

59. Cardiac Function and Valvular Heart Disease

The MR evaluation of cardiac chambers and valves typically encompasses the assessment of volumes and flow velocities. For quantification of ventricular size and function typically a stack of parallel short axis cine sequences is acquired covering the cardiac chambers from base to apex. Dedicated post-processing software aids to segment the blood-myocardium-interface in end diastole and end systole. Additionally acquired 2- and 4-chamber view sequences help to delineate the atrioventricular valve planes and to display the longitudinal strain of the cardiac chambers. Based on this planimetry, volumes of cardiac chambers and myocardium at end systole and end diastole can be obtained and subsequently parameters such as ejection fraction, stroke volume or cardiac output can be calculated thereof.

Regurgitant flow or valve pressure gradients may be obtained with velocity-encoded MRI. In this sequence, a gradient is applied in the expected direction of blood flow and phase (as well as magnitude) information acquired. Velocity of protons within a given voxel is obtainable based on proportionality between velocity and degree of phase change. Flow volume may be calculated by multiplying velocity by the cross-sectional area (acquired from magnitude information). Integration of flow volume over the cardiac cycle yields the total ejection volume. Comparing the ratio of retrograde to anterograde flow over the cardiac cycle yields a regurgitant fraction, while pressure gradients may be estimated from peak flow rates across a valve.

The morphology of the typically low SI cardiac valves is not easily assessed on MR due to their small size, rapidity of movement, and surrounding (low SI) turbulent flow. The normal tricuspid aortic valve—insufficiency of which was shown in Fig. 57.1D—normally lacks a significant pressure gradient when open and exhibits a total area of 3-4 cm². Stenosis is graded by surface area as severe (< 1 cm²), moderate (between 1 and 1.5 cm²), and mild (> 1.5 cm² but with a measurable pressure gradient). A pressure gradient resulting from stenosis can be obtained by applying the Bernoulli equation ($\Delta \text{ pressure} = 4 \times \text{peak velocity}^2$) to peak velocity values obtained by velocity-encoded MRI. In Fig. 59.1A a low SI jet, correlating with rapidly dephasing protons in this area of turbulent flow, extends across the stenotic left ventricular outlet into the aorta, consistent with aortic stenosis. In this patient with rheumatic valve disease, a similar jet is seen extending across the closed but regurgitant mitral valve into the left atrium. Mitral insufficiency may also be caused by prolapse, perforation, or annular dilatation with myxomatous degeneration constituting the most common underlying etiology. Nonbacterial vegetations of Libman-Sack's endocarditis are occasionally seen. If possible, valve morphology and root size should be assessed. In prolapse, identification of a culprit leaflet is crucial, as a solitary dysfunction is repairable

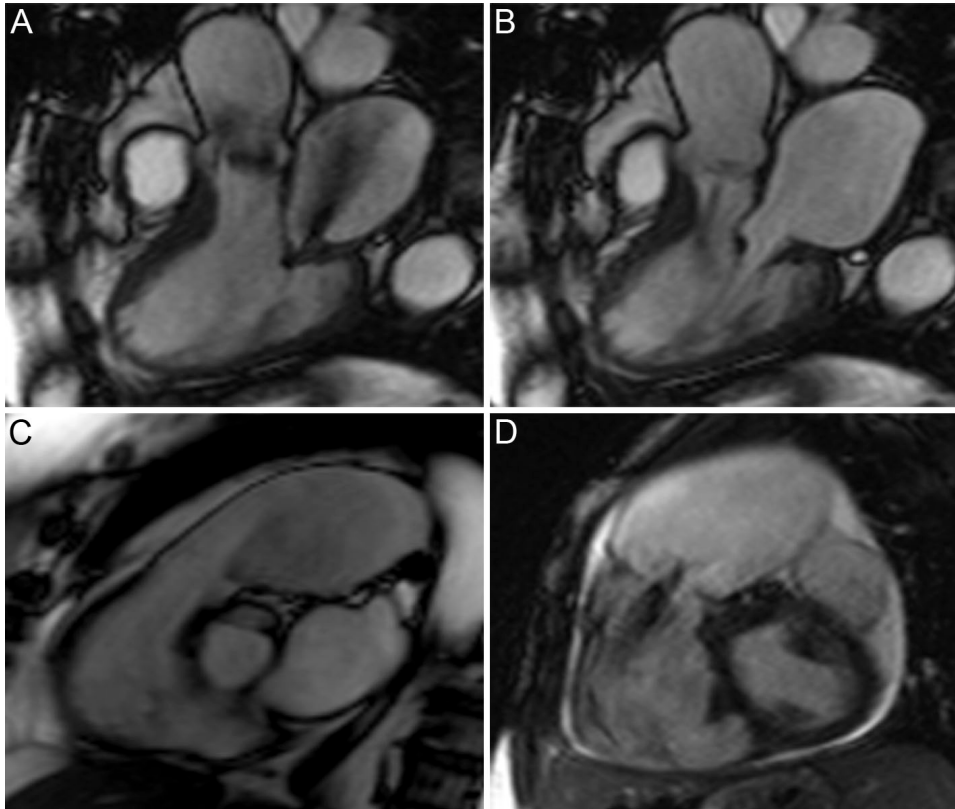


Fig. 59.1

without full surgical replacement. Quantification of regurgitation severity, ejection fraction, and chamber dimensions should be made. In isolated mitral regurgitation, a regurgitant fraction can be calculated by (in addition to previously described methods) computing the difference in ejection fraction between the right ventricle, which is elevated secondary to systolic ejection of the regurgitant volume, and left ventricle. Mitral stenosis is rare and seen almost exclusively with rheumatic disease in which leaflet scarring limits motion. The area of the mitral valve is typically between 4-5 cm², and the valve normally lacks a pressure gradient when open. An open, stenotic mitral valve is shown in the diastolic three-chamber cine images of Fig. 59.1B. Significantly increased pressures across the valve may be transmitted retrograde, resulting in dilation of the left atrium, as also seen in the figure. Aortic insufficiency is concurrently present, the regurgitation correlating with a low SI diastolic jet crossing the left ventricle into the aorta. Possible etiologies include endocarditis, rheumatic disease, congenital valve defects, and Marfan's syndrome. Quantitative flow calculations should not be performed too close to the aortic valve, as retrograde flow filling the coronary arteries naturally occurs during diastole. An ejection fraction less than 45% or left ventricular end diastolic diameter greater than 55 mm warrants surgical intervention. Pulmonic valve stenosis is often congenital, as in the tetralogy of Fallot (see Chapter 60). Moderate and severe disease are defined by valve

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pressures greater than 50 and 75 mmHg, respectively. The cine right-ventricular outflow systolic view of Fig. 59.1C demonstrates a stenotic right ventricular outlet with marked dilatation of the trunk and turbulent low SI flow present therein. While some degree of pulmonary regurgitation is considered normal on echocardiogram, hemodynamically significant lesions may occur secondary to pulmonary hypertension or as a result of a prior valvotomy to correct pulmonary stenosis. Clearly regurgitant flow across an insufficient pulmonary valve is visible as a low SI jet on the diastolic cine image of Fig. 59.1D with marked accompanying dilatation of the pulmonary trunk. When regurgitation is suspected to have resulted from postoperative valvotomy, MR evaluation is often useful.