A 32-year-old man presented to the emergency department with progressive dyspnea on exertion, light-headedness, and fatigue. The patient was an avid runner and owned a gymnasium where he worked as a personal trainer. His medical history was notable for asymptomatic paroxysmal supraventricular tachycardia diagnosed at 15 years of age that resolved spontaneously, and a lumbosacral radiculopathy, which he managed with physical therapy and exercise. He took ibuprofen only as needed for his radiculopathy. He grew up in Minnesota and never lived abroad.

Three days before presentation, he ran his usual 2 miles without any difficulty. However, 2 days before presentation, he began experiencing shortness of breath precipitated by mild physical activity. The day before presentation, he became markedly lightheaded and “almost fainted” while he was coaching wrestling. This 5-minute episode was associated with tunnelled vision and resolved spontaneously without any loss of consciousness. He denied any chest pain radiating to the jaw, the shoulder, or the back. He also denied any palpitations, concomitant nausea, diaphoresis, weight gain, lower extremity swelling, paroxysmal nocturnal dyspnea, or orthopnea.

His family history was negative for sudden cardiac death. Although he did spend some time outside running, landscaping, and doing yard work, he did not recall having a tick attached to his skin and he denied arthralgias, fevers, or rashes.

In the emergency department, he was afebrile, with a heart rate of 43 beats/min, blood pressure of 121/68 mm Hg, a respiratory rate of 16 breaths/min, and saturation of 99% on room air. He had a body mass index of 25.1 kg/m². On physical examination, the patient was comfortably lying in bed and in no acute distress. His head, ear, eye, nose, and throat examinations were unremarkable. His neck examination revealed neither jugular venous distension nor lymphadenopathy. His cardiovascular examination disclosed bradycardia with a regular rhythm and without any murmurs, rubs, or gosples. His peripheral pulses were palpable and strong. His