in both groups, but was greater and declined at a slower rate in the anesthetized than in the conscious animals ($P < 0.025$). In another 4 dogs injections of 150 μg./kg. of glucagon caused in both conscious and anesthetized animals an immediate increase in blood glucose which was greater during anesthesia. *Comment:* Progressive hypoglycemia accompanies halothane anesthesia in dogs. Factors exist during halothane anesthesia which impede removal of glucose from plasma following either an exogenous or endogenous glucose load, but insulin given exogenously counteracts this inhibitory effect. The rapid removal of fructose from plasma and its subsequent conversion to blood glucose suggest that halothane anesthesia does not block this metabolic pathway. The sensitivity of the homeostatic mechanisms controlling the output or action of insulin and glucagon appears diminished. While not delineating the specific mechanisms responsible for these phenomena, our results suggest areas for further investigation. A hypothesis is proposed that halothane anesthesia impedes plasma removal of glucose by interference with cellular membrane transport in muscle and/or adipose tissue. (Supported by Grant HE-06967-03, National Institutes of Health, Bethesda, Maryland.)

Dynamic Pulmonary Compliance in Anesthetized Humans. MARTIN I. GOLD, M.D., and MARTIN HELRICH, M.D., *University of Maryland School of Medicine and University Hospital, Baltimore, Maryland.* It is generally accepted that during general anesthesia, total and pulmonary compliance decreases (Howell and Pecket: J. Physiol. *136:* 1, 1957). However, the lung volume history immediately preceding the compliance measurement also affects respiratory mechanics (Mead and Collier: J. Appl. Physiol. *14:* 669, 1959). Since spontaneous respirations during anesthesia are frequently depressed, the changes in pulmonary mechanics may be due to this and not to the anesthetic agent (Fry, D. L., and others: Amer. J. Med. *16:* 80, 1954). This investigation is a chronologic study of mean dynamic pulmonary compliance in 8 anesthetized subjects from the induction of anesthesia to the emergence from anesthesia. *Technique:* Having received barbiturate-anticholinergic premedication 1–1½ hours previously, each presurgical patient had a thin latex balloon inserted transnasally under topical anesthesia so that it lay in the lower one-third of the esophagus. By means of appropriate differential transducers, a carrier amplifier (Electronics for Medicine DR-8), a pneumotachograph (Fleisch no. 1), and electronic integrator, the following three parameters of respiratory mechanics were monitored: transpulmonary pressure, airflow, and tidal volume. Continuous scalar traces of these parameters allowed for the measurement and calculation of long series of dynamic pulmonary compliances. The apparatus for all measurements in the awake and anesthetized states consisted of: a tight-fitting face mask with a nipple for pressure measurements in the airway, a 450-g. to-fro carbon dioxide absorption canister, a pneumotachograph, and anesthesia rebreathing bag with low resistance pop-off valve. This apparatus was housed on a convenient swivel stand attached to the operating table. Metal to metal connections insured a leak-proof system. Conscious control measurements were made in the supine position on an operating table after a steady state was observed. All compliance measurements were derived from a series of from 20 to 60 consecutive breaths allowing for the calculation of a mean. At an appropriate time, halothane nitrous oxide-oxygen anesthesia was induced by means of the system mentioned above. In six of eight instances this induction was facilitated with a small amount (100–200 mg.) of thiopental. Measurements of dynamic pulmonary compliance were made in chronologic fashion so that all data could be plotted against time from the awake control state through the induction of anesthesia to the end of the investigation. Measurements were made: (a) during light and deep general anesthesia monitored by an electroencephalogram, (b) with and without an oral airway and with and without an endotracheal catheter, (c) during spontaneous respirations, (d) during assisted or controlled respirations, and (e) before surgery in eight subjects and after in three of these subjects. *Results:* There is apparently little correlation between levels of anesthesia and pulmonary compliance if other factors remain unchanged. An example of one

of the variables which may influence compliance was observed in two patients who had endotracheal intubation. In these subjects, an unexplained but significant reduction in compliance occurred while the catheter remained in place. When it was removed, compliance increased almost to the control level. Other variables may significantly influence compliance. Measurements made after periods of positive airway pressure were significantly higher than before (23 of 28 instances). Measurements made during the application of positive airway pressure were significantly lower than measurements made before or after (22 of 26 instances). A positive relation was demonstrated between tidal volume and pulmonary compliance. As anesthesia deepened and spontaneous respirations remained unassisted for periods of time approximating 15 to 45 minutes, pulmonary compliance decreased. (Supported by P.H.S. Grant HE-06429-03 and AMA-ERF Grant 64 and AMA-ERF Grant 150.)

