

# Delayed Enhancement MR Imaging: Utility in Myocardial Assessment<sup>1</sup>

## CME FEATURE

See accompanying test at [http://www.rsna.org/education/lrg\\_cme.html](http://www.rsna.org/education/lrg_cme.html)

## LEARNING OBJECTIVES FOR TEST 3

After reading this article and taking the test, the reader will be able to:

- Describe the technique of delayed enhancement myocardial MR imaging.
- Discuss the use of delayed enhancement MR imaging in ischemic myocardial disease.
- Identify the features of nonischemic myocardial diseases at delayed enhancement MR imaging.

## TEACHING POINTS

See last page

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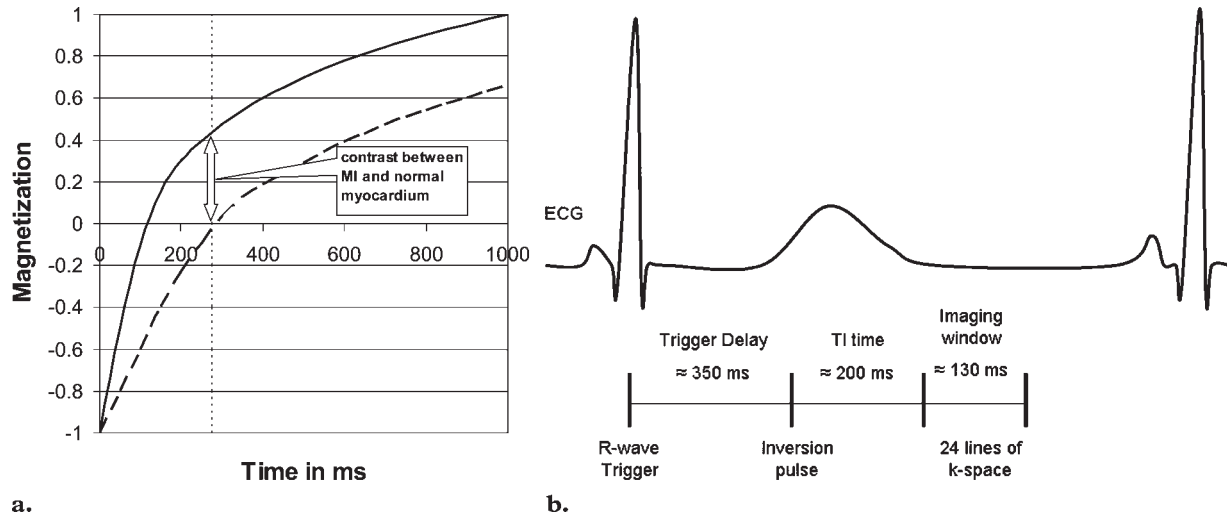
Use of magnetic resonance (MR) imaging for diagnosis of cardiac diseases and treatment monitoring is expanding. Delayed myocardial enhancement MR imaging is performed after administration of paramagnetic contrast agents and is used for a growing number of clinical applications. This technique was developed primarily for characterization of myocardial scarring after myocardial infarction. On delayed enhancement MR images, scarring or fibrosis appears as an area of high signal intensity that is typically subendocardial or transmural in a coronary artery distribution. However, delayed myocardial enhancement is not specific for myocardial infarction and can occur in a variety of other disorders, such as inflammatory or infectious diseases of the myocardium, cardiomyopathy, cardiac neoplasms, and congenital or genetic cardiac conditions, as well as after cardiac interventions. In nonischemic myocardial disease, the delayed enhancement usually does not occur in a coronary artery distribution and is often midwall rather than subendocardial or transmural. Therefore, the patient's clinical history is critical in the evaluation of delayed myocardial enhancement MR images.

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**Abbreviation:** ARVD = arrhythmogenic right ventricular dysplasia

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**a.** **Figure 1.** (a) Relaxation of myocardium with delayed enhancement and normal myocardium. Optimal image contrast between myocardial areas of delayed enhancement (solid line) and normal myocardium (dashed line) is achieved by imaging at the null point of normal myocardium and adjusting the inversion time for each patient (3). In this example, the inversion time (dotted line) is 275 msec. *MI* = myocardial infarction. (b) Diagram of a delayed myocardial contrast-enhanced MR imaging sequence. The image acquisition starts after or around the center of the individually selected inversion time (*TI*), depending on the vendor. *ECG* = electrocardiogram.

## Introduction

Delayed enhancement cardiac magnetic resonance (MR) imaging for myocardial infarction was first described more than 10 years ago (1). Since then, owing to recognition of the clinical importance, extensive experimental studies, higher-field-strength magnets, and high-performance gradient hardware have led to improved pulse sequences within the past few years. Currently, delayed myocardial enhancement MR imaging is rapidly becoming the standard of reference for evaluation of myocardial scar due to infarction. After administration of gadolinium contrast material, the technique involves an inversion-recovery preparatory pulse to null normal myocardium, followed by a segmented k-space gradient-echo acquisition. Retention of contrast material results in T1 shortening and thus increased signal intensity on T1-weighted images relative to that of the normal myocardium.

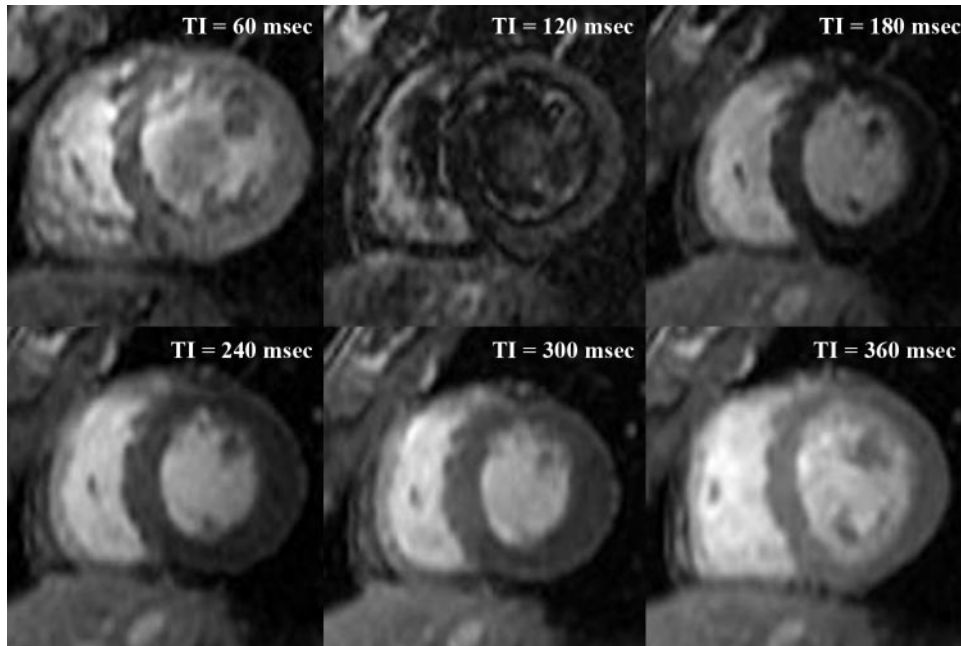
Differential contrast enhancement of myocardial tissue is seen in many pathophysiologic scenarios: retention of contrast material by fibrous tissue in chronic myocardial infarction, increased

volume of contrast material distribution in acute myocardial infarction and inflammatory conditions, and differential contrast enhancement in primary or secondary cardiac malignancies due to tumor neovasculature and tumor necrosis. **It is important to recognize that delayed myocardial enhancement is not specific for myocardial infarction and can occur in a variety of other disorders, such as inflammatory or infectious diseases of the myocardium, cardiomyopathy, cardiac neoplasms, and congenital or genetic cardiac pathologic conditions.** In this article, we present the technique for delayed enhancement cardiac MR imaging and a range of clinical applications, including ischemic and nonischemic diseases of the myocardium.

