CORRESPONDENCE

Effect of Cyclopropane on Myocardial Contraction and Membrane Potentials

EDITOR'S NOTE: *Dr. Henry L. Price of the University of Pennsylvania was asked to comment on the paper, "Effect of Cyclopropane on Myocardial Contraction and Membrane Potentials," by Drs. Levy, Ichiyanagi and Frederickson, appearing on page 185 of this issue. The authors in turn were given the opportunity to defend the methodology and interpretation.*

To the Editor. I enjoyed reading the article by Levy and co-workers, but I find it discouraging that, in the end, they cannot be certain whether cyclopropane or lack of oxygen caused the results which they observed. In fairness to themselves they could have pointed out that certain effects of anoxia (decreased membrane resting and action potentials) were different from those caused by cyclopropane. But in metabolically-active unperfused preparations the oxygen tension below which function deteriorates may be several hundred millimeters of mercury, as these authors found. The most convincing way of excluding an effect from changing oxygen tension under these conditions is to keep that tension constant. This can be done by substituting some inert gas for cyclopropane in making the control observations.

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To the Editor. It is evident in the paper that we, too, were somewhat discouraged that we could not get a definite answer as to the effect of cyclopropane on transmembrane action potentials. We attempted to do this by another method which is described in the article; and using this method we thought that we had reasonably ruled out the effect of hypoxia in the experiment. As mentioned in Dr. Price's comment, the effect of hypoxia is also usually associated with a decreased membrane resting potential and a decreased action potential, but the early effects do sometimes resemble the effects shown in this experiment; therefore, the attempt to have an adequate control. In checking the effect of other anesthetic agents on transmembrane action potentials, it is possible to achieve an adequate pharmacologic effect without decreasing the tension of oxygen to any significant degree with a more potent agent. This work will be reported in the near future, and we hope to be able to generalize on the action of anesthetic agents on membrane electrical functions. The method described by Dr. Price will work well in future experiments on agents requiring a high partial pressure to achieve an effect. We sincerely appreciate his interest in this work.

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HYPERKALEMIA Four adult patients with dangerous states of hyperkalemia, as evidenced by characteristic ECG patterns and markedly elevated serum potassium levels, were rapidly improved by the intravenous administration of 200–250 ml. of hypertonic saline (5 per cent). Within minutes the ECG showed reversion toward normal and serum potassium and sodium levels improved over the next several hours. This treatment may be life saving. (*Garcia-Palmieri, M.R.: Reversal of Hyperkalemic Cardiotoxicity with Hypertonic Saline, Amer. Heart J. 64: 483 (Oct.) 1962.*)