ABSTRACTS

JOSEPH F. ROSS, M.D., Editor

ABSTRACTERS

Helen W. Belding, M.D., Winston-Salem, N.C.
Conrad Maier, M.D., Zurich, Switzerland
Robert B. Chodos, M.D., Boston, Mass.
Cesar Merino, M.D., Lima, Peru
Roger C. Crafts, Ph.D., Cincinnati
Miloš Netoušek, M.D., Praha, Czechoslovakia
C. R. Das Gupta, M.D., Calcutta, India
Charles E. Rath, M.D., Washington
Solomon Estren, M.D., New York
Jean P. Soulier, M.D., Paris, France
Conrad Maier, M.D., Zurich, Switzerland
Robert B. Chodos, M.D., Boston, Mass.
Cesar Merino, M.D., Lima, Peru
Roger C. Crafts, Ph.D., Cincinnati
Miloš Netoušek, M.D., Praha, Czechoslovakia
C. R. Das Gupta, M.D., Calcutta, India
Charles E. Rath, M.D., Washington
Solomon Estren, M.D., New York
Jean P. Soulier, M.D., Paris, France
Conrad Maier, M.D., Zurich, Switzerland
Robert B. Chodos, M.D., Boston, Mass.
Cesar Merino, M.D., Lima, Peru
Roger C. Crafts, Ph.D., Cincinnati
Miloš Netoušek, M.D., Praha, Czechoslovakia
C. R. Das Gupta, M.D., Calcutta, India
Charles E. Rath, M.D., Washington
Conrad Maier, M.D., Zurich, Switzerland
Robert B. Chodos, M.D., Boston, Mass.
Cesar Merino, M.D., Lima, Peru
Roger C. Crafts, Ph.D., Cincinnati
Miloš Netoušek, M.D., Praha, Czechoslovakia
C. R. Das Gupta, M.D., Calcutta, India
Charles E. Rath, M.D., Washington
Conrad Maier, M.D., Zurich, Switzerland
Robert B. Chodos, M.D., Boston, Mass.
Cesar Merino, M.D., Lima, Peru
Roger C. Crafts, Ph.D., Cincinnati
Miloš Netoušek, M.D., Praha, Czechoslovakia
C. R. Das Gupta, M.D., Calcutta, India
Charles E. Rath, M.D., Washington

LEUKOCYTES and LEUKOCYTIC DISEASE


The presence of $^{35}$S in the mast cells of rats is demonstrated by the authors by means of stripping film autoradiography after injection of labeled sulphates. A maximum uptake was found forty-eight hours after injection and the decrease after this maximum appeared fairly small up to eighteen days after the injection.—C.M.

CYTOLOGY OF INFECTIOUS MONONUCLEOSIS. Johannes A. Horster. From the Medical Department, Academy of Düsseldorf, Düsseldorf, Germany. Acta haemat. 8: 378-387, 1952.

The author classifies the reticular cells obtained from lymph nodes in cases of infectious mononucleosis into seven groups. He especially stresses the question of the nature of the inclusion bodies within the monocytoid cells. They are considered as phagocyted leukocytes or virus bodies. The giant cells in i.m. show peculiarities which allow a separation from other types of giant cells.—C.M.

THE PATHOGENESIS OF THE ESSENTIAL CHRONIC GRANULOCYTOPENIA IN CHILDHOOD. C. Gasser. From the Pediatric clinic, University of Zurich, Zurich, Switzerland. Helvet. paediat. acta 7: 426-451, 1952.

Description of a new case of chronic benign granulocytopenia first seen by Vrtilek in 1951. The 15 year old girl suffered from the usual chronic infection of the upper respiratory tract. The author found, in the bone marrow, neither a disturbance of the mitosis nor an inhibition of the evacuation in the blood stream. There seems to be a defect in the maturation of the myelocytes to the segmental forms, in the author's opinion due to dienccephalic disorder. The accompanying symptoms of cranial asymmetry and vegetative and allergic manifestations would agree with this hypothesis.—C.M.

BUTAZOLIDIN AND IBUPROFEN IN THEIR RELATIONS TO AGRANULOCYTOSIS. K. H. Rechenberg. From the Medical Department, Kantonsspital, St. Gallen, Switzerland. Acta haemat. 9: 354-370, 1953.

