Correspondence

Hepatic Halothane Metabolism

To the Editor:—In our paper "Concentration Dependence of Hepatic Halothane Metabolism" (ANESTHESIOLOGY 34:230–235, 1971) we have misled several readers, including Dr. Brown in his otherwise perceptive editorial "Minipigs, Microsomes, Metabolism, and Maupassant" (ANESTHESIOLOGY 34:217–218, 1971). We unwittingly convinced Dr. Brown "that halothane acutely inhibits its own metabolism. . . ." We do suggest this as one of two possible explanations for our inability to detect removal of halothane by the liver at higher concentrations. This inability, however, is probably a function of the crudeness of our measurement. It may well be that a small fraction of halothane brought to the liver is biotransformed at higher concentrations. In that case, substrate saturation of the metabolizing enzyme system would explain our results. Both substrate saturation and inhibition by high substrate concentrations are common findings in enzyme chemistry (Dixon, M., and Webb, E. C.: Enzyme Kinetics, Enzymes, New York, Academic Press, Inc., 1958, pp. 73–89). Our data cannot be used to discriminate between these two possibilities for halothane.

It appears that we may have confused the reader on one other point: the definition of "metabolism" or "rate of metabolism." Rate of metabolism may be defined by the milliliters of a substance biotransformed per unit time. This definition is simple and widely accepted. Rate of metabolism also may be defined by the fractional rate of biotransformation. This definition is equally accepted (for example, see: Mark, L. C., Perel, J. M., Brand, L., and Dayton, P. G.: Studies with thiohexital, an anesthetic barbiturate metabolized with unusual rapidity in man, ANESTHESIOLOGY 29:1159–1166, 1968), and though a bit more complex is more useful in a description of drug kinetics.

EDMOND I. EGER, II, M.D.
Department of Anesthesia
School of Medicine
University of California,
San Francisco
San Francisco, California 94122

DONALD C. SAWYER, D.V.M., PH.D.
Department of Small Animal
Surgery and Medicine
College of Veterinary Medicine
Michigan State University
East Lansing, Michigan 48823

Coronal Discharge during Countershock

To the Editor:—The range of interest and activity of today's anesthesiologist takes him far from his classic area of endeavor, the operating room. Interest in electrical safety, at one time limited to the operating room, now extends throughout the hospital setting.[1] With this in mind, we wish to cite a phenomenon which recently occurred in the Coronary Care Unit of our Hospital.

The history and physical findings of the patient involved are irrelevant except that the patient had had repeated episodes of tachyarrhythmia, each deteriorating into ventricular fibrillation. Each episode required electrical defibrillation. Also pertinent is the fact that the patient was asthmatic. Over a six-day period, until his death, he received 14 countershocks. At the onset of each arrhythmia, the patient became markedly diaphoretic. Because of his bronchopulmonary problem, he received an open, ultrasonically-generated mist at his bedside. At the time of the thirteenth coun-