

Acute ST Depressions in a Patient With Idiopathic Hypertrophic Subaortic Stenosis and Normal Coronary Arteries

Eldrin F. Lewis, MD; Elazer R. Edelman, MD, PhD

A 49-year-old woman with a history of idiopathic hypertrophic subaortic stenosis diagnosed in 1982 presented with progressive dyspnea on exertion, paroxysmal nocturnal dyspnea, and palpitations. A physical examination revealed a heart rate of 74 bpm, blood pressure of 106/65 mm Hg, room air saturation of 87%, mild jugular venous distension, and a III/VI systolic ejection murmur at the left lower sternal border that was accentuated by the Valsalva maneuver. A baseline ECG was obtained (Figure 1), and an echocardiogram revealed severe left ventricular hypertrophy with an interventricular septum that was disproportionately thicker (19 mm) than the remainder of the ventricle. There was prominent systolic anterior motion of the mitral valve, with a left ventricular outflow gradient of 70 mm Hg and moderate-to-severe mitral regurgitation. She remained stable with an esmolol drip, and the following day she underwent cardiac catheterization, which revealed no epicardial coronary artery disease. On arrival to the coronary care unit after the angiogram, she developed severe chest pain with diaphoresis, nausea, vomiting, and hypotension with a

systolic blood pressure of 60 mm Hg. A repeat ECG demonstrated prominent down-sloping ST depressions (Figure 2) that were not present on her previous ECG. Despite aggressive therapy with intravenous fluid, inotropic therapy, and positional maneuvers, her clinical status continued to deteriorate, with refractory ischemia and pulmonary edema requiring intubation and urgent surgery. Her postoperative ECG demonstrated improved ST segments (Figure 3).

This case illustrates the tendency of patients with idiopathic hypertrophic subaortic stenosis to develop hemodynamic compromise and ischemia as a result of catheterization, possibly in relation to increased myocardial oxygen demand, inadequate capillary density, abnormal intramural coronary arteries, systolic compression of arteries, or impaired vasodilatory reserve. Cardiac catheterization may result in fluid shifts that can change the preload conditions of the heart and result in a larger gradient across the left ventricular outflow tract and a decrease in cardiac output.

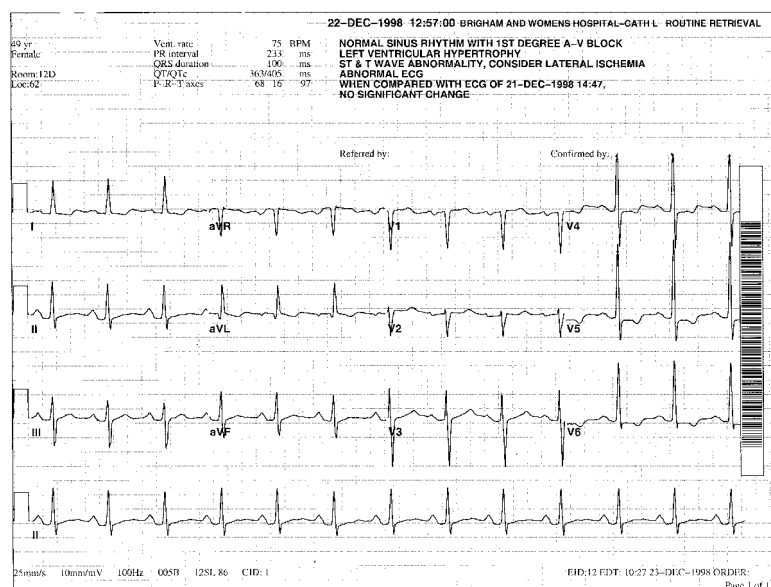


Figure 1. ECG in a patient with idiopathic hypertrophic subaortic stenosis demonstrating normal sinus rhythm with first-degree atrioventricular block and left ventricular hypertrophy with strain pattern.

From the Division of Cardiology, Brigham and Women's Hospital, Boston, Mass.

