The Heart

7

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7.1 Introduction to Techniques and Applications

Magnetic resonance imaging of the heart has become a central noninvasive tool for the evaluation of patients with cardiovascular disease.

MRI provides excellent soft tissue contrast and tissue characterization, and advances made over the last decade have improved the evaluation of both cardiac morphology and function (ACCF/ACR/AHA/NASCI/SCMR 2010). A variety of MRI protocols are available for analysis of cardiac function, including wall motion at rest and stress, identification of inflammation and ischemia, viability imaging, and evaluation of valve function, making MRI a versatile imaging modality for a wide range of diagnostic tasks in patients with cardiac disease (ACCF/ACR/AHA/NASCI/SCMR 2010).

Currently, the most important indications for cardiac MRI are (Jiji and Kramer 2011):

- Assessment of left ventricular function and myocardial viability in ischemic heart disease
- Imaging of patients with suspected infiltrative cardiomyopathies, hypertrophic (obstructive) cardiomyopathies, or arrhythmogenic right ventricular cardiomyopathy
- Patients with ischemic heart disease or cardiac failure in whom echocardiography does not allow adequate evaluation of cardiac structures or function
- Imaging of myocardial viability prior to revascularization
- Evaluation of pericardial tissue or constrictive pericarditis
- Assessment of myocardial mass and evaluation for thrombus
- Analysis of nearby vessels prior to ablation therapy

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All cardiac MRI examinations are performed during breath-hold and require careful timing using a reliable triggering technique (electrocardiogram or, when a good ECG signal cannot be obtained, peripheral pulse monitoring).

7.1.1 Cardiac Function Imaging

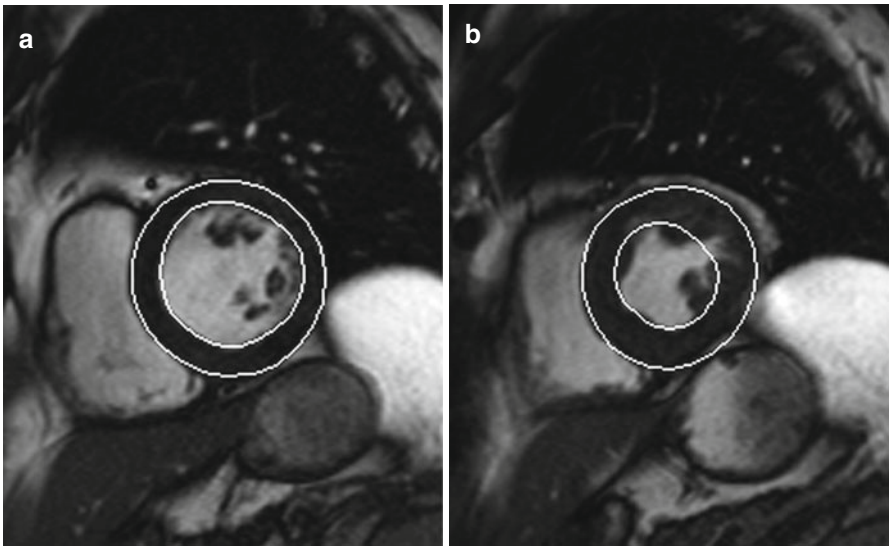
Four-dimensional cine MR imaging is highly accurate and reproducible. Hence, it has become the method of choice (gold standard) for assessing left ventricular function and identifying cardiac wall motion abnormalities (Palumbo et al. 2010). Images are acquired over several cardiac cycles, which eliminates motion artifacts and ensures adequate resolution per cardiac phase.

MRI is an excellent modality for the assessment of cardiac function, allowing determination of standard functional parameters such as left ventricular ejection fraction (LVEF), left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), and left ventricular mass (LVM) (Walsh and Hundley 2007).

Cardiac function can be determined using long-axis or short-axis views:

1. Long-axis views: Cardiac function in the long axis is assessed using the area-length method (ALM), which provides a very accurate measure of cardiac dimensions in individuals with normal-sized hearts and normal contractile function. When using this method, it is important to exclude the papillary muscles, or else left ventricular mass may be overestimated and left ventricular volume underestimated.
2. Short-axis views: This method is more accurate but also more time-consuming, and the postprocessing software required for it is provided by most manufacturers as part of the standard package. Functional parameters are calculated using the slice summation method (SSM). In a stack of contiguous short-axis slices covering the complete ventricle from apex to base (valvular plane), the end-systolic, end-diastolic, and epicardial areas are outlined in each slice. The outlined areas are converted to volumes and summed to produce estimates of end-diastolic and end-systolic volumes and myocardial mass. In contrast to the first method, here it is important to include the papillary muscles, as they account for approx. 8–10 % of the left ventricular mass, and the SSM allows their size and location to be determined accurately (Nassenstein et al. 2009) (Fig. 7.1).

Fig. 7.1 Assessment of the left ventricle using the slice summation method (SSM). Short-axis views in end diastole (**a**) and end systole (**b**). (**c**) Parameters determined in assessing left ventricular function are *EF* ejection fraction, *EDV* end-diastolic volume, *ESV* end-systolic volume, *SV* stroke volume, *CO* cardiac output, and myocardial mass (at end diastole and average); measures of ventricular ejection and filling rates: peak ejection rate, time to peak ejection rate, peak filling rate, and time from end systole to peak filling rate



Pat. ID:	00008...	Untersuch. Datum:	22.07.2008		
Pat.Größe:	178.00 cm.	Pat.Gewicht:	79.30 kg.	Herzrate:	66 Schläge/min

c **Linker Ventrikel - Absolut**

Herzfunktion			Normaler Bereich (M) (MRI)	Einheiten
Auswurffraktion	EF	62.5	56.00 ... 78.00	%
ED-Volumen	EDV	174.7	77.00 ... 195.00	ml
ES-Volumen	ESV	65.5	19.00 ... 72.00	ml
Schlagvolumen	SV	109.1	51.00 ... 133.00	ml
Herzminutenvolumen	CO	7.20	2.82 ... 8.82	l/min
Myokardmasse (in ED)		130.7	118.00 ... 238.00	g
Myokardmasse (Mittelwert)		151.6 ± 15.0	118.00 ... 238.00	g
Füll- und Auswurfdaten				
Max. Auswurfrate		412.6	n.a.	ml/sec
Max. Auswurfzeit		153.7	n.a.	msec
Max. Füllrate		353.8	n.a.	ml/sec
Max. Füllzeit nach ES		133.2	n.a.	msec

Prüfen Sie die Konturen. Diese können von den anatom. Strukturen abweichen.

Cine sequences also permit visual assessment of ventricular contraction, detection of global or locoregional wall motion abnormalities, and evaluation of valve function (Figs. 7.2 and 7.3).

Quantitative estimation of stenosis severity and valve incompetence requires the use of a flow-sensitive imaging technique. Flow across the valve is typically assessed by acquisition perpendicular to the valvular plane and is known as through-plane acquisition.

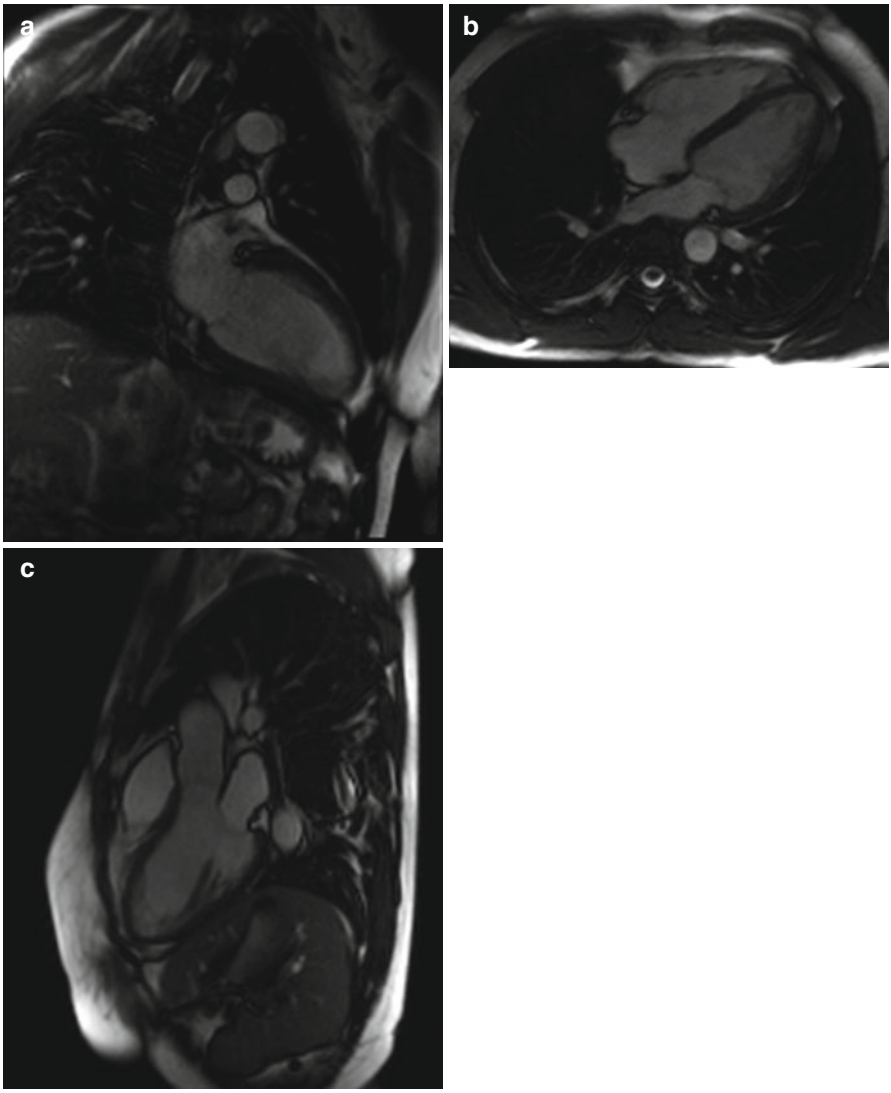


Fig. 7.2 Cine images from a healthy subject. (**a**) Two-chamber view, (**b**) four-chamber view, and (**c**) three-chamber view

