The post–myocardial infarction scarred ventricle and congestive heart failure: The preeminence of magnetic resonance imaging for preoperative, intraoperative, and postoperative assessment

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An operative procedure can be precise and perfect only if it is based upon the surgeon's profound knowledge of anatomy, his understanding of the alteration of this normal anatomy by the pathology with which he is dealing and his ability to use the anatomic information in organizing and affecting his surgical procedure.

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Magnetic resonance imaging (MRI), with its ability to precisely define myocardial anatomy, echoes the teachings of one of cardiac surgery's most distinguished pioneers. Since the 1990s, several studies have demonstrated the preeminence of cardiac magnetic resonance (CMR) for analysis of wall motion abnormalities after myocardial infarction (MI).[2,3] Both cine-MRI to determine precise scar location and the late enhancement technique to assess the transmural extension of infarcted myocardium illustrate the utility of CMR. CMR is the preeminent method of assessing left ventricular remodeling and aneurysmal progression after MI. Moreover, thrombus formation; the site, depth, and extension of the scarred myocardial wall; precise localization of asynergic areas; disorganization of the mitral apparatus; and estimation of left ventricular volume and performance are all circumstances in which CMR has proved more accurate than other techniques.[4] In our opinion, a complete MRI evaluation of cardiac function is paramount before deciding how to appropriately treat patients after MI.

The core problem with ischemic congestive heart failure (CHF) is the undue demand put on the residual viable left ventricular myocardium. As noted by Klein and colleagues[5] more than 40 years ago, ''When the myocardium in the aneurismal area functions improperly, or has been replaced by fibrosis in 20%–25% of the surface area of the left ventricle, the extent of shortening required of the remaining functioning heart begins to exceed physiological limits.'' This was later confirmed by McKay and associates,[6] who showed that left ventricular dilatation occurs when the asynergic (dyskinetic or akinetic) left ventricular circumference exceeds 40%.

MRI can be used as a complementary tool in the surgical care of post-MI ventricular dysfunction and the ischemic CHF that may ensue. What follows is concise summary of MRI's applicability to surgical care to treat this complex disease process.

POSTISCHEMIC MYOCARDIAL ASSESSMENT

Immediately after MI, MRI shows no reflow to the endocardium.[7,8] This can be helpful, because it aids in predicting the degree of benefit to be expected from successful recanalization. Indeed, in patients seen many hours after coronary occlusion, demonstration by MRI of total lack of transmural reflow is highly suggestive that there is little in the way of viable tissue and that further intervention to reperfuse the myocardium is unlikely to improve on the residual impairment that remains.[9] In addition, 5 to 15 days after MI, CMR again becomes useful in the decision-making process, providing additional information about the scarred ventricle and its potential consequences.

To calculate reliable values of end-systolic volume index (ESVI), end-diastolic volume index (EDVI), and left ventricular ejection fraction (LVEF) true volumes of multiple parallel disks are added together. An increase of 20% in EDVI is considered to be the definition of left ventricular remodelling,[10] and an ESVI greater than 60 mL/m^2 is a criterion for chronic advanced heart failure.[11]

The site, depth, and extension of the scarred wall may be evaluated completely with 4 MRI projections. The 2-chamber view is used to assess the anterior and posterior walls of the left ventricle, the 4-chamber view allows assessment of the septum and lateral wall, the short-axis view enables a staged analysis of the septum and papillary muscles, and the projection that illustrates the left ventricular outflow tract (accordingly entitled the left ventricular outflow tract view) also provides a detailed analysis of the mitral apparatus (Figure 1).

The silhouette of an asynergic left ventricle cannot be correctly characterized by a simple plane angiogram in the right oblique projection, because this view cannot ''see'' the septum and lateral wall. The classification of left ventricular aneurysms into four types by means of this technique is thus obsolete. The type of asynergy is often correlated with the depth of the scar, being dyskinesia when the scar is transmural and akinesia when the scar is subendocardial (Figure 2).[12]

The extension of wall motion abnormality was for decades analyzed by the center-line method, which is still

