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An intermittently paced rhythm: Deciphering the etiology of depolarization

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Abstract We present a case of an intermittently paced rhythm in a patient with prior placement of a St. Jude Medical (SJM) dual chamber pacemaker (PPM). The patient had a history of difficult-to-control hypertension, insulin-dependent diabetes mellitus, gastroparesis and end-stage renal disease receiving hemodialysis presenting for admission due to hypertensive urgency. Initial electrocardiogram upon arrival showed sinus tachycardia, however, a follow up electrocardiogram was notable for ventricular pacing only every other beat with a shortened atrioventricular delay beyond the programmed settings of the PPM.

Keywords: Permanent pacemaker (PPM); Pseudofusion; Fusion

A 55 year old Latin American female presented with one day of elevated blood pressure and intractable nausea and vomiting. She had a past history of difficult-to-control hypertension, insulin-dependent diabetes mellitus, gastroparesis, end-stage renal disease receiving hemodialysis, and placement of a St. Jude Medical (SJM) dual chamber permanent pacemaker (PPM). Her PPM was placed 6 years prior due to symptomatic bradycardia and intermittent atrioventricular (AV) nodal block. Last known device settings were: DDD pacing mode, lower rate interval of 60 bpm, upper tracking rate of 120 bpm, AV delay of 250 milliseconds (ms) without rate responsive changes and a post ventricular atrial refractory period (PVARP) of 170 ms. Rate responsive AV delay (RRAVD) changes were set to "off". The patient was now admitted for hypertensive urgency and started on a nicardipine drip at 5 milligrams per hour. Examination was unremarkable except for a blood pressure of 203/113 mm Hg, mild distress secondary to abdominal discomfort and diffuse abdominal tenderness with deep palpation. Laboratory data were remarkable for an elevated creatinine of 3.7 milligrams per deciliter. Serum electrolytes were in normal range. Electrocardiogram (ECG) showed sinus rhythm with a left axis deviation. Electronic pacemaker spikes were not seen (see Fig. 1).

On hospital day 5 the patient developed acute onset chest pressure and a repeat ECG showed sinus tachycardia at a rate of 108 bpm with intermittent ventricular pacing stimuli (see Fig. 2) following an AV delay of 120 ms. Serial cardiac biomarkers were negative for acute coronary syndrome. Plain chest radiography revealed clear lung fields, a normal heart border with an overlying dual chamber pacemaker with a ventricular lead appropriately placed in the right ventricular apex and atrial lead in the right atrial appendage.

What is the most likely diagnosis?

a. ECG processing artifact
b. Sinus tachycardia with pacemaker malfunction (no sensing, no pacing).
c. Sinus tachycardia with pseudofusion complexes
d. Sinus tachycardia with fusion complexes
e. Pacemaker mediated tachycardia (PMT)

Initial analysis of the ECG tracing in Fig. 2 shows sinus tachycardia at a rate of 108 bpm. An electronic pacemaker stimulus is seen at the beginning of every other QRS. The PR interval in the complexes not preceded by the pacemaker stimulus is 160 ms, and the P-to-ventricular pacemaker stimulus in the complexes preceded by the pacemaker stimulus is 120 ms. At this step of the review a differential diagnosis may include failure of the device to sense atrial depolarization, true intermittent pacing, and a failure of the ECG machine to register every output from the pacing device in a 2:1 fashion.

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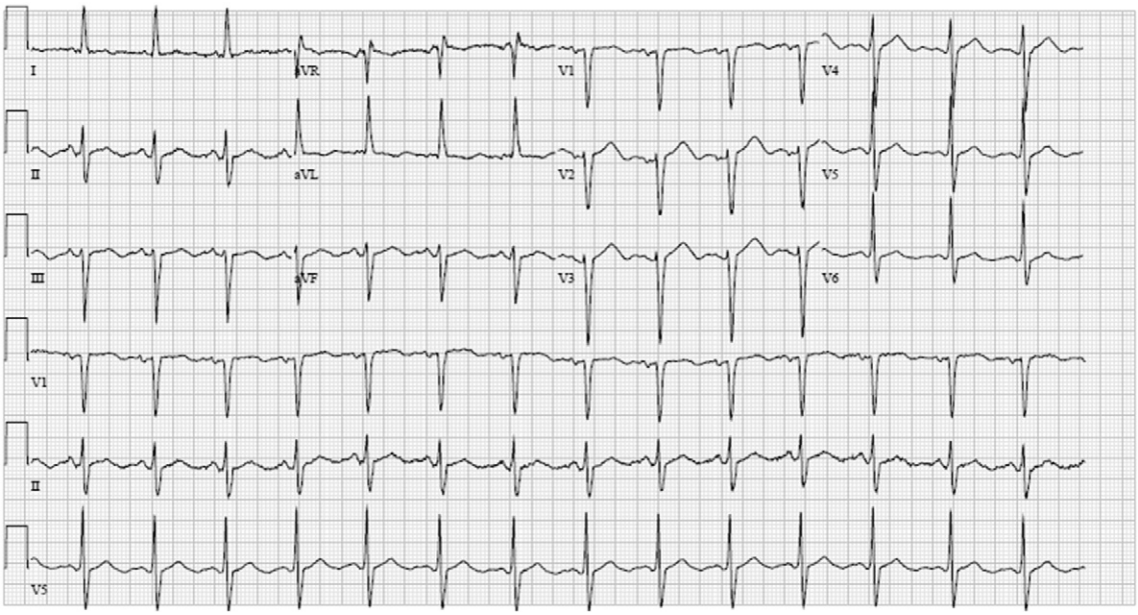

