fected areas were Lissauer's tract and the posterior funiculus. The latter was involved in all cases but one. The involvement of the posterior funiculus was usually a narrow demyelinated band in the fasciculus cuneatus at the level of the injection only. The changes in the dorsal funiculus are apparently the effect of the alcohol on touch pressure fibers. Degenerative changes in the lateral portion of the dorsal funiculus represent progressive degeneration while those of the medial part of the dorsal funiculus represent the direct effect of alcohol on the cord. Changes in nerve cell bodies and Clarke's Column occurred regularly below, at, and above the injection site. There is no apparent reason for this. Since Clarke's Column is a spinal cord center for visceral efferent fibers, the changes are probably unrelated to pain relief obtained. Another puzzling feature revealed in this series is that regeneration of nerve fibers has been found to occur in the cord itself, where according to classical neuropathology, none should occur. This may be nonfunctional regeneration. It is apparent that the presence of alcohol in the subarachnoid space may directly affect the cord itself. Usually this change is limited to the periphery of the cord near the site of injection and the structures adjacent to the dorsal median fissure, where presumably a high concentration of alcohol may have been present. This phenomenon is thought to account for the occasional focal demyelinization seen in the dorsal spinocerebellar tract. Interruption of ascending tracts by this same mechanism is believed to be responsible through retrograde change for the degeneration observed in various nerve cells in the cord at levels below the site of injection. Accidental injury to the cord by direct intramedullary injection of alcohol occurred in two of the sixteen cords examined. Judging from the extent of damage, only small amounts of alcohol had been deposited in the cord, the balance entering the subarachnoid space as intended. There was very little clinical evidence of any neurological deficit as a result of this occurrence. Histopathologic evidence indicates that the posterior root is interrupted with all sensory modalities being involved. No patient who had a change in his pain pattern had any subjective sensory changes in the skin. There is apparently enough overlapping of contiguous dermatomes that the sensory loss to the patient is imperceptible. A more detailed report will be published elsewhere.

The Effects of THAM on Cerebrospinal Fluid Pressure During the Acute Carbon Dioxide Phase of Apneic Oxygenation. E. C. JORDON, M.D., H. C. SLOCUM, M.D., AND G. G. NAHAS, M.D. *Department of Anesthesiology, Walter Reed Army Hospital, Washington, D. C.* In another paper (*Nahas, G. G., and Jordon, E. C.: Effects of "CO_2 Buffer" on Hypercapnia of Apneic Oxygenation, to be published*) we have shown that a 0.33 molar intravenous infusion of nontoxic 2-amino-2 hydroxymethyl-1-3 propanediol (THAM) administered to dogs at the rate of 0.34 m*M*/kg./minute maintained cerebrospinal fluid pressure constant during a one hour period of apneic oxygenation. The present study demonstrates a possible clinical application of THAM to counteract or reverse the acute effects of hypercapnia on cerebrospinal fluid pressure. Using the preparation of apneic oxygenation, a total of 12 mongrel dogs were subjected to 30 minutes of apneic oxygenation induced with succinylcholine in a series of three experiments. Experiment one; 4 dogs were subjected to 30 minutes of apneic oxygenation without receiving THAM. Experiment two; 5 dogs were subjected to 30 minutes of apneic oxygenation while receiving a 0.33 molar intravenous infusion of THAM at a rate of 0.34 m*M*/kg./minute. Experiment three: 3 dogs were subjected to 15 minutes of apneic oxygenation without receiving THAM, followed by 15 minutes of apneic oxygenation during which time they received a 0.66 molar intravenous infusion of THAM at the rate of .66 m*M*/kg./minute. Just prior to apnea and at 15 minute intervals during apnea, arterial blood samples were withdrawn for determination of *p*H, P_{CO_2}, and hematocrit. Measurements were also made of mean arterial blood pressure, and cerebrospinal fluid pressure. Comparing the control value to those obtained after 30 minutes of apneic oxygenation in experiment one, there was a decrease in arterial *p*H from 7.54 to 6.64; P_{CO_2} increased from 25.1 mm. Hg to 247.7 mm. Hg; mean arterial blood pressure increased from 150 mm. Hg to 176 mm. Hg; and

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