Sodium Bicarbonate and Epinephrine in Cardiac Resuscitation. BULENT KIRIMLI, M.D., LEROY C. HARRIS, JR., M.D., and PETER SAFAR, M.D., *Department of Anesthesiology, University of Pittsburgh School of Medicine and Health Center Hospitals, Pittsburgh, Pennsylvania.* The value of epinephrine in the treatment of asystole is established. The value of epinephrine, alkalinizing agents and combinations of alkalinizing agents with sympathomimetic amines prior to defibrillation in cardiac resuscitation has not been established and requires controlled studies. Correction of the metabolic acidosis due to cessation of circulation by THAM in dogs facilitated internal a-c defibrillation (Clark, L. C.: New York Acad. Sci. 92: 687, 1960). *Method:* Ventricular fibrillation was induced electrically in four groups of 5 dogs each, under light pentobarbital anesthesia and controlled ventilation with air. After 5 minutes of ventricular fibrillation, mechanical controlled ventilation with air and external cardiac compressions were started. At 9 minutes, d-c external counter-shocks were applied. The initial shock was 10 watt-seconds. This was increased every 10 seconds in increments of 10 watt-seconds, until successful defibrillation occurred. The experiment was repeated 2 to 3 times after 30 minutes of adequate spontaneous circulation. At 7 and 8 minutes, respectively, each group was given the following drugs intravenously. Control group: physiologic saline solution; epinephrine group: physiologic saline and epinephrine 0.5 mg.; bicarbonate group: $NaHCO_3$ 2.5 mEq./kg. and physiologic saline; bicarbonate-epinephrine group: $NaHCO_3$ and epinephrine. *Results:* The *arterial* *p*H, in all groups (first trials only), was initially 7.23–7.52 (mean 7.39) and after 5 minutes of fibrillation 7.12–7.54 (mean 7.30). After 3 minutes of heart-lung resuscitation (following medication), the *p*H was in the control group 7.10–7.30 (mean 7.23); in the epinephrine group 7.02–7.48 (mean 7.23); in the bicarbonate group 7.38–7.70 (mean 7.58); and in the bicarbonate-epinephrine group 7.29–782 (mean 7.61). Fifteen minutes after defibrillation most dogs in all four groups had low *p*H values. Changes in arterial P_{CO_2} were minimal and variable. *Arterial* PO_2 values, checked periodically, were over 95 mm. of mercury. For successful *defibrillation,* the average energy required was lower in the bicarbonate and in the bicarbonate-epinephrine groups (mean 21 watts/second) than in the control group (mean 28 watts/second) and in the epinephrine group (mean 36 watts/second). All resuscitation attempts were successful in the bicarbonate-epinephrine group, but there was a great individual variation in the defibrillation response of the other groups. Following defibrillation, ventricular fibrillation recurred in 2 of the 5 dogs of the epinephrine group and in none of the control, bicarbonate, and bicarbonate-epinephrine groups. Following the first successful defibrillation, the return of *spontaneous circulation* required sternal compressions in 2 of the 5 dogs of the control group; in 2 of the 5 dogs of the bicarbonate group; and in none of the dogs which received epinephrine. Considering all successful defibrillations, spontaneous circulation returned in 65 per cent of the control group, 70 per cent of the epinephrine group, 36 per cent of the bicarbonate group, and 100 per cent of the epinephrine-bicarbonate group. Mean carotid blood flows, 30 minutes after defibrillation (in percentage of initial values), were: control group, 55 per cent; epinephrine group, 65 per cent; bicarbonate group, 90 per cent; and bicarbonate-epinephrine group, 100 per cent. *Conclusion:* The combination of bicarbonate and epinephrine was superior for the restoration of spontaneous circulation to the use of each drug alone. (Supported by Army Contract DA-49-193-MD-2160.)

