ABSTRACTS

BLOOD VASCULAR PROBLEMS

SCHÖNLEIN-HENOCH PURPURA. *E. G. L. Bywaters, I. Isdale and J. J. Kempton.* From the Canadian Red Cross Memorial Hospital, Taplow, Bucks., the Postgraduate Medical School, London and the Royal Berkshire Hospital, Reading, Berks., England. Quart. J. Med. *26*:161–175, 1957.

Although attention has been called to a relationship between Schönlein-Henoch purpura and acute nephritis and rheumatic fever, both of which are now accepted as being due to a preceding group A β-hemolytic streptococcus infection, such organisms have not been isolated from the throat during or before an attack of Schönlein-Henoch purpura, nor has a raised antibody titer been demonstrated in the serum.

In 64 cases of Schönlein-Henoch purpura, mostly children, serum antistreptolysin O titers were raised in a third of the cases, an incidence equal to that found in a control series of non-rheumatic children, and half of that seen in children with rheumatic fever that were admitted to hospital. Group A β-hemolytic streptococci were isolated in a quarter of the cases investigated, the incidence being intermediate between that found in rheumatic fever and that found in a control series of children admitted for nonrheumatic conditions. Although there are clinical and pathological similarities between the condition and rheumatic fever and acute nephritis, that is no clear evidence of infection with group A β-hemolytic streptococci as an invariable causative factor.—*R. H. G.*

PARTIAL GASTRECTOMY IN VON WILLEBRAND'S DISEASE. *R. E. Irvine and J. D. T. Jones.* From the Royal Infirmary, Newcastle-on-Tyne, England. Brit. M. J. *1*:1101–1102, 1957.

A 42-year-old male with a family history of bleeding had a history of bleeding episodes from childhood. The bleeding time was prolonged, but the platelet count, clotting time and clot retraction were normal, as were the results of the one and two stage prothrombin time and the thromboplastin generation test. On account of a gastric ulcer with recurrent hemorrhages, a Bilroth I partial gastrectomy was done. There was continued oozing of

The initials of abstracters who are not listed in the above masthead refer to those abstracters listed in the masthead of the December 1958 issue of *Blood*, p. 1206.

blood into the stomach for 48 hours after operation and then a brisk hemorrhage, for which 5 pints of blood were transfused. In addition 100 mg. of corticotrophin were given daily for 5 days. The patient recovered without further trouble. Although all authors stress the danger of operation in this disease, the literature contains only one record of a fatal operation. This contrasts very favourably with hemophilia.—*R. H. G.*

BONE MARROW

THE CHANGING CELLULAR DISTRIBUTION IN BONE MARROW OF THE NORMAL ALBINO BETWEEN ONE AND FIFTY WEEKS OF AGE. *C. Harris and W. T. Burke.* From Fels Research Institute, Temple University Medical School, Philadelphia, Pa. Am. J. Path. *33*:931–951, 1957.

Changes in cell lineage taking place in adult bone marrows are in too small a number to permit an observer to relate them to cells from which they derived, or to cells which derive from them. However, it has been found that there are two periods in the life of the young rat during which mass alterations in distribution of marrow cells occurred, so that at least half of the observed cells were in one phase of transition or another. First, at two weeks of age, the transition involved a series of mononuclear cell reactions that eventually resulted in the production of myeloblasts and definitive erythroblasts (normoblasts). Second, there is a period between 6–10 weeks of age in which granulocytic (benzidine positive) cells were produced from lymphocyte-like small mononuclear cells. These two changes occur as an orderly response to separate biologic influences, probably extracellular in nature, which become dominant at the second and eighth weeks of life, respectively. The author suggests that perhaps the classical myeloblast is not the only pathway of granulocytopoiesis, but the link in an occasional pathway of hematopoiesis, and may be by-passed in many instances by more direct transformations. This, of course, is just what Downey has emphasized in his writings on the myeloblast and leukemic reticuloendotheliosis.—*O. P. J.*

THE NORMAL BONE MARROW IN ALBINO RATS. *N. N. Sen, S. Kumar and V. S. Mangalik.* From the Division of Hematology, Department of Pathology and Bacteriology, Lucknow University, Lucknow, India. Indian J. M. Sc. *10*:951–954, 1956.