In a series of three patients with known tendency to agranulocytosis following the use of dipyrine, the new pyrazole compound, sodium-dioxy-diphenyl-butylpyrazolidine (Butazolidine), was tested. This medicament had no influence on the white cells of these cases. A recurrent gonarthritis in one case was successfully treated with butazolidine without the development of agranulocytosis.—C.M.
ABSTRACTS


A total of nineteen patients received one or more doses of arsenic\textsuperscript{76} in doses varying from 12 to 90 mc. The patients were grouped as follows: acute leukemia, one; subacute myelogenous leukemia, three; chronic myelogenous leukemia, five; subacute lymphatic leukemia, two; chronic lymphatic leukemia, four; polycythemia vera, one; multiple myeloma, two; metastatic carcinoma, one.

"In general, there was either no evidence or insufficient evidence of cytolyis or inhibition of cellular proliferation to account for the remissions produced. There is no evidence that immature, rapidly dividing cells are more sensitive, or that irradiation by As\textsuperscript{76} induces a right shift in benign or neoplastic hematopoietic tissues.

"A review of the literature reveals similar experience with nitrogen mustard."

On the basis of their data the authors conclude that "irradiation in particular and cytotoxic therapy in general will probably not achieve a cure of these diseases." It seems to the reviewer that the evidence presented offers no basis for assuming that these agents will be effective; but neither do they prove that this conclusion is a valid one.—T.R.T., Jr.


This is the case of a girl, aged 6 years, who was quite well until an attack of measles. A month later she had flitting joint pains. This was followed by intermittent pyrexia up to 101 F., a persistently high pulse-rate of 130 unrelated to temperature, a progressive rise in sedimentation rate, loss of weight, and increasing anemia and granulopenia. A bone marrow biopsy led to the diagnosis. The characteristic cell had a large round or oval nucleus with a finely reticular distribution of the chromatin and usually two nucleoli. Some cells had punched-out intranuclear vacuoles. The basophilic cytoplasm was without a perinuclear pale zone, but did have an indefinite margin with pseudopodial processes. The cytoplasm was packed with innumerable clear vacuoles, ranging in size from 3 down wards. A very small number of cells resembling the smaller type of abnormal marrow cell appeared in the peripheral blood in the last week before death. Treatment with aminopterin, which has been shown to produce remission in some patients with acute leukemia, failed in this case of myeloid reticulosis. In fact, it possibly hastened death by destroying the few remaining normal red and white cell precursors. Postmortem examination revealed a reticuloendothelial hyperplasia affecting the bone marrow and to a lesser extent the lymph nodes, with only minimal involvement of other organs. The hyperplastic reticulum cells showed little tendency to differentiate into hemic or fixed-tissue cells.—O.P.J.


Five children with severe or moderately severe forms of reticuloendotheliosis were treated with cortisone and ACTH. In one, an infant with a fulminating case of Letterer-Siwe disease, therapy was a failure. The dramatic relief of acute symptoms during therapeutic courses in the other four patients and the gradual return to a state of health in two suggests the value of these agents in the management of these diseases. It is not implied that these hormones offer a cure nor that they should replace other methods of treatment such as x-ray therapy or antifolic acid drugs. Further clinical trial should determine their place in the therapy of the reticuloendothelioses.—H.W.B.
ABSTRACTS

BLOOD COAGULATION and HEMORRHAGIC DISEASE


This excellent review of prothrombin and accessory factors includes a summary of some of the author's own highly significant experimental work which led to the discovery of the clotting factors: proaccelerin, accelerin, proconvertin, and convertin. While the role of these accessory factors in the conversion of prothrombin to thrombin is still controversial, the author presents his concept of this process in a logical and convincing manner. Details on the preparation of these principles and methods for their quantitative determination are included as well as an analytic approach to the recognition of disturbances of prothrombin and its accessory factors in various clinical conditions.—H.W.B.

MECHANISM OF BLOOD COAGULATION IN NORMAL AND PATHOLOGIC CONDITIONS. M. Stefani. From the Ziskind Laboratories (Hematology Section) of the Joseph H. Pratt and New England Center Hospitals, and the Department of Medicine, Tufts College Medical School, Boston, Mass. Am. J. Med. 14: 64–86, 1953.

The author discusses in considerable detail the properties and mechanism of action of the several factors participating in blood coagulation, the fibrinolytic system, and the general disturbances in coagulation which underlie hemorrhagic disorders.

This scholarly presentation helps to clarify a complex subject which has been made all the more confusing by conflicting concepts and terminology. The objective presentation of the current theories of the coagulation process, including the author's own, and the liberal use of tables and diagrams make this a valuable reference paper.—H.W.B.


