TOXICITY The influence of hydergine, chlorpromazine, promethazine and meperidine on the toxicity of procaine, procaine amide and tetracaine was studied in 860 mice. Different combinations of chlorpromazine, meperidine and promethazine increase the toxicity of procaine. Addition of hydergine causes a lesser increase of toxicity. The classical cocktail also increases the toxicity of tetracaine. (*Wellhöner, H. H., and others: Change of Toxicity of Procaine Amide and Tetracaine by Components of Lytic Mixtures. Der Anaesthesist 8: 235 (Aug.) 1959.*)

VAGAL INHIBITION The action of morphine (0.1 to 100 mg./kg.) on the cardiac slowing produced by stimulation of the right vagus nerve varied in the four species studied. In the guinea pig, morphine had no inhibitory effect and in the cat the effect was small, but in the rat and the rabbit stimulation of the vagus slowed the heart much less after administration of morphine than before. In the last two species morphine delayed the onset of cardiac slowing. Nalorphine, which, given alone had a slight morphine-like action, partly reversed the effect of morphine. (*Kosterlitz, H. W., and Taylor, D. W.: Effect of Morphine on Vagal Inhibition of Heart, Brit. J. Pharmacol. 14: 209 (June) 1959.*)

PRESSOR AMINES Methylphenidate, a central nervous system stimulant, can alter the pressor responses in the dog to a series of phenylalkylamines. The amines are affected in three different ways. They may be (1) potentiated or not altered, (2) reversibly blocked, or (3) irreversibly blocked. Differences in structure of the phenylalkylamines may be some of the factors involved. (*Povalski, H. J. and Goldsmith, E. D.: Effect of Methylphenidate on the Cardiovascular Actions of Pressor Amines, Proc. Soc. Exp. Biol. & Med. 101: 717 (Aug.–Sept.) 1959.*)

ISOPROTERENOL Six patients with complete heart block and one with a 2:1 block were given graded doses of isoproterenol and data collected with dynamic recording systems to evaluate the hemodynamic effects of the drug, in contrast to the already known effects on the cardiac conduction system. After isoproterenol average cardiac index increased from 2.45 liters to 3.58 liters. Average resting ventricular rate increased from 40.7 to 45.8 beats per minute. Stroke volume increased on an average of 32.2 per cent. Average left ventricular work rose from 4.52 to 7.19 kg.-meters/minute. Left ventricular stroke work was also increased. The data would indicate that sublingual isoproterenol has both a chronotropic and inotropic effect on both heart block and normal hearts. (*McGaff, C. J., Cohen, H. K., and Leight, L.: Hemodynamic Effects of Isoproterenol in Complete Heart Block, Arch. Int. Med., 104: 242 (August) 1959.*)

CATECHOL AMINES Both local and systemic lesions can be produced by the administration of epinephrine and norepinephrine. Two different mechanisms are responsible for pathologic changes. One is mechanical, due to hemodynamic factors; the other is chemical, producing derangement in cellular oxidation by uncoupling oxidation phosphorylation. Best examples of the first mechanism are the massive cerebral hemorrhage and hemorrhage into the mitral valve. These lesions depend more on the rapidity of infusion than on total dose. The second mechanism is best illustrated by the myocardial necrosis following prolonged dosages of norepinephrine, not associated with marked changes in blood pressure. Hemodynamic changes combine with the hypoxiating effects of norepinephrine in the production of lesions of the liver, kidneys, and large vessels. In the production of skin necrosis, the additional factors of hydrostatic pressure of the infiltrating fluid and local anatomic peculiarities are important. (*Szakacs, J. E., and others: Pathologic Implication of Catechol Amines, Epinephrine and Norepinephrine, U. S. Armed Forces Med. J. 10: 908 (Aug.) 1959.*)

PREMEDICATION The administration of morphine 10 mg. and scopolomine 0.4 mg., one and a half hours prior to anesthesia, produces an air of indifference with amnesia without circulatory and respiratory depression. This is superior to preanesthesia sedation from barbiturates. Because of its hypotensive action, the authors are reluctant to anesthetize anyone who has received chlorpromazine or any of its allies of similar potency within 48