Femoral bone of rats shows pink marrow almost throughout its length. The M:E ratio was found to be 1.55 : 1. Histologic examination did not show any lymph follicles.—*J. B. C.*

THE BONE MARROW IN MALIGNANT DISEASE. *E. M. Kingsley Pillers, John Marks and J. S. Mitchell.* From the Departments of Radiotherapeutics and Pathology, University of Cambridge. Brit. J. Cancer *10*:458–471, 1956.

Sternal marrows of 601 patients were evaluated with particular reference to the identification of malignant cells in the marrow, the presence and significance of plasma cell hyperplasia in the marrow, the significance of bone marrow eosinophilia, relationship between the marrow findings and the clinical follow-up, and the value of sternal puncture in the treatment of carcinoma. The majority of the patients were clinically well and at an early stage of the disease. Four hundred six patients with carcinoma of the breast, bronchus, cervix uteri, and ovary were considered in detail. Thirty-seven of the 601 patients (6.2%) showed tumor cells in the marrow, and 180 showed an increase in plasma cells. The prognosis was worst in those patients who have tumor cells in the marrow. In carcinoma of the bronchus, cervix uteri, and ovary, increase in the number of plasma cells in the marrow was associated with a shorter average survival than in patients without this finding. Of 406 patients analyzed for eosinophilia, 18 showed eosinophilia over 7.5%. There is at present no apparent correlation between eosinophilia and prognosis in these patients. No eosinophilia was detected in any of the 37 patients with cancer cells in the

marrow smears. The authors suggest that sternal marrow examination has a place in assessing the type of treatment and providing cytologic material for diagnosis of malignancy. —*H. R.*

EOSINOPHILS

THE INTERPRETATION OF EOSINOPHILIA AND EOSINOPENIA. *R. Gross.* Med. Univ. Klinik Marburg, Lahn. Deutsche med. Wschnschr. No. 15, p. 507–510, 1957.

For clinical purposes the bone marrow may be considered as the only site of production of eosinophilic leukocytes. They circulate in the peripheral blood only for about 2 days of their life, before they are destroyed in the gastrointestine, the lung, and the spleen. The author points out the discrepancy between bone marrow findings and peripheral blood eosinophilia in eosinophilic reactions. Average values are given for the percentage of eosinophilic cells in the bone marrow with respect to their different stages of maturity under normal and pathologic conditions. Conditions leading to an eosinophilia and causes of eosinopenia are discussed.—*M. -H. H.*

THE THORN TEST AND PARASITIC INFECTIONS. *Terezinha Ferreira de Lorenzi and Michel Jamra.* 1ª Clínica Médica—Prof. A. B. Ulhoa Cintra. Servico de Hematologia do Hospital das Clínicas da Fac. de Med. da Univ. S. Paulo. S. Paulo, Brazil. Revista do Hospital das Clínicas. *12:*234–238, 1957.

The Thorn test with cortocotrophin and adrenalin was studied in 20 patients infected by different kinds of parasites and with variable degrees of eosinophilia. An eosinophilic depression was observed after the stimulants mentioned, irregular in some cases when compared to that obtained in persons not parasitized.—*M. A. J.*

EOSINOPHILIA AFTER SPLENECTOMY. *T. K. Saha and R. N. Chaudhuri.* From the Clinical Research Unit, I.C.M.R., School of Tropical Medicine, Calcutta. Indian J. M. Sc. *10:*967–970, 1957.

Following splenectomy in 15 out of 18 cases of "tropical splenomegaly" there was fluctuating high eosinophilia in the peripheral blood. Between 1 and 9 months after splenectomy the maximum eosinophil counts varied from 1900 to 10,800. During febrile bouts due to malaria or other infections the eosinophils temporarily decreased.—*J. B. C.*

BLOOD EOSINOPHILIA. *Horacio A. Podesta.* Hospital de Clinicas, Cuba. Revista Medica Cubana. *66:*354–369, 1955.

After mentioning the several causes of eosinophilia the author discusses some rare cases in which an isolated eosinophilia is present with minor or any clinical symptomatology.

