John Haggerty, DO; William Goldenberg, MD

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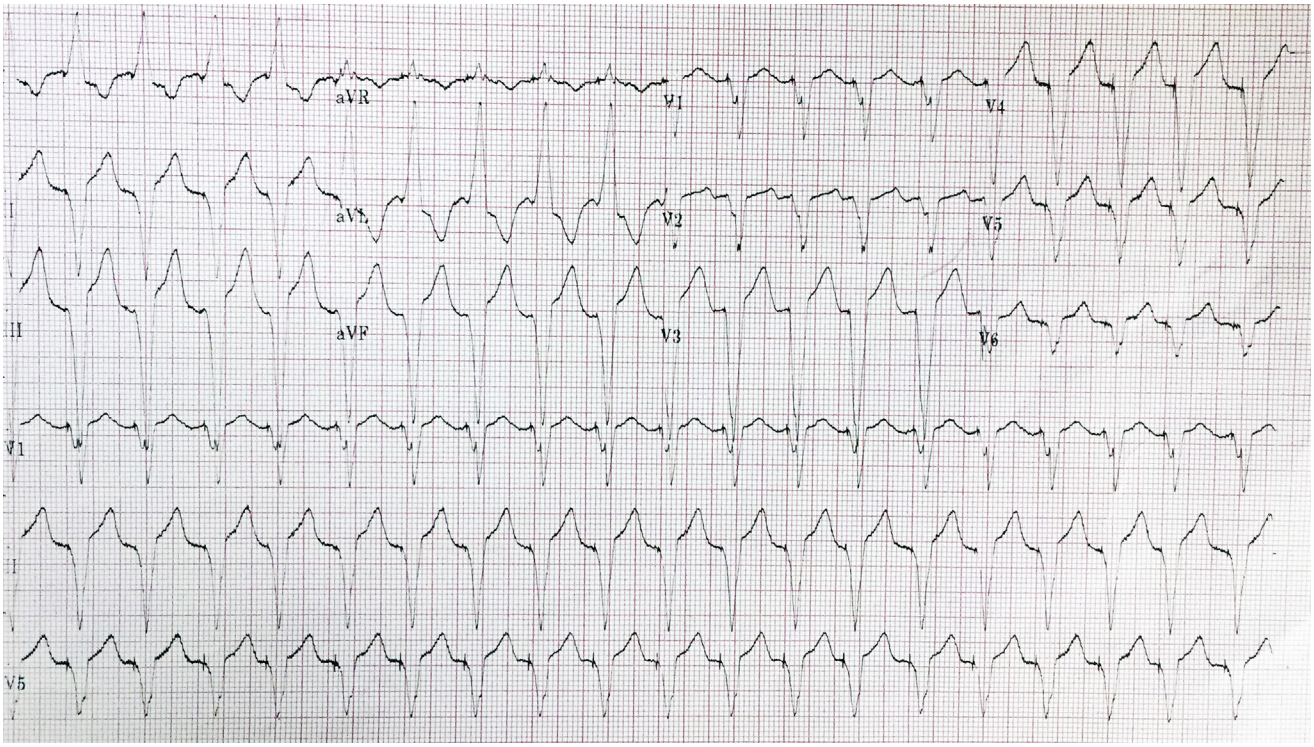


**Figure 1.** The patient's presenting ECG, showing an atrially sensed, ventricularly paced rhythm at 120 beats/min, with a PR interval of 104 ms, QRS-interval duration of 160 ms, prolonged QTc interval at 599 ms, and QRS axis of −65.

[Ann Emerg Med. 2019;73:627-630.]

A 61-year-old woman reported to her pacemaker clinic with a 1-week history of continuous dyspnea, chest tightness, and palpitations, which were worsened by exertion. Pacemaker interrogation revealed atrial flutter, but no malfunction. Subsequently, the patient was sent into the emergency department (ED). She reported dual-chamber pacemaker placement for sick sinus syndrome several months previously and warfarin usage for mitral valve replacement. ECG revealed paced atrial flutter at 120 beats/min, with widened QRS interval to 160 ms (Figure 1). Chest radiograph revealed normal pacemaker lead placement. Laboratory evaluation revealed undetectable troponin levels and therapeutic-range international normalized ratio.

Because of her symptoms, synchronized cardioversion was performed at 120 J, which the patient tolerated well. Postcardioversion ECG was obtained (Figure 2) and can be compared with the patient's baseline ECG (Figure 3).

