57. Cardiomyopathy

Motion from the beating heart poses a particular challenge to acquiring high quality MR images, one often solved by electrocardiographic (ECG) or pulse synchronization based on prospective triggering or retrospective gating. In prospective triggering, image acquisition is triggered by the R wave of the ECG or the peak of the pulse wave, allowing for imaging during relatively quiescent diastole. In retrospective gating MR images are acquired along with an electrocardiogram or a pulse curve, allowing visualization of the heart within the context of the cardiac cycle. Normal motion from respiration is also problematic, although breath holding may be employed. The short acquisition time of the steady-state free precession (SSFP) sequence makes it ideal for evaluation of wall motion, cardiac function and blood flow via cine images. On these intermediately T1 and T2-weighted sequences, blood demonstrates high SI, and thus SSFP is also known as a bright blood sequence. In distinction, prospectively triggered sequences obtained during diastole are best utilized for evaluation of peri- and myocardial morphology. Flowing blood within the cardiac chambers and great vessels appears as signal void, and these sequences are thus known as black blood sequences. With ultrafast gradient echo sequences first pass perfusion imaging after application of contrast media is feasible and may depict reduced myocardial perfusion both at rest or during physical or pharmacological stress. Myocardial SI may be suppressed by applying a preliminary inversion recovery (IR) or phase-sensitive inversion recovery (PSIR) pulse scheme. Combining this with CE T1-weighted sequences allows for assessment of myocardial viability. Residual enhancement after 10-15 minutes implies myocardium with inflammation or nonviable tissue, consistent with myocardial scarring (i.e. fibrosis). Typical imaging planes in cardiac MR include short axis (Fig. 57.1A,B), three-chamber (Fig. 57.1C), and horizontal long axis or four chamber (Fig. 57.1D) views. MR is frequently utilized to distinguish constrictive pericarditis, which warrants surgical treatment, from restrictive cardiomyopathy, which is managed medically. The normal parietal pericardium is best seen during systole on T1WI as a low SI band less than 2 mm in thickness. The pericardium is often incompletely visualized in MR. This makes diagnosing agenesis difficult, although a posteriorly, leftward shifted heart is suggestive. The short-axis, CE IR T1WI in Fig. 57.1A demonstrates a thickened (> 4 mm) pericardium, enhancing superiorly and inferiorly with a small effusion posteriorly. As evident here, the motion-induced flow voids of simple effusive fluid lead to a low SI on SE T1WI. Such effusions demonstrate high SI on cine sequences. In restrictive cardiomyopathy, the pericardium is seldom thickened, while the atrium and right-sided venous structures may be dilated based on increased filling pressure of the left and right ventricular chambers.