A case is presented, 37 years old, with weakness and pallor for a year, and a blood picture of 4.2 millions erythrocytes per cu.mm., 18,000 leukocytes per cu.mm., with 48.0% eosinophils. Other laboratory investigations, especially the search for intestinal parasites were negative; there was no pulmonary modification. Biopsy of an axillary lymph node revealed no definite lesions on histopathologic examination. Gastrointestinal series and cutaneous test for trichina and other allergens were negative. Benzidine tests in feces were negative. After careful consideration of similar cases studied in the literature, the author approximated this case to those cases of Vucentich and Panaia (Instituto de Patologia General del Norte, Jujuy) in which there was found therapeutic response only to a mixture of vitamins of the B complex, since the eosinophilia is resistant to arsenic and other measures. Vitamin B complex in this case, given for 3 weeks, relieved the weakness and decreased the eosinophils from 58.0% to 7.0%.—*M. A. J.*

THE MECHANISM OF EOSINOPENIA PRODUCED BY ACTH AND CORTICOIDS IN THE HORSE. *R. K. Archer.* From Equine Research Station of the Animal Health Trust, Newmarket, England. J. Path. & Bact. *74*:387–395, 1957.

Eosinopenia of peripheral blood occurs after the "alarm reaction," injection of ACTH, or injection of compound F, provided the suprarenal cortex is intact. The fate of the eosinophil leukocytes, which disappear from circulating blood, has been variously explained. A series of experiments were devised to test these on horses. By using intradermal technics the direct action of corticoids and ACTH upon eosinophils in vivo were investigated. In vitro experiments were conducted by incubation of blood or marrow aspirate with these agents. Studies were also made of the bone marrow eosinophil content during corticoid- and ACTH-induced peripheral eosinopenia. The results indicate that the reduction of circulating histamine which follows injection of corticoids may remove the necessary stimulus to the release of eosinophils by the bone marrow. Cortisone does not prevent the development of localized eosinophilia in the skin followng local injection of histamine. Histamine is not in direct control of eosinopoiesis, but is perhaps necessary for the release of eosinophils held in the marrow as well as for chemotactic attraction of the cells. The peripheral eosinopenia which follows corticoid administration is due to the absence of the normal stimulus to release provided by histamine, since the corticoids have produced a reduction of circulating histamine.—*O. P. J.*

FIBRINOGEN

THE TREATMENT OF THROMBOCYTOPENIC HEMORRHAGES BY THE INJECTION OF BIG DOSES OF HUMAN FIBRINOGEN. *P. Cazal, R. Graafland, P. Izarn, M. Mathieu, G. Paleirac and J. Fischer.* Montpellier, France. Presse méd. *64*:670–671, 1956.

Cohn's fraction I was employed for the treatment of 10 episodes of hemorrhages in 4 cases of thrombocytopenias with doses of 3 to 10 Gm. In 99 instances a beneficial effect was obtained. The mechanism of action is still uncertain. Possibly, in physiopathologic conditions an excess of fibrinogen might compensate the platelet deficiency.—*P. d. N.*

EVALUATION OF THE LIFE SPAN OF FIBRINOGEN IN MAN AND RABBIT. *K. Gerdes and W. Maurer.* From the Medizinische Universitätsklinik, Köln, Germany. Biochem. Ztschr. *328*: 552, 1957.

The biologic half time of fibrinogen was studied in man and rabbit by means of radioisotopes, i.e., S^{35}-yeast, S^{35}-yeast-hydrolysate or S^{35}-methionine, as well as S^{35}-plasma from a rabbit donor to rabbits. The decrease of S^{35}-activity was measured in the fibrin obtained from plasma samples at intervals. For human fibrinogen the half time was 6.7 days (upper limits). For rabbit fibrinogen the values were 3.5 days. The infused S^{35}-fibrinogen had a half time of 2.3 days.—*P. d. N.*

PURIFICATION OF HUMAN AND BOVINE FIBRINOGEN. *B. Blombäck and M. Blombäck.* From the Chemistry Department II, Karolinska Institutet, Stockholm, Sweden. Arckiv f. Kemi *10*:415–443, 1956.

Description of an improved method for the purification of human fibrinogen for intravenous use from Cohn's fraction I and of bovine fibrinogen. A human fibrinogen with a coagulability of 88%, containing practically all the antihemophilic globulin of fraction I, was obtained, but no prothrombin and plasmin. Further purifications of bovine fibrinogen made it possible to obtain a purity of 95%. Such preparations are easily soluble and stable both in lyophilized condition and in sodium chloride solution.—*P. d. N.*

LATENT FIBRINOLYSIS IN LIVER CIRRHOSIS. *J. Dausset, A. Paraf, Y. Bergerot-Blondet and J. Caroli.* From the Centre National de la Transfusion Sanguine, Paris, France. Ann. Rech. Med. *13*:1, 1956.

