

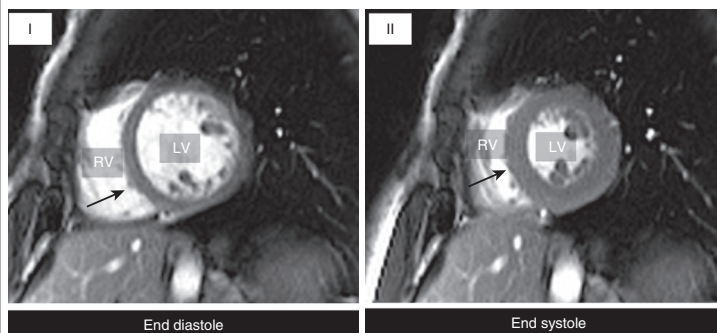


Abnormal Motion Patterns of the Interventricular Septum

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THE INTERVENTRICULAR SEPTUM (IVS) IS A COMMON WALL SHARED BY THE LEFT VENTRICLE (LV) AND the right ventricle (RV). Functionally, the IVS normally behaves like a wall of the LV, with the RV attached to its side, thus maintaining a concave curvature (toward the LV) throughout the cardiac cycle. Normal motion of the IVS is an inward motion toward the left during systole and an outward motion toward the right during diastole. The net septal motion is determined by the pressure on the right and left sides of the IVS, as well as the active tension developed within the wall of the IVS and the intrinsic stiffness of the septal tissue. Variations of the normal septal motion are often termed “paradoxical” septal motion. However, because there are many different variations of the septal motion, this generic term is very broad. **Table 1** is a list of causes of abnormal IVS motion (1,2). We have here gathered representative examples of some of the different types of IVS motion patterns to illustrate the differences between them. Although the images presented were acquired with cardiac

FIGURE 1 Normal Motion of the Interventricular Septum



The interventricular septum (IVS) (**arrow**) moves inward toward the left ventricle (LV) from end-diastole (I) until end-systole (II) and then moves back outward toward the right ventricle (RV) during diastole. In a normal heart, post-systolic shortening may be noted whereby the IVS moves normally toward the LV in systole, and then in early diastole, it initially moves normally toward the RV; however, in mid-diastole it briefly moves back toward the LV and then finally moves back normally toward the RV in late diastole ([Online Video 1](#)).

TABLE 1 Some Causes of Abnormal Interventricular Septal Motion

Right ventricular pressure or volume overload
• Pulmonary hypertension • Pulmonary insufficiency
Cardiomyopathy (intrinsic muscle disease)
• Ischemic heart disease • Infiltrative disease (amyloid, Fabry disease) • Hypertrophic cardiomyopathy • Left ventricular hypertrophy
Abnormal electrical activation
• Left bundle branch block • Right bundle branch block • Paced rhythm
Post-surgical status
Pericardial disease
• Constrictive pericarditis
Congenital heart disease
• Ebstein anomaly • Ventricular septal defect • Transposition of great arteries • Septal aneurysm
Post-systolic shortening
Mass in the interventricular septum

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Manuscript received May 5, 2017; revised manuscript received June 8, 2017, accepted June 13, 2017.

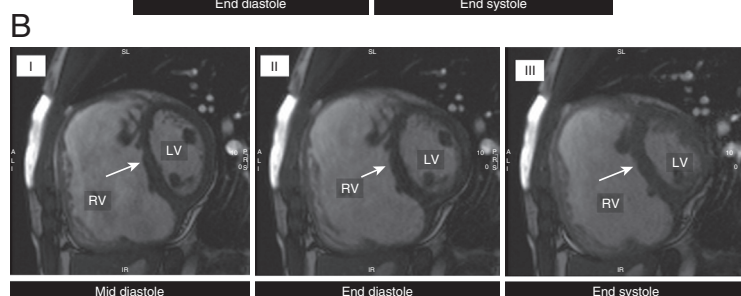
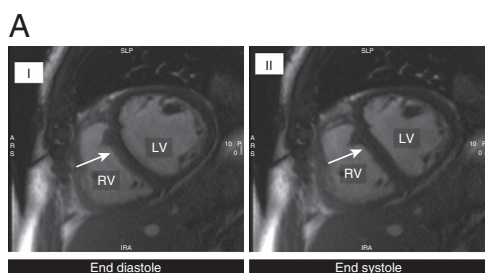


FIGURE 2 Abnormal Septal Motion Secondary to Altered Trans-Septal Loading Conditions

(A) Abnormal septal motion from RV pressure overload (secondary to pulmonary hypertension). A 40-year-old man with no significant past medical history presented with congestive heart failure, and a transthoracic echocardiogram demonstrated evidence of biventricular dysfunction. Cardiac magnetic resonance (CMR) was performed to further evaluate the cause of congestive heart failure. The CMR demonstrates an enlarged RV and LV with severely decreased biventricular function. There was also evidence of multiple pulmonary emboli associated with pulmonary hypertension leading to RV pressure overload. Demonstrated in the image is flattening of the IVS (**arrows**) during diastole (I), becoming more pronounced during systole (II). Also noted is a transient leftward motion of the IVS at end-systole as a result of a drop in the LV pressure before the RV pressure ([Online Video 2](#)). **(B)** Abnormal septal motion from RV volume overload (secondary to left-to-right shunt). A 21-year-old man with no significant past medical history presented with dyspnea and dizziness on exertion. A transthoracic echocardiogram demonstrated severe right atrial and right ventricular dilation. CMR was performed to further evaluate the cause of right-sided heart dilation. The CMR demonstrates severe RV dilation with normal function. The LV is normal in size and function. A large inferior sinus venosus atrial septal defect was identified as the cause of RV volume overload. The IVS (**arrows**) progressively flattens in mid- to end-diastole (I and II), moving abnormally toward the LV, losing the normal curvature. In systole (III), the IVS moves normally toward the LV and regains the normal curvature. See [Online Video 3](#). Abbreviations as in [Figure 1](#).

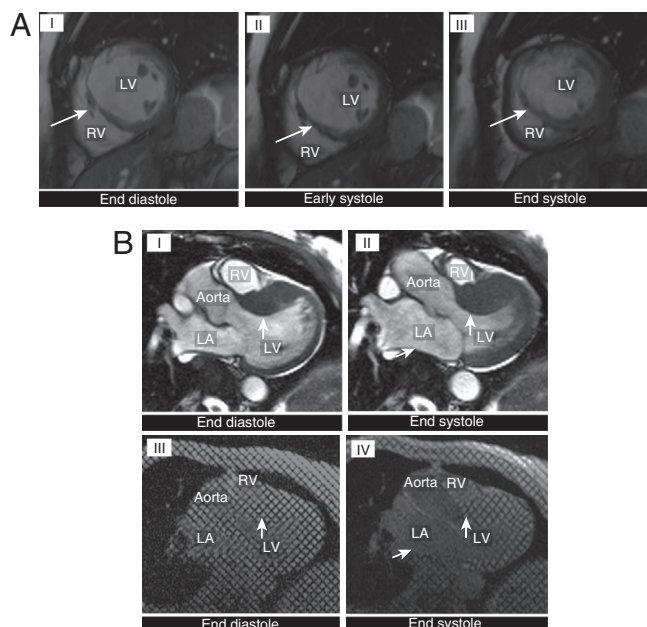


FIGURE 3 Abnormal Septal Motion Secondary to Altered State of the Myocardium

(A) Abnormal septal motion from ischemic cardiomyopathy (from infarction of the left anterior descending artery). A 42-year-old woman with a history of stroke, who presented with chest pain, had a diagnosis of non-ST-segment elevation myocardial infarction. Cardiac catheterization and coronary angiography demonstrated occlusion of the mid-left anterior descending artery that was treated with percutaneous coronary intervention. An echocardiogram demonstrated an ejection fraction of 50%. CMR was performed to evaluate for myocardial scar because of significant, ongoing, nonsustained ventricular tachycardia after revascularization. The CMR demonstrates a dilated LV with thinning of the midlevel anterior and anteroseptal segments. In diastole (I), the IVS (**arrows**) moves normally outward toward the RV; however, in systole (II and III), the anterior IVS fails to move toward the LV ([Online Video 4](#)). **(B)** Abnormal septal motion from hypertrophic cardiomyopathy (with increased intrinsic stiffness of the septal tissue). A 56-year-old woman with a past medical history of hypertrophic cardiomyopathy, who presented for risk stratification of sudden cardiac death, underwent CMR to evaluate the septal thickness, as well as to detect any evidence of late gadolinium enhancement. The CMR demonstrates asymmetrical septal hypertrophy and a normal-size LV cavity. There is limited motion of the affected IVS (**arrows**), between end-diastole (I) and end-systole (II); the limited septal deformation (strain) is also demonstrated on tagged CMR images in end-diastole (III) and end-systole (IV) ([Online Videos 5](#) and [6](#)). LA = left atrium; other abbreviations as in [Figures 1](#) and [2](#).

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