assumed that the "headaches" were nitrate-induced and proceeded to encourage patients to persevere through the initial phases of therapy. Furthermore, the side effects requiring crossover or termination were not judged to be "hypersensitivity" to nitrates because a documented history of nitrate intolerance was clearly an exclusion criterion of the study. We therefore felt that these subjects should be included for analysis.

Those patients who were titrated to maximal therapy may well represent a limitation of protocol design, and this probably should have been emphasized more clearly as one limitation of this type of study. We believed, however, that we employed dosage schedules that are commonly used in the community at large and that comparative data at these levels would be useful. A future investigation might well address the comparison of a calcium channel blocker with long-acting nitrates in an open label fashion where the dosage of nitrates can be confidently advanced to "the limits of tolerance" without fear of jeopardizing patient comfort or safety.

Finally, in response to the question of costs of respective drug therapies, there is little or no argument that use of any of the calcium channel blocking agents is more costly than conventional therapy with long-acting nitrates. This fact, however, has not dampened any of the enthusiasm for these agents nor has it precluded their widespread use. Perhaps not cost, but cost-effectiveness, should also be addressed in future clinical trials.

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Closed Chest Catheter Ablation of an Accessory Pathway in a Patient With Permanent Junctional Reciprocating Tachycardia

Gang et al. (1) describe successful catheter ablation of the accessory pathway in the permanent form of junctional reciprocating tachycardia. We disagree that theirs is "the first report of successful application of the procedure in this rhythm disorder," because a similar report has been published (2). In addition, we believe that some concepts concerning the initiating mechanisms of this arrhythmia, as reported in the article, should be modified. Our previous report (3), which is quoted by Gang et al., demonstrates that several mechanisms other than critical acceleration of the atrial rate can be responsible for the tachycardia initiation. Thus, we observed initiation of permanent junctional reciprocating tachycardia without any apparent antecedent event or after an atrial or ventricular premature contraction, as well as after a junctional escape beat.

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Reply

At the time of submission of our manuscript, we were not aware of a previous English language report of a successful catheter ablation of an accessory pathway in a patient with the "permanent" form of AV reciprocating tachycardia. Clearly, such a report had already been published in the Italian cardiologic literature. For this oversight, we offer our apology.

We certainly accept the comments of Monda et al. regarding mechanisms of initiation of tachycardia in patients with permanent junctional reciprocating tachycardia. Our introductory comments reviewed the common mode of initiation of the tachycardia (acceleration of the atrial rate prior to onset of the tachycardia), which constitutes a sufficient but by no means necessary condition for the onset of the tachycardia. This was invariably the way in which the tachycardia was initiated in our patient. Our comments do not *exclude* other modes of tachycardia initiation. Regardless of the way the tachycardia is described, we are gratified that both patients appear to have been cured of their troublesome tachycardias. In our case, the follow-up period has now extended to 17 months without a single recurrence of the tachycardia.

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