LEUKOCYTES

In this study, administration of glucose to normal individuals caused lymphocytopenia, whereas moderate hypoglycemia caused lymphocytosis. In the latter instance the patients showed no symptoms of hypoglycemia, and it is assumed the stress factor was avoided. The lymphocytopenia after glucose administration is inferred to be due to adrenocortical stimulation caused by the change in the blood sugar level. The absolute lymphocytosis after insulin is assumed to be due to adrenocortical inhibition resulting from moderate hypoglycemia. The authors suggest that these responses serve to maintain homeostasis.—R.B.C.

This author has observed that distention of the colon is at times accompanied by a "degenerative" type of white cell reaction, the outstanding features of which are unexpectedly low to leukopenic total counts and very high nonfilamental neutrophil percentages. It occurs only with distention of the large bowel and may develop within twenty-four hours of onset. He presents 6 cases of small intestine distention and 2 cases of minimal large bowel distention that did not show these changes. Five cases of large bowel distention demonstrated these changes.—R.B.C.

When purified protein derivative is injected intravenously into tuberculous rabbits there is a decrease in the total number of white blood cells and lymphocytes. In six to nine hours this response reaches its maximum, with a gradual return to normal. There is often a delayed increase in neutrophils.—R.B.C.

The plasma of patients acutely ill with disseminated lupus erythematosus contains a factor which induces the formation of rosettes of leukocytes and a characteristic inclusion-containing cell, the lupus erythematosus (LE) cell, when mixed with normal bone marrow preparations. This study was designed to identify the fraction of the LE plasma responsible for inducing this phenomenon. Of the various plasma fractions tested, only the gamma globulin fraction was found to contain such ability.—G.E.C.
An eosinophilia during the acute stage of infectious mononucleosis is not rare. In a personal series of 82 cases, 41.5 per cent had 4 per cent or more eosinophils and 16.8 per cent had 5 per cent or more. In a hospital series of 63 cases, 24.2 per cent showed 4 per cent or over and 12.9 per cent showed 5 per cent or over. In the differential counts recorded in the series of several authors there was an eosinophilia of 4 per cent or more in 10 to 20 per cent of cases. A smear showing numerous atypical mononuclear cells, especially with a decrease in percentage of neutrophils and a shift to the left, suggests infectious mononucleosis; another point in favor of that diagnosis is an eosinophilia of 4 to 10 per cent or slightly higher.—G.E.C.


Four cases are reported. In the first, a 6-year-old girl, acute appendicitis was diagnosed, but the blood count, showing 42,000 white cells with 80 per cent normal lymphocytes, delayed surgery. Complete clinical and hematologic recovery took place within twenty-one days.

The second child, 7 years old, brother of the first case, showed the same acute abdominal symptoms: 17,500 leukocytes with 82 per cent lymphocytes twenty days after his sister’s episode. At the same time the mother developed symptoms of right colitis with 15,700 leukocytes and 74 per cent lymphocytes (mother and son had had normal blood values 2.2. days before).

The fourth case was a 9-year-old child, with symptoms of subacute appendicitis. The white cell count was 31,500 with 76 per cent lymphocytes. He recovered promptly, as did the other 3 cases.—J.P.S.


In a study of 12 cases observed in Rabat (Morocco), an anemia with 2.960,000 erythrocytes was found in one case, and with 3.350,000 erythrocytes in the other case. There were typical changes in the leukocyte percentages in the peripheral blood, and generalized symptoms which completely disappeared spontaneously. This justified the diagnosis of acute infectious lymphocytosis of Carl Smith with its favorable prognosis, whereas the presence of the marked anemia had originally aroused serious suspicion of an acute leukemia.—J.P.S.

MALIGNANT LYMPHOMA AND LEUKEMIA


In an analysis of 406 patients operated upon at the Lahey Clinic between 1941 and 1945 for malignant disease of the stomach, 16 had sarcomas of the stomach. Two of these were leiomyosarcomas and 14 were tumors of lymphoid origin. This represents an incidence of 4.0 per cent, which is considerably higher than that reported in the literature.

