EDITORIAL COMMENT

Myocardial Viability Assessment by Contrast-Enhanced Magnetic Resonance Imaging*

Joao A. C. Lima, MD
Baltimore, Maryland

Recent progress in the diagnosis and treatment of coronary artery disease has intensified the need to differentiate viable from nonviable myocardium with accuracy and high spatial resolution. Contrast-enhanced magnetic resonance imaging (MRI) had been investigated as a potential technique to assess myocardial viability for several years (1), before the development of gradient echo-imaging techniques provided the ability to temporally distinguish perfusion abnormalities created by microvascular obstruction from myocardial hyperenhancement secondary to myocardial necrosis (2,3). The prognostic value of contrast-enhanced MRI in patients with acute myocardial infarction (AMI) is well established (4). However, recent work on the specific utilization of this technique for the purpose of predicting local functional recovery after AMI (5,6) has further expanded its potential utilization in state-of-the-art cardiology practice. The work by Beek et al. (7) in this issue of the Journal represents an important addition to our knowledge in this field.

See page 895

Beek et al. (7) compared MRI studies performed in the acute post-infarction phase with similar studies obtained 2 to 4 months later in 30 patients. Of 500 dysfunctional segments, 273 (55%) improved at follow-up, with little or no difference in rate of improvement between segments with no necrosis, compared to those with necrosis involving up to 25% of the left ventricular (LV) wall thickness. As involvement extended across the LV wall, the likelihood of functional recovery diminished, underscoring the power of contrast-enhanced MRI to discern viable from nonviable tissue across the LV wall. The development of improved techniques to amplify the differences between hyperenhanced necrotic tissue and normal myocardium (10) has also contributed to the growing clinical incorporation of these techniques by generating images that are obvious to the naked eye and preclude offline analyses to determine the exact boundaries between normal and abnormal tissue (5–7,9).

Beek et al. (7) demonstrate the relationship between functional recovery and transmural necrosis using visual assessment of systolic endocardial motion and wall thickening. Previous studies used similar methods to document similar relationships both in the acute (5) and chronic (11) MI settings. Beek at al. (7) confirmed the low interobserver variability associated with these methods, particularly when performed in a before and after setting (late vs. early post-MI in this case) by trained observers. Ideally, however, myocardial deformation during systole and diastole would be objectively quantified to allow for less biased comparisons. Unfortunately, planar systolic wall thickening measurements obtained by endocardial and epicardial border delineation are also quite variable because of technical and geometric factors intrinsic to LV architecture and function (12,13). In addition, systolic wall thickening measurements obtained from border contours do not contain information on endocardial motion and thus may not be much more accurate to assess segmental function than visual assessment performed by well-trained observers. On the other hand, noninvasive MRI methods to measure myocardial deformation in multiple orientations (myocardial strains) have been available for more than a decade (6,13–18), and novel ways to rapidly acquire and analyze these complex imaging data sets have been recently developed (6,17,18). Similar methods using ultrasound to measure myocardial strains in different directions have also been developed (19), creating the possibility of a new paradigm for noninvasive objective functional assessment in the not-so-distant future. It is very important to realize, however, that all functional indices based on myocardial deformation (including direct visual

*Editorials published in the Journal of the American College of Cardiology reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

From the Division of Cardiology, Johns Hopkins Hospital, Baltimore, Maryland.
assessment, wall thickening, and strain measurements) are load-dependent, and in particular, afterload-dependent. This load or local stress dependence is even more complex in the regionally ischemic or infarcted LV, where the mechanical behavior of infarcted subendocardial myocardium is in large part dictated by that of the preserved subepicardial layers and vice versa (17,20,21). Similarly, the function of preserved myocardium adjacent to the infarct border is heavily dependent on the mechanical behavior of remote noninfarcted regions (22–24). Load interdependence at both the global and regional levels is an important factor to keep in mind when planning and interpreting viability studies designed to investigate predictors of functional recovery.

The main mechanism underlying the functional improvement of viable segments after AMI is the resolution of myocardial stunning in regions that underwent temporary ischemia but survived owing to either reopening of the infarct-related artery or reperfusion through collaterals (25). Myocardial stunning is believed to result from calcium overload at the time of reperfusion with secondary damage to contractile proteins of the troponin complex (26). Several days or sometimes weeks are required to repair the damaged contractile apparatus, accounting for the typical time course of functional recovery associated with this condition (5–7,25,26). However, the mechanistic interpretation of findings from studies performed in patients with chronic ischemic heart disease (11,27) is much less obvious. The pathophysiology of chronically dysfunctional myocardium has been the subject of intense debate in the past two decades (27–29), and the mechanisms of functional improvement after revascularization in that setting remain unresolved (27–29). Moreover, in the case of chronic myocardial dysfunction, the issues related to local functional interdependence are of even greater importance given that patient cohorts tend to be less uniform and medical management less standardized than in the acute setting. Further investigation on the fundamental mechanisms underlying functional recovery of “hibernating” myocardium will likely provide fundamental insights into the pathophysiology of heart failure due to chronic ischemic myocardial damage.

Today, some of the greatest limitations to the routine use of contrast-enhanced MRI to assess viability in patients with ischemic heart disease relate to economic and organizational factors such as cost, easy access to equipment, and availability of personnel trained to perform and interpret MRI studies. This is unfortunate because from both the clinical and scientific perspectives, greater utilization of this technology by cardiologists and other cardiovascular scientists would be highly desirable. From a clinical perspective, the available body of literature indicates that contrast-enhanced MRI can be very useful in patients with AMI and also in those with chronic LV dysfunction due to ischemic heart disease. From a scientific standpoint several directions require further exploration in the quest for a more complete understanding of myocardial ischemic injury. Among potential directions for further research, the link between viability and malignant arrhythmias ranks high given the persistently elevated incidence of sudden death among patients with ischemic heart disease (30). In this regard, although extremely useful clinically, currently used techniques to assess myocardial viability may obscure the presence of “gray areas” corresponding to necrotic tissue intermingled with normal myocardium. The latter limitation may be of particular importance to investigational efforts directed at further clarifying the role of border-zone and/or non-transmural myocardial injury in the genesis of ventricular arrhythmias (31). Future lines of investigation should also include: 1) further research on microvessel damage and its role in the ultimate viability of segments injured by ischemia and reperfusion (32); 2) the development of ionic distribution maps including sodium (32–35) and eventually potassium (34) MRI to further probe cellular responses to ischemia and reperfusion; and 3) the potential to superimpose structural myocardial alterations to detailed measurements of function (6,16–18) and metabolism (32–35). The results accomplished so far, as well as the possible pathways of discovery that lay ahead, place MRI and spectroscopy at the forefront of techniques likely to produce future breakthrough knowledge on myocardial viability. The paper by Beek et al. (7) is an important step in the right direction.

Reprint requests and correspondence: Dr. Joao A. C. Lima, Johns Hopkins Hospital, Division of Cardiology, Blalock 569, 600 North Wolfe Street, Baltimore, Maryland 21287. E-mail: jilima@jhmi.edu.

REFERENCES


