Only one of these, phosphatidyl serine, which is present in both platelet and red cells but not in plasma, is able to substitute for the whole platelet lipid extract *in vitro* coagulation systems. The coagulant activity of phosphatidyl serine is enhanced by the presence of lecithin. Phosphatidyl ethanolamine, inactive alone, displays some coagulation activity when combined with lecithin and a lesser activity when combined with sphingomyelin. The coagulant activity of the complete platelet lipid extract can be reproduced by employing only the amounts of phosphatidyl serine and lecithin contained in the whole lipid extract. (*Troup, S. B., and others: Thromboplastic Factors in Platelets and Red Blood Cells: Observations on Their Chemical Nature and Function in "In Vitro" Coagulation, J. Clin. Invest. 39: 342 (Feb.) 1960.*)

THROMBOLYSIS Determination of the release of radioactivity from isotopically-labeled human plasma clots immersed in unaltered plasma is a sensitive measure of plasma thrombolytic activity. Using the isotopic clot assay, thrombolytic activity was determined in plasma from healthy adults, adults following stress and following the administration of drugs and from individuals with disease. The results indicate that plasma from normal adults contains a plasminogen activator capable of lysing human plasma clots under conditions similar to those seen *in vivo*. The quantity of this material varies in response to stress, drug administration and disease. (*Sawyer, W. D., and others: Studies on the Thrombolytic Activity of Human Plasma, J. Clin. Invest. 39: 426 (Feb.) 1960.*)

FIBRIN Fibrinogen concentration of adequately heparinized blood is unchanged by severe degrees of agitation for periods approximating surgical cardiopulmonary bypass times. The amorphous material deposited in the extracorporeal circuit is not fibrin removed from heparinized blood by mechanical trauma. The clot-like material occasionally seen on filter screens during experimental extracorporeal circulation is made up chiefly of fragmented red blood cells. (*Gadboys, H. L., and others: The Effect of Mechanical Trauma on Fibrinogen in Heparinized Blood, Ann. Surg. 151: 399 (March) 1960.*)

PAIN Analgesia in humans increases phagocytic activity of leucocytes during severe pain and decreases it in dull pain. These changes are mediated via the influence of the central nervous system. (*Pelts, D. G.: Influence of Pain on Basic Immuno-Reaction. IV. Influence of Pain and of Analgesia on Phagocytosis in Humans, Zh. Mikrob. Epid. i Immunobiol. 10: 70, 1958.*)

CEREBRAL ISCHEMIA Total arrest of cerebral circulation in 55 dogs revealed that all animals subject to periods of cerebral ischemia up to ten minutes recovered completely. Certain transient neurological damage was noted in dogs subjected to a ten-minute period of cerebral anoxia. All dogs died in the immediate postoperative period without awakening after a 14-minute period of circulatory arrest. A greater tolerance to cerebral hypoxia than had previously been reported was accomplished by this method for two reasons: (1) The heart was very well oxygenated during the period of arrest of cerebral blood flow; and (2) A high venous pressure in the brain was prevented by allowing a small venous return through the azygos vein during the period of anoxia. (*Brockman, S. K., and Jude, J. R.: The Tolerance of Dog Brain to Total Arrest of Circulation, Bull. Johns Hopkins Hospital 106: 74 (Feb.) 1960.*)

VENTRICULAR FIBRILLATION Ventricular tachycardia and fibrillation were terminated by externally applied electric countershock more than 532 times in eight patients; five having survived for one month to two and a half years. Prevention of recurrent ventricular tachycardia and fibrillation in patients with complete heart block remains an unsolved problem. Drugs are largely ineffective; indeed, quinidine and procaine amide are contraindicated. External electric cardiac stimulation at rates above the basic idioventricular rate has been effective in preventing these recurrent ventricular arrhythmias, but long-term stimulation is difficult. (*Zoll, P. M., Linethal, A. J., and Zarsky, L. R.*

N.: Ventricular Fibrillation. Treatment and Prevention by External Electric Currents, N. E. J. Med. 262: 105 (Jan. 21) 1960.)