This report covers 23 cases of primary lymphoid gastric tumors. The clinical course of patients with these tumors is indistinguishable from those with carcinoma as to history, physical examination, laboratory findings, and results of treatment. Indigestion, pain, or discomfort is invariable. There were no instances of hematemesis or tarry stools. Prognosis was felt to be better for these patients than for those with carcinoma of the stomach. Treatment should consist first of a biopsy to establish a definite pathologic diagnosis. Resection of the malignant tumor should then be carried out, if possible followed by x-ray treatment. If resection is impossible, x-ray therapy should be given. Several lengthy survivals following x-ray therapy alone have been reported.—R.B.C.

Plasma-Cell Tumor of the Stomach, with Report of a Case. N. Ende, P. D. Baron, L. K. Richardson, L. Raider, and J. Ziskind. From the Clinical Laboratory, Departments of Surgery and Radiology, Veterans Administration Hospital, New Orleans; the Departments of Radiology and Surgery, Louisiana State University School of Medicine; and the Department of Pathology, Tulane University School of Medicine, New Orleans, La. Radiology 55: 207–213, 1950.
In a search of the literature to 1943, it was found that, of 127 cases of extramedullary plasma-cell tumor, only one case included involvement of the stomach. Subsequently, 2 further instances of involvement of the stomach by this disease were reported. The present authors report a third, in which plasma-cell tumor was apparently localized entirely to the stomach and neighboring tissues.

The patient was a 63 year old Negro with upper abdominal pain, eructation, nausea, and weight loss for a period of three years. X-ray studies showed filling defects along the greater curvature of the stomach; at operation a subtotal resection of the stomach together with removal of the gastro-colic ligament, part of the omentum, and part of the transverse colon were successfully performed. Pathologic examination revealed plasmacytoma. Studies of the blood were unremarkable, and the urine was normal. A single bone marrow puncture showed normal results, as did skeletal x-ray studies. The patient received postoperative irradiation, and was living and well, with no laboratory abnormalities, seven months after the operation.—S.E.

A Study in Renal Function in Patients with Multiple Myeloma. J. B. Armstrong. From the Department of Medicine, Duke University School of Medicine, Durham, N. C. Am. J. M. Sc. 219: 488-493, 1950.

Eighteen renal clearance studies were performed on 15 patients with multiple myeloma who presented no evidence of other renal disease. The glomerular filtration rate (GFR), effective renal plasma flow (ERPF) and maximal tubular excretory capacity (TmPAH) were determined. There was no simple correlation between the renal functional impairment, the age of the patient, the duration of the disease, the presence or degree of Bence Jones proteinuria, the hematologic findings, or the quantity of abnormal serum protein. The glomerular filtration rate was found to be impaired to an equal or greater extent than the tubular excretory capacity. This finding was more evident in the patients with poorer kidney function. The filtration fraction was not consistently altered. No final conclusion can be drawn from these studies as to the cause or morphologic site of the renal lesion in multiple myeloma. The lowering of the GFR/Tm ratio suggests that glomerular as well as tubular changes occur, but the absence of a consistent fall in filtration fraction does not indicate a primary and predominant glomerular lesion.—G.E.C.


Satisfactory sustained remissions were achieved in 4 of 37 cases of chronic myelocytic leukemia in which urethane was administered. In an additional 13 cases, significant early remissions were followed by the development of a urethane-refractory state and the appearance of clinical and hematologic relapse. In the remaining 20 cases, treatment with urethane was not clinically effective, although in 14 cases a moderate reduction in the total leukocyte count of the peripheral blood was effected. The administration of therapeutically active amounts of urethane resulted in distressing side effects in many instances, although no serious side effects were observed.

The authors conclude that ionizing roentgen radiation is much more consistently effective in the palliative treatment of chronic myelocytic leukemia than is urethane.—G.E.C.


Twelve patients with acute leukemia were administered cortisone. The daily dose for adults was 200 to 300 mg. of cortisone and that for children was 50 to 75 per cent of the adult dose. When possible, the hormone was administered for at least twenty to thirty days. Two patients became clinically well, while the blood and bone marrow were so improved that a diagnosis of leukemia could not be made. The remissions lasted three to five weeks. Nine patients experienced no improvement. Six patients with acute leukemia received ACTH. One had a complete remission lasting sixteen weeks after administration of 1 Gm. of ACTH over a period of five days. After relapse a second remission could not be induced. A moderately severe leukopenia may be induced with such hormone therapy. Three patients with
chronic lymphatic leukemia were given one or more courses of cortisone and all showed a 40 to 60 percent decrease in liver, spleen and lymph nodes for ten to twenty days. Three patients with Hodgkin's disease received a full course of cortisone. The spleen and nodes of all 3 patients regressed for fifteen to thirty days. Biopsy specimens before and after therapy revealed no significant differences.—P.F.W.

