ABSTRACTS

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ANEMIA

THE ANEMIA OF INFECTION. X. THE EFFECT OF INFECTION ON THE ABSORPTION AND STORAGE OF IRON BY THE RAT. C. J. Gubler, G. E. Cartwright and M. M. Wintrobe. From the Department of Medicine, University of Utah College of Medicine, Salt Lake City, Utah. J. Biol. Chem. 184: 563-574, 1950.

Previous studies have shown that in the presence of inflammation there is a hypoferrremia and a decrease in the concentration of the metal-binding serum protein. The greatest proportion of radioactive iron injected intravenously into animals in which inflammation has been produced experimentally is diverted primarily to the liver and spleen rather than to the inflamed tissues. In the experiments reported here, the influence of experimentally induced inflammation in rats on the absorption of iron from the gastro-intestinal tract has been investigated. In the presence of infection there was a decrease in the amount of iron retained.—P.F.W.

THE ANEMIA OF INFECTION. XI. THE EFFECT OF TURPENTINE AND COBALT ON THE ABSORPTION OF IRON BY THE RAT. C. J. Gubler, G. E. Cartwright and M. M. Wintrobe. From the Department of Medicine, University of Utah College of Medicine, Salt Lake City, Utah. J. Biol. Chem. 184: 575-578, 1950.

The influence of turpentine-induced abscesses and of cobalt administration on the absorption of iron was investigated in rats. Turpentine was found to decrease the absorption of iron. This effect could be reversed by the administration of cobalt. Cobalt administration did not significantly affect the absorption of iron in normal rats.—P.F.W.


Repeated injection of colloidal thorium dioxide into dogs resulted in a marked hyperferremia and abolished the hypoferrremia-producing effects of turpentine. As injected colloidal thorium is found largely in macrophagic tissue and as injected colloidal substances result in at least a temporary depression of macrophagic function, the suggestion is made that administration of colloidal thorium resulted in a temporary functional alteration in the reticulo-endothelial system. It is also suggested that possibly the injection of turpentine so enhances or stimulates the functional state of the reticulo-endothelial system that iron is rapidly removed from the serum and hypoferrremia results. Plasma copper levels were essentially unaffected by either turpentine or colloidal thorium injection.—P.F.W.


This is another report on the use of saccharated oxide of iron in the treatment of hypochromic anemia. Since the preparation is much better tolerated than other forms of intravenous iron, an attempt was made to treat 11 patients with hypochromic anemia with from 300 to 900 mg. at a single injection. One patient tolerated 500 mg. without reaction, but the remainder had reactions of varying severity.
It was clear that it is unwise to use such a procedure and that the initial dose should be of the order of 100 mg., increased gradually, if the patient tolerates it well, to 200 or 300 mg. per injection—S.C.


Blood counts on a group of women between 20 and 60 years of age were performed. Twenty per cent had an insulin resistant achlorhydria and 14 per cent an incomplete achlorhydria. No differences as regards hemoglobin, erythrocytes or hematocrit were found. Serum iron was not determined. Achlorhydria is not regarded as an important factor determining the absorption of iron.—J.W.

Heme, Porphyrin and Bile Pigment Metabolism


The role of hematin in hemooglobin and bile pigment metabolism has not been clearly defined. The studies of Fairley suggested that hematin in plasma unites immediately with albumin to form methemalbumin. Injections of hematin followed by measurements of bile pigment excretion have yielded conflicting results. In the experiment reported here of the total amount of N^14-labeled hematin injected intravenously in a day, 18 per cent was converted to bile pigment during the first nine-day period. This rate of conversion indicates that at least a portion of hematin is metabolized much more rapidly than previously suggested. Conversion of hematin to bile pigment supports the view that a portion of bile pigment may originate from the heme or hematin not utilized for hemooglobin formation.—P.F.W.


This is a report of observations on a 14 year old white girl with manifestations of congenital porphyrin and excreting large amounts of uroporphyrin I and coproporphyrin I. Although she had no anemia and there was a normal pattern of red cell survival there was erythroid hyperplasia of the bone marrow and a reticulocytosis of 6 per cent in the peripheral blood. Glycine was specifically utilized in the biologic synthesis of porphyrins related to the etioporphyrin I configuration. At least 31 per cent of the sterobilin in this subject was derived from sources other than the hemoglobin in mature circulating erythrocytes. Among the possible sources of this sterobilin are hemoglobin from young cells destroyed in the bone marrow or soon after entering the circulation, formation of bile pigment directly from pyrroles, rapid degradation of increased quantities of uroporphyrin III and coproporphyrin III as well as myoglobin, respiratory heme pigments and heme which is not utilized for hemooglobin production.—P.F.W.


