PULMONARY CIRCULATION One of the major difficulties in interpreting drug-induced alterations in pulmonary flow is the distensibility of the pulmonary vessels. The pharmacologic agents studied were: (1) respiratory gases, (2) cardiac drugs, (3) autonomic drugs, (4) anesthetic agents, (5) serotonin, and (6) miscellaneous drugs. Evidence indicates that the pulmonary vasculature may respond to drugs independently of the systemic circulation. Hypoxia increases to some extent flow resistance in the pulmonary circulation. Carbon dioxide increases such flow resistance temporarily in the perfused lung but not persistently in intact animals. Acetylcholine decreases pulmonary vascular resistance and serotonin appears to be a potent pulmonary vasoconstrictor. (*Fowler, N. O.: Effects of Pharmacologic Agents on Pulmonary Circulation, Amer. J. Med. 28: 927 (June) 1960.*)

PULMONARY CIRCULATION In man the pulmonary hypertensive response to anoxia is either unchanged or slightly reduced by administration of barbiturates as anesthetics, aminophylline, tolazoline, trimethaphan, and hexamethonium. In animals the hypertensive response to anoxia is resistant to general anesthesia, adrenergic blocking drugs and ganglion blocking drugs. The reduction in pulmonary blood flow in patients anesthetized with cyclopropane is overshadowed by an increase in pulmonary vascular resistance which is not dependent on accumulation of carbon dioxide. Inhalation of ether causes an increase in calculated pulmonary vascular resistance apparently due to pulmonary vasoconstriction. Epinephrine usually causes a rise in pulmonary arterial pressure because of local pulmonary vasoconstriction and cardiac stimulant action. Methoxamine is more likely to cause a fall in pulmonary arterial pressure because it does not cause pulmonary vasoconstriction and usually reduces pulmonary blood flow. No presently available drugs are reliable for reduction of pulmonary hypertension because cardiac and systemic vascular functions are also altered. (*Aviado, D. M.: Pharmacology of Pulmonary Circulation, Pharmacol. Rev. 12: 159 (June) 1960.*)

CARDIAC ARREST Analysis of 157 cases of cardiac arrest led to the conclusion that in 50 of these the sole cause was potassium intoxication induced by the rapid infusion of large quantities of bank blood. Bank blood contains high concentrations of extracellular potassium which increase progressively the longer blood is in storage. Endogenous release of potassium from the liver during hemorrhagic hypotension further increases the circulating potassium concentration. Fresh blood, or blood treated with ion-exchange resin, has a low potassium concentration and is relatively safe. The proper management of cases in which massive blood replacement may be required involves the following considerations: (1) Monitor blood loss so that hypovolemia does not occur. Hemorrhagic hypotension is an intense stimulus for the liberation of epinephrine, which in turn liberates glucose and potassium from the liver. (2) Do not rapidly transfuse ice-cold blood. Selective refrigeration of the heart and great vessels results. Furthermore, refrigeration causes potassium to leave the erythrocytes; this potassium enters the cell again after two to three hours of warming. (3) Since the contractility of the myocardium, which is impaired in potassium intoxication, can often be restored by the administration of digitalis, this drug may be used to antagonize the effects of potassium. (4) While citrate infusion alone does not induce cardiac arrest, it can increase the toxic effect of potassium by temporarily altering the potassium-calcium ratio. During rapid blood transfusion, the injection of calcium salts is advisable. (5) Should cardiac resuscitation become necessary, isopropylarterenol (Isuprel) rather than epinephrine should be used as a cardiac stimulant. The direct cardiac effect of isopropylarterenol is ten times that of epinephrine, and furthermore, isopropylarterenol has practically no influence on the liberation of glucose and potassium from the liver. (*LeVeen, H. H., and others: Hemorrhage and Transfusion as Major Cause of Cardiac Arrest, J.A.M.A. 173: 770 (June 18) 1960.*)