RETICULOENDOTHELIAL GRANULOMA: A REVIEW WITH A REPORT OF A CASE OF LETTERER-SIWE DISEASE.

The authors report a fatal case of Letterer-Siwe disease in a 6 month old infant, and discuss the relationships between eosinophilic granuloma of bone, Hand-Schüller-Christian disease, and Letterer-Siwe disease—all varieties of reticuloendothelial granuloma.—S.E.

LETTERER-SIWE'S SYNDROME: REPORT OF A CASE WITH UNUSUAL PERIPHERAL BLOOD CHANGES.

This report concerns a 3½ year old boy who died after a four-month illness characterized by pallor, ankle pain, malaise, lymphadenopathy, and splenomegaly. Autopsy revealed a diffuse, generalized granuloma involving the lymph nodes, spleen, liver, lungs, and kidneys. The characteristic cell pathologically was a large, reticulum cell. During life, identical cells appeared in the peripheral blood, numbering from 1 to 30 per cent of the white cells (3,100 to 7,000 leukocytes per cu. mm.). A marrow puncture during life showed replacement of the marrow with such “hemohistiocytes” or “reticulum cells.” The blood picture was one of pancytopenia with granulocytopenia, relative lymphocytosis, and reticulum cells in the blood.—S.E.

BLOOD COAGULATION
NEWER CONCEPTS OF THE COAGULATION OF BLOOD.

This review article presents a theory of the coagulation of blood based on observations carried out in Dr. Armand J. Quick’s laboratory. The analytical findings in support of this theory are referred to in the bibliography. The theory presented states that there are three phases in the coagulation of blood. In phase one, thromboplastin is formed through the action of an enzymatic agent supplied by the platelets (thromboplastinogenase) on a plasmatic precursor (thromboplastinogen). In phase two, thrombin is formed by interaction between prothrombin, thromboplastin, labile factor, and calcium. The thrombin “labilizes” platelets leading to the production of more thromboplastin. Thrombin also acts to convert the labile factor into an accelerator agent (Factor VI, serum Ac-globulin, and possibly SPCA [serum prothrombin conversion accelerator]) capable of accelerating the formation of thrombin from prothrombin. In phase three, thrombin reacts with fibrinogen to produce fibrin and a clot. Factors which antagonize these substances are discussed. A classification of hemorrhagic diseases on the basis of their abnormality in the process of coagulation of blood is presented and various hemorrhagic diseases are briefly discussed in relation to this classification. It is emphasized that several abnormalities of the hemostatic mechanism are usually present in hemorrhagic diseases.—R.B.C.

PROTHROMBIN UTILIZATION DURING CLOTTING: COMPARISON OF RESULTS WITH THE TWO-STAGE AND ONE-STAGE METHODS.

This study presents comparative data on prothrombin utilization during clotting, as indicated by the two-stage and one-stage methods. Simultaneous one- and two-stage determinations of prothrombin activity were performed on a series of bloods during and after clotting. By the two-stage method, progressive disappearance of prothrombin from serum was demonstrated. By the one-stage method, an initial period of hypoactivity in the plasma was followed by a hyperactive phase in the serum. In slowly clot-
ABSTRACTS


The authors briefly review the classic theory of blood coagulation and summarize the action of a number of more recently described factors which are believed to exert an influence on the clotting mechanism. These factors include plasma Ac-globulin, SPCA, and Factor V of Owren. In this investigation the comparative effect of storage at low temperature upon plasma prothrombin activity of newborn infants and adults was studied, and second, the effect of storage on the prothrombin activity of plasma was observed. The prothrombin activity of plasma of normal adults and newborn infants was determined at varying intervals after initiating storage at low temperature (10-20°C). Similar determinations were made on a group of newborn infants who had received vitamin K at birth. The method of Quick was used for prothrombin determinations. The results were expressed in percentage concentration of normal adult plasma. These results showed a decrease in the plasma prothrombin concentration which followed a hyperbolic course. This occurred with adult plasma and with newborn infant plasma both before and after vitamin K therapy. The authors feel that these results lend further support to the theory that prothrombin in the newborn infant is qualitatively and physiologically similar to that in the adult. They feel that other accelerator and inhibiting factors in newborn infants and adults are similar. The study also showed that plasma stored longer than one week would be of little value as an effective source of prothrombin.—R. B. C.


