pattern of arousal. In the experiments reported, atropine is shown to similarly block the electrocortical effects of adrenergic and cholinergic drugs. Atropine can induce an electroencephalographic picture of sleep without concomitant behavioral signs in the rabbit. This suggests that there is little correlation between electroencephalographic patterns and behavior. They also indicate that an electroencephalographic arousal pattern is not necessary for consciousness. (*White, R. P., and Daigneault, E. A.: Antagonism of Atropine to Electroencephalographic Effects of Adrenergic Drugs, J. Pharmacol. & Exper. Therap. 125: 339 (April) 1959.*)

MORPHINE Eighty-five nonaddict, nonpatient, adult male college and graduate students received a dose of 10 mg. of morphine per 70 kilograms of body weight, or 1 ml. of saline subcutaneously. In comparison with the placebo, morphine did not reduce the hunger responses studied in the majority of these individuals. The demonstration is important because of the importance which has been attributed to the hunger reducing power of morphine in addicts. (*Smith, C. M., and Beecher, H. K.: Effect of Morphine on Subjective Response of Hunger in Normal Subjects, J. Pharmacol. & Exper. Therap. 129: 63 (May) 1959.*)

MORPHINE Sixty-one nonaddict, nonpatient, adult male, college and graduate students were given subcutaneous injections of a placebo or morphine (10 mg./70 kg. of body weight). Before and after medication, the subjects gave information concerning sensations and moods. The major subjective responses to morphine in the 'somatic' area were dizziness, nausea, pruritis, headache, and feeling of warmth. The responses in the 'nonsomatic' area were principally mental clouding, physical inactivity, and mental inactivity. (*Smith, C. M., and Beecher, H. K.: Measurement of "Mental Clouding" and Other Subjective Effects of Morphine, Surg. Gynec. & Obst. 126: 50 (May) 1959.*)

DIPIPANONE Clinical use of a new synthetic narcotic, dipipanone hydrochloride (Pipadone), in anesthesia revealed certain characteristics different from other narcotics now used. Given as premedication, the respiratory rate is slowed and minute volume decreased in spite of an increase in tidal volume. During thiopental anesthesia, apnea is easily produced with intravenous dipipanone. A slowing of the cardiac rate was evident, as well as peripheral vasodilatation, leading to hypotension in some cases, especially when Fowler's position was utilized. As a postoperative analgesic, 25 mg. of dipipanone gave greater pain relief than 100 mg. of meperidine. Dipipanone demonstrated little or no hypnotic effect, and sleep patterns on electroencephalogram were absent. Amnesia was not produced. Complete disappearance of the cough reflex was noted in most patients. (*Lamoureaux, L., and others: Preliminary Clinical Study of Dipipanone Hydrochloride (Pipadone) in Anaesthesia, Canad. M. A. J. 80: 968 (June 15) 1959.*)

PHENOTHIAZINE A new phenothiazine drug, Trimeprazine, has been compared with a barbiturate as a premedicament in 200 young children. Its effects on recovery and vomiting after operation have been assessed. It proved to be palatable and easy to administer. Its hypnotic effect was equal to that of a barbiturate. It did not prolong the period of recovery, and fewer children vomited or were restless after operation, although the difference was not statistically different. (*Cope, R. W., and Glover, W. J.: Trimeprazine Tartrate for Premedication of Children, Lancet 1: 858 (April 25) 1959.*)

NAUSEA AND VOMITING Effects of Trilafon on vomiting in 265 obstetrical patients was compared with the effect of routine sedation in 264 similar patients in active labor. The group receiving Trilafon showed an incidence of nausea and vomiting that was 75 per cent lower than that in the control group. (*Anderson, G., and others: Effect of Trilafon on Nausea and Vomiting During Labor, Obst. & Gynec. 13: 504 (April) 1959.*)

VASOPRESSORS Restoration of the systemic blood pressure to normal levels by vaso-

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*