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-