Hepatic Function Following Trichlorethylene Anesthesia. DAVID M. LITTLE, JR., M.D., and JAMES B. GIVEN, III, M.D., *Department of Anesthesiology, Hartford Hospital, Hartford, Connecticut.* The present study was designed to elucidate the effects of trichlorethylene in comparison with other types of anesthesia upon hepatic function. *Method:* Liver function tests included the determinations of serum cholinesterase activity, percentage of cholesterol esterification, serum alkaline phosphatase activity, total serum bilirubin concentration, and cephalin-cholesterol flocculation. These tests were performed preoperatively, on the second postoperative day and

on the seventh postoperative day, on a group of healthy women undergoing gynecologic surgery. The patients were grouped in three series according to the major anesthetic agent employed, trichlorethylene, cyclopropane, or ether. *Results:* The findings indicate that these hepatic functions were not affected more following anesthesia produced by trichlorethylene than following anesthesia produced by either cyclopropane or ether. The level of serum cholinesterase activity was decreased following all three types of anesthesia: arithmetic mean values of 16 patients receiving trichlorethylene were 244.7 units preoperatively, 190.0 units on the second postoperative day (3 abnormal values), and 185.9 units on the seventh postoperative day (7 abnormal values); the arithmetic mean values of 20 patients receiving cyclopropane were 197.7 units preoperatively, 170.7 units on the second postoperative day (5 abnormal values), and 168.9 units on the seventh postoperative day (7 abnormal values); and the arithmetic mean values of 20 patients following ether anesthesia were 216.6 units preoperatively, 182.6 units on the second postoperative day (4 abnormal values), and 169.4 units on the seventh postoperative day (8 abnormal values). Cholesterol esterification was abnormal following trichlorethylene anesthesia in 2 patients on the second postoperative day and 2 patients on the seventh postoperative day; following cyclopropane anesthesia in one patient on the second postoperative day and 5 patients on the seventh postoperative day; and following ether anesthesia in 2 patients on the second postoperative day and 3 patients on the seventh postoperative day. The level of alkaline phosphatase was abnormal following trichlorethylene anesthesia in 2 patients on the second postoperative day and one patient on the seventh postoperative day; abnormal following cyclopropane anesthesia in only one patient on the seventh postoperative day; and abnormal following ether anesthesia in 2 patients on the second postoperative day and 4 patients on the seventh postoperative day. Total bilirubin was abnormal following trichlorethylene anesthesia in one patient on the second postoperative day; abnormal following cyclopropane in 2 patients on the second postoperative day and one patient on the seventh postoperative day; and abnormal following ether anesthesia in 2 patients on the second postoperative day. The cephalin-cholesterol flocculation determinations were normal in all instances following anesthesia produced by trichlorethylene; showed a 2+ or 3+ determination after 48 hours on the second postoperative day in 3 patients following cyclopropane anesthesia; and showed a 2+ determination after 48 hours in 4 patients on the second postoperative day following ether anesthesia, and a 3+ determination in 48 hours in one patient on the seventh postoperative day. *Discussion:* The results are of interest since the virtual epidemic of recent reports of postoperative liver dysfunction following the administration of halogenated hydrocarbon anesthetics, halothane and methoxyflurane, has suggested once again the inherent hepatotoxicity of the halides. Neither the present limited study or a very similar study following halothane anesthesia performed several years ago reveals any more effect upon the normal liver than that following cyclopropane or ether anesthesia, yet massive hepatic necrosis is reported to have occurred following both halothane and trichlorethylene, as well as after cyclopropane and ether. It may be that this type of pharmacological testing is inadequate to reveal hepatotoxicity because of the insensitivity of the liver function tests employed, or because of the size of the sample tested; or it may be that massive hepatic necrosis following anesthesia occurs as a result of other factors than the anesthetic drugs themselves.

Influence of Inhalation Anesthetics on Potassium Content of Rat Atria. R. R. PARADISE, PH.D., and L. K. GRIFFITH, B.S., *Departments of Anesthesiology and Pharmacology, Indiana University, Indianapolis, Indiana.* Research involving the effects of inhalation anesthetics on isolated contractile tissues has generally been hampered by the lack of a suitable method of metering quantities of anesthetics into the bathing medium which will affect the tissue in a constant and reproducible manner. *Apparatus:* An electronic device which will accomplish this function has been developed recently in our laboratory. With our device, the Anesthetistat, any reasonable control lever can be obtained