

A “Tag Along” Pacemaker

George Veenhuizen, MD



About the Author

George Veenhuizen is an adult cardiac electrophysiologist at the Libin Cardiovascular Institute of Alberta in Calgary. He is interested in the diagnosis and management of all arrhythmias, particularly using catheter ablation.

A 62-year-old woman was brought to the emergency room by her neighbour with progressive confusion developing over several days, and weakness. She had a pacemaker implanted several years previously because of recurrent syncope due to intermittent complete heart block. What is going on?

pacemaker is probably sensing the ventricular escape rhythm since pacemaker spikes are synchronous with that rhythm. In fact, the pacemaker spikes occur approximately 1,000 ms after every QRS complex, indicating a VVI pacemaker set at a lower rate of 60 bpm. To summarize: (1) slow wide complex rhythm with (2) QRS, (3) Q–T

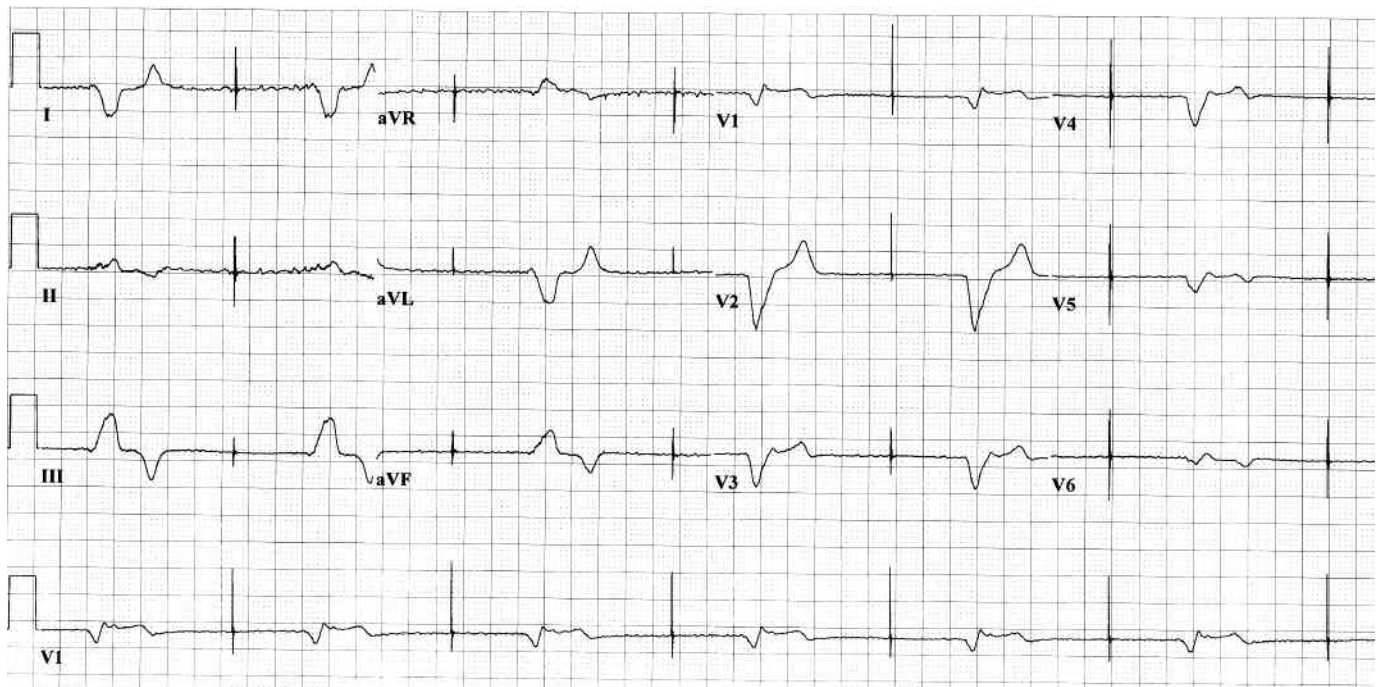


Figure 1

The tracing in the EKG (Figure 1) reveals a slow, regular, bizarre morphology, wide complex rhythm at 36 bpm. There is no evidence of atrial activity (i.e., there are no P waves and no evidence of atrial fibrillation), so AV block is not currently the causing bradycardia. That is, the problem is not that atrial impulses are *being formed and not conducted* but, rather, that *none of the heart's usual pacemakers are forming impulses* (other than the one generating the slow wide complex rhythm). The QRS complex duration (240 ms!) and Q–T interval (560 ms) are markedly prolonged. There are also pacemaker spikes occurring at a rate of 36 bpm, synchronous with the wide complex rhythm, but none of them capture the atrium (no P waves after the spikes) or ventricles (no QRS complexes after the spikes). The

prolongation, and (4) failure of pacemaker to capture.

