Fumble or Interception?
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An 85-year-old man is admitted to hospital following an acute left hemispheric stroke. As part of the management protocol, he is placed on cardiac telemetry and subsequently found to be intermittently bradycardic. The ECG shown in Figure 1 is obtained.

![Figure 1. Initial ECG.](image)

1. What abnormality of cardiac rhythm is shown?

The initial rhythm is sinus bradycardia at a rate of approximately 50 beats per minute (bpm). Following the first three beats, the rate slows somewhat and a rhythm with a different QRS morphology emerges at a rate of approximately 40 bpm. The initial QRS complexes show a normal duration with relatively high voltage in leads I and aVL and are accompanied by downsloping ST segment depression. These changes suggest hypertensive heart disease. The subsequent QRS complexes are abnormally wide, with a right bundle branch block (RBBB) morphology. This rhythm may be either a slightly accelerated idioventricular rhythm, or a junctional rhythm with associated RBBB. Although bradycardia-dependent RBBB may occur, it is not common and so an idioventricular rhythm is a much more likely diagnosis.
Figure 2. Rhythm strip from initial ECG showing a blocked atrial extrasystole.

2. **What is the likely etiology and clinical significance?**

Approximately 10% of stroke patients will demonstrate transient bradycardias, presumably due to imbalances of autonomic tone and relative vagotonia. It was established that this patient had been taking a beta-blocker for hypertension, an important contributor to the initial sinus bradycardia. The mechanism of the initiation and subsequent perpetuation of the idioventricular rhythm is interesting. Following the third QRS complex, a premature P wave indicating a blocked atrial extrasystole can be clearly seen (Figure 2). This resets the sinus node cycle length and the resultant pause is sufficiently long to permit the lurking idioventricular rhythm to emerge and capture the ventricles before the next sinus beat arrives. The sinus node is now relegated to spectator status, unable to regain control of the ventricles (see arrows in Figure 2). Eventually, the sinus node will regain capture of the ventricles when it arrives out-with the refractory period of the preceding QRS complex. We may consider the arrhythmia both a fumble and an interception; the collision of an atrial extrasystole and a drowsy sinus node provides an opportunity for the ventricles to run with the ball, albeit briefly.

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