The purpose of this investigation was to determine the effect of sulfadiazine in the usual therapeutic dosage upon the prothrombin of the newborn infant. Prothrombin determinations were done on the plasmas of 6 normal newborn infants during the first forty-eight hours of life, and again after seventy-two hours of sulfadiazine administration in doses of 1 grain per pound of body weight per twenty-four hours. The results uniformly showed neither a decrease in the prothrombin concentration nor did the sulfadiazine prevent the expected rise in prothrombin concentration from the usual hypoprothrombinemic level present in the neonatal period.—R. B. C.

THE SIGNIFICANCE OF DIFFERENT METHODS FOR PROTHROMBIN ESTIMATION AND THEIR RELATIVE VALUES. J. H. Olwin. From the Department of Surgery, Presbyterian Hospital, affiliated with the University of Illinois College of Medicine, Chicago, Ill. Surg., Gynec. & Obst. 90: 413-419, 1950.

The author presents a practical discussion of the tests in current use which are based on the one- and two-stage methods for measuring prothrombin. The accuracy and significance of each method are reviewed, and it is emphasized that an understanding of what each method represents should be the determining factor in the selection of the particular test needed.—H. W. B.

The factors taking part in the formation of thrombin are not totally utilized during coagulation. In the one-stage technic, after the thrombin formed has been neutralized by the natural antithrombin, three proteins active in the promoting of blood coagulation may be present. These are unconverted prothrombin, unconverted labile factor (Factor V, plasma Ac-globulin), and an accelerator substance (serum Ac-globulin, Factor VI, SPCA).

This paper presents data on the utilization of labile factor during coagulation in healthy subjects and in patients with different disorders of the clotting mechanism. About 8 per cent of the labile factor was utilized during coagulation in healthy subjects, and smaller amounts in those presenting various pathologic conditions. The percentage of labile factor utilized during coagulation is roughly proportional to that of the prothrombin consumed and in inverse proportion to the concentration of accelerator developed in serum. In the sera of healthy subjects, a very small percentage of labile factor and prothrombin activity and a high value of accelerator substance are found. Low values of accelerator and a large percentage of prothrombin and labile factor activity are found whenever the formation of thrombin is deficient because of depletion of either thromboplastin (hemophilia, thrombocytopenic purpura) or prothrombin and labile factor (liver dysfunction). In dicumarol hypoprothrombinemia, the available prothrombin is well utilized during coagulation, but the percentage in serum of unconverted labile factor is high and that of the accelerator is low.—R.B.C.


The relationship between the proteolytic activity of plasma and blood coagulation has yet to be clarified. Recently, considerable evidence has accumulated in favor of the view that proteolysis plays an essential role in the coagulation of blood.

In the present study, a fraction of human globulin was prepared which was deficient in prothrombin, thrombin, fibrinogen, plasma thromboplastin and accelerator globulin, but which contained potential proteolytic activity. For reasons as yet not clear this globulin fraction accelerated the clotting of normal platelet-deficient plasma. Its clot-accelerating effect, however, was the same whether or not its proteolytic property had been activated by streptococcal fibrinolysin. Furthermore, the addition of streptococcal fibrinolysin directly to platelet-deficient plasma (normal and hemophilic) activated the proteolytic enzyme in this plasma without accelerating coagulation.

This study casts doubt on the concept that there is a direct and obvious relationship between proteolysis and the physiologic coagulation of blood. The possibility that the precursor of plasma proteolytic enzyme may promote coagulation, however, cannot be excluded.—H.W.B.


Data have been obtained which indicate that fibrinogen may play a role in protecting Ac-globulin against various physical and chemical agents. Data also indicate that in addition it may inhibit Ac-globulin activity in the conversion phase of the normal clotting process. The fibrinogen apparently acts as a buffer or protective substance to Ac-globulin. It is suggested that the Ac-globulin "molecules" may form a complex with fibrinogen molecules. The conversion of an inert pro-enzyme plasma Ac-globulin to an active serum Ac-globulin may be mediated by thrombin or by any chemical or physical agent which effectively removes fibrinogen without concomitantly removing the Ac-globulin.—R.B.C.