These findings suggest a metabolic derangement that is suppressing the activity of the heart's usual pacemakers, slowing depolarization (wide QRS) and repolarization (long Q–T), and increasing the capture threshold of the pacemaker. Diagnoses to consider include overdose of a cardiac membrane active drug (e.g., an antiarrhythmic drug), hypothermia, or a severe electrolyte disturbance. The history suggests a process that has progressed over several days. In this case, the patient had acute renal failure with hyperkalemia. When hyperkalemia causes a loss of discernible atrial activity, QRS widening, and failure of a pacemaker to capture, the serum potassium

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concentration is usually >7 mmol/L.

It is noteworthy that although there is technically a failure to pace, the pacemaker is functioning normally. Intact sensing of the intrinsic escape rhythm inhibits pacemaker output until 1,000 ms pass without any sensed intrinsic rhythm. This triggers the pacemaker to fire; only the output of the pacemaker is insufficient to generate a propagating wavefront of depolarization (because the high potassium

concentration has raised the threshold for capture), so no QRS complexes are produced. This is followed by the next beat of the escape rhythm, which is appropriately sensed by the pacemaker, and this "tag along" cycle continues. When the serum potassium concentration was lowered, normal sinus rhythm resumed and pacing was not required. Pacemaker interrogation revealed, as expected, a normally functioning pacemaker.

Clinical Review

Stress-Induced (Takotsubo) Cardiomyopathy

John Robb, MD



About the Author

John Robb is a community general internist in Magog, Quebec, and a professeur enseignement clinique at Université de Sherbrooke.

Clinical Case

A 55-year-old woman presents with 2 days of squeezing retrosternal chest pain on walking, lasting 1 hour. Her medical history includes hypertension on lisinopril and hyperlipidemia on atorvastatin, but no diabetes, and she is an ex-smoker, quitting 20 years ago. She has been on a diet and exercise program for 6 months with a weight loss of 14.5 kg

(32 lb). She has been under stress lately for financial (credit card) debt and a possible company closure. Her vital signs and physical examination are normal except for a possible S_4 . An ECG shows anterior T wave inversion (Figure 1) and her troponin has increased to 0.16 ng/mL. The following day she has a normal coronagraphy. What is her diagnosis and how should she be treated?

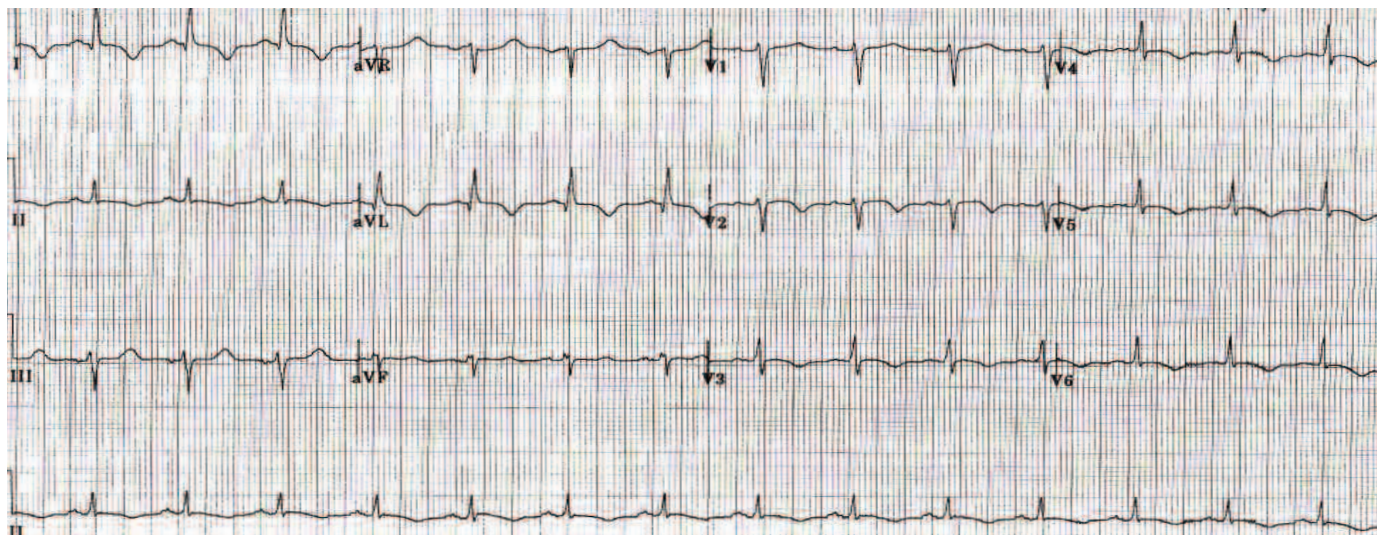


Figure 1. ECG at presentation showing sinus rhythm at 88 bpm with diffuse T wave inversion.