in a semiclosed carbon dioxide absorption system with assisted or controlled respiration. Anesthesia was monitored electroencephalographically in an attempt to maintain comparable electroencephalographic patterns in each patient throughout operation. Arterial blood samples were drawn at the time that the cardiac cycle records were taken during halothane anesthesia, and were analyzed for *p*H, P_{CO_2} and O_2 content and hematocrit. The mean arithmetic average of the Q wave to first tone interval during the control period was 0.063 second, while the average for the same interval during fluothane anesthesia was 0.073 second. This difference was not statistically significant. The first tone to carotid pulse rise interval, was 0.070 seconds prior to anesthesia, and 0.091 seconds during fluothane anesthesia. This was a statistically significant difference. The Q wave to carotid pulse rise interval was 0.132 seconds before operation, and 0.164 seconds during halothane anesthesia. The difference was significant. The first tone to second tone interval was 0.332 seconds before anesthesia, and 0.364 seconds during the administration of halothane anesthesia. This was also a statistically significant difference. The R-R′ interval was 0.773 seconds, representing an average heart rate of 78 beats per minute, before anesthesia, and 0.848 seconds, representing a slight decrease to 70 beats per minute, during halothane anesthesia. The arterial *p*H, at the time that these cardiac cycle measurements were made, averaged 7.36; the arterial P_{CO_2} averaged 41.5 mm. Hg; the arterial oxygen content averaged 17.98 volumes per cent and the hematocrit averaged 39.8 per cent. The results indicate that the administration of halothane does decrease the rapidity of mechanical contraction of the ventricle, but does not affect the electrical spread of the depolarizing wave in the ventricular myocardium. The data are in contrast to similar measurements made during ether anesthesia, which appeared to inhibit both of these processes; and are also in contrast to similar measurements made during cyclopropane anesthesia, which appeared to inhibit neither of these processes. [*This work was supported by a grant from Burroughs Wellcome & Company (U.S.A.) Inc.*]

Effect of Phenylephrine on Survival and Acid-Base Balance in Dogs with Acute Hemorrhagic Hypotension on Constant Volume Ventilation. ROBERT W. LOEHNING, M.D., ISSAKU UEDA, M.D., AND VASIL P. CZORNY, M.D. *Division of Anesthesiology, University of Utah, Salt Lake City, Utah.* The aims of these experiments were: (1) to determine the survival rates of dogs on constant volume ventilation subjected to acute arterial hypotension and then treated with phenylephrine to raise and maintain blood pressure at 120 mm. Hg or over in one series, and 70–80 mm. Hg in another series, and (2) to observe the effects of the drug on improving or preventing blood acidosis which accompanies hemorrhagic hypotension. Forty nonfasting mongrel dogs weighing from 9–18 kg. were anesthetized with pentobarbital (25 mg./kg.) and given galamine triethiodide for relaxation. The animals were ventilated with a constant volume respirator with sufficient volumes of a 30 per cent oxygen 70 per cent nitrogen gas mixture to maintain a "steady state" and an end-tidal carbon dioxide tension of 20–40 mm. Hg. After a stabilization period of 30–60 minutes the animals were bled from a catheter in the aorta, within a period of five minutes to a mean pressure of 40 mm. Hg. Five minutes later the animals were given phenylephrine intravenously. Fifty-nine per cent of the dogs maintained at 120 mm. Hg blood pressure or over died. All 8 of the animals maintained at 70–80 mm. Hg and 10 out of 11 of the controls survived. All animals became acidotic following hemorrhage, and after phenylephrine the dogs maintained at the higher blood pressures were more acidotic than the other groups. End-tidal carbon dioxide tensions fell during hemorrhage and rose concomitantly with the rise in blood pressure after treatment with the vasopressor. The control group did not attain normal levels until 20 minutes later. Therefore, the animals maintained at pre-existing blood pressures became more acidotic, in spite of greater carbon dioxide output, than untreated animals or those maintained at lower blood pressures.