The utilization of tagged epsilon carbon of dl-lysine by a dog, depleted of both hemoglobin and plasma proteins, was studied for a period of 134 days. The alterations observed in the concentration of C^14 in plasma and in circulating blood cells appeared to provide a valid measurement of the average life span of the red blood cell, which in this experiment was 115 days. Of interest was the evidence which strongly suggested that the globin from the destroyed red cells was not used with any particular preference for the synthesis of new hemoglobin, but rather that the amino acids entering into the formation of new red blood cells were drawn from a larger and more labile body protein store.

The question of the life span of the erythrocyte is still somewhat unsettled. The more recent use of isotopes, particularly those not necessarily reutilized in the formation of new cells, has greatly extended the investigative possibilities and should provide information not only about the normal life span but also as to how this may be altered by various pathologic and physiologic processes.—H.W.B.


A study was conducted to examine the reliability of a porphyrin test of the urine of workers exposed to lead. To 10 cc. of urine in a test tube, 2 drops each of glacial acetic acid and 3 per cent hydrogen peroxide, and 1.5 cc. of ether were added; the stoppered test tube was shaken and the fluorescence in the ether layer noted by use of ultraviolet radiation from a Wood lamp. The authors concluded the method was reliable as a screening test for workers with increased lead absorption.—P.F.W.

THE INFLUENCE OF BILE ACIDS ON THE DIAZO-REACTION OF BILIRUBIN. Hans Adolf Kuhn.

From the Department of Medicine, University of Freiburg (Germany). Ztschr. f. d. ges. exper. Med. 115: 371-385, 1950.

The author approached the problem experimentally, with regard to the new concept of Baumgärtel and Kuhn, who claim chemical identity of direct and indirect bilirubin, and think that only the presence of bile acids is responsible for the difference in reaction to the Diazoreagent. In vitro, under the influence of cholic acid, dehydrocholic acid and desoxycholic acid, he could find acceleration and intensification of the Diazoreaction. The concentrations necessary for eliciting this phenomenon were higher, however, than those occurring (usually) in vivo. If the concept of the above-mentioned workers is correct, we have to assume the presence in vivo of factors enhancing the reaction, or lack of inhibiting agents (proteins).—C.M.

LEUKOCYTIC DISEASES AND ABNORMALITIES

LYMPHOPENIA IN THE COURSE OF ACUTE PanCREATIC NECROSIS. K. Herfort and V. Letošák.

From the First Medical Clinic, Charles University, Prague, Czechoslovakia. Casop. lék. čes. 88: 1006, 1949.

In 38 cases of acute pancreatic necrosis observed between 1942 and 1949, 31 of which were verified objectively, lymphopenia was a constant finding. In 23 cases (63.1 per cent) lymphopenia was absolute, in 14 cases (36.9 per cent) it was relative. In 8 cases the blood picture was followed during the course of acute pancreatic necrosis up to the return to the normal lymphocyte count. In 5 of the 8 cases there was a slow return of the lymphopenia to normal values without these ever having been exceeded either during the course of the disease or during convalescence. The more extensive the process, the slower was the disappearance of lymphopenia. In 3 cases of slight acute necrosis (37.5 per cent) the original lymphopenia was replaced by lymphocytosis. The lymphocytes returned only later to normal values. According to previous experience, the occurrence of lymphocytosis in the course of acute pancreatitis is a sign of good prognosis. Based on these findings in 38 cases of acute pancreatic necrosis, 33 of which were verified objectively, the authors consider lymphopenia found within forty-eight hours of the onset to be a valuable diagnostic sign.—M.N.

REILLY'S GRANULATION ANOMALY OF LEUKOCYTES IN FAMILIAL DISOSTOTIC NANOISM OF PEINZL-HERL'S VARIETY (GARGOYLISM). Herbert Brugsch.


