

Item 33

A 28-year-old man is evaluated in the office for a 5-year history of palpitations. These episodes used to occur once or twice a year, but over the past 6 months he has been experiencing them on a monthly basis. He reports that his heart starts “racing” suddenly for no reason, and the episode usually terminates abruptly after he takes a few deep breaths. Episodes typically last 10 to 15 minutes, although one episode last month lasted 30 minutes. He is otherwise healthy, has no other symptoms, and takes no medications. Physical examination is normal.

A baseline electrocardiogram is shown.

Which of the following is the most likely cause of his arrhythmia?

- (A) Atrial flutter
- (B) Atrial tachycardia with block
- (C) Atrioventricular re-entrant tachycardia (Wolff-Parkinson-White syndrome)
- (D) Atrioventricular nodal re-entrant tachycardia

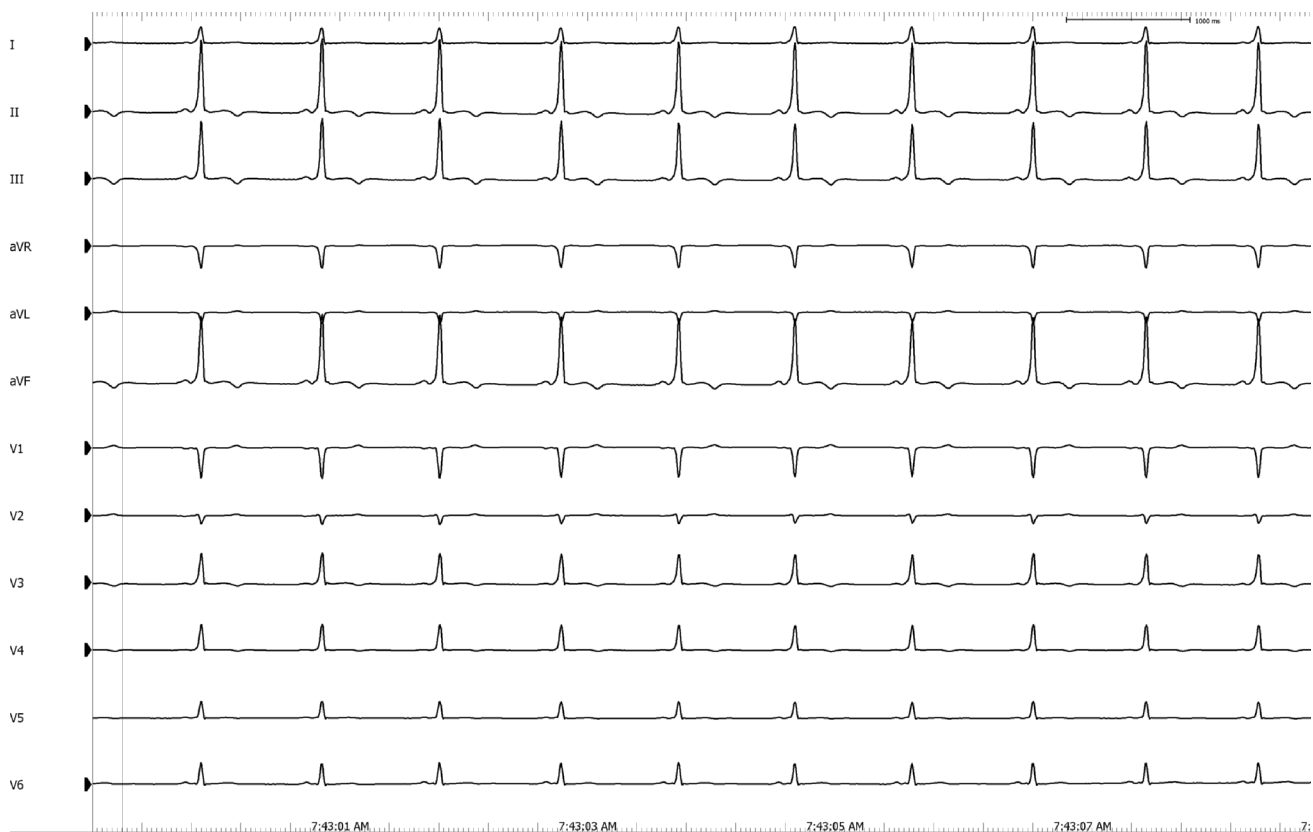
Item 34

A 50-year-old man is evaluated during a routine follow-up office visit for heart failure, which was diagnosed 1 year ago. A stress test at the time of diagnosis was negative for ischemia. At his most recent evaluation 4 months ago, an echocardiogram showed left ventricular enlargement and hypertrophy, a left ventricular ejection fraction of 40%, and no significant valvular disease. An electrocardiogram was unchanged, showing left ventricular hypertrophy but no evidence of previous myocardial infarction. The patient is currently asymptomatic, and his medications are hydrochlorothiazide and lisinopril.