## Technique

The technique for delayed enhancement MR imaging involves intravenous infusion of gadolinium chelate contrast material (0.1–0.2 mmol/kg) followed 10–30 minutes later by a cardiac-gated T1-weighted pulse sequence. Imaging too early (eg, less than 5 minutes after the initial contrast material injection) results in reduced contrast difference between infarcted and normal myocardium because insufficient contrast material has

Teaching  
Point



**Figure 2.** The inversion time ( $TI$ ) is optimized for each patient by using low-resolution breath-hold images. The optimal inversion time (180 msec in this example) is visually selected to maximize both signal-to-noise ratio and contrast-to-noise ratio of the left ventricle.

been washed out of the normal myocardium. This can lead to overestimation of the infarcted region (2). However, imaging too late (eg, more than 30 minutes after initial contrast material injection) may result in excessive washout of the contrast agent from the infarcted tissue and poor signal-to-noise ratio.

The typical pulse sequence for myocardial delayed enhancement is a segmented inversion-recovery-prepared fast gradient-echo sequence. Imaging occurs over nine to 12 heartbeats, in a breath hold. In patients who have difficulty holding their breath, navigator-assisted free-breathing techniques can be used without breath holding.

An inversion-recovery sequence is used to optimize the contrast between gadolinium-retaining infarcted or scar tissue and healthy or viable myocardium. The inversion-recovery pulse is used to null the normal myocardium and optimizes the contrast difference compared with the gadolinium-retaining infarcted tissue (Fig 1a) (4). The optimal inversion time depends on contrast material clearance from the normal myocardium.

The contrast material clearance from the normal myocardium is determined by several factors: the washout rate of contrast material from the normal myocardium, overall cardiac function, renal function, and possibly the administered dose of contrast material. Therefore, the inversion time is optimized for each patient just before the image acquisition by using low-resolution breath-hold images (Fig 2). The optimal inversion time is visually selected to maximize both signal-to-noise ratio and contrast-to-noise ratio of the left ventricle. Typical values for inversion time are 175–250 msec.

Following the R wave of the electrocardiogram, a delay period (trigger delay) is used to ensure that the image acquisition occurs in diastole to minimize cardiac motion. The magnetization of the heart is then prepared by a nonselective  $180^\circ$  inversion pulse. Then, the center of acquisition of the segmented k-space lines occurs at the visually selected inversion time (Fig 1b). Typically, short-axis and horizontal and vertical long-axis views are obtained. A delayed enhancement

**Teaching Point**

**Table 1**  
**Parameters for Delayed Enhancement MR Imaging**

Parameter	Description or Value*
Type of acquisition	GRE segmented k-space acquisition, ECG gated
Timing of acquisition	10–20 min after administration of contrast material
Contrast material	0.2 mmol/kg <sup>†</sup> dose of IV gadolinium
Repetition time (msec)	5.4
Echo time (msec)	3.0
Flip angle (degrees)	20
Segmentation factor	24
Trigger delay (msec)	350
Inversion time (msec)	175–250 (adjusted to suppress normal myocardium)
Matrix	256 × 160–192
Number of signals acquired	2
Section thickness (mm)	8
Section spacing (mm)	0
Imaging planes	8 short-axis and horizontal and vertical long-axis images

\*ECG = electrocardiographically, GRE = gradient-echo, IV = intravenous.

<sup>†</sup>Note the administration of a “double dose” (0.2 mmol/kg) of contrast material, which results in improved contrast-to-noise ratio.

MR imaging protocol from our institution is shown in Table 1.

### Delayed Enhancement MR Imaging in Ischemic Heart Disease

The term *viable myocardium* refers to myocardium that may recover function following coronary revascularization. Hibernating myocardium is viable myocardium with reduced blood flow and will recover function, but scar or fibrosis in an area of infarction will not recover function. After myocardial infarction, accurate assessment of myocardial viability is crucial for optimal clinical decision making.

To identify viable versus nonviable myocardium, delayed enhancement MR imaging is used. Scar or fibrosis is depicted as an area of high signal intensity on delayed enhancement MR images that is typically subendocardial or transmural in a coronary artery distribution.

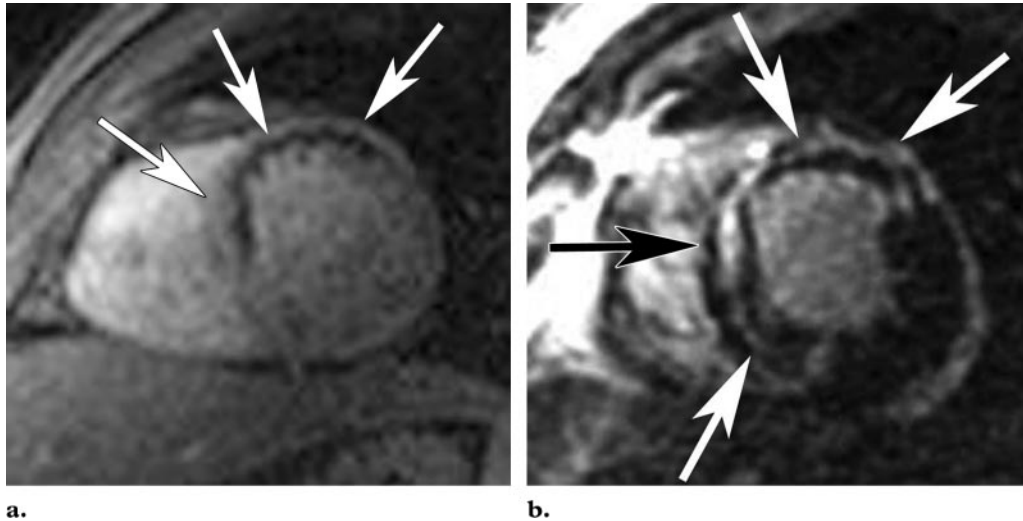
**Teaching Point**

The concept that “bright is dead” is that myocardium with high signal intensity on MR images corresponds to retention of gadolinium contrast material in non-contracting scar or fibrotic tissue. The basis for this relates to both animal models following myocardial infarction (5) and human studies (6) that show lack of contraction of the enhanced myocardium following coronary revascularization. In general, myocardial scar or fibrosis involving more than 50% of myocardial wall thickness at cardiac MR imaging is unlikely to recover contractile function following coronary revascularization (6).