From this analysis of a group of one hundred forty infants and children with purpura some informative data are presented which should prove useful as a guide in the management of this variable symptom-complex in children. Patients with known primary conditions, such as leukemia, were excluded and the cases considered were classified broadly as nonthrombopenic purpura and thrombopenic purpura.

One of the most striking findings was the close association of infection, usually respiratory in type, with both types of purpura. Another finding of note was the definitely increased incidence of personal and family histories of allergy among the entire group. These two observations in combination suggest that many cases of purpura may be the result of a reaction to a bacterial antigen in a hypersensitive patient with subsequent damage to platelets, capillaries, or megakaryocytes or any combination thereof.

In general the patients with nonthrombopenic purpura in this series did well and, although in some instances the disease ran a protracted course, all recovered without residual damage. Of special interest were the few in this group who showed signs of extensive renal involvement but who recovered completely within six months.

While thrombopenic purpura in infants and children may be extremely serious, it is emphasized that the disease in this age group is far more often an acute self-limited illness. —H.W.B.

DIFFERENTIAL DIAGNOSIS, PATHOGENESIS AND TREATMENT OF THE THROMBOCYTOPENIC PURPURAS. E. L. Lozner. From the Department of Medicine, State University of New York at Syracuse, College of Medicine, Syracuse, N. Y. Am. J. Med. 14: 459–468, 1953.

In this excellent discussion of the thrombocytopenic purpuras, emphasis is placed on the more recent studies which have influenced, and are influencing, our present day concept of pathogenesis and, in turn, management of idiopathic thrombocytopenic purpura.
Among the several important points considered here are: evidence for the existence of a platelet agglutinin; possibility of platelet isosagglutination phenomena; the ever puzzling role of the spleen; limitations of the bone marrow aspiration in diagnosis and prognosis; and the place of splenectomy, ACTH and cortisone, and transfusions of platelet-rich blood or of platelet suspensions in treatment of the thrombocytopenic purpuras.—H.W.B.


It is concluded that the fundamental lesion in purpura is increased capillary fragility, although the hemorrhagic tendency may be increased if there is a coexistent thrombocytopenia. Evidence to date suggests an antigenic relationship between platelets and the vascular endothelium, and it is quite possible that thrombocytopenia and capillary damage have a common etiology.

Allergic purpura is classified here as: (1) purpura associated with an erythema exanthem and also with joint and visceral symptoms (Henoch-Schönlein syndrome); and (2) true purpura (without associated exanthem) due to infections or drugs. A detailed description of the Henoch-Schönlein syndrome is given. The discussion of the mechanisms underlying purpura due to infections is approached from the point of view that this type of purpura may be thrombocytopenic or nonthrombocytopenic, may occur with mild or severe infections, and may occur at the height of an infection or during convalescence.

A summary of the author's own significant immunologic studies of thrombocytopenic purpura due to Sedormid closes this comprehensive and informative presentation.—H.W.B.


A hemorrhagic diathesis was observed in a newborn on the fourth day of life. It was resistant to vitamin K therapy, and a few weeks after birth the prothrombin time (Quick was still greatly prolonged. Prothrombin was found normal by the two-stage procedure; Ac globulin was also normal. The defect was related to a deficiency of convertin (factor VII, SPCA). It was corrected by normal plasma or serum but not by Dicumarol or BaSO4 plasma. This case seems identical to another case described by Alexander et al. and termed congenital SPCA deficiency. It is shown that about 4 per cent of convertin is necessary for complete transformation of prothrombin into thrombin, thus this factor is not only an accelerator but also a converting factor.—J.P.S.

Hemorrhagic Diathesis Due to Absence of Christmas Factor. S. van Creveld and M. M. P. Paulissen. From the Paediatric Clinic, University of Amsterdam, Amsterdam, Holland. Lancet i: 823-824, 1953.

A report of a boy aged 10 years, with Christmas disease. The condition differed from hypoproconvertinemia in that the plasma proconvertin was 60 per cent of normal, a small quantity of thromboplastin greatly reduced the coagulation time, and the one-stage prothrombin time was normal.—R.H.G.


