FIBRINOLYSIS Fibrin deposition is an integral part of all acute inflammatory reactions, and the fibrinolytic process is best conceived as a basic repair mechanism operative throughout the organism. All mammalian sera contain an enzyme precursor, plasminogen (profibrinolysin). Spontaneously, or in the presence of serum, tissue, urine, or bacterial activators, it is converted to plasmin (fibrinolysin). These activators may conceivably be inhibited by a naturally occurring plasma inhibitor, but definite studies have not been reported. However, free plasmin in blood is inhibited by one or more antiplasmins found in serum and possibly in platelets. Normal lysis of clot is thought to be due to activation of plasminogen which had been adsorbed onto fibrin during clot formation by circulating activator. The increased fibrinolytic activity occurring after exercise, ischemia, pyrogens, electroshock, epinephrine and acetylcholine is also associated with increased levels of circulating plasminogen activator. The release of activator into the circulation is believed to depend on a cholinergic effector mechanism in vessel walls (veins, arteries and capillaries) which respond to ischemia (locally and reflexly) by releasing activator. Hyperplasminemia occurs when the rate of activation of plasminogen exceeds the inhibitory activity of blood. Plasmin hydrolyzes fibrin, but in addition, it will hydrolyze fibrinogen, accelerator globulin, some of the components of complement, ACTH, growth hormone and glucagon. Clinically, this is manifested by hypofibrinogenemia and decreased factor V concentration. (*Alkjaersig, N.: Fibrinolysis and Fibrinolytic Activity in Man, Physiol. Rev. 39: 343 (April) 1959.*)

PLASMINOGEN INHIBITOR Plasminogen in the presence of activators is rapidly converted to plasmin, a proteolytic enzyme active at neutral hydrogen ion concentrations. Activators specific for plasminogen include streptokinase and staphlokinase of bacterial origin, urokinase and plasma activator found in body fluids, and fibrokinase derived from tissue. Arginine and lysine esters are competitive inhibitors of both plasmin and its activators. This study describes the effects of ϵ-aminocaproic acid. The results indicate that its primary action is to inhibit the activation of plasminogen, but it also possesses, depending upon its concentration, the dual property of either inhibiting or enhancing the action of plasmin. (*Alkjaersig, N., Fletcher, A. P., and Sherry, S.: ϵ-Aminocaproic Acid: An Inhibitor of Plasminogen Activator, J. Biol. Chem. 234: 832 (April) 1959.*)

LIPID MOBILIZATION Lipid mobilizer hormone is released during surgical stress, leading to mobilization of triglycerides from the omental depot. There is a shift in paper electrophorograms from alpha-lipoprotein to beta-lipoprotein predominance in omental vein blood during surgery. Omentum also appears to remove cholesterol from blood circulating through it. (*Zarafonetic, C. J., and others: Lipid Mobilization as a Consequence of Surgical Stress, Am. J. M. Sc. 237: 418 (April) 1959.*)

BLOOD LOSS ESTIMATION A comparison is made between the blood loss measured by weighing sponges and that determined by measuring the pre- and post-operative blood volume. To avoid errors due to evaporation, the sponges are weighed immediately after use. There is no correlation between the operative blood loss and the difference between the pre- and post-operative hematocrit. There is a high correlation between the operative blood loss as measured by the gravimetric method and the one estimated through determination of blood volume. Calculations indicate that the value of the blood loss determined gravimetrically, plus 25 per cent, is equal to the true operative blood loss. (*Cáceres, E., and Whittemburg, G.: Evaluation of Blood Losses During Surgical Operations, Surgery 45: 681 (April) 1959.*)

WOUND SHOCK Civilian injuries resemble battle injuries in the extent of blood loss and the uniformity with which this loss is usually underestimated, particularly in closed injuries with fractures of the limbs and trunk. A patient with a normal blood pressure and pulse rate, pink, warm and with good arm veins, may already have lost 30 to 40 per cent of his total blood volume. Severe collapse usually indicates a blood loss of at least 50 per cent. Even when the total blood volume has been returned to 80 per cent of normal by hemodilution or

plasma transfusion, a raised pulse rate may still indicate a lowered red cell volume. (*Clarke, R.: On the Nature and Treatment of Wound Shock, Ann. Roy. Coll. Surgeons England 24: 239 (April) 1959.*)

