ABSTRACTS

JOSEPH F. ROSS, M.D., Editor

Abstracts

HELEN W. BELLINO, M.D., WINSTON SALEM, N.C.
SHERL T. CALLENDER, M.D., OXFORD, ENGLAND
GEORGE E. CARTWRIGHT, M.D., SALT LAKE CITY
ROGER C. CRAFTS, PH.D., BOSTON
CHARLES P. EMERSON, M.D., CINCINNATI
SOLOMON ESTREN, M.D., NEW YORK
OLIVER P. JONES, PH.D., BUFFALO

CONRAD MAIER, M.D., ZURICH, SWITZERLAND
MILOS NETOULEK, M.D., PRAGUE, CZECHOSLOVAKIA
JEAN P. SOUSSER, M.D., PARIS, FRANCE
RAMÓN M. SUÁREZ, M.D., SAN JUAN, PUERTO RICO
WM. N. VALENTINE, M.D., PACIFIC PALISADES, CAL.
PHILIP F. WAGLEY, M.D., BALTIMORE

ERYTHROCYTES AND ERYTHROCYTIC DISEASES


Twenty cases of tetralogy of Fallot were studied with the purpose of determining the rate of blood destruction and formation before and after operation. Prior to operation, both urobilin excretion and reticubocyte formation were above normal. Subsequent to operation, with diminution in anoxemia, the reticubocytes were diminished to a very low point or even disappeared, while the urobilin excretion was markedly increased over a period of about one week and then fell rapidly to normal levels. The findings are interpreted to indicate that both cessation of blood production and increased blood destruction were responsible for the reduction in red cells and hemoglobin after operation. During the week following operation, blood destruction was considered to be the major factor.

The similarity of events reported here and those occurring in the newborn period are pointed out. It is suggested by the author that blood destruction in these cases is the result of a physiologic mechanism which has as its "purpose" the reduction of red cells.—W. N. V.


The demonstration of erythrocytes in paraffin sections is made difficult by postmortem lysis as well as destruction of erythrocytes by certain fixatives. If fresh tissue is used, fixation in either formol-sublimate or Zenker-formol is suitable. On the other hand, if lysis has already begun, a preliminary fixation in a plain formalin mixture (four to six hours) followed by further fixation in saturated aqueous mercuric chloride will fix the erythrocytes with the least amount of additional lysis. The most selective staining of erythrocytes has been obtained with the Krag (kitron-red almond-green) method. The time of staining in kitron red is not acute, but the dye must be dissolved in cellosolve. Muscle, fibrin, the granules of Paneth cells and the Russell bodies of plasma cells all lose the red on washing with water, while erythrocytes and acidophilic granules of eosinophils retain the red.—O. P. J.


Human group O red cells, when treated with potassium periodate, develop a new antigenic specificity which is distinct from that occurring in cells treated with a Vibrio preparation. Periodate-treated cells are susceptible to a panagglutinin present in human serum; this panagglutinin is distinct from the panagglutinin in serum from Vibrio-treated cells. It appears not only that periodate treatment effects a specific change in the cell but also that the changes occurring in periodate-treated and in vibrio-treated cells have no common antigenic feature.—O. P. J.

Etude Clinique, Sérologique et Thérapeutique de la Maladie de Marchiafava-Micheli. (Clinical, Serologic and Therapeutic Study of a Case of Marchiafava-Micheli Disease.) J. BOISSER and P. 526

All well studied cases of Marchiafava-Micheli disease warrant reporting as they are of considerable interest with regard to the very special hemolytic mechanism in these patients. The case reported here deals with a woman 23 years of age and is of interest regarding the following points: she had a hemoglobinuria when lying down rather than a purely nocturnal hemoglobinuria; a persistent hemosiderinuria, a serum iron level of 180 micrograms per 100 cc., a disappearance of the serum haptoglobin, and the unusual finding of leukopenia in an hemolytic anemia. The osmotic resistance was normal, and both Donath-Landsteiner and Coombs tests were negative. The acid test of Ham was positive. Of particular interest was the testing of the resistance of erythrocytes to heat, as done by Hegglin and Maier, which showed an abnormal hemolysis (hemolysis at 37 C. after six hours and twenty-four hours). From tests in which cells and serum were mixed, this hemolysis was found to be due to an abnormality of the patient's red cells, since the serum was devoid of activity. A series of previous experiences seemed to show that complement plays an essential role in hemolysis (Ham).

