anesthetized two weeks later with halothane. Thus each animal served as its own control. *Results:* With halothane 7 of 9 dogs survived the initial hemorrhage. With methoxyflurane all of these dogs survived the subsequent hemorrhage. However, of the 6 dogs who survived the initial methoxyflurane hemorrhage, only 3 survived the subsequent procedure with halothane. *Conclusions:* These results suggest that tolerance to severe hemorrhage during methoxyflurane anesthesia may be greater than during halothane anesthesia in animals who had been previously stressed. Furthermore, there appeared to be a similar quantitative relation between the degree of hypotension and the degree of hypovolemia with methoxyflurane and halothane anesthesia.

**A Comparison of the Effects of Atropine and Scopolamine on Fetal Responses (Pulse Rate and Arterial Oxygenation) to Maternal Hemorrhage and Subsequent Vasopressor Administration.** ANTONIO BOBA, M.D., E. JURGEN PLOTZ, M.D., and DANIEL M. LINKIE, M.S., *Department of Anesthesiology and Obstetrics and Gynecology; The Albany Medical College of Union University and The Albany Medical Center Hospital, Albany, New York.* It is possible, by means of specialized experimental techniques to define fetal distress in terms of fetal arterial oxygenation. The technique, reported elsewhere (Boba, A. and others: Surgery *58:* 267, 1965), allows withdrawal of microsamples of fetal carotid blood, for $P_{O_2}$ determinations, while the fetus is still *in utero. Methods and Results:* GROUP ONE: Experiments carried out in 7 dogs showed that, repeated maternal hemorrhages (each being equal to 1.0 per cent of the body weight and each being followed by a phenylephrine injection capable of restoring the maternal blood pressure to prehemorrhage values) led to fetal bradycardia and hypoxia. Fetal bradycardia and hypoxia were simply and directly related to one another. GROUP TWO (9 Dogs): Atropine was injected, intravenously (0.13 mg./kg.) into 5 mothers and intra-arterially (0.03 mg.) into the fetus four times, approximately five to ten minutes prior to the hemorrhage-vasopressor sequence. It was noted that fetal hypoxia developed in a predictable fashion. However, fetal bradycardia was either prevented or delayed for at least 120 minutes (at which time hypoxia had reached nearly catastrophic proportions). GROUP THREE (8 Dogs): Scopolamine was injected, intraveonusly (0.07 mg./kg.), into 4 mothers and intra-arterially (0.3 mg.) into the fetus four times, about five to ten minutes before beginning the hemorrhage-vasopressor sequence. Again, as after atropine injection, it was noted that fetal hypoxia developed in a predictable fashion. However, fetal bradycardia was either prevented or delayed for at least 120 minutes. GROUP FOUR: Complete arrest of the maternal circulation was achieved in 15 animals through electrically-induced ventricular fibrillation. In 6 animals atropine was injected intra-arterially into the fetus, and scopolamine in 4 animals. In all instances a prompt but transient increase in heart rate was noted, but no changes were noted in the hypoxic state of the fetus. Such spontaneous increases in fetal heart rate were not seen in the 5 control animals. *Conclusion:* On the basis of our experimental evidence, fetal distress can be dissociated from bradycardia by means of vagolytic drug injections. On the other hand, if no vagolytic drugs are injected fetal distress and bradycardia proceed *pari passu.* It is suggested that the use of vagolytic drugs may deprive the physician of an otherwise prompt and reliable indicator of fetal distress, namely, fetal bradycardia. Since there is no assurance that patients in labor may not develop complications leading to fetal distress, the wisdom of employing vagolytic drugs during labor and delivery is questioned. (Supported by N.I.H. Grant HD 00106.)