Reilly, in 1941, reported unusually pronounced, coarse granulation of the leukocytes in gargoylism. This author discusses a case recently observed by himself, and two cases observed in 1941. He discusses whether or not these findings are identical with those observed by Alder in 1937 of an anomaly of granulocytes in 2 healthy siblings who showed bone disease later on. Alder commented on this at the meeting of Swiss hematologists in 1950, and assumed that the abnormal granulation is not specific and may occur in various disorders of development.—C.M.

THE APPEARANCE OF LEUKOCYTIC DISINTEGRATION FORMS IN THE PERIPHERAL CAPILLARY BLOOD IN CASES OF INFLAMMATORY DISEASES. E. Koch and P. Lubbers.

From the Medical Department of the City Hospital, South Lübeck (Germany). Deutsche Arch. klin. Med. 196: 768-778, 1949.

With reference to the investigations of Undritz, Heilmeyer, Motius and Tischendorf, the authors report 106 cases of inflammatory diseases in which they searched for disintegration forms of leukocytes in the peripheral blood. Accordingly these cells, found in many inflammatory affections, show but a transient appearance; a specific correlation with age, sex, temperature, leukocytosis, and treatment...
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with penicillin or sulfonamides could not be found. There seems to be, however, a higher incidence in special irritative states of the reticulo-endothelial system, such as subacute bacterial endocarditis, and acute polyarthritis.—C.M.

CONTRIBUTION TO THE PROBLEM OF TUBERCULOSIS OF LYMPHATIC GLANDS FROM A HEMATOLOGIC POINT OF VIEW. F. Hermanský. From the Medical Department, State Hospital, Motol, Czechoslovakia. Časop. lék. čes. 88: 1361, 1949.

The value of glandular puncture in tuberculous lymphadenopathy is discussed on the basis of 16 cases; in 13 of them the diagnosis was made possible by this diagnostic procedure, while in the remaining 3 a glandular biopsy was necessary to establish the diagnosis. It is suggested that the epithelioid cells originate from the large lymphoreticular elements (Stahel's "round" cells). Microphotographs are presented to confirm this view.

Five patients with a very chronic and benign course are presented in detail. Tuberculosis with caseation was present in numerous glands and a simultaneous enlargement of liver, spleen or both of these organs suggested a blood disease. Lymphopenia was the most frequent finding in the peripheral blood and in a single case normocytic normochromic anemia was found. Leitner's generalized caseating tuberculous of lymphatic (and hemopoietic) tissue is discussed and the importance of the knowledge of such atypical forms of tuberculosis by the hematologist is stressed. The explanation of the hepatosplenomegaly in these cases meets with great diagnostic difficulties.

Therapeutic effects of chlorethylamines in 4 of these patients are discussed; it was possible to achieve a regression of the glands and a clinical remission of variable length. However, the difficulty of evaluating these effects in a chronic disease with such a variable course is stressed.—M.N.


The authors publish a case representing a syndrome characterized by the presence of symptoms from the central nervous system with paresis of extremities and increased protein content of the spinal fluid, together with slight lymphocytosis. The neurologic symptoms seem to have been very slight. The clinical picture was dominated by a long-drawn tendency to epistaxis, intra-ocular bleeding and chronic anemia with slight swelling of lymph nodes, marked increased sedimentation rate, bone marrow picture showing lymphocytosis with atypical small cells. The first diagnosis was lymphatic leukemia. The patient was followed during eleven years. His serum globulin was increased and electrophoretic examination showed the presence of 44 per cent of a B-globulin. Ultracentrifugation revealed the same percentage of a high-molecular globulin of the type described by Pedersen and Waldenström as macroglobulin. The clinical picture is a classic example of the macroglobulinemia described and studied by these authors. The patient died without presenting any radiologic evidence of multiple myeloma. The autopsy showed normal structure of the bones. There were no tumors in the osseous system. Histologically, the bone marrow was rich in cells, especially in lymphocytes.—J.W.

INDUCTION OF THE LUPUS ERYTHEMATOSUS ("L.E.") CELL IN VITRO IN PERIPHERAL BLOOD. R. H. Hamburger. From the Department of Internal Medicine, Yale University School of Medicine, New Haven, Conn. Yale J. Biol. & Med. 22: 407-410, 1950.

