Scenario: An 81-year-old female with a medical history of hypertension, type II diabetes mellitus, atrial fibrillation, congestive heart failure, and coronary artery disease comes to the emergency department with complaints of weakness following a syncopal episode. She had been in her normal state of health until after her doctor added an antihypertensive (verapamil) to her routine medications, which included metoprolol. She denies any chest pain or shortness of breath, but states that she feels very weak and a little nauseous.

For every ECG, we recommend that readers systematically examine the following 9 features (check all that apply):

1. Rate
   - Normal (60-90 beats per minute)
   - Bradycardia (<60 beats per minute)
   - Tachycardia (>90 beats per minute)

2. Rhythm
   - Regular
   - Irregular
   - Irregular-regular

3. P waves
   - One P wave for every QRS complex
   - Fewer P waves than QRS complexes
   - More P waves than QRS complexes
   - Cannot determine

4. PR interval
   - Normal (≤0.20 seconds)
   - Short (<0.11 seconds)
   - Lengthened (>0.20 seconds)
   - Cannot determine

5. QRS complex duration
   - Normal (≤0.12 seconds)
   - Wide (>0.12 seconds)

6. QRS complex direction lead V1
   - Negative and ≤0.20 seconds (normal)
   - Positive and >0.20 seconds
   - Cannot determine

7. ST segments
   - Normal
   - Elevated (≥2 mm)
   - Depressed (≥2 mm)
   - Elevation/depression 2 contiguous (side by side) leads (≥1 mm)

8. T wave
   - Normal
   - Inverted

9. QT
   - Normal
   - Lengthened (>0.47 seconds)

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Interpretation

Complete heart block with atrial tachycardia at 250/min and slow junctional escape rhythm at 30/min.

Rationale

The P waves of rapid atrial tachycardia can be appreciated in lead V1. Importantly, there is no relationship between the P waves and the QRS complexes; hence there is AV dissociation. The QRS complex is about 0.11 seconds wide, suggesting a junctional escape rhythm with an intraventricular conduction delay.

Calcium channel blockers (CCBs) and β-blockers are ubiquitous in clinical practice; however, in combination these may lead to cardiosuppression. The mechanism underlying cardiosuppression is decreased intracellular Ca²⁺ in the myocardium. Impaired impulse generation and conduction may act to slow the heart rate or cause atrioventricular nodal block. In this patient, the heart block and subsequent syncopal episode was likely triggered by the addition of verapamil.

Nursing Actions

Clinicians should be aware that this risk exists for any combination of CCB and β-blocker. Intravenous calcium gluconate is a proven, simple, and specific way to treat this complication because it acts as intracellular Ca²⁺, which then triggers a calcium-induced Ca²⁺ release from the sarcoplasmic reticulum, thus reversing metoprolol-verapamil induced complete heart block. Finally, this ECG illustrates the importance of multilead monitoring for accurate interpretation because, in many leads, the interpretation would be atrial standstill with a slow junctional escape rhythm.

ANSWERS

1. Rate
   - Normal (60-90 beats per minute)
   - Bradycardia (<60 beats per minute)
   - Tachycardia (>90 beats per minute)

2. Rhythm
   - Regular
   - Irregular
   - Irregular-regular

3. P waves
   - One P wave for every QRS complex
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   - Normal (≤0.12 seconds)
   - Wide (>0.12 seconds)

6. QRS complex direction lead V1
   - Negative and ≤0.12 seconds (normal)
   - Negative and >0.12 seconds
   - Positive and >0.12 seconds
   - Cannot determine

7. ST segments
   - Normal
   - Elevated (≥2 mm)
   - Depressed (≥2 mm)
   - Elevation/depression 2 contiguous (side by side) leads (≥1 mm)

8. T wave
   - Normal
   - Inverted

9. QTc
   - Normal
   - Lengthened (>0.47 seconds)
Pharmacological Cardiosuppression
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