The authors postulate that in man a portion of the prothrombin exists as prothrombino-gen, an inactive form, and another portion as free or active prothrombin. They conclude that it is only the latter fraction that is measured by the unmodified one-stage procedure. On storage of oxalated human plasma, the prothrombinogen is converted to free prothrombin. However, this does not result in increased prothrombin activity of stored plasma because labile factor disappears. This postulate is discussed in the light of some clinical conditions and compared with the theory of prothrombin accelerator.—P.F.W.
1056

ABSTRACTS


A two-stage method was used to investigate problems of the quantitative relationship of the clotting factors V and VII. Factor VII can partially compensate for a deficiency of factor V, but its accelerator effect in the conversion of prothrombin is limited by the concentration of factor V. Optimal acceleration only occurs with high concentration of both factors. Factor VII is more active in the enhancement of the speed of thrombin formation. The prothrombin content does not influence the activation time but is proportional to the amount of prothrombin converted. The effects of factor VII do not depend on the concentration of prothrombin and cannot be replaced by prothrombin.—C.M.


Fibrin can be prepared in two different forms, one of which contains much stronger bonds than the other. The latter is obtained from highly purified fibrinogen and thrombin; the former is prepared in the presence of small amounts of calcium salts and an unidentified component of serum. The author now reports that this serum factor can be removed from fibrinogen by exposure to several reagents such as 3.0 M urea or 2.0 M sodium bromide. Serum factor activity can be restored merely by addition of a solution of bovine serum albumin.—R.H.G.


Tromexan (ethyl biscoumacetate) appears to have action similar to Dicoumarol on the mechanism of blood coagulation in that it depresses factor VII rather than prothrombin. The addition of serum containing factor VII will greatly shorten the clotting time of Tromexan-treated plasma. Plasma containing factor V does not have this effect.

A comparison of Quick's one-stage method for estimating prothrombin activity with Owren's method employing added Factor V in eighty cases where Tromexan was given in varying amounts, suggests that Quick's method is suitable for routine use.—R.H.G.


This is an interesting report of two patients developing painful, symmetrical hemorrhagic lesions involving the lower extremities and occurring approximately three weeks after the onset of relatively mild scarlet fever. Both cases were fatal; one autopsy was done and revealed a necrotizing vasculitis.—P.F.W.


Transfusion experiments carried out by the author prove the presence of a thrombocytopenic and a leukopenic factor in the blood of patients with thrombocytopenia and leukopenia, respectively. A relation of the factor and some immunologic reaction of the patients is stressed. In one case there was a sensitization of the red cells and two of the cases showed a marked increase in the gamma globulin fraction of the plasma.—C.M.

ABSTRACTS

An account of forty cases from Korea treated at the British Commonwealth General Hospital (Japan). The features included petechial hemorrhages in the skin and mucous membranes with bleeding from the respiratory, gastrointestinal, and urinary tracts. Thrombocytopenia was not noted. Prothrombin times, clotting times, and bleeding times were normal. The etiology remains obscure.—R.H.G.

BLOOD GROUPS


The third example of this antibody is in a Scotswoman, aged 29, whose blood is of group O, R, r (CDe/cde) and who was investigated when she had just given birth to healthy twins. There had been seven previous pregnancies. No transfusions or injections of blood had been given.—R.H.G.


A new Rh antibody, anti-f, has been found in the serum of a hemophilic man, aged 30, who has received about thirty-five blood transfusions.

Anti-f does not promise to be of any great practical importance in routine Rh blood group work.—R.H.G.


A survey of three thousand six hundred thirty-two cases of gastric carcinoma was carried out in a number of hospitals in England and Scotland. The frequency of blood group A was greater and of blood group O less in patients with gastric carcinoma than in the general population of their area.—R.H.G.


"Private" blood factors are characterized by the limitation of the positive or negative reactions to members of a particular family. The authors suggest that it is necessary to enquire into the racial origin of the stimulator of an antibody defining a "private" blood factor, and if he is found to be of a different race from the person who has developed the antibody, members of his race should be tested. Sometimes a hint of foreign ancestry may be given by detailed blood grouping. (The original paper should be studied by those interested.)—R.H.G.


The incomplete cold antibody present in all normal human sera gives the reactions expected of anti-H. The strength of the reactions of this antibody depends on the H content of cells.

