ERYTHROCYTE MORPHOLOGY


In the peripheral blood of patients with uremia, carcinoma of the stomach, and bleeding peptic ulcer, peculiar poikilocytes were frequently observed which the authors have designated as "burr" cells. These peculiar red cells measure about 7.5 micra or less in diameter and have one or more large spiny projections along their peripheries. "Burr" cells occurred in the bloods of 73 per cent of the patients with uremia, 68 per cent of the patients with carcinoma of the stomach, and 54 per cent of the patients with bleeding peptic ulcers. They were rarely observed in normal blood but did occur occasionally in the blood of patients with diseases other than those mentioned above.—G.E.C.

OBSERVATIONS ON ACHROMOCYTES. U. Haenen. From the Medical Outpatient Department, University of Berne, Switzerland. Schweiz. med. Wchnschr. 79: 843-844, 1949.

Heilmeyer has shown that it is possible with the 12-hour staining process with Giemsa solution to stain blood cells that cannot be found with an ordinary staining method; since they contain vital staining granules he termed them "achromoreticulocytes." It is very easy to examine them in the dark field. In the author's opinion, the achromocytes are hemoglobin-free erythrocytes which are composed only of stroma and membrane. They contain merely rests of basophilic protoplasmic substances in the form of substantia granulofilamentosa. This conception is supported by the fact that experimental hemolysis may cause similar pictures in normal erythrocytes in the dark field.—C.M.

DARK FIELD INVESTIGATIONS ON RETICULOCYTES AND ERYTHROCYTES WITH BASOPHILIC PUNCTATION. G. Riva. From the Medical Outpatient Department of the University of Berne, Switzerland. Schweiz. med. Wchnschr. 79: 840-841, 1949.

Blood-smears were examined in the dark field, using the method of Nizet. Cells with basophilic punctations and reticulocytes can be counted very easily with this method. With the dark field, the author found more cells of this type than with the ordinary brilliant cresyl blue method. Normal persons show figures up to 79 per cent. It is the author's opinion that erythrocytes with basophilic punctation and reticulocytes are identical. Cells with basophilic punctation show merely coarser granulation than reticulocytes. In cases of lead poisoning, there seems to occur an increase especially of the coarsely granulated reticulocytes.—C.M.


Investigations of 4 healthy persons by liver puncture showed an increase of the volume of the erythrocytes and a decrease of the osmotic resistance in hepatic blood. The author found the same change in 36 cases of liver disease with jaundice. In addition to the normal peak in the Price-Jones curve, there was a peak of microcytes which could not be found in the peripheral blood. The author assumes from these
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findings a blood-disintegration in the liver via fragmentation (formation of schistocytes), similar to that found in the peripheral blood in Cooley’s anemia.—C.M.

IRON AND COPPER METABOLISM


The plasma iron level was estimated by the electrophotometric method, the color complex obtained with potassium thiocyanate being extracted into a mixture of ether and butanol. In acute epidemic hepatitis, the plasma-iron level was raised; this increase began a few days after the onset of the illness and remained elevated in the period of convalescence even at the time when the patient was clinically perfectly well. In obstructive jaundice, the plasma-iron level was only slightly elevated; in cirrhosis, the plasma-iron level was normal (in chronic forms), elevated (in progressive forms) or decreased. In liver carcinoma, the plasma-iron level was low (with one exception). No direct relation has been found between the plasma-iron level and bilirubin or any other liver function test. The increase of the plasma-iron level seems to be related to the degree of damage to the liver parenchyma and might be an indication of the degree of repair present in acute hepatitis.—M.N.