Finally, treatment by transfusion of washed erythrocytes (method of Dacie) has maintained the patient and relieved the symptoms.—J.P.S.

RECENT ADVANCES IN TREATMENT OF HEMATOLOGIC DISORDERS. C. C. Sturgis. From the Department of Medicine, University of Michigan School of Medicine, Ann Arbor, Mich. J.A.M.A. 141: 969-973, 1949.

The purpose of this review article is to emphasize the treatment of the common hematologic disorders which are seen by the practitioner of medicine. The author discusses especially specific treatment of the macrocytic anemias (liver extract, folic acid, vitamin B12) and recent advances in the treatment of the leukemias (x-ray, urethane, anti-folic acid compounds, and nitrogen mustards).—S.E.

TONICITY-VOLUME RELATIONS IN PARTIALLY HEMOLYZED HYPOTONIC SYSTEMS. E. Ponder. From the Nassau Hospital, Mineola, N. Y. J. Gen. Physiol. 33: 277-293, 1950.

Since linear relations between red cell volume and the reciprocal of the tonicity is not the one expected on the basis of the van't Hoff-Mariotte law, attempts were made to explain this anomalous result. The form of the tonicity-volume relation can be changed by treating the cells with resorcinol, colloidal silicic acid, iodoacetate, or sodium oxalate. Temperature changes, pH, and allowing the cells to stand for twenty-four hours produce only minor changes in this relation.—O.P.J.

PIGMENT METABOLISM


The results of these experiments indicate glycine is, in dogs, a direct nitrogenous precursor for coproporphyrin I and stercobilin as well as hemoglobin protoporphyrin. It is postulated that coproporphyrin I is formed as a by-product during the biosynthesis of coproporphyrin III, which in turn gives rise to protoporphyrin IX. Coproporphyrin I is not a hemoglobin derivative, but protoporphyrin IX, excreted in feces of dogs, is a hemoglobin derivative. Stercobilin is derived in part from sources other than circulating hemoglobin breakdown. Increases in coproporphyrin III excretion, observed frequently in association with a disturbance of hemoglobin metabolism (as in metal intoxication and sulfonamide therapy), may be explained by a blockage in the transformation of coproporphyrin III to protoporphyrin IX or by a decrease in the utilization of protoporphyrin IX for hemoglobin metabolism.—P.F.W.

Isotopic Studies of Porphyrin and Hemoglobin Metabolism. II. The Biosynthesis of Coproporphyrin III in Experimental Lead Poisoning. M. Grinstein, H. M. Wikoff, R. Pimenta de Mello, and C. J. Watson. From the Department of Medicine, University of Minnesota Hospital, Minneapolis, Minn. J. Biol. Chem. 182: 713-726, 1950.

Data are presented to support the idea that coproporphyrin III excreted in excess in the urine of lead-poisoned rabbits is not related to hemoglobin catabolism. A new formation of coproporphyrin III seems to occur as a result of lead poisoning.—P.F.W.
ABSTRACTS


Observations are reported on the absorption spectra of a pigment observed in the hemolysate of washed erythrocytes obtained from rabbits fed a purified diet for a period on one to two years. This pigment did not appear to be heme, porphyrin, methemoglobin, choleglobin, or sulfhemoglobin.—P.F.W.


In the five species studied, the quantity of hemoglobin was relatively constant per kilo of body mass. The total content of cytochrome c, however, was shown to be proportional in four of the five species to the estimated body surface area. In all species studied, the concentration of cytochrome c in cardiac muscle was higher by a factor of 3 to 6 than in skeletal muscle.—P.F.W.

LEUKOCYTES AND LEUKOCYTIC DISEASES


The authors report the occurrence of agranulocytosis in a newborn infant on the third day of life. At this time, a blood count was done in an effort to explain the occurrence of sudden fever. The white cell count was 4,200, and neutrophils numbered 2 per cent. The child was acutely ill in the next three weeks; blood studies during this time continued to show white cell levels from 3,500 to 11,000, with granulocytes from 1 to 6 per cent. The red counts and hemoglobin values were normal. A bone marrow aspiration showed hypocellularity and increased numbers of promyelocytes, lymphocytes, and monocytes. Forceful treatment was undertaken with penicillin, streptomycin, whole blood, clyses, and the use of an oxygen tent and, after a stormy course, the infant began to improve rapidly from the age of 20 days on. The white count at this time was 12,000 with 19 per cent granulocytes; and on subsequent days it became and remained normal. At 3 months and 9 months of age, the child was apparently completely normal; a bone marrow examination at the age of 9 months was also normal.

