cerebrospinal fluid pressure increased from 64.24 mm. H_2O to 225.41 mm. H_2O. Results from experiment two, showed the following: arterial *p*H changed by only .05 units; P_{CO_2} increased from 29.9 mm. Hg to 56 mm. Hg; no significant change in arterial blood pressure; no significant change in cerebrospinal fluid pressure. The results from experiment three are significant. Signs of progressive hypercapnia were noted to occur during the untreated apneic oxygenation; however, they were reversed during the treated apneic oxygenation. The *p*H which had decreased to 6.98 returned to 7.38 P_{CO_2} decreased from 139.6 mm. Hg to 78.6 mm. Hg; mean arterial blood pressure decreased from 164 mm. Hg to 146 mm. Hg; cerebrospinal fluid pressure decreased from 133 mm. H_2O to 57.64 mm. H_2O. In view of our observations we believe that THAM may have a clinical value in counteracting one of the acute deleterious effects of progressive hypercapnia; namely, an increased cerebrospinal fluid pressure.

Audio-Visual Demonstration of the Fetal Heart Beat. PETER G. LEHNDORFF, M.D. *Department of Anesthesiology, Burbank Hospital, Fitchburg, Massachusetts.* The instrument for the audio-visual demonstration of fetal heart beats, previously described (*Anesthesiology 19: 104, 1958*), has been considerably improved. The audiomonitor has been made simple, compact, sparkproof and sufficiently sensitive to be of considerable use. ECG and EEG methods for monitoring fetal heart beats, while more valid, are not practical for smaller hospitals; and audio-monitoring has become our method of choice. The monitor now consists of: (1) a transistorized, battery driven (4 v.) preamplifier-amplifier, which "clips" soundwaves, eliminates treble sounds, and suppresses (to a large extent) bowel sounds, maternal heartsounds and other random noises; (2) two microphones, one for location, the other (light and sensitive) remains taped to the patient; (3) a loud speaker or earphones and a needle indicator for audio-visual "reading" of the signal; (4) an electronic pulse-counter to facilitate counting high pulse rates. The instrument has been useful in: location of heart sounds not heard with the stethoscope, observation during contractions, demonstration of changes of fetal heart beat during contraction and their improvement during oxygen inhalation by the mother. Using this monitor, severe irregularities in fetal heart beat were discovered which made possible changes in obstetrical management so that 2 cesarean sections were done in time to save the distressed infants. In most cases the monitor was used to attract attention to changes which were usually confirmed by more standard means. Plans for further development include: a recording device for making permanent records, elimination of forceps noise, and simultaneous monitoring of mother and unborn child. [*Sponsored by Worcester North Chapter, The Massachusetts Heart Association. Equipment furnished by E. & J. Manufacturing Company.*]

Measurement of Electrical and Mechanical Events of the Cardiac Cycle During Halothane Anesthesia. DAVID M. LITTLE, JR., M.D., AND JAMES B. GIVEN, M.D. *Department of Anesthesiology, Hartford Hospital, Hartford 15, Connecticut.* Most previous observations of the effect of anesthetic agents upon cardiac activity have been concerned primarily with two aspects of cardiac activity, output or rhythm. The present study was designed to investigate some of the other effects of halothane (Fluothane) anesthesia upon the heart by measuring the relationship between certain of the electrical and mechanical events of the cardiac cycle. Simultaneous records of the electrocardiogram and phonocardiogram, and of the electrocardiogram and the carotid pulse tracing, were obtained on a Sanborn Twin-Beam photographic recorder. The following time intervals were then measured from the recordings: (1) Q wave to first tone (electrical ventricular systole); (2) first tone to carotid pulse rise (approximate isometric contraction period); (3) Q wave to carotid pulse rise (indirect isometric contraction period); (4) first tone to second tone (mechanical ventricular systole); and (5) R-R′ interval (heart rate). Control records were taken on 10 normal, healthy female patients following premedication for pelvic surgery, and repeat records were taken towards the end of operation during nitrous oxide-halothane anesthesia. Halothane was administered from a Fluotec vaporizer in concentrations of 0.7–1.5 per cent

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-