Continuous murmur and cardiac failure in a 53-year-old woman

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Figure. Electrocardiogram recorded in a 53-year-old woman with a continuous murmur and congestive heart failure. See text for explanation.

A 53-year-old woman first had a heart murmur noted at age 3. She was symptom free until age 47 but since then has had repeated episodes of cardiac failure. On this occasion she noted increasing shortness of breath, bilateral leg edema up to her knees, and a 10-lb weight gain over a week's time. Increasing her furosemide from 20 to 40 mg daily did not relieve her symptoms.

On physical examination, this small, slender woman had a blood pressure of 140/84 mm Hg, bilateral expiratory wheezes, and crackles at both lung bases. Neck veins were elevated to 15 cm above the angle of Louis, and there was 1–2+/4+ edema up to her knees. A 2+/4+ right ventricular lift was felt at the left sternal edge. A hyperdynamic 4+/4+ left ventricular impulse was felt and a third heart sound was heard in the anterior axillary line. A 4+/6+ continuous murmur peaked at the second heart sound and was best heard

in the second left intercostal space. There are many causes of continuous murmurs. When the intensity of the murmur peaks at the second heart sound and the murmur is loudest in the second left intercostal space or just below the medial portion of the left clavicle, the cause is almost always a patent ductus arteriosus. When the murmur is maximal elsewhere, the cause usually is not a ductus.

An electrocardiogram (*Figure*) recorded at half standard, i.e., 1 mV = 5 mm, showed sinus rhythm with atrial premature complexes, biatrial enlargement, left axis deviation of the QRS

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complex (-35°), and left ventricular hypertrophy with repolarization abnormality, including a long QT interval (405 ms with a QTc of 473 ms). Although left ventricular hypertrophy with repolarization abnormality, i.e., a wide angle between the QRS and the ST-T vectors, is classically seen with pressure overload of the left ventricle, such as occurs with systemic hypertension or aortic stenosis, this repolarization change may occur whenever left ventricular hypertrophy is severe, no matter the cause (1).

The chest radiograph demonstrated a huge cardiac silhouette, prominent pulmonary vasculature, bilateral pulmonary edema, and scoliosis of the spine, which is more common in patients with congenital heart disease than in the general population.

In the past, patients with patent ductus arteriosus occasionally have lived to be far older than our patient (2), but for 75 years now closure of the arterial duct, first by surgical ligation (3) and more recently by catheter-delivered closure devices (4), has been available as a highly effective and relatively low-risk cure for the malformation. Consequently most arterial ducts are now closed in infancy or early childhood. Our patient, however, has always refused any invasive treatment. In her, the duct is large enough to allow a moderately large left-to-right shunt that over time has produced severe left atrial and left ventricular enlargement. Although her left

ventricular ejection fraction 18 months ago was 50% as judged by echocardiogram, the left ventricle clearly is failing and the resulting pulmonary arterial hypertension has caused right ventricular and right atrial enlargement. This has been accentuated by chronic obstructive pulmonary disease caused by her smoking $1\frac{1}{2}$ packs of cigarettes per day for 25 years and manifested by a forced expiratory volume in 1 second of 35% and wheezing even when she is not in overt cardiac failure, and by a hemoglobin of 17 g/dL and a hematocrit of 53% on the current admission.

The patient responded to medical treatment for congestive heart failure but was lost to follow-up after discharge.

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