

# Sudden Unexpected Acceleration

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### **Vignette**

An 83-year-old man presents with unstable angina, having experienced three prolonged episodes of chest pain at rest in the preceding 24 hours. His cardiac examination is considered normal and his initial electrocardiogram (ECG) is shown in Figure 1. He is admitted to hospital for observation and further investigation. Two days later, another ECG is obtained (Figure 2).

#### **Ouestions**

- 1. What is the initial cardiac rhythm?
- **2.** What is the explanation for the sudden increase in heart rate?

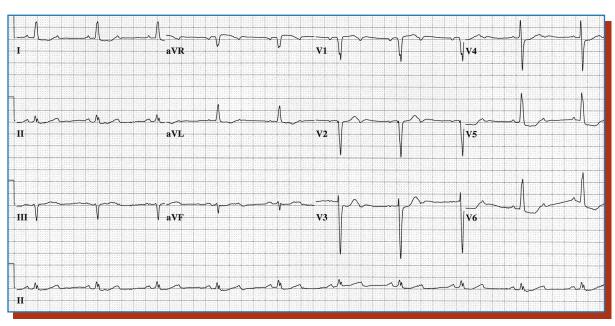


Figure 1. Initial ECG.

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## ECG clinic

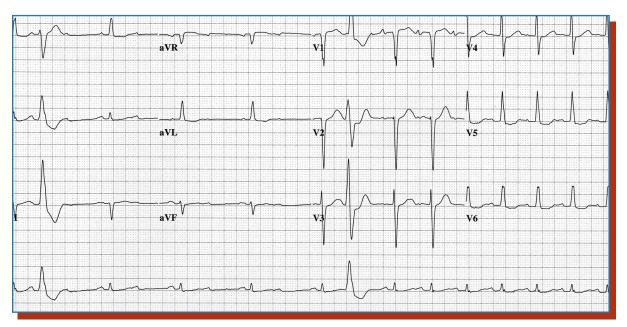


Figure 2. ECG two days after presentation.

#### **Answers**

- 1. The initial cardiac rhythm is sinus in origin, with a ventricular rate of 60 beats per minute (bpm). There is downsloping ST segment depression in the lateral leads, which may be due to ischemia, digitalis effect, or both. At first glance, this appears to simply be a sinus bradycardia, but the sudden increase in heart rate seen on the second ECG is difficult to reconcile with this interpretation. An important observation is that the increase in heart rate shown in Figure 2 is an exact doubling of the baseline heart rate recorded during the first half of the ECG.
- 2. Closer inspection of the ECG in Figure 1 reveals P waves concealed within the terminal portion of the T waves. This is best seen in limb lead 1 and chest leads  $V_5$  and  $V_6$ . The rhythm is indeed sinus, but the atrial rate is 120 bpm, not 60 bpm, and 2:1 atrioventricular (AV) block is present. A careful cardiac examination at this point might have detected regular cannon "A"

wave in the neck veins and an atrial filling sound in early diastole. The presence of the ventricular extrasystoles is probably an incidental finding, although one would have to consider the possibility of concealed His bundle extrasystoles masquerading as second-degree heart block. By rendering the AV node intermittently refractory, they could interfere with conduction of the sinus node impulses. Their occasional conduction to the ventricles could produce extrasystoles, as seen in this case.

There is no clearly defined explanation for the sudden improvement in AV node conduction seen here. However, the atrial rate in Figure 2 is slightly slower than in Figure 1, and is probably right at the threshold for 1:1 conduction. Slight changes in autonomic tone and the withdrawal of the patient's digoxin (his serum level was above the desired therapeutic range) are the likely explanation. Fortunately, unlike the phenomenon described in automobiles some years ago, the "sudden unexpected acceleration" seen in this case is without adverse consequences. Desired