biochemical processes in the platelet must be functioning. (*Zucker, M. B., and Borrelli, J.: A Survey of Some Platelet Enzymes and Functions: The Platelets as the Source of Normal Serum Acid Glycerophosphatase, Ann. New York Acad. Sc. 75: 203 (Oct. 13) 1958.*)

PROTHROMBIN ACTIVATION Prothrombin is the only necessary activating factor for thrombin. The other clotting factors are seen as aids to thrombin when it exercises its qualities as an enzyme. These include: calcium ions, Ac-globulin, platelet 3 factor, etc. Opposed to these, and almost in exact balance are: heparin, sphingosine, antithrombin, fibrinogen, etc. (*Seegers, W. H.: Is Thrombin the Only Enzyme Involved with Prothrombin Activation?, Ann. New York Acad. Sc. 75: 182 (Oct. 13) 1958.*)

THROMBIN-FIBRINOGEN INTERACTION The conversion of fibrinogen to a fibrin clot in the presence of thrombin (and absence of serum factor and calcium ions) may be formulated in three reversible equations: (1) the proteolysis of fibrinogen in the presence of thrombin to a fibrin monomer; (2) the polymerization of the fibrin monomer to intermediate polymers; and (3) the formation of the fibrin fibril. Each of these equilibriums is studied. Thrombin is necessary only for the first equation and does not take part in the polymerization steps. (*Scheraga, H. A.: Thrombin and Its Interaction with Fibrinogen, Ann. New York Acad. Sc. 75: 189 (Oct. 13) 1958.*)

BLOOD CELLS In sleep a single dose of barbiturates caused leucopenia with a fall of the number of reticulocytes and granulocytes and a slight shift to the left. These changes were well developed after the administration of barbamyl but only slightly so after noctal, nembutal, medinal or sonbutal. In experimentally produced anaemia by repeated bleeding of mice, barbamyl delayed the time of full blood regeneration, whereas other barbiturates had no such effect. (*Runova, M. F.: Effect of Barbiturates on the Blood Picture in Normal Animals and Animals with Experimentally Produced Anaemia, Byull. Eksper. Biol. i Med. Suppl. 1, p. 38–41 1957.*)

TRANSFUSION The development of jaundice in 12 injured and 4 burned subjects soon after transfusion with stored blood has been studied. In 9 injured and 2 burned patients the jaundice was also related to operation and general anesthesia. The jaundice, which generally disappeared within a week, was deemed to be hepatic in origin because it was associated with direct serum van den Bergh reaction, bilirubinuria, urobilinuria, and often a raised serum alkaline phosphatase level. No evidence of blood incompatibility or intravascular haemolysis were noted. It was concluded that the jaundice resulted from a combination of two factors. Temporary hepatic impairment probably resulted from a lowered hepatic circulation produced by injury, burning, subsequent operations, and anesthesia, singly or in combination. An acute loading of the bloodstream with bilirubin derived from extra-vascular destruction of the non-viable red cells in stored blood was superimposed upon this temporary hepatic dysfunction. The blood transfusion appeared, thus, to have acted as a bilirubin-loading test of liver function, and since this was acutely disturbed, jaundice with biliuria appeared. (*Sevitt, S.: Hepatic Jaundice After Blood Transfusion in Injured and Burned Subjects, Brit. J. Surg. 46: 68 (July) 1958.*)

EXCHANGE TRANSFUSION The use of exchange transfusions in the management of an adult with acute renal failure due to a hemolytic transfusion reaction is reported. Relief of convulsions was almost immediate, the level of consciousness was raised, and the patient went on to complete recovery following one exchange. (*Smiley, R. K.: The Use of Exchange Transfusions in Acute Renal Failure, Canad. M. A. J. 79: 740 (Nov. 1) 1958.*)

CEREBRAL BLOOD FLOW Cerebral blood flow (Nitrous Oxide technique) was significantly depressed in 12 of 13 patients with mitral stenosis. Rebreathing of 5 per cent carbon dioxide was followed by increases in cerebral blood flow which were somewhat less than reported to occur in normal volunteer subjects. (*Dewar, H. A., and Davidson, L. A. G.: The Cerebral Blood Flow in Mitral*