
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, syndrome X (1).

In diagnostic practice with stress imaging, not all patients follow the reassuring imaging paradigm proposed by the classical ischemic cascade. Indeed, the typical behavior of coronary microvascular disease during stress testing is the frequent induction of chest pain, ST-segment depression, and perfusion abnormalities without regional or global wall motion changes (1). The high specificity of pacemaker stress echocardiography in these patients is a particular case of the generally higher specificity of wall function markers in coronary microvascular disease (1). Left bundle branch block is pathophysiologically germane to chronic ventricular pacing in that a functional coronary microvascular disease can occur for an altered sequence of ventricular activation, inducing abnormally increased myocardial extravascular resistances in diastole (2). Vigna et al. (3) reported 95% specificity of stress echocardiography and—in the same patients—a specificity of 43% for sestamibi dipyridamole perfusion imaging if reversible and/or fixed perfusion defects were taken as positivity criteria in 37 patients with left bundle branch block.

Previous studies described wall motion abnormalities by radionuclide ventriculography to occur in patients with no significant coronary artery disease. This is also not surprising. Wall motion—as evaluated by stress nuclear ventriculography—can be very unreliable in patients with ventricular paced rhythm, and during stress echocardiography, primary reliance is placed on regional wall thickening—not motion. 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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doi:10.1016/S0735-1097(02)02977-7

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