On physical examination, heart rate is 85/min and blood pressure is 135/85 mm Hg. There is no jugular venous distention or peripheral edema. The lungs are clear. There is a soft S₄ but no murmur.

Which of the following medications should be added to the patient’s regimen?

- (A) Carvedilol
- (B) Digoxin
- (C) Diltiazem
- (D) Losartan
- (E) Spironolactone



Item 33

Item 35

A 22-year-old man is evaluated in the emergency department for a rapid heart rate and lightheadedness. He reports episodes of a “racing heart” a few times each year since his early teens. He describes today’s episode as different: it started as one of his regular episodes but then became erratic. In addition, the lightheadedness has never happened before. He is otherwise healthy and takes no medications.

On physical examination, the patient is diaphoretic. Blood pressure is 72 mm Hg palpable. His lungs are clear, and cardiac examination demonstrates a rapid irregular rhythm with no murmurs. The electrocardiogram is shown.

Which of the following is the most appropriate therapy for this patient?

- (A) Direct-current cardioversion
- (B) Intravenous procainamide
- (C) Intravenous verapamil
- (D) Overdrive atrial pacing

Item 36

A 43-year-old woman with idiopathic cardiomyopathy (most recent left ventricular ejection fraction, 30%) is evaluated in the office. She has fatigue and shortness of breath with minimal activity. Her medications are lisinopril, 2.5 mg daily; sustained-release metoprolol, 12.5 mg daily; digoxin, 0.125 mg daily; and furosemide, 80 mg daily.

