoglobin was discovered in the amount of 0.76 Gm. per 100 ml. The total hemoglobin concentration was 12.6 Gm. No significant increase in methemoglobin was detected. Upon withdrawal of all medications, the cyanosis gradually subsided over the next few weeks. Ascorbic acid and intravenous methylene blue had no effect on the cyanosis. It is postulated that the administration of the sulfur and molasses tonic favored the conversion of methemoglobin to sulfhemoglobin. This would explain the unusual finding of a high sulfhemoglobin concentration without a concomitant rise in methemoglobin.—R. S.


The case histories of five patients with sulphaemoglobinæmia are presented. In all but one, the blood disturbance was due to the excessive use of analgesic medications containing
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phenacetin. The diagnosis was established by demonstration in the laked blood of the typical absorption band at 615-630 nm. Treatment consisted in withholding the offending drug or replacing it with aspirin. Only in the most severe case were blood transfusions required because of anoxia.—R. S.

METHODS


A short method is described for determining the latent iron-binding capacity of serum. The method, which was first presented by Ventura (1952), involves measurement of the light transmission of a diluted serum sample before and after saturation of its iron-binding capacity, and requires the calibration of a photoelectric instrument. A simple procedure is described for this calibration.—G. C. de G.

VISOSITY MEASUREMENTS OF BLOOD AND OTHER FLUIDS WITH A FULLY AUTOMATIC RAPID-VISCOSMETER. A. Reis and J. Miller. From the Laboratory (Leiter Prof. Dr. Dr. K. Dirr) of II. Medizinischen Universitätsklinik (Dir. Prof. Dr. Dr. Bodechtel) München. Klin. Wehnschr. 34: 197-199, 1956.

A viscosimeter is described which allows fully automatic measuring of small amounts of fluid (1 drop). The exactness of the measurements, close to 2%, equals those of the Höppler precision viscosimeter. Substances which undergo changes (for instance blood by clotting or synthetic materials by polymerization) can be measured within 2 seconds.—M. H. H.


To 1 ml. of packed rabbit red cells are added 10 ml. of pooled human serum heated for 30 minutes at 56 C. The tubes are then left at room temperature for 30 minutes, being shaken several times. The agglutinated red cells are washed thrice with saline, suspended in 3 ml. of saline and injected into the ear vein. For immunization, 2 injections weekly are carried out for 6 weeks. By this method 4 antiglobulin sera were obtained and all were satisfactory.—R. H. G.


The authors report their methods for the determination of red cell volume using sodium chromate containing Cr⁶¹ and plasma volume using chromic chloride containing Cr⁶¹. The important part of this paper concerns the fact that when chromic chloride was added to saline solution there was considerable adsorption of chromium onto the glass surface. This could be overcome by the addition of nonradioactive chromium chloride to the distilled water before the addition of the radioactive chromium. No adsorption to glassware of sodium chromate was found.—T. R. T.


A brain extract was used as a substitute for platelet suspension in the thromboplastin-generation test. Rabbit brain was used as follows: 100 mg. of rabbit brain (powdered, acetone treated) were mixed with 5 ml. of chloroform for 15 minutes at room temperature and
filtered through paper, dried at 37 C. and resuspended in 5 ml. of saline. This material can be stored at −20 C. without loss of activity for several months, and is diluted 1 to 100 in saline for the use in the thromboplastin-generation test.—P. d. N.

**Modified Westergren Method for Determination of Sedimentation Rate of Erythrocytes.** 

A modified Westergren's method in which oxalated venous blood mixed with 3.8 per cent sodium citrate solution in the ratio of 1 to 4 was allowed to sediment in standard Westergren tube proved to be convenient and dependable.—J. B. C.

**Blood Sedimentation.** 

A method is described in which a pipet with blood and sodium citrate is fixed at an angle of 62 degrees from the horizontal and the rate of sedimentation is taken after 11 and 20 minutes. Sedimentation values correspond approximately to those obtained in one and two hours in tubes placed in a vertical position.—M. N.

**Evolutionary Serum-Plasma Time (ESP). Clinical Importance and Evaluation.** 

The serum-plasma time is described as a simple and quick test for clinical evaluation of bleeding conditions. The principle of this method is the mixing of serum with plasma 3, 4, 5 and more minutes after artificially made coagulation. In hemophilies, the behaviour of the ESP is specific. It is enormously prolonged and of a steeply descending character. In thrombocytopenia, the ESP is slightly prolonged and of a brief descending character. In the course of the treatment with Pelentan (Tromexan), the ESP is slightly prolonged, always of a rising tendency. After the return of Quick's time to normal values, the ESP remains prolonged in case of overdosage.—M. N.

**The Differential Leucocyte Count: Observations on the Error Due to Method of Spreading.** 

Observations are described indicating that with rapidly spread blood smears the leucocytes are distributed fairly uniformly throughout the smear. This is in contrast to the slowly spread smears in which the leucocytes become concentrated in the edges and tail. The extent to which this concentration takes place varies according to the type of leucocyte. It follows that a differential count performed upon a rapidly spread smear will be much closer to the true count than one performed upon a slowly spread smear.—G. C. de G.

**Isotope Technique for Mediastinal Tumors.** 

By means of 111I and 32P, the authors are able to divide mediastinal tumors into three groups: (1) thyroid; (2) fast growing tumors, such as the lymphomata; and (3) slow growing masses, such as cysts. Their method depends upon the known uptake of 111I by thyroid tissue, and the fact that 32P is metabolized by all growing structures in direct proportion to their rate of growth. According to their method, they first give a tracer dose of 50 μc of 111I, and then scan the thyroid and mediastinal areas by means of a contact Geiger-Müller counter. If the mediastinal mass does not show uptake of 111I, the authors then give 100 μc of P32 by mouth, and repeat the scanning process after 24 hours. Malignant lymphomata are rapidly growing neoplasms and usually pick up considerable P32; in this way, the authors are able to diagnose with considerable certainty the probable nature of a mediastinal tumor.—S. E.
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It was found that the disappearance rate of 131 serum albumin from the circulation of ten subjects with severe generalized edema was exponential after mixing up to at least one hour. The average mixing time in this group of patients was longer than in the group of patients without edema. It is concluded that samples drawn after thirty minutes and up to two hours should give a single exponential slope from which accurate calculations can be made.—T. R. T.

The Other Journals of Hematology


The abstracts:


