the untoward side effects of the latter. (*Chilton, N. W., Lewandowski, A., and Cameron, J. R.: Double-Blind Evaluation of New Analgesic Agent in Post-Extraction Pain, Amer. J. Med. Sci. 242: 702 (Dec.) 1961.*)

PRESSOR AMINES A new technique for simultaneously measuring total myocardial blood flow and cardiac output with the (dog) heart intact and functioning in a physiological manner has been developed using radioiodinated (I^{131}) human serum albumin. When given to hypovolemic dogs, norepinephrine caused a substantially greater blood flow through the myocardium than did any other amine tested. This ability of norepinephrine to increase myocardial flow was followed in order of decreasing effectiveness by metaraminol, epinephrine, and phenylephrine. Since there was little or no change in myocardial blood flow when phenylephrine was used to raise blood pressure, it can be assumed that this drug causes about the same degree of vasoconstriction in the coronary arteries as it does in the systemic vessels. In contrast, the effects of norepinephrine, metaraminol, and epinephrine on myocardial blood flow are due to coronary vasodilation as well as to systemic vasconstriction. (*Dunn, H. K., and others: Effect of Pressor Amines on Myocardial Blood Flow in Dog, J. A. M. A. 178: 1090 (Dec. 16) 1961.*)

NOREPINEPHRINE After the intravenous administration of H^3-norepinephrine, it was taken up by the heart and slowly released over a period of days. Long-acting monoamine oxidase inhibitors blocked the slow release of H^3-norepinephrine. Long-acting and short-acting monoamine oxidase inhibitors blocked the releasing action of reserpine on H^3-norepinephrine in the heart. On the basis of these observations it is proposed that monoamine oxidase inhibitors elevated the catechol-amine concentration in certain tissues by blocking the release of the hormone from its binding site. (*Axelrod, J., Hertting, G., and Patrick, R. W.: Inhibition of H^3-Norepinephrine Release by Monoamine Oxidase Inhibitors, J. Pharmacol. Exp. Ther. 134: 325 (Dec.) 1961.*)

NOREPINEPHRINE UPTAKE Tyramine, amphetamine, cocaine, chlorpromazine, imipramine, reserpine, guanethidine and dibenzyline (phenoxybenzamine) markedly reduced the concentration of administered H^3-norepinephrine in the heart, spleen and (except for guanethidine) adrenal gland. These drugs lowered the H^3-catechol-amine concentration to a moderate degree in the liver. Imipramine was the only drug that lowered the H^3-catechol-amine in skeletal muscle. The level of H^3-normetanephrine was decreased by all these drugs in heart and spleen and in some cases in the adrenal gland and liver. Tyramine, amphetamine, cocaine, chlorpromazine, imipramine, reserpine, guanethidine and dibenzyline also elevated the plasma levels of administered H^3-norepinephrine for the first 5 minutes. The plasma levels of H^3-normetanephrine were also raised to varying degrees. These drugs appear to be acting by preventing the entry and/or the binding of H^3-norepinephrine. The following drugs had no significant effect on the tissue and plasma concentration of H^3-norepinephrine and H^3-normetanephrine: regitine (phentolamine), dichlorisoproternol, TM 10 B, hexamethonium and ouabain. (*Hertting, G., Axelrod, J., and Whitby, L. G.: Effect of Drugs on the Uptake and Metabolism of H^3-Norepinephrine, J. Pharmacol. Exp. Ther. 134: 146 (Nov.) 1961.*)

CATECHOL-AMINE UPTAKE Epinephrine and norepinephrine can be stored in tissues other than those in which they were synthesized. Most of the accumulation is in sympathetic nerve endings, which have a storage capacity considerably greater than their normal catechol-amine content. Quantitative aspects of this uptake suggest that it may play an important role in terminating the actions of exogenous and endogenous catecholamines. (*Strömblad, B. C. R., and Nickerson, M.: Accumulation of Epinephrine and Norepinephrine by Some Rat Tissues, J. Pharmacol. Exp. Ther. 134: 154 (Nov.) 1961.*)

MEPHENTERMINE Mephentermine, in humans with mitral stenosis or chronic pulmonary emphysema, caused bronchodilatation, pulmonary vasodilatation, direct stimulation of the respiratory center, and an increased cardiac output through myocardial stimulation. (*Barrera, F., and others: Cardiovascular-Re-*