During the process of normal coagulation of blood, the conversion of prothrombin to thrombin occurs with increasing velocity. It is considered to be an "autocatalytic" phenomenon. Certain sub-
stances which arise during coagulation accelerate, activate, or otherwise act as ancillary agents in the conversion of prothrombin to thrombin in the presence of thromboplastin and calcium. These include the serum Ac-globulin, Factor VI of Owren, SPCA of Alexander, prothrombin kinase and thrombin kinase of Milstone, and the "labile factor" of Quick. It is the opinion of Alexander et al. that it is unlikely that these substances are entirely different. Dissimilarities may be due to differences in the technics of study, in purity and source of material, and in interpretation. This paper reports a comparative study of the action and properties of SPCA and serum Ac-globulin. The authors conclude that SPCA accelerates the conversion of prothrombin to thrombin in the presence of a labile plasma component. They believe that "serum Ac-globulin" comprises a mixture, or chemical combination, of at least two clotting components—plasma Ac-globulin and SPCA. The former is a plasma component, is labile, and is not readily adsorbed by BaSO₄ or BaCO₃, whereas SPCA is a serum factor which is relatively stable and is adsorbed by these substances. The labile plasma Ac-globulin is probably identical with the "labile factor" of Quick and is essential for rapid prothrombin conversion. As SPCA is evolved during coagulation, the reaction is further accelerated, and it appears to Alexander et al. that SPCA is the true "auto-catalytic accelerator" of prothrombin conversion.—R.B.C.

**THE PROBLEM OF THE LIPOID THROMBOPLASTINS.** J. H. Milstone. From the Laboratory of Pathology, Yale University School of Medicine, New Haven, Conn. Yale J. Biol. & Med. 22: 675-687, 1950.

The author discusses the possible role of lipoid thromboplastin (in these studies, that fraction of brain soluble in ether but not in acetone) and the possible role of trypsin on the clotting mechanism. In the presence of calcium, either trypsin or crude cephalin is found to activate crude prothrombin. However, as prothrombin is purified, it reaches a state which is readily activated by crystallized trypsin (a protein, not a lipoprotein) but not by crude cephalin. In the presence of platelets, minute amounts of trypsin (in terms of micrograms and assayable proteolytic activity) produced a large effect on the conversion of prothrombin. Without platelets, the amount of trypsin necessary readily to activate the prothrombin had to be increased severalfold. The author points out that the behavior of thrombokinase is consistent with the possibility that it is an enzyme, and that there is no established case of a lipoprotein which is an enzyme. The actions of lipoid thromboplastins remain an unsolved problem with several possibilities yet to be explored.—W.N.V.

**HYPOCOAGULABILITY OF CERTAIN IRRADIATED PLASMAS.** S. S. Cutler, B. Burbank and E. R. Marullo. From the Department of Medicine, Long Island College Hospital and Long Island College of Medicine, Brooklyn, N. Y. J.A.M.A. 143: 1057-1059, 1950.

Irradiated plasma which is currently supplied is prepared in two ways, the one involving irradiation with energy chiefly at 2.537 Å (Sharp and Dohme method), the other with irradiation at both 2.537 Å and 1849 Å (Michael Reese method). Although such ultraviolet irradiation has been felt to cause no chemical or electrophoretic alterations in plasma, the authors found certain changes in the coagulability of the Michael Reese plasma, which suggested to them that radiation of energy 1849 Å may cause profound biochemical changes in plasma.

They found that, while nonirradiated plasma, and Sharp and Dohme irradiated plasma, could be promptly coagulated by means of a small and constant amount of thrombin, certain lots of Michael Reese prepared plasma could not be coagulated, even when large amounts of thrombin were employed. Protamine sulfate and toluidine blue were capable of partially restoring the coagulability of these plasmas; hence, it was felt that the irradiation had resulted in the formation of excessive amounts of heparin or heparin-like substances. These lots of plasma also increased the coagulation time of normal blood.

In addition, however, since enormous amounts of thrombin failed to induce coagulation, it was felt that the heparin theory was only part of the answer, and that some more complex alteration (perhaps in the character of the fibrinogen) occurred following irradiation.

