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-

sion of blood in animals destined to recover while it is not in those that ultimately go on to die, in spite of the well-known temporary restoration of blood pressure in these doomed animals. This phenomenon correlated well with the degree of oxygen debt of tissues deprived of adequate perfusion. Brain stem reticular formation inhibitory influence on spinal reflexes is relatively resistant to homorrhagic hpyotension, whereas the reticular formation facilitatory influence is more vulnerable. Carotid sinus mechanism is a shock resistant, high-gain servomechanism which is not significantly impaired at mean arterial pressures well under 50 mm. of mercury. (*Peterson, C. G., and Haugen, F. P.: Hemorrhagic Shock and the Nervous System, Amer. J. Surg. 106: 233 (Aug.) 1963.*)

FLUID VOLUME CONTROL Moderate changes of blood volume primarily affect the low-presusre system, which contains 80 to 85 per cent of the total blood volume and is 100 to 200 times as distensible as the arterial tree. The very distensible pulmonary vascular bed and the chambers of the heart are part of the low-pressure system. Adequate filling of these components assures fast adaptability of left ventricular performance to varying loads. Homeostatic control of the "fullness of the blood stream" is predominantly achieved by an adjustment of volume to anatomical size of the vascular bed rather than by active contraction or relaxation of the tension of the vascular smooth muscles. Changes of total blood volume are related to intravascular pressures throughout the low-pressure system of the circulation. With changes in total blood volume or the distribution of blood, usually an increase in intrathoracic blood volume occurs together with diuresis while a decrease is always associated with oliguira. Mild volume loss almost exclusively involves the central baroreceptors of the low-pressure system, which inhibit water excretion. Severe volume loss impairs cardiac performance and involves the arterial baroreceptor regions. At this stage, reflex changes of renal hemodynamics and aldosterone secretion induce sodium retention in addition to water retention. In circulatory shock with severe blood loss, the kidney shuts down, and the capacitance vessels constrict. (*Gauer, O. H., and Henry, J. P.: Circulatory Basis of Fluid Volume Control, Physiol. Rev. 43: 423 (July) 1963.*)

CARDIAC OXYGEN CONSUMPTION Oxygen consumption of the heart is correlated with the product of pulse frequency times mean blood pressure, the normal value being 7,000. The value is an index for coronary perfusion. *Gross, D.: Practical Significance of the Formula Pulse Frequency Times Blood Pressure in Judging the Oxygen Consumption of the Heart, Z. Kreislaufforsch. 52: 770 (Aug.) 1963.*)

CARDIAC OUTPUT Cardiac output can be measured by the Fick method using carbon dioxide. Carbon dioxide output is measured volumetrically. With this method arteriovenous carbon dioxide difference is found to be high and values for cardiac output therefore low because there is no emotional stirring up by this method. The critical point is achievement of a constant alveolar ventilation by the rebreathing technique before recirculation takes place. A special formula can be used to find the needed concentration for the rebreathing of carbon dioxide which must be higher than the venous value. (*Ulmer, W. T., Berta, G., and Berkel, H. A.: Estimation of the Cardiac Output With the Carbon Dioxide-Rebreathing Technique, Arch. Kreislaufforsch. 41: 292 (Sept.) 1963.*)

HYPOXIA Cardiovascular effects of hypoxia were studied with methods that kept arterial blood *p*H constant and recorded blood oxygen tension, which was varied. The acute circulatory crisis which supervenes when men or animals are suddenly exposed to arterial oxygen tensions below 44 mm. of mercury was not reproducible unless blood having a P_{O_2} below 45 mm. of mercury reached the common carotid arteries. Arterial perfusion of the carotid arteries, but not of other vascular territories, with moderately hypoxic blood caused reflex bradycardia, with simultaneous constriction of systemic arteries and veins, and acute severe heart failure. (*Salisbury, P. F., and others: Circulatory Effects of Arterial Hypoxia, Aerospace Med. 34: 935 (Oct.) 1963.*)