

A Bigeminal Bradycardia

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A 76-year-old man presents to the ER complaining of fatigue, lethargy and recurrent lightheadedness over the preceding several days. He gives a history of diabetes, hypertension and heart failure and says he is taking heart medications but is unable to name them. On examination he is found to be extremely bradycardic with a pulse rate around 30 bpm. His initial ECG is shown (Figure 1).



Figure 1. ECG on presentation.

1. What is the cardiac rhythm?

2. What diagnosis should be suspected?

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This Month's ECG Diagnosis

1. The ECG shows a bigeminal rhythm of uncertain etiology. No P waves can be identified. The first QRS complex of each couplet shows an intraventricular conduction delay with an incomplete left bundle branch block pattern; the second is much wider, more bizarre and not in keeping with a typical bundle branch block. A reasonable working hypothesis is that the


rhythm is junctional with a pre-existing intraventricular conduction delay and ventricular extrasystoles with a fixed coupling interval, in a pattern of bigeminy. The patient's pulse rate is much slower than the actual ventricular rate because the closely coupled ventricular extrasystoles do not generate an adequate stroke volume.

2. In an elderly patient with symptomatic bradycardia, possible renal dysfunction and an uncertain medication burden drug toxicity should be considered. It was determined that the patient was taking digoxin and ramipril and he was subsequently found to have a serum creatinine level of 210 $\mu\text{mol/L}$. His serum digoxin level was markedly elevated at 5.7 mmol/L . He received prompt treatment with digoxin immune Fab and an ECG one hour later (Figure 2) showed a persisting junctional rhythm at 50 bpm with resolution of the extrasystoles. He subsequently reverted to controlled atrial fibrillation (AF), a rhythm which



Figure 2. Subsequent ECG

had been noted on previous ECGs and which was presumably the original indication for the prescription of digoxin.

Digoxin toxicity has been reported to cause almost any kind of cardiac arrhythmia with the possible exception of atrial flutter. The rhythm in Figure 2 is probably fine AF with complete heart block and a junctional escape rhythm. The bigeminal extrasystoles in Figure 1 are probably due to digoxin-triggered delayed after depolarizations secondary to intracellular calcium overload. Digoxin enhances myocardial excitability and automaticity and depresses atrioventricular conduction and so the combination of bradycardia or heart block and atrial or ventricular irritability should raise suspicion about possible digoxin toxicity. 

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