We describe the features of a rare form of left dominant coronary circulation in which the left anterior descending wraps around the apex forming the posterior descending artery. Similar cases have been reported,14 but this is the first in which the artery extends beyond the crux into both atrioventricular grooves and supplies the AV node.

**CASE REPORT**

A 54-year-old man presented after 1 h of chest pain, diaphoresis, and dyspnea. Electrocardiography revealed anterior and inferior ST-segment elevation, inferior Q-waves, and inferior T-wave inversion. He promptly received tissue plasminogen activator, his angina improved, and ST segment elevation resolved. The peak creatine phosphokinase was 2,418 U/L. Cardiac catheterization was declined, and he was discharged after nine days.

He returned two days later with chest pain and dyspnea. On examination, blood pressure was 134/86 mm Hg, and pulse was 92 beats per minute. Rales were present over the lung bases. An S4 and a grade 1/6 apical holosystolic murmur were heard. Electrocardiography revealed sinus rhythm, inferior Q-waves, and a tall R-wave in lead V1.

Cardiac catheterization demonstrated a large LAD (Fig 1) which wrapped around the apex forming the entire PDA, including a branch to the atrioventricular node. At the crux, the PDA bifurcated giving rise to branches traversing both AV grooves. The branch to the left AV groove in turn gave rise to posterolateral branches. There was a severe stenosis in the proximal LAD which appeared to have superimposed thrombus. A ramus intermedius branch had stenosis in its midportion, and a small right coronary artery had a proximal stenosis. A small left circumflex artery was free of disease. Left ventriculography revealed anterior, septal and apical akinesis and inferior hypokinesis with an ejection fraction of 0.26.

He underwent coronary artery bypass surgery with a left internal mammary artery graft placed to the mid-LAD and saphenous vein grafts to the ramus intermedius and first obtuse marginal branches. He was discharged after an uneventful postoperative recovery.

**DISCUSSION**

There is considerable variation in the blood supply to the inferior and posterior myocardium as first described by

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**Left Coronary Dominance Due to Direct Continuation of the Left Anterior Descending to Form the Posterior Descending Coronary Artery**

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Continuation of the left anterior descending to form the posterior descending artery is rarely observed. We describe the first patient with this variant in whom the coronary artery extends beyond the crux, supplying branches to the atrioventricular node and both atrioventricular grooves. (Chest 1992; 102:319-20)

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**Figure 1** A (left). Right anterior oblique left coronary angiogram demonstrating a large left anterior descending artery which wraps around the cardiac apex, ascends along the posterior interventricular groove and forms the entire posterior descending artery. This artery gives a branch to form the atrioventricular nodal artery and branches which traverse both atrioventricular grooves. A proximal stenosis, which appeared to have superimposed thrombus, is present in the left anterior descending artery. B (right). Left anterior oblique left coronary angiogram in the same patient. LAD, left anterior descending artery; PDA, posterior descending artery; AVNA, atrioventricular nodal artery; and L, lesion.
Silent Maladie de Roger*
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A patient with no cardiac murmur was found to have a ventricular septal defect by Doppler echocardiography yet no evidence of pulmonary or right ventricular hypertension. This array of findings is distinctly unusual and appears to be at odds with the clinical teachings concerning small ventricular septal defects. (Chest 1992; 102:320-22)

Doppler echocardiography has demonstrated aspects of cardiac physiology undetectable by physical examination or other imaging techniques. Physiologic degrees of atrioventricular valve regurgitation and pulmonic valve insufficiency are frequently seen. Unsuspected mild aortic regurgitation is often documented.

Recently, we evaluated a patient with no abnormal cardiac findings upon physical examination, yet a flow-restrictive ventricular septal defect was unexpectedly found by color flow Doppler imaging and was confirmed by pulsed and continuous-wave Doppler studies.

CASE REPORT
A 30-yr-old man presented to the outpatient clinic for a preemployment physical examination. He gave a history of a cardiac murmur as a child, for which he vaguely remembered having undergone cardiac catheterization. He received no specific treatment. Information concerning the findings from catheterization could not be traced. He was normally active and denied chest pain, dyspnea, palpitation, cyanotic spells, and syncope.

Physical examination demonstrated a thin man with a blood pressure of 110/80 mm Hg and a pulse rate of 80 beats per minute. The neck veins and carotid upstrokes were normal. The thoracic cage was normal, and the lungs were clear. The cardiac apex impulse was normal. The S1 was normal; S2 was normal and physiologically split. No rub, gallop, click, or murmur could be heard in any posture. No murmur was elicited with Valsalva’s maneuver, handgrip, or standing from a squat. The abdomen and extremities were normal. Specifically, no cyanosis or clubbing was present. The electrocardiogram and chest roentgenogram were normal. A right antecubital fossa scar was noted, probably the site of his childhood catheterization.

Echocardiographic Findings
M-mode and two-dimensional images in all standard views were obtained. The chamber sizes, wall motion, and valvular motion were all normal. Doppler echocardiography demonstrated no valvular stenosis or regurgitation. Color flow Doppler imaging revealed a narrow, high-velocity systolic jet entering the right ventricle at the level of the membranous septum (Fig 1A and 1B) compatible with a ventricular septal defect. Continuous-wave Doppler interrogation of this jet, as depicted in Figure 1C, documented a 4 m/s peak systolic flow velocity. The highest velocity we could obtain was 4.3 m/s. Using the modified Bernoulli equation, this corresponds to a left ventricular-right ventricular peak systolic gradient of 75 mm Hg. Given a systemic arterial systolic pressure of 110 mm Hg, this suggests a right ventricular systolic pressure of no greater than 35 mm Hg.

Because of the disparity between the echocardiographic findings

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