In the tests, saliva from secretors was used as a source of H substance, and a 1 per cent solution of purified H substance was also employed. Rabbit anti-H serum and the serum of the eel, anguilla anguilla, were the sources of anti-H.—R.H.G.
ABSTRACTS

EXPERIMENTAL ERYTHROBLASTOSIS FETALIS IN RABBITS. A. Kellner and E. F. Hedal.
From the Department of Pathology and the Central Laboratories, The New York Hospi-

I. CHARACTERIZATION OF A PAIR OF ALLELIC BLOOD GROUP FACTORS AND THEIR SPECIFIC IMMUNE ISOANTIBODIES.
Two new allelic blood group antigens in rabbits, designated G and g, and the character-
istics of their specific immune isoantibodies are described. Despite a few dissimilarities,
there was a remarkably close parallel between the serological properties of the antigens
and antibodies of this rabbit G-g system and the Rh-Hr system in man.

One or the other or both of these blood group factors were identified in the red blood
cells (but not in other tissues or body fluids) of every one of a large group of mongrel and
inbred rabbits. Specific antibodies were produced only by the injection of specific red cells
or as a result of the isoimmunization of pregnancy. In most cases a given antiserum con-
tained both agglutinating and coating antibodies to the G or g factors. The coating anti-
bodies, which did not behave as true blocking antibodies, could be detected by the Coombs
test or by trypsin-modified red cells. The antibodies were heat-stable, active over a wide
temperature range, and were capable of hemolyzing red cells readily in the presence of
complement.

II. THE PASSAGE OF BLOOD GROUP ANTIGENS AND THEIR SPECIFIC ISOANTIBODIES ACROSS THE PLACENTA.
The development of specific antibodies in female rabbits to blood group factors G or g
was observed in five of the ten pregnancies in which the fetal red cells carried one of the
factors absent in the mother. These antibodies were of low titer, disappeared within six
weeks postpartum, and were not present in increasing titer with successive pregnancies.

It was shown that antibodies to the G-g factors, whether produced as a result of preg-
nancy or by the injection of specific red cells, readily pass from the maternal to the fetal
circulation and can be found coating the fetal red cells or in the serum of the fetus both
in utero and at birth prior to nursing. Unlike the human, the rabbit placenta appeared to
be equally permeable to the agglutinating and coating antibodies.

The implication of these findings are well discussed. From investigations already in
progress on the fetal changes resulting from this intrauterine transfer of specific antibodies
may come elucidation of some of the obscure aspects of human erythroblastosis fetalis.—
H.W.B.

THE INFLUENCE OF ANTIHISTAMINIC AGENTS ON ERYTHROCYTE AGGLUTINATION AND RELATED PHENOMENA. S. P. Lucia, M. L. Hunt and H. R. Bierman. From the Department of Medicine, University of California School of Medicine, San Francisco, Calif. J. Lab. & Clin. Med. 41: 574–582, 1953.

During a study of the effects of Benadryl on transfusion reactions it was observed that
the addition of Benadryl to anti-A and anti-B sera significantly retarded the specific ag-
glutination in vitro of group A and group B corpuscles respectively. Accordingly, a de-
tailed study was undertaken. Eight anti-histaminic compounds were tested, but only one
was used in crystalline form, the others being used “as commercially available.” Several
of these compounds caused hemolysis.

Although this work is of considerable interest it is difficult to evaluate because of the
failure to use pure compounds.—T.R.T., Jr.
ABSTRACTS

METHODOLOGY


A modification of Ehrlich's hematoxylin and eosin technique—in which an equal parts mixture of glycerol and water is used as a solvent for the eosin—stains specifically the granules of eosinophile leukocytes in methyl alcohol fixed blood films. The advantage of this method over those employing the Romanowsky type of stains is that the location of eosinophils may be made with the lower power of the microscope, and no confusion with neutrophile cells is likely because the cytoplasmic elements of these remain practically unstained.—O.P.J.

AN IMPROVED HEMATOXYLIN-EOSIN STAIN FOR SECTIONS OF MARROW UNITS. E. M. Schleicher. From Department of Pathology, St. Barnabas Hospital, Minneapolis, Minn. Stain Technol. 28: 119-123, 1953.

The procedure recommended involves the use of dilute Harris' hematoxylin for 2 to 3 minutes, followed by rinsing in distilled water, acid-alcohol, and weak ammonia water. Following a brief treatment in aqueous phosphotungstic acid and additional rinsing, the slides are checked for blueness and nuclear differentiation in 80 per cent alcohol. Counter-staining is accomplished with a 0.5 per cent alcohol-eosin stock solution 10 parts and 95 per cent alcohol 90 parts and checked under microscope for staining quality. After dehydration, clearing, and mounting, this staining schedule brings out minute structural detail of bone marrow tissue heretofore not demonstrable.—O.P.J.