A method for the determination of copper in serum or plasma, red blood cells, urine and feces is presented; it is based on the colorimetric determination of this element by means of sodium diethylthiocarbamate. Values of copper in serum, red blood cells, urine and feces in normal males and females are given. In 166 normal individuals, the plasma copper in males was 101.4 ± 18.57 per cent, in females 116.6 ± 19.87 per cent. In 18 normal individuals, the copper content of the red blood cells in males was 73.1 ± 3.77 per cent, in females 75.4 ± 3.67 per cent. In 28 normal individuals, the copper content of urine/24 hours in males was 351 ± 4.77 per cent, in females 336 ± 4.47 per cent. In 10 normal individuals, the copper content of feces/24 hours was 2.68 ± 0.5 mg. per cent. No significant difference in copper content of plasma and urine was noted during the day and in menstruation. In 2 cases a slight elevation of copper content of the red blood cells was found in the evening.—M.N.


This study was made to determine the effects of sterile inflammation (induced by repeated subcutaneous turpentine injections over a two-week period) on the utilization of intravenous radioactive iron in normal dogs and in anemic, iron-deficient dogs on a diet low in iron. The normal dogs developed a moderate anemia associated with a fall in plasma iron concentration and marked impairment in the uptake of radioactive iron by the red cells. The anemic dogs, on the other hand, showed neither an increase in degree of anemia nor an alteration in ability to utilize the injected iron. These observations and those of other investigators have demonstrated quite clearly that iron is not the limiting factor in the impaired hemoglobin formation associated with the anemia of infection. That the erythropoietic stimulus of depletion of body iron stores is sufficiently great to overcome this marrow inhibition is of interest. It has already been shown that cobalt is capable of stimulating the rate of erythropoiesis in humans with chronic infections, and it is quite likely that other ways to alter the state of marrow activity will be found. The actual reason for erythropoietic depression in infection, however, remains a mystery.—H.W.B.


Accidental ferrous sulfate poisoning was observed in 6 children in Dundee over a period of five years, five months. Two of the cases were fatal. All cases were due to ingestion of about 10 to 39 tablets of ferrous sulfate compound, B.P.C.
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There was an early onset of pallor, drowsiness and vomiting, in 4 cases, hematemesis. In 3, there followed a deceptive period of apparent improvement with subsequent sudden fatal collapse.

Treatment was designed to eliminate the iron while still in pill form by emesis and gastric lavage. Use of sodium bicarbonate for lavage may convert the iron to the less irritant ferrous carbonate. Bismuth is suggested to protect the mucosal lesions.

Emphasis is laid on the prevention of the condition by warning parents of the danger, and securing safe packing and storing of the pills.—S.C.

VITAMIN B₁₂


A detailed description is given of the methods used by Lester Smith and his co-workers in the isolation of the red crystalline anti-pernicious anemia factor from liver. A second red factor, active both clinically and microbiologically, has been isolated but not crystallized.

The authors relied heavily on physical procedures such as adsorption and partition chromatography rather than chemical methods. The results of various physical and chemical analyses are detailed, including crystallography, x-ray crystallography, measurement of refractive indices, absorption spectrum, optical rotation, electrometric titration, electrical conductivity, mobility and polarography.

From the chemical analysis the formula C₉₁₂H₁₄₀₂₀�₁₄P⁺₁₀⁻ Co is suggested very tentatively. The presence of 4.0 per cent cobalt indicates a molecular weight of 1500. The values obtained with x-ray crystallography of 1360 to 1575 are in agreement with this.

The crystalline factor is destroyed slowly by cold dilute acid and alkali, by light and by strong oxidizing or reducing agents.

Attempts to produce labeled anti-pernicious anemia factor with radioactive cobalt or phosphorus by direct chemical exchange have not been successful but a biologic synthesis using radioactive cobalt is now being attempted.

(This article cannot be adequately summarized and should be read in the original.)—S.C.


No difference in microbiologic activity for L. derner or L. leichmannii could be shown as a result of incubation of minced lean beef at 37 C. for two and one-half hours with normal gastric juice, as compared with similar treatment with water or boiled gastric juice.