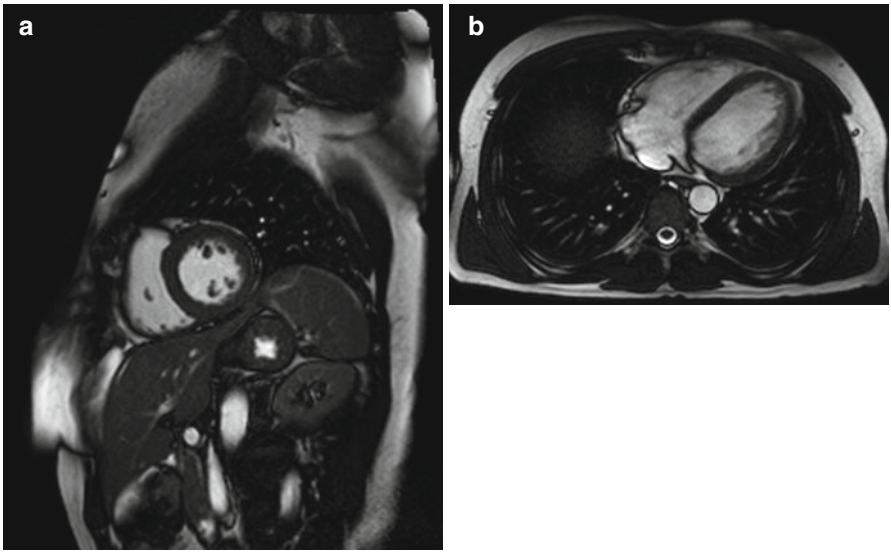


Fig. 7.3 Cine images from a healthy subject. (**a**) Short-axis view (midventricle) and (**b**) true axial view (midventricle)

7.1.2 Edema Imaging

A clinical cardiac MRI protocol used in patients with suspected myocarditis or cardiomyopathy should always include a fat-saturated T2-weighted sequence (Carbone and Friedrich 2012). Because it suppresses signal from fat and flowing blood, this pulse sequence is highly sensitive for detecting fluids in tissues. It has approx. 80 % sensitivity in identifying myocardial edema and is also well suited for detecting concomitant pericardial effusion. Myocardial edema imaging using newer pulse sequences with much improved image quality provides useful additional diagnostic and prognostic information, allowing identification of acute or recent myocardial ischemic injury in patients with acute chest pain, distinguish acute from chronic myocardial infarction and contributing to the determination of myocardium at risk (Eitel and Friedrich 2011). Despite these advances, cardiac MRI is only a supplementary tool and not the first-line imaging modality in patients undergoing imaging for evaluation of myocarditis or cardiomyopathy.

Inflammation damages the cell membrane, resulting in a larger distribution volume of contrast medium in injured myocardium. This is one of the mechanisms underlying late enhancement in cardiac imaging.

However, the presence of late enhancement alone does not allow discrimination between inflammatory processes and scar tissue. Areas of late enhancement resulting from retention of contrast medium are seen in a variety of disorders associated with myocardial injury and edema, necrosis, or fibrosis (Manrique et al. 2009). Some additional features such as distribution and location of delayed enhancement may help the radiologist in narrowing the differential diagnosis among ischemic and nonischemic disorders. For instance, when contrast medium accumulates in the epicardium, a

myocardial scar of ischemic origin is unlikely because such a scar is typically seen in a subendocardial location. Furthermore, delayed enhancement occurring in coronary artery territories can contribute to the differentiation between scar and edema. In unclear cases, acquisition of the delayed enhancement sequence can be repeated after 15–20 min. At this point in time, a scar still has high signal intensity due to delayed washout of contrast medium from injured myocardium, while edema has low signal intensity (Abdel-Aty and Schulz-Menger 2007; Aletras et al. 2008).

7.1.3 Ischemia Imaging

Imaging of ischemic myocardial injury is an important component of a cardiac MRI protocol and is done by assessing late enhancement, typically 12–15 min after administration of a Gd-DPTA-based contrast agent (e.g., gadobutrol as in the SHIP). Differential enhancement of infarcted myocardium is primarily due to diffusion and delayed clearance of contrast medium from the zone of infarction. In addition, myocardial injury shortens longitudinal relaxation (T1). Compared with other imaging methods, contrast-enhanced cardiac MRI is more sensitive in detecting even small subendocardial infarcts. Furthermore, the extent of delayed enhancement on MRI closely correlates with the size of myocardial injury determined by established methods (Kim et al. 1999; Saraste et al. 2008). In contrast, PET and SPECT usually detect large transmural infarcts or nearly transmural defects but may miss smaller, subendocardial infarcts (Wagner et al. 2003).

Cardiac MRI may also detect complications of acute myocardial infarction such as the development of left ventricular pseudoaneurysm (Jiji and Kramer 2011).

Single-shot sequences are widely used for delayed enhancement imaging, offering the advantage that all data can be acquired in a single cardiac cycle. This comes at the cost of a loss of image sharpness.

After administration of gadolinium-based contrast medium, infarcted myocardium exhibits delayed enhancement and can be identified using an inversion recovery (IR) sequence, which greatly improves contrast between viable myocardium and infarction. When magnitude reconstruction is used, IR delayed enhancement imaging is highly sensitive to the inversion time (TI) selected (Kellman et al. 2002). The optimal TI to null signal from viable myocardium (i.e., to render it dark) for an individual can be determined by obtaining a TI scout series with progressively larger TIs (TI surfing). Typical TIs are between 200 and 300 ms, depending on the amount of contrast medium administered (usually 0.1–0.2 mmol/kg body weight), time of image acquisition after contrast administration, and individual contrast medium clearance (Stork et al. 2007). An optimal TI enhances the contrast between dark viable myocardium and bright infarcted tissue. When delayed enhancement studies are performed with a phase-sensitive inversion recovery (PSIR) sequence, it is possible to use a nominal value of TI, which eliminates the need for TI surfing and achieves a consistent contrast over a wide range of TIs without artifacts due to incorrect polarity (Kellman et al. 2002).

Phase-sensitive reconstruction is used to remove the background phase while preserving the sign of the desired magnetization during IR. The phase-sensitive reconstruction method reduces the variation in apparent infarct size observed in magnitude images as TI is changed. Phase-sensitive detection also has the

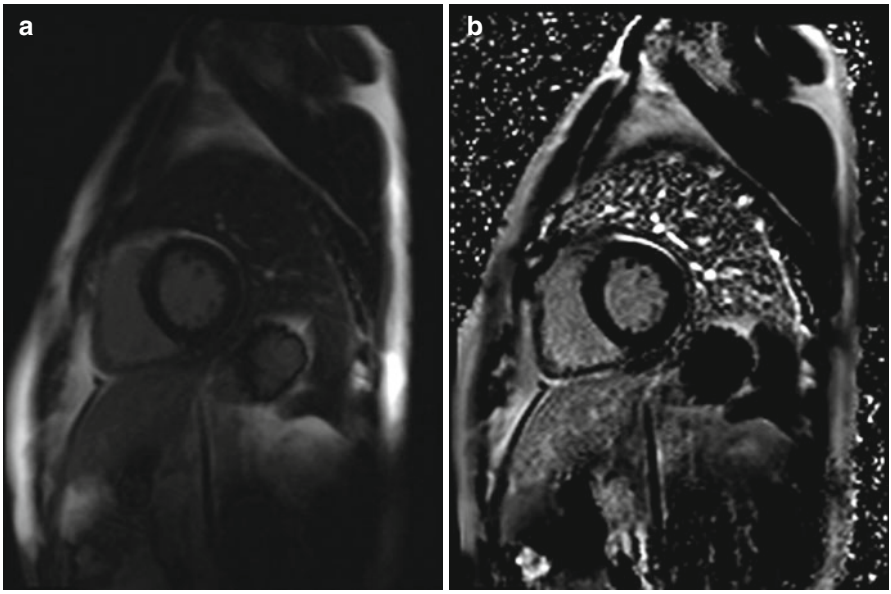


Fig. 7.4 Single-shot inversion recovery sequences (short axis) for delayed enhancement imaging in a healty subject. (**a**) magnitude reconstruction (**b**) phase-sensitive reconstruction

advantage of decreasing the sensitivity to changes in tissue T1 with increasing delay from contrast medium injection (Kellman et al. 2002) (Fig. 7.4).

There are certain pitfalls one must be aware of when interpreting delayed enhancement images. Mural thrombi do not take up contrast medium and will appear dark, just like normal myocardium. Regions of microvascular obstruction after acute myocardial infarction also have low signal intensity (Jiji and Kramer 2011). Hence, it is not possible to reliably differentiate thrombus, microvascular obstruction, and viable myocardium, precluding accurate estimation of infarct size (Karamitsos et al. 2009).

7.1.4 Viability Imaging

The use of specialized pulse sequences allows myocardial perfusion imaging by assessing contrast medium inflow and clearance from the myocardium. Myocardial perfusion can be assessed at rest and stress. Pharmacologic stress is induced by administration of adenosine or dobutamine. Areas of reduced myocardial perfusion show delayed contrast medium uptake, i.e., after administration of contrast medium, they remain dark longer than well-perfused myocardium (Karamitsos et al. 2011).

Myocardial perfusion MRI allows noninvasive assessment of myocardial viability and differentiation of infarcted areas from dysfunctional but viable myocardium (Saraste et al. 2008; Wellnhofer et al. 2004). In conjunction with cine MRI and delayed enhancement imaging, cardiac perfusion MRI enables reliable detection of coronary stenosis >70 % (De Mello et al. 2012).

7.1.5 Imaging in Rare Diseases of the Heart

Cardiac MRI also has an important role in diagnosing less common diseases of the heart such as arrhythmogenic right ventricular cardiomyopathy, cardiac sarcoidosis, myocardial involvement in amyloidosis, and tumors of the heart. Moreover, it can contribute to the detection of pericardial or endocardial disease (ACCF/ACR/AHA/NASCI/SCMR 2010).

7.1.6 Cardiac Imaging in Whole-Body MRI

Due to time constraints, a cardiac MRI examination performed as part of a whole-body MRI protocol cannot include all components of a comprehensive cardiac MRI study. The cardiac MRI protocol used in the SHIP included cine sequences for functional assessment in four-chamber, three-chamber, and two-chamber views as well as short-axis views for left ventricular function assessment. Strict axial cine sequences were acquired for evaluation of right ventricular function. A fat-saturated T2-weighted pulse sequence was deliberately not included although edema may persist for some time after a myocardial event. Delayed enhancement imaging was performed using an inversion recovery single shot sequence with magnitude reconstruction, highly sensitive to the inversion time (TI) selected and a phase-sensitive inversion recovery (PSIR) single shot sequence acquired 15 min after administration of 0.15 mmol/kg body weight of gadobutrol. The PSIR sequence used in the SHIP MRI protocol creates two image data records. First dataset contains images with magnitude reconstruction, in the second dataset phase-sensitive reconstruction images are generated.

The following sections give an overview of the most common incidental findings detected by cardiac MRI performed as part of a whole-body screening examination.

