animals, 15 serving as controls, were on diets for 6 weeks. Following this, they were anesthetized with halothane 1 per cent in oxygen for 45 minutes and then sacrificed in batches of 10 on the second, fifth and tenth days post-anesthesia. Subgroup 3: Twenty-five animals, 10 serving as controls, were on diets 6 weeks. Following this, they were anesthetized with halothane 1 per cent in oxygen for 45 minutes every other day for five exposures. Five were sacrificed immediately and ten two days post-anesthesia. All animals were sacrificed by decapitation and livers were fixed in formalin and examined both grossly and histologically (H and E stain). The tissue sections were prepared and examined by the same technician and pathologist respectively by a blind technique. No gross changes were noted. The microscopic changes were degeneration (primarily nonspecific cytoplasmic alterations, i.e., pallor, increased eosinophilia, intracytoplasmic coagulation and vacuolization) or focal necrosis. Approximately 20 per cent of the "normal" control animals showed some pathological liver changes. In the normal diet group, an increased incidence of changes was seen in the animals anesthetized with halothane 1 per cent in oxygen as compared with halothane-air. No explanation for this was apparent. The low protein diet per se gave an increased incidence of nonspecific cytoplasmic changes through 5 weeks. Exposure to halothane 1 per cent in oxygen further increased the incidence of pathological liver changes, particularly following multiple administrations.

An Increased Incidence of Paradoxical Hypertension Following Resection of Aortic Coarctation Under Halothane Anesthesia. Thomas B. Davis, M.D., Dean H. Morrow, M.D., Clarence L. Herbert, M.D., and Theodore Cooper, M.D., Anesthesiology Department, Clinical Center, National Institutes of Health, and Surgical Laboratory, National Heart Institute, Bethesda, Maryland. In 1953 Sealy reported a series of cases of unexpected hypertension that followed surgical correction of aortic coarctation. He termed this clinical situation "paradoxical hypertension." Since then, others have reported that "paradoxical hypertension" occurs in 10 to 20 per cent of patients who have undergone surgical repair of coarctation of the aorta. In the last three years, 24 patients with aortic coarctation had surgical treatment at the Clinical Center of the National Institutes of Health. Seven of this group were noted to have developed unexplained postoperative hypertension requiring the use of sympatholytic drugs in their management. Halothane had been employed as an anesthetic agent in five of the seven cases whereas nitrous oxide-thiopental had been used in the other two patients. The other 17 patients received nitrous oxide—thiopental anesthesia. This suggested that the use of halothane might be a contributing factor in the high incidence of postoperative hypertension. Halothane is known to have profound cardiovascular effects, part of which may be attributed to autonomic blockade. Patients with coarctation of the aorta have a lesion in which arterial flow distal to the obstruction occurs at a lower mean pressure and a greatly modified pulse pressure. Therefore, we considered that the hypertension seen in these patients postoperatively could possibly be an expression of adaptation of the peripheral vascular bed to increased arterial pressure in a situation of autonomic imbalance following halothane. It is well known that in dogs the release of acute constriction of the aorta produces profound effects on total and regional hemodynamics. It seemed reasonable, therefore, that a study of the hemodynamic changes following acute aortic occlusion in dogs with different anesthetic agents might be a logical approach to the problem. Eleven mongrel dogs were subjected to 15 minutes of total occlusion of the thoracic aorta, distal to the subclavian artery, utilizing two different anesthetic drugs on two separate occasions. On one occasion the experiment was carried out under nitrous oxide—thiopental anesthesia, and on a second occasion nitrous oxide—halothane anesthesia was used in the same dog. Intra-arterial blood pressure were measured continuously before, during and after occlusion. In the nitrous oxide—thiopental experiments, neither the systolic nor diastolic pressures in the post-occlusion, post-anesthetic period ever exceeded their control levels. Whereas, in the halothane series the systolic and diastolic pressures rose 55 and 75 per cent respectively over the control levels. The greater percentage increase in the diastolic
pressures than in the systolic pressure in the halothane series, might indicate an increased peripheral resistance in the postanesthetic period. The detrimental effects of aortic occlusion on renal hemodynamics are well known. It has been suggested by some that the kidneys may be involved in the production of “paradoxical hypertension.” Therefore, we elected to investigate the changes in renal resistance that might occur following aortic occlusion under the influence of the different anesthetic agents. The same methodology is being employed in this series as in the previous one, but in addition direct renal venous flow is being measured simultaneously with intra-arterial blood pressure. From these measurements the changes in calculated renal resistance are being studied under the circumstances of the experiment. Preliminary studies in 8 animals have shown a rise in renal resistance following aortic occlusion under both types of anesthesia. In the group receiving halothane there was a 125 per cent increase as compared with a 55 per cent increase for the nitrous oxide-thiopental group at one hour postanesthesia, postocclusion. The results of the two experiments seem to indicate that there is more tendency toward autonomic imbalance following relief of aortic occlusion under nitrous oxide-halothane than under nitrous oxide-thiopental anesthesia.