*For the diagnosis and teaching points, see page 629.*
*To view the entire collection of ECG of the Month, visit www.annemergmed.com*

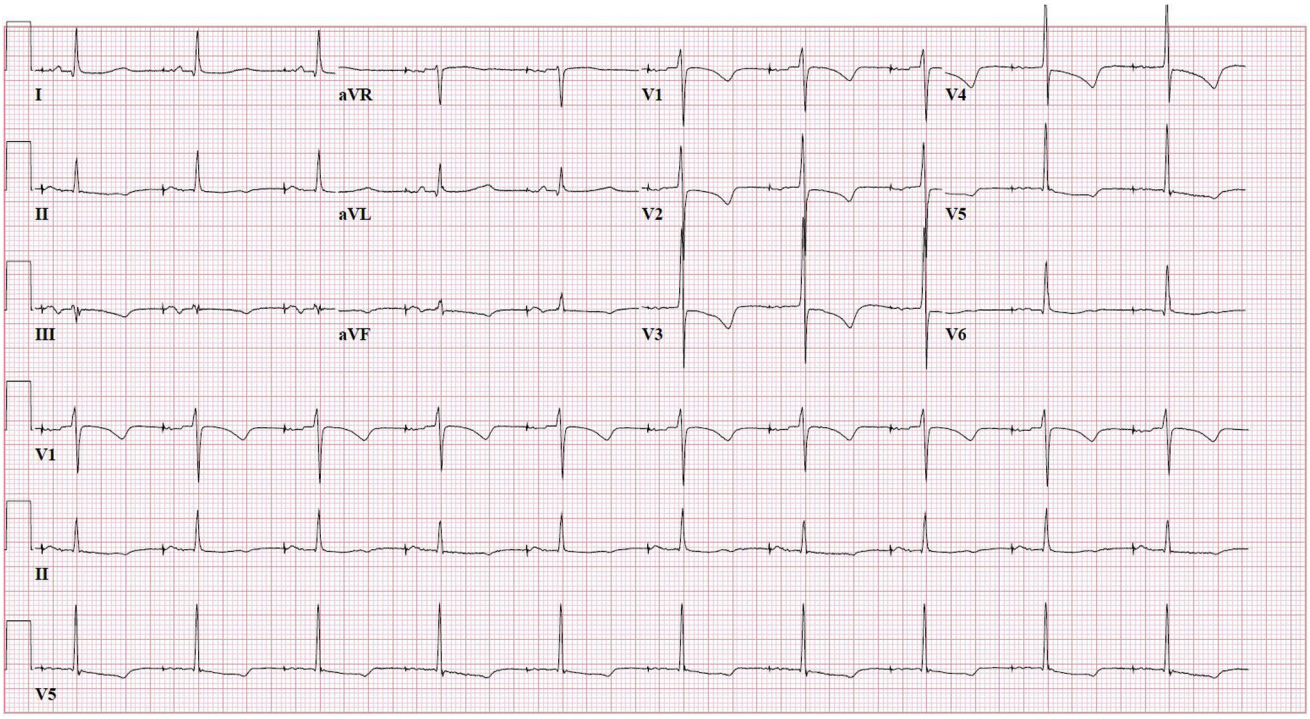


**Figure 2.** The postcardioversion ECG, showing atrially paced rhythm at 60 beats/min, with a normal QRS axis of 24, prolonged QTc interval at 510 ms, and new T-wave inversions in leads II, III, aVF, and V3 to V5.