7.2 Diseases of Myocardium

Areas of delayed myocardial enhancement are the most common incidental findings in the heart at screening MRI. Note, however, that delayed enhancement is a mere descriptive term and provides no clues to possible underlying causes, which are multifarious (Kim et al. 2006). Classic causes include acute and chronic myocardial infarction, cardiomyopathy, and acute or chronic myocarditis (Stork et al. 2007). In a cardiac MRI protocol, late enhancement sequences enable accurate and noninvasive quantification of the extent of myocardial infarction (gold standard). The transmural extent of delayed enhancement may be used to predict functional outcome in ischemic heart disease (Saraste et al. 2008).

7.2.1 Ischemic Heart Disease

Ischemic heart disease (IHD) or myocardial ischemia is characterized by reduced blood supply to the heart muscle and is usually due to atherosclerosis of the coronary arteries (ACCF/ACR/AHA/NASCI/SCMR 2010) (Table 7.1).

Table 7.1 Ischemic heart disease

Frequency	Common, prevalence of up to 20 %, increases with age M:F ratio of 4:1
Causes	Endothelial damage by atherogenic risk factors, e.g., hypercholesterinemia, hyperlipoproteinemia, smoking, diabetes mellitus, arterial hypertension, obesity, and familial predisposition Subsequent development of atheromatous plaque with narrowing of arterial lumen (critical when lumen loss is 70 % or greater) Resulting mismatch between myocardial oxygen supply and demand
Clinical presentation	Angina pectoris, exercise-induced dyspnea, cardiac insufficiency, cardiac dysrhythmia, myocardial infarction in coronary artery occlusion

Flow-limiting coronary stenosis causes coronary insufficiency, or a mismatch between oxygen supply and demand. The resulting myocardial ischemia has different clinical presentations:

- Angina pectoris (stable/unstable; Canadian Cardiovascular Society (CCS) classification system)
- Acute coronary syndrome (ACS)/myocardial infarction
- Ischemic cardiomyopathy with cardiac insufficiency
- Cardiac arrhythmia/sudden cardiac death

In patients first presenting with IHD, angina pectoris (55 %) is most common, followed by myocardial infarction (25 %) and sudden cardiac death (20 %).

IHD remains a leading cause of mortality and morbidity.

Cardiac MRI is highly accurate and has robust prognostic value in the evaluation of patients with both acute and chronic IHD (Heydari and Kwong 2014).

MRI Features

- Lines of myocardial calcification or subendocardial fatty metaplasia.
- Myocardial thinning with akinesia, hypokinesia, and dyskinesia.
  - Akinesia is common in areas of transmural infarction and manifests as absence of systolic thickening and contraction in affected wall segments.
  - Hypokinesia is reduced systolic thickening and contraction of myocardium.
  - Dyskinesia is paradoxical motion, often involving predominantly the septal segments of the left ventricle (septal dyskinesia).
- Myocardial aneurysm or pseudoaneurysm can develop.
- Subendocardial to transmural delayed enhancement, usually without an increase in T2 signal intensity, which can be localized to the area supplied by a particular coronary artery (Stork et al. 2007). In acute infarction, concomitant edema of affected wall segments may be detectable on fat-saturated T2-weighted images (Karamitsos et al. 2011).

The severity of wall motion abnormality varies with the transmural extent of infarction. Wall motion disturbance and myocardial thinning are usually more marked when MRI shows full-thickness late enhancement compared with subendocardial enhancement (Fig. 7.5).

Myocardial infarction involving the papillary muscles can impair mitral valve closure. Short-axis views are well suited to localize infarcted myocardial segments to specific coronary artery territories.

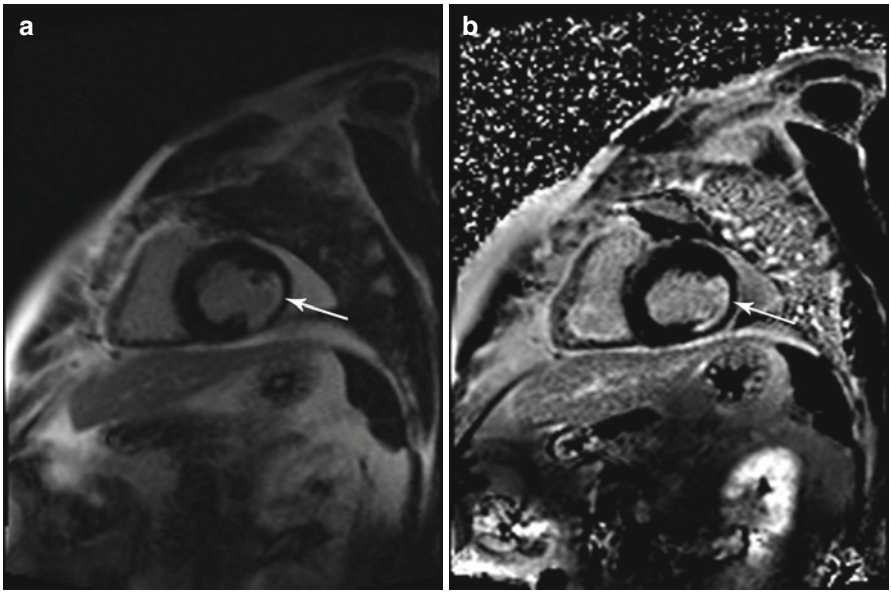


Fig. 7.5 Incidental findings in a 57-year-old subject. Typical subendocardial delayed enhancement in the lateral walls of the left ventricle (*arrow*). Late enhancement imaging with single shot inversion recovery sequences (short axis) (**a**) phase sensitive reconstruction (**b**) magnitude reconstruction

Among coronary dominance patterns, so-called balanced circulation is the most common type (80 %). In balanced circulation, the left coronary artery supplies the anterior wall of the left ventricle and most of the interventricular septum. The right coronary artery supplies the right ventricle and the posterior wall segments. The left coronary artery divides into the left anterior descending artery (LAD), or anterior interventricular branch, and the circumflex branch (LCX) (Cerqueira et al. 2002) (Fig. 7.6).

Differential Diagnosis

- *Delayed enhancement of other cause* (Stork et al. 2007)
- *Acute myocardial infarction* (edema on fat-saturated T2-weighted sequences, myocardial thickening, fatty metaplasia, calcification, or aneurysm detectable) (Perazzolo et al. 2011)
- *Acute myocarditis* (see below; edema on fat-saturated T2-weighted sequences)
- *Nonischemic cardiomyopathy* (see below; nonischemic myocardial fibrosis or scar has a variety of delayed enhancement patterns but is never subendocardial, making it easy to differentiate from ischemic infarction) (Stork et al. 2007)

In most patients, a variety of further cardiac and myocardial changes can be observed secondary to a myocardial infarction. Ventricular function may be impaired by left ventricular remodeling. Left ventricular dilatation is common and can evolve into full-blown dilated cardiomyopathy (De Smet et al. 2012). Initially the Frank-Starling mechanism maintains a constant left ventricular ejection fraction by responding to an increase in volume with an increase in wall tension. At some point,

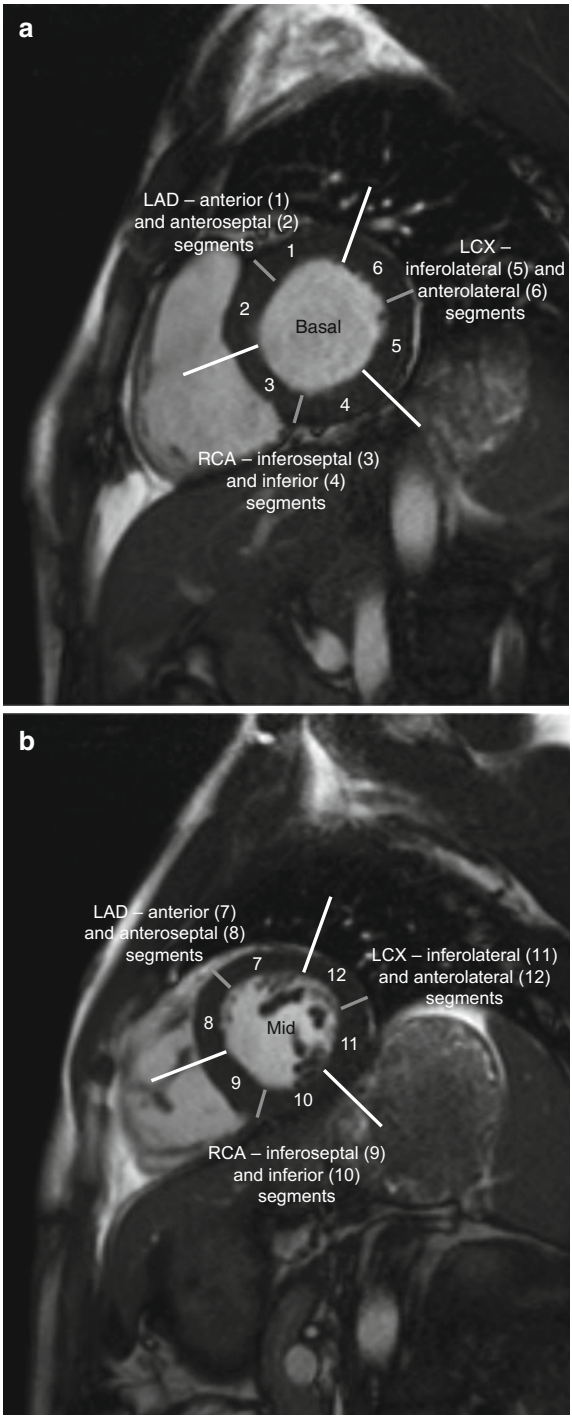


Fig. 7.6 Myocardial segmentation and assignment of the 17 myocardial segments to coronary artery territories. Short-axis views at basal level (**a**), at midventricular level (**b**), and at apical level (**c**) and apical long-axis view (**d**) (*LAD* left anterior descending artery, *LCX* left circumflex branch, *RCA* right coronary artery) (According to Cerqueira et al. (2002))

