

Prediction of Functional Recovery (Viability and Perfusion)
Christopher M. Kramer MD
University of Virginia Health System

There are 2 settings in cardiovascular medicine where prediction of functional recovery is important. The first is in the setting of acute myocardial infarction (MI) in which recovery of infarcted myocardium and stunned myocardium (transiently ischemic but not infarcted) is a significant clinical concern. The second clinical scenario is in the patient with chronic ischemic heart disease and reduced left ventricular (LV) function where prediction of recovery of function in dysfunctional myocardium may determine whether or not the patient is revascularized either percutaneously or with coronary artery bypass surgery. There may be other reasons to revascularize patients, but functional recovery is a tried and true endpoint. Cardiac magnetic resonance (CMR) has made important contributions to our understanding of functional recovery in acute MI and chronic ischemic heart disease through the use of 3 distinct methodologies. One is first-pass contrast enhanced perfusion to determine the status of the microvasculature. The second is late gadolinium enhancement to determine whether myocardium is scarred or not. Lastly, low dose dobutamine cine CMR as a means to interrogate contractile reserve has been used to predict recovery.

In acute MI, first-pass contrast enhanced perfusion has demonstrated that those infarcted segments with first-pass hypoperfusion demonstrate less functional recovery than those with normal perfusion(1). Late gadolinium enhancement was initially validated in a canine model of acute MI as a closely correlated marker of necrosis(2). Subsequent studies in acute MI demonstrated an inverse relationship between the transmural extent of necrosis and functional recovery(3). The presence of persistent low signal at the core of an infarcted segment is a marker of microvascular obstruction and is a predictor of complete lack of functional recovery in that segment(1;4). Response to low doses of dobutamine can also predict recovery of dysfunctional myocardium after acute MI, although the response within subendocardial segments is quite complex(5).

In chronic ischemic heart disease, first pass contrast-enhanced perfusion has a less reliable relationship with functional recovery. In this setting, like in acute MI, there is a well-described inverse relationship between functional recovery and the transmural extent of late enhancement or scar(6). However, the anatomy of scar alone may not be the best predictor of functional recovery. CMR is a powerful technique that can combine anatomic imaging with physiology (response to low dose dobutamine). In 2 studies, the response to low dose dobutamine in a chronically dysfunctional myocardium has been more predictive of functional recovery with revascularization than late gadolinium enhancement alone(7;8). In summary, CMR can bring to bear multiple techniques for optimal prediction of functional recovery in both acute MI and chronic ischemic heart disease.

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