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Received for publication April 22, 2008; accepted for publication July 31, 2008.
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J Thorac Cardiovasc Surg 2008;136:1405-12
0022-5223/$34.00

doi:10.1016/j.jtcvs.2008.07.044

Abbreviations and Acronyms

CHF = congestive heart failure
CMR = cardiac magnetic resonance
DVBS = diastolic volume balloon sizing
EDVI = end-diastolic volume index
ESVI = end-systolic volume index
EVCPP = endoventricular circular patch plasty
IFV = ischemic failing ventricle
LVEF = left ventricular ejection fraction
LVR = left ventricular reconstruction
MI = myocardial infarction
MRI = magnetic resonance imaging
SI = sphericity index

used, but limited to the right oblique projection.[13] The gadolinium injected during MRI is especially fixed on dead tissue, outlining the limit of irreversibly scarred myocardium.[14] The ratio between scar length (by late gadolinium enhancement [LGE]) and length of the left ventricular circumference gives a result similar to the center-line method, but this result can be analyzed with the 4 projections, allowing a more precise estimation of the percentage of asynergic wall. LGE is helpful in verifying the percentage of remaining contractile myocardium on the anterior, lateral, and septal walls, allowing the surgeon to learn whether the endoventricular reconstruction is feasible and how it can be carried out inside the ventricle.

The analysis of mitral valve anatomy and dysfunction can be fully assessed by CMR. The presence of mitral regurgitation, coaptation of leaflets, diameter of the annulus, distance between the papillary muscles (analyzed at the level of the tips), and the distance between basal interpapillary muscles are all accurately evaluated with CMR. In the case of left ventricular dilatation, the tips of the papillary muscles are often increased. Conversely, for both anteroseptoapical and posterior aneurysms, even when extremely large, the interpapillary muscles are never affected. Only posterolateral aneurysms related to occlusion of the obtuse marginal (less than 5% of cases) result in distention of the interpapillary muscles. In practice, however, infarction from obtuse marginal occlusion is more often complicated by limited free-wall rupture or papillary muscle rupture than is a lateral wall aneurysm.

This absence of involvement of the papillary muscle in anteroseptoapical aneurysms and most posterior or posteroseptal aneurysms is explained by pathoanatomy.[15] The papillary muscles are not inserted onto the septum. Instead, the anterior papillary muscle is inserted on the lateral wall and the posterior papillary muscle onto the posterolateral wall (Figure 3). As illustrated by McAlpine[16] and well known to anatomic pathologists, the orifice of an anteroseptal aneurysm is septoapical, because the septum is more affected than the anterior wall during an obstruction of the left anterior descending artery. The orifice of the posterior aneurysm is also posteroseptal. The basal papillary muscle can be remote from the septum, but the interpapillary muscle remains stable (Figure 4). Consequently, the diagram often used to explain mitral insufficiency complicating left ventricular remodeling by distention between septal and lateral papillary muscles does not correspond to normal anatomy or to pathoanatomy. In reality, for an anteroseptoapical aneurysm,

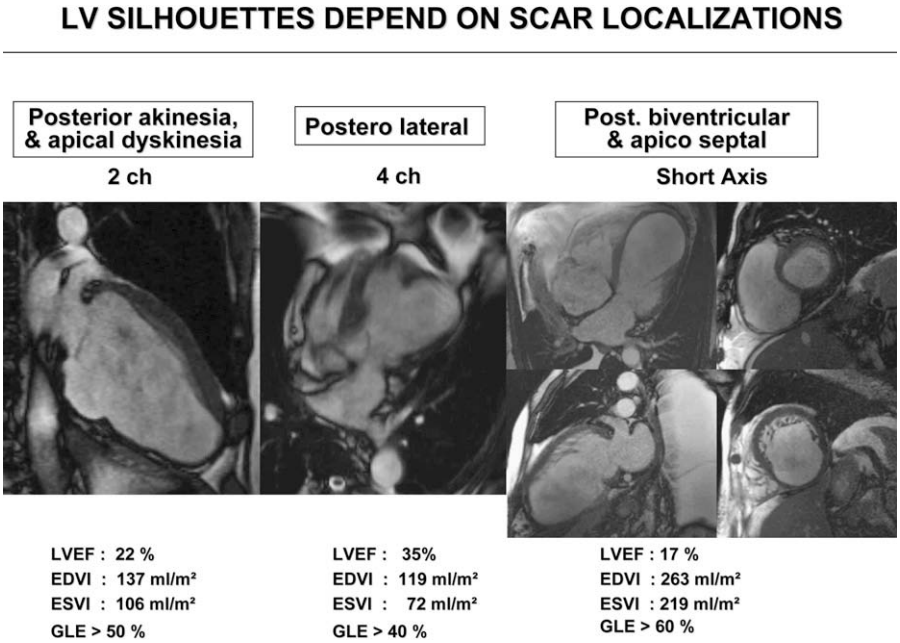

FIGURE 1. Analysis of 2-chamber and short-axes views showing exactly localization of wall motion abnormality.