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