

protein contains  $^{75}\text{Se}$ . By plotting the concentration of  $^{75}\text{Se}$  versus time on a full logarithmic scale, we obtained two distinct curves: a falling straight line and a rising straight line. We have observed similar changes in nonanesthetized dogs. At this stage, we have the impression that the distribution pattern and breakdown of  $^{75}\text{selenate}$  may serve three purposes: (1) the retrograde extrapolation of the decay curve to the 10-minute time interval will indicate the selenate space which closely approximates the sulfate space, a measure of ECF, (2) the slope of the decay curve to indicate liver perfusion rate, and (3) the slope of the ascending curve to indicate liver function, the ability of the liver cells to incorporate  $^{75}\text{Se}$  during protein synthesis. For this purpose we are, at present, trying (1) to determine in which fraction of protein  $^{75}\text{Se}$  is incorporated, (2) to substantiate the slope of the rising curve with liver function tests, and (3) to determine the effect of anesthetic agents on the liver by virtue of selenate metabolism.

**Methoxyflurane and Halothane Anesthesia During Controlled Bleeding In Dogs.** I. C. ANDREWS, M.D., HOWARD L. ZAUDER, M.D., and LOUIS R. ORKIN, M.D., *Department of Anesthesiology, Albert Einstein College of Medicine, New York City.* For many years cyclopropane was considered the inhalation anesthetic agent of choice when acute hemorrhagic hypovolemia was present. However, recent work by Fabian (*Anesth. Analg.* 40: 137, 1961), comparing halothane and cyclopropane in the shocked animal, indicated the superiority of halothane in survival following hemorrhagic shock. The increased use of the fluorinated hydrocarbons has made it essential to study the effects of these agents in the hypovolemic patient. As yet, no knowledge of the effects of methoxyflurane in shock are known. Using the method outlined by the study of Fabian *et al.*, we undertook a pilot study comparing the survival of acutely bled dogs under halothane and methoxyflurane anesthesia. *Method:* Unpremedicated healthy female mongrel dogs of similar weights were used as experimental animals. Blood volume determinations were made by the Evans-blue dye technique before subjecting the animals to hemorrhage. Each animal was lightly anes-

thetized with 2 per cent thiamylal in amounts just sufficient to allow endotracheal intubation and cannulation of femoral vessels. The animal was allowed to awaken completely before reinduction of anesthesia with either halothane or methoxyflurane. After the animals were stabilized hemorrhage was induced. Blood was withdrawn through a catheter threaded from the femoral vein into the inferior vena cava. A 30-ml. syringe was used for withdrawal and reinfusion of blood, and all blood was collected in a sterile heparinized flask. The blood was withdrawn at intervals of 15 minutes in the following proportions: (1) 10 per cent of the total blood volume, (2) an additional 20 per cent of the total blood volume, and (3) another 20 per cent of the blood volume, reducing the blood volume 50 per cent in 45 minutes. Fifteen minutes following withdrawal, infusion of blood was begun on the same percentage basis and in the same sequence as used for withdrawing the blood. An 8-channel Offner Type R Dynograph and appropriate transducers were used to monitor arterial blood pressure, central venous pressure, electrocardiogram, electroencephalogram and end-expired  $\text{CO}_2$  concentration. Anesthesia was maintained according to electroencephalographic patterns attempting to maintain all animals at EEG levels II or III. Halothane was vaporized from a standard Foregger Copper Kettle while a Pentec was used for methoxyflurane. A partial rebreathing system was used throughout the study with 4 liters of oxygen as diluting gas. For halothane anesthesia, the induction concentrations ranged from 1.5 to 3.0 per cent. The maintenance concentration ranged from 0.6 to 1.2 per cent in inspired air. For methoxyflurane anesthesia the induction concentrations ranged from 0.6 to 1.0 per cent. The maintenance concentrations ranged from 0.3 to 0.7 per cent. Anesthetic concentrations were measured within the breathing circuit by a Perkin-Elmer vapor fractometer model 154. In a group of 16 animals, 9 were anesthetized with halothane and 7 were anesthetized with methoxyflurane prior to hemorrhage. Seven animals that survived the initial hemorrhagic stress with halothane were anesthetized two weeks later with methoxyflurane. Six animals that survived the initial hemorrhagic stress with methoxyflurane were

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 de-

layed for at least 120 minutes (at which time hypoxia had reached nearly catastrophic proportions). GROUP THREE (8 Dogs): Scopolamine was injected, intraveinously (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