**Teaching Point**

### Acute versus Chronic Myocardial Infarction

In acute myocardial infarction, there is loss of integrity of myocyte cellular membranes. In addition, large myocardial infarction may be associated with capillary occlusion and plugging with cellular debris, termed *microvascular obstruction*.



**Figure 3.** Acute myocardial infarction in a 53-year-old man with chest pain. **(a)** Short-axis first-pass perfusion MR image shows lack of subendocardial enhancement in the territory of the left anterior descending artery (arrows) due to microvascular obstruction. **(b)** Short-axis delayed MR image shows nearly transmural delayed myocardial enhancement in the territory of the left anterior descending artery (arrows). In addition, there is persistence of the region of microvascular obstruction.

After acute myocardial infarction, first-pass MR perfusion images obtained immediately after bolus administration of the gadolinium contrast agent may demonstrate lack of enhancement at the region of microvascular obstruction, which sometimes persists on delayed enhancement MR images (Fig 3). This is typically at the “core” of the area of myocardial necrosis. If present, lack of enhancement of this infarct core, or microvascular obstruction, is related to poor patient prognosis as documented by Wu et al (7), with increased incidence of congestive heart failure, recurrent infarction, and chest pain.

Delayed enhancement MR imaging in acute myocardial infarction typically shows diffuse enhancement of the entire infarct area—the zone of myocardial necrosis. The zone of myocardial necrosis may be either subendocardial or transmural in a distribution reflecting one or more of the affected coronary arteries. The mechanism of enhancement is likely related to increased volume of distribution of the gadolinium contrast agent due

to disruption of cell membranes associated with myocardial necrosis. Areas of stunned myocardium, which have decreased function but have intact cell membranes, do not show delayed enhancement on MR images. Cine MR imaging, usually performed in conjunction with the delayed enhancement MR imaging, shows akinesis in areas of myocardial stunning.

Distinguishing acute versus chronic infarction may be difficult with these MR imaging methods. Both acute and chronic infarctions show high signal intensity on delayed enhancement MR images (Table 2). In general, if the infarction is transmural and chronic, the myocardium will show thinning (Fig 4), whereas the myocardium is normal thickness in acute infarction (Fig 5). However, chronic subendocardial infarction will also frequently have normal thickness of the myocardial wall.

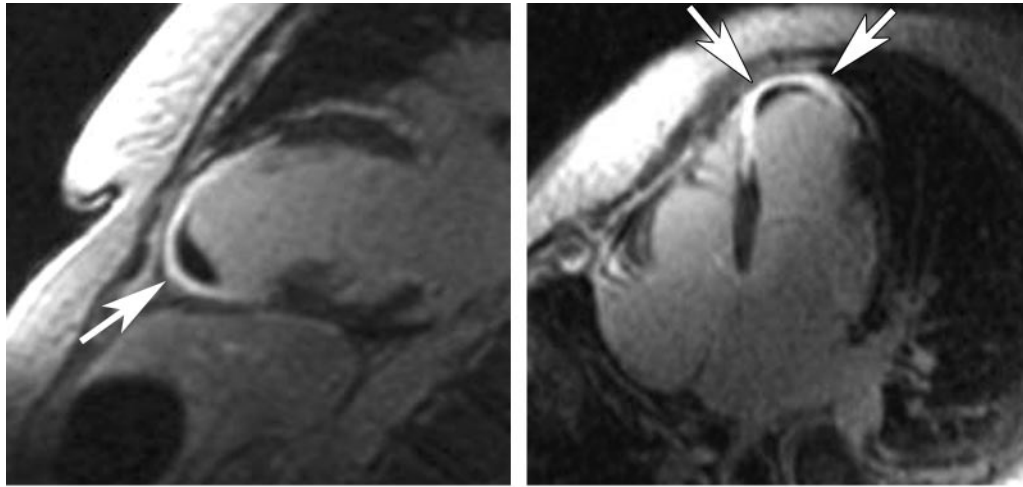
**Table 2**  
**Overview of Pathologic Features, MR Imaging Findings, and Prognosis in Acute and Chronic Myocardial Infarction**

Myocardial Disease	Pathologic Features	Findings at Resting First-Pass Perfusion MR Imaging	Findings at Delayed MR Imaging	Significance of Findings
Acute infarction with reperfusion	Myocardial necrosis	Normal appearance	High signal intensity in the affected coronary artery segments due to delayed washout of contrast material	Increased transmural of delayed enhancement is associated with decreased likelihood of recovery of function
Acute infarction without reperfusion	Myocardial necrosis, microvascular obstruction	Low signal intensity (usually in the core of the infarct)	High signal intensity in the affected coronary artery segments due to delayed washout of contrast material	Microvascular obstruction is associated with increased complications (eg, heart failure, recurrent infarction)
Stunned myocardium*	Normal myocardial perfusion but decreased function	Normal appearance	Normal appearance	Good prognosis for recovery of function of these myocardial segments
Chronic infarction	Fibrous tissue replacing the myocardium	Normal to slightly delayed first-pass perfusion	High signal intensity in the affected coronary artery segments due to delayed washout of contrast material	Function is unlikely to be recovered in myocardial segments with enhancement of >50% of wall thickness
Hibernating myocardium†	Noninfarcted myocardium, decreased function, decreased blood flow	Normal appearance	Normal appearance	Good prognosis for recovery of function of these myocardial segments after revascularization

\*Associated with acute infarction.

†Associated with high-grade chronic coronary artery disease.

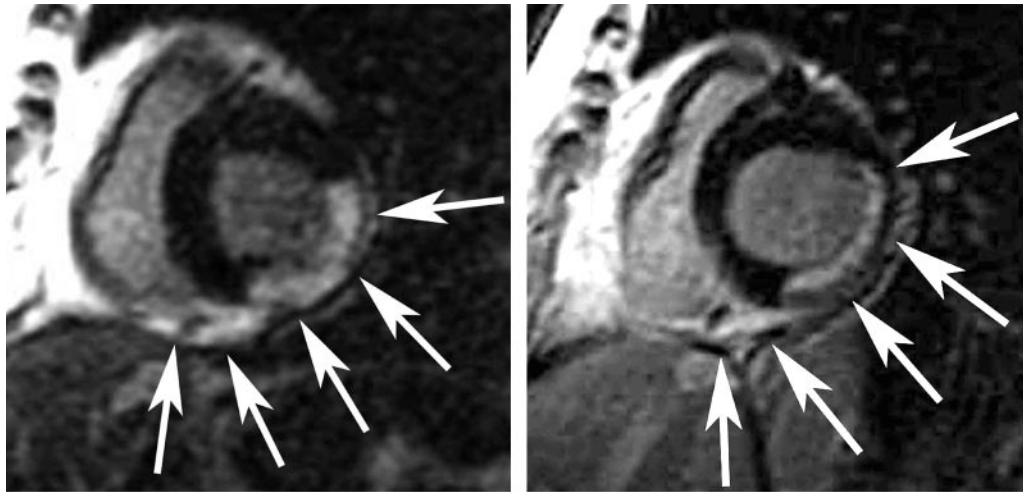




a.

b.