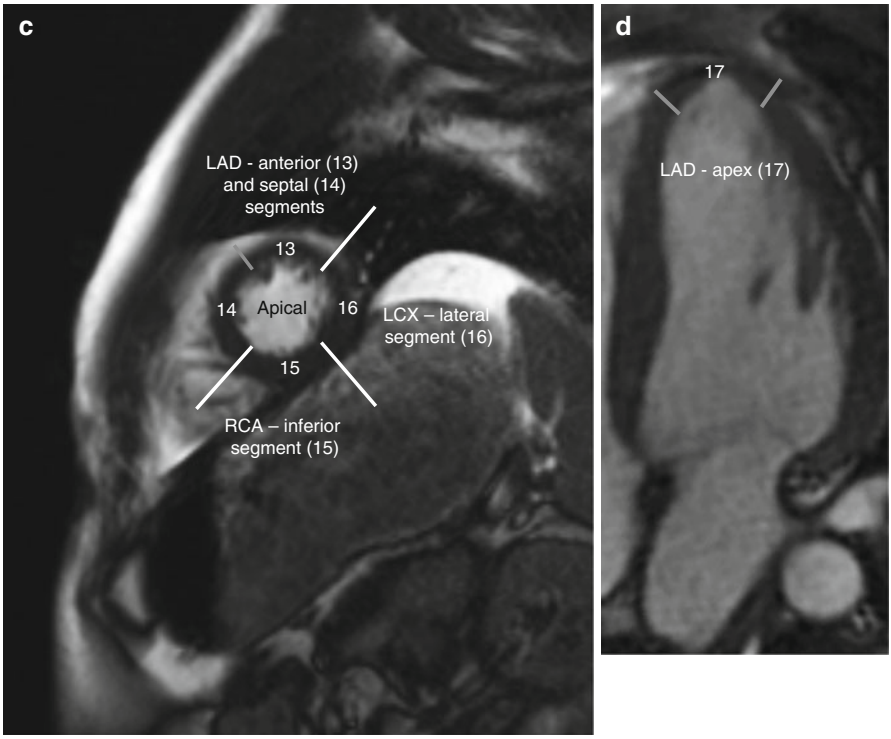


Fig. 7.6 (continued)

however, the bulge becomes so large that a marked drop in LVEF results. Mitral regurgitation due to remodeling of the chordal fibers of the papillary muscles is another common sequela.

Finally, pseudoaneurysm may develop, typically involving the posterior and lateral walls, less commonly the anterior wall.

Clinical Management

A previous small subendocardial or transmural myocardial infarction with well-preserved pump function requires no further clinical diagnostic evaluation. A larger subendocardial scar is critical even when pump function is preserved. It should be disclosed if a subject has another significant pathologic finding elsewhere in the body that may require surgery. The same holds true for transmural late enhancement with wall motion abnormalities. Markedly impaired pump function due to myocardial scar formation, which may be associated with concomitant pericardial or pleural effusion, should always be revealed to the subject. In this case, electrocardiography, ergometry, and echocardiography are recommended as supplementary diagnostic tests. Depending on the findings, cardiac catheterization may be the next step.

7.2.2 Cardiomyopathies

According to a consensus of the WHO and the International Society and Federation of Cardiology Task Force (1995), cardiomyopathies are diseases of the myocardium associated with cardiac dysfunction. Five main types are distinguished: dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM and HOCM), restrictive cardiomyopathy (RCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and unclassified cardiomyopathies, such as noncompaction cardiomyopathy and Takotsubo cardiomyopathy. They are primarily not based on valvular, pericardial, or cardiovascular disorders (ACCF/ACR/AHA/NASCI/SCMR 2010) (Tables 7.2 and 7.8).

Diseases of the heart muscle associated with cardiac or systemic disorders are referred to as specific cardiomyopathies in the WHO classification. They comprise ischemic, valvular, hypertensive, inflammatory, and metabolic cardiomyopathies, general system diseases, muscular dystrophies, neuromuscular disorders, sensitivity and toxic reactions, and peripartum cardiomyopathy. Dilated cardiomyopathy is the most common (Richardson et al. 1996) (Tables 7.2 and 7.3).

New scientific insights led to a revision of the definition and classification of cardiomyopathies in 2006. According to this revised definition, the cardiomyopathies are a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that frequently are genetic. Cardiomyopathies either are confined to the heart or are part of generalized systemic disorders, often leading to cardiovascular death or progressive heart failure-related disability (Maron et al. 2006).

The revised classification distinguishes two major groups based on predominant organ involvement: primary cardiomyopathies, in which the clinically relevant disease processes solely or predominantly involve the myocardium, and secondary

Table 7.2 WHO classification of cardiomyopathies

Type	Abbreviation	Characteristics
Dilated cardiomyopathy	DCM	Systolic dysfunction with reduced ejection fraction
Hypertrophic cardiomyopathy, subtype: hypertrophic obstructive cardiomyopathy	HCM, subtype: HOCM	Thickening of ventricular walls prevents adequate left ventricular expansion during diastole, impairing diastolic compliance
Restrictive cardiomyopathy	RCM	Increased wall stiffness due to endocardial fibrosis, impairing diastolic compliance
Arrhythmogenic right ventricular cardiomyopathy	ARVC	Right ventricular diastolic and systolic dysfunction and concomitant ventricular tachycardia as well as localized aneurysms of the right ventricle
Unclassified cardiomyopathies	UCCM	For example, noncompaction cardiomyopathy, Takotsubo cardiomyopathy

Modified according to Richardson et al. (1996)

Table 7.3 Dilated cardiomyopathy

Frequency	6/100,000 population/year
Causes	Idiopathic Genetic (familial) Primary and secondary causes Postinfectious (viral, bacterial, fungal, parasitic), toxic (alcohol, medications), autoimmune (vasculitis, systemic lupus erythematosus), endocrine (thyroid dysfunction, pheochromocytoma), neuromuscular and metabolic, infiltrative (amyloidosis), inflammatory (sarcoidosis) End stage of long-standing arterial hypertension, ischemia based on coronary artery disease, and progressive valvular dysfunction
Clinical presentation	Progressive left ventricular failure with exercise-induced dyspnea; global insufficiency and ventricular arrhythmia in advanced disease

cardiomyopathies, which show pathologic myocardial involvement as part of a large number and variety of generalized systemic (multiorgan) disorders.

Primary cardiomyopathies are further subdivided by etiology: genetic (hypertrophic cardiomyopathy, hypertrophic obstructive cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and noncompaction cardiomyopathy), mixed (dilated cardiomyopathy and restrictive cardiomyopathy), and acquired (inflammatory cardiomyopathy based on myocarditis, Takotsubo cardiomyopathy). Secondary cardiomyopathies appear in the context of various diseases: infiltrative (amyloidosis), inflammatory (sarcoidosis), storage (hemochromatosis), endomyocardial (endomyocardial fibrosis, hypereosinophilic syndrome (Löffler's endocarditis)), endocrine (diabetes mellitus, hyperthyroidism, pheochromocytoma), and neuromuscular diseases; nutritional deficiencies; autoimmune disorders (vasculitis, systemic lupus erythematosus); and exposure to toxic substances (cancer therapy) or drugs (Maron et al. 2006).

Dilated cardiomyopathies, which may be genetic or acquired, are the most common type. Causes of acquired dilated cardiomyopathy include a history of myocarditis or alcohol abuse. In addition, dilated cardiomyopathy may develop as the terminal stage of a variety of preexisting cardiac diseases, such as coronary heart disease or arterial hypertension (Richardson et al. 1996) (Tables 7.2 and 7.3).

Cardiac MRI has an important role in the diagnostic and clinical management of patients with cardiomyopathies, allowing evaluation of myocardial morphology and function in a single examination and hence also enabling identification of less common underlying causes (De Smet et al. 2012). The five main types of cardiomyopathy distinguished in the WHO classification are presented in more detail in the following sections.

7.2.2.1 Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is characterized by left ventricular dilation and systolic dysfunction with a left ventricular ejection fraction of less than 40 % with or without concomitant right ventricular involvement (ACCF/ACR/AHA/NASCI/SCMR 2010) (Fig. 7.7).

The severity of left ventricular dysfunction determines the patient's prognosis.

DCM is probably the result of myocardial damage due to a variety of primary and secondary underlying causes: infectious agents; viral or chronic myocarditis; other bacterial, fungal, and parasitic infections; toxic myocardial damage (alcohol, cocaine); chemotherapy (primarily doxorubicin); metabolic defects (e.g., hyperphosphatemia, hypocalcemia, uremia); myocardial involvement in other tissue disorders (vasculitis, e.g., Churg-Strauss syndrome, systemic lupus erythematosus); endocrine disorders (thyroid dysfunction, pheochromocytoma, Cushing disease); infiltrative or inflammatory diseases such as amyloidosis or sarcoidosis; and neuromuscular diseases. In rare cases, DCM is associated with pregnancy (peripartum cardiomyopathy). DCM is also the end stage of other myocardial conditions, such as long-standing arterial hypertension, ischemia based on coronary artery disease, or progressive valvular dysfunction. Approx. 20–35 % of DCM cases are assumed to be familial. In many cases, no etiology is apparent (idiopathic DCM) (Table 7.3) (De Smet et al. 2012; Maron et al. 2006).

DCM is characterized by progressive heart failure and a decline in left ventricular contractile function, ventricular and supraventricular arrhythmias, conduction system abnormalities, thromboembolism, and sudden or heart failure-related death (Maron et al. 2006). Patients with symptomatic DCM have a mortality rate of 11–13 %.

Cardiac MRI provides very accurate estimates of left and right ventricular volumes, atrial dimensions, myocardial mass, and left ventricular ejection fraction, and it also offers a quick and reliable means of detecting areas of fibrotic transformation (Schalla et al. 2010). The prognosis of idiopathic DCM depends on the severity of left ventricular dysfunction, with delayed enhancement being an additional prognostic factor. Areas of delayed enhancement indicate more extensive fibrotic transformation of myocardium, which is usually associated with lower LVEF and an increase in end-diastolic volume (Lehrke et al. 2011).

However, the estimates should be verified by echocardiography to confirm an initial MRI-based diagnosis of DCM.