The "L.E." cell was found in small numbers in the peripheral blood of patients with lupus erythematosus and in the buffy coat of normal blood incubated with oxalated lupus erythematosus plasma. The author suggests that this may be useful as a diagnostic test, especially since the leukopenia of the patient frequently makes an adequate buffy layer difficult to obtain.—W.N.V.

HEMORRHAGIC DISEASES


Senile purpura is a clinical entity, affecting both sexes equally, and unrelated to ascorbic acid deficiency or to any systemic disorder or the presence of any blood dyscrasia.
Capillary fragility tests were found to be within the normal range in the patients investigated. The lesions are characteristically found on the extensor surface and radial border of the forearm and on the back of the hand but do not extend onto the fingers. The individual lesions are large and irregularly shaped, dark purple, and possess a clean-cut margin in contrast to the ordinary bruise. They are considered to be due to the rupture of dilated venules as a result of minor external trauma in areas in which the skin shows marked senile degenerative changes. The vasculature of this area is thought to be inadequately supported. Degenerate collagen fibers are found histologically in affected areas but it was not possible to demonstrate any increase in true elastic tissue.—W.N.V.

HEMOPHILIA IN TWINS. A. J. Quick and J. P. Conway. From the Department of Biochemistry, Marquette University School of Medicine and Milwaukee Children's Hospital, Milwaukee, Wis. Am. J. Med. 7: 841-843, 1949.

The case history of a set of presumably identical twins is presented, in which one twin is a typical hemophiliac and the other entirely normal. The authors are unable to explain this paradoxical situation.

This unusual observation is of interest and serves to stress the inadequacy of our present knowledge of the genetic aspects of this disease. It is to be hoped that investigations, perhaps those being made on canine hemophilia, may in time shed light on situations such as this.—H.W.B.

A NEW HEMOPHILIA-LIKE HEREDITARY HEMORRHAGIC DIATHESIS WITH DISTURBANCE OF THE FIRST COAGULATION PHASE. F. Koller, G. Krüsi and P. Luchsinger. From the Department of Medicine, University of Zurich (Switzerland). Arch. der Julius Klaus-Stiftung für Vererbungsforschung 25: 16-21, 1950.

The authors describe a hemorrhagic diathesis, with disturbance of the first coagulation phase, in a family from Southern Austria (Tyrol). The heredity is very similar to that of hemophilia (manifestation only in male descendants and transmission by "healthy" females). Quick's hemophilia test and the retraction test of Fonio with a plasma-platelet mixture were negative. The plasma prothrombin was normal but the serum prothrombin consumption test showed a disturbance of thrombin formation. The disease is to be differentiated from hemophilia and also from parahemophilia (lack of factor V) because of normal plasma prothrombin concentration.—C.M.


Although the majority of authors sustain that the histopathology of the spleen in thrombocytopenic purpura offers no characteristic data, Polak and Bomchil studied the reticulo-histiocytic sector of the spleen in 4 cases. Three of these were of chronic, and one of acute evolution. To carry out this study, the authors used the specific technic of Rio Ortega.

It was found that the malphigian follicles were totally or nearly deprived of their reticulo-endothelial components, while in the extrafollicular regions there was a hyperplasia of reticulo-endothelial cells. Several photomicrographs are shown illustrating these findings. The authors admit the difficulty in interpreting the facts observed.—R.M.S.

BLOOD COAGULATION

CONGENITAL HYPOTHROMBINEMIA. A CASE STUDY WITH PARTICULAR REFERENCE TO THE ROLE OF NON-PROTHROMBIN FACTORS IN THE CONVERSION OF PROTHROMBIN. G. Landwehr, H. Lang and B. Alexander. From the Medical Research Laboratory, Beth Israel Hospital, the Department of Medicine, Harvard Medical School and the Children's Medical Center, Boston, Mass. Am. J. Med. 8: 255-259, 1950.

A case of a 5 week old infant with true hypoprophrombinemia, probably congenital, is presented. That the deficiency was one of prothrombin and not accessory prothrombin factors is very adequately demonstrated.

It was found that the disappearance of injected prothrombin from the patient's plasma was far more rapid than the in vitro deterioration of prothrombin in plasma and also than that observed in heavily dicumarolized dogs. The authors, therefore, present the interesting possibilities that normally there is a rapid consumption and regeneration of prothrombin, and secondly, whenever prothrombin synthesis...
in the liver is interfered with; i.e., by dicumarol, there may be other body stores from which prothrombin may be drawn.