BLOOD OXYGEN TENSIONS A catheter-type P_{O_2} electrode has been developed which permits the polarographic continuous recording of the blood oxygen tension *in vivo*. The electrodes have the size of ordinary cardiac catheters in yield continuous P_{O_2} tracings in the animal over several hours, with a standard deviation of less than three per cent. With changing inspiratory oxygen concentration, the time for reaching a new equilibrium was found to be three to four minutes. While the method has not yet been applied in man there should not be any practical obstacles to doing so. *(Kreuzer, F., and others: A Method for Continuous Recording in Vivo of Blood Oxygen Tension, J. Appl. Physiol. 15: 77 (Jan.) 1960.)*

CONTINUOUS *p*H MEASUREMENT When hypothermia is rapidly inducted by corporeal cooling system and adequate oxygenation is maintained by a respirator, the blood *p*H increases. Myocardial irritability also increases at reduced temperatures. The increased blood *p*H further accentuates the myocardial irritability. Reducing blood *p*H at lowered body temperature reduces myocardial irritability. An accurate monitoring system providing continuous blood *p*H measurements is described. *(Edmark, K. W.: Continuous Blood pH Measurement with Extracorporeal Cooling, Surg. Gynec. & Obst. 109: 743 (Dec.) 1959.)*

OXIMETRY Oximetry is based on the differences between the absorption spectra of reduced and oxygenated hemoglobin. The details of this technique are discussed, particularly the problems arising from the corpuscularity of blood, the choice of wave length and photocell, inhomogeneity of tissues, and the effect of blood flow on transmission of light. The most important difficulty is that the indicated saturation values are not absolutely reliable. This is aggravated by the notorious tendency of selenium cells to show individual variation in their sensitivity to illumination. *(Nilsson, N. J.: Oximetry, Physiol. Rev. 40: 1 (Jan.) 1960.)*

OXYHEMOGLOBIN DISSOCIATION A shift to the right of the oxyhemoglobin dissociation curve had previously been shown to exist in anemic patients, by *in vitro* methods. To confirm its presence *in vivo*, 23 subjects with hemoglobin levels ranging from 3.4 to 13 gm. per 100 ml. were examined. All cases were of chronic anemia with no abnormal hemoglobin present, and no significant cardiopulmonary derangement demonstrated. A steady state was achieved in each patient by allowing him to breathe oxygen for 25 minutes before obtaining arterial samples. To obtain points at the lower end of the wave occasionally mixed venous blood was used. The curve was corrected to *p*H 7.4 Moderate displacement of the curve to the right was demonstrated with hemolobin below 9 Gm. per 100 ml., while below 6.5 Gm. per 100 ml. it was marked. The change is desirable in that it facilitates oxygen uptake by the tissues. *(Rodman, T., Close, H. P., and Purcell, M. K.: The Oxyhaemoglobin Dissociation Curve in Anemia, Annals of Int. Med. 52: 295 (Feb.) 1960.)*

AMINE OXIDASES The amine oxidases are classified according to five general groups, amine oxidase, histaminase, mescaline oxidase, spermine oxidase, and menzylamine oxidase. The substrate specificity of amine oxidase is in a large part dependent upon the source of the enzyme. With the possible exception of spermine oxidase, very little is known about their functions. The only function which may be tentatively ascribed to amine oxidases in general is that they may act to detoxicate amines entering the body from the alimentary canal. This assumption finds support in the high concentration of these enzymes in the intestinal mucous membrane and liver and, in the case of spermine oxidase and benzylamine oxidase, in blood. Much has been made of the relation of the biosynthesis and metabolism of amines in the brain to cerebral activity but it is well to remember that our knowledge of the function of these amines is still so limited as to make it difficult to implicate amine oxidases in these processes. *(Hagen, P., and Weiner, N.: Enzymic Oxidation of Pharmacologically Active Amines, Fed. Proc. 18: 1005 (Dec.) 1959.)*