The etiology of this instance of agranulocytosis is discussed, but no conclusions are drawn. Efforts to implicate drugs (in mother or child), maternal illnesses, sepsis, and some sort of allergic anaphylactic reaction, were unsuccessful, although it was felt that possibly sepsis might have been the underlying causative agent. The dramatic effect of anti-infection therapy is emphasized.—S.E.

AGRANULOCYTOSIS DURING THERAPY WITH THE ANTI-HISTAMINIC AGENT METHAPHENILENE (DIATRIN). T. G. Drake. From the Department of Internal Medicine, Washington University School of Medicine, and the Missouri Baptist Hospital, St. Louis, Mo. J.A.M.A. 142: 477-478, 1950.

An 81 year old man received 50 milligrams of Diatrin every eight hours for seven weeks, because of a diagnosis of chronic bronchitis and rhinitis. There was clinical relief of symptoms during this time. At the end of the seven-week period, however, there was a sudden acute febrile illness, and a blood count at this time showed 3,600 white cells, with no granulocytes on the smear. Therapy with aureomycin, penicillin, streptomycin, and blood transfusion resulted in ultimate return of the patient to normal health. Blood counts became normal after two weeks, and remained normal thereafter.

In the absence of other etiologic agents, the agranulocytosis was attributed to Diatrin therapy. It is noted that this is the fifth published report of agranulocytosis following antihistamine therapy, three having occurred following the use of Pyribenazine, and one the use of a synthetic compound in France. The author points out that in all five instances, the particular drug used contained the benzamine linkage, C6H5-N-, which is also found in Aminopyrine, and which is absent from other antihistamine drugs (notably, Benadryl).—S.E.

LYMPHOID TISSUE AND ITS RELATION TO SO-CALLED NORMAL LYMPHOID FOI AND TO LYMPHOMATOSIS. II. QUANTITATIVE ANALYSIS OF LYMPHOID AREAS IN THE PANCREAS OF LABORATORY AND FARM CHICKENS.
ABSTRACTS


In a previous article (Am. J. Path. 25: 1197, 1949) it was shown that no qualitative differences could be determined in lymphoid tissue of the pancreas, regardless of the size or amount, until such areas were occupied by wildly proliferative large lymphoid cells. The present study reports the results of a quantitative analysis of laboratory chickens from strains resistant and susceptible to lymphomatosis, and chickens obtained from farms. The percentage and size of lymphoid areas was less in the resistant lines. For practical purposes, in making a positive diagnosis of lymphomatous lesions the arbitrary point of 1 per cent lymphoid tissue was found to be a workable value. Microscopic examinations should be supplemented with gross examinations, since analysis of one or a few sections misses some positive cases. It is concluded that all ectopic lymphoid areas in an organ such as the pancreas are probably abnormal. However, this does not mean that all such areas are specific for lymphomatosis or incompatible with life.


In sarcoma-bearing hamsters, plasmacytes infiltrate hepatic periportal connective tissue. Since many plasmacytes are in the venous sinuses of the spleen and other portal organs, it was thought that perhaps this was their source. Terminal branches of the portal and splenic veins, the pancreas, spleen, and left anterior and cystic lobes of the liver were obtained from control, 16 pregnant, 6 lactating and 16 sarcomatous hamsters and fixed in sublimate-alcohol. Toluidine blue and methyl green-pyronin were among the stains used. The percentage of plasmacytes was the highest in the spleen of all groups, intermediate in the splenic vein and lowest in the portal vein. Plasmacytes disintegrated within the circulation. There was no evidence that splenic plasmacytes acted as a source for those in periportal connective tissue. They most likely arise there from lymphocytes in response to the presence of globulins in the blood of the sarcomatous hamsters.

E. H. Reinhard, J. T. Good, and E. Martin. From the Department of Medicine, Washington University School of Medicine, and the Barnes Hospital, St. Louis, Mo. J.A.M.A. 142: 383-390, 1950.