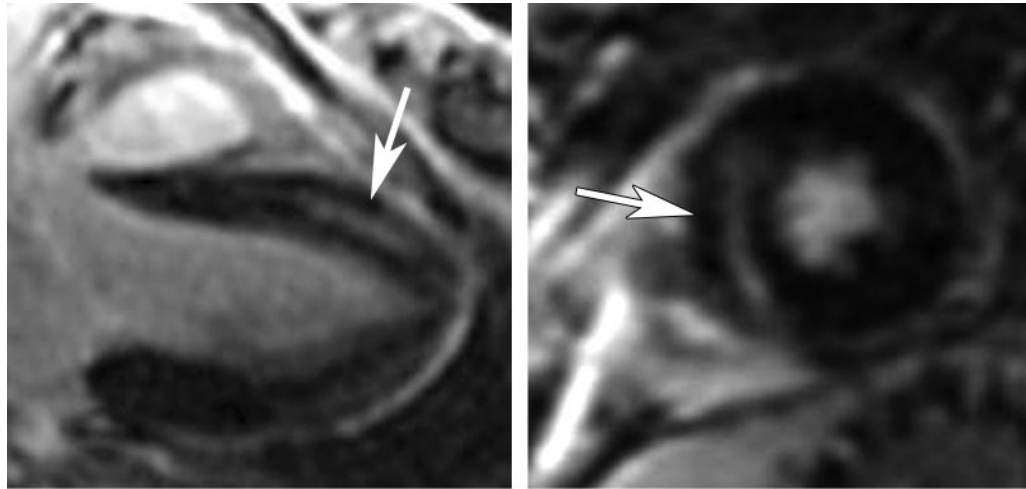
**Figure 4.** True aneurysm and thrombus in a 67-year-old man after a myocardial infarction. Two-chamber (a) and four-chamber (b) MR images show moderate thinning and delayed myocardial enhancement of the anterior left ventricular wall (arrows) with an aneurysm; these findings were due to a chronic full-thickness myocardial infarction. The aneurysm is complicated by a mural thrombus. True aneurysms, which are composed of pericardium adherent to underlying epicardium and scar tissue from infarcted myocardium, can be distinguished from false aneurysms, which consist of pericardium that contains a ruptured left ventricle. False aneurysms are not expected to demonstrate high signal intensity in the wall on delayed enhancement MR images due to lack of scar tissue. Because false aneurysms represent contained myocardial ruptures, they require urgent surgical repair.



a.

b.

**Figure 5.** Acute and chronic transmurular myocardial infarction in a 68-year-old man with chest pain. (a) Short-axis delayed enhancement MR image shows a transmurular (100%) acute myocardial infarction in the lateral and inferior left ventricular wall (arrows), a site compatible with a dominant right coronary artery territory. No left ventricular atrophy in the infarcted territory has occurred. (b) Corresponding image obtained 5 months later shows persistent delayed enhancement in the infarcted area (arrows), which demonstrates thinning relative to its appearance on the acute-phase image.



**a.** **b.**  
**Figure 6.** Idiopathic myocarditis in a 22-year-old woman. The patient presented 3 months earlier to an outside hospital with chest pain and fever. Follow-up MR imaging was performed to assess left ventricular function and evaluate scar extent. Four-chamber (**a**) and short-axis (**b**) delayed enhancement MR images show characteristic enhancement of the myocardial midwall at the septum and apex (arrow), an appearance typical of prior myocarditis.

Complications of myocardial infarction, such as aneurysm and thrombus formation, can also be assessed with delayed enhancement MR imaging. Acute thrombi may appear masslike, whereas chronic thrombi often conform to the contour of the cardiac cavity. The vascular supply of myocardial thrombi is poor, so that the majority do not enhance after the administration of gadolinium contrast material (Fig 4). Rarely, thrombi may demonstrate delayed contrast enhancement due to organization of the thrombus (8).

### Delayed Enhancement MR Imaging in Nonischemic Heart Disease

Delayed myocardial enhancement is not specific for myocardial infarction and can be observed in many other cardiac diseases and after cardiac interventions. **Unlike in ischemic heart disease, delayed enhancement in nonischemic myocardial disease generally does not correspond to any particular coronary artery distribution and is often midwall rather than subendocardial or transmural (9).** Moreover, in the acute phase, the first-pass perfusion study usually does not show any focal

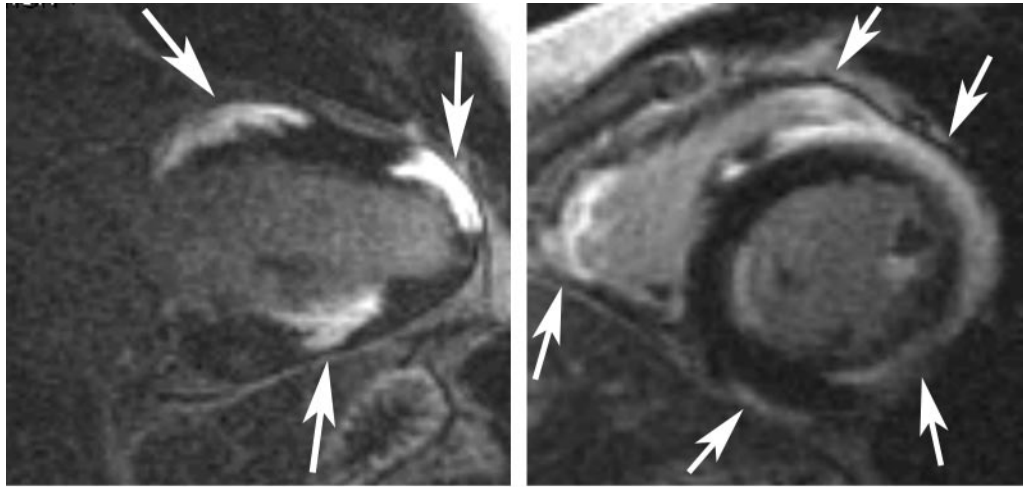
perfusion defect in nonischemic cardiomyopathy but instead may show normal results or early increased enhancement (10).