The microbiologic activity for L. leichmannii in the contents of the alimentary canal at various levels in a man, a horse and a sheep was assayed. Both total activity and alkali stable activity were measured in an attempt to eliminate the effect of interference with desoxy-ribosides. In the horse and sheep the vitamin B₁₂ content of the diet was known and it was evident that considerable synthesis must occur in the intestine. In the ruminant, this occurs in both upper and lower parts of the digestive tract; in the horse and man, largely in the cecum.

From the contents of the horse’s intestine, red fractions were isolated which showed clinical activity in 3 cases of pernicious anemia.

It is suggested that the normal requirements of vitamin B₁₂ are met by bacterial synthesis in the colon and that an absorptive defect in pernicious anemia may be more extensive in the alimentary tract than has been previously thought.—S.C.

BLOOD GROUPS AND BLOOD TRANSFUSIONS


A hemophilic who had received previous transfusions had a hemolytic transfusion reaction. His serum
was found to contain two immune antibodies—anti-Rh and another antibody detected with certainty only by the indirect Coombs test. This antibody is independent of all previously known blood groups. It is proposed that the antibody system should be called the Duffy system and that the antigen with which the serum reacts be called Fy*. An allele, Fyb, is postulated, the approximate gene frequencies being $Fy^* = 0.41; Fyb = 0.59.$

THREE CASES OF SYMPLIS TRANSMITTED BY BLOOD TRANSFUSION. L. DONNER. From the Second Medical Clinic, Charles University, Prague. Časop. lék. čes. 85: 885, 1949.

Three cases of syphilitic infection due to blood transfusion are described. Two patients were infected by a donor with latent syphilis and negative serologic blood test. Within one year this infected donor donated 5480 cc. blood to 18 different recipients; 12 of them were examined for syphilis, 10 being negative, and 2 presenting signs of recent syphilitic infection. Another patient was infected by a donor who had recently been treated for early syphilis and who at the time of transfusion was in the prehumoral seronegative state.

The reliability of serologic tests for the prevention or transmission of syphilitic infection is discussed. Periodic physical examination of the donors immediately before drawing the blood is suggested. The advantage of banked blood stored for more than three days is stressed.—M.N.


A report of five years' experience on the actual knowledge of diagnosis, prevention and treatment of erythroblastosis is presented. In Czech population, the occurrence of Rh negative persons has been noted in 15.3 per cent. From 716 families with positive Rh/Hr isoimmunization, 665 women were Rh negative, i.e., 92.9 per cent; in 7.1 per cent, isoimmunization was proved in Rh positive women. Exchange transfusion, started at the beginning of 1947 for the treatment of erythroblastosis, has been performed in 35 cases, of which 28 were successful. The prevention of erythroblastosis fetalis is in this country largely based on the antenatal investigation in dispensaries; it has been proved statistically, that erythroblastosis fetalis is a more frequent cause of neonatal mortality than syphilis.—M.N.


The frequencies of the ABO blood groups in a sample of both Rh-positive and Rh-negative child-bearing women varied slightly and insignificantly. These data indicate that there is no relationship between the incidence of Rh-positive and Rh-negative types and the ABO blood groups. The incidences of ABO blood groups in this sample, segregated according to race (white and Negro), are similar to those previously published. The numbers of primigravid and multigravid women in this sample were found to be approximately equal. The incidence of ABO blood group compatibility between mother and child was determined. Of the total group of Rh-negative women, only 4.3 per cent bore infants suffering from hemolytic disease of the newborn. Of the group of Rh-negative women who demonstrated antepartum sensitization, 45.4 per cent bore infants afflicted with hemolytic disease of the newborn. Fifty per cent of the afflicted infants died of hemolytic disease of the newborn.—G.E.C.