This review article summarizes present knowledge and applications of a number of chemotherapeutic agents in the treatment of the leukemias and related diseases. Included in the discussion are the use of stilbamidine and ethylstilbamidine in myeloma; urethane, in leukemia and myeloma; teropterin and dipterin, in various forms of cancer; aminopterin and related compounds, in leukemias; and the nitrogen mustards, in Hodgkin’s disease.

M. S. Sacks, G. T. Bradford and E. B. Schoenbach. From the Department of Medicine of the University of Maryland School of Medicine and the Department of Preventive Medicine, Johns Hopkins University School of Medicine, Baltimore, Md. Ann. Int. Med. 32: 80-215, 1950.

Detailed studies of 14 patients with acute leukemia treated with aminopterin and a-methopterin are presented. The therapeutic regimens employed consisted of the liberal use of whole blood transfusions, various antibiotics and the folic acid antagonists aminopterin (0.5 to 1.0 mg. per day intramuscularly) and a-methopterin (3.0 to 4.0 mg. daily). Two patients were considered to have undergone complete temporary clinical and hematologic remissions of two to three months’ duration. Both subsequently relapsed and died. Partial temporary remissions were observed in 3 patients, all of whom subsequently died. The course of the disease in 9 patients was considered to have been uninfluenced by the therapy. Stomatitis and diarrhea are probably the earliest toxic signs to be noted. Diffuse alopecia is common later in the therapeutic course.

D. P. Barr, G. G. Reader and C. H. Wheeler. From the Department of Medicine of the

This paper reports interesting clinical observations on a patient with a plasma protein precipitated by cooling and dissolved by warming to 37° C. Cryoproteins have been seen most often in association with multiple myeloma but may occur in leukemia, arthritis, disseminated lupus and in kala-azar. Because they may settle at room temperature with the blood cells, when proteins are determined by routine laboratory methods, they are probably missed on occasions. It is suggested that sensitivity to cold with Raynaud’s syndrome or purpura and with a tendency to excessive bleeding from many mucous membranes are the most prominent manifestations attributable to the presence of cryoproteins.—P.F.W.

Current Views Concerning the Nature and Management of Leukemia and Allied Disorders. M. M. Wintrobe. From the Department of Medicine, University of Utah College of Medicine, Salt Lake City, Utah. Am. Pract. 1: 68-74, 1950.

A brief review of current experimental approaches to the determination of the fundamental nature of leukemia and allied disorders is given, with a discussion of modern forms of therapy.—G.E.C.


A total of 120 patients with a variety of neoplastic diseases have been treated with nitrogen mustard. The authors conclude that Hodgkin’s disease responds favorably. Other conditions which responded favorably but not dramatically were fibrosarcoma, leukosarcoma, chondrosarcoma and monocytes leukemia. Only slight improvement was noted in melanoma, liposarcoma and carcinoma. Contrary to the opinion of many other workers in this field, the authors conclude: “Nitrogen mustard is probably contraindicated in lymphatic and myeloid leukemia, due to the magnitude of lymphoblastic destruction.” —G.E.C.


Twenty-four patients with tumors of the hematopoietic tissues, two with polycythemia rubra vera, and one with metastatic carcinoma were treated with intravenous injections of radioarsenic (As14). The authors conclude that this isotope resembles but is not superior to numerous other agents such as x-ray, urethane, nitrogen mustard, radioactive phosphorus and radio-sodium in that it is capable of producing a remission in certain of the more chronic and benign tumors of the hematopoietic tissues.—G.E.C.


This report deals with 9 cases of acute myelocytic leukemia treated according to the suggestion of Bessis and Bernard with exchange transfusion. It is the author’s opinion that this type of therapy is not satisfactory, and that after these transfusions there may be noted peculiar changes in the blood and bone marrow. Also there is frequently a transient improvement of the clinical picture. He presents the hypothesis that in the normal blood there is a substance which compensates for an unknown deficiency or acts as an antileukemic agent.—C.M.