### Inflammation

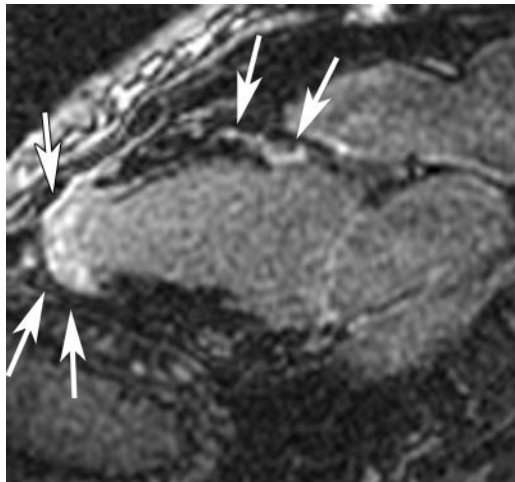
**Myocarditis.**—Myocarditis is defined clinically as inflammation of the heart muscle. Although the cause of myocarditis often remains unknown, a large variety of infections, systemic diseases, drugs, and toxins have been associated with myocarditis (11).

The presence of focal delayed enhancement in a non-coronary artery distribution together with wall motion abnormalities correlates strongly with acute or subacute myocarditis in the correct clinical setting (Figs 6–8). The enhancement pattern has been described as becoming less intense and more diffuse over a period of weeks to months. Myocarditis lesions occur predominantly in the lateral free wall and originate from the epicardial quartile of the ventricular wall. In a study by Mahrholdt et al (13), contrast enhancement was never seen to originate from the subendocardium, a pattern that is otherwise typical for myocardial infarction. Sometimes it can be difficult to distinguish myocardial infarction from myocarditis with





**Figure 7.** Acute idiopathic myocarditis in a 30-year-old man with frequent premature ventricular contractions and normal results at coronary angiography. Two-chamber long-axis MR image of the left ventricle (**a**) and short-axis MR image (**b**) show extensive scattered delayed enhancement in the anterior, lateral, and inferior wall and apex of the left ventricle and the right ventricular wall (arrows). The endocardium is relatively spared by the inflammation, which occurs predominantly in the epicardium and mid myocardial wall. This distribution is typical of myocarditis. (Case courtesy of Dr John Freeby.)



**Figure 8.** Chagas myocarditis in a 34-year-old man from Brazil. Two-chamber MR image shows patchy delayed enhancement at the apex and mid anterior wall of the left ventricle (arrows). The patient was infected with *Trypanosoma cruzi*, and the myocardial areas of delayed enhancement represent inflammation due to Chagas disease (12). This parasite can spread via the bloodstream into many organ systems and often affects the heart.

imaging alone (14). Therefore, adequate patient history and examination are crucial for correct image interpretation.

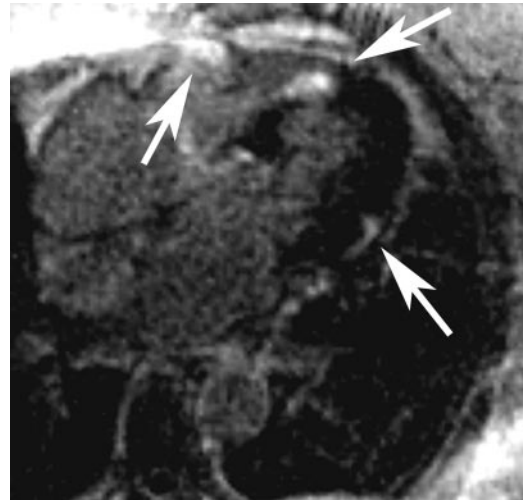
In myocarditis, myocardial damage is more diffuse than damage due to infarction. Islands of necrotic cells are scattered throughout the focus of acute myocarditis. Thus, contrast enhancement in acute myocarditis may not be as intense as in myocardial infarction. During healing, inflammatory cells infiltrate the myocardial regions of myocarditis, and eventually necrotic myocytes are replaced by areas of fibrous tissue. Therefore, delayed contrast enhancement can persist in the chronic phase. The area of myocarditis diminishes in size as it is replaced by scar, potentially explaining the observation that contrast enhancement typically decreases significantly over time (13,15).

**Sarcoidosis.**—Sarcoidosis is a multisystem inflammatory disease of unknown etiology. Cardiac involvement is symptomatic in 5% of patients with sarcoidosis (16). Clinical disease often includes heart block, dilated cardiomyopathy, and

ventricular arrhythmias. Patients with sarcoidosis are at increased risk of sudden death. Endomyocardial biopsy can demonstrate the diagnosis; however, it is invasive, and sampling errors can occur. Cardiac MR imaging is a useful noninvasive method for the early noninvasive diagnosis and follow-up of cardiac sarcoidosis (16). In a study by Shimada et al (17), eight of 16 patients with sarcoidosis and suspected cardiac involvement demonstrated gadolinium enhancement of the myocardium. Endomyocardial biopsies of all eight patients with MR imaging abnormalities were positive.

In acute myocardial sarcoidosis, increased focal signal intensity can be noted on both T2-weighted and early gadolinium-enhanced MR images because of edema associated with inflammation. Delayed myocardial enhancement may also be observed, probably reflecting accumulation of gadolinium as a result of differences in contrast agent distribution volume (Fig 9). Focal myocardial thickening is often seen because of the edema, a feature that can mimic hypertrophic cardiomyopathy or a cardiac mass (18). Myocardial inflammation in sarcoidosis often involves the septum and sometimes the left ventricular wall, whereas papillary muscle and the right ventricular wall are rarely affected. In advanced postinflammatory sarcoidosis, dilated cardiomyopathy and delayed enhancement can be seen, most likely related to areas of myocardial fibrosis (10).

**Vasculitis.**—Various forms of arteritis or vasculitis exist. Some of these vasculitides may affect the heart. In young children, Kawasaki syndrome is well known to cause coronary artery aneurysms, stenosis and occlusion due to thrombi, and myocarditis (19). Myocarditis and especially myocardial infarction in these cases can readily be depicted with delayed enhancement MR imaging (Fig 10). For these children, MR imaging is the preferred method of monitoring the cardiac involvement due to Kawasaki syndrome. A case report of gadolinium-enhanced cardiac MR imaging described focal inflammation in the location of the atrioventricular node in a patient with Wegener granulomatosis, causing a complete heart



**Figure 9.** Cardiac sarcoidosis in a 56-year-old woman with chronic coughing and occasional wheezing. Four-chamber delayed enhancement MR image shows foci of myocardial enhancement in the septum, lateral left ventricular wall, and right ventricular wall (arrows). Other foci of delayed enhancement were seen in the anterior-lateral segments and inferior wall.

