LETTERS TO THE EDITOR

Letters to the Editor will be published, if suitable, and as space permits. They should not exceed 1,000 words (double spaced) in length, and may be subject to editing or abridgment.

Role of the Vagus in Acute Myocardial Infarction

To the Editor:

In a recent paper (Circulation 47: 291, 1973) Kent and his colleagues attempted to define certain electrophysiologic alterations induced by changes of heart rate or vagal nerve stimulation after coronary artery ligation. The authors conclude that any increase in heart rate, or an inhibition of vagal tone, has deleterious effects on the electrical stability of ischemic myocardium.

The first conclusion by these authors was that increasing heart rates (from 60 to 120 or 180 beats/min) increased the disparity of refractory periods in ventricular muscle. My concern is that refractory period measurements were apparently confined to the ischemic zone. Since ischemia shortens the action potential of the involved muscle, and an increase of heart rate shortens the action potential of normal muscle, it is entirely possible that the net effect of increasing heart rate was to decrease the disparity of refractory periods of the ventricle as a whole. It also concerns me that alterations of conduction within the ischemic zone were not considered in the analysis of refractory periods; and finally, that nowhere in the paper are actual refractory period measurements presented. We cannot tell if ischemia and/or heart rate shortened or lengthened the refractory periods.

A second conclusion was that increasing heart rate enhanced vulnerability to fibrillation. Trains of pulses starting 80 msec after the onset of the QRS complex and ending 50 msec after the T wave were employed. We do not know whether this train produced one or more premature beats prior to its termination. We do not know whether the duration of the train was adjusted when changes of the QT interval occurred. In figures 3 and 5 the fibrillation threshold was presented in milliamperes, but in figures 4 and 6 it was presented as a percent of control measurements. In figure 3 results were presented which allegedly show that an increase in heart rate decreased the fibrillation threshold; the control fibrillation threshold was 89 + 31 milliamperes.

In figure 5 which allegedly depicts that vagal stimulation increased fibrillation threshold, the control values were 26 ± 3 milliamperes. Why was there a greater than threefold difference in the control observations in the two series of animals? Why was the fibrillation threshold 89 milliamperes in the one series, a value 3 to 4 times higher than reported by other investigators?

Finally, it was concluded that a decrease in vagal tone led to a decrease in fibrillation threshold. In the methods section it stated that, "When heart rate was controlled by vagal stimulation, the right and left cervical vagi were isolated and stimulated through platinum electrodes." In the legend to figure 5 it is stated that, "In three of the animals, the vagi were decentralized with no significant change in the response." Are we to assume, therefore, that all of the experiments which were presented and which employed vagal stimulation, were based on stimulation of undivided vagi? Certainly the authors would agree that coincident central and distal stimulation of the vagi complicates unnecessarily the interpretation of their results and is less than a precise or controlled way to characterize cholinergic influences on the ventricle.

I believe there are serious methodologic faults with these experiments. I do not believe they provide a basis for discarding the experiments by Han and his colleagues which show the opposite effect of heart rate on ventricular fibrillation threshold. Finally, I do not believe that stimulation of undivided cervical vagi permits valid interpretations regarding cholinergically-mediated changes in the electrical stability of ischemic myocardium.

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The authors reply:

To the Editor:

We are surprised at Dr. Wallace’s comments, since they reflect several serious misconceptions regarding the results reported in our recent investigation1 as well as those reported by Han and coworkers.2 We therefore appreciate this opportunity to clarify the issues raised by Dr. Wallace.

Dr. Wallace incorrectly interprets our data as purporting to provide a basis for discarding the data by Han et al. In fact, we have confirmed Dr. Han’s important findings that increasing heart rate decreases disparity of refractory periods in nonischemic myocardium. Dr. Han, however, did not study the responses to changes in heart rate in the presence of myocardial ischemia. Unfortunately, Dr. Wallace, as well as others, apparently assumed that Dr. Han’s results could be extrapolated to the ischemic situation. Our study was designed to determine the validity of just such an assumption, and our data clearly demonstrate that the above extrapolation is invalid. Increasing heart rate in the presence of ischemia adversely affects both disparity of refractory periods and ventricular fibrillation threshold.

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