electromagnetic magnetic resonance fields may occur. Complete inhibition of pacemaker output in pacemaker-dependent patients and/or false triggering with tracking to the upper rate limit in patients with reduced left ventricular function may be detrimental.

Therefore, we cannot support the approach of Martin and co-workers—that is, to leave the pacemaker sensing function activated. In contrast, we recommend deactivating the sensing function in pacemaker-dependent patients by programming the pacemaker device to an asynchronous mode, to ensure continuous pacing, and to program the pacemaker device to a sensing-only mode (0X0) or subthreshold pacing in nonpacemaker-dependent patients to avoid MRI-related triggering.

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REPLY

We thank Dr. Sommer and colleagues for their interest in our work (1). We agree that the magnet mode of the pacemaker is not always maintained during magnetic resonance imaging (MRI). We have in fact observed this phenomenon during our experience. This phenomenon is related to the position of the pulse generator relative to the isocenter of the magnetic field. However, this does not alter our recommendations for imaging these patients.

Their statement regarding the possibility of an open reed switch leading to false inhibition and/or false triggering is valid. However, it was for this reason that pacemaker-dependent patients were excluded from our study. We were also concerned about thermogenic damage at the lead-tissue interface with subsequent loss of capture, which would also be detrimental in pacemaker-dependent patients. Finally, we did not alter pacemaker sensing, because in our experience over-sensing on the atrial channel occurs extremely infrequently.

Pacemaker-dependent patients can likely undergo MRI as long as sensing is disabled. However, the possibility of thermogenic damage and loss of capture cannot be overstated.

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RESTORE—From Deduction to Leap of Faith

In a recent study in *JACC* by the RESTORE Investigators, the researchers equate postoperative elevation in left ventricular ejection fraction (LVEF) (29.6% to 39.5%) and decrease in left ventricular end-systolic index (LVESI) (80 ml to 56 ml) to improved LV function. However, an increase in LVEF and a decrease in LVESI are geometric necessities of the operation, which involves a concentric shrinking of the infarcted anterior/septal myocardial area with a purse-string stitch, and closure of the small residual defect with an oval patch. They are predicated within the notion of the ventricular reduction itself, assuming the remaining sarcomeres continue their usual function. To say that ventricular function is improved, one would need data to show that stroke volume or, secondarily, pulmonary artery pressures or cardiac output improved. None of these data were provided; indeed, one would expect stroke volume and cardiac output not to change, and pulmonary artery pressures to fall based on Laplace's law.

The investigators make a good case in their discussion that they have helped their patients based on historical series involving individual components of the operation in subjects with dilated hearts (coronary artery bypass graft, ventricular aneurysmectomy, mitral repair). But the leap from what is essentially a deductive tautology (A = A) to their empiric finding of improved clinical symptoms requires hemodynamic data for inductive reasoning about cause and effect (1).

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REPLY

The RESTORE registry was accumulated to confirm the extensive experience of Dor and his colleagues, which dates back about 20 years. The primary reason for presenting these data was to

demonstrate that volume reduction and shape changes are valuable components in the treatment of congestive heart failure following infarction. Ejection fraction improved, volume was reduced, and clinical status improved by New York Heart Association (NYHA) functional classification status. Moreover, five-year survival was very gratifying, especially when compared to conventional therapy.

The effects of ventricular restoration have been studied and referenced in our report (1). These include improvement of systolic and diastolic function, confirmed by the centerline method and pressure-volume loop studies. The operation's primary physiological impact is on the remote noninfarcted myocardium and has been well described.

Our study did not report hemodynamics because we believe that little can be extrapolated from such data. It is well known that heart failure progresses independently of hemodynamic status and is directly related to ventricular size and shape changes (2,3). Indeed, patients with markedly dilated hearts and advanced heart failure often have normal cardiac output and pulmonary pressures at rest. Our reporting is, therefore, consistent with the majority of clinical trials of heart failure, few of which assert that acute resting hemodynamic changes are of functional or prognostic significance.

Hemodynamics, however, may be of value during exercise. Consequently, conversion of 67% in NYHA functional class III/IV category to 85% in class I/II seems an effective functional counterpart. In the meanwhile, the recently documented neurohormonal consequences of ventricular restoration are very pertinent to the reviewer's comments regarding "cause and effect" (4).

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Cardiac Magnetic Resonance-Directed Intervention in Non–ST-Segment Elevation Acute Coronary Syndrome

I read with great interest the study by Plein et al. (1) on the use of cardiac magnetic resonance (CMR) imaging to determine the presence of significant coronary stenosis in patients with non–ST-segment elevation acute coronary syndromes (NSTE-ACS). The investigators showed an impressive sensitivity (96%) and specificity (83%) to predict significant coronary stenosis using combined myocardial function, perfusion, viability (employing late enhancement), and coronary anatomy. They also pointed out that the diagnostic yield of combining only perfusion, wall motion, and late enhancement was similar without the addition of CMR angiography, representing a significant reduction in scanning time.

The advent of evidence-based international guidelines that all patients with NSTE-ACS should undergo early (<72 h) revascularization (2,3) has created both an enormous burden on worldwide health care systems and long inpatient waiting times for intervention (4). A more precise risk-stratification of those patients with a diagnosis of NSTE-myocardial infarction, such as that advocated by the Plein et al. (1) study, would prevent exposing patients to unnecessary risk and markedly alleviate the extra burden on health care systems. This is particularly relevant to this study as only 53% of subjects had a positive troponin level and thereby fulfilled the definition of NSTE-ACS and evidence-based criteria for early intervention.

The difficulty of accurate risk-stratification highlights the importance of the study by Plein et al. (1), but the real strength of CMR lies in not only detecting coronary artery stenoses, but in directing interventional therapy. Although 56 of the 68 patients studied were found to have coronary artery disease, a more profound question is whether the stenotic vessel supplied a territory that was viable or ischemic, as these are the stenoses that, when treated, result in beneficial ventricular remodeling, prognosis, or reduction in symptoms (5). This information is inherent in the CMR technique and therefore readily already available to the investigators. Further analysis of the data would determine how appropriate the intervention was, and allow the potential cost benefit of a single CMR scan to be calculated.

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