and 45-minute intervals after the intravenous injection of 0.6 mg./kg. of *d*-tubocurarine. The average fall in mean arterial blood pressure at the 15- and 45-minute intervals was 11 and 13 per cent respectively. However, immediately after the injection of *d*-tubocurarine, mean arterial blood pressure fell to an average of 16 per cent (range − 8 to − 28). The duration of this change varied from 10 to 90 minutes. In all instances blood pressure returned to control levels. The average cardiac output and stroke volume fell 15 per cent during the 15- and 45-minute intervals, but there were no significant changes in heart rate and total peripheral resistance. In group B during the continuous intravenous infusion of 0.2 per cent solution of succinylcholine (8 mg. per minute), the determinations were repeated at similar time intervals. Mean arterial blood pressure fell 12 per cent (range: 3 to 16 per cent) within one to 6 minutes. Immediately following this transient fall, mean arterial blood pressure gradually increased to an average of 14 per cent above the control level (range: + 9 to + 35 per cent). The duration of this increase varied from 4 to 20 minutes. Cardiac output was unchanged at the 15-minute interval and was decreased 14 per cent at the 45-minute interval (range: + 7 to − 42 per cent). The reduction of the stroke volume paralleled the decrease of cardiac output while heart rate and mean circulation time remained unchanged. Total peripheral resistance was increased 18 per cent at the 15-minute interval and 28 per cent at the 45-minute interval. The results indicate that the reduction of cardiac output following the administration of *d*-tubocurarine is primarily due to reduction of stroke volume. The fall of blood pressure can be related to diminished cardiac output since there was no significant change in the total peripheral resistance. The cause for the transient drop of mean arterial blood pressure during succinylcholine infusion is obscure. Although cardiac output was reduced, there was a subsequent rise in mean arterial blood pressure which can be attributed to the increase in total peripheral resistance. It is of interest to note that reduction of stroke volume appears to be the primary cause for the decrease in cardiac output with both drugs. Further study on the mechanisms of these changes is in progress. (*Supported by a grant from the U. S. Public Health Service No. H-1711 C5.*)

Measurement of Electrical and Mechanical Events of the Cardiac Cycle During Cyclopropane Anesthesia. DAVID M. LITTLE, JR., M.D., AND JAMES B. GIVEN, M.D. *Department of Anesthesiology, Hartford Hospital, Hartford, Conn.* Most previous observations of the effect of cyclopropane anesthesia on cardiac activity have been concerned primarily with two aspects of cardiac activity, output and rhythm. The present study was designed to investigate some of the other effects of cyclopropane 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 the 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 second tone (mechanical ventricular systole); (3) first tone to carotid pulse rise (approximate isometric contraction period); (4) Q wave to carotid pulse rise (indirect isometric contraction period); and (5) R to 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 under cyclopropane anesthesia. Cyclopropane was administered by the closed circle, carbon dioxide absorption technique with controlled and/or assisted respiration, and anesthesia was monitored electroencephalographically to maintain pattern four as described by Possati and coworkers. Arterial blood samples were drawn at the time that the cardiac cycle records were taken during cyclopropane anesthesia, and were analyzed for *p*H, pCO_2, O_2 content and hematocrit. The mean arithmetic average of the Q wave to first tone interval during the control period was 0.062 seconds, while the average for this same interval during cyclopropane anesthesia was 0.069 seconds. The difference between these two figures is not statistically significant. The total duration of

mechanical ventricular systole, as measured by the first tone to second tone interval, was 0.339 seconds before anesthesia, and 0.362 seconds during the administration of cyclopropane anesthesia. This, also, is not a statistically significant difference. The approximate isometric contraction period, as measured by the first tone to carotid pulse rise interval, was 0.078 seconds prior to anesthesia, and 0.089 seconds during cyclopropane. Once again, this is not a statistically significant difference. The Q wave to carotid pulse rise interval, representing the indirect isometric contraction period, was 0.141 seconds before operation, and 0.160 seconds during cyclopropane anesthesia. The statistical difference between these two figures is not significant. The R to R′ interval was 0.824 seconds, representing an average heart rate of 74 beats per minute, before anesthesia, and 0.983 seconds, representing a relative bradycardia of 63 beats per minute, during cyclopropane. The arterial *p*H, at the time that these cardiac cycle measurements were made, averaged 7.39; the arterial pCO_2 averaged 36.9 mm. of mercury; the arterial O_2 content averaged 20.76 volumes per cent; and the hematocrit averaged 39.8 per cent. The results indicate that the administration of cyclopropane anesthesia during pelvic surgery did not alter the normal relationships of the measured intervals of these electrical and mechanical events of the cardiac cycle. The data are in contrast to similar measurements previously reported during deep ether anesthesia, which showed relative prolongations of the Q wave to first tone interval, the first tone to carotid pulse rise interval, and the Q wave to carotid pulse rise interval; those findings were interpreted as suggesting that deep ether anesthesia produced a relative inhibition of both the electrical spread of the depolarizing wave in the ventricular myocardium and the rapidity of the mechanical contraction of the ventricle. (*Supported by a grant from Burroughs Wellcome & Co. (U.S.A.) Inc.*)

Arterial Oxygen Desaturation in Severely Burned Patients: Its Significance to the Anesthesiologist. FRED K. MCCUNE, CAPT. MC, GLEN K. ARNEY, LT. COL. MC, CHARLES R. BAXTER, CAPT. MC, JEROME J. DEGOSSE, CAPT. MC, AND JOHN A. JENICEK, LT. COL. MC. *Brooke Army Medical Center, Fort Sam Houston, Texas.* Anesthesia or analgesia in the severely burned patient causes deleterious physiological effects, the nature of which is not fully understood. Many investigators have made the clinical observation that oxygenation of these patients is often inadequate despite anesthetic techniques providing maximum concentration of oxygen. A study of oxygen uptake by the lungs was begun. Sixty-six determinations, including oxygen saturation, pO_2, pCO_2 and blood *p*H were made on 24 patients in this series. These studies revealed that oxygen saturation is below normal following the fifth post-burn day in patients with a burn index greater than 20 per cent. It remains below normal for varying periods, depending upon the extent of the burn. The return to normal blood oxygenation, in most cases, correlates closely with wound coverage. Oxygen desaturation in the acutely burned patient is of physiological significance to the anesthesiologist. It reduces the margin of safety in these patients, particularly for administration of inhalation techniques. Analyzing the records of 17 patients, the incidence of anesthetic administration was plotted against post-burn day. Forty-four per cent of all anesthetics were administered between the fifth to thirtieth post-burn day. This is the period in which all patients showed oxygen desaturation. A laboratory study of cardiopulmonary response to acute lethal burn was made in 12 dogs. Parameters measured were arterial blood pressure, venous pressure, intrapulmonic pressure, cardiac output, mean circulation time, intrathoracic blood volume, total peripheral resistance and ECG. The results are being evaluated to determine clinical applicability. Using the Waters-Conley ear oximeter, an attempt is being made to determine if there is a point during the course of anesthesia when sustained refractory arterial oxygen desaturation occurs. Recordings are made before preoperative medication is given, one hour after administration of preoperative premedication, during induction of anesthesia, throughout the course of anesthesia and post-operatively during the recovery period. Evidence thus far indicates that such a point does exist.