a thiopental-nitrous oxide-oxygen-halothane sequence is simple and safe. However, during heart block, care must be taken during intravenous induction of anesthesia; and nitrous oxide and oxygen alone is safer, with a succinylcholine drip to provide relaxation and apnea. After pacing is satisfactory, halothane may be used safely in low concentration. (*Howat, D. D. C.: Anaesthesia for the Insertion of Indwelling Artificial Pacemakers, Lancet 1: 855 (Apr. 20) 1963.*)

CARDIAC DENERVATION Total extrinsic cardiac denervation was carried out on dogs. Their responses to hypoxia and hypercapnia were then tested and compared to those of normal dogs. Both groups had similar responses to hypercapnia: reduced blood pressure, heart rate and myocardial contractile force. During hypoxia the control dogs had the usual changes: elevated blood pressure, heart rate, and myocardial contractile force. The animals with cardiac denervation, however, had minimal responses to hypoxia. (*Greenfield, L. J., and Ebert, P. A.: Cardiac Denervation Effect in Hypoxia and Hypercapnia, Arch. Surg. 87: 717 (Nov.) 1963.*)

CARDIAC SYMPATHETICS The isolated heart can synthesize norepinephrine and the enzyme O-methyl transferase can inactivate norepinephrine. Norepinephrine at the sympathetic nerve ending is not in a homogeneous store but is partitioned into several pools or functional compartments. Guanethidine is able to block the adrenergic neuroeffector junction and is not dependent on the depletion of tissue norepinephrine stores, while these stores have to be lowered markedly in order for reserpine to block the effects of stimulating sympathetic nerves. Guanethidine releases free norepinephrine into the coronary venous blood, but its norepinephrine-depleting action is not dependent on this property. Reserpine, on the other hand, lowers tissue norepinephrine stores more rapidly than does guanethidine but does not release norepinephrine, as the free amine, into the circulation. (*Braunwald, E., and others: Studies on the Function of the Adrenergic Nerve Endings in the Heart, Circulation 28: 958 (Nov.) 1963.*)

CARDIAC SYMPATHETICS Norepinephrine is formed continuously regardless of sympathetic tone and is stored inside a lipoid membrane. The amine is present in two pools—a reserve pool in granules in equilibrium with a mobile pool which is maintained against a concentration gradient by active transport. Monamine oxidase (MAO) controls the amount of norepinephrine in the nerve endings so that at the steady-state level the amine does not freely diffuse onto receptor sites. In the absence of sympathetic tone, norepinephrine can leave the storage compartments by simple diffusion through the lipoid membrane onto the receptor sites and reaches the blood stream in the form of bases. (*Brodie B.: Recent Views on Mechanisms for Lowering Sympathetic Tone, Circulation 28: 970 (Nov.) 1963.*)

HYPOTENSION Circulatory reflexes of man have been investigated by standard procedures, such as intra-arterial measurement of pulse pressure curves, tilt table, and the Valsalva maneuver. These reflexes were found to be absent in neuritis due to alcohol, porphyria, and infective polyneuritis. Acute loss of circulatory reflexes was found in alcoholic intoxication and in poisoning due to barbiturates and drugs used in psychotherapy. Cerebrovascular accidents also caused acute interruption of the reflex pathways. Severe hypotension in the supine position was usual in the acutely ill group, was precipitated in chronic neuritis by minor decreases in blood volume, artificial respiration and therapeutic doses of hypnotics or drugs used for psychotherapy. (*Barraclough, M. A., and Sharpey-Schafer, E. P.: Hypotension from Absent Circulatory Reflexes, Lancet 1: 1121 (May 25) 1963.*)

NERVOUS SYSTEM AND SHOCK There are predictable effects at various sites of the central nervous system in "reversible" and "irreversible shock." There is a roughly linear depression on both monosynaptic and multisynaptic spinal reflex responses with graded hemorrhagic hypotension such that clear reduction of evoked responses is evident at 50 mm. mean arterial pressure and nearly complete loss of excitability at 30 mm. of mercury. This depression is reversed by reinfu-