Data is presented to show that BaSO₄ removed one of the accessory prothrombin plasma constituents, possibly Factor V of Owren, from this patient’s plasma. If this is true of normal plasma, the use of BaSO₄ plasma as a diluent in one-stage prothrombin determinations is necessarily limited.

This case is of particular interest since, with the discovery of nori-prothrombin factors important in prothrombin conversion, some doubt has been cast on the existence of idiopathic hypoprothrombinemia as such.—H.W.B.

LABILE FACTOR OF PROTHROMBIN CONVERSION: ITS CONSUMPTION IN NORMAL AND ABNORMAL BLOOD COAGULATION. B. Alexander, G. Landerbr and R. Goldstein. From the Medical Research Laboratories, Beth Israel Hospital, and the Department of Medicine, Harvard Medical School, Boston, Mass. Federation Proc. 9: 4, 1950.

“Labile factor” (L.F.), a substance necessary for the rapid conversion of prothrombin to thrombin, was measured by its ability to rectify the retarded prothrombin conversion of aged plasma devoid of L.F. It was found that normal serum contains much less L.F. than normal plasma, and that serum from blood whose coagulation had been accelerated by the addition of thromboplastin had even less L.F. than normal serum. Conversely, it was found that the sera in hemophilia and in thrombocytopenic purpura contained a large amount of L.F.; and that dicumarolized blood yielded serum which was especially rich in L.F.

The conclusion was therefore reached that L.F. is consumed in relation to the amount and the velocity of prothrombin conversion, and that L.F. is not a catalyst but an actual reagent used in the process of prothrombin conversion.—S.E.


The deterioration of solutions of thromboplastin was studied in regard to the effects of contamination, oxidation, and denaturation. It was found that none of these three factors could be implicated causally in the deterioration of the solutions: such deterioration did not depend upon the presence or absence of bacterial contamination, oxidation, and denaturation.

It was suggested that deterioration is perhaps actually due to the presence of a naturally occurring antithromboplastin in the solutions.—S.E.


A method for measuring the prothrombin consumption in venous blood is given and its importance for the diagnosis of hemorrhagic disorders is discussed. It was found to be a much more sensitive test than the determination of the clotting time. This does not reach pathologic values until below 30 per cent of prothrombin consumption. An isolated vascular defect as cause of a hemorrhagic syndrome may be diagnosed with the aid of this method.—J.W.


In a group of 13 subjects treated with nitrogen mustard (TS 160 -HN₃ and TS 160/4 - HN₃), administered in maximum doses up to 40 mg, divided in four doses of 10 mg per day and two doses of 15 mg, given within twelve hours, changes of clotting time were studied. In 9 of these patients clotting time of recalcified oxalate plasma (recalcification time) was estimated simultaneously. Variations of clotting time, estimated by the Lee-White method, were rather irregular and small and they never exceeded 15 minutes. The recalcification time was increased and it was definitely prolonged in 3 patients. However, manifestations of a slight hemorrhagic diathesis appeared in a single patient, who also had a thrombocytopenia; it occurred at a time when the clotting and recalcification times were already returning towards normality. It is concluded that current treatment with chlorethylamines of Czechoslovak origin does
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not lead to marked disturbances of the clotting mechanism which could provoke hemorrhagic diathesis.—M.N.

STUDY OF THE COAGULATION MECHANISM OF PLEURAL BLOOD IN HEMOPNEUMOTHORAX. S. W. Cosgriff.
From the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Presbyterian Hospital, New York, N. Y. Am. J. Med. 8: 57-61, 1950.
The reason for the fluidity of blood obtained from the pleural cavity by thoracentesis or at autopsy has never been adequately explained, although it has been suggested that the intact mesothelial pleural membranes may behave like untraumatized blood vessel endothelium or may even secrete an anticoagulant at the site of hemorrhage.

Observations on the pleural and venous blood coagulation mechanisms in a patient with hemopneumothorax are reported. There was no defect in the clotting mechanism of the peripheral blood. The specimens of pleural blood, which failed to clot after thoracentesis, were comparable in hemoglobin content, hematocrit and plasma specific gravity to the circulating venous blood. The pleural blood was, however, completely devoid of the essential coagulation constituents: prothrombin, thrombin and fibrinogen. Anticoagulant activity was not demonstrated in any form. It was concluded, therefore, that coagulation had occurred prior to thoracentesis and had been followed either by normal fibrinolysis or by defibrination with deposition of fibrin on the lung surfaces. Excessive fibrinolytic activity was not detected in the pleural blood.—H.W.B.

BLOOD GROUPS AND TRANSFUSION

MULTIPLE ANTIBODY RESPONSE TO REPEATED TRANSFUSIONS. R. H. Malone and J. Cowan.

A patient with hemolytic anemia was transfused in the course of a month with blood from 24 different donors. It became increasingly difficult to find cells which were compatible, her own group being A, NNS, iP, Rhi, (CDe/CDe); Lewis, Lutheran, Kell and Willis negative. Detailed laboratory investigation showed the presence of anti-E, anti-M (active at 37 C.) and a cold antibody, anti-P, in her serum.—S.C.


A woman suffering from anemia associated with osteosclerosis, was treated by twenty transfusions over the course of twelve years. She had had a splenectomy for splenic enlargement, before anemia developed. She had two mild hemolytic reactions and two febrile reactions to the blood transfusions. Investigation of her serum showed the presence of antibodies to antigens which she lacked in five blood group systems.

Anti-A and anti-B were present, the patient being group O. Anti-N and anti-Leb could be demonstrated in the cold and were probably naturally occurring antibodies. The remainder, anti-S, anti-E, anti-C*, anti-K and anti-Lea, were probably all immune antibodies.—S.C.

AN UNUSUAL RH CHROMOSOME COMBINATION. Ruth Sanger.

A family is described in which the very rare Rh chromosome C*D*e has been recognized in the genotype C*D*e/cde.—S.C.

ANTI-M ANTIBODIES AND LATENT N-ANTIGEN IN MAN. L.A.M. van der Spek and L. de Kromme.

A woman whose serum contains anti-M agglutinins is described and the serologic properties of her blood are analyzed. These antibodies were considered to have been present constitutionally as there
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were no indications of active immunization of the mother. The observation shows once more the absolute necessity to make a cross-testing and not be content with ordinary blood grouping.

The 'significance of iso- and autoimmunization for pathology' is discussed by the same authors in a further paper on page 455 in the same issue.—J.W.


Sixteen cases showing neurologic sequelae of severe jaundice associated with erythroblastosis fetalis are discussed and the neurologic features of the disease reviewed and analyzed in 63 cases selected from the literature. The main features consist of severe neonatal illness with jaundice, opisthotonos, and muscular twitching. After a latent period of a few weeks or months, stiffness or hypotonia and retardation may be evident. Of the older patients, over 80 per cent show athetosis, chorea, or choreo-athetosis, and in 30 per cent of the authors' cases deafness was found. Mental defect is often but not always present.—W.N.V.


Transfusion reactions which occurred in certain patients who were given plasma transfusions, did not occur when the same patients were given only washed red blood cells devoid of plasma. The authors looked for involvement of the coagulation mechanism in the production of the reactions, and describe the following findings: (1) The platelet count fell just before the chill; (2) the coagulation time was reduced; (3) clot retraction became incomplete; (4) the serum coagulation accelerator activity fell during the reaction, then rose to excessively high levels; (5) the plasma 'labile factor' content increased; (6) the fibrinogen level fell; (7) fibrinolysis increased markedly.

The authors suggest that perhaps platelet embolization is the initiating mechanism in the clinical symptomatology of these plasma reactions.—S.E.


An apparatus for intra-arterial transfusion is described. The authors feel there are advantages to intra-arterial transfusion in shock including a rapid return to normal blood pressure and the early reestablishment of renal function.—P.F.W.


This paper reports that when the blood of dogs and rabbits is mixed with strong solutions of NaCl and the formed elements are subsequently resuspended in normal saline, a depressor substance is released from the cells. This depressor substance has not been completely identified but the authors find it is not potassium ion, acetylcholine, or histamine. It resembles the adenosine-like compounds released from mammalian erythrocytes under various conditions. The authors think that the release of this depressor substance accounts, at least in part, for the potentiation of hypotensive effects of hypertonic solutions resulting from their admixture with homologous blood.—R.C.C.