However, proarrhythmia did not occur during PST in the present study. This is remarkable in a patient population with reduced left ventricular function and may be attributable to the fact that the clinical arrhythmia was VT in all but one patient. In addition, six patients were hospitalized for incessant VT, which typically persists rather stably for a long time without degenerating into VF.

Each paired stimulus produced a mechanical contraction. The magnitude of left ventricular pressure increase, however, was so little that the pressure level either did not or hardly exceeded aortic pressure. Thus, in some patients, the pressure curve did not show any systolic wave associated with the paired stimulus, whereas in others a very small systolic "hump" was present. In addition, analysis of the 12-lead electrogram showed that the tail end of the paced QRS complex sometimes fused with the next VT beat, especially when longer coupling intervals were applied. The supposed (small) re-entrant circuit of the VT origin, however, could not excite most of the ventricular tissue as this was still refractory because of the previous stimulus.

With regard to possible mechanisms, a prolongation of the diastolic interval may be operative: the postpacing cycle length was longer than the VT cycle length, thereby prolonging the ventricular filling time and augmenting the pressure wave elicited by the next VT beat. However, other factors are likely to contribute: when comparing ventricular pacing with ventricular pacing at higher rates but with interpolated paired stimuli so that both stimulation algorithms result in the same rate of arterial pressure waves, paired stimulation increased systolic pressure (3). In addition, after termination of coupled stimulation in dogs (3) and in our study the pressure effect persisted for several beats. This may be because of an elevated myoplasmic calcium concentration contributing to a postextrasystolic potentiation of contractile force, probably attributable to an increased calcium release from the sarcoplasmic reticulum (5).

Possible limitation of PST may include an increase in myocardial oxygen consumption. A supposedly higher oxygen demand, however, may be compensated by an increased coronary perfusion pressure during PST.

We did not evaluate cardiac output, thus we could not assess any potential contribution of the peripheral vascular resistance to the hemodynamic effect of PST. Because most patients reported subjective improvement of symptoms (dizziness, shortness of breath) during PST, this may be taken as some evidence for an improved cardiac output and clinical status.

We do not know whether PST will be effective and safe during spontaneous VT or for longer than 1 h. Although the mean cycle length of induced VTs was rather long, PST was also effective in eight patients with fast VTs. Still, a major challenge of the algorithm if applied during fast incessant VT will be to prevent VT detection breakdown because of necessary blanking periods.

In conclusion, PST during VT significantly augments systolic and mean arterial blood pressure. In patients with a high risk of VT, short-term PST up to 1 h did not accelerate VT or induce VF. The algorithm may help to hemodynamically stabilize patients with refractory or incessant VT.

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Letters to the Editor

Sensitivity and Specificity Versus Calcium Score in the St. Francis Heart Study

Regarding the recent paper in *JACC* concerning the St. Francis Heart Study by Arad et al. (1), I discovered an error in their Figure 2. The sensitivity goes to 100% as the calcium score goes to 0 while specificity goes to 0%. This is not a possible result. As the *x*-axis in Figure 2 (calcium score) goes to 0, it defines two separate groups of individuals, those with no (0) coronary calcium and those who have any coronary calcium. According to the figure, the positive predictive value of a coronary calcium score (CCS) above 0 would be equal to the event prevalence, and the negative predictive value of 0 calcium score would be indeterminate (0÷0).

The *y* intercept for sensitivity should go to 30% and specificity should go to 98% as the calcium score goes to 0. These latter numbers are in line with the natural curvature of the graph, which was artificially broken to get to 100% and 0% and also in line with the result of Kondos et al. (2) whose study reported 37-month follow-up data from 4,151 men and 1,484 women (mean age 52 years) without known coronary disease who were either self- or physician-referred for a screening electron-beam tomographic (EBT) examination. A total of 191 events were seen in the male population, but only 32 events were observed in the female population. Because so few events occurred in the

Table 1. Coronary Calcium Versus Events in Asymptomatic Men

	CCS > 0	CCS = 0
Event (191)	187	4
No event (3,960)	2,878	1,082

Thirty-seven-month data from Kondos et al. (2) on 4,151 middle-aged men undergoing screening electron-beam tomographic (EBT) heart examination. This matrix gives a sensitivity of 98% and a specifity of 27% for the presence of any coronary calcium and the prediction of events.
CCS = coronary calcium score.

female population, this group was excluded from this analysis. The 37-month matrix for the distribution of events and the presence of coronary calcium for asymptomatic men is detailed in Table 1.

Finally, assuming a sensitivity of 30%, a specificity of 98%, and a 10-year event risk of 10%, the positive predictive value of a non-0 calcium score would be 13.5%, and the negative predictive value of a 0 calcium score would be 99%, numbers in line with published data.

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REPLY

In reply to Dr. Boyar's letter, using data from Figure 2 in our study (1) and rounding off, we calculated a sensitivity of 0.94 and a specificity of 0.33 for all calcium scores ≥1. The values in our Figure 2, 0.91 and 0.39, respectively, differ slightly because the computer program that generated Figure 2 grouped all square roots less than the next whole number with the previous whole number. Thus, calcium scores of 1, 2, and 3, with square roots of 1.0, 1.4, and 1.7, respectively, were all plotted as corresponding to a value of 1.0 on the abscissa. Calcium scores of 4 to 8, with square roots of 2.0 to 2.8, were lumped together as 2, and so on. We apologize for any confusion created by Figure 2 in our report (1).

We are not aware of any standard that a threshold means > the threshold value rather than ≥ the threshold value. In the case of calcium scores of 0, we chose ≥0 because this makes a useful *reductio ad absurdum* point about the test, and because we believed the rest of the graph makes better sense if the threshold values were included.

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Left Atrial Remodeling in Competitive Athletes

Pelliccia et al. (1), as so frequently in the past, have added to our knowledge of "athlete's heart." One can only agree with their conclusions, but I believe their quantitative results might have been significantly different had they not restricted the search for prolonged P-wave duration to leads I, II, and V_1. Except for V_1, use of the limb leads only puts us in the anachronistic standards of the 1920s when there were only three leads (2). (Curiously, some current textbooks still rely on lead II.) We have shown several times that one needs to evaluate all 12 leads of the standard electrocardiogram (ECG) to get true P-wave durations. Indeed, if we had relied on lead II only, we would have recognized only just over one-half of the prolonged P waves despite utilizing calibrated magnifying graticules (2,3). Indeed, leads V_3 and V_4 gave substantially more prolonged P waves than did lead II. Another quantitative effect of the protocol may have occurred because the investigators used M-mode echocardiography when it is quite clear that, when assessing the left atrium volumetrically, two-dimensional echocardiography would have significantly been more sensitive (4).

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Competitive Athletes and Left Atrial Remodeling

In a recent issue of the *Journal*, Pelliccia et al. (1) assessed the prevalence and clinical significance of left atrial (LA) enlargement in competitive athletes. Enlarged LA size was common and present in 20% of examined athletes, and we agree with the investigators that the possible determinants of these changes remain incompletely resolved. They found that LA enlargement occurred in association with left ventricular (LV) enlargement and were largely dependent on the type of sport practiced, with cycling, rowing, and canoeing showing maximal impact. In their opinion these changes are due to the increased preload as they revealed normal resting LV diastolic filling and systolic function.

Rowing and cycling represent typical strength and endurance sports involving combined dynamic and static exercise of large