The importance of this work at present is purely theoretic, but clinical applications are discussed.—S.E.

**A FURTHER REPORT ON DICUMAROL PROPHYLAXIS AGAINST VENOUS THROMBOSIS IN WOMEN UNDERGOING SURGERY.** G. Van S. Smith. From the Free Hospital for Women, Brookline, Mass., and the Depart-
The purpose of the present study, still in progress, was to determine whether dicumarol in conservative doses (so as to avoid hemorrhagic complications as well as to maintain extensive laboratory control) would reduce appreciably the incidence of postoperative thromboembolic phenomena.

The study covered a period of over six years and included 3,078 ward patients over 35 years of age undergoing major and vaginal plastic surgery. One-half the group received dicumarol one to two days postoperatively and again five days later. The others received it the night before operation and in most instances four or five days later. The dose was 100 mg. for those weighing 60 kilograms, and 100 mg. for those weighing less. Hemorrhagic manifestations were few and minor; no death was attributable to the drug, and only rarely did this dosage require laboratory control.

Comparisons between the dicumarol-treated patients and similar groups of untreated patients indicated that conservative dicumarol prophylaxis is of value in helping to reduce the incidence of postoperative thromboembolism, although the results were not as striking as those reported by others using larger doses. It would appear that a conservative program such as this could be used with relative safety where laboratory control of dicumarol dosage was poor. It is pointed out, however, that this dosage schedule is of value only during its course of action, since the incidence of thrombotic complications after the fourteenth postoperative day was not appreciably reduced.—H.W.B.

HEMORRHAGIC DISEASE


In connection with the newer concepts of the mechanism of hemostasis there has developed an expanding battery of diagnostic tests. These tests assess the different function and phases of the hemostatic mechanism. The author outlines the various diagnostic methods available and classifies the methods in relation to (1) the study of platelet function, (2) the study of vascular function, and (3) the study of the clotting mechanism. He describes the tests in detail in many instances and in chart form presents the diagnostic features of hemorrhagic diseases as determined by laboratory procedures. The hemorrhagic diseases are divided into (1) those primarily due to platelet dysfunction, (2) those due to vascular dysfunction, (3) those primarily due to deficient clotting mechanism, and (4) those secondary to systemic disease. The evaluation of the etiology of a bleeding condition has become an increasingly complex problem. This article in compact form provides the working framework by which this problem may be approached.—R.B.C.


The authors report the results of their investigation into the nature of the hemostatic defect in 118 cases of hepatocellular and obstructive jaundice with a bleeding tendency. They found prolongation of the coagulation time in 92 per cent of hepatocellular and 84 per cent of obstructive cases, a decrease in the prothrombin activity in most of the cases of both groups, an increase in plasmin activity in the majority of cases of hepatocellular jaundice but in only a few of the obstructive cases, low normal or decreased antithrombin activity in hepatocellular and increased activity in obstructive cases, decreased fibrinogen reactivity in hepatocellular and increased reactivity in obstructive cases. Fibrinogen, platelet counts, and accelerator globulin activity were not significantly abnormal. On the basis of these studies the authors conclude that the coagulation defect in obstructive jaundice is due to decreased prothrombin activity and increased antithrombin activity. In hepatocellular jaundice a decrease in activity of clotting proteins and an increase in plasmin activity was observed. The increase in plasmin activity was felt to be responsible for the coagulation defect in hepatocellular jaundice. The therapeutic implications of these conclusions are briefly discussed.—R.B.C.
ABSTRACTS


A case is reported of dramatic gastro-intestinal hemorrhage in a 36 year old male. In spite of two blood transfusions, persistent bleeding necessitated gastrectomy. The resected stomach and the varioliform gastritis are well illustrated photographically. Histologic examination revealed a striking gastric metaplasia. Such a syndrome seems to be quite exceptional, and only five published reports (all French) have been found on the subject.—J.P.S.


One hundred cases of idiopathic thrombocytopenic purpura were analyzed in an attempt to correlate the bone marrow eosinophilic index (the number of eosinophils per 1000 neutrophils, including metamyelocytes) with prognosis.

