with congenital heart disease may have prolonged prothrombin times, and these tend to become longer under hypothermia. Bleeding episodes may be due to an increase in thrombin inhibitor. (*Von Kaulla, K. N., and Swan, H.: Clotting Deviations in Man Associated with Open-Heart Surgery During Hypothermia, J. Thoracic Surg. 36: 857 (Dec.) 1958.*)

HYPOFIBRINOGENEMIA Dogs who have fibrinogen levels drastically reduced by intravenous infusion of thrombin at controlled rates do not form intravascular clots, but show marked decrease in fibrinogen levels and a very great reduction in the levels of labile factor and thromboplastinogen. They tend to bleed profusely from venipuncture and have prolonged clotting times and poorly organized clots. It is possible that defective hemostasis in acquired clinical fibrinogenemia may be due primarily to loss of other factors rather than to low levels of fibrinogen. (*Quick, A. J., and others: Occult Intravascular Clotting, J. Lab. & Clin. Med. 52: 935 (Dec.) 1958.*)

AFIBRINOGENEMIA Against the concept that intravascular clotting is a starting point in the etiology of fibrinogenemia is the difficulty of conceiving a mechanism by which tissue thromboplastin could produce thrombin intravascularly at such a rate that all the fibrinogen would be removed by coagulation without causing occlusion of blood vessels. On the other hand intravenous administration of fibrinolysin in both animals and humans produces a decrease both in fibrinogen and in inhibitor levels. It is suggested that while partial intravascular coagulation may occur in a number of obstetrical accidents, most of the drop in fibrinogen is due to its hydrolysis by fibrinolysin. Tissue extracts containing significant fibrinogenolytic activity and activator are found in placental, decidual and myometrial extracts. In *abruptio placentae* these substances may be forced into the maternal blood stream and there may initiate fibrinogenolysis. Clinically, fibrinolysins have been demonstrated in patients who develop hypofibrinogenemia. More significant, however, is the consistently low levels of profibrinolysin and inhibitor which suggests that a fibrinolytic process is involved. (*Phillips, L. L.: Etiology of a Fibrinogenemia: Fibrinogenolytic and Fibrinolytic Phenomena, Ann. New York Acad. Sc. 75: 676 (Jan. 9) 1959.*)

AFIBRINOGENEMIA Afibrinogenemia is diagnosed from a clinical history suggesting abruptio placentae, fetal death, or amniotic fluid embolism, and from the "clot observation test." Failure of blood to clot or fragmentation and dissolution of a clot within an hour of its formation is evidence of relative afibrinogenemia. Initial treatment is directed to identification and correction of the clotting defect and treatment of shock. To deliver a patient before this is done is to invite uncontrolled hemorrhage. Blood transfusion with supplementary fibrinogen are indicated. The amount of fibrinogen administered will vary with the deficit, and serial clot observation tests will indicate the amount of fibrinogen needed. In the average case 1 to 2 liters of blood and 4 to 8 Gm. of fibrinogen are required. With restoration of the clotting mechanism, the patient may be delivered safely. (*Reid, D. E.: Clinical Considerations in Hypofibrinogenemia, Ann. New York Acad. Sc. 75: 685 (Jan. 9) 1959.*)

FIBRINOGENOPENIA Fibrinogenopenia occurs in many acquired hemorrhagic syndromes, particularly, separation of placenta, amniotic embolism, intrauterine fetal death, and attempts at criminal abortion. At the acme of bleeding most cases also had thrombocytopenia, appearance of circulating anticoagulants, and changes in the fibrinolytic system which included appearance of fibrinolysins, fibrinogenolysins and antifibrinolysins. A considerable variation was noted among these factors suggesting that the fibrinogenopenic syndrome may encompass a number of different entities. Hemorrhagic shock alone may produce a similar picture. (*Stefanini, M., and Turpini, R. A.: Fibinogenopenic Accident of Pregnancy and Delivery: Syndrome with Multiple Etiological Mechanisms, Ann. New York Acad. Sc. 75: 601 (Jan. 9) 1959.*)