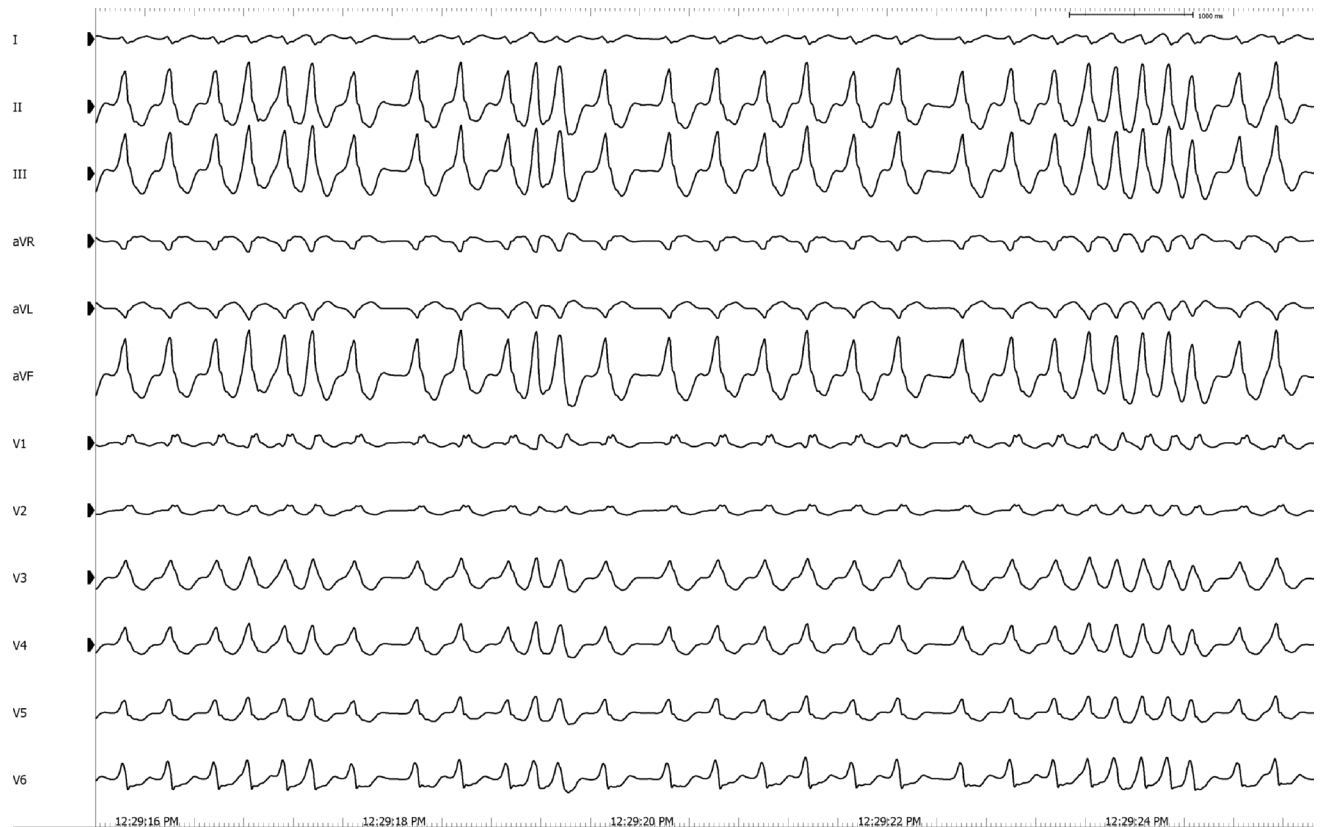
On physical examination, blood pressure is 110/75 mm Hg and heart rate is 94/min. There is jugular venous distention to 2 cm above the clavicle at a 45-degree incline. Cardiac rhythm is regular, with normal S₁ and S₂ and no gallops. There is no peripheral edema.

Laboratory studies:

Blood urea nitrogen	43 mg/dL
Potassium	5.1 meq/L
Creatinine	2.7 mg/dL
Digoxin	0.9 µg/L

Which of the following would be the most appropriate medication change?

- (A) Increase digoxin
- (B) Increase furosemide
- (C) Increase metoprolol
- (D) Start spironolactone



Item 35

tomatic heart failure in the absence of other indications (such as rate control for atrial fibrillation). Spironolactone is indicated for patients with heart failure who have severe symptoms (New York Heart Association class III or IV).

KEY POINT

- **Angiotensin-converting enzyme inhibitors are indicated for all patients with systolic heart failure, regardless of ejection fraction or functional status, barring contraindications.**

Bibliography

1. Dagenais GR, Pogue J, Fox K, Simoons ML, Yusuf S. Angiotensin-converting-enzyme inhibitors in stable vascular disease without left ventricular systolic dysfunction or heart failure: a combined analysis of three trials. *Lancet*. 2006;368:581-8. [PMID: 16905022]

Item 33 Answer: C

The combination of manifest pre-excitation (short PR segment and delta wave [slurred initial upstroke of the QRS complex]) in this patient's baseline electrocardiogram plus tachycardia establishes the diagnosis of the Wolff-Parkinson-White syndrome, which can cause an atrioventricular (AV) re-entrant tachycardia. AV re-entrant tachycardia is a bypass-tract-mediated re-entrant tachycardia, in which the anterograde conduction (atria-to-ventricle) is typically via the AV node, and retrograde conduction is via the bypass tract. Because bypass-tract conduction is typically faster than conduction via the AV node, during episodes of atrial tachycardia, atrial activation occurs rapidly after the QRS complex, resulting in a "short RP" tachycardia and the P wave is usually located within the ST segment. Because the ventricle is activated normally during tachycardia, the QRS complex is narrow. Patients with Wolff-Parkinson-White syndrome should be offered radiofrequency catheter ablation as first-line therapy. This recommendation is based on the high success rate of catheter ablation and the presence of a small but persistent risk of sudden cardiac death if the arrhythmia is untreated.