Immunologic data from 138 cases of sensitized Rh-negative women were subjected to analysis. Five antepartum maternal factors were evaluated with regard to their relative importance in the antepartum prediction of hemolytic disease of the newborn. These are, in the order of significance: (a) definitely higher amounts of maternal Rh antibodies in 'albumin' diluent than in 'saline'; (b) appreciable amounts of Rh antibodies (3+ or over) in the maternal serum during the last month before delivery; (c) antibodies appearing earlier than ten weeks before the expected date of confinement; (d) the presence of Rh antibodies on all successive testings after the first appearance; and (e) the presence of ABO
compatibility between the mother and child. Although there were instances of normal Rh-positive infants being born to mothers who showed the presence of all 5 factors, there was no instance of an afflicted child being born to a mother who lacked two or more of the factors.—G.E.C.

HYPERSPLENISM


The author discusses the normal and pathologic physiology of the spleen and reviews the evidence to support his concept of hypersplenism; namely, the withdrawal and hypersequestration of the cellular elements in the circulating blood by an unstable and pathologically hyper-reactive spleen; a normal bone marrow which responds by compensatory hyperplasia and accelerated delivery of marrow cells; and the persistence of a low level of circulating cellular elements due to their increased withdrawal by the spleen. His hypothesis of the spleen as the sole pathologic focus in primary hypersplenic states is based on supravital studies of marrow and spleen; study, during surgical exploration, of the cellular components of the blood entering and leaving the spleen before and after adrenalin; and the repeated observation of differential splenic selectivity in the same individual. Splenectomy, its contraindications and failures, and secondary hypersplenism are also discussed.

The author’s concept of the pathogenesis of hypersplenism is by no means universally accepted. Equally as convincing is the evidence presented by the investigators who postulate marrow inhibition by an humoral agent elaborated by the spleen. The spleen remains a poorly understood organ, and there is obvious need for continued study of its complex interrelationship with the peripheral blood and bone marrow.—H.W.B.

APLASTIC ANEMIAS IN CHILDHOOD. REPORT OF A PRIMARY IDIOPATHIC REFRACTORY TYPE, WITH SPLENECTOMY, IN AN ELEVEN YEAR OLD GIRL. A. F. Abt. From the Sarah Morris Hospital for Children, Michael Reese Hospital, and the Department of Pediatrics, Northwestern University Medical School, Chicago, Ill. Am. J. Dis. Child. 78: 516-536, 1949.

The clinical and pathologic findings in an 11 year old Negro girl with an unusual case of idiopathic aplastic anemia are presented. Salient features were premature birth, probable anemia in the mother at time of birth, moderate anemia and leukopenia during an episode of otitis media at the age of 6 years, and the observation five years later of pancytopenia and splenomegaly. The sternal marrow was hypoplastic and later attempts to obtain marrow by aspiration and biopsy were unsuccessful. Splenectomy was performed after the epinephrine test failed to indicate extramedullary hematopoiesis in the spleen. Histologic examination of the spleen revealed follicular hyperplasia and erythrophagocytosis. Clinical improvement ensued but was maintained for only six months when rapid deterioration and death occurred. There was no significant change in the anemia and thrombocytopenia after splenectomy, although fewer transfusions were required. Of interest was the marrow specimen obtained from the iliac crest prior to death, which showed almost normal composition although diminished megakaryocytosis. Autopsy findings were multiple hemorrhages, fatty degeneration of the liver with hemosiderosis, marrow hypoplasia with primarily immature cells and paucity of megakaryocytes, and no evidence of leukemia.

The long history of anemia and leukopenia, the enlargement and histologic appearance of the spleen, the temporary clinical improvement following splenectomy, and the variability in marrow cellularity at different sites make one wonder whether this case was not one of hypersplenism in which marrow hypoplasia rather than hyperplasia was observed because of the exhaustion of a (?) constitutionally inadequate marrow.—H.W.B.