A new anomaly of leukocytic granulation is described in the case of a 34 year old infant. In nearly all of the neutrophils, as well as in the eosinophils, there were found coarse, partly triangularly indented clumps, staining deeply blue-red with the Giemsa method. The form of the nucleus was found pathologic too, insofar as the segmented ones rarely showed complete segmentation. At the same time the lympho-
cytes have unusual azurophilic granules, the size ranging up to 3 μ. The pathologic changes of these cells can be found in the myeloblasts. This anomaly is not identical with the abnormal granulation found by Alder (Deutsche Arch. f. klin. Med. 183: 372, 1949).—C.M.


The leukocytes have a tendency for aggregation (similar to the erythrocytes) which is determined by the amount of negative charge of the membrane, the surface tension and the properties of the plasma. By means of a special method, the "leukocyte-aggregation reaction," the author found that only mature leukocytes aggregate. Myeloblasts and lymphoblasts do not show this phenomenon. The capacity for aggregation increases with enlarging capacity for phagocytosis, whereby the electric charge usually diminishes.—C.M.

BLOOD COAGULATION AND HEMORRHAGIC DISEASES


The authors found that 13 per cent of a series of 105 surgical patients on routine admissions showed prothrombin levels considered abnormal. They found postoperative hemorrhage and "deficient" wound healing more frequent in the patients with hypoprothrombinemia. Wounds produced in rats receiving dicumarol had less tensile strength on the third and fifth postoperative days than did comparable wounds produced in rats not receiving dicumarol. They suggest hypoprothrombinemia may contribute to impaired wound healing. There was no correlation between the amount of hemorrhage and the tensile strength of the experimental wounds; several of the wounds with decreased tensile strength showed no hemorrhage at all.—P.F.W.


The data indicate that meals with random selection of foods and strenuous exercise that is not prolonged or exhausting produce no significant changes in prothrombin times. It is concluded that it is probably not necessary that blood for prothrombin tests be taken with the patient fasting and at bed rest.—P.F.W.


In animals maintained on dicumarol, vitamin K1 and K1 oxide may effect a prompt return of prothrombin time toward normal. Similar results were found in patients. A 1 gram dose of vitamin K1 intravenously seemed optimal in man to correct the hemorrhagic tendency within a matter of hours. In contrast to this, the water-soluble synthetic vitamin K preparations tested showed no antagonistic effect. It is suggested that the apparent effectiveness of the water-soluble vitamin K compounds reported previously may be evident only after much of the dicumarol has been disposed of by the body. If further anticoagulant therapy is required following a recent injection of vitamin K1 or K1 oxide, it may prove more convenient to change temporarily to heparin.—P.F.W.

Dicumarol Prophylaxis of Thromboembolic Disease in Congestive Heart Failure. W. P. Harvey and C. A. Finch. From the Medical Clinic, Peter Bent Brigham Hospital and the Department of Medicine, Harvard Medical School, Boston, Mass. New England J. Med. 242: 208-211, 1950.

One hundred and eighty patients with congestive heart failure were studied. One group received dicumarol and a control group did not. The groups were similar in types of heart disease, age distribution and severity of heart failure. It is suggested that dicumarol administered in amounts sufficient to lower
the prothrombin level to 30 to 50 per cent of normal results in a significant reduction in thromboembolic disease in patients hospitalized with congestive failure.—P.F.W.


This is a report on a series of surgical cases studied over a four-year period of time. Early postoperative ambulation did not seem to alter the incidence of thromboembolic disease. Prophylactic levels of 40 to 50 per cent prothrombin index attained on dicumarol therapy decreased the rate of thromboembolism.—P.F.W.


This is a review of the literature on the chemistry, mechanism of action, mode of administration and toxicity of heparin and dicumarol, with a brief discussion on the clinical use of anticoagulants.—P.F.W.


Increased capillary fragility in premature infants may be considered a presumptive factor favoring frequent central nervous system hemorrhage found in this group of infants. Seven of 8 premature infants having intracranial hemorrhage were found to have a capillary resistance less than 30 cm. of mercury. In 23 of 24 premature infants who were normal, the capillary resistance was greater than 30 cm. of mercury.—J.P.S.

**Idiopathic Thrombocytopenic Purpura.** H. N. Robson. From the Department of Medicine, University of Edinburgh, Edinburgh, Scotland. Quart. J. Med. 28: 299-318, 1949.

An investigation was made of bleeding time, capillary resistance, platelet counts, and megakaryocytic cytology before and after splenectomy in subjects with idiopathic thrombocytopenic purpura and subjects with and without thrombopenia undergoing splenectomy for other causes. Similar studies were made on patients undergoing abdominal operations other than splenectomy and, in addition, bone marrow studies were made on 3 patients with secondary purpuras due to toxic agents. The latter group did not undergo splenectomy.