In 55 cases of liver cirrhosis, 3 cases of infectious hepatitis and 10 cases of obstructive jaundice, fibrinolysis was studied by means of a personal method, which is based on the dilution of the blood by serum and saline. An increased fibrinolytic activity was observed in 47% of the cases of liver cirrhosis, but not in the other cases. In a few cases there is also a hemorrhagic syndrome. These findings are supposed to be due to an excess of a plasmatic activator or a deficiency of antiplasmin. During the lysis, a substance is liberated, which, after heating at 56 C. for 30 minutes, is able to react with tanned erythrocytes, coated with a plasmatic substance present in fraction I.—*P. d. N.*

HEMORRHAGIC DISEASES

PRIMARY HEMORRHAGIC DISEASES. *J. H. Lewis, J. H. Ferguson, J. W. Fresh and M. B. Zucker.* From the University of North Carolina, Chapel Hill. J. Lab. & Clin. Med. *49:*211–232, 1957.

The study concerns the hemostatic profiles obtained from a large series of normal subjects and from 101 patients with congenital hemorrhagic disorders. In 12 of the patients the disorder was of vascular origin; and in this group 4 had local vascular lesions (hemangiomata, telangectasia), the remainder being classified as "pseudohemophilia." Although an occasional mild coagulation abnormality was found, no consistent blood finding accounted for the hemorrhagic symptoms. One patient, a 2-year-old child, had symptoms of thrombocytopenic purpura for a year and responded to splenectomy. Other members of the family were also found to have thrombocytopenia. Two patients had normal platelet counts but deranged platelet function, as evidenced by a prolonged bleeding time, poor clot retraction, or reduced serum serotonin. There were 52 hemophiliacs. These included one female with a 2% blood AHF content whose parents were both normal. In the remaining typical cases, AHF assays ranged between 0–20% of normal, and 5 of the group showed blood anti-AHF activity. Patients with PTC deficiency numbered 26, an astonishingly high incidence compared to AHF deficiency. Two of these patients had circulating anticoagulants. In addition, there were five patients lacking Ac globulin, two with reduced factor VII, and one with afibrinogenemia.—*T. H. S.*

ANTI-AcG: SPECIFIC CIRCULATING INHIBITOR OF THE LABILE CLOTTING FACTOR. *J. H. Ferguson, C. L. Johnson and D. A. Howell.* From the University of North Carolina, Chapel Hill. Proc. Soc. Exper. Biol. & Med. *95:*567–570, 1957.

An elderly man developed a hemorrhagic disorder following surgery, and the condition was worsened by transfusion. The patient was found to have a prolonged Quick prothrombin time which was not significantly improved by an equal volume of normal plasma. Partial improvement was produced by an AcG concentrate; concentrated proconvertin was ineffective. The anticoagulant effect of the patient's plasma was not due to antithromboplastin.

The patient's plasma contained less than 1% AcG, and it reduced the AcG assay of potent sources of AcG. A method of titrating the anticoagulant was developed and showed activity even in a 1:320 dilution. The anticoagulant was not adsorbed on barium sulfate and was precipitated by saturation of ammonium sulfate between 33 and 50%. It was not lost during clotting, and was stable to storage and heating to 60 C. There was also stability over a wide pH range, and extraction with ether did not cause loss of activity.

A variety of coagulation studies applied to the patient's blood gave abnormalities consistent with AcG deficiency; the authors conclude that the patient had a true and specific anti-AcG.—*T. H. S.*

COMBINED MILD PTC (PLASMA THROMBOPLASTIN COMPONENT) AND FACTOR VII DEFICIENCIES. *H. B. Stein and Olga L. Abrahams.* From the Department of Clinical Pathology, University of the Witwatersrand, Johannesburg. South African J. M. Sc. *21:*13–22, 1956.

The propositus showed a normal whole blood coagulation time with relatively poor prothrombin consumption. The 1-stage prothrombin time was slightly prolonged and found

to be due to Factor VII deficiency, while thromboplastin generation studies revealed a deficiency in PTC. This is probably the third recorded case of such a combined deficiency. —*T. H. B.*

HYPOPROCONVERTINEMIA. (FACTOR VII, STABLE FACTOR, SPCA DEFICIENCY.) *Eurico Coelho, Mercedes Zindel and Michel Jamra.* 1ª Clínica Médica—Prof. A. B. Ulhoa Cintra. Servico de Hematologia do Hospital das Clínicas da Fac. de Med. da Univ. S. Paulo. São Paulo, Brazil. Revista do Hospital das Clínicas *12*:220–225, 1957.

