Focused Perspectives

Exercise-Induced Premature Ventricular Beats
Should We Do Anything Differently?

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Exercise testing has typically been used to diagnose myocardial ischemia and to risk stratify patients with known coronary disease. The interpretation of stress testing has rested largely on the development of ST-segment changes and the presence of anginal symptoms. Stress tests have also been used to help categorize and treat various cardiac arrhythmias.

Single ventricular premature beats (VPBs) that occur at rest or during exercise are a cause of anxiety for patients and their physicians. However, the implications of exercise-induced VPBs are unclear. The prevalence and clinical significance of VPBs during and after exercise have been investigated for many years. Most of the studies have focused on the predictive value of exercise-induced VPBs in patients referred for diagnostic exercise testing. Of these, 3 studies showed no correlation between any exercise-induced VPBs and all-cause mortality over a follow-up period of 3 to 5 years. One study showed an association between frequent VPBs and mortality, but only after 8 years, and another large study determined that frequent VPBs occurring after exercise might be more predictive of mortality than VPBs occurring during exercise.

Over the last several years, 4 studies, including that by Morshedi-Meibodi et al in the present issue of Circulation, have been conducted in asymptomatic subjects with no evidence of cardiovascular disease. Busby et al followed 1160 men and women ranging from 21 to 96 years of age from the Baltimore Longitudinal Study of Aging for a mean of 5.6 years. Cardiac events and all-cause mortality were not increased in the 80 subjects with frequent exercise-induced VPBs when compared with an age- and gender-matched control group.

Mora et al examined a cohort of 2994 asymptomatic women 30 to 80 years of age enrolled in the Lipid Research Prevalence study over 20 years of follow-up. There was no relationship between frequent exercise-induced VPBs and all-cause mortality, but their presence did correlate to an increased risk of cardiovascular death. This finding is in contrast to the Busby et al study; however, the follow-up was significantly longer.

Jouven et al exercised 6106 asymptomatic French male civil servants ranging in age from 42 to 53 years. They reported that frequent VPBs during exercise, defined as >10% of all ventricular depolarizations during any 30-second recording, occurred in 138 (2.3%) subjects and were associated with an increased risk of cardiovascular death over a period of 23 years. They also noted that frequent VPBs occurring after exercise were associated with an increase in noncardiovascular and all-cause mortality. In the 540 (9.5%) subjects with fewer VPBs than 10% of all beats in any 30-second recording during exercise, there was no increase in either cardiovascular or noncardiovascular mortality.

In the present issue of Circulation, Morshedi-Meibodi et al correlate exercise-induced VPBs to 15-year outcome in 2885 asymptomatic participants of the Framingham offspring study. They recorded VPBs in 792 (27%) of their subjects. However, the VPBs during exercise exceeded 10% of all ventricular depolarizations in only 4 (0.1%) of the participants. For that reason, they defined the VPBs as frequent when they exceeded the median of 1 VPB per 4.5 minutes of exercise (0.22 VPBs/min). They concluded that neither infrequent nor frequent VPBs during exercise were associated with a greater risk of “hard” cardiovascular end points, defined as coronary insufficiency, myocardial infarction, or sudden cardiac death. However, in their secondary analysis, frequent VPBs were associated with an increased risk of death due to coronary heart disease, stroke, heart failure, and other vascular diseases. They also reported that VPBs, both infrequent and frequent, were associated with an increased risk of all-cause mortality, with many of the deaths due to malignancy.

It is difficult to compare the results of this study with those of earlier studies because the authors chose to isolate hard cardiovascular end points from “secondary” cardiovascular end points. However, there are noteworthy study design differences between this and previous studies. The cohort in this study experienced a much lower frequency of exercise-induced VPBs than in the prior studies. For this reason, the authors defined “frequent” VPBs differently than in previous studies. Unlike some of the other studies (the Jouven et al study, for example), subjects with VPBs at rest were excluded, and the exercise and ECG monitoring protocols and the follow-up periods differ. In addition, the age groups and percentages of women varied between the studies, and there were probably differences in racial diversity and tobacco use.

Important similarities were also present. As in the present study, the study by Jouven et al failed to establish a
The relationship between less frequent VPBs and cardiovascular events, and none of the studies demonstrated an interaction between exercise-induced VPBs and ST-segment changes, indicating that the exercise-induced VPBs could not be attributed to ischemia.

Morshedi-Meibodi et al. also recorded fractional shortening, determined by echocardiogram, and concluded that the presence of exercise-induced VPBs was unaffected by ventricular systolic function.

How then do these studies, taken together, impact on our understanding of the electrophysiological cause of the VPBs, their etiology, and our response to their presence?

It seems reasonable to assume that the presence of VPBs indicates a region of increased excitability and that their relationship to exercise suggests catecholamine sensitivity. But the studies provide no further insight into whether the relationship to exercise suggests catecholamine sensitivity. How then do these studies, taken together, impact on our understanding of the electrophysiological cause of the VPBs, their etiology, and our response to their presence?

It seems reasonable to assume that the presence of VPBs indicates a region of increased excitability and that their relationship to exercise suggests catecholamine sensitivity. But the studies provide no further insight into whether the VPBs are due to reentry, to enhanced automaticity, or to afterdepolarizations. As noted above and mentioned in other editorials, their presence is not linked to ischemia but is related in a linear fashion to increasing age. This suggests that a change in myocardial substrate over time might have been related to the increase in cardiovascular mortality associated with frequent exercise-induced VPBs.

The studies suggest that exercise-induced VPBs in asymptomatic individuals do not predict an increased risk of cardiovascular end points over the short term (5 years). Thus, there is no evidence to suggest that further evaluations other than a careful history, physical examination, and possibly an echocardiogram be routinely performed in these patients.

The relationship between infrequent exercise-induced VPBs and all-cause mortality reported in the present study is difficult to interpret. Nonetheless, the possibility that exercise-induced VPBs may identify patients at risk for noncardiovascular deaths warrants additional large cohort studies to determine whether this relationship does indeed exist. In addition, the relationship between frequent exercise-related VPBs and cardiovascular end points needs further investigation. Lastly, research focused on the possible mechanisms of exercise VPBs is needed. Perhaps the relationship between exercise-induced VPBs and age, heart rate variability, heart rate turbulence, and other markers of autonomic tone would contribute to our understanding of these anxiety-producing events.

References


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