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*

of Thermal Responses to Pentylenetetrazol, J. Pharmacol. & Exper. Therap. 126: 143 (June) 1959.)

ANALEPTICS The effect of Megimide and Metrazol were studied in mice, rabbits and dogs. Megimide proved to be a convulsive agent similar to Metrazol. It also acted as an antagonist to the depressant action of pentobarbital, urethane and ethyl alcohol. Subconvulsant doses in dogs and mice would lead to convulsions if the animals had previously received large doses of morphine. Megimide is not a specific barbiturate antagonist, since it will antagonize depression produced by drugs of unrelated chemical structures. No data were obtained to indicate that Megimide was a better antagonist than metrazol, in acute barbiturate poisoning. *(Zapata-Ortiz, V., De-LaMata, C. R., and Campos-Iturrizaga, A.: Effect of Bemegride (Megimide) and Metrazol on Some Neurodepressors, J. Pharmacol. & Exper. Therap. 125: 347 (April) 1959.)*

BARBITURATE ANTAGONIST Treburon, primarily an anticoagulant drug, has been noted to have analeptic actions in dogs anesthetized with pentobarbital. This activity was investigated in dogs, rabbits and pigeons. A reduction of time for return of righting reflexes in animals anesthetized with barbiturates and given Treburon was statistically significant. No toxic effects were noted. *(Joseph, A. D., Jindal, M. N., and Patel, M. A.: Treburon as Barbiturate Antagonist, Lancet 1: 815 (April 18) 1959.)*

INTRA-ARTERIAL THIOPENTAL The effects of thiopental injected intra-arterially have been studied on spiral strips of rabbit aorta, after intra-vascular injections into the ears of rabbits, and the profused hind leg of dogs. Thiopental causes a contraction of the isolated strips, a constriction of the profused vessels of the rabbit ear. This constriction is due to release of nor-epinephrine from structures in or near the artery wall. The constriction was not due to the alkaline *p*H of the solution injected. Hexobarbital which has not caused vascular thrombosis has almost no constrictor action in the rabbit ear. The attempts to observe a similar constrictor action of thiopental in the profused dog's hind leg were unsuccessful. *(Burn, J. H., and Hobbs, R.: Mechanism of Arterial Spasm Following Intra-Arterial Injection of Thiopentone, Lancet 1: 1112 (May 30) 1959.)*

PHENOBARBITAL EXCRETION The acid-base equilibrium of the blood of chloralized dogs was altered by experimentally induced hyper- and hypoventilation or by the intravenous injection of hydrochloric acid N/4 solution or sodium bicarbonate solution 3.5 per cent. Phenobarbital was determined with a spectrophotometric method. In a group of 11 nephrectomized dogs, respiratory or metabolic alkalosis increased the level of phenobarbital in the blood. In a group of 47 dogs, the renal excretion of phenobarbital was found to occur by a filtration-reabsorption process and to increase with alkalosis. The *p*K of phenobarbital is 7.26; changes in *p*H of the plasma will therefore exert an influence upon the ionization of phenobarbital and concomittantly affect its excretion through the kidney. *(Mollaret, P., and others: Acid-Base Balance and Renal Excretion of Phenobarbital, Compt. rend. Acad. Sc. 248: 2257 (April) 1959.)*

BARBITURATE INTOXICATION Based upon experimental data in dogs that alkalosis increased the excretion of phenobarbital by the kidneys by inhibiting tubular reabsorption, 50 cases of barbiturate intoxication were treated with injections of 3 per cent solutions of sodium bicarbonate (up to 5–6 liters per 24 hours). Artificial ventilation was maintained in the cases with marked respiratory depression. The authors report very favorable results in their series and no deaths. *(Mollaret, P., and others: Treatment of Barbiturate Intoxication, Compt. rend. Acad. Sc. 248: 2424 (April) 1959.)* *(Abstractor's Comment:* No blood levels of barbiturate, *p*H or carbon dioxide substantiate these data.)