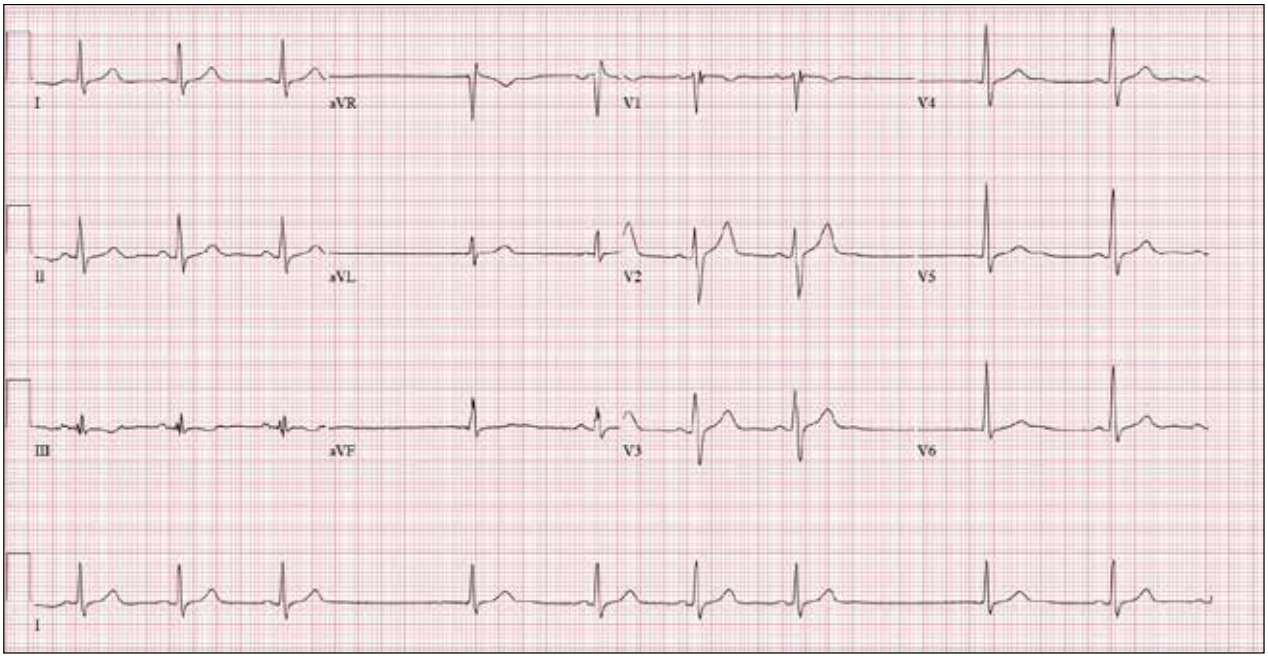
One Heck of a Check-up



A 63-year-old man with coronary artery disease (CAD) and New York Heart Association Class I heart failure presents for a routine semi-annual clinic visit. He was first diagnosed with CAD nine years ago, when he presented with exertional chest pain. Following an abnormal stress test, cardiac catheterization revealed moderate right CAD and severe left anterior descending artery disease. His left ventricular ejection fraction (LVEF) was 59%. Two stents were placed in the left anterior descending artery (LAD) and he was started on a ß-blocker, aspirin, clopidogrel, and a statin.

He remained stable for six years, but then his exertional angina returned. Repeat catheterization showed progressive disease in the right coronary artery (RCA) with new disease in an obtuse marginal (OM) branch of the circumflex artery. His LVEF had also diminished to 38%. Stents were placed in the RCA and OM arteries, and his ß-blocker dose was increased.

Today, he reports that over the past six months, his heart rate has been slow and often skips beats. He stopped taking his ß-blocker, hoping it would help speed up his heart; it didn't. He says he feels fine right now (although his heart continues to skip beats). He says he can climb a flight of stairs without difficulty and denies chest pain, dyspnea, dizziness, or syncope. At night, he sleeps on one or two pillows. He denies orthopnea, paroxysmal nocturnal dyspnea, or peripheral edema.

Medical history includes type 2 diabetes (controlled with diet and exercise), degenerative joint disease, and hyperlipidemia. Surgical history includes a tonsillectomy and trigger finger repair of the left third digit. His current medications include clopidogrel, losartan, pravastatin, spironolactone, and aspirin. He has no known drug allergies.

The patient used to be a heavy smoker (> 2 packs/d) but quit after his first stent was placed. He drinks one to two glasses of wine per week and denies recreational drug use.

Family history is positive for coronary artery disease and stroke. His father died of CAD at age 60, and his mother of heart failure at 58.

A review of systems is noncontributory.

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Vital signs include a blood pressure of 118/80 mm Hg; pulse, 53 beats/min; temperature, 37°C; respiratory rate, 14 breaths/min^{-1}; and O_2 saturation, 96% on room air. His weight is 214 lb and his height, 70 in.

Physical exam reveals an alert, well-kept male in no distress. Pertinent findings include a regularly irregular pulse and normal S_1 and S_2, with no murmurs, gallops, or rubs. The lungs are clear bilaterally. The abdomen is soft and benign with no organomegaly. Peripheral pulses are 2+ bilaterally, and there is no peripheral edema or calf tenderness. Neurologic exam shows a grossly intact sensory and motor system with no focal signs.

An ECG reveals a ventricular rate of 55 beats/min; PR interval, 146 ms; QRS duration, 122 ms; QT/QTc interval, 424/405 ms; P axis, 60°; R axis, 38°; and T axis, 29°. What is your interpretation?

ANSWER

The correct interpretation includes sinus bradycardia with marked sinus arrhythmia and junctional escape beats, and an intraventricular conduction delay.

This type of ECG is not ideal for calculating the ventricular rate via the 30/150/100 method. An easier method is to multiply the number of QRS complexes in the rhythm strip by six (an ECG at standard paper speed takes 10 s; 6 × 10 s = 60 s). In the absence of a permanent pacemaker, variation of a few beats/min from the computer reading is acceptable. In this case, multiplying 9 x 6 yields a rate of 54 beats/min (close to the computer reading of 55) and reveals sinus bradycardia.

Looking at the lead I rhythm strip, notice that while the QRS complexes in the fourth and eighth beats look similar to the others, they are not preceded by P waves; the T waves of these two beats are also not similar to the others. These represent junctional escape beats, with a possible retrograde P wave in the T-wave complex. The long pauses and the absence of a P wave prior to the fourth and eighth beats make this a sinus arrhythmia.

The diagnosis of an intraventricular conduction delay can be made by the duration of the QRS complex (122 ms), which is above normal limits. This ECG does not meet the clear criteria for a right bundle branch block (QRS ≥ 120 ms, terminal broad S wave in lead I, RSR' in V_1) or left bundle branch block (QRS ≥ 120 ms, ST depressions and inverted T waves, particularly in I, aVL, V_5, and V_6). **CR**