Eight cases of malignant melanoma were studied by means of marrow aspiration. The aspirated material was stained with Wright's stain and supravital staining was done using neutral red and Janus green. Four of these cases were shown to have melanoma cells in the marrow. The authors state that careful palpation will often elicit areas of bone tenderness, which, if possible, should be the site of aspiration.

A grossly black marrow aspirate or the presence of black pigment in tumor cells is diagnostic of metastatic melanoma, and even in the absence of pigment it is felt that the melanoma cells have a rather characteristic cytologic appearance.

Good photomicrographs are included.—T.R.T., Jr.


A method is described which is a modification of Ham's modification of the method of Bing and Baker. Benzidine base is purified according to the method of McFarlane and Hamilton. The method is otherwise very similar to the original except for greater final dilution, and the use of a spectrophotometer at 485 to 500 mμ.

It is of interest that low plasma recoveries were always observed, averaging 61.2 per cent at plasma dilutions of 1:25 and over 85 per cent at 1:100. The recovery at any one dilution with a given batch of reagent, however, seems constant.

The method appears to be a good one, but the perfect method for plasma hemoglobin has not yet been described.—T.R.T., Jr.

IDENTIFICATION OF ABNORMAL HEMOGLOBINS BY MEANS OF PAPER ELECTROPHORESIS. T. H. Spaet. From the Department of Medicine, Stanford University School of Medicine, San Francisco, Calif. J. Lab. & Clin. Med. 41: 161-165, 1953.
ABSTRACTS

The author describes an apparatus which is a modification of that described by Durrum. The apparatus is simple and economical. Satisfactory separation of a, b, and c hemoglobins was achieved.—T.R.T., Jr.

POST TRANSFUSION SURVIVAL OF ERYTHROCYTES STORED IN A SOLUTION OF ETHYLENE DIAMINE TETRA-ACETIC ACID AND DEXTROSE. C. C. Sprague, J. B. Shapleigh, S. Mayes, R. D. Lange, and C. V. Moore. From the Department of Medicine, Washington University School of Medicine, St. Louis, Mo. J. Lab. & Clin. Med. 41: 84–90, 1953.

The disodium salt of ethylenediamine tetra acetic acid (EDTA) has been found to be an effective anticoagulant because of its ability to act with calcium to form a soluble complex of calcium.

Blood was collected from thirty-two healthy group O donors. Sixteen collections were made into 1.5 Gm. EDTA in 100 ml. of 5 per cent dextrose. The remaining sixteen were collected in 75 ml. of ACD solution. All were stored at 4 to 6 C.

Sixteen recipients each received one of each of the above bloods, stored for periods from one to twenty-eight days.

The data indicate that ACD and EDTA were equally satisfactory under these conditions.—T.R.T., Jr.


The author reviews briefly the history of blood transfusions, and points out again that "there is no substitute for whole blood." From a functional point of view the only criteria for the degree of preservation is whether or not the preserved red cells remain in the recipient's circulation after transfusion. A brief description is given of the different methods for measuring the persistence in the circulation of transfused red cells. These methods include the Ashby technic, the use of blood from donors who have received radioactive iron, and incubation of blood with either radioactive phosphorus or radioactive chromium.

It is pointed out that there is not as yet any satisfactory in vitro method for predicting whether or not red cells will survive in the circulation after transfusion.

Emphasis is given to the fact that blood should be kept at storage temperatures of 4 to 10 C., and that warming for even a few hours may cause significant deterioration.

A description is given of recent work by the author on preservation of whole blood in 15 per cent glycerol at −79 C. for eight months. Normal survival of these red cells was demonstrated, although some cells were lost before transfusion as a result of the processing. Similar studies have shown that this can be accomplished at −15 C. Red cells stored at −79 C. undergo slow but continuous destruction, and the same is true to a greater degree at −15 C. The author is optimistic concerning the finding of the cause for this destruction.—T.R.T., Jr.

AN IMPROVED TEST FOR BILIRUBIN IN URINE. G. Klatskin and L. Bungards. From the Department of Internal Medicine, Yale University School of Medicine, New Haven, Conn. New England J. Med. 248: 712–717, 1953.

A simple, sensitive, and specific test for urine bilirubin is described. The paper is worthy of study by anyone interested in clinical laboratory diagnostic procedures; however, it is questionable whether the test justifies general use because negative results were obtained in individuals with elevated serum bilirubin levels.—P.F.W.