treatment of angina and cardiac arrhythmias, may precipitate cardiac failure, produce bradycardia or precipitate asthmatic attacks. Isoprenaline, an effective and potent beta-receptor agonist, is not readily available. Orciprenaline, also a stimulator of beta-receptors, is readily available. Used intravenously, it is 10 to 40 times less potent than isoprenaline. Propranolol does not antagonize the positive inotropic action of digitalis and rapid digitalization may be of benefit in failure caused by blockade. Atropine may be of value in reducing parasympathetic tone and severe bradycardia associated with blockade especially under anesthesia. Clinically, increase in heart rate may be useful in judging the adequacy of dosage with orciprenaline. The drug also causes increased cardiac contractile force and forearm blood flow. (*Shanks, R. G., and others: Stimulation of Adrenergic Beta-receptors by Orciprenaline, Brit. Med. J. 1: 610 (March) 1967.*)

CARDIAC TRANSPLANTATION Performance of the autotransplanted canine heart (which is accompanied by complete extrinsic autonomic denervation) is compared with the normal canine heart. The transplanted heart was able to achieve a level of performance comparable to the normal heart. The primary difference between the two preparations was the decreased efficiency of the autotransplanted heart as demonstrated by increased total left ventricular oxygen consumption at comparable level of work. In addition, the normal dogs showed no net change in circulating catecholamines across the coronary bed whereas autotransplants always showed a net uptake of catecholamines. This alteration in utilization of circulating catecholamines may aid the hemodynamic adaptation to the cardiac denervation. (*Pagget, W. M., and others: Work Capacity and Efficiency of the Autotransplanted Heart, Circulation 35 (Supp. 1): 96 (April) 1967.*)

BETA ADRENERGIC BLOCKAGE Beta adrenergic blocking drugs such as propranolol inhibit the positive chronotropic and inotropic action of epinephrin on the heart. The metabolic effects of epinephrin, rise of blood sugar and liberation of free fatty acids, are likewise blocked. The decrease of circulating eosinophils by intramuscular injection of 0.3 mg. epinephrin averages 55 per cent. This effect could be inhibited if 10 mg. propranolol was given intravenously 10 minutes before injection of epinephrin. Instead, eosinophils increased by an average of 21 per cent. It is concluded that the decrease of eosinophils after epinephrin is probably mediated by adrenergic beta receptors located in the hypothalamic area or in the pituitary gland. (*Braunsteiner, H., and Dienstl, F.: Inhibition of Eosinopenia after Epinephrin by Means of Beta Adrenergic Blockage, Klin. Wschr. 45: 48 (Jan.) 1967.*)

MYOCARDIAL INFARCTION The measurement of blood levels of activity of certain enzymes has helped in the diagnosis of myocardial infarction. As little as one gram of infarcted heart muscle may be detected while enzyme elevations are generally not found after reversible myocardial ischemia. The principle tests presently in favor include: (1) Serum glutamic oxalocetic transaminase (SGOT) rises in 6–12 hours, peaks in 24–48 hours and returns to normal by the fourth to seventh day after infarct. (2) Lactic Dehydragenase (LDH) rises in 12–24 hours, peaks in 2–4 days and returns to normal by the eighth to fourteenth day. (3) Serum hydroxybutyrate dehydrogenase (SHBD). This modification of enzyme activity has been proposed as the most useful enzyme test available for the confirmation of myocardial infarction. (4) Heat stable LDH proposed as more specific than LDH for myocardial damage. (5) Serum creatine phosphokinase (SCPK) rises in 6 hours, peaks at 12 to 24 hours, returns to normal several days post infarct. All of these enzymes tests are somewhat nonspecific and critical clinical judgment is essential. The present concensus indicates that SGOT and SCPK are the tests of choice in the first two days after suspected infarction; LDH is a standard routine for later evaluation (10–14 days); while increasing emphasis is being placed upon SHBD and stable LDH as reflections of the more cardiac specific components of LDH isoenzymes. (*Hamolsky, M. W.: Editorial—Enzymes in Acute Myocardial Infarction, Circulation 35: 427 (March) 1967.*)