ERYTHROCYTES AND ION TRANSPORT An adenosine triphosphatase (ATPase) activity in broken human erythrocyte membranes has been identified as part of an active

transport system for sodium outward and potassium inward across the membranes of intact erythrocytes. The ATPase and active sodium and potassium transport have the following unusual group of properties in common. (1) Both were located in the membrane. (2) Both utilized adenosine triphosphate (ATP) in contrast to inosine triphosphate. (3) Both required sodium and potassium ions together. Either sodium or potassium ion alone was ineffective. (4) Potassium ion activation was completely inhibited by high concentrations of sodium ion in both systems. (5) Ouabain inhibited both systems. (6) Ammonium ion substituted for potassium ion but not for sodium ion in both systems. (7) The concentrations at which sodium ion, potassium ion, ouabain, and ammonium ion showed half of their maximal effects were the same in both systems. The identification of this ATPase activity as part of a transport system implies that sodium and potassium ions are more than simple cofactors; as substrates for transport they should be moved from one part of the system to another at a rate dependent on the rate of dephosphorylation of ATP. The movement is not apparent, of course, in a suspension of broken membranes. The identification suggests that the reciprocal competitive inhibition between sodium and potassium may mean that on each side of the membrane the linked transport system must free itself of one substance as transported product before it can take on the other as transportable substrate. (*Post, R. L., and others: Membrane Adenosine Triphosphatase as Participant in Active Transport of Sodium and Potassium in Human Erythrocyte, J. Biol. Chem. 235: 1796 (June) 1960.*)

BUFFERING OF BLOOD PLASMA Buffering of blood plasma in man depends upon red blood cells, chemical buffers in blood and tissue cells, respiratory system, and the kidneys. The role of each in maintaining blood plasma *p*H is examined in detail. At a constant P_{CO_2}, the total blood buffer value is 76.8 mEq./l. for a change in one *p*H unit. Of this total, 3 per cent is due to plasma bicarbonate, 28 per cent to other chemical buffers in the blood, and 69 per cent to the presence of a gas phase. When the alveolar ventilation changes in response to variations in *p*H and P_{CO_2}, the blood buffer value increases substantially. The tissues and kidneys have the capacity to contribute substantially to the buffer value of blood, but they react more slowly than the respiratory system and chemical buffers in the blood. (*Gilbert, D. L.: Buffering of Blood Plasma, Yale J. Biol. & Med. 32: 378 (April) 1960.*)

PUMP-OXYGENATOR In 1926 S. S. Broukhonenko devised a machine for sustaining life in mammals while cardiopulmonary bypass was performed. It consisted of two diaphragm pumps and a donor lung which was rhythmically inflated while being perfused by one of the pumps. It was used successfully three times. The inventor suggested that such machines could be perfected for the uses to which they are in fact being presently put. (*Probert, W. R., and Melrose, D. G.: Early Russian Heart-Lung Machine, Brit. Med. J. 1: 1047 (April 2) 1960.*)

PUMP-OXYGENATOR A pump-oxygenator system requiring no blood for priming adds greatly to the safety of, and makes possible a wider application of heart-lung bypass procedures. The patient's blood is collected, oxygenated, and returned through a low volume extra-corporeal circuit in which little trauma occurs. (*Neptune, W. B., Bougas, J. A., and Panico, F. G.: Open-Heart Surgery Without Need for Donor-Blood Priming in Pump-Oxygenator, New Eng. J. Med. 263: 111 (July) 1960.*)

CARDIAC SURGERY For 71 operations on the heart and great vessels a 0.1 per cent solution of Arfonad (trimetaphen) in 5 per cent glucose was injected by the drip method. The use of ganglion-blocking drugs in operations on the heart and great vessels is justified by the possible prevention of operative shock, the lowering of the blood pressure and by the creation of satisfactory conditions for the surgeon. (*Meshalkin, E. N., and Stadnikova, E. I.: Ganglion Block in Operations on Heart and Main Blood Vessels, Khirurgiya 9: 3, 1959.*)

TRANSAMINASE The serum glutamic oxaloacetic transaminase levels in 80 patients, undergoing cardiac repair and with total body perfusion, were markedly increased when po-