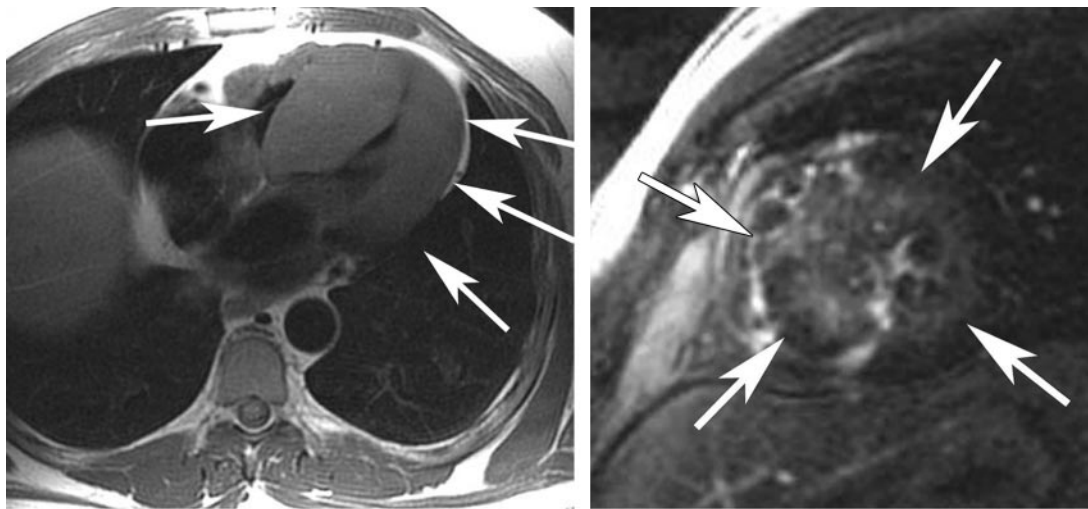
block (20). New developments in MR imaging unit technology will further improve spatial and temporal resolution in the near future. This may enable visualization of gadolinium enhancement directly within the coronary artery wall in patients with vasculitis or atherosclerotic plaques, as already shown in larger and more superficial arteries (21,22).

### Cardiomyopathy

**Hypertrophic Cardiomyopathy.**—Hypertrophic cardiomyopathy is a primary myocardial disease characterized by focal or diffuse left ventricular wall thickening in the absence of dilatation. Hypertrophic cardiomyopathy is often characterized by impaired regional myocardial function, arrhythmias, and decreased coronary flow reserve. In hypertrophic cardiomyopathy, cardiac MR imaging is used to assess left ventricular wall thickness and mass, regional myocardial function, and degree of left ventricular outflow tract obstruction. At histologic analysis, hypertrophic cardiomyopathy is characterized by myofibrillar disarray and abnormal increase in connective



**a.** **b.**  
**Figure 10.** Kawasaki syndrome in an 18-year-old man. **(a)** Maximum intensity projection steady-state free precession image shows a 1.5-cm aneurysm (arrow) at the origin of the right coronary artery. There was also an aneurysm in the distribution of the left coronary artery with thrombosis; this aneurysm resulted in an extensive infarction of the left anterior descending artery. **(b)** Short-axis delayed enhancement MR image shows wall thinning and delayed enhancement (arrows) due to infarction.

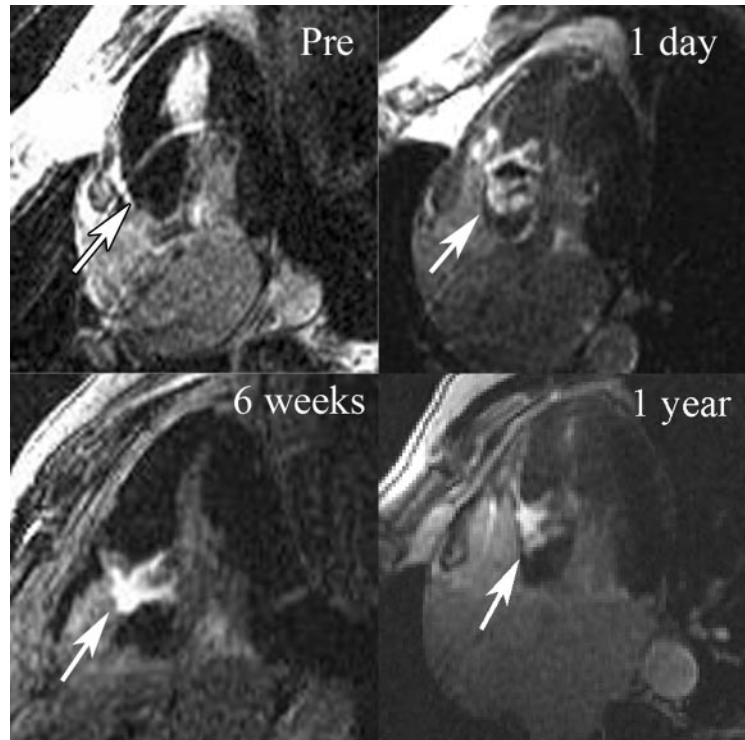


**a.** **b.**  
**Figure 11.** Hypertrophic obstructive cardiomyopathy in a 48-year-old patient with chest pain. **(a)** Four-chamber black blood MR image shows marked hypertrophy of the ventricular septum and left ventricular wall (arrows). **(b)** Short-axis delayed gadolinium-enhanced MR image shows subendocardial areas of delayed myocardial enhancement (arrows) in the left ventricular wall and septum. These areas of delayed enhancement are related to the myofibrillar disarray and scattered fibrosis present in hypertrophic obstructive cardiomyopathy.

(fibrotic) tissue within the left ventricular wall, which demonstrates near transmural, scattered, patchy delayed gadolinium enhancement in the hypertrophied myocardium (Fig 11). The degree

of delayed enhancement is inversely correlated with regional contraction and positively correlated with regional hypertrophy (23).

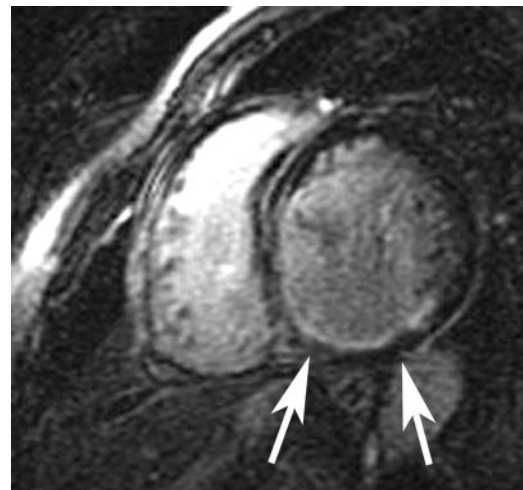




**Figure 12.** Hypertrophic obstructive cardiomyopathy in a 46-year-old man with dyspnea, chest discomfort, and exertional syncope. Transarterial alcohol ablation was performed via the first perforating branch off the left anterior descending artery for treatment of left ventricular outflow tract obstruction. MR images show delayed myocardial enhancement in the proximal septum (arrow) beginning directly after the ablation procedure and persisting over time. Note the gradually decreasing septal wall thickness due to iatrogenically induced myocardial infarction and subsequent scar formation.