Fig. 1. ECG at presentation showing sinus rhythm at a rate of 90 bpm.

Even though pacemaker stimuli are seen only with every other QRS complex, it cannot be assumed that ventricular pacing is truly intermittent. ECG processing is known to lead to missed stimuli based on the fact that the PPM stimuli are short in duration and at times can be of low amplitude. Sampling on modern machines occurs by taking analog output information and processing it into a digital ECG. The sampling of electrical cardiac signals is typically set at a rate 500–1000 Hertz (1 sample cycle every 1–2 ms). However, given PPM output duration is frequently less than 0.5 ms it is not uncommon that PPM spikes are missed by routine ECG sampling. In addition, newer bipolar leads have a much lower output than traditional unipolar leads. This can lead to missed signals from the PPM and lack of a spike on the digital ECG [1].

In the case of our patient, however, careful inspection of the ECG in Fig. 2 shows subtle changes in the QRS morphology between complexes preceded and not preceded by electronic pacemaker stimuli. Given the slight variation of the QRS in the paced beats, these complexes likely represent fusion, rather than pseudofusion beats. Ventricular fusion complexes occur when two distinct sources of electrical depolarization combine to separately activate the ventricular myocardium. The further an ectopic foci is from the native ventricular conduction system (i.e., His–Purkinje system) and the earlier the foci depolarizes the ventricular myocar-

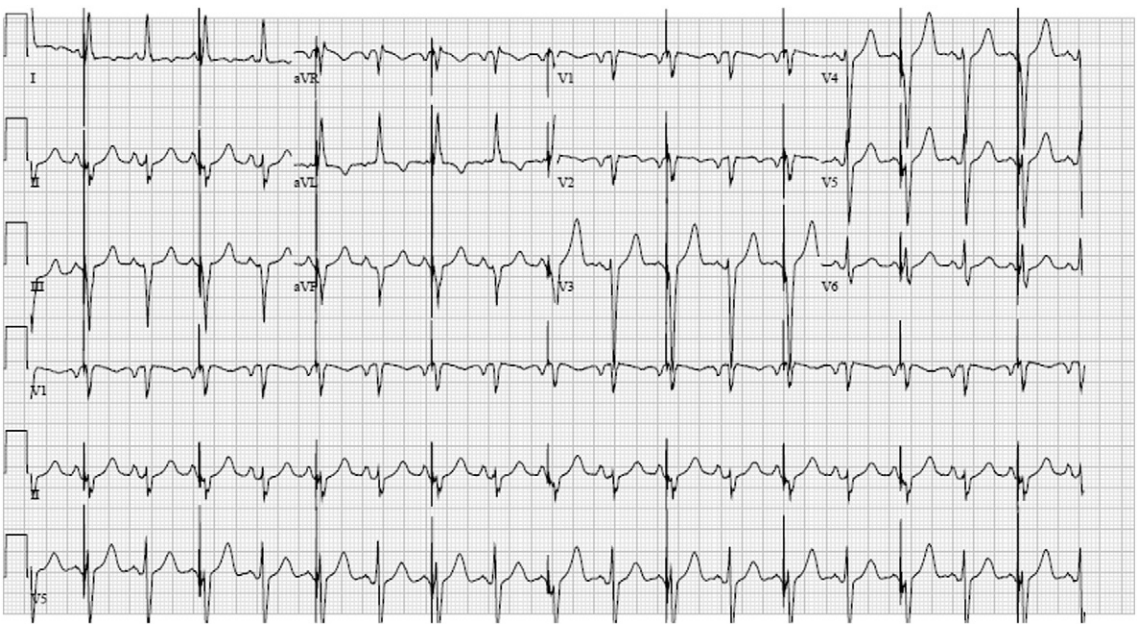

Fig. 2. Repeat ECG showing sinus tachycardia at a rate of 108 bpm and intermittent electronic ventricular pacing spikes.