A deficiency of factor VII (stable factor, proconvertin) of serum is described in a peculiar congenital case. The Quick prothrombin time was normal. The thromboplastin generation tests revealed slight deficiency curves with the patient's plasma and serum. The addition of normal plasma to the patient's serum was not able to regenerate entirely the thromboplastin generation curves as well as the addition of normal serum to the patient's plasma.

P.T.A. deficiency was ruled out by mixing normal tricalcium-phosphate treated serum with the patient's plasma, the mixture being abnormal. The deficiency was associated with anomalous capillaries and was revealed only by sensitizing the "thromboplastin generation test" using 1/50 dilutions of serum instead of the regular 1/10 dilution, and by tests of Quick prothrombin time mixing the patient's plasma with factor VII-deficient bovine plasmas. Slight deficiency states may be followed, as in the case described, by apparently normal values in the thromboplastin generation tests and they need several ways of approach to reach the definite picture of the involved coagulopathy.—*M. A. J.*

HEMORRHAGIC DIATHESIS DUE TO FACTOR VII DEFICIENCY. *C. P. Barnett.* From the Mary Washington Hospital, Fredericksburg, Va. Arch. Int. Med. *99*:280–284, 1957.

The propositus was a 32-year-old Negro man with hemorrhagic symptoms since childhood. Positive family history was confined to one sister who died at the age of 13 of "vaginal bleeding."

Positive laboratory findings were as follows: The one-stage (Quick) prothrombin time varied between 150 and 270 seconds, but the "true prothrombin" content of the plasma (Owren) was normal. The defect was corrected by normal plasma, aged plasma and normal serum, but not by adsorbed (adsorbing agent not stated) or dicumarol plasma. Factor VII (de Vries) was markedly reduced, and the patient's serum failed to improve clotting in dicumarol plasma.

Additional abnormalities included prolonged clotting times of whole blood and recalcified plasma, and impaired prothrombin consumption. In view of these latter findings, the defect is probably that of Stuart Factor rather than factor VII. The author is evidently unaware of this distinction.—*T. H. S.*

CONGENITAL HYPOPROCONVERTINEMIA: CLINICAL STUDY AND GENETIC FINDINGS. *U. M. Serafini and F. Pericoli.* From the Instituto di clinica Medica Generale e Terapia Medica, University, Roma, Italy. Progresso med. *12*:577–588, 1956.

Two cases of congenital, hemorrhagic hypoproconvertinemia and three cases of congenital hypoproconvertinemia with no hemorrhagic manifestations were described. The two patients were aged 9 and 17 years, respectively. Prothrombin times were of 27 and 33 seconds (normal 17–18 sec.). Factor VII activity was of 30 and 28%. A complete coagulation and genetic study of the family was carried out, as well as a complete review of the literature. —*P. d. N.*

STUART CLOTTING DEFECT. I. SEGREGATION OF AN HEREDITARY HEMORRHAGIC STATE FROM THE HETEROGENEOUS GROUP HERETOFORE CALLED "STABLE FACTOR" (SPCA, PROCONVERTIN, FACTOR VII) DEFICIENCY. *C. Hougie, E. M. Barrow and J. B. Graham.* From the University of North Carolina, Chapel Hill. J. Clin. Invest. *36*:485–496, 1957.

The subject of this study is a 36-year-old man with a congenital hemorrhagic disorder that had been previously been reported as factor VII deficiency. The coagulation defect was characterized by a slightly prolonged whole blood clotting time, marked prolongation of the Quick prothrombin time, normal prothrombin concentration, and impaired prothrombin consumption. The prolonged one-stage prothrombin time was corrected with normal plasma and serum, but not by plasma adsorbed with aluminum hydroxide. Unlike factor VII deficiency, the present defect failed to give a normal clotting time of plasma with the addition of Stypven, and the serum gave an abnormal thromboplastin generation curve. Serum from patients with PTC deficiency and from patients on short-term dicumarol therapy corrected the defect, but correction was impaired in sera from patients treated for longer periods with dicumarol. The agent missing from this patient has been named *Stuart Factor.*

Stuart Factor is necessary for blood thromboplastin formation, as shown by the inability of Stuart-deficient serum to form active thromboplastin in the presence of AHF, calcium ion, and platelets. It is moderately storage-stable, but is destroyed in plasma at 56 C. within 30 minutes.

