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-