concentrations with V_{max} reduced 22 per cent to 34 per cent and P_0, 19 per cent to 58 per cent, in proportion to the concentration of halothane. Maximum work and power, and the loads at which these maxima were achieved, also fell. All length-developed tension curves were symmetrically depressed by halothane. Stress-strain curves of series elasticity were moved upwards and to the left by 2.0 per cent and 3.1 per cent halothane but were unchanged by the 0.7 per cent concentration. The changes in developed tension and series elasticity associated with 2.0 per cent and 3.1 per cent halothane, and all regression lines relating anesthetic-induced effects to the three halothane concentrations studied, were statistically significant. There were no statistically significant changes in resting tension. Simultaneous display of changes with time of velocity of shortening of the contractile element (CE) and tension indicated that both were initially depressed by halothane. However, from approximately half the time to peak tension to the point at which the CE stopped shortening, CE velocity was unaffected by halothane. During this period, the developed tension remained decreased in the presence of the anesthetic. When the extent of the CE shortening plotted against the load was extrapolated to zero load, this also was unchanged by halothane. In addition, the force velocity constant "a" and "b" were not altered by the anesthetic. *Discussion:* Although the velocity of shortening of the CE under zero load (V_{max}) is depressed by halothane, given enough time at zero load the CE could shorten as much as it did in the absence of halothane. Also, even though the CE produces less shortening with loads above zero in the presence of halothane, the mechanical equivalents of the amount of heat produced per unit of shortening ("a"), and the rate of liberation of extra energy per unit change in load ("b"), remain unchanged. These results are consistent with the concept that halothane directly depresses heart muscle, with some increase in series elasticity and no change in parallel elasticity. However, the effect of halothane on the contractile element may be an indirect one, with the primary site of action on those mechanisms which link excitation to contraction. (Supported in part by USPHS Grant GM-HE-14719-01 and Career Development Award 5-K3-HE-17949-02.)

Effects of General Anesthesia on Sicklemic Patients. WALTER S. GUINEE, M.D., JAMES A. HEATON, D.D.S., LUIS BARRERAS, M.D., and L. W. DIGGS, M.D., *Departments of Anesthesiology and Medicine (Hematology), University of Tennessee, Memphis, Tenn.* Patients with sickle-cell anemia were studied to determine whether general anesthesia is particularly hazardous in this disease. *Methods:* Patients with SS, SC, or S-thalassemia hemoglobin who were to undergo elective surgery were investigated. The selection of anesthetic drugs was systematically randomized between flammable and nonflammable agents depending upon the need for electrocautery. Premedication consisted of atropine alone or combined with hydroxyzine. Records were kept of drugs and anesthetic gases used, types and amounts of fluid given, and urinary output. Arterial and venous blood samples were analyzed for P_{O_2}, P_{CO_2}, *p*H, buffer base, HCO_3^-, and percentage of sickled erythrocytes as determined by the Sherman Test (Sherman, I. J.: Bull. Johns Hopkins Hosp., July, 1940: p. 309), before and after premedication, during induction, during operation, in the immediate post-anesthetic period, and 24 hours postoperatively. At each sampling, blood pressure, pulse, temperature, tidal volume and minute volume were measured. *Results:* Twelve patients have been studied. In all, as would be expected, the blood oxygen tension was increased during anesthesia. The P_{CO_2} and *p*H were not significantly altered. In all patients, however, the percentage of sickled cells was markedly reduced in both arterial and venous samples. This was also true in the 24-hour post-anesthetic samples when all other measurements had returned to pre-anesthetic values. In one patient this reduction persisted for 96 hours after anesthesia. *Conclusions:* Though no definite conclusions can be drawn from this small group, it suggests that general anesthesia is not necessarily harmful to patients with sickle-cell disease.