Effect of Anesthesia on the Tolerance of Dog Brain to Anoxia. ALEXANDER GOLDSTEIN, JR., M.D., BUFORD A. WELLS, M.D., and ARTHUR S. KEATS, M.D., *Division of Anesthesiology, Baylor University College of Medicine, Houston, Texas.* This study was stimulated by clinical observations (Wells, B. A., Keats, A. S., and Cooley, D. A.: Surgery *54:* 216, 1963) which suggested that the brain was more tolerant to ischemia during general anesthesia than without anesthesia. To test the validity of these observations, a study was undertaken using the technique of Brockman and Jude (Bull. Johns Hopkins Hosp. *106:* 74, 1960) to produce cerebral ischemia of precise duration. *Methods:* Under local or general anesthesia, 9–28 kg. adult dogs were given intravenous succinylcholine, intubated with cuffed endotracheal tubes, and given intermittent positive pressure ventilation with a respirator. Through a right thoracotomy, umbilical tapes were passed around the azygos vein, the inferior and superior venae cavae, and the ascending aorta. By simultaneous occlusion of both cavae and the ascending aorta, blood flow was confined to the coronary and pulmonary circulations, and complete cerebral ischemia was produced for measured periods of 8 to 15 minutes. Neurologic sequelae were evaluated and graded for 48 hours postoperatively. Dogs eating normally and performing coordinated spontaneous activities were considered to have no neurologic sequelae (grade I). Dogs able to stand alone, but with ataxia or visual disturbances, were estimated to be moderately damaged (grade II). Comatose or markedly ataxic dogs were regarded as severely damaged (grade III). Dogs expiring within 48 hours following the anoxic episode were placed in a fourth category (grade IV). *Results:* To date total arrest of the cerebral circulation has been performed in 132 animals divided into 5 groups with the following results: GROUP A: (20 dogs) Local Anesthesia and Air—8 minutes ischemia: Grades I, 3; II, 2; III, 2; IV, 3— 10 minutes ischemia: Grades III, 2; IV, 8. GROUP B: (20 dogs) Local Anesthesia and 100 Per Cent Oxygen—8 minutes ischemia: Grades I, 1; III, 3; IV, 6—10 minutes ischemia: Grades I, 1; III, 1; IV, 8. GROUP C: (20 dogs) Local Anesthesia and 5 per cent Carbon Dioxide, 95 per cent Oxygen—8 minutes ischemia: Grades I, 3; III, 4; IV, 3— 10 minutes ischemia: Grades I, 2; III, 2; IV, 6. GROUP D: (30 dogs) Morphine Sulphate (15 mg./kg. body weight) and 100 per cent Oxygen—8 minutes ischemia: Grades I, 3; II, 1; IV, 6—10 minutes ischemia: Grades I, 1; III, 2; IV, 7—13–15 minutes ischemia: Grade IV, 10. GROUP E: (42 dogs) Pentobarbital (30 mg./kg. body weight) and 100 per cent oxygen—10 minutes ischemia: Grades I, 7; II, 3—11 minutes ischemia: Grades I, 5; IV, 1— 12 minutes ischemia: Grades I, 2; II, 4; III, 4—13 minutes ischemia: Grades I, 1; II, 1; III, 4; IV, 4—15 minutes ischemia: Grades III, 4; IV, 2. *Conclusions:* Although there was no marked difference between the three groups with local anesthesia, there was a suggestion that ventilation with 100 per cent oxygen produced increased cerebral damage. Morphine failed to protect against cerebral damage. Pentobarbital anesthesia, however, extended the duration of cerebral ischemia required to produce 100 per cent grade III and IV dogs to 15 minutes compared to 10 minutes for this degree of damage under local anesthesia. (This study was supported by a grant from the Houston Heart Association.)