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 sarcomeses 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...