VASOACTIVE POLYPEPTIDES Recently a number of polypeptides exhibiting vasoactive properties have been isolated, purified, synthesized, and their biologic activities intensively studied. Examples of vasopressor polypeptides are: Angiotensin II and related peptides, pepsitensin, oxytocin, vasotocin and the vasopressins. Vasodepressor substances are: bradykinin, kallidin II, substance P, substance U and peptide B. It is apparent that there is a structure–function relationship among these compounds but its specific nature is as yet not known. Some of these substances may be important in the physiologic regulation of blood pressure in man. (*Wolf, R. L.: Vasoactive Polypeptides, Amer. Heart J. 64: 427 (Sept.) 1962.*)

QUINIDINE Quinidine is frequently misused in the treatment of auricular fibrillation. The fact that a patient experiences no unfavorable reactions following a test dose of quinidine is no indication that he may safely be given almost any multiple of the initial dose at frequent intervals without the risk of serious toxic complications. Convulsions, shock-like syndromes, and unexplained sudden deaths accompany this form of therapy, sometimes many hours after the last dose is given. There are wide variations in the tolerance to this drug by individual patients. The fibrillating heart is not a fully compensated heart but a more or less depressed one. Quinidine is a myocardial as well as a respiratory depressant. Treatment of auricular fibrillation with quinidine means the addition of a myocardial and respiratory depressant to an already embarrassed myocardium and a physiologically impaired respiratory center. This results in serious toxic complications such as cardiac standstill, respiratory paralysis and so-called unexplained sudden deaths. The heart must first be "conditioned." This is done by the use of digitalis, which is a myotonic agent. The more completely that restoration is accomplished in this fashion, the smaller will be the amount of quinidine required to restore normal sinus rhythm without the danger of toxic effects. (*Weisman, S. A.: Misuse of Quinidine in the Treatment of Auricular Fibrillation, Geriatrics 17: 421 (July) 1962.*)

OCTAPRESSIN Octapressin is a new synthetic analogue to vasopressin, a polypeptide hormone with a very marked vasoconstrictive action. It has been used as an additive to local anesthetic solutions and also for elevation of systemic blood pressure. It was given intranvenously during anesthesia with cyclopropane, trichlorethylene and halothane. No arrhythmias were seen. (*Huegin, W.: Compatibility of Octapressin with Different Anesthetics, Der Anaesthesist 11: 185 (June) 1962.*)

MEPHENTERMINE Dual hemodynamic effects of mephentermine were demonstrated in a group of 19 human subjects. These effects were a primary positive inotropic and chronotropic effect followed by a secondary action causing an increase of the total peripheral resistance. The dual effects have a different time sequence of onset. During the first 5 to 20 minutes the increase in arterial blood pressure is due primarily to the increase in the cardiac output and in the latter period, the vasopressor response is due mainly to an increase in total peripheral resistance. A significant lowering of the arterial blood carbon dioxide tension and an increase in the *p*H, presumably owing to central stimulation, followed mephentermine administration. (*Li, T.; Shimosato, S., and Etsten, B.: Hemodynamics of Mephentermine in Man, New Engl. J. Med. 276: 180 (July 26) 1962.*)

TACHYPHYLAXIS Studies on isolated rat hearts previously perfused with tritium (H^3)-labelled noradrenaline show that tyramine releases catecholamine from the heart stores. Repeated injection of small doses of tyramine releases progressively smaller amounts of catecholamine. Similar tests on reserpinized hearts gave accelerated depletion of the labelled catecholamine. Tachyphylaxis under these conditions is undoubtedly due to progressive diminution of heart catecholamine content with serial injections of the releasor drug. (*Axelrod, J., and others: Mechanism of Tachyphylaxis to Tyramine in the Isolated Rat Heart, Brit. J. Pharmacol. Chem. 19:56 (Aug.) 1962.*)