It was shown that a nonspecific lowering of bleeding time and an increased capillary resistance accompanied abdominal operations whether or not splenectomy was part of the procedure. In this respect, however, although improvement in these tests was evident by the time the splenic pedicle was ligated, the maximal response occurred later in splenectomies for idiopathic thrombocytopenias than in controls with no preoperative thrombopenia. The platelet count showed little correlation with changes in the other tests in the thrombopenic groups. The author points out that in 8 of 12 patients checked three months after splenectomy for idiopathic thrombocytopenic purpura, one or more of the preoperative hemostatic tests had again become abnormal. In 4 patients, all tests were still normal three months after splenectomy. However, only 2 of 16 patients had return of clinical bleeding.

No evidence of an increase in primitive megakaryocytes was thought to be present in the marrows of patients with idiopathic thrombocytoppenic purpuras. However, diminished platelet production was evident and this was alleviated by splenectomy.

The author subscribes to the view that the cause of idiopathic thrombocytoppenic purpura is the elaboration of some factor by the spleen and other reticulo-endothelial tissues which alters the state of the capillaries and reduces platelet formation from megakaryocytes.—W.N.V.


Three cases of thrombocytoppenic purpura following rubella are described. One patient died of cerebral hemorrhage. Some lowering of platelet counts and some increase in capillary fragility were also noted at the onset of uncomplicated rubella, these abnormalities disappearing during convalescence. The degree
of these abnormalities varied considerably from patient to patient and bore no close relationship to the severity of the disease. The author states that similar changes occur in many acute infections and that purpura may complicate either mild or severe infections. He concludes that the capillary abnormalities and purpura occurring early in an infectious disease are more dependent upon the susceptibility of patients' tissues—particularly the capillaries—to damage than upon the intensity of primary infection. Thrombocytopenia is also sometimes a cause, as in the 3 cases here reported. The development of purpura in convalescence is best explained on the assumption of an allergic basis.—W.N.V.


A new coumarin compound, bis-3,3'-(4-oxy coumarinyl) ethyl acetate (B.O.E.A.), was used in treatment of 126 patients with thromboembolic conditions. This anticoagulant is about 4 times less active than dicumarol. In general 0.9 to 1.2 Gm. were given on the first two days, followed by 0.3 to 0.6 Gm. per day according to the prothrombin level.

The advantages over dicumarol are the more rapid initial action and quicker return to normal after stopping treatment. In over 80 per cent of cases the prothrombin was reduced to under 0.1 per cent within thirty-six hours and returned to normal in a similar short period at the end of treatment. As with dicumarol, treatment has to be controlled by frequent prothrombin estimations to avoid dangerously low levels.—S.C.


The effects of 4-hydroxycumarin anticoagulant No. 63 (1-methyl-2-methoxy-4-phenyl-5-oxodihydropyranos-(3,2-C) (1) benzopyran) have been studied in dogs and man. An optimal effect from a single dose of this new compound was usually obtained in twenty-four to forty-eight hours. The anticoagulant potency is two to three times that of dicumarol. In the dogs, it appeared that the new compound had a greater and longer anticoagulant activity without the appearance of hemorrhagic manifestations than did dicumarol. The microscopic findings in the dogs given lethal doses of this compound did not reveal evidence of toxic lesions in the small vessels such as have been reported with lethal doses of dicumarol. Further evaluation will be necessary before the clinical use of this compound can be defined. It is possible that as greater experience with the new anticoagulant accumulates, a certain fixed dose for each patient may be given either daily or every third or fourth day and the necessity for frequent prothrombin estimations curtailed.—G.E.C.


