

toxicity is not a causative factor. (McGee, R., and Tellis, I.: *Ventricular Tachycardia*, *Am. J. Cardiology* 3: 300 (March) 1959.)

ST DEVIATION Experimental data show that electrocardiographic changes characteristic of myocardial ischemia (S-T segment depression) are related largely to a change in the balance between intra- and extracellular electrolytes. This change in electrolyte balance occurs in ischemic heart disease as well as in a wide variety of noncardiac conditions. (Prinzmetal, M., and others: *Angina Pectoris: Demonstration of Chemical Oxygen of ST Deviation in Classic Angina Pectoris, Its Variant Form, Early Myocardial Infarction, and Some Noncardiac Conditions*, *Am. J. Cardiology* 3: 276 (March) 1959.)

CELL VOLUME The maintenance of individual cell volume is dependent on aerobic metabolism, and cells incubated anaerobically increase in volume as solute and water enter the cell. One explanation implicates the mechanisms that normally maintain cellular ionic concentration gradients. Intracellular fluid is high in potassium and low in sodium and chloride concentrations; the opposite is present in extracellular fluid. Since cell membranes are permeable to those ions and to water, maintenance of transcellular ionic gradients requires the expenditure of energy. Specific active transport systems have been described for sodium, potassium and chloride. When metabolism is inhibited, energy for ion transport is no longer available. Sodium which defuses into cells along its concentration gradient can no longer be extruded; while potassium leaks out of cells into the extra-cellular fluid. The result of accumulation of sodium and loss of potassium is that the negative cell membrane potential is reduced, and the negative chloride ion can more easily penetrate the smaller negative surface charge. This results in a greater entry of sodium than loss of potassium by the cell. The net gain of solute by tissue is accompanied by an isotonic gain of water and the tissue becomes swollen. (Leaf, A.: *Maintenance of Concentration Gradients and Regulation of Cell Volume*, *Ann. New York Acad. Sc.* 72: 396 (Feb. 6) 1959.)

HYPERVOLEMIA Intravenous infusions of distilled water, 3 per cent glucose or physiologic saline solution resulted in increase in blood volume of 5 to 8 per cent. Infusion of 6 or 8 per cent dextran solution resulted in a 20 to 35 per cent increase in blood volume. In normal subjects a significant linear relationship was found between the degree of blood volume expansion and the percentage change in cardiac output, stroke output and the change in right ventricular stroke work. In patients with mitral stenosis a significant linear relationship was found only between the degree of blood volume expansion and the percentage change in cardiac output. Relatively small alterations in cardiac function occurring with moderate to marked elevations in right heart filling pressure suggest the importance of factors other than filling pressure in the control of cardiac function in man. (Schnabel, T. G., Jr., and others: *The Effect of Experimentally Induced Hypervolemia on Cardiac Function in Normal Subjects and Patients with Mitral Stenosis*, *J. Clin. Invest.* 38: 117 (Jan.) 1959.)

SHOCK The influence of environmental temperature on the development of shock produced by a clamping technique in rats was studied. When the animals were exposed to different environmental temperatures during the period of limb ischemia, the best survival was obtained with an air temperature of 15 C. When the clamping was carried out at a standard temperature of 27 C. and the rats then transferred to a different temperature just before clamp release, the best survival was obtained near a temperature of 24 C. When chlorpromazine was given at the time of clamp removal, survival was prolonged at air temperature of 20 C. but not at 10 C., 24 C. or 30 C. Changes in humidity had no significant influence on survival. (Haist, R. E., and others: *The Influence of Environmental Temperature and Humidity on Shock Produced by a Clamping Procedure*, *Canad. J. Biochem. Physiol.* 37: 165 (Feb.) 1959.)

HEMORRHAGIC SHOCK Hemorrhagic shock in rabbits showed significant hemostatic alterations. "Hypercoagulability" of the blood, characterized by thrombocytosis, accelerated generation of thromboplastin, and rapidly