

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.

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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-

tershock, the nurse who administered the shock noted arcing between the electrode paddles. Moments later, another attempt at defibrillation again resulted in arcing. However, at this time the entire cubicle, including articles therein, glowed with an aura of light. The odor of singed hair was noted. The odor of ozone was denied, although with uncertainty. The cardiologist in charge abandoned further attempts at defibrillation. Since we are deeply involved in cardiopulmonary resuscitation, and since Inhalation Therapy is under our auspices, we were asked to ascertain what had happened.

The entire incident was reminiscent of descriptions of coronal or brush discharge, known as St. Elmo's fire. In this case, we surmise that repeated applications of electrode paste and profuse diaphoresis provided a preferential electrical pathway which permitted arcing over the skin surface. This, in turn, ionized the heavy droplet concentration from the ultrasonic nebulizer, thereby providing further alternate pathways for the subsequent discharge to follow. This resulted in the coronal discharge observed by nursing personnel in the unit.

We thought that perhaps an ultrasonic nebulizer might impart an electrical charge to each droplet, thereby creating an electrical conductor.² This explanation would appear to be

invalid in this instance. While an ultrasonic nebulizer does impart a charge to the droplet it generates, the net charge of the mist is zero.³ Apparently, this was purely an ionization phenomenon followed by coronal discharge.

We believe this is another instance in the growing list of electrical hazards. While the coronal discharge itself is allegedly harmless, the arcing may not be, and therefore we are: 1) wiping the skin free of perspiration and previously applied electrode paste prior to countershock; and 2) banning the use of "open"-type high-humidity generators in the vicinity of patients potentially in need of countershock.

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REFERENCES

1. Electrical Hazards in Hospitals: Proceedings of a Workshop. Edited by C. Walter. Printing and Publishing Office, National Academy of Sciences, 2101 Constitution Avenue, Washington, D. C.
2. Lelacheur RM: Personal communications
3. Gauthier WD, Lierke EG: Determination of particle charges on ultra-sonically generated aerosols. *J Asthma Res* 7:165 (June) 1970

Vascular Responses in Arteriosclerotic Patients

To the Editor:—The article by Bonica *et al.*¹ provided long overdue information about vascular responses in normal awake man. We have recently completed a study of peripheral circulatory effects of peridural block, as an adjunctive technique, in patients undergoing vascular surgery in the lower limb.² We agree in general terms with Bonica's warning about abolishing sympathetic tone in the presence of myocardial disease, but we would like to comment on this. The patients in our series had myocardial disease ranging from mild to

severe and were under light general anesthesia for insertion of the vascular graft and subsequent pre-block blood-flow measurements. After peridural block to T8-9, there was no significant fall in blood pressure or CVP, while graft blood-flow measurements showed a highly significant rise.

The application of the dramatic increase in limb blood flow reported by Bonica has been demonstrated in our series of arteriosclerotic patients. The achievement of an increase in graft blood flow with little change in systemic