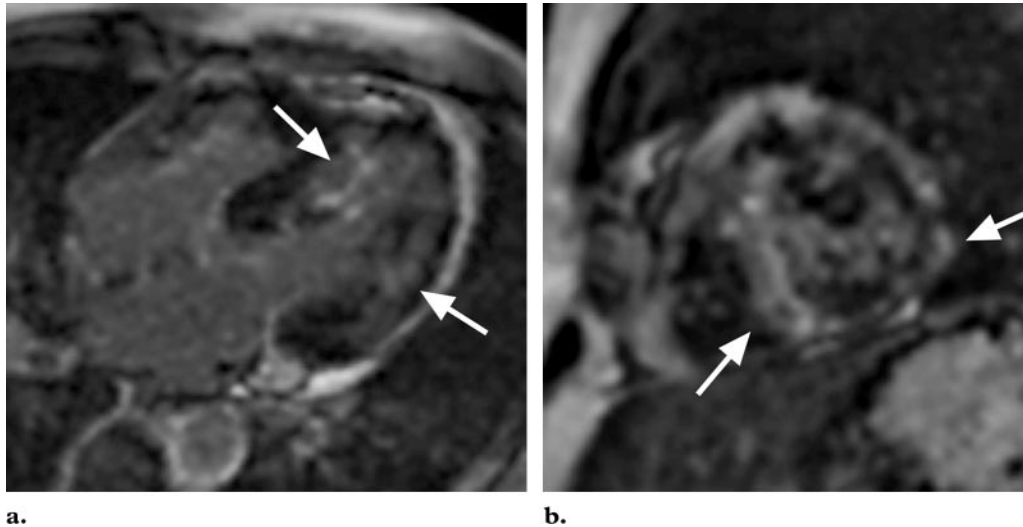
Owing to the limited coronary flow reserve, ischemic necrosis is frequently observed on delayed enhanced MR images in a centripetal fashion starting in the subendocardium and leading to a decrease in myocardial thickness in the chronic phase. Transarterial alcohol ablation via the first perforating branch off the left anterior descending coronary artery for treatment of the left ventricular outflow tract obstruction can be monitored with serial delayed enhancement MR imaging (Fig 12).

**Dilated Cardiomyopathy.**—The most common causes of dilated cardiomyopathy are idiopathic or inherited, viral infection, and alcoholism. Delayed enhancement MR imaging may help identify dilated cardiomyopathy caused by coronary artery disease. McCrohon et al (9) examined 63 patients with dilated cardiomyopathy and unobstructed coronary arteries. Of their patients with dilated cardiomyopathy, 59% did not show gadolinium enhancement; 28% demonstrated longitudinal or patchy midwall enhancement, which was unrelated to any coronary territory and most likely reflected focal segmental fibrosis, myocardial degeneration, or necrosis. Finally, 13% of the patients had subendocardial or transmural en-

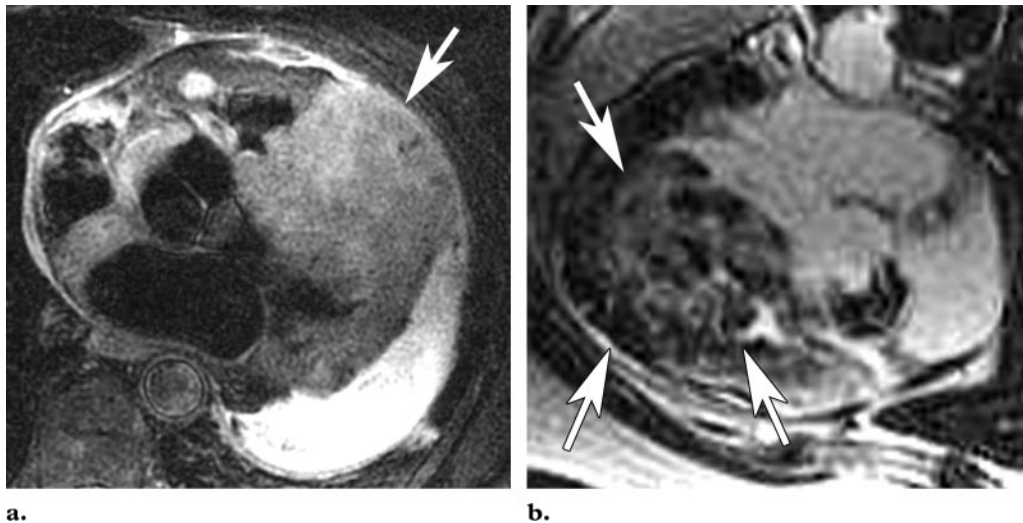


**Figure 13.** Ischemic dilated cardiomyopathy in a 60-year-old woman with three-vessel coronary artery disease. Short-axis delayed enhancement MR image shows marked dilatation of the left ventricle, which was 8 cm in diameter. There is mild subendocardial delayed enhancement and thinning of the inferior wall (arrows) due to an old infarction of the right coronary artery. Delayed enhancement is not usually seen in non-ischemic dilated cardiomyopathy.

hancement that was indistinguishable from that in ischemic patients with coronary artery disease (Fig 13). This subgroup was reassigned as cardiac failure due to coronary artery disease with recanalized coronary arteries. Koito et al (24) report that



**Figure 14.** Cardiac amyloidosis in a 56-year-old man with chest pain, renal failure, and diabetes. Short-axis (a) and two-chamber (b) MR images show delayed, patchy, inhomogeneous gadolinium enhancement of the left ventricular wall in a nonvascular distribution (arrows). A low normal ejection fraction of 55% and a restrictive wall motion pattern were observed at cine MR imaging.



**Figure 15.** Metastatic breast cancer involving the heart and pericardium in a 55-year-old woman. (a) Axial T2-weighted MR image shows a large bulky mass at the left ventricular apex (arrow) and a moderate-sized pericardial effusion. (b) Axial delayed enhancement MR image shows diffuse delayed enhancement of the mass (arrows).

the degree of myocardial enhancement correlates with the severity of left ventricular dysfunction in patients with dilated cardiomyopathy.

