Electrocardiogram in a 55-year-old woman with an endocrine disorder

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Figure 1. Electrocardiogram recorded in August 2006. See text for explication.

The salient features of the electrocardiogram in *Figure 1* are sinus bradycardia, low QRS voltage, flat T waves in all leads in the absence of ST-segment depression, and a long QT interval. Each of these findings is typical of hypothyroidism, and the combination of all four in the same electrocardiogram has a high positive predictive value (1, 2). Indeed, the patient's serum levels of thyroid-stimulating hormone (183.71 μ IU/mL; reference range, 0.50–5.00) and free thyroxine (<0.15 ng/dL; reference range, 0.60–1.15) indicate profound hypothyroidism. An earlier electrocardiogram, recorded when the patient was euthyroid, shows sinus bradycardia but otherwise is normal (*Figure 2*).

The patient has been hypothyroid for 10 years due to Hashimoto disease, which, along with the use of radioiodine to treat Graves disease, has replaced iodine deficiency as the leading cause of hypothyroidism in this country (2). Six months before the electrocardiogram in *Figure 1* was recorded, she had stopped taking levothyroxine because of fatigue, dizziness, nausea, and diarrhea. These symptoms may have been due to Addison disease, because soon thereafter adrenal insufficiency was diagnosed

on the basis of low levels of serum cortisol both at baseline (7.2 μ g/dL; reference, $\geq$ 8.0) and 30 and 60 minutes after the intravenous injection of cosyntropin (9.4 μ g/dL at 30 minutes, 11.4 at 60 minutes; reference, $\geq$ 20). She started on cortisol but never resumed levothyroxine.

When the patient was seen just before the electrocardiogram shown in *Figure 1* was recorded, she complained of cold intolerance, decreased appetite, constipation, and weight gain. Examination revealed dry skin, puffy eyes, slow and slurred speech with hoarseness, and thyromegaly. Thus, her clinical picture matched the laboratory values.

Electrocardiographic changes in hypothyroidism are usually accompanied by other cardiovascular abnormalities (2). Decreased blood volume, decreased stroke volume, and decreased

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Figure 2. Electrocardiogram recorded in May 2004 when the patient was taking 100 µg/d of levothyroxine shows sinus bradycardia but is otherwise normal.

heart rate result in a low cardiac output. Despite this, a significant rise in systemic vascular resistance often causes systemic arterial hypertension. Left ventricular contractility is reduced, and a prolonged myocardial relaxation time impairs early diastolic ventricular filling. A pericardial effusion occurs in up to 30% of overtly hypothyroid patients and occasionally may be large. Cardiac tamponade, however, is rare. Cardiac failure is unusual in the absence of coexisting heart disease.

Total and low-density lipoprotein cholesterol levels rise in proportion to the rise in thyroid-stimulating hormone, beginning at a level of 5 µIU/L (2). Thus, hypothyroidism is a risk factor for coronary arterial disease. A large study of elderly women in Rotterdam found that, compared with the euthyroid state, subclinical hypothyroidism increased the odds ratio of developing atherosclerosis and myocardial infarction to 1.7 and 2.3, respectively, and was an independent risk factor for both (3).

Because all of the adverse effects of hypothyroidism are reversed when adequate amounts of thyroid hormone are given and because 3% to 4% of the population have overt hypothy-

roidism and another 7% to 10% have milder disease (2), the potential public health benefit of early diagnosis is enormous. The extreme sensitivity of elevated levels of serum thyroid-stimulating hormone for diagnosing hypothyroidism, even when serum thyroxine levels are in the normal range, make early diagnosis easy, provided one thinks of it. Such screening has been recommended for virtually all adults, especially older ones and those with hyperlipidemia, hypertension, evidence of atherosclerosis, unexplained pericardial or pleural effusions, and several musculoskeletal problems (2).

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