Samples of Stuart plasma sent to Alexander showed mutual correction with SPCA-deficient plasma in the prothrombin test, but a patient also previously reported as factor VII deficiency was found to be another case of Stuart Factor deficiency. Possibly about half of the patients currently diagnosed as factor VII deficiency actually lack Stuart Factor instead.—*T. H. S.*

STUART CLOTTING DEFECT. II. GENETIC ASPECTS OF A "NEW" HEMORRHAGIC STATE. *J. B. Graham, E. M. Barrow and C. Hougie.* From the University of North Carolina, Chapel Hill. J. Clin. Invest. *36*:497–503, 1957.

Stuart Factor was estimated in plasma by correction of the one-stage prothrombin time of Stuart-deficient plasma, and in serum by correction of thromboplastin generation with Stuart-deficient serum. Studies of the Stuart family with these methods revealed the patient's mother and his 4 children to have a partial deficiency of Stuart factor. The father was not available for testing, but a paternal aunt and uncle proved to be partially deficient. The patient's parents were consanguinous. The data show that the gene for Stuart factor deficiency is incomplete recessive and autosomal, and the full blown disorder represents the homozygous state. Heterozygotes showed a 1½ to 3 second prolongation of the prothrombin time and thromboplastin generation time.—*T. H. S.*

HAGEMAN TRAIT (HAGEMAN FACTOR DEFICIENCY). *R. T. S. Jim and S. Goldfein.* From Washington University School of Medicine, St. Louis, Mo. Am. J. Med. *23*:824–831, 1957.

The patient was a 26-year-old woman with no history of a hemorrhagic disorder and no excessive bleeding following tonsillectomy or appendectomy. The family history was negative for hemorrhagic symptoms and consanguinity. The patient presented a prolonged whole blood clotting time, impaired thromboplastin generation, but normal prothrombin consumption. Matching studies on patients with known defects established the diagnosis of Hageman factor deficiency; and previously described properties of the factor were confirmed. This report adds further credence to the existence of Hageman factor as a unique clotting entity.—*T. H. S.*

COAGULATION DEFECT IN HORSE PLASMA. *K. E. Sjølin.* From the Carlsberg Foundation, Copenhagen, Denmark. Proc. Soc. Exper. Biol. & Med. *94*:818–820, 1957.

Compared to human blood, coagulation in the horse is greatly retarded. Previous studies have alleged AHF or PTC deficiency in horse blood.

In the present study, horse plasma was shown to have delayed thrombin generation and a prolonged recalcified clotting time of about 4 minutes. These abnormalities were corrected by untreated or adsorbed human plasma or serum, and by serum heated to 56°

for 30 minutes. Serum or plasma from a patient with Hageman trait (source and identification not given) was ineffective. Surprisingly, the defect was corrected by frozen platelets, even from the deficient animal.

It is concluded that horse blood is deficient in Hageman factor. If confirmed, this would be another example of normal hemostasis in the face of coagulation currently considered to be defective.—*T. H. S.*

HYPOFIBRINOGENEMIC SYNDROMES IN POLYCYTHEMIA VERA. *O. Arlotti and G. Ballerini.* From the Istituto di Semeiotica Medica, University, Ferrara, Italy. Arch. Patol. Clin. Med. *33*:233–262, 1957.

Three cases of polycythemia vera with unclottable or hypocoagulable blood are described. The cause of this finding was assigned to the hypofibrinogenemia which should take place as a result of an increased fibrinogen consumption owing to the concomitant presence of a thrombotic diathesis. A relative lack of fibrinogen is assumed to be almost always present in polycythemia vera and is considered as the cause of both the hemorrhagic and thrombotic diathesis.—*P. d. N.*

HEMOPHILIA-LIKE SYNDROME DUE TO CIRCULATING ANTICOAGULANTS IN PEMPHIGUS VULGARIS: PATHOLOGIC-ANATOMIC STUDY. *H. Gasser.* From the Pathologischen Institut, University, Bonn, Germany. Deutsches Arch. klin. Med. *203*:617–629, 1957.

Postmortem findings of a case of *Hemmkörperhämophilie* complicating a pemphigus vulgaris. The findings were not different from those of classical hemophilia. The collagen alterations in pemphigus are considered as significant for the development of the coagulation defect. Three other cases of the literature include postmortem findings.—*P. d. N.*