**Blood Viscosity: Effects of Surgery, Inhalation Anesthesia and Plasma Expanders.** C. PAUL BOYAN, M.D., PATRICIA S. UNDERWOOD, M.D., and WILLIAM S. HOWLAND, M.D., *Department of Anesthesiology, Memorial Hospital for Cancer and Allied Diseases, New York City.* The effects of surgery, inhalation anesthesia with diethyl ether, halothane and cyclopropane and of various plasma expanders on blood viscosity were studied on 162 unselected adult patients at Memorial Hospital. *Method:* Venous blood samples were drawn in heparinized vacutainers from premedicated but nonanesthetized patients. After thiopental induction the anesthesia was maintained in light

planes and muscular relaxation was secured with divided doses of muscle relaxants. Respiration was either assisted or controlled by the anesthesiologist. At the end of operation, a second venous sample of blood was drawn. The viscosity of whole blood was measured at 37° C. with a cone plate viscometer at 46, 115 and 230 seconds shear rate and compared with that of the preoperative sample. Hematocrits were measured on capillary blood samples centrifuged for three minutes at 12,000 rpm. Toward the end or at the termination of operation, the following plasma expanders were given within 30 minutes to 21 patients divided into four groups: (1) 500 ml. of dextran-40 (40,000 mol. weight) as 15 per cent solution in 5 per cent dextrose in water, (2) 500 ml. of dextran-75 (75,000 mol. weight) as 6 per cent solution in saline, (3) 500 ml. of 5 per cent solution of human proteins in buffered saline, Plasminate, and (4) 1,000 ml. of isotonic electrolyte replacement solution, Normosol-R. The volumes of the dextrans and Plasminate represented less than 16 per cent and that of Normosol-R less than 27 per cent of the total blood volume of each patient. Total blood volume and plasma volume were measured before the infusion was started and at 0, 60 and 120 minutes after it was completed. Blood volume was determined by a semi-automatic blood volume computer (Volemetron) using RISA $^{131}$I. Blood viscosity and microhematocrits were measured on venous samples taken before and after the end of the infusion and at 10, 30, 60 and 120 minutes thereafter. *Results:* Operation and routine inhalation anesthesia with diethyl ether, halothane and cyclopropane produced an insignificant reduction of blood viscosity, less than 0.20 centipoise, at 46, 115 and 230 inverse seconds shear rates, Five hundred milliliters of 15 per cent solution of dextran-40 (40,000 molecular weight), 6 per cent solution of dextran-75 (75,000 molecular weight), or 5 per cent solution of human plasma proteins, Plasminate, given in thirty minutes and representing less than 16.0 per cent of total blood volume did not improve the blood viscosity significantly (at 46, 115 and 230 inverse seconds shear rates) in patients undergoing operation or recovering from anesthesia. Isotonic electrolyte replacement solution, Normosol-R, in amounts less than 27 per cent of the blood volume did not improve blood viscosity under the same conditions.

**Central and Peripheral Venous Oxygen Saturations During Spinal Anesthesia.** WILLIAM E. CAIN, M.D., and WILLIAM K. HAMILTON, M.D., *Department of Anesthesia, University Hospitals, University of Iowa, Iowa City, Iowa.* Proper management of hypotension occurring with spinal anesthesia is a common problem. Tissues of many patients with low arterial pressure appear to be adequately perfused. Indiscriminate use of vasopressors may be detrimental. Many studies of circulatory dynamics have been done; however, they are largely concerned with arterial pressure and its alteration by various vasopressors. Maintenance of blood pressure even with well-oxygenated blood does not insure perfusion and may result in tissue hypoxia. The question still remains, when do we treat spinal hypotension and by what method? The purpose of this study was to obtain information concerning adequacy of circulation during spinal anesthesia, utilizing venous oxygen saturation as an indirect measurement of the relation of perfusion volume to tissue needs (Theye and Tuohy: ANESTHESIOLOGY, *26:* 49, 1965). Therefore, venous oxygen saturation in the right atrium and upper and lower extremities was studied as an index of adequacy and distribution cardiac output. *Method:* Unpremedicated, elderly patients (17) were selected from the daily operative schedule. All patients were positioned supine and 5 degrees head down. Catheters were placed in the brachial artery, right atrium, mid-forearm vein, and mid-calf vein. Arterial and right atrial pressures were measured electronically by appropriate transducers and recorders. Arterial, mixed venous ($S\bar{v}_{O_2}$), upper and lower extremity venous oxygen saturations were determined by an American Optical oximeter. Baseline measurements were recorded at three 15-minute intervals. Spinal anesthesia, produced by tetracaine-epinephrine-glucose combinations, was then administered with patients in lateral position. Measurements were repeated after spinal anesthesia at 10-minute intervals until blood pressure became stable. Patients were then given intravenous vasopressor, either ephedrine or methoxamine selected by a dou-