On the basis of the eosinophilic index, the authors divided the cases into two groups: (1) those with a high index (i.e., increased numbers of marrow eosinophils), and (2) those with a low index (i.e., no increase in marrow eosinophils). Significant prognostic differences were noted between the two groups. Of 65 patients with a high eosinophil index, 54 (83 per cent) were cured (45 per cent spontaneously), and, more significant prognostically, none of the splenectomized patients died; whereas of 35 patients with a low eosinophil index, 16 (46 per cent) were cured (23 per cent spontaneously), and, most significant prognostically, 8 (44 per cent) of the splenectomized patients died of their disease despite surgery. The total deaths in this series was 9 per cent of the patients with a high eosinophil index and 43 per cent of the patients with a low index, for an over-all mortality of 21 per cent.—G.E.C.


This case report describes the clinical and laboratory features occurring in a case of thrombocytopenic purpura in a newborn infant. Some 36 such cases have been reported in the literature. The essential features are a generalized purpuric eruption, absence of splenomegaly, a usually positive tourniquet test, and a reduction in blood platelets. There was a general mortality rate of 2.5 per cent in the 36 cases reported in the literature. The prognosis is felt to be better if the mother herself has not had evidence of purpura.—R.B.C.


A fatal case of acute hemolytic anemia, purpura hemorrhagica, thrombocytopenia, mild icterus, central-nervous-system manifestations and platelet thromboses of the arterioles, capillaries, and venules occurring in a 23 year old woman is reported.—P.F.W.


Two cases of purpura fulminans are described, which developed in the course of scarlet fever. One patient died five days after onset of purpura, the second was saved by exchange transfusion. In the first case, Factor V was completely missing in the blood; in the second case, Factor V was also missing, but besides, an antithrombin of the type of heparin-antithrombin was found.

As far as is known, this is the first time that such a deficiency of Factor V has been found in fulminating purpura. In previous observations, a considerably prolonged prothrombin time was repeatedly found, which might also be caused by deficiency of Factor V.—C.M.
ATTACHMENT OF SERUM IRON TO PLASMA PROTEIN.

The iron, which resides in Fraction I, is supplied in this disease. R.B.C. observations also suggested the release of iron from the tissues to the blood when additional metal-combining globulin is supplied to the plasma. In Mediterranean anemia, the blood has an elevated serum iron and no latent or complete reserve iron-binding capacity. The determination of the serum iron level measures the degree of saturation of this protein. In Mediterranean anemia, the blood has an elevated serum iron and no latent or complete reserve iron-binding capacity. In the present study, Fraction IV-7 of plasma was administered to 4 patients with Mediterranean anemia in an effort to see if the serum iron and iron-binding capacity could be altered by its administration. Experimental results showed a consistent rise in the serum iron values in all and a transitory appearance of a latent capacity further to bind iron in 3 of the 4 subjects. The observations also suggested the release of iron from the tissues to the blood when additional metal-combining globulin is supplied in this disease.—R.B.C.

IRON METABOLISM


Hematologic determinations were made in a group of normal pregnant women, receiving an adequate diet, at frequent intervals throughout the pregnancy and during labor and the postpartum period. Particular emphasis is placed on iron metabolism, and determinations of the level of serum iron and iron-binding protein were correlated with the other routine hematologic data. It was shown that the anemia which develops during pregnancy is normocytic and associated with a slight increase in both serum iron and iron-binding protein. That no evidence was obtained to indicate iron deficiency at any stage of the pregnancy in this group of women is of interest, in the light of several other reported studies to the contrary. The authors suggest that the geographic factor may in part account for this variation. Since the blood changes in pregnancy are still poorly understood, there is need for continuation of such studies. Of interest would be data relative to the state of iron metabolism in a similar group of women receiving iron medication throughout the pregnancy. It was also noted that the polymorphonuclear leukocytosis, lymphopenia and decrease in total eosinophil count which develop during pregnancy and become intensified during labor are similar to the changes produced by adrenocortical stimulation.—H.W.B.


Iron is transported in the serum only attached to a β-globulin, called the "metal-combining globulin," which resides in Fraction IV-7 of the plasma. Except under certain conditions this globulin is not completely saturated with iron. The determination of the serum iron level measures the degree of saturation of this protein. In Mediterranean anemia, the blood has an elevated serum iron and no latent or reserve iron-binding capacity. In the present study, Fraction IV-7 of plasma was administered to 4 patients with Mediterranean anemia in an effort to see if the serum iron and iron-binding capacity could be altered by its administration. Experimental results showed a consistent rise in the serum iron values in all and a transitory appearance of a latent capacity further to bind iron in 3 of the 4 subjects. The observations also suggested the release of iron from the tissues to the blood when additional metal-combining globulin is supplied in this disease.—R.B.C.