Three cases with the syndrome of splenomegaly, erythroblastosis and myelosclerosis are described. The author is of the opinion that he deals with a syndrome where the symptoms may vary. He believes that the reticulo-endothelial system is primary in eliciting the changes. Of these 3 cases, one showed a transition from splenic erythroblastosis to erythroleukocytosis. The second case is described as a pure one and the third case was characterized by a high number of megakaryocytes.—C.M.
HYPERSPLENISM WITH ARTHRITIS. W. R. Gauld. From the Aberdeen Royal Infirmary, Aberdeen, Scotland.
This is a full case report of a woman, aged 38, with mild rheumatoid arthritis of about thirteen years' duration, accompanied by gross splenomegaly, hypochromic anemia, severe neutropenia and mild thrombocytopenia. She was treated with chemotherapy for complicating skin infection and by transfusion and finally underwent splenectomy. The blood picture returned to normal and was still normal seventeen months after operation. There was a temporary relapse of the arthritis within a year of the splenectomy.—S.C.

GAUCHER'S DISEASE AMELIORATED BY SPLENECTOMY. Z. Gepel. From the Department of Surgery, State Hospital, Prague. Časop. lěk. čes. III: 536, 1949.
A case of Gaucher's disease is described. The patient was a boy, aged 5, with delayed physical development, marked cachexia, splenomegaly, enlargement of the liver, hypochromic anemia with leukopenia and concomitant tuberculosis of the peritoneum with slight ascites. Osseous changes and pigmentation of the skin were not manifest. Following splenectomy (spleen 1930 Gm., with milliary nodules in the capsule), a rapid improvement with increase of weight and satisfactory improvement of the blood picture took place. Six months after splenectomy, the patient became a healthy and vigorous child. The results of splenectomy and prognosis of this disease are discussed.—M.N.

HEMORRHAGIC DISEASES

Blood coagulation studies were carried out on a group of patients with a variety of hemorrhagic disorders. Patients were classified into two groups: those with and those without evident hemocytologic disorders. Abnormalities in the first group were confined chiefly to an increased protamine titration and prolongation of whole blood clotting time as determined by a special technic. The patients comprising the second group had severe marrow disturbances due either to their primary disease or therapy. In most of this group thrombocytopenia and vascular disorders were present as well as an increased protamine titration and prolonged clotting time. The clinical and laboratory response of these patients to the administration of protamine sulfate and toluidine blue is discussed.

Observations are presented to show that whereas the clotting defect was frequently found in association with thrombocytopenia and vascular abnormalities, it bore no direct relationship to them, and that clinical bleeding was influenced by protamine sulfate or toluidine blue administration only to the extent that this clotting abnormality contributed to the over all hemorrhagic picture.

Considerable disagreement has arisen among those studying this problem with respect to the nature of this clotting abnormality and its relationship to human heparin, whether such an anticoagulant actually exists, and to the clinical effectiveness of these two antihemorrhagic agents. This aspect of blood coagulation is of sufficient interest and concern, however, to warrant continued investigation. Efforts at the present time are being directed toward refinement of tests and control of the many variables (e.g., use of platelet-free plasma, oxalated plasma, etc.).—H.W.B.

Blood studies (protamine titration, whole blood clotting time, prothrombin time, observation for lysis of clots, clot retraction and platelet count) were carried out during the menstrual period, on the day of the heaviest flow, and intermenstrually in 43 patients with menorrhagia. Twenty-three normal women and 36 normal men were used as controls. The most significant abnormality was that revealed by the protamine titration. While an increased protamine titration was detected in some of the members of the three groups in each of the various periods tested, this abnormality was more pronounced and more commonly observed during the menorrhagic periods than during menstruation in normal women. Many of the women with menorrhagia and an increased protamine titration responded to a single injection of...
prolamine sulfate with a reversion to normal flow throughout the rest of the period. Similarly good results were not obtained in the small group given toluidine blue. Some degree of thrombocytopenia and a slight to moderate increase in clotting time during menstruation in many of the patients were reported. The tabulated results of these latter studies were not impressive, however, possibly due to lack of detail.

The nature of the prolamine titratable abnormality and particularly its relationship to changes in the menstrual cycle is not understood. More extensive studies to determine the normal variation would certainly appear to be worthwhile.—H.W.B.