Plasma Volume Changes Incident to Open-Heart Surgery: Analysis of Patients-Donor Blood Exchange. THOMAS N. MAC-

KRELL, M.D., CARL R. BOGARDUS, JR., M.D., AND W. NEALE BENNETT, M.D. *Department of Anesthesiology, Louisville General Hospital, University of Louisville, School of Medicine, Louisville, Kentucky.* During open-heart surgery large quantities of blood are administered and lost. Accurate measurements indicate that much more blood is administered than is lost. Yet, there is little clinical evidence that these patients are over-transfused. This study was undertaken to explain the discrepancy between blood administered and blood lost and has included objective measurement of changes in patient's blood volume incident to open-heart surgery. The following studies were made upon 5 patients undergoing open-heart surgery using the Kay-Cross rotating disk oxygenator: (1) Accurate body weights were determined immediately preceding and following open-heart surgery on these patients. Any items that might be attached to the patient postoperatively were weighed preoperatively with the patient, such as adhesive tape, elastoplast, bandages, chest and urinary catheters and hemostats. Unused adhesive tape, bandages and catheters were again included on the scales with the patient postoperatively. We had, therefore, an accurate indication of the weight gain incident to heart surgery. (2) A control blood volume was determined shortly after induction of anesthesia. Another blood volume was performed at the completion of surgery before the patient was removed from the table. The source of radioactive iodinated serum albumin was injected intravenously, and after equilibrium, a sample was taken from another vein or from an artery. This was analyzed for the dilution of radio-activity which is a function of the patient's plasma volume and is directly related to patient's blood volume. (3) The amount of blood administered was accurately determined by weighing the bottles of blood before and after administration. Weight was converted to volume by dividing it by the average specific gravity of blood, 1.056. The volume of anti-coagulant solution was subtracted from total volume, leaving the amount of blood administered from each bottle. (4) The measured blood loss was determined by weighing sponges used and by the volume of blood aspirated by suction into graduated cylinders. At present, the study is not adequate to explain discrepancy between blood administration and blood loss. However, we have the impression that the pump oxygenator is probably the greatest source of this apparent discrepancy and is presently under investigation.

There were several factors we discovered relating to this study that bear mentioning. First, an average unit of citrated blood contained only 410 ml. of blood and 110 ml. of A.C.D. solution. Each patient was given less blood and more solution than we commonly realized. The total amount of anticoagulant solution given our patients ranged between 500 and 1,000 cc. Secondly, each unit of heparinized blood as an average contained 475 ml. of blood and 30 ml. of physiological saline with 18 mg. of heparin. When 10 units of heparinized blood were used to "prime" the heart-lung machine, the total volume, 300 ml. was saline solution. After several minutes of circulation through the patient, this saline solution was removed into the patient's extracellular fluid space. The actual priming volume of the oxygenator falls. However, the operator of the heart-lung machine was not aware of this and in an attempt to "end up even" with the original priming volume, will "steal" 300 ml. of blood from the patient's blood volume. This is one of the sources of "loss of blood" that occurs during cardiac bypass. Thirdly, when the patient's weight gain was correlated with the change in blood volume and total fluid administered, the patient's net weight gain was equal to the total fluid administered minus 100–200 ml. fluid loss (due to evaporation) plus the patient's blood volume change. The following formula has been devised: Patient's weight change in grams = Fluid gain in ml. + blood volume change in ml. This expression is totaled algebraically with the patient's weight change and blood volume change expressed as positive values when there was a gain and as negative values when there is a loss. Thus, in addition to clinical signs such as the patient's blood pressure and amount of vascularity from the lower eyelid conjunctivae as proposed by Mendelsohn (*Mendelsohn, D. et al.: Anesthesiology 18: 231, 1957*), we have a clinical method of estimating the patient's change in blood volume during open-heart surgery. All that is necessary is an accurate de-

termination of weight change during operation and an accurate account of fluid administered to the patient, not only as fluid by itself, but also as anticoagulant solution accompanying each unit of blood.