MRI Features

- Enlargement of the left ventricle without an increase in mass; globally reduced systolic inward motion and wall thickening as well as markedly reduced LVEF (De Smet et al. 2012).
- Markedly reduced pump function and enlarged volume lead to circular movement of blood with an increased risk of thrombus formation.
- Ventricular dilation additionally causes regurgitation through the mitral valve and possibly through the tricuspid valve as well.
- This can result in enlargement of both atria.
- Depending on the severity of DCM, end-diastolic and end-systolic volumes (EDV and ESV) may be increased.
- In idiopathic DCM, first-pass perfusion usually appears normal.
- Concomitant enlargement of the right ventricle indicates advanced DCM.
- Thirty to forty percent of patients with idiopathic cardiomyopathy show midwall enhancement from the base to the midventricle (De Smet et al. 2012) (Fig. 7.8);

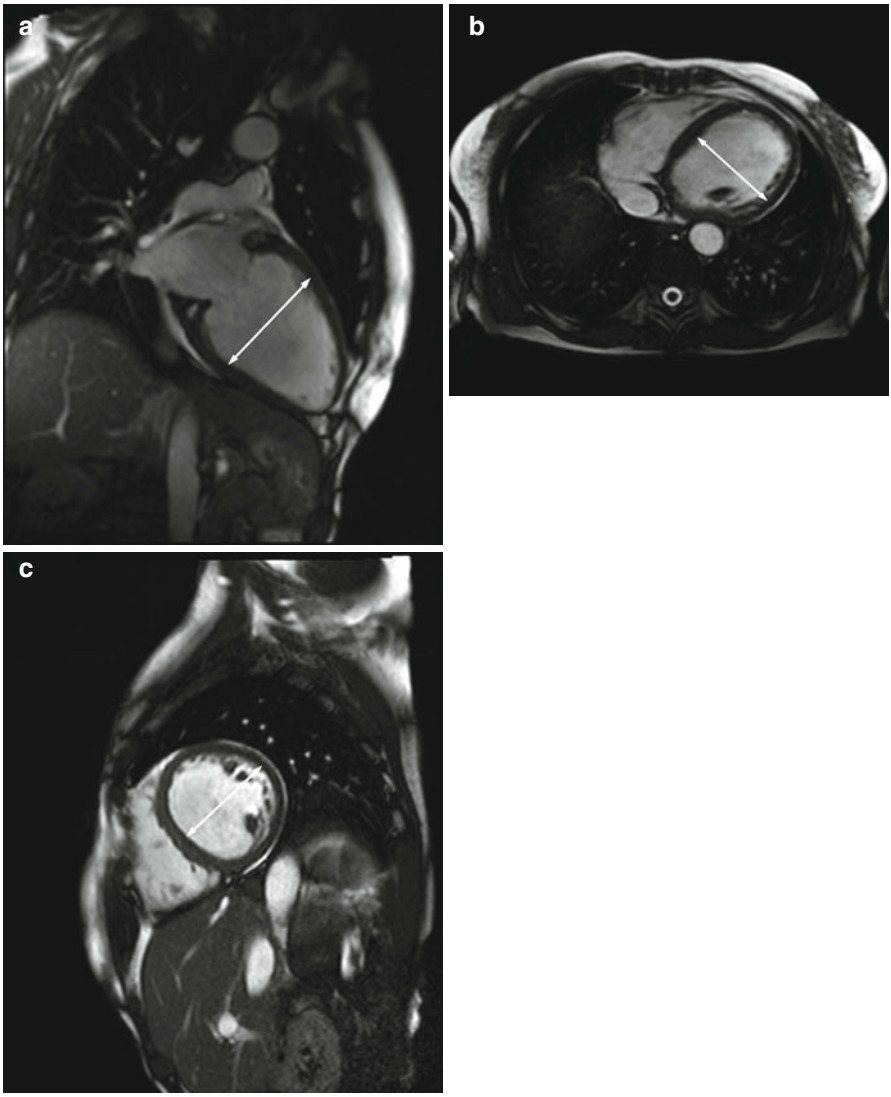


Fig. 7.7 DCM in a 66-year-old subject with reduced pump function (approx. 45 % EF) and global hypokinesia in all wall segments. Left ventricular enlargement (*arrow* indicates left ventricular diameter). (**a**) Two-chamber cine view, (**b**) true axial cine view, and (**c**) short-axis cine view

the typical pattern of late enhancement is in the form of longitudinal striae not corresponding to a coronary artery territory (Schalla et al. 2010; Stork et al. 2007).

- As DCM can also develop secondary to ischemic heart disease, marked subendocardial or transmural delayed enhancement may be seen in conjunction with left ventricular enlargement (McCrohon et al. 2003).
- An occasional patient may show no delayed enhancement at all (Jiji and Kramer 2011).

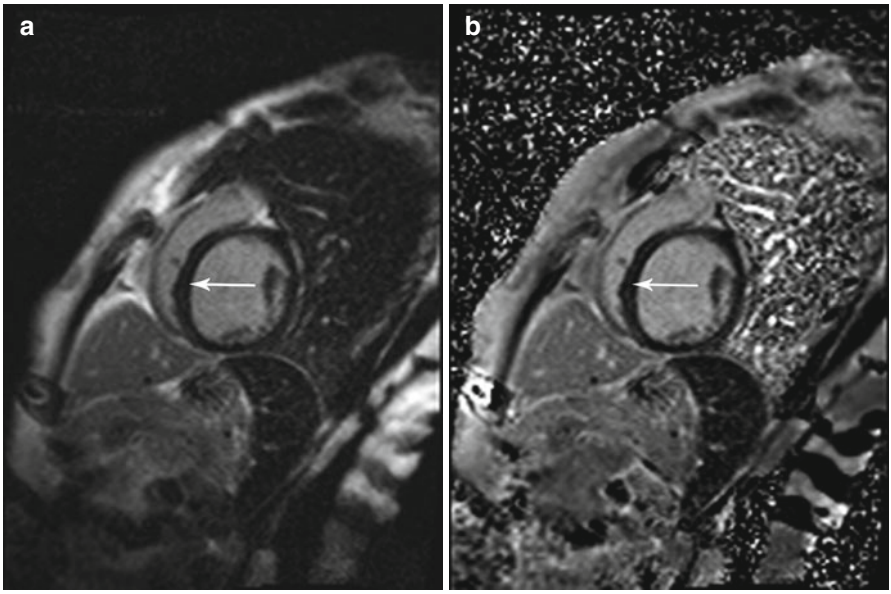


Fig. 7.8 Phase-sensitive inversion recovery (PSIR) single shot sequence for delayed enhancement imaging in the same subject as in Fig. 7.7. Typical midwall enhancement predominantly involving the septum (*arrow*). (**a**) Magnitude reconstruction and (**b**) phase-sensitive reconstruction

Differential Diagnosis

- *Restrictive cardiomyopathy* (see below; typically, isolated enlargement of both atria with normal ventricular size and diastolic dysfunction) (Mookadam et al. 2011)
- *Hypertrophic or hypertrophic obstructive cardiomyopathy* (see below; no dilatation but left ventricular wall thickening, predominantly of the basal interventricular septum) (De Smet et al. 2012)
- *Valvular diseases* (predominantly chronic reflux through the aortic or mitral valve, resulting in increased volume load and reduced systolic function) (Bonow et al. 2008)

Clinical Management

When dilated cardiomyopathy (DCM) with markedly reduced pump function is suspected, the subject should be informed and undergo further diagnostic workup. The ventricular size measured by MRI should be confirmed by echocardiography as a second imaging modality and vice versa. In addition, most patients subsequently require myocardial biopsy.

7.2.2.2 Hypertrophic and Hypertrophic Obstructive Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) and its obstructive form, hypertrophic obstructive cardiomyopathy (HOCM), are inherited heart diseases that run in families. They are characterized by left ventricular hypertrophy, predominantly of the anteroseptal wall segments near the base (70 % of cases) (Table 7.4). In the obstructive form, hypertrophy causes obstruction of the left ventricular outflow tract (Fig. 7.9). Atypical patterns of hypertrophy with involvement of midventricular and/or apical segments have been described as well (De Smet et al. 2012).

Electrocardiogram (ECG) data obtained in large cohorts indicate that sudden deaths in HCM are attributable to rapid ventricular tachycardia or ventricular fibrillation (Hoey et al. 2014).

MRI Features

- Asymmetric or symmetric thickening of left ventricular wall segments; septal involvement is very common.
- Reduced LV diastolic compliance (prolonged diastolic relaxation), in most cases caused by increased intracellular calcium.
- In HOCM, there is additional end-systolic narrowing of the left ventricular outflow tract (LVOT) due to asymmetric septal hypertrophy and a suction effect of the turbulent jet at the insufficient mitral valve during systole (Bernoulli effect) with systolic anterior motion (SAM) of the anterior mitral leaflet (Guarise et al. 2011).
- Diffuse or focal delayed enhancement in areas of myocardial fibrosis; can occur anywhere in the left ventricle and is not confined to the distribution of a single coronary artery, most commonly involves septal and anteroseptal segments (De Smet et al. 2012; Stork et al. 2007).
- The extent of delayed enhancement correlates with the risk of sudden cardiac death (Rickers et al. 2005; Rochitte et al. 2006).

Differential Diagnosis

- *Aortic stenosis* (thickening, calcification, or fusion of aortic valve leaflets, typically resulting in concentric rather than focal LV hypertrophy) (Proctor et al. 2011)
- *Restrictive cardiomyopathy* (see below; disease of unknown etiology or terminal stage of a variety of diseases, e.g., amyloidosis, sarcoidosis, hemochromatosis, damage caused by chemotherapy or radiotherapy, endocardial thickening with

Table 7.4 Hypertrophic and hypertrophic obstructive cardiomyopathy

Frequency	Up to 0.2 % of the population; ratio of HCM to HOCM of approx. 3:1
Cause	Usually hereditary
Clinical presentation	Uncharacteristic, often asymptomatic (incidental finding); some patients present with dyspnea, angina pectoris, and higher-grade ventricular arrhythmia or even ventricular tachycardia; most common cause of sudden cardiac death in competitive athletes If the critical heart weight of 500 g is exceeded, the resulting reduction in perfusion reduction can trigger small infarctions

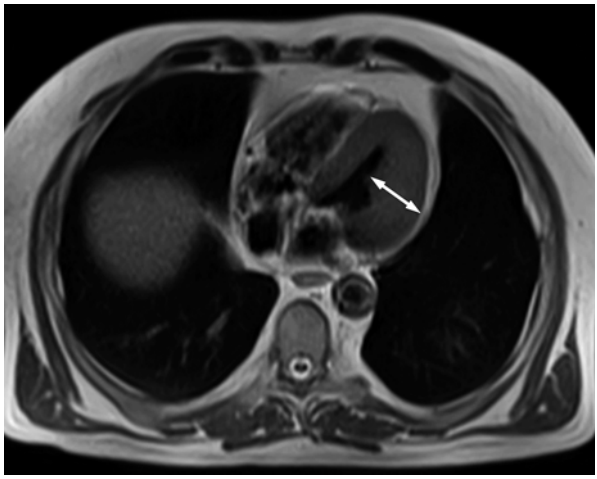


Fig. 7.9 Severe, more concentric, hypertrophy of the left ventricular wall on a T2-weighted HASTE image obtained in a 58-year-old subject (*arrow* indicates thickness of LV free wall)

deposits, thrombus formation and risk of embolism, and development of diastolic dysfunction) (Penugonda 2010)

- *Systemic arterial hypertension* (pressure overload typically results in concentric LV hypertrophy)

Clinical Management
If MRI detects focal or concentric LV hypertrophy, the finding should be communicated to the subject along with a recommendation to undergo further cardiac diagnostic evaluation including auscultation (HOCM is characterized by a late-onset systolic ejection murmur with a crescendo-decrescendo configuration, best heard at the apex and accentuated by physical activity or Valsalva maneuver, and the presence of a fourth heart sound), an electrocardiogram (to detect signs of LV hypertrophy), and an echocardiographic examination. In addition, a blood test should be performed. If these examinations provide no diagnosis, a left heart catheter examination should follow.

