compared to a nine-month event rate of 32% in the corresponding patients within GISSOC.

Undoubtedly, as stent designs improve, late complications will become less frequent; thus, use of drug-eluting stents (3) in TOAT and GISSOC could have resulted in fewer restenoses and recurrences. The use of post-stent clopidogrel and other thienopyridines for only two weeks is validated and supported by Mishkel et al. (4) and Berger et al. (5).

Unfortunately, Doppler color flow mapping was not a protocol requirement in our study; therefore, quantitative assessment of resting mitral regurgitation is incomplete. Nevertheless, because all patients had single-vessel disease with left anterior descending artery occlusion, the posterior papillary muscle is likely to have been spared, making annular dilation the most probable mechanism of mitral incompetence. We agree that the increased exercise endurance observed in open-artery patients (despite increased left ventricular volumes) is paradoxical, and likely to be mediated through a placebo effect.

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Specificity of Noninvasive Pacemaker Stress Echocardiography in Diagnosis of Coronary Artery Disease

We read with great interest the recently published study by Picano et al. (1) in the October 2, 2002, issue of JACC. The investigators concluded that noninvasive pacemaker stress echocardiography is a diagnostically efficient method for patients with a permanent pacemaker and suspected or known coronary artery disease (CAD).

Nevertheless, some concerns arise based on our experience and from careful review of the published reports.

First, the main problem in detecting CAD in patients with permanent ventricular pacing is the low specificity of noninvasive techniques related to abnormalities of microvascular flow arising from chronic functional and/or structural abnormalities induced by abnormal ventricular excitation (2). We do not believe that the study by Picano et al. (1) could solve this problem; the studied group consisted of patients with a high prevalence of risk factors for CAD (>50%) and/or previous myocardial infarction (37%): it was, therefore, an excellent way to assess the sensitivity of the method, but not the specificity. In addition, 15 of 45 patients were in AAI pacing mode, and even for the remaining patients we do not know if there was partial or full ventricular excitation from the pacemaker electrode during daily life or during the stress protocol. We also do not know whether the studied group represents a total population of patients with a pacemaker, as the manner of the patients’ recruitment is unclear.

Second, the allegation that perfusion defects are more common than wall motion abnormalities during stress in patients with alterations of coronary flow reserve and normal epicardial coronary arteries conflicts with the findings of a study by Tse et al. (3), who observed wall motion abnormalities by radionuclide ventriculography to occur in the same proportion of patients with permanent ventricular stimulation and no significant CAD as did perfusion defects detected by dipyridamole thallium myocardial scintigraphy. Thus, the advantage of the stress echocardiogram remains in question.

Third, given the deterioration of myocardial perfusion over time that is observed in some studies (3,4), it is crucial to know the mean duration of pacing, especially in patients without wall motion abnormalities. Such data were not provided in the study by Picano et al. (1).

In conclusion, although no one has doubt about the diagnostic accuracy of the noninvasive pacemaker stress echocardiogram in patients with AAI pacing mode, or, more generally, about the sensitivity of the same method in detecting CAD, the specificity of the method remains in doubt. Based on data currently available, a specificity of 50% is the best we can expect from noninvasive techniques (2–5), at least in patients with permanent ventricular stimulation.

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REPLY

We thank Drs. Vardas and Skalidis for their thoughtful comments. Permanent ventricular pacing can induce an alteration in coronary flow reserve independently of epicardial coronary artery stenosis. Frequently, this may give rise to stress-induced perfusion abnormalities, which are "false positive" if we consider the anatomic, angiographic gold standard and "true positive" if we consider the functional gold standard of coronary flow reserve. This often happens in patients with microvascular disease: hypertensives, left ventricular hypertrophy, hypertrophic cardiomyopathies, diabetes, and wall motion changes (1). The unsurpassed spatial and temporal resolution of stress echocardiography, and the need to rely on systolic thickening to assess ischemia, explains the high specificity of the procedure, which was observed regardless of the (very variable) duration of pacing in patients with normal coronary arteries and ventricular pacing.

Finally, to judge a priori that "a specificity of 50% is the best we can expect from noninvasive techniques" on the basis of available data on stress perfusion scintigraphy and radionuclide ventriculography is perhaps a bit pessimistic. After all, however beautiful the strategy, one should occasionally look at the results. With pacemaker stress echocardiography, the diagnostic results are good, especially for specificity. Moreover, the strategy (the underlying rationale) is not so bad if we consider that: 1) not all diagnostic ischemic markers are the same—and regional perfusion is not synonymous with regional function (4); 2) not all techniques are the same—and wall motion by nuclear ventriculography is not synonymous with systolic thickening by two-dimensional echocardiography, especially with a ventricular paced rhythm (5); and 3) that our monolithic view of the classic ischemic cascade should be integrated with the awareness of the at least equally frequent alternative ischemic cascade—linked to coronary microvascular disease.

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