Postoperative Ventilation Studies in the Recovery Room. THOMAS E. MACNAMARA, M.B., CH.B., BEATRIZ L. DE NAVA, M.D., TAPAN SARKAR, M.D., AND THOMAS F. MCDERMOTT, M.D. *Department of Anesthesiology, Georgetown University Medical Center, Washington, D. C.* The use of the Bennett Respiratory Ventilation Meter in random fashion as a teaching aid disclosed the fact that respiratory depression occurred frequently in the operating and recovery rooms. These deficits occurred in patients whose gross clinical condition appeared satisfactory to the anesthetist. Pulmonary ventilation measurements were made in 487 unselected, nonconsecutive adult surgical patients (from 15 to 70 years of age) who underwent general anesthesia following premedication with a narcotic, pentobarbital, promethazine, and a belladonna drug in varying doses. The measurements of respiratory exchange were made using the Bennett Respiratory Ventilation Meter. With the face mask being closely applied, measurements were made during 30-second periods with duplicate or triplicate determinations. Of these 487 patients:—256 had four measurements of ventilation: 45 minutes after premedication, upon arrival in the recovery room, upon leaving the recovery room, and day following operation; 123 had three measurements of ventilation: upon arrival in the recovery room, upon leaving the recovery room, and day following operation; 61 also had three measurements of ventilation: 45 minutes after premedication, upon arriving in the recovery room, leaving the recovery room; and 47 had two measurements of ventilation: upon arriving in the recovery room and leaving the recovery room. We observed that an inspiratory volume of 4 liters per minute was a normal average for this series. Of these 487 patients, 90 (16 per cent) showed 15 per cent or more pulmonary ventilation depression on arriving in the recovery room, followed by gradual recovery. Nine of the 317 patients who were measured preoperatively showed reduced ventilation of more than 15 per cent. Two patients showed more than the 15 per cent reduction on the following day. Sixteen patients had a reversal of this trend, being depressed on leaving the recovery room (or conversely having stimulation of respiration on entry to the Recovery Room). The following factors have been considered in this study; and, so far, there has been no obvious correlation between adequate pulmonary ventilation and: (1) type of operation, intra-abdominal, intrathoracic and others, (2) experience of the anesthetist, (3) use of relaxant drugs, (4) type of premedication, and anesthetic agent. In a few instances, gross depression of respiration could be traced to inexperience of the anesthetist. In conclusion, we found that this technique of mechanically measuring ventilation made us more conscious of adequate ventilation. The depression of respiration was readily reversed when detected. There was no obvious morbidity or mortality. The simplicity of the method allows for an easy adoption in any hospital.

Comparison Studies of Hepatic Function Following Anesthesia with the Halogenated Agents. LUCIEN E. MORRIS, M.D. *Division of Anesthesiology, University of Washington School of Medicine, Seattle, Washington.* The increasing interest in fluorinated or otherwise halogenated anesthetic agents during the past five years necessitates the evaluation of the effect of these new agents upon the functions of various systems. Therefore, a study has been made to compare possible hepatic changes subsequent to the use of each of several halogenated anesthetics, both in laboratory animals and in clinical practice. Rats were anesthetized with trifluoroethylvinyl ether, halothane, or chloroform under conditions of (1) high oxygen-low carbon dioxide; (2) high oxygen-high carbon dioxide; (3) low oxygen-low carbon dioxide or (4) starvation. Liver biopsies at 24, 48, and 72 hours were studied histologically. Liver biopsies were studied from a control group of animals given no anesthesia. Dogs were also studied for liver function changes after anesthesia with either halothane or chloroform under similar alterations of carbon dioxide and oxygen in the respired air. In these studies the chloroform and trifluoroethylvinyl ether anesthetics were most frequently followed