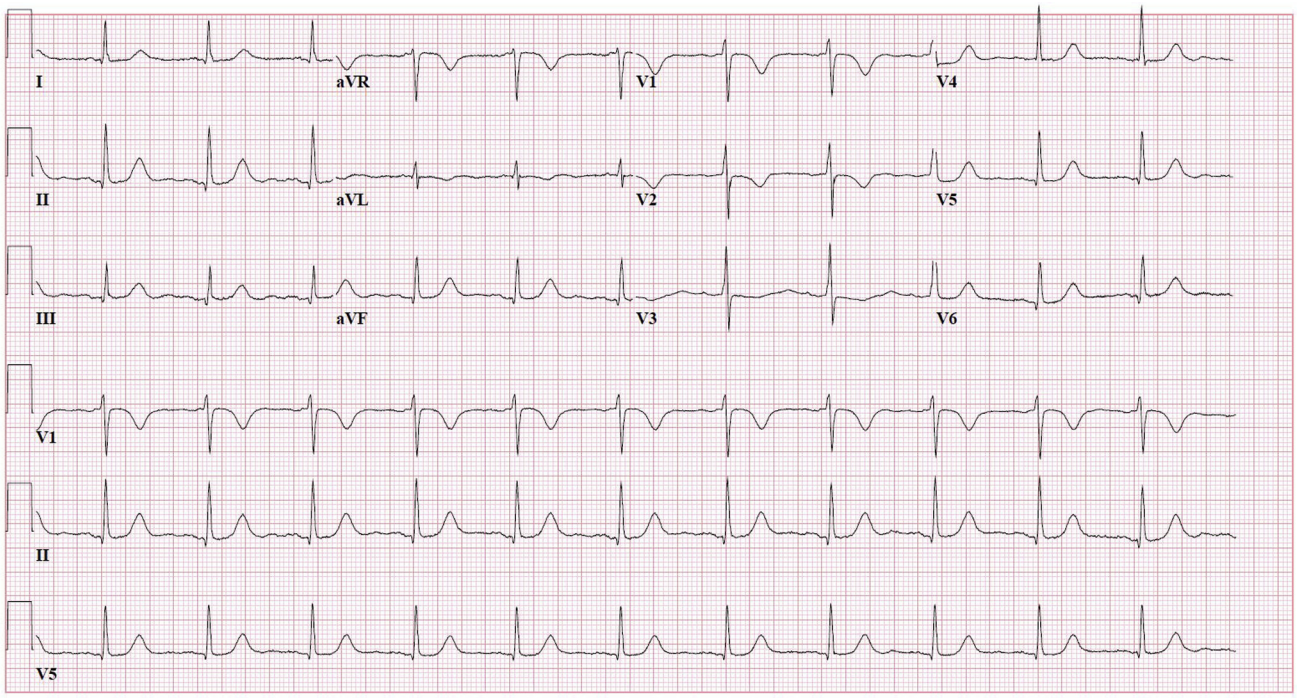


**Figure 3.** Patient's baseline ECG from several months before her visit to the ED, showing nonpaced, normal sinus rhythm at 69 beats/min, PR interval of 118 ms, QRS duration of 80 ms, QTc interval of 475 ms, and a QRS axis of 55, with only T-wave inversions in V1 and V2.

ECG OF THE MONTH

(continued from p. 627)

**DIAGNOSIS:**

The patient's ECG and clinical picture were consistent with a diagnosis of cardiac memory.

**CLINICAL COURSE**

After cardioversion, the patient reported resolution of the palpitations and dyspnea but continued chest tightness, which was relieved with nitroglycerin. Given the patient's symptoms and new T-wave inversions, cardiac ischemia was a concern, and her cardiology team was consulted and noted that the T-wave inversions followed a unique pattern consistent with cardiac memory. Although this in and of itself did not rule out ischemia, laboratory testing revealed negative troponin results and no changes in troponin levels, and her symptoms resolved. Because of this, her cardiologist thought that the patient could be safely discharged from the ED and closely followed up in their clinic for further risk stratification. The patient was discharged and followed up with the cardiologist, with no further symptoms or events.

**DISCUSSION**

Cardiac memory is defined as persistence of T-wave changes after restoration of normal ventricular conduction after a wide QRS-complex rhythm. The extent and persistence of these changes are proportional to the duration of the preceding wide QRS rhythm. Rosenbaum et al[1] first demonstrated cardiac memory by inducing left bundle branch block in a volunteer and noting new T-wave changes that persisted even after resolution of the block. Similar findings were shown in other volunteers who received right ventricle pacing for various periods. These findings were later attributed to short-term changes in proteins and ion channels and longer-term changes in gene transcription and protein synthesis.[2] A retrospective study conducted by Shvilkin et al[2] compared a percutaneous coronary intervention-proven ischemia group with an induced cardiac memory group and found 92% sensitivity and 100% specificity for cardiac memory when the following criteria were met in individuals who had resolution of a prolonged wide QRS rhythm:

1. A positive T wave in lead aVL and a positive or isoelectric T wave in lead I
2. Precordial T-wave inversions with a larger magnitude than any T-wave inversion in lead III[2]

Given the small numbers of patients in this study (13 with induced cardiac memory and 47 with proven ischemia), wide confidence intervals, although not reported, could be expected. Several case reports describe patients who met the above criteria after a period of abnormal depolarization and who went on to receive cardiac catheterization demonstrating no significant blockage.[3-5]

**PEARLS**

There is an increasing body of literature describing cardiac memory, a benign, nonischemic process that can occur after cardioversion or spontaneous resolution of a wide QRS-complex rhythm.

The presence of cardiac memory in and of itself is not "protective" from coronary artery disease, and appropriate exclusion of cardiac ischemia may still be warranted.

In the absence of other evidence of ischemia, costly hospitalizations and further invasive testing may be avoided if cardiac memory is properly understood.

Consultation with a cardiologist may be useful in decisions about disposition and further testing.

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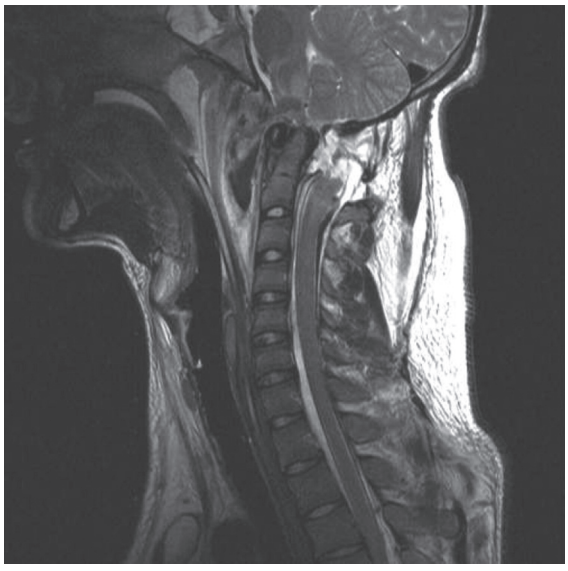

"Long-Term Survival Following Complete Medulla/Cervical Spinal Cord Transection" by Gautschi and Zellweger, April 2007, Volume 49, #1, pp. 540, 545.