tion of the arrhythmia may produce full compensation and clearing of symptoms. Oral quinidine is the drug of choice for conversion. A slower method using quinidine gluconate (Quinaglute Dura-Tabs) 0.65 Gm., every six to eight hours for one to 21 days until conversion occurs, then usual maintenance doses, removes the need to stop treatment because of drug reaction. (*Brill, I. C.: Changing Concepts in the Treatment of Sustained Atrial Fibrillation, Dis. Chest 41: 334 (Mar.) 1962.*)

HYPOGLYCEMIC ARRHYTHMIAS Severe cardiac arrhythmias may follow insulin-induced hypoglycemia in diabetic patients. This has been attributed solely to the accompanying hypokalemia. It was suggested that the hypokalemia increased the sensitivity of the myocardium to elevated levels of circulating epinephrine, the latter associated with the adrenal medullary discharge of hypoglycemia. Intravenous glucose promptly converted this arrhythmia in one case in spite of a continuing low serum potassium level (3.1 mEq./liter), presumably by causing an abrupt reduction in adrenal output of epinephrine. (*Leak, D., and Starr, P.: Mechanism of Arrhythmias During Insulin-Induced Hypoglycemia, Amer. Heart J. 63: 687 (May) 1962.*)

ARRHYTHMIA The observed frequency of cardiac arrhythmias, excluding tachycardia and bradycardia of sinus origin, in 569 unselected surgical patients during anesthesia was 29.9 per cent. The most frequent arrhythmia was atrioventricular dissociation with ventricular premature contractions ranking second. Preexisting heart disease was the most important factor. They occurred less frequently with thiopental and occurred more frequently during intra-abdominal operations. (*Dodd, R. B., and others: Cardiac Arrhythmias Observed During Anesthesia and Surgery, Surgery 51: 440 (Apr.) 1962.*)

PULMONARY CIRCULATION Constant infusion of acetylcholine directly into the pulmonary artery in patients with various forms of chronic pulmonary disease showed small changes in pulmonary artery pressure and pulmonary resistances. Arterial oxygen saturations did not fall. Possibly anatomic factors are more important in regulating ventilation perfusion than vasoconstriction. This study argues against the thesis that hypoxia in areas of lung poorly ventilated induces vasoconstriction. In advanced pulmonary disease, as in mitral stenosis, maldistribution of blood flow has become primary and overshadows uneven ventilation. (*Charms, B. L., and others: Effect of Acetylcholine on the Pulmonary Circulation in Patients with Chronic Pulmonary Disease, Circulation 25: 814 (May) 1962.*)

PULMONARY CIRCULATION Unilateral pulmonary artery occlusion is followed by homolateral bronchial constriction, reducing ventilation to the affected side and increasing it on the other and minimizing ventilation of nonperfused alveoli. This bronchoconstriction is initiated by the fall in P_{CO_2} in the alveolar air of the affected lung when no longer perfused by mixed venous blood. This mechanism reduces useless ventilatory effort in unilateral embolization but has serious implications for the patient with bilateral emboli. The reduction in ventilation of the affected lung falls to such an extent that there is no ventilation after one to two days though it almost returns to normal in a month. The roentgenographic and pathological findings of congestive atelectasis coincide with these variations in ventilation. The lungs of infants dying of hyaline membrane disease resemble these lungs and those with atelectasis produced in a variety of ways including bronchial occlusion and postperfusion atelectasis. Extracts of lungs atelectatic because of all these insults also showed a marked reduction in the surface active principle which reduces surface tension in the undistended state, thus keeping alveoli open, and which by reducing surface tension also reduces the pressure necessary to inflate the lung. This surface active agent appears to be the phospholipid portion of a lipoprotein. (*Comroe, J. H., Jr.: Pulmonary Arteral Blood Flow, Amer. Rev. Resp. Dis. 85: 179 (Feb.) 1962.*)

MUSCLE BLOOD FLOW Muscle blood flow in the calf and forearm was examined with continuous heat conductivity measurement in healthy subjects during intravenous and intra-arterial injections of epinephrine.