Role of Preanesthetic Medication in Modifying the Hypersynchronous Electrical Waves in Rhinencephalic Structures Induced with Inhalation Anesthetics. Edward F. Domino, M.D., Department of Pharmacology, University of Michigan, Ann Arbor, Michigan. The effects of various general anesthetics including diethyl ether, divinyl ether, halothane, methoxyflurane, cyclopropane, nitrous oxide, and ethylene were determined on the spontaneous electrical activity of neocortical and rhinencephalic structures of dogs. Both acute preparations as well as animals with chronically implanted electrodes were used. An attempt was made to correlate the changes in spontaneous electrical activity with the stages and planes of general anesthesia. The changes in spontaneous electrical activity were related to the depth of anesthesia and speed of induction. In neocortical structures diethyl ether and the other potent inhalation anesthetics produced the following EEG changes with increasing depth of anesthesia: (a) low voltage, fast frequency desynchronization, (b) some low voltage, slow wave activity of approximately 3 to 6 cycles per second, (c) marked flattening of all spontaneous electrical activity. Diethyl ether and the other potent inhalation anesthetics produced in the amygdala, olfactory bulb, posterior hypothalamus and related rhinencephalic structures the following EEG changes with increasing depth of anesthesia: (a) reduction of respiratory burst discharges, (b) spike-like 40 cycle per second activity which decreased in frequency during light plane I of surgical anesthesia, (c) slowing of the frequency of the spontaneous asynchronous activity, (d) increasing hypersynchrony and amplitude of electrical activity during the lower level of plane 1 or in plane 2 of surgical anesthesia, (e) persistent maximal hypersynchrony of 10 to 18 cycles per second lasting 30 seconds or more depending upon the level of anesthesia. Occasionally such activity bore a striking resemblance to seizure-like discharges. This was especially true with trichlorethylene which was observed to produce grand mal seizures during recovery from anesthesia, (f) loss of synchrony and decreasing amplitude and frequency of the spontaneous activity in plane 3 stage III, and finally, (g) further decrease in the amplitude of spontaneous activity in plane 4 stage III of anesthesia. Although the majority of these effects were readily produced by diethyl ether and most other inhalation anesthetics during induction, such phenomena were usually best seen during recovery from the deeper planes of surgical anesthesia. Nitrous oxide and ethylene produced only fast frequency high voltage waves in concentrations that were not hypoxic. The role of preanesthetic medication with pentobarbital, morphine, scopolamine, reserpine, and chlorpromazine on this phenomenon in rhinencephalic structures induced by diethyl ether anesthesia was determined. It was found that preanesthetic medication with pentobarbital reduced the frequency and duration of the hypersynchronous waves. On the other hand, morphine either had no effect or in some animals prolonged the duration of hypersynchrony. Scopolamine induced high voltage