Using a stable thromboplastin of high activity, 184 paired prothrombin determinations have been done by the bedside test and the Quick one-stage test on 16 patients undergoing dicumarol therapy and on 38 normal persons. A correlation coefficient of 0.810 was found between the prothrombin times obtained by the two methods. When the thromboplastin was diluted to give a bedside prothrombin time of 24 to 43 seconds for normal persons, it was found that the clotting powers as derived from the bedside tests were in agreement with those obtained by the use of the more active thromboplastin. In sixty-eight paired tests the correlation coefficient was found to be 0.872. The authors suggest that with the use of a stable, active thromboplastin, the bedside test is adequate for the control of dicumarol therapy. However, it is essential to obtain frequent values on normal blood to ensure that the thromboplastin retains its activity. In order to convert prothrombin times, as obtained by the bedside test, to prothrombin activity, they recommend the preparation of a dilution curve by the one-stage method at weekly intervals, or more frequently if the values show any tendency to vary.—G.E.C.
THE SPLEEN


This comprehensive article details experiences with splenic puncture in a series of 55 patients with splenomegaly, ranging in age from 6 months to 71 years. The procedure is considered to be of value in the diagnosis of obscure cases of leukemia, myeloid metaplasia of the spleen, and fat storage diseases, as well as in the diagnosis of tropical splenomegalies. In addition, it is an excellent method for following the effects of therapy on a diseased spleen.

A total of 91 punctures were made in the 55 patients. Blood studies, including hemorrhagic tests, were performed before the puncture, and patients with idiopathic thrombocytopenic purpura and with abnormal bleeding tests were usually omitted from the procedure. Four patients bled intraperitoneally following the procedure; all recovered. Two patients died: the first, of a dissecting hematoma originating in the rectal muscle; the second, of rupture of the splenic pedicle. In neither case was the hemorrhage at or near the site of the splenic puncture, and it was felt that the procedure had not caused the deaths.

In one patient (aged 6 months), no material was obtained. In all the others, adequate splenic material was obtained.

The authors detail the technic of puncture, and emphasize the importance of absolute bedrest following the procedure. Disregard of this rule resulted in increased pain, intraperitoneal hemorrhage, and perhaps also the death of one of the two patients who died. Contraindications to splenic puncture include a hemorrhagic tendency, and nonpalpability of the spleen. On the whole, however, it is felt that the procedure is of sufficient value to justify its cautious use in patients with splenomegaly.—S.E.


By means of nine case reports, the authors amplify their concepts that hypersplenism is a disorder in which the spleen destroys excessive numbers of red cells, white cells, or platelets; and detail their criteria for diagnosis and treatment. Two cases of idiopathic thrombocytopenic purpura are presented; 2 of familial spherocytosis; and one each of acquired idiopathic hemolytic anemia, primary splenic leukopenia, primary splenic panhematopenia, secondary hypersplenism, and hypoplastia of the marrow.

Emphasis is placed on four criteria for diagnosis: enlargement of the spleen, cytopenia, normal bone marrow, and positiveness of the epinephrine tests. As the authors point out, however, exceptions are so numerous as to make rigidity of these criteria incorrect. Splenomegaly does not occur in idiopathic thrombocytopenic purpura, nor is the epinephrine test positive in this disease. The phagocytic role of the spleen in all these conditions is considered to be similar to that of the spleen in familial spherocytosis; yet, in the latter condition, it is known that the disease is probably one of the red cell, not the spleen itself.

The curative nature of splenectomy is pointed out in the case reports. Although the authors' pathogenesis of hypersplenism is not agreed on by others, it is evident that the diagnosis is becoming more standard and more tenable; and that splenectomy is therefore curing patients who previously went undiagnosed and ineffectively treated.—S.E.

CYST OF THE SPLEEN IN SICKLE CELL ANEMIA. H. R. Pratt-Thomas and P. K. Switzer. From the Departments of Pathology and Medicine, Medical College of the State of South Carolina, Charleston, S. C. Arch. Path. 49: 159-162, 1950.

A splenic mass was removed from a 24 year old Negro woman. She was undernourished and studies of her blood showed sickle cell anemia. Microscopic examination of the tissue showed that all the vascular channels were packed with sickle cells, and that siderofibrosis with the formation of "Gamma-Gandy nodules" was conspicuous. The cyst wall was composed of organizing fibrous tissue in its inner portion, with a coating of fibrin and fibrinoid material covering its inner portion. This cyst probably belongs to the group of degeneration cysts arising from secondary changes in areas infected as a result of arterial degeneration or embolic occlusion of blood vessels with consequent necrosis of the splenic pulp. A spleen of this size (13 x 13 x 13.5 cm.) is rare in sickle cell anemia and the occurrence of a solitary cyst is even more unusual.—O.F.J.