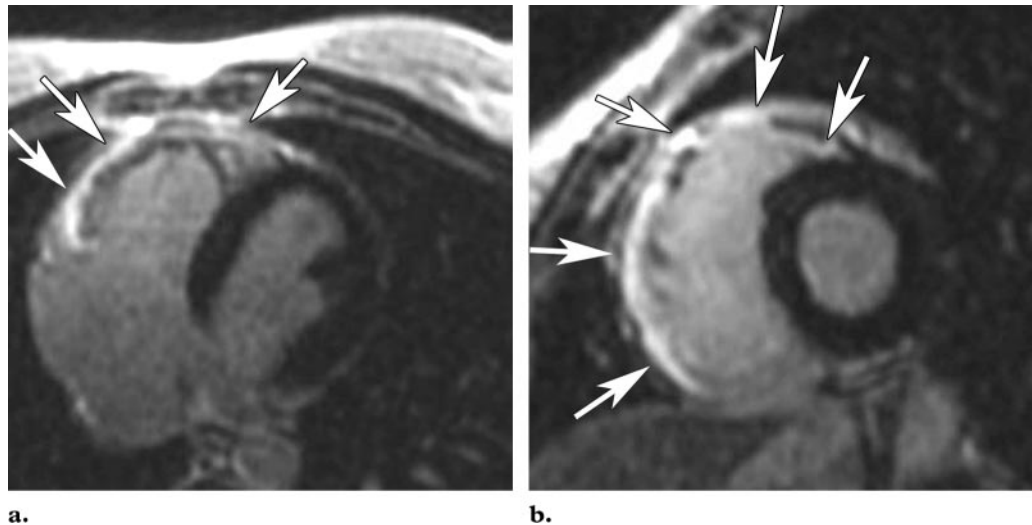
**Restrictive Cardiomyopathy.**—Apart from idiopathic primary restrictive cardiomyopathy, amyloidosis is a common cause of secondary restrictive cardiomyopathy. It is an infiltrative deposition disease of fibrillar proteins and can be primary or secondary in origin. Cardiac amyloidosis may lead to restrictive cardiomyopathy due to loss of compliance and diastolic function. MR imaging can show functional impairment, biventricular hypertrophy, and nonspecific inhomogeneous

gadolinium enhancement (Fig 14). Ventricular enhancement may also occur in certain forms of glycogen storage diseases (25).

### Cardiac Masses

Metastases to the heart and pericardium are 20–40 times more common than primary tumors (26). Primary tumors of the heart are rare and are most often benign. Most primary and secondary cardiac tumors enhance with gadolinium and may demonstrate delayed enhancement (Fig 15).





**Figure 16.** ARVD in a 43-year-old woman. Long-axis (a) and short-axis (b) delayed enhancement MR images show diffuse delayed enhancement (arrows). Delayed enhancement is present in about two-thirds of patients with ARVD. Delayed enhancement of the right ventricle in association with reduced function or an aneurysm is highly suggestive of ARVD in patients who have nonsustained ventricular tachycardia with left bundle-branch block.

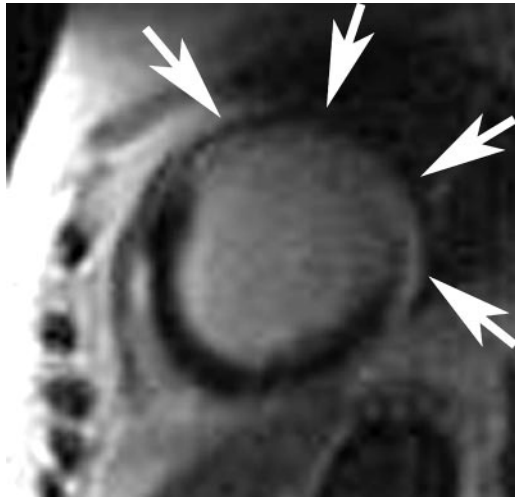
However, in general, delayed enhancement sequences in addition to the routine gadolinium-enhanced sequences do not help in further characterizing a cardiac mass. Morphologic features, signal intensity characteristics, and a good patient history are usually more important in determining the cause of cardiac masses.

### Genetic or Congenital Conditions

**Arrhythmogenic Right Ventricular Dysplasia.**—Arrhythmogenic right ventricular dysplasia (ARVD) is characterized by structural and functional abnormalities of the right ventricular wall leading to ventricular arrhythmias and progressive right ventricular failure. ARVD is associated with sudden cardiac death. The diagnosis of ARVD especially at early stages remains challenging and is based on major and minor criteria including family history, genetic factors, conduction and repolarization abnormalities, biopsy results, and abnormal right ventricular morphology and function.

In addition to the morphologic and functional evaluation of the right ventricle with cardiac MR imaging, delayed enhancement MR imaging can demonstrate diffuse or segmental replacement of myocardium in the right ventricular free wall by fibrofatty tissue (Fig 16). In a recent study, we observed myocardial delayed enhancement in eight of 12 patients with ARVD (67%), whereas none of the patients without ARVD demonstrated delayed myocardial enhancement of the right ventricle. An increasing number of enhancing segments of the right ventricle correlates with a decreased right ventricular ejection fraction and increasing end-diastolic volume (27). Myocardial delayed enhancement MR imaging may help improve the specificity of MR imaging for ARVD diagnosis.

**Muscular Dystrophy.**—Becker muscular dystrophy and Duchenne muscular dystrophy are X-linked recessive neuromuscular disorders characterized by progressive skeletal and cardiac weakness. Cardiac myocyte dystrophin deficiency leads to fiber necrosis causing biventricular replacement of morbid myocardium with connective tissue and fat, which demonstrate high signal intensity at myocardial delayed enhancement MR



**Figure 17.** Duchenne muscular dystrophy in an 18-year-old man with heart failure. Coronary angiography showed normal coronary arteries. Short-axis MR image shows extensive, nearly transmural delayed enhancement of the anterior and lateral left ventricular wall (arrows) and left ventricular dilatation (ejection fraction of 34%). The findings are compatible with fibrosis of the left ventricular wall due to muscular dystrophy.

imaging, predominantly in the midwall (28) (Fig 17). Patients often have dilated cardiomyopathy. In the presence of midmyocardial delayed enhancement in a patient with dilated cardiomyopathy, muscular dystrophy should be considered as a rare but important differential diagnosis.

### Conclusions

Delayed myocardial enhancement cardiac MR imaging is a reliable tool for assessing chronic and acute myocardial infarction and predicting patient outcome after myocardial infarction treatment. However, delayed myocardial enhancement is not specific for myocardial infarction and can be seen in many other cardiac pathologic conditions and after cardiac interventions. Therefore, clinical history is critical in the evaluation of myocardial delayed enhancement cardiac MR images.

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## Delayed Enhancement MR Imaging: Utility in Myocardial Assessment

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### Page 796

It is important to recognize that delayed myocardial enhancement is not specific for myocardial infarction and can occur in a variety of other disorders, such as inflammatory or infectious diseases of the myocardium, cardiomyopathy, cardiac neoplasms, and congenital or genetic cardiac pathologic conditions.

### Page 797

The contrast material clearance from the normal myocardium is determined by several factors: the washout rate of contrast material from the normal myocardium, overall cardiac function, renal function, and possibly the administered dose of contrast material.

### Page 798

Scar or fibrosis is depicted as an area of high signal intensity on delayed enhancement MR images that is typically subendocardial or transmural in a coronary artery distribution.

### Page 798

In general, myocardial scar or fibrosis involving more than 50% of myocardial wall thickness at cardiac MR imaging is unlikely to recover contractile function following coronary revascularization (6).

### Page 802

Unlike in ischemic heart disease, delayed enhancement in nonischemic myocardial disease generally does not correspond to any particular coronary artery distribution and is often midwall rather than subendocardial or transmural (9).