7.2.2.3 Restrictive Cardiomyopathy

Restrictive cardiomyopathy (RCM) is characterized by severe diastolic dysfunction due to endocardial thickening in the presence of normal ventricular systolic function (Mookadam et al. 2011). Typically, both atria are markedly dilated with backup of blood into the inferior vena cava and hepatic veins (Table 7.5). Sporadic and familial forms have been described (Maron et al. 2006).

One of the clinical challenges in patients with suspected RCM is its differentiation from pericardial constriction, which shares a similar clinical presentation but is treatable with pericardectomy. Cardiac MRI is helpful because it allows anatomic evaluation of pericardial thickening, assessment of the hemodynamic effects of constriction, and detection of abnormality of the underlying myocardium (Quarta et al. 2011).

Table 7.5 Restrictive cardiomyopathy

Frequency	Rare
Causes	Endocardial thickening with thrombus formation on the endocardium Chronic RCM with progressive endocardial fibrosis and diastolic ventricular dysfunction as well as development of therapy-refractory cardiac insufficiency with inflow congestion at the entrance to the right heart
Clinical presentation	Signs of cardiac failure including dyspnea, fatigue, edema, and pleural effusion; atrial and/or ventricular arrhythmia

MRI Features

- Small LV with normal wall thickness and normal LVEF
- Enlargement of both atria
- Abnormal diastolic filling
- Abnormal, typically patchy, delayed enhancement in secondary forms with myocardial involvement or inflammation (Stork et al. 2007)
- Normal pericardium (<3 mm) without adhesions, no abnormal contrast enhancement (Rochitte et al. 2006)

Differential Diagnosis

- *Constrictive pericarditis* (see below; pericardial thickening >4 mm with pericardial delayed enhancement seen in pericardial inflammation or fibrosis, which is not a feature of RCM. Constrictive pericarditis is also characterized by conspicuous ventricular filling obstruction during diastole; however, obstruction occurs secondary to pericardial inflammation and can be eliminated by surgical excision of pericardium (Francone et al. 2006; Hancock 2001)
- *Secondary cardiomyopathies with myocardial restriction* (myocardial involvement in systemic diseases such as amyloidosis, hemochromatosis, or sarcoidosis; after radiotherapy or chemotherapy; also characterized by patchy delayed enhancement (ACCF/ACR/AHA/NASCI/SCMR 2010))

Clinical Management

Subjects with suspected RCM should be referred to a cardiologist for further diagnostic evaluation including echocardiography, electrocardiography, CT, and possibly endomyocardial biopsy. The combined results of all of these modalities are usually necessary to establish the diagnosis of RCM.

7.2.2.4 Arrhythmogenic Right Ventricular Cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterized by fibrofatty replacement of myocardium on myocardial biopsy and localized aneurysms of the right ventricle. The pathogenesis is unknown, but it is assumed that myocyte loss is the result of generalized apoptosis.

In addition to structural changes, patients with ARVC also have functional impairment of the right ventricle due to dilatation, increased trabeculation, and

Table 7.6 Arrhythmogenic right ventricular cardiomyopathy

Frequency	1/5,000 population/year before age 30
Causes	Unknown, gene mutations have been identified (autosomal dominant inheritance)
Clinical presentation	Right heart failure, arrhythmia, syncopes, ventricular tachycardia, and even sudden cardiac death

regional wall motion abnormalities (dyskinesia) (Table 7.6). Moreover, the ECG demonstrates conduction abnormalities. ARVC is often demonstrated by autopsy or biopsy in patients with familial cardiomyopathy. In severe ARVC, the left ventricle may be affected as well (De Smet et al. 2012; Murphy et al. 2010).

MRI Features

- Regional RV wall motion abnormalities.
- RV dilatation for which no other cause – such as shunt defects, pulmonary hypertension, or tricuspid insufficiency – can be found.
- Microaneurysms in the RV (Ferrari et al. 2005).
- Increased RV trabeculation ("stack of dishes" sign) with thinning of the wall.
- On T1-weighted images (dark blood spin echo sequence), fatty dysplasia has high signal intensity (subepicardial, predominantly involving the lateral wall and the right ventricular outflow tract (RVOT) anteriorly) (Jain et al. 2008; Tandri et al. 2005).
- Delayed enhancement of RV areas of fibrofatty degeneration; the most common sites of involvement are the subtricuspid area, the RV apex, and the RVOT (triangle of dysplasia) (Ferrari et al. 2005; Stork et al. 2007).

Differential Diagnosis

- *Uhl disease* (anomaly of the right ventricle, characterized by near or complete absence of RV myocardium)
- *Valve defects* (RV dilatation caused by valve defects, e.g., tricuspid insufficiency, ASD, VSD)
- *Idiopathic right ventricular tachycardia* (absence of fibrofatty transformation of RV myocardium)

Clinical Management

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is rare.

When ARVC is suspected, the subject must be informed so that further diagnostic tests can be done.

Although not consistently present on an electrocardiogram, a characteristic epsilon wave (a terminal notch in the QRS complex) in leads V1–V3 is a sure diagnostic sign of the disease. Fibrofatty deposits in the right ventricular wall, which are often demonstrated by MRI, can be confirmed by myocardial biopsy.

7.2.2.5 Unclassified Cardiomyopathies

7.2.2.5.1 Takotsubo Cardiomyopathy

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy or broken heart syndrome, is characterized by sudden onset of transient akinesia or dyskinesia of the LV apex without significant coronary stenosis (>50 %). The left ventricular ejection fraction is reduced (Eitel et al. 2011).

Takotsubo cardiomyopathy is triggered by intense emotional stress or a traumatizing event. Findings include acute chest pain, reversible electrocardiogram changes, and elevated cardiac enzymes (Table 7.7).

MRI Features

- Akinesia or dyskinesia due to LV apical ballooning (Eitel et al. 2011).
- Delayed enhancement imaging usually reveals no signs of myocardial damage.
- Fat-saturated T2-weighted images may occasionally show myocardial edema (high signal intensity) (Celik et al. 2009; Eitel et al. 2011).

Differential Diagnosis

- *Acute myocardial infarction* (While patients also present with chest pain and dyspnea, cardiac MRI typically detects significant atherosclerotic plaques in the coronary arteries and demonstrates perfusion deficits, often with concomitant myocardial edema in affected areas (fat-saturated T2-weighted pulse sequence) (Abdel-Aty and Schulz-Menger 2007). Another characteristic of myocardial infarction is subendocardial or transmural delayed contrast enhancement in a vascular distribution.) (Perazzolo et al. 2011)

Table 7.7 Unclassified cardiomyopathies

Frequency	Altogether rare
Cause	Heterogeneous group of diseases of different etiologies Stress-induced, increased blood catecholamines (Takotsubo cardiomyopathy) Failure of myocardial development during embryogenesis due to gene mutation (noncompaction cardiomyopathy)
Clinical presentation	Takotsubo cardiomyopathy Symptoms resemble those of myocardial infarction, in particular angina pectoris and dyspnea Noncompaction cardiomyopathy Affected individuals remain asymptomatic for a long time, first diagnosed in adults; signs of cardiac insufficiency, tachyarrhythmia, sudden cardiac death, thromboembolism

- *Acute myocarditis* (see below; delayed enhancement tends to be subepicardial or to appear as diffuse enhancement of the entire myocardial wall not corresponding to a coronary artery territory. Fat-saturated T2-weighted images will show high-signal-intensity edema in 80 % of cases.) (Stensaeth et al. 2012)
- *Coronary vasospasms*

7.2.2.5.2 Noncompaction Cardiomyopathy

Noncompaction cardiomyopathy (NCCM) is an anomaly resulting from arrested myocardial development during embryogenesis. The failure of the initial meshwork of interwoven myocardial fibers to compact results in a myocardial wall with a thickened appearance due to the presence of prominent trabeculations and deep intertrabecular recesses, known as spongy myocardium. Noncompaction predominantly affects the apex (Yousef et al. 2009) (see Table 7.7 and Fig. 7.10). The left ventricular ejection fraction may be reduced. There is a risk of thrombus formation within the intertrabecular recesses, and arrhythmia may occur (De Smet et al. 2012).

MRI Features

- Prominent trabeculation with deep intertrabecular recesses of the LV mainly involving the apical and midventricular inferior and lateral segments.
- The abnormal trabeculations make the wall appear thickened, but due to the deep intertrabecular recesses, the wall is actually thinner than normal; contractility is preserved in most patients.
- An occasional patient has a dilated LV and reduced LVEF.
- Subendocardial perfusion defects or delayed enhancement may be observed in some patients (Petersen et al. 2005).

Differential Diagnosis

- *HCM or HOCM* (see above; primarily when there is apical involvement)
- *DCM* (see above; primarily when there is LV dilation and reduced pump function)
- *Secondary cardiomyopathy* (cardiomyopathy caused by a known medical condition, e.g., amyloidosis or sarcoidosis, or occurring secondary to radiotherapy or chemotherapy) (Penugonda 2010)

Clinical Management

Noncompaction cardiomyopathy is altogether rare and should be communicated especially if LV function is impaired.

