Atrial fibrillation with QRS voltage low in the limb leads and high in the precordial leads

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Figure. Electrocardiogram recorded during the last month of the patient’s life. See text for explication.

In the electrocardiogram illustrated here (Figure), the ventricular rhythm is irregularly irregular with baseline undulation but no definite P waves; thus, the rhythm is atrial fibrillation. No time lines are visible on the tracing, but it was recorded at 25 mm per second and is 250 mm, or 10 seconds, long. Nineteen QRSs occur in the 10 seconds, making the ventricular rate 114 beats per minute. The fourth QRS is wide and unlike the other QRSs. It is not typical of right or left bundle branch block and therefore is more likely to be a ventricular premature complex than an aberrantly conducted supraventricular impulse. The calibration signals on the left side of the electrocardiogram are 10 mm/mV, and the R waves in V6 are 34 mm tall (3.4 mV), exceeding the 26 mm (2.6 mV) upper limit of normal for V5 or V6 (1). Likewise, the S wave in V1 plus the R wave in V6 equals 41 mm (4.1 mV), exceeding the 35 mm (3.5 mV) upper limit of normal for SV1 plus RV5 or V6 (2). In addition to meeting these two voltage criteria for left ventricular hypertrophy, the repolarization change in V6 is typical of the condition: depressed ST segment with asymmetric inversion of the T wave (3).

Perhaps the most interesting and least appreciated aspect of the electrocardiogram is the low QRS voltage in the limb leads in the presence of high QRS voltage in the precordial leads. This pattern was recognized as a clue to a dilated cardiomyopathy in 1985 by Goldberger et al (4), and others subsequently have
confirmed it (5, 6). A less common, but perhaps more specific, electrocardiogram pattern indicating a dilated cardiomyopathy is left bundle branch block with right axis deviation (6–8).

This electrocardiogram was recorded in the last month of life of a 40-year-old man who died of cardiac failure. At necropsy, he had four-chamber cardiac dilatation. His valves and coronary arteries were normal, and his left ventricular wall was hypertrophied and the cavity dilated. He had never been hypertensive. Thus, he had an idiopathic dilated cardiomyopathy.