

chondrial performance could have arisen from a variety of secondary effects induced by halothane; for example, altered levels of endogenous metabolites or metabolic derivatives of the anesthetic agent itself could induce transient or longer-lasting alterations in mitochondrial function. Therefore, extrapolating the findings of *in vitro* models to our study overlooks a fundamental distinction between the two kinds of study.

Our assay media contained 8.5 mM K_2HPO_4 and 10 mM tris buffer with a pH of 7.4. We regret not including this information. However, workers studying isolated mitochondria are generally aware that measurement of such indices as RCR and ADP/O ratio according to the method of Chance and Williams requires an adequately-buffered medium and the presence of inorganic phosphate.⁵

In conclusion, we believe that our work offers a valid alternative approach to the study of the effects of anesthetic agents on mitochondrial function, and hope that it will be of value to other investigators in this field.

W. SCHUMER, M.D.
P. R. ERVE, Ph.D.
Department of Surgery
University of Illinois College of Medicine
at Veterans Administration
West Side Hospital
Chicago, Illinois 60612

REFERENCES

1. Mela L, Miller LD, Diaco JF, et al: Effect of *E. coli* endotoxin on mitochondrial energy-linked functions. *Surgery* 68:541-549, 1970
2. Johnson CL, Schwartz A: Some effects of local anesthetics on isolated mitochondria. *J Pharmacol Exp Ther* 167:365-373, 1969
3. Schumer W, Erve PR: Bovine serum albumin effect on endotoxin-challenged mitochondria. *Surgery* 69:699-701, 1971
4. Cohen PJ, Marshall BE: Effects of halothane on respiratory control and oxygen consumption of rat liver mitochondria, *Toxicity of Anesthetics*. Edited by BR Fink. Baltimore, The Williams and Wilkins Company, 1968, pp 24-36
5. Chance B, William GR: Respiratory enzymes in oxidative phosphorylation. Kinetics of oxygen utilization. *J Biol Chem* 217:383-393, 1955

Crawford Long of Athens/Jefferson, Georgia

To the Editor:—In his recent article about anesthetic history (ANESTHESIOLOGY 35: 515, 1971), Dr. Greene stated, "In 1842, Crawford Long of Athens, Georgia. . . ." This contrasts to a statement in *The History of Surgical Anesthesia* by T. E. Keyes (1945), p. 22, ". . . Crawford W. Long of Jefferson, Georgia. . . ." Having once lived in Athens, Georgia, I know Jefferson, Georgia is close by, but distinctly separated from Athens. I wonder, therefore, why the discrepancy about Long's habitat.

JAMES E. HEAVNER, D.V.M., Ph.D.
Anesthesia Research Center
University of Washington
Seattle, Washington 98195

To the Editor:—The confusion about Crawford Long's habitat centers about the fact that although Long was in practice in Jefferson in 1842, at the time he first administered ether anesthesia, he spent the majority of his professional career (1851-1878) in Athens. He was, accordingly, identified by his contempo-

raries as a physician from Athens at the time his role in the discovery of anesthesia became widely recognized. One of the more important events in this belated recognition was, of course, the visit of Charles T. Jackson to Athens in 1854 to talk to Long. Jackson, not without controversy himself in the discovery of ether anesthesia, became so impressed with Long's priority in this matter that he wrote a letter to the U. S. Senate, at that time considering a congressional award to the "discoverer of anesthesia." Jackson's letter was so convincing that it eliminated Morton, as well as Jackson himself, as contenders for the award, an idea subsequently abandoned.

So, perhaps, to be entirely precise, we should say "Crawford Long, of Athens, Georgia, who first administered ether anesthesia in Jefferson, Georgia, in 1842." This seems a bit awkward.

NICHOLAS M. GREENE, M.D.
Yale University School of Medicine
New Haven, Connecticut 06510