Pauses in the electrocardiogram of a woman with chest pain and lightheadedness

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Figure. Electrocardiogram in a woman with chest pain and near syncope. See text for explication.

A 64-year-old woman with systemic arterial hypertension, dyslipidemia, cigarette smoking, and a history of stent placement in the proximal left anterior descending coronary artery for an 80% diameter stenosis returned to the hospital a year later because of pain similar to the pain she had before stent placement. She also described one episode of dizziness and a feeling of faintness while walking.

An electrocardiogram recorded during this admission showed group beating with four sinus-initiated complexes separated by a pause from three sinus-initiated complexes; pauses also began and ended the tracing (*Figure*). All seven sinus-initiated impulses had P-R intervals of 0.15 seconds. The intervals separating the first four P waves were 1.04, 1.02, and 0.99 seconds, respectively, and the P-P intervals of the group of three complexes were 1.03 and 0.99, respectively. The P-P interval of the middle pause was 1.87 seconds and, thus, was shorter than 2 times any single P-P interval. These are the findings of typical second-degree

sinoatrial (SA) block, type I, with 5:4 and 4:3 Wenckebach periods. The SA conduction times increased in progressively smaller increments so that the P-P intervals became progressively shorter, and the P-P interval containing the nonconducted impulse was shorter than 2 times any single P-P interval.

Unlike typical second-degree atrioventricular (AV) block, type I, where the lengthening of the P-R intervals in progressively smaller increments can be measured, because sinus node depolarization does not register on the standard electrocardiogram, SA conduction times cannot be measured. SA block, type I, is diagnosed by finding progressive shortening of the P-P

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intervals before the pause with no P wave. This is analogous to the progressive shortening of the R-R intervals before the nonconducted P wave in typical second-degree AV block, type I. Also, analogous to the situation in type II AV block, with type II SA block, SA conduction time does not increase before the nonconducted impulse; the P-P intervals do not decrease; and the pauses are exact multiples of the basic P-P interval (1).

The patient's electrocardiogram also showed several published voltage criteria for left ventricular hypertrophy when $1 \text{ mV} = 10 \text{ mm}$: $\text{RI} > 13 \text{ mm}$, $\text{SaVR} > 14 \text{ mm}$, $\text{SV}_1 + \text{RV}_5$ or $\text{V}_6 > 35 \text{ mm}$, $\text{R} + \text{S}$ in any limb lead $> 19 \text{ mm}$, and in a woman ≥ 40 years old $\text{RaVL} + \text{SV}_3 > 12 \text{ mm}$ when $\text{TV}_1 \geq 2 \text{ mm}$ (2–4). Widespread slight nonspecific ST-segment sagging also was present. Her echocardiogram was normal.

The patient underwent repeat coronary arteriography, which revealed a patent left anterior descending stent and nonobstructive coronary arterial disease, and further invasive interventions were considered unnecessary. She continued to have near-syncope episodes. A review of her electrocardiograms over the past 18 months revealed another episode of SA block and a number of other findings suggesting sick sinus syndrome: sinus bradycardia as slow as 32 beats per minute, at times with a junctional escape rhythm; slow ectopic atrial bradycardia; and sinus pauses of up to 2 seconds that did not fulfill criteria for SA block. Most of this time she was on a beta-blocking drug, at first atenolol 25 mg per day and subsequently carvedilol 6.25 mg once or twice per day, that may well have accentuated her bradyarrhythmias.

Her primary physicians thought the beta-blockers a necessary part of her drug regimen for coronary arterial disease, and the bradyarrhythmias were managed with an AV sequential electronic pacemaker (5).

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