Table 7.8 MRI features of the different cardiomyopathies

Parameter	DCM	HCM/HOCM	RCM	ARVC	NCCM
Atrial size	Typically both LA and RA are enlarged	Typically both LA and RA are enlarged	Typically both LA and RA are enlarged	LA of normal size, RA occasionally enlarged	LA and RA of normal size
Ventricular size	Enlargement of both LV and RV or of LV alone	LV and RV of normal size, rarely reduced in size	LV and RV of normal size	RV enlarged, LV usually of normal size	LV and RV typically of normal size, LV enlarged in rare cases
LV mass	LV normal or slightly hypertrophied; LV and RV mass normal or increased	Generalized hypertrophy of LV; septal hypertrophy, primarily apical, in 25 % of cases	LV and RV of normal size	RV wall involved at apex and inflow/outflow tract Wall thinning giving rise to microaneurysms Prominent trabeculation (stack of dishes) and fat deposition in RV wall	LV wall normal or thickened at apex and/or midventricle
LVEF	LVEF reduced	LVEF usually normal; may be increased in HOCM due to obstruction	LVEF normal	LVEF normal RVEF reduced	LVEF normal or reduced
Delayed enhancement	Atypical delayed enhancement (in 10–40 % of cases): intramural, subepicardial	Diffuse or confluent delayed enhancement; often patchy enhancement of septum in HOCM	Absent	Diffuse patchy delayed enhancement of RV (often difficult to evaluate)	Absent

Modified according to Hombach (2006)
LA left atrium, *RA* right atrium, *LV* left ventricle, *RV* right ventricle, *LVEF* left ventricular ejection fraction, *RVEF* right ventricular ejection fraction

Table 7.9 Acute and chronic myocarditis

Frequency	Cardiac involvement in approx. 1 % of patients with cardiotropic viral infection, approx. 4–5 % in Coxsackievirus infection
Causes	Infectious Viruses (50 % of cases), especially enteroviruses, e.g., Coxsackie B1-B5 viruses, but also influenza or herpes viruses Bacteria, especially staphylococci, enterococci Fungi, especially in immunocompromised individuals Protozoa, e.g., toxoplasmosis Parasites, e.g., trichina, echinococci Noninfectious Rheumatoid arthritis, connective tissue diseases, vasculitis, mediastinal radiotherapy, hypersensitivity myocarditis caused by certain medications
Clinical presentation	Highly variable, from asymptomatic or mild forms (common) to fulminant fatal disease (rare) Infectious myocarditis presents with fatigue, feeling of weakness, fever, tachycardia, arrhythmia (especially premature beats), and signs of cardiac insufficiency

7.2.3 Acute and Chronic Myocarditis

Myocarditis is an acute or chronic recurrent inflammatory process of the myocardium, which may show focal or diffuse involvement (Table 7.9). It can result in necrosis and myocyte loss.

Chronic myocarditis can progress to dilated cardiomyopathy (De Smet et al. 2012; Ling et al. 1999).

Because of the nonspecificity of its symptoms, signs, and test findings, myocarditis is often diagnosed by exclusion of other cardiac diseases (Friedrich and Marcotte 2013).

When performed as part of a whole-body imaging protocol, the examination is less comprehensive than a dedicated MRI study of the heart. The whole-body protocol used in the SHIP does not include a fat-saturated T2-weighted pulse sequence for edema imaging, precluding the detection of chronic or acute myocarditis. Cine sequences typically reveal systolic dysfunction in the presence of generalized hypokinesia; less commonly they reveal diastolic dysfunction. However, this findings are not specific for myocarditis and may also be seen in DCM and other cardiac diseases.

Most patients with myocarditis have concomitant relative mitral insufficiency due to mitral ring dilatation. Isolated pericardial effusion may also point to an inflammatory condition of the myocardium (Ong et al. 2011).

MRI Features

- On fat-saturated T2-weighted images, inflamed myocardium has high signal intensity (edema); areas of inflamed myocardium cannot be identified without acquisition of fat-saturated T2-weighted images; on the other hand, even these sequences will detect edema in only 80 % of cases (Mahrholdt et al. 2006).

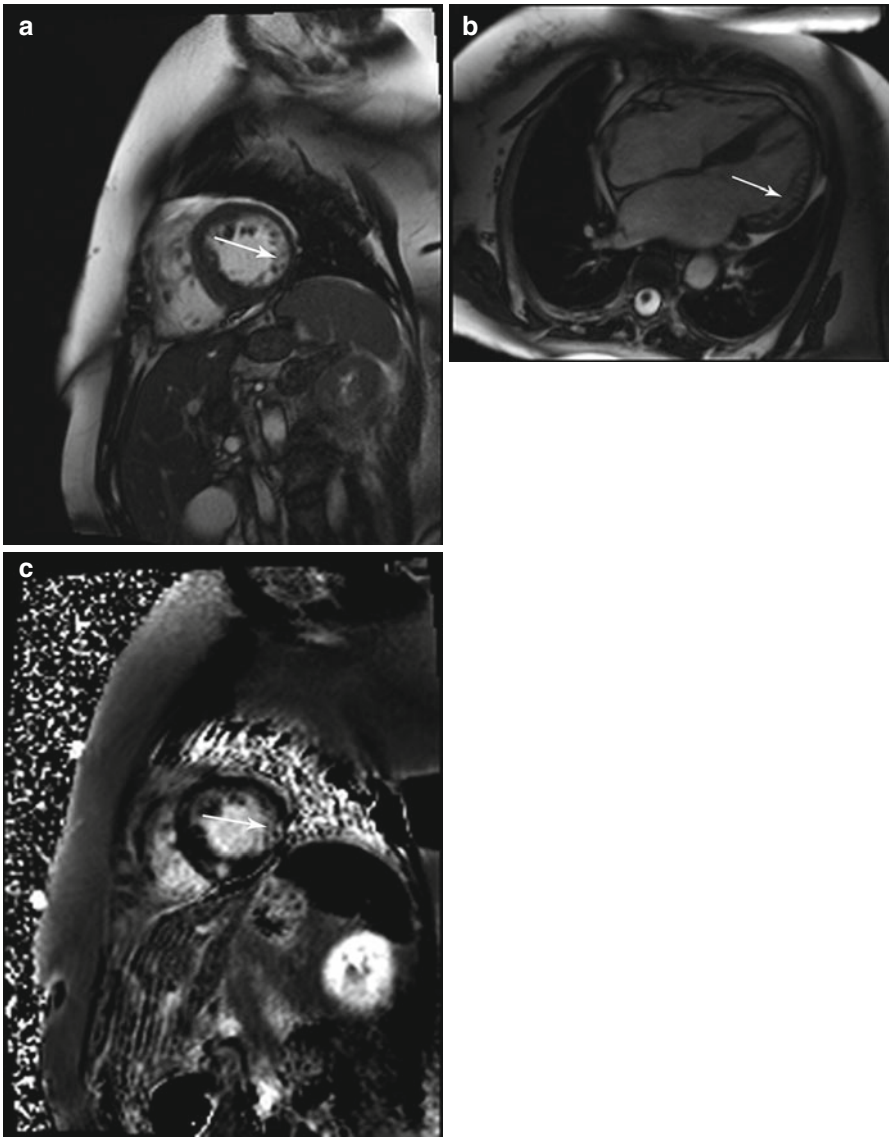


Fig. 7.10 Noncompaction cardiomyopathy in a 58-year-old subject. (**a**) Short-axis cine view. Prominent trabeculation of anterior and lateral wall segments of the left ventricle (*arrow*). (**b**) Four-chamber cine view. Spongy myocardium with deep intratrabecular spaces is apparent (*arrow*). (**c**) Phase-sensitive inversion recovery (PSIR) image showing atypical patchy delayed enhancement (*arrow*)

- T1-weighted imaging after contrast medium administration enables calculation of an enhancement ratio of inflamed myocardium to skeletal muscle; an enhancement ratio >4.0 is highly indicative of acute myocarditis (the normal ratio is <2.5) (Friedrich et al. 1998; Stensaeth et al. 2012).
- On delayed enhancement images, acute myocarditis typically shows subepicardial enhancement in the inferolateral wall, predominantly in the midventricular portion (Goitein et al. 2009). However, some patients may show diffuse delayed enhancement that includes subendocardial segments and some may show no delayed enhancement at all (De Smet et al. 2012; Gahide et al. 2010).
- Nevertheless, it is usually possible to differentiate myocarditis from myocardial infarction, which is characterized by subendocardial delayed enhancement confined to a vascular territory (Stensaeth et al. 2012; Stork et al. 2007).

Differential Diagnosis

- *Ischemic cardiomyopathy* (subendocardial delayed enhancement in the distribution of a coronary artery)
- *Nonischemic dilated cardiomyopathy* (typical midwall enhancement primarily involving the septum)
- *Other forms of cardiomyopathy* (nonspecific patchy delayed enhancement)

Clinical Management

Pericardial effusion is not an uncommon finding in whole-body screening. It should be communicated to the subject if the width of the effusion is 1 cm or more or if the perfusion is hemodynamically relevant.

Echocardiography allows rapid and sensitive confirmation of pericardial effusion (when 50 mL or more is present).

7.3 Diseases of Pericardium

Cardiac MRI is well suited for evaluating conditions affecting the pericardium such as inflammatory processes or thickening, lesions such as cysts, calcification, and pericardial masses or malignant tumors. Because of its three-dimensional capabilities, MRI enables complete evaluation of the heart and surrounding anatomy (Bogaert and Francone 2013).

Fat-saturated T2-weighted pulse sequences reliably identify inflammatory processes of the myocardium and pericardium. In most cases, pericardial effusion is already visible on cine images. Cardiac MRI using a combination of different T1-weighted and T2-weighted pulse sequences even enables identification of the type of fluid present (transudate, exudate, hemorrhagic effusion) (Abbara and Miller 2005).

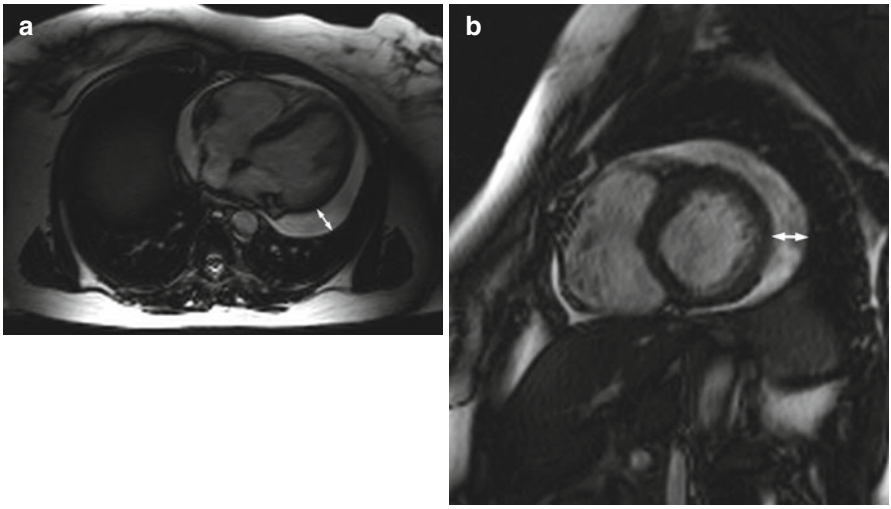


Fig. 7.11 Pericardial effusion in a 48-year-old subject. (**a**) Strict axial cine image (**b**) short-axis cine image (*arrow* indicates width of pericardial effusion near the base)

7.3.1 Pericardial Effusion

Pericardial effusion is an abnormal fluid collection in the pericardial space (Fig. 7.11). Excess fluid collects in the pericardial space for a variety of reasons including inflammatory and infectious processes, cardiac congestion, and tumors (Ong et al. 2011).

