Exercise and VPCs

To the Editor:

The following is in response to Dr. David S. Sheps article entitled, Decreased frequency of exercise-induced ventricular ectopic activity in the second of two consecutive treadmill tests.

We cannot agree with the author's statement that consecutive testing is a poor method for studying exertional arrhythmia using standardized maximal treadmill tests. In the first place, none of our repeat tests were done at 45 minutes; this is clearly insufficient time for recovery as noted by the authors. Our studies showed slightly longer second tests with slightly higher maximal oxygen consumption (Ann Intern Med 80: 711, 1974). Recovery rate may be affected by fitness, a factor not considered by the authors.

Another factor which may influence the arrhythmia prevalence is selection of subjects. Many of our subjects were tested several times and showed frequent reproducible ventricular arrhythmia. Currently, we have in press a double-blind study which shows reproducibility as high as $r = .99$ for arrhythmia frequency.

There is some week to week variation but this can be partially adjusted for by considering % reduction with two consecutive tests. However, qualification of success with 100% suppression would resolve many uncontrolled variables when testing an antiarrhythmic drug.

We agree that physiological variables should be reproduced insofar as possible since pressure-rate product is indirectly related to myocardial work. This is clearly altered with many of the antiarrhythmic drugs and may be the mechanism by which they are effective.

Psychological stress remains an uncontrolled variable related to arrhythmia which is only partially resolved with maximum testing in that variability of maximum values is significantly less than those at rest or submaximal exercise.

It is surprising that the study which shows an $r$ correlation of .92 for frequency of arrhythmia and no significant change in severity of arrhythmia is probably the best correlation for reproducibility published for ambulatory patients. An optimistic view would be more realistic, $r^2 = .85$ with .15 to be explained.

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The authors reply:

To the Editor:

We agree with Dr. Gey that one of the factors which may influence arrhythmia prevalence is selection of subjects. We stated clearly in the Materials and Methods section of our manuscript that the patients selected for our study were previously identified as having frequent ventricular premature beats (>10 VPCs) during or after maximal treadmill exercise. Thus, our patients represented a select group in which arrhythmias were previously shown to be reproducible on two separate tests done on separate days. Therefore, our results cannot be explained by nonreproducibility of exercise-induced VPCs.

Careful study of figure 1, which shows the relationship between the number of VPCs on Test 1 and Test 2, demonstrates that there is a good correlation ($r = .92$) between these two values. However, there is always a lower number of VPCs during the second test.

The study by Dr. Gey which he refers to employed a protocol similar to ours except for a rest period of one hour between tests, as compared to 45 minutes in our study. Reference to their table 1

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The authors reply:

To the Editor:

It is not clear from his letter what statistical method Dr. Wu used to analyze our data. The correct method of analysis in this situation would be the paired $t$-test. We had performed this latter analysis in our initial evaluation of our data. This type of analysis of course assumes a unimodal distribution and it is clear from the amount of deviation that considerable scatter is present. It was for this very reason that we stated clearly in our paper that no a priori prediction of the effect of digitalis could be made. The direction and magnitude of change observed depended on the individual patient and not the dose of digoxin nor the value of the shortest R-R interval. Thus, despite the fact that there was no overall mean change in the intervals recorded before or after digitalis in the total group, definite shortening of the cycle length of the most rapid ventricular response was noted in one-third of the patients.

We agree with Dr. Wu that progressive hemodynamic deterioration over a prolonged period of time could certainly contribute to susceptibility to ventricular fibrillation and in this situation, delay of appropriate therapeutic measures because of the administration of digoxin could have contributed to a false association between drug administration and the onset of ventricular fibrillation. However, details of the clinical history not included in our manuscript because of space limitation suggest that this was not the case in most instances. Patient DE had multiple episodes of tachycardia lasting more than 4 hours. However, ventricular fibrillation only occurred after he had been administered intravenous digoxin. Atrial fibrillation induced in the absence of medication did not result in ventricular fibrillation. Patient KH had several episodes of tachycardia prior to his digitalization. Subsequent to his oral digitalization, he suffered ventricular fibrillation with his first and only episode of tachycardia. Patient SH had multiple episodes of atrial fibrillation prior to digitalization which lasted 5 to 6 hours or longer at a time. None of these deteriorated into ventricular fibrillation. However, after three separate courses of oral digoxin, she suffered ventricular fibrillation on each occasion she experienced atrial fibrillation. Once digoxin was discontinued, no further episodes of ventricular fibrillation occurred. Patient JM had multiple episodes of atrial flutter with 1:1 A-V conduction at 300 per minute lasting up to several hours. Only after oral digitalization did she develop ventricular fibrillation within 1 hour of onset of atrial flutter. Patient RR had multiple episodes of tachycardia prior to digitalization. However, following institution of digoxin, her next episode of arrhythmia deteriorated in one hour into ventricular fibrillation. The other patients mentioned in our paper had ventricular fibrillation occurring more than 4 hours after the onset of their tachyarrhythmia.

Since the submission of this manuscript, we have identified two additional patients who developed ventricular fibrillation only after digitalization. Since the response of an individual patient to digoxin cannot be predicted a priori, and since the ventricular response to atrial fibrillation became more rapid in one third of our patients after administration of digoxin, we believe that digoxin should be avoided in patients with the WPW syndrome unless the ventricular response during atrial fibrillation has been observed.

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