An 83-year-old woman with a history of atrial fibrillation and prior pacemaker implantation was brought to the emergency room with pacemaker non-capture, slow idioventricular rhythm, and cardiogenic shock. During resuscitation, it became apparent that the patient had been maintained on flecainide: 150 mg PO bid with no recent follow-up. The pacemaker was programmed to maximal unipolar output settings and vasopressors administered. Subsequent 3:1 capture (Figure 1) was noted without hemodynamic benefit. Serum potassium was normal, and mild acidosis was corrected with sodium bicarbonate. Magnesium sulfate was administered and an intra-aortic balloon pump was placed. Following these measures, the patient’s blood pressure stabilized, and improved pacemaker capture was observed, initially manifesting progressive latency from the pacemaker spike to the QRS onset with associated progressive QRS widening prior to block in a 3:2 Wenckebach pattern (Figure 2 - arrows). She gradually improved and sinus rhythm with 1:1 pacemaker capture was evident prior to discharge. Flecainide level obtained on admission was elevated (2.36 μg/dl).

This patient manifested hallmarks of flecainide toxicity, including marked bradycardia, pacemaker non-capture, QRS prolongation, and cardiovascular collapse. The cardiac rhythm disturbances observed mirrored the sodium channel-blocking electrophysiologic effects of flecainide, including slowing of conduction and action potential prolongation that is most pronounced on the infra-Hisian conduction system and ventricular myocardium.1 These effects are particularly demonstrated in the electrocardiogram showing Wenckebach periodicity with both progressive latency from the pacemaker spike to the QRS complex and increasing QRS widening evident. Toxic levels exacerbate these properties with the potential for elevation of pacing and defibrillation thresholds and cardiovascular collapse.2,3

Without prompt resuscitative maneuvers, mortality rates of over 20% have been reported.4 Our therapeutic approach mirrored previously described successful therapies including administration of intravenous sodium bicarbonate, as achieving an alkalotic state may be beneficial, and magnesium. Mechanical support is often essential until the severe clinical manifestations of drug toxicity resolve with either intra-aortic balloon pumping or, in non-responders, total cardiopulmonary support with an extracorporeal membrane oxygenation device.5

References
Figure 1: Rhythm strip after reprogramming pacemaker to maximal output and then higher rate. Pacemaker capture in a 3:1 pattern is evident with one ventricular escape complex present. The captured QRS complexes are significantly prolonged (large spikes due to unipolar pacing).

Figure 2: Twelve-lead electrocardiogram obtained early after successful resuscitation demonstrating intermittent pacemaker capture with progressively increasing latency from the pacing spike to the captured QRS complex and lengthening of the QRS duration prior to block in a 3:2 Wenckebach conduction pattern (arrows).