Venous myocardial contrast echocardiography is a new method for myocardial perfusion imaging. It has been shown to accurately evaluate risk area and infarct size in the experimental setting of acute myocardial infarction and has recently become clinically available. We used this method to assess myocardial salvage after coronary reperfusion in a patient with acute coronary syndrome.

A 68-year-old man with known single-vessel coronary artery disease presented with 6 hours of moderate chest pain typical of unstable angina. Four years earlier, a percutaneous transluminal coronary angioplasty of the left anterior descending coronary artery had been performed; the patient was asymptomatic throughout the following years. On admission, the 12-lead ECG showed atrial fibrillation and descending ST-segment depression up to 0.25 mV in leads V3 through V6. Cardiac enzymes, including troponin I, were normal at that time. After medical treatment with heparin, nitroglycerin, β-blocker, and aspirin, the chest pain and ECG changes resolved completely. Eight hours later, the patient reported reoccurrence of mild chest pain; by then, troponin I was elevated, at 0.8 ng/mL. Cardiac imaging at rest with venous myocardial contrast echocardiography (digitally processed and color-coded echocardiographic images, Figure, A) and 99mTc-sestamibi single photon emission computed tomography (SPECT; B) showed perfusion defects of the basal inferior left ventricular wall. On cardiac catheterization, a subtotal occlusion of the left circumflex coronary artery (C) was seen, and uncomplicated stenting of the lesion was performed (F). Creatine kinase reached a maximum of 256 U/L (MB fraction, 34 U/L) during the next day. After 7 days, venous myocardial contrast echocardiography (D) and 99mTc-sestamibi SPECT (E) were repeated and demonstrated almost complete reperfusion of the inferior left ventricular wall. The patient was discharged 10 days after admission.
2  Myocardial Perfusion in Acute Coronary Syndrome

A, Venous myocardial contrast echocardiography before reperfusion; apical 2-chamber view; contrast defect (arrows) of basal inferior left ventricular myocardium. Precontrast images were digitally subtracted from contrast-enhanced images, and resulting image was color-coded, with gradual transition from red to orange to yellow and white representing increasing contrast enhancement. B, 99mTc-sestamibi SPECT before reperfusion (99mTc-sestamibi injected simultaneously with A); vertical long-axis slice; reduced tracer uptake of basal inferior left ventricular myocardium (arrows). C, Coronary angiography; right anterior oblique angulation; subtotal occlusion (arrow) of left circumflex coronary artery. D, Venous myocardial contrast echocardiography 7 days after stenting of left circumflex coronary artery; almost normal contrast enhancement (arrows) of inferior myocardium. E, 99mTc-sestamibi SPECT 7 days after stenting of left circumflex coronary artery; nearly normal tracer uptake of basal inferior left ventricular myocardium. F, Coronary angiography; result after successful stenting of left circumflex coronary artery.