Sinus tachycardia, AV nodal re-entrant tachycardia, atrial tachycardia, and atrial flutter can cause paroxysmal episodes of palpitations, but none of these conditions is associated with a short PR segment and delta wave.

KEY POINT

- **Atrioventricular re-entrant tachycardia (Wolff-Parkinson-White syndrome) is characterized electrocardiographically by a short PR segment, delta wave, and tachycardia.**

Bibliography

1. Esberger D, Jones S, Morris F. ABC of clinical electrocardiography. Junctional tachycardias. *BMJ*. 2002;324:662-5. [PMID: 11895828]

Item 34 Answer: A

Treatment with an angiotensin-converting enzyme (ACE) inhibitor and a β -blocker is indicated for all patients with any degree of systolic heart failure, including this asymptomatic patient with a low ejection fraction, because treatment with

both agents has been shown to reduce morbidity and mortality.

Losartan, an angiotensin-receptor blocker (ARB), is an acceptable alternative in a patient who cannot tolerate an ACE inhibitor, but there is no benefit to adding an ARB to an ACE inhibitor. Calcium-channel blockers are indicated in patients with heart failure who have hypertension or angina that is not adequately controlled with an ACE inhibitor or β -blocker. First-generation calcium-channel blockers, such as nifedipine, diltiazem, and verapamil, cause a reactive increase in sympathetic activity in response to peripheral vasodilatation and negative inotropic effects, whereas second-generation calcium-channel blockers, such as amlodipine, are more vasoselective, less cardiodepressant, and do not appear to have a deleterious effect on outcome in patients with heart failure. Spironolactone and digoxin are not indicated for patients with asymptomatic systolic heart failure. Spironolactone reduces mortality in patients with severe symptomatic heart failure (New York Heart Association class III or IV) and a left ventricular ejection fraction $\leq 35\%$. Digoxin alleviates symptoms and reduces hospitalizations related to heart failure, but has not been shown to reduce mortality.

KEY POINTS

- **An angiotensin-converting enzyme inhibitor and a β -blocker are indicated in all patients with systolic heart failure, including asymptomatic patients with low ejection fractions.**
- **Spironolactone and digoxin are not indicated in patients with asymptomatic systolic heart failure.**

Bibliography

1. O'Connor CM. The new heart failure guidelines: strategies for implementation. *Am Heart J*. 2007;153:2-5. [PMID: 17394896]

Item 35 Answer: A

This patient is unstable, as demonstrated by his symptoms and low blood pressure. In unstable patients, electrical cardioversion should always be considered the treatment of choice regardless of the tachycardia mechanism. This electrocardiogram is consistent with atrial fibrillation in a patient with an accessory atrioventricular pathway. In patients with atrial fibrillation and an accessory pathway (bypass tract), the rapid atrial impulses travel to the ventricle through both the normal conduction system and the accessory pathway. Because of the chaotic electrical activity in the atria, the degree of conduction via both pathways is quite variable. Therefore, the electrocardiogram shows variable degrees of pre-excitation (manifested by variable QRS complex widths) and great irregularity.

Verapamil and other atrioventricular nodal blocking drugs should not be given to patients with pre-excitation tachycardias, especially pre-excitation atrial fibrillation, because blocking the atrioventricular nodal conduction may enhance conduction down the accessory pathway, leading to faster heart rates and, possibly, hemodynamic collapse. For such patients, drugs capable of directly slowing conduction on the pathway,

such as procainamide and ibutilide, are the best options, provided the patient is stable and does not require immediate cardioversion. Overdrive atrial pacing is not possible; this patient is in atrial fibrillation, therefore, pacing would not result in capture of the electrical impulse.

KEY POINT

- Tachycardic patients who are unstable should undergo immediate direct-current cardioversion.