MRI Features

- Transudate has low T1 signal intensity and high T2 signal intensity.
- Exudate, due to its high protein content, has moderate to high signal intensity on T1-weighted images and low signal intensity on T2-weighted images.
- Hemorrhagic effusion has high T1 signal intensity, especially when fresh blood is present, while it is iso- to hyperintense with T2 weighting.
- LV and RV function is usually normal as long as the fluid collection has no hemodynamic effects.
- Large pericardial fluid collections impair diastolic filling (Hombach 2006).

Differential Diagnosis

- *Myocarditis* (see above; edema confined to affected wall segments, high signal intensity on fat-saturated T2-weighted sequences; subepicardial or patchy nonspecific pattern of delayed enhancement) (Stensaeth et al. 2012)
- *Acute myocardial infarction* (typical pain pattern, edema on fat-saturated T2-weighted images, subendocardial delayed enhancement) (Perazzolo et al. 2011)
- *Cardiac disease of other etiology* (e.g., rheumatic disease, usually asymptomatic)
- *Pericarditis* (see below; typically with pericardial edema, high signal intensity on fat-saturated T2-weighted images, pericardial thickness usually >4 mm, pericardial delayed enhancement) (Goyle and Walling 2002)

Table 7.10 Pericarditis

Frequency	Cardiac involvement in approx. 1 % of patients with cardiotropic viral infection, approx. 4–5 % in Coxsackievirus infection
Causes	Infectious Viruses are the most common infectious agents; the pathogen spectrum is the same as in myocarditis Immunologic Systemic lupus erythematosus and rheumatic fever, postmyocardial infarction syndrome/Dressler syndrome (febrile pericarditis/pleuritis 1–6 weeks after myocardial infarction or cardiac surgery), uremia, tumors, radiotherapy
Clinical presentation	Typically, sharp chest pain, often located behind the sternum, which intensifies with deep inspiration/coughing or when lying down Pericardial friction rub on auscultation Electrocardiogram in acute pericarditis: concave upward ST segment elevation originating from S wave; transition to a negative T wave with disease progression. Absence of loss of R wave allows differentiation from myocardial infarction!

7.3.2 Pericarditis

Pericarditis has infectious and noninfectious causes. Infectious pericarditis is caused by a variety of viral and bacterial agents. Noninfectious pericarditis can develop after surgery or after myocardial infarction (Table 7.10). Pericarditis presents with fever, coughing, and other general symptoms of inflammation. Most patients report retrosternal chest pain (Abbara and Miller 2005).

MRI Features

- Pericardial thickening ≥4 mm (normal 1–3 mm).
- Cine sequences reveal no wall motion abnormalities; most patients have normal LV and RV function.
- Fat-saturated T2-weighted sequences depict high-signal-intensity pericardium; a reliable diagnosis of pericarditis or inflammatory pericardial involvement cannot be made without use of a fat-saturated T2-weighted sequence (Friedrich et al. 1998; Mahrholdt et al. 2006).
- Global delayed enhancement of the pericardium (Goyle and Walling 2002).

Differential Diagnosis

- *Cardiac disease of other etiology* (e.g., rheumatic disease, usually asymptomatic)
- *Acute myocardial infarction* (typical pain pattern, edema on fat-saturated T2-weighted images, subendocardial delayed enhancement) (Perazzolo et al. 2011)
- *Myocarditis* (see above; edema confined to affected wall segments; high signal intensity on fat-saturated T2-weighted sequences; subepicardial or patchy nonspecific pattern of delayed enhancement) (Stensaeth et al. 2012)

Table 7.11 Constrictive pericarditis

Frequency	Idiopathic (33 %); secondary to pericarditis or infection (19 %); mechanical, e.g., after trauma or cardiac surgery (18 %); after radiotherapy (13 %, e.g., for breast cancer, Hodgkin disease); metabolic (uremia); in association with rheumatic diseases
Causes	Abnormal pericardial thickening with fibrosis and calcification secondary to one of the conditions listed above, most severe after tuberculosis
Clinical presentation	Symptoms secondary to right ventricular inflow obstruction (right heart failure, dyspnea, hepatomegaly with ascites, edema, congestive proteinemia) Low-cardiac-output syndrome with physical weakness and dyspnea on exertion

Clinical Management

A subject with a suspected myocardial or pericardial condition should see a cardiologist for further examinations including an electrocardiogram (possibly long-term) and echocardiography. Laboratory testing is required and should cover cardiac enzymes (CK/CK-MB and troponin) and inflammatory parameters (BSR, blood count, CRP). The initial laboratory work should be followed by special bacteriologic/virologic tests (fecal enteroviruses, antibody titer, etc.).

7.3.3 Constrictive Pericarditis

Constrictive pericarditis results from fibrotic thickening of the pericardium secondary to pericarditis, cardiac surgery, trauma, radiotherapy, or chemotherapy (Table 7.11).

Fibrosis and calcification of the pericardium can lead to the development of a rigid shell around the heart. The shell impairs expansion of the heart, resulting in inadequate filling of the ventricles during diastole. This is seen as inflow obstruction. Patients with longer-standing constrictive pericarditis develop myocardial atrophy (Ling et al. 1999).

If the heart size is normal but clinical signs of right-sided heart failure are present, this should alert the physician to the possibility of constrictive pericarditis. Surgical pericardiectomy is potentially curative in constrictive pericarditis (Mookadam et al. 2011).

MRI Features

- Pericardial thickening ≥4 mm.
- Intrapericardial calcification will be seen as areas of very low signal intensity.
- Restricted diastolic filling with secondary dilatation of the right atrium and congestion of blood at the entrance to the right heart.
- Clinical signs of right heart failure (Francone et al. 2006)
- Pericardial delayed enhancement seen in pericardial inflammation or fibrosis.

Differential Diagnosis

- *Restrictive cardiomyopathy* (typically no pericardial thickening, no delayed enhancement) (Hancock 2001; Cheng et al. 2011)
- *Right heart failure of other etiology*

Clinical Management
Hemodynamically significant valve defects should be communicated.

7.4 Diseases of Endocardium

The endocardium lines the cardiac chambers and covers the cardiac valves (Bonow et al. 2008). Infectious endocarditis remains a diagnostic and therapeutic challenge. Patient outcome depends on a rapid diagnosis and accurate risk stratification (Bruun et al. 2014).

7.4.1 Valvular Disease

Most acquired abnormalities of the cardiac valves occur secondary to endocarditis, typically appearing several years later. Acquired valvular disease predominantly affects the left heart (mitral/aortic valves) because it is subject to greater mechanical stress and pressure, causing the valves to become incompetent. The severity of valve incompetence varies with the presence of prior cardiac damage, virulence of the pathogen involved, and the patient's immune status (Bonow et al. 2008; Morris et al. 2010) (see Table 7.12 and Fig. 7.12).

Other causes of valvular disease are myocardial ischemia and cardiomyopathies.

Ischemic damage of the myocardium and papillary muscles can compromise the valves. In dilated cardiomyopathy, incomplete closure of leaflets may result when the left ventricle is markedly dilated.

Cine imaging allows visual evaluation of the jet across the valve, but this is not a definitive diagnostic test and can only suggest the possibility of valve incompetence. With the whole-body MRI protocol used here, it is not possible to grade the severity of stenosis or valve incompetence. Grading requires measurement of flow velocities across the valve. However, with adjustments to the protocol, MRI can provide quantitative and reproducible information on valvular stenosis and insufficiency. In addition, MRI allows detection of dilatation or narrowing of the major arteries such as the aortic root or ascending aorta (D'Arcy and Myerson 2010).

With its poorer temporal resolution compared with Doppler echocardiography, MRI underestimates the peak gradient. In determining the mean pressure gradient, however, there is good agreement between the two modalities. Overall, MRI only enables incomplete valvular assessment and is markedly inferior to echocardiography.

Table 7.12 Diseases of endocardium

Frequency	Infectious causes are most common: 5–10/100,000 population/year
Causes	Infectious (a) Acute (bacterial infection) (b) Subacute (endocarditis lenta) (c) Viral or mycotic Noninfectious (a) Rheumatic endocarditis = complication of acute fever, in which a rheumatic immune reaction to streptococci produces an autoimmune attack against elements of the endocardium (antigen-antibody reaction) (b) Nonbacterial thrombotic endocarditis = vegetations on mitral and aortic valves occurring in the terminal stages of severe chronic diseases (e.g., cancer) (c) Libman-Sacks endocarditis = associated with systemic lupus erythematosus; presence of fibrin vegetations on cardiac valves and thickening of chordae tendineae; often with concomitant pericarditis or pleuritis (aggregation of immune complexes) (d) Eosinophilic endocarditis (Löffler syndrome) = accumulation of eosinophils in the lung and other organs (e.g., heart, brain, gastrointestinal tract) due to an allergic drug reaction or in response to parasitic or fungal infection; often also idiopathic Endocardial fibrosis
Clinical presentation	Acute onset: may present with sepsis (fever and chills); rapid progression to acute cardiac failure due to valve dysfunction Subacute onset: weight loss, fatigue, subfebrile temperatures, night sweat

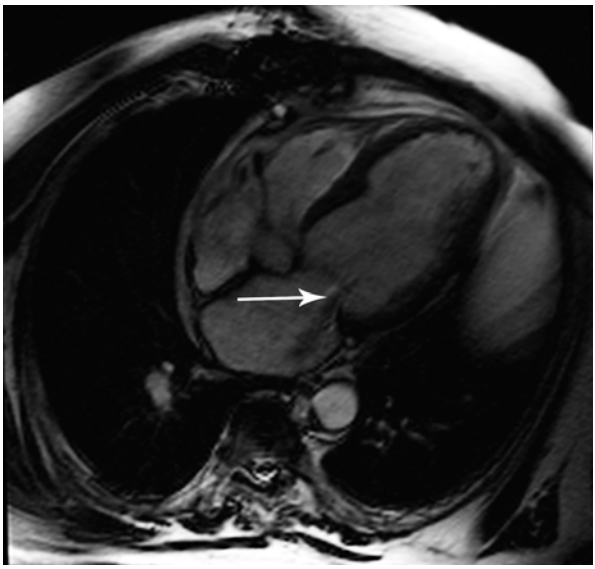


Fig. 7.12 Marked regurgitation through the mitral valve (*arrow*) in a 63-year-old subject (four-chamber cine view)

With MRI, the mitral, aortic, and tricuspid valves are depicted. The most common valvular abnormality encountered in the setting of whole-body MRI is a jet across the mitral valve. In most instances, the jet has no hemodynamic relevance (Bonow et al. 2008).

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