The Attachment of Serum Iron to Plasma Protein. F. Theodorin, Jr. From the Medical Clinic of the University of Tübingen (Germany). Acta haematologica 3: 310-320, 1950.

Until now, there has been no uniform opinion about whether the plasma iron is bound to the globulin or albumin fraction. The author performed cataphoretic and precipitation studies and postulates the following: (1) The iron absorbed from the intestine is carried in a trivalent albumin complex to the bone marrow. (2) Intravenously injected iron is bound in trivalent form to globulin. The same complex may be found also as destruction product from liberated iron by physiologic or pathologic hemolysis. (3) Ferritin is found only in the intestine wall and in the reticulo-endothelial system as storage iron, but apparently not in the plasma. (4) An insoluble bivalent iron complex which has no intravascular metabolic value, is excreted by the liver and the bile ducts in the intestines. Only from there may it be reabsorbed and start a new circulation in the body.—C.M.
BOOK REVIEWS

BONE MARROW


Transplantation of normal bone marrow to intramedullary sites or to spleen or liver of irradiated host animals was without benefit. In the case of intramedullary transplants, intramedullary bleeding with subsequent organization and fibrosis rather than stimulation of marrow growth was the result. Transplantation of bone and marrow results in death and replacement of the grafted tissue. In transplants to liver and spleen, only residual deposits of fat were demonstrable.

The intravenous injection of bone marrow resulted in multiple heterotopic bone spicules within the pulmonary veins. In animals irradiated and treated with intravenous injections of marrow, the mortality was slightly lower than in the control groups irradiated only and the hemograms were slightly less depressed than in controls in this series.

Transplantation of bone marrow to such regions as the anterior chamber of the eye, the subcutaneous tissue and to the intramuscular and intraperitoneal sites was uniformly unsuccessful. Autologous or homologous marrow transplanted to extramedullary sites resulted in heterotopic bone formation. Transplantation of selected autologous homologous, and heterologous hematopoietic embryonal tissues to irradiated hosts did not alleviate the irradiation toxicity, nor could conclusive evidence of successful transplantation of these tissues be obtained.—W.N.V.

Bone Marrow in Rheumatic Arthritis. Š. Šišaj and E. Kesvánsk. From the Rheumatologic Institute, Trenčianske Teplice, Czechoslovakia. Časop. lék. žes. 88: 1444, 1949.

The bone marrow was studied in 48 patients with rheumatic arthritis, some having a typical progressive course and some with an atypical, and with a rather subacute or acute clinical picture. All patients had marked articular changes and an increased erythrocyte sedimentation rate. Eight patients had enlarged lymphatic glands and splenomegaly; one patient had splenomegaly and a granulocytopenia.

The red marrow exhibited a definite depression in the progressive cases, corresponding to a slight microcytic anemia. In the acute and subacute cases, anemia is rare, appearing however at the beginning of the disease, when it is marked and microcytic. In the bone marrow the red component prevails.

The white component is practically normal and only occasionally is there an increase in younger elements. Plasmatic cells are markedly increased whenever the erythrocyte sedimentation rate is high. The number of the reticulum cells is sometimes increased; this increase could not be correlated either with the course or any other feature of the disease.

In the patient with splenomegaly, enlarged lymphatic glands and a cyclic granulocytopenia (Felty syndrome), there is a depression of the white marrow with a shift to the left with every bout of the granulocytopenia. During remissions with normal white blood cell count there is only a shift to the left. In the remaining patients with splenomegaly, the bone marrow picture is not typical. In 2 patients a definite shift to the left was found.—M.N.

BOOK REVIEWS


As the author says in his introduction, this book does not pretend to be a treatise on hematology, but rather a practical work dealing with hematologic technic and its application to the study of the patient. The first section deals with the various technics for studying the blood cells in their diverse manifestations. Beautiful lithographs in color illustrate the various points made. The second section, devoted to clinical hematology, deals with the application of these technics to various hematologic abnormalities, including anemia, leukemia and the like.