Bibliography

1. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, Alpert JS, Calkins H, Camm AJ, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias). *Circulation*. 2003;108:1871-909. [PMID: 14557344]

Item 36 Answer: C

In general, β -blockers for the treatment of systolic heart failure should be up-titrated toward the doses used in clinical trials, with the aim of achieving the morbidity and mortality benefits demonstrated by such increases. However, this dose increase may be limited by the patient's blood pressure, heart rate, or symptoms. There is evidence that heart rate response to the dose of β -blocker may be more indicative of an adequate level of treatment than the absolute dose itself. This patient is on the lowest dose of sustained-release metoprolol, and her blood pressure and heart rate are well within ranges that would tolerate a higher dose at this time. In contrast, there is no definitive dose-dependent benefit when using angiotensin-converting enzyme inhibitors, and the patient's elevated serum potassium and creatinine concentrations limit the ability to increase the dose.

Although spironolactone is indicated for patients with severe heart failure, it is important to be aware of the other criteria for using spironolactone: left ventricular ejection fraction $\leq 35\%$, serum creatinine ≤ 2.5 mg/dL, and serum potassium ≤ 5 meq/L. This patient's creatinine and potassium levels are too high to start spironolactone. Her digoxin level is therapeutic, and thus there is no need to increase the digoxin dose; in fact, higher therapeutic levels of digoxin have been associated with greater mortality. The physical examination and laboratory test results suggest a stable state of euolemia, and there is no need to change the furosemide dose at this time.

KEY POINT

- In patients with systolic heart failure, β -blocker therapy should be titrated to heart rate.

Bibliography

1. O'Connor CM. The new heart failure guidelines: strategies for implementation. *Am Heart J*. 2007;153:2-5. [PMID: 17394896]

Item 37 Answer: D

This patient has ventricular tachycardia. Ventricular tachycardia is defined as three or more consecutive beats originating below the atrioventricular node (wide-complex beats not associated with a conducted P wave), with a heart rate >100 – 120 /min. Patients with sustained ventricular tachycardia in the setting of significant structural heart disease have a high risk of future recurrence, with a mortality rate as high as 25% per year. An implantable cardioverter-defibrillator reduces sudden cardiac death in patients with ventricular fibrillation or sustained ventricular tachycardia associated with hemodynamic compromise and is superior to amiodarone therapy. It is therefore recommended in this population unless there is a contraindication.

The electrocardiogram for a patient with atrial fibrillation shows a rapid, irregularly irregular rhythm with no discernible P waves and atrial fibrillatory waves at a rate between 350 and 600/min. The fibrillatory waves vary in amplitude, morphology, and intervals, creating a rough, irregular baseline between the QRS complexes. Unless there is co-existing bundle branch block, the QRS complex is narrow. Electrocardiographically, left bundle branch block is associated with absent Q waves in leads I, aVL, and V₆; a large, wide, and positive R wave in leads I, aVL, and V₆ (“tombstone” R waves); and prolongation of the QRS complex to >0.12 sec. Repolarization abnormalities are present consisting of ST segment and T wave vectors directed opposite to the QRS complex. In right bundle branch block, lead I will show a small Q wave and tall R wave; lead V₆ will show a small positive R wave and a small negative S wave followed by a large positive deflection (the “rabbit ear”). There is ST depression and T wave inversion in right precordial leads and upright T waves in left precordial and limb leads. The QRS complex is >0.12 sec.

KEY POINTS

- Patients with sustained ventricular tachycardia in the setting of significant structural heart disease are at high risk of future recurrence with a high mortality rate.
- An implantable cardioverter-defibrillator improves survival in patients with sustained ventricular tachycardia and concomitant structural heart disease.

Bibliography

1. Kokolis S, Clark LT, Kokolis R, Kassotis J. Ventricular arrhythmias and sudden cardiac death. *Prog Cardiovasc Dis*. 2006;48:426-44. Erratum in: *Prog Cardiovasc Dis*. 2006;49:58. [PMID: 16714162]

Item 38 Answer: D

The principles of management of heart failure in the pregnant patient parallel therapy in the nonpregnant patient, with the modification that some medications should be avoided during pregnancy. This patient with a low left ventricular ejection fraction should be started on afterload reduction therapy. Because angiotensin-converting enzyme inhibitors may cause