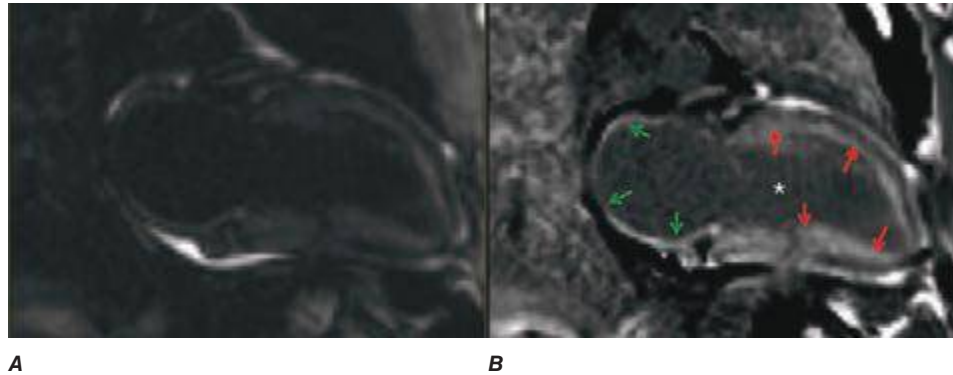
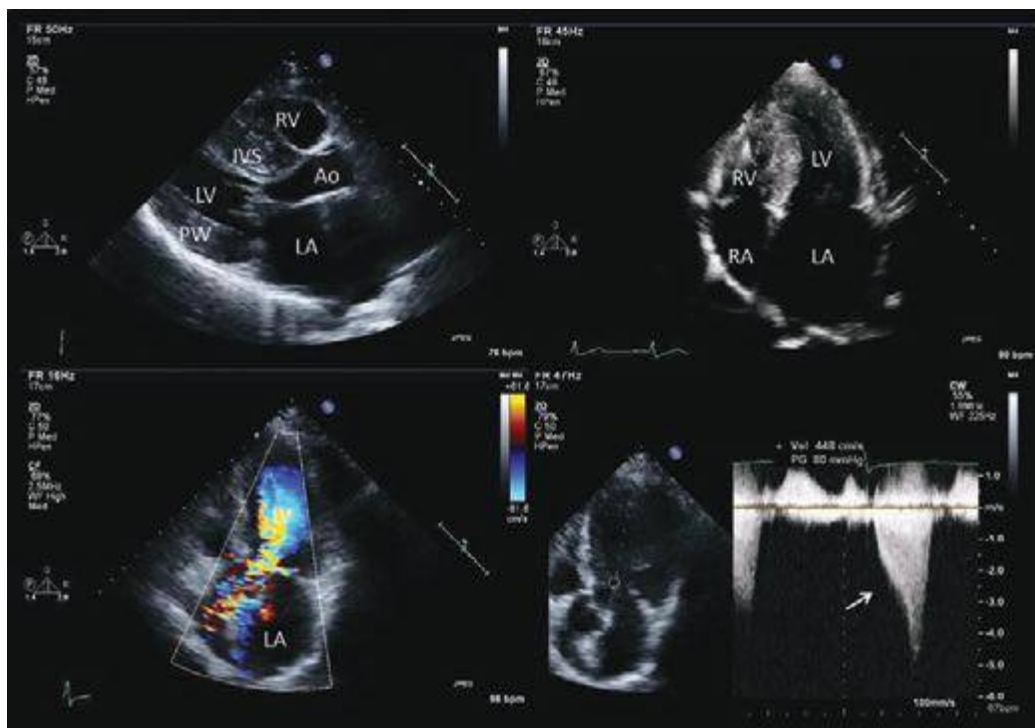




**FIGURE 271e-13** A 66-year-old patient with multiple myeloma and progressive shortness of breath. Echocardiography shows features typical of cardiac amyloidosis, including thickened myocardium with a “sparkly” appearance and left atrial enlargement. Systolic function is mildly reduced, and diastolic function is severely reduced. LA, left atrium; LV, left ventricle; RV, right ventricle. (See Videos 271e-11 and 271e-12.)



**FIGURE 271e-14** Magnetic resonance image with contrast enhancement in magnitude (A), and phase-sensitive reconstructed images (B) 5–10 min after injection of gadolinium in a patient with transthyretin (TTR)-mediated amyloidosis. The phase-sensitive reconstruction (B) enhances the region of abnormal collection of gadolinium, making gadolinium enhancement in the ventricle (red arrows) and the atrium (green arrows) more prominent. Amyloidosis causes accumulation of abnormal interstitial proteins, which results in late gadolinium enhancement in a diffuse subendocardial pattern (red arrows). Blood pool signal is characteristically dark (asterisk) owing to sequestration of gadolinium into other organs.



**FIGURE 271e-15** A 34-year-old woman with known cardiac murmur and syncope with a family history of sudden cardiac death. Echocardiogram shows classic findings of hypertrophic cardiomyopathy, including marked left ventricular wall thickness, particularly in the interventricular septum, notable in the parasternal long-axis view (upper left) and apical view (upper right). Note reverse septal curvature in the apical view (upper left). There is substantial flow acceleration through the left ventricular outflow tract (lower left) with evidence of a late peaking systolic gradient (arrow, lower right) caused by outflow tract obstruction. Ao, aorta; IVS, interventricular septum; LA, left atrium; LV, left ventricle; PW, posterior wall; RV, right ventricle. (See Videos 271e-13, 271e-14, and 271e-15.)