TRANSFUSION REACTIONS A transfusion reaction exhibits three clinical phases: (1) acute reaction, (2) oliguric phase, (3) diuretic phase. Anesthesia masks most of the classical symptoms of acute transfusion reactions; during an operation, the most prominent sign may be bleeding from cut surfaces. Emergency measures of the acute reaction phase are: (1) Immediate discontinuance of the transfusion. The needle should be left in place for treatment of shock should it develop: intravenous fluids, dextran, or compatible blood. Arterenol may be required in addition. (2) Establishment of the diagnosis. The physician himself should draw blood from the patient, examine it for hemolysis, and have it cross-matched with blood from the incriminated bottle. He should refuse to accept any previously used samples. (3) Immediate intravenous administration of five per cent solution of dextrose in water (two liters) with 300 mEq. of sodium bicarbonate (to prevent blocking of renal tubules with acid heme). (4) Administration of Mannitol, 50 or 100 ml. of a 25 per cent solution, in the hope of increasing urinary flow by osmotic diuresis. Medical management of the oliguric phase includes restriction of fluid intake and prohibition of sodium intake. Protein catabolism is discouraged by a high-calorie, low-protein regimen. Prophylactic antibiotic treatment is indicated. Indications for the artificial kidney are chemical and clinical. Dialysis is an important adjunct to therapy and should be employed relatively early in the oliguric phase before the patient's clinical condition deteriorates. (*Barlas, G. M., and Kloff, W. J.: Transfusion Reactions and Their Treatment, Especially with the Artificial Kidney, J. A. M. A. 169: 1969 (April 25) 1959.*)

AMMONIA INTOXICATION Ammonia in stored blood increases at an average rate of 20 micrograms per 100 ml. per day. The most practical method at present for reducing or controlling ammonia intoxication that may result from massive transfusion in a cirrhotic patient is to administer intravenously arginine simultaneously with the blood as described. (*Britton, R. C.: Ammonia Intoxication from Bank Blood in Patients with Cirrhosis of the Liver, Cleveland Clinic Quarterly 26: 81 (April) 1959.*)

CARDIAC ARRHYTHMIAS Direct coronary artery pressure and flow, coronary sinus flow, and systemic blood pressure were measured in 264 dogs. After control studies, cardiac arrhythmias were induced. Auricular and ventricular premature contractions, paroxysmal auricular tachycardia, auricular fibrillation and flutter, ventricular tachycardia and fibrillation all were found to decrease coronary artery pressure and flow and systemic arterial pressure significantly, particularly when irregular and rapid rates (190 and above) were present. Direct brachial artery pressure measured in humans, showed significant decrease in blood pressure when heart rate was 180 or above. Vasopressor drugs may abolish arrhythmias plus correcting hypotension. On the basis of animal experimental evidence, therapy of cardiac arrhythmias should aim toward rapid correction with vasopressors and later permanent correction should be secured with quinidine and digitalis drugs. Patients with coronary artery disease should have arrhythmias treated to maintain adequate coronary perfusion since both clinical and electrocardiographic evidence of myocardial ischemia may become evident with even very small reduction of coronary artery pressure and flow. (*Corday, E., and others: Effect of Cardiac Arrhythmias on the Coronary Circulation, Annals. Int. Med. 50: 535 (March) 1959.*)

VENTRICULAR FIBRILLATION Fibrillation of the ventricle in the human is produced by an electric charge made within the heart itself. A checkerboard distribution of coronary artery blood produces these electric charges. They do not appear when the heart is uniformly deprived of oxygenated blood, but when only part of the muscle is deprived of oxygenated blood. Conversion of oxygen differentials into electric charges requires further investigation. Hearts with adequate inflow but with checkerboard distribution require even distribution for