pressor drugs may often terminate cardiac arrhythmias such as premature systoles, atrial and ventricular tachycardias, atrial fibrillation, sinus bradycardia and heart block when they are associated with hypotension. If hypotension results from tachycardias in patients with severe coronary-artery disease, vasopressor therapy should be instituted as soon as vagal-stimulating measures fail to terminate the arrhythmia. Even if vasopressor drugs fail to terminate a tachycardia, they have a salutary effect because they sustain the coronary blood flow until other antiarrhythmic agents take effect. (*Gold, H., and Corday, E.: Vasopressor Therapy in Cardiac Arrhythmias, New England J. Med. 260: 1151 (June 4) 1959.*)

VASOPRESSORS The induction of hemorrhagic shock in the experimental dog is accompanied by an initial fall in blood flow to brain, heart, liver, bowel, and kidney followed promptly by a "compensatory" rise in coronary, hepatic, and usually in carotid artery flows and further fall in renal and portal vein flow. The administration of vasopressors (norepinephrine, metaraminol) at this time further increases carotid, coronary, and hepatic artery flow, but renal and portal vein flows fall even further. This is probably the most economical redistribution of the circulation during the hypotensive period of shock, although it must be assumed that the use of vasopressors increases the danger of renal and intestinal necrosis. (*Williams, J. H., and Corday, E.: Rationale of Vasopressor Treatment of Cerebrovascular Insufficiency and Coronary Insufficiency, Dis. Chest 35: 561 (May) 1959.*)

EPINEPHRINE Epinephrine (E) and norepinephrine (NE) exhibit weak vasoconstrictor effects when applied to the cerebral cortex. Given intravenously their pressor effect overcomes cerebral arterial vasoconstriction and produces passive dilation. E may cross the blood-brain barrier sufficiently to produce slight central effects. E and NE increase the activity of carotid and aortic baroreceptors both by direct stimulation and sensitization, and by raising the blood pressure. E seems to be important in facilitating the actions of acetylcholine in the spinal cord, but excessive concentrations may have an opposite effect. E also seems to have a definite facilitating effect on motor reflex mechanisms. Small doses of E intravenously causes arousal from natural sleep. Larger doses produce prolonged stupor generally preceded by vomiting, hyperventilation, and excitement. Analgesia without stupor has been reported in man, but E seems actually to antagonize the analgesic effect of narcotics. E in clinical dosage has only slight effect on electroencephalogram. E produces respiratory stimulation which may be central in origin. (*Rothballer, A. B.: The Effects of Catecholamines on Central Nervous System, Pharm. Rev. 11: 494 (June) 1959.*)

NORADRENALINE Following an infusion of noradrenaline for 1–2 hours, during which time the systolic blood pressure was maintained at a 160–190 mm. Hg, hypotension occurred and lasted up to two hours. In four subjects given infusions for seven hours with constant-rate infusors it was found that the blood pressure declined during the infusion for the first four hours and then remained steady. There was a considerable degree of hypotension after the infusion was stopped. There was a decrease in skin blood flow during and after the infusion but an increase in muscle blood flow during and after the infusion. There is probably a circulating vasodilator present since the increase in muscle blood flow is not due to a nerve reflex nor due to the local action of noradrenaline. (*Lever, A. F., Mowbray, J. F., and Peart, W. S.: Blood Flow and Pressure after Noradrenaline Infusion, J. Physiol. 146: 43 (May 19) 1959.*)

ANALEPTIC Tetrazol given in repeated convulsant doses to dogs completely paralyzed with *d*-tubocurarine or decamethonium produced a consistent hyperthermia at ambient temperatures above a range of 23 to 25 C, and a consistent hypothermia at lower temperatures. Increased cutaneous blood flow with resulting loss of body heat appears to be the main factor in the hypothermic response, whereas increased heat production independent of skeletal muscle activity appears to be the major factor in the hyperthermic response. (*Shemano, I., and Nickerson, M.: Mechanisms*