plantation Test (I.T.), which guarantees their freedom from toxicity should be used. The major cause of reactions, however, is ethylene oxide or its products, ethylene glycol and chlorohydrin, which are formed in the presence of moisture and chloride. All of these are highly irritant, absorb into the sterilized item and only slowly elute. *Results:* The rate of elution is slowed if the article is PVC; if the thickness of the plastic is increased; if ambient temperature rather than 50 C. temperature is used during elution; if Freon/Eto mixtures rather than CO_2/Eto mixtures are used for sterilization; if the article is wrapped in polythene rather than cloth or paper; if the article is PVC and has been previously gamma-ray sterilized. This forms HCl in PVC and more ethylene chlorohydrin (boiling point = 139) is formed. *Summary:* Since maximum tolerable levels of these residuals are unknown, complete elimination must be attempted. Recommendations for achieving this include: (a) adequate aeration of gas-sterilized materials for a minimum of five to seven days at ambient temperature or, better still, aeration at 50 C for six to eight hours in a properly designed aerator with bacterial filters; (b) avoidance of the use of 3-ml polythene wrap, plastics containing acid phthallic ester plasticizers which absorb Eto selectively, and any previously gamma-ray-sterilized items; (c) increased use of disposable items.

Alteration, by Halothane, of the Effect of Isoproterenol on Maximum Acceleration of Ejected Left-ventricular Blood. B. F. Rusy, M.D., R. J. Tallarida, Ph.D., A. I. Karetas, M.D., and M. H. Loughnane, M.S., *Departments of Anesthesiology and Pharmacology, Temple University School of Medicine, Philadelphia, Penna.* The maximum acceleration of ejected left ventricular blood ($\dot{Q}_{max}$) has been shown by Noble *et al.* (Circ. Res. 19: 139, 1966) to be a satisfactory index of the inotropic state of the myocardium. The effects of isoproterenol on $\dot{Q}_{max}$ in the conscious dog have been compared with similar effects observed during halothane anesthesia. The questions asked are to what extent halothane antagonizes the contractile response to isoproterenol, and what the nature of the antagonism is, *i.e.*, is it simple competitive inhibition involving a single receptor, or do these drugs act independently at separate receptor sites? *Methods:* Electromagnetic flow probes were implanted on the ascending aortas of mongrel dogs one or more weeks prior to study. $\dot{Q}_{max}$ was computed electronically as the first time derivative of instantaneous aortic flow. The effects on $\dot{Q}_{max}$ of 10, 15 and 20 μg isoproterenol, injected intravenously, were observed first in the conscious animal and then during 1.25 per cent halothane anesthesia. *Results:* The $\dot{Q}_{max}$ response to isoproterenol is markedly depressed by 1.25 per cent halothane. The absolute value of the response to 15 μg isoproterenol during halothane anesthesia is 56 per cent of the response observed during the conscious state ($P < 0.05$). During halothane, a definite plateau in the curve of $\dot{Q}_{max}$ response *vs.* dose of isoproterenol is observed. In order to predict what the maximum obtainable $\dot{Q}_{max}$ responses to isoproterenol would be, Lineweaver-Burke plots of $1/\dot{Q}_{max}$ *vs.* 1/dose isoproterenol were constructed. The y intercepts of these plots (representing the maximum responses theoretically obtainable) were significantly ($P < 0.02$) less during halothane anesthesia. *Summary:* The antagonism of the positive inotropic effect of isoproterenol by halothane is not one of simple competitive inhibition; rather, these agents probably act independently at separate receptor sites.

Correlation of Mechanical and Electrical Events in Depolarization Paralysis of Isolated Human Intercostal Muscle. P. B. Sabawala, M.D., *Baylor University College of Medicine, Houston, Tex.* When depolarizing drugs are allowed to remain in contact with isolated nerve-muscle preparations in unchanged concentrations, a paralysis develops due to a persistent depolarization at the endplate and at the small area of muscle membrane immediately surrounding it (J. Physiol. 115: 41, 1951). Later evidence showed that this period of depolarization is short and that the muscle remains paralyzed in spite of repolarization of the membrane (Acta Physiol. Scand. 34: 218, 1955). *Methods:* We have repeated these experiments using the isolated human intercostal muscle and extracellular electrodes in