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