Reply

Alves and Rose have commented on their experience using relatively low doses of amiodarone to control ventricular and supraventricular arrhythmias in patients with congestive heart failure. They first describe the use of amiodarone in patients with complex ventricular arrhythmias associated with a low left ventricular ejection fraction. Although not stated, it is implied that many of these patients have symptomatic ventricular arrhythmia, with control judged by clinical assessment and subsequently confirmed by electrocardiographic monitoring. In contrast, our patients were relatively asymptomatic from any ventricular arrhythmia associated with their heart failure and were given the drug for prophylactic reasons rather than to control current symptoms. Alves and Rose found a low incidence of serious side effects with the use of small doses of amiodarone. We would agree with this observation, although we found a substantial incidence of nuisance side effects such as chronic nausea. Our protocol did not allow for dosage adjustment, and in particular a 50% reduction in dosage of amiodarone if side effects occurred might have helped in our patients. The success rate of low dose amiodarone in the patients described by Alves and Rose is consistent with their patients having frequent ventricular premature complexes, or nonsustained ventricular tachycardia. However, it is the experience of most clinicians that life-threatening episodes of sustained ventricular tachycardia, particularly in association with poor left ventricular function, often require higher loading and maintenance doses of amiodarone for control, with the consequence of a higher incidence of side effects and a relatively high discontinuation rate during long-term follow-up.

Alves and Rose also describe the use of low dose amiodarone to control the ventricular response during atrial fibrillation in patients with congestive heart failure with or without a mitral valve prosthesis. These patients have previously had their ventricular rate poorly controlled by digoxin. Whether their congestive heart failure is related to the rapid ventricular response rate during atrial fibrillation, poor left ventricular function, or both, is not stated. Nonetheless a dramatic benefit from slowing the ventricular rate by amiodarone was apparent, and their experience is in accord with that of others (11), with emphasis on the use of low dose therapy. Interactions with digoxin if this medication is continued) and coumadin used for anticoagulation in patients with a mitral valve prosthesis, must be remembered.

Amiodarone has previously been shown to be effective in a wide spectrum of cardiac arrhythmias (11). Alves and Rose are to be applauded for emphasizing the use of the lowest dose possible at all times, particularly in the treatment of arrhythmias that are not immediately life threatening or for prophylaxis (13, in our study). This has been the practice in Europe and elsewhere since the introduction of the drug, but its initial use in the United States in high doses to control drug-resistant life-threatening episodes of sustained ventricular tachycardia was often associated with a plethora of side effects, and this experience may still be causing some clinicians to overlook the benefits of low dose therapy.

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Exercise ECG and Silent Restenosis

Lasman and colleagues (1) conclude, in part, that “exercise ECG [electrocardiographic] testing is not the technique of choice to detect silent restenosis after coronary angioplasty of single vessel coronary artery disease.” This is somewhat misleading, because all but 3 of 13 patients with a false positive treadmill test had a percent diameter stenosis of <50% and nearly one-third of the false negative tests were associated with a percent diameter stenosis of >40%. If the authors had concluded that exercise ECG testing is insensitive for the detection of coronary artery stenoses of approximately 50%, there would be little cause for comment. Additionally, the clinical significance of coronary artery restenosis of 50% to 55% is unclear. Certainly many interventionists would leave such a vessel alone in an asymptomatic patient. Finally, if they wished to test the ability of the exercise ECG to detect the presence or absence of restenosis, the treadmill test should have been performed within a day or two of the angiography—a condition that is not reported within their Methods section.

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Reference


Reply

It seems that Vine did not read our article very carefully. 11 In our study, bicycle instead of treadmill exercise was used. 2) Vine mentions 13 patients with false positive test results; in the text this is false negative test results. Conversely, nearly one-third of the false positive tests were associated with a percent diameter stenosis >=40%. We agree that a sharp division in “normal” (no restenosis) versus “abnormal” (restenosis) using the 50% diameter stenosis as a cutoff point has certain limitations associated with such a dichotomous classification. We also applied other thresholds such as minimal luminal diameter >=1.5 mm and minimal luminal cross-sectional area >=2 mm² (Table 1). The diagnostic value remained essentially unchanged and therefore the conclusion applies irrespective of the angiographic criteria used.

3) We fully agree that the clinical significance of borderline restenoses is unclear and that these obstructions perhaps should be left alone in asymptomatic patients. We never advocated repeat angioplasty of such lesions. On the contrary, we concluded our article with the question: “. . . will the long-term prognosis be