FIBRINOPENIA Fibrinopenia may be: congenital, associated with infection severe enough to cause inadequate plasma protein production, and produced by fibrinogen re-

moval. One possible mechanism of excessive fibrinogen removal is by activation of fibrinolysins. Profibrinolysin can be activated to fibrinolysin by fibrinokinase, a possible cause of fibrinolytic activity during surgery. Because the gravid uterus contains large amounts of fibrinokinase, effective concentrations may be released from the damaged conceptus into the maternal circulation and initiate fibrinogenolysis. However, although fibrinolysin may attack both fibrin and fibrinogen in purified systems, the antifibrinolysin mechanism in the blood prevents attack on dissolved fibrinogen but not on coagulated fibrin. Thus the fibrinolysin-antifibrinolysin system appears to be well adapted to removal of fibrin formed during fibrination but has a physiological mechanism which minimizes fibrinogenolysis. Another possible mechanism of fibrinogen removal is fibrination-defibrination. During fibrination, fibrin is formed in the blood. Fibrin formation activates fibrinolysin. Hence the blood becomes fibrinopenic but remains liquid and continues to circulate. The depletion of coagulation and thrombus-forming mechanisms may be so severe that a hemorrhagic tendency results. Outstanding examples occur during abruptio placentae, delivery of a hydatid mole, and abortion of late pregnancy. (*Schneider, C. L.: Etiology of Fibrinopenia: Fibrination Defibrination, Ann. New York Acad. Sc. 75: 634 (Jan. 9) 1959.*)

BLOOD CONTAINERS Plastic containers (polyvinyl chloride) made of certain basic plastics or plasticizers release leachable substances exerting a toxic effect on red blood cells shortening their in vitro and in vivo survival. Red blood cells are well preserved in silicone coated glass, but uncoated glass also releases a toxic material. Presence of liquid-air interfaces or the degree of wettability of the container wall have no effect on the degree of red blood cell damage. (*Strumia, M. M., Colwell, S. S., and Dugan, A.: Preservation of Blood for Transfusion. III. Mechanism of Action of Containers on Red Blood Cells, J. Lab. & Clin. Med. 53: 106 (Jan.) 1959.*)

PLASTIC TRANSFUSION SETS A comparison of a disposable plastic transfusion set with the standard rubber and glass reuseable transfusion sets has revealed that the plastic sets reduce the incidence of thrombophlebitis by approximately 50 per cent. It was found impossible to guarantee that the interior of a reuseable set is free of pyrogens. The incidence of air embolism and the risk of infection has been reduced materially, since the advent of the plastic disposable set. (*Jenkins, W. J., and others: Experiences with Disposable Plastic Transfusion-Giving Set, Lancet 1: 139 (Jan. 17) 1959.*)

BANKED BLOOD Stored blood, regardless of the method of preservation, undergoes certain biochemical alterations with the passage of time. To investigate such changes, blood was drawn from 27 normal male volunteers and divided into three groups. In one group, blood was stored in ACD solution in silicone coated vacuum bottles; in the second group in the same solution and stored in plastic bags; and, in the third group run through a cation-exchange resin and then stored in a plastic container. In all three groups, potassium levels continued to rise for 21 days, with levels of about 20 mEq./l. at that time. Ammonium levels responded similarly. Passage of the blood through resin columns reduced the potassium and ammonium to or below normal levels. (*Schechter, D. C., Nealon, T. F., Jr., and Gibbon, J. H., Jr.: Removal of Excessive Potassium and Ammonium from Bank Blood Prior to Transfusion, Surg. Gynec. & Obst. 108: 1 (Jan.) 1959.*)

TRANSFUSION REACTIONS Blood transfusions were given in 81,392 instances at the Kings County Hospital Center between 1952 and 1957. Thirty patients were transfused with incompatible blood. Eighteen of these patients had a hemolytic transfusion reaction for an incidence of 1:4,520. Seven of the 18 patients died. In 5, the appearance of a coagulation defect contributed significantly to death. The conservative management of acute renal failure produced the best results. (*Binder, L. S., Ginsberg, V., and Harmel, M. H.: Six Year Study of Incompatible Blood Transfusions, Surg. Gynec. & Obst. 108: 19 (Jan.) 1959.*)