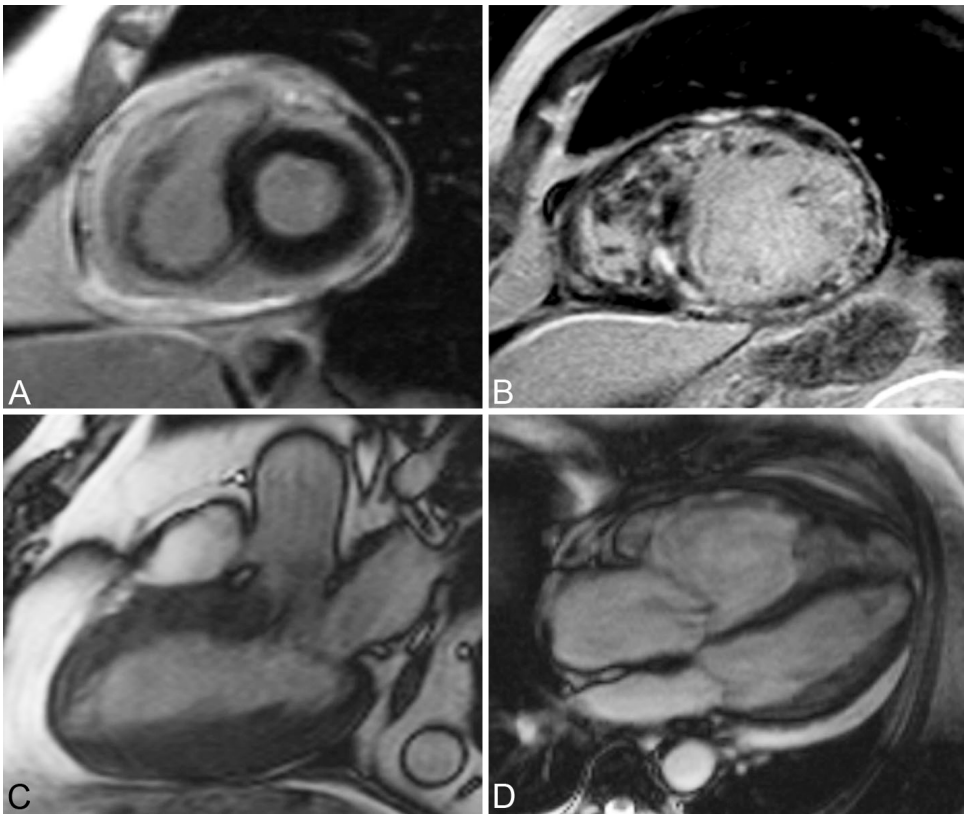

Fig. 57.1

Endomyocardial fibrosis and amyloidosis are two potential causes of restrictive cardiomyopathy. The cardiac wall may enhance in both. The myocardium in amyloidosis often demonstrates low SI on T1 and T2-weighted sequences and shows extensive and heterogenous late gadolinium enhancement (LGE). Dilated cardiomyopathy may also be detected on MR, thinning the walls of both ventricles, despite an overall increased myocardial mass. Asymmetric wall thinning in combination with subendocardial or transmural late gadolinium enhancement are typical findings of myocardial infarction as the underlying cause. Myocarditis-related fibrosis may also cause dilated cardiomyopathy. Acute (usually viral) myocarditis is demonstrated in the short axis CE IR images of Fig. 57.1B. Here, patchy myocardial enhancement involves the subepicardium and mid myocardium as opposed to the subendocardial enhancement pattern in ischemia. Dilated cardiomyopathy from hemochromatosis is distinguished by its low SI appearance on T1 and T2WI due to susceptibility effects from iron deposition. Hypertrophic cardiomyopathy is a potential cause of sudden death and is best assessed on three-chamber (inflow-outflow) views as in Fig. 57.1C. Here cine SSFP sequences show marked hypertrophy of the left ventricular myocardium involving the interventricular septum and significantly constricting the outflow of blood from the left ventricle. This turbulent flow through this narrowed left ventricular outlet manifests as a low SI jet. Septal thickness greater than 1.5 times that of

the posterolateral wall is diagnostic. In severe cases, mid myocardial or subepicardial enhancement may be present in hypertrophic areas indicating fibrosis or ischemic injury. Similar fibrotic or fatty infiltration also occurs in arrhythmogenic right ventricular dysplasia. In this disorder, myocardial high SI fat from the epicardial surface may displace lower SI myocardial fibers within the right ventricle. Pathognomonic for this condition and demonstrated in the four-chamber view cine SSFP sequence in Fig. 57.1D are aneurysmatic dilatations within the "dysplastic triangle" consisting of the right ventricle inflow-outflow tracts and its apex. The right ventricle is markedly dilated as well, and a low SI jet is located at its junction with the atria, representing tricuspid valve regurgitation. A final entity warranting mention is myocardial noncompaction of the ventricle—a congenital cardiomyopathy, characterized by a thickness ratio greater than 2.3 at end diastole between noncompacted, trabeculated, endocardial myocardium and compacted, epicardial myocardium.