One hundred premature infants were studied by means of daily prothrombin estimations. Of 83 of these infants, vitamin K was given neither to the mother prior to delivery nor to the child after birth. The other 17 received 2.5 mg. of vitamin K immediately after birth and vitamin K was given to nearly all of these mothers before delivery. The untreated premature infants had slightly higher prothrombin levels for the first fourteen days than those observed previously for full term infants. There was an increase in the prothrombin content of the blood of the treated premature infants above that seen in the untreated group. The administration of vitamin K, however, appeared to have no effect on the clinical course of these infants. The incidence of both hemorrhagic manifestations and mortality (due to causes other than hemorrhage, except possibly in 2 infants with cerebral hemorrhages who died despite adequate vitamin K) was far greater in the treated than in the untreated group. This difference was attributed to the fact that the treated group was born outside this hospital and received poorer initial care.

While there have been conflicting reports as to the prothrombin content of the blood of premature infants and its relationship to hemorrhagic manifestations, this study demonstrates rather effectively the doubtful value of routine vitamin K administration in premature infants and emphasizes that factors other than the prothrombin content of the blood are usually responsible for the increased hemorrhagic incidence in premature infants.—H.W.B.


The authors have described previously the structure of mature clots formed from bovine fibrinogen and thrombin as they appeared under the electron microscope (J. Exper. Med. 88: 285, 1947). In the present study, using a modification of the same technics, observations have been made on the sequence of events leading to the formation of these clots. It appeared that fibrinogen molecules were polymerized by the action of thrombin to form small needle-like structures which increased in length and diameter by lateral association with other similar structures to form larger fibrils or so-called unit fibers. The unit fibers in turn aligned by lateral association to form the compound fibers of the developing clot, but retained their identity within the strands.

In order to correlate these morphologic observations with what is known about physical constants and observations made on x-ray diffraction patterns, the authors propose that the fibrinogen molecules are disk-shaped and that when activated by thrombin they become aligned edge to edge as well as face to face to form fibrils giving a striated appearance.

The microscopic observations and proposed model of the fibrinogen molecule are well illustrated.—H.W.B.


A family showing evidence of hereditary hemorrhagic telangiectasia is recorded. The disease manifested itself either as repeated epistaxis and postoperative hemorrhages, with no visible evidence of angiomata, or as hemorrhages associated with typical angiomata. In this family there appeared to be
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Three more families with hereditary hemorrhagic telangiectasia are recorded. The authors consider that the condition is not particularly rare, as this makes a total of twenty-three families observed by them in the West Riding of Yorkshire. Epistaxis is the most frequent manifestation. Two of the present group suffered from arteriovenous aneurysm of the lung. Both were successfully treated by operation.—S.C.


A known hemophiliac, aged 57, was admitted to the hospital with severe respiratory obstruction due to a spontaneous subpharyngeal hemorrhage. This complication was dealt with successfully by direct laryngoscopy and intubation under soluble pentothione anesthesia. After thirty-six hours the tube was removed and convalescence was uneventful.—S.C.

LEUKOCYTES


In 3 patients who received therapeutic doses of chloramphenicol (2 because of typhoid fever, one for brucellosis), the authors noted the appearance of leukopenia with granulocytopenia. This appeared on the seventh day of therapy in one patient, and not until or after the completion of therapy in the other 2. Associated with the neutropenia was the bone marrow picture of a quantitative decrease in granulocytopoietic tissue, with a maturation arrest of granulopoiesis. In addition, one of the patients showed similar marrow arrest of the erythroid series, and a corresponding severe anemia. In none of the 3 cases were megakaryocytes or platelets altered.

Cessation of therapy was followed by rapid return of bone marrow and blood to normal. It was felt that the hematologic alterations were directly due to the drug.—S.E.


Nitrogen mustard produces widespread cellular destruction in the bone marrow and lymphoid organs. Some of these changes are similar to those found in the "alarm reaction." In the latter, changes are less marked in adrenalectomized animals. The present investigation on mice was undertaken to study the effect of adrenalectomy on the involution of lymphoid tissue in animals poisoned with nitrogen mustard, contrasted with the effect of formalin. In the experiment with nitrogen mustard, the injected and noninjected animals were divided into three groups: Unoperated, sham-operated and adrenalectomized. In the formalin experiments, the animals were divided into unoperated, anesthetized-unoperated, sham-operated and adrenalectomized groups. Intrapertoneal injections of pontamine blue were given to mark the lymphoid organs. The initial and final body weights, superficial lymph nodes, thymus, spleen and the estimated weight of splenic lymphoid tissue and total lymphoid tissue were studied. Nitrogen mustard differs from formalin in its effect on the weight of the lymphoid organs and adrenalectomy protects against lymphoid involution induced by formalin but affords little protection against the nitrogen induced involution.—O.P.J.

The author believes there is much to be learned about the functions of leukocytes from their enzyme content. He has worked out a method for obtaining cell-free preparations of rabbit polymorphonuclear leukocytes. The author summarizes the article as follows. (1) Surface active substances, such as saponin, alkyl sulphate, or bile salts, increase the alkaline phosphatase activity of suspensions of rabbit polymorphonuclear leukocytes. (2) Saponin acts by liberating the enzyme from the cell. The amount of saponin necessary is related stoichiometrically to the number of cells in the suspension. (3) The same surface-acting substances release esterase. (4) High concentrations of alkyl sulphate or bile salt, but not of saponin, inhibit the enzymes studied. There is a discussion concerning the methods used.—R.C.C.

From the Institute of Experimental Medicine and Surgery, University of Montreal, Montreal, Quebec, Canada. Am. J. Path. 26: 211-233, 1950.
Pathologists have reported the presence of fat cells, round cell infiltrations and lymphatic nodules, and bone marrow tissue in the adrenals. These lesions occurred spontaneously in patients suffering with advanced carcinomatosis, atherosclerosis, renal disease, heart disease, or hypertension. Since it was observed that impure corticotrophin-containing, bovine anterior pituitary extracts would produce leukocytic islets in the rat adrenal and that testoid compound, especially methyl testosterone, would produce fat cells there similar to those in bone marrow, experiments were devised to study the effects of synchronous treatment with these substances. A metaplasia of typical adrenal cortical cells into hematopoietic cells was observed, although it was impossible to preclude the participation of reticuloendothelial cells. Apparently products of tissue necrosis are essential for these results because purified corticotrophin is ineffective. Myeloid metaplasia was also observed in other tissues of the rat.—O.P.J.

BOOK REVIEW

The author has set himself the task of “informing students and teachers of science, of medicine, and of pharmacy, about the results of biochemical research since 1939,” and has succeeded admirably. Twenty-four chapters, many of them less than 20 pages long, summarize the use of isotopes in biology, mineral metabolism, the chemistry of carbohydrates, fatty acids, sterols, proteins, and the nucleic acids, the protein hormones, the vitamins and growth factors, the properties of enzyme systems, the chemistry of muscular contraction and the excitation of nerve, and advances in immunochemistry and in cytochemistry. Additional chapters deal with intermolecular forces in living matter and with thermodynamics and kinetics in biologic reactions.
Our present state of knowledge of each of these subjects is presented with a rare combination of exactness and clarity which will make the book as acceptable to the expert as it is informative to the student. This is achieved partly by the arrangement of the material of each chapter into short subsections, and by each new or unfamiliar idea being explained concisely as it is encountered. There is neither preoccupation with detail nor wandering into generalities. The effect is not only to leave the reader with a surprisingly large amount of varied information, but to give him an appreciation of how the present position with respect to each subject has been evolved. References are given throughout the text and are collected at the end of each chapter; speaking generally, they are the key references to the subject, and as such are very well chosen.—Eric Ponder