Correspondence to Eldrin F. Lewis, MD, Division of Cardiology, Brigham and Women's Hospital, 75 Francis St, Boston, MA 02115. E-mail eflewis@partners.org

The editor of Images in Cardiovascular Medicine is Hugh A. McAllister, Jr, MD, Chief, Department of Pathology, St Luke's Episcopal Hospital and Texas Heart Institute, and Clinical Professor of Pathology, University of Texas Medical School and Baylor College of Medicine.

Circulation encourages readers to submit cardiovascular images to the *Circulation* Editorial Office, St Luke's Episcopal Hospital/Texas Heart Institute, 6720 Bertner Ave, MC1-267, Houston, TX 77030.

(*Circulation*. 2002;106:757-758.)

© 2002 American Heart Association, Inc.

Circulation is available at <http://www.circulationaha.org>

DOI: 10.1161/01.CIR.0000024108.79612.E3

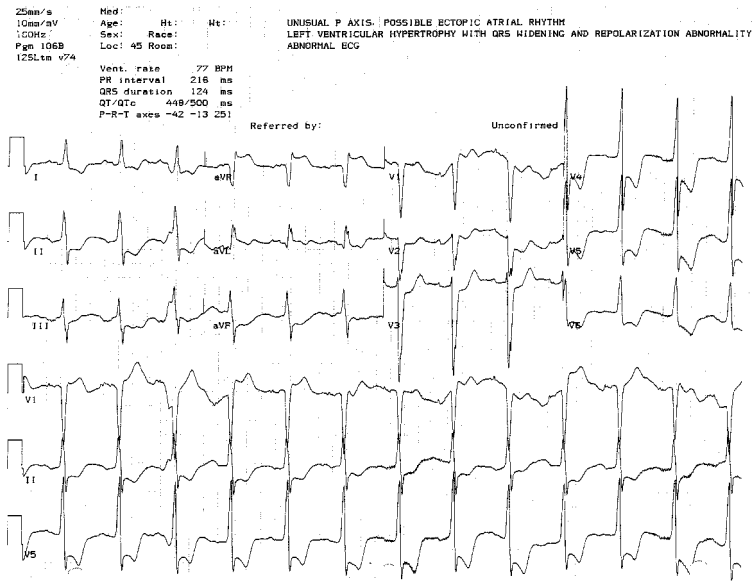


Figure 2. ECG obtained during chest pain and hypotension after cardiac catheterization. Note the diffuse, prominent ST depression.

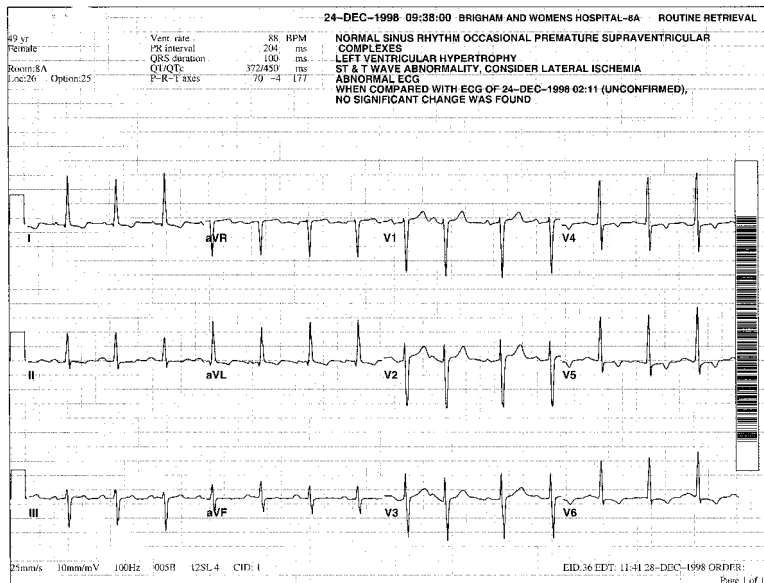


Figure 3. The ST depressions have resolved after emergent cardiac surgery necessitated by refractory chest pain and hypotension.