curarine is of both theoretical and practical importance. Experimental studies previously undertaken have failed to resolve this question satisfactorily. Under normal circumstances, there is little likelihood that intravenously administered d-tubocurarine should penetrate the central nervous system. It has been suggested, however, that stress situations of asphyxia, electrolyte imbalance, hemorrhage, etc., may weaken this selectiveness of entry. The availability of a sensitive fluorescent method for the quantitative analysis of d-tubocurarine in plasma and cerebrospinal fluid has permitted the evaluation of this problem. Method: Fifteen mongrel dogs were given d-tubocurarine by intravenous or intraarterial route and studied under stress conditions. The study groups included a control, those receiving massive intravenous doses of d-tubocurarine, those made acidic, those subjected to hypoxia, those made hypokalemic, and a group given d-tubocurarine into the carotid artery. Anesthesia was induced with sodium pentobarbital, and mean arterial pressure, electrocardiogram, and electroencephalogram were monitored via a multichannel recorder. Cisternal puncture was performed with a 20 gage spinal needle and adapted for sampling of spinal fluid. Determinations of pH, P02, Piones, and serum potassium were made at appropriate intervals. Following a paralyzing dose of d-tubocurarine (0.3 mg./kg.), plasma and cerebrospinal fluid samples were drawn at five, fifteen, thirty, and sixty-minute intervals and analyzed in duplicate for d-tubocurarine content. Results: Despite plasma levels in the control group of 0.6–2.6 γ/ml, no d-tubocurarine could be found in the cerebrospinal fluid. Likewise, in the group given massive doses of d-tubocurarine (1.5–3.0 mg. kg.), resulting in plasma levels of 4.7–11.8 γ/ml., no drug was found in the cerebrospinal fluid. The group rendered acetic (pH 6.91–7.02), and the hypoxic group (P02 26–36 mm. of mercury) also failed to show presence of any muscle relaxant in the central nervous system. The hypokalemic animals (K+ – 3.4 mEq./l.) evidenced a reduced tolerance to both the anesthesia and to the intravenous d-tubocurarine, yet none of the latter was measurable in the cerebrospinal fluid. Finally, the injection of large amounts of d-

Experimental Studies of Coronary Arterial Flow Using the Square-Wave Electromagnetic Flowmeter. D. LeRoy Crandell, M.D., Edgar Lee Marston, M.D., and Jesse H. Meredith, M.D., Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina. This study deals with the application of the square-wave electromagnetic flowmeter to quantitate the volumetric rate of blood flow through an unopened coronary artery. An experimental method for the study of coronary arterial flow alterations resulting from surgical manipulation and drugs used in anesthetic practice is of clinical importance. Previous studies have used cannulating techniques with heparinization to measure coronary arterial flow. With the square-wave electromagnetic flowmeter (Denison, A. B., Jr., Spencer, M. P., and Green, H. D.: Circulat. Res. 3: 39, 1955), the pulsatile and mean flow through the intact coronary artery can be recorded. Young adult mongrel dogs, ranging in weight from 16 to 40 kg., were studied. They were lightly anesthetized with intravenous pentobarbital sodium, their tracheas intubated, and their lungs ventilated with a piston-type respirator. Through a left thoracotomy, the proximal portion of the anterior descending branch of the left coronary artery was isolated to allow the placement of a small magnetic probe measuring 0.8 × 1.0 × 3.0 cm. Adjusting gain and damping of probe currents permitted phasic and mean flow measurements. Zero flow references were secured by momentary occlusion of the vessel.
An electronic difficulty, ECG pick-up interference, was encountered early in the investigation. The QRS causes an imperceptible disturbance presumably because frequencies dominant in the QRS are efficiently rejected by the flowmeter. The P wave is the major source of disturbance which is of random phase and polarity from beat to beat. Its magnitude was reduced by retracting the atrial appendage, and by placing a 1 cm. × 1 cm. piece of tinfoil between the artery and the underlying myocardium. (This makes it more likely that the ECG will affect both electrodes equally and so be cancelled out.) The arterial pressure was monitored simultaneously from the femoral artery using a Statham gauge transducer. Lead 2 of the electrocardiogram was monitored continuously. Results: The normal pattern of coronary blood flow did not deviate grossly from that reported by Gregg and Green (Gregg, D. E.: Coronary Circulation in Health and Disease. Phila., Lea & Febiger, 1950) with the differential pressure flowmeter. The effect on coronary arterial flow from momentary occlusion of the coronary artery was studied. After a fall of arterial flow to zero, a measurable reactive hyperemia regularly occurred. The normal physiological relation of systemic arterial pressure to coronary flow was demonstrated. Also, there was noted an increase in coronary flow with increases in heart rate up to 150 beats per minute but a decrease with further increases in heart rate. Conclusion: A series of observations are planned on alterations in mean coronary arterial flow and pulsatile characteristics produced by the administration of drugs and various situations encountered during anesthetic practice. The results should be of value to the anesthesiologist in the anesthetic management of the patient with coronary artery disease.

Effects of N-Allyloxy morphine-Narcotic Mixtures in Anesthetized Subjects. G. M. Davidson, M.B., F. F. Foldes, M.D., D. Dunclaf, M.D., E. S. Seiker, M.D., and S. Kuwabara, M.D., Departments of Anesthesiology, Mercy Hospital and the University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania. (Dr. Foldes' present address: Montefiore Hospital, New York.) Method: Sixty subjects, given premedication of 100 mg. pentobarbital and 0.3 to 0.4 mg. scopolamine hydrobromide, were lightly anesthetized with thiopental sodium and nitrous oxide-oxygen. Control values of respiratory rate, tidal and minute volumes and of pulse rate and blood pressure were recorded after induction of anesthesia. The 60 subjects were divided into six groups of ten. The following drugs were administered intravenously over a 30-second period to the members of the groups: group I, 0.02 mg./kg. oxy morphine hydrochloride; group II, 0.02 mg./kg. oxy morphine plus 5 µg./kg. N-allyloxy morphine hydrochloride; group III, 0.2 mg./kg. morphine sulphate; group IV, 0.2 mg./kg. morphine plus 5 µg./kg. N-allyloxy morphine; group V, 2.0 mg./kg. meperidine hydrochloride; group VI, 2.0 mg./kg. meperidine plus 5 µg./kg. N-allyloxy morphine. Results: When administered alone, the three narcotics caused marked respiratory depression. The respiratory rate and minute volume decreased to about 25, 55, and 70 per cent of control after meperidine, oxymorphine and morphine, respectively. When administered together with 5µg./kg. N-allyloxy morphine, the respiratory rate and minute volume decreased only to about 75, 85 and 90 per cent of control with meperidine, oxymorphine and morphine, respectively. These doses of narcotics caused a 10 to 20 per cent decrease of pulse rate and systolic and diastolic blood pressure. These circulatory effects were not significantly affected by the simultaneous administration of N-allyloxy morphine. Judging from the reaction to the skin incision made 12 to 15 minutes after the administration of the narcotics or the narcotic–N-allyloxy morphine mixtures and from the mg./kg./minute thiopental doses, the admixture of N-allyloxy morphine caused no significant decrease in the analgesic potency of the narcotics. In ten other subjects, who, in addition to pentobarbital and scopolamine, also received 50 to 100 mg. meperidine in their premedication, the apparent protective effect of the admixture of 5 µg./kg. N-allyloxy morphine to 0.02 mg./kg. oxymorphine was greater than in the subjects of group II not premedicated with a narcotic. Conclusion: Our findings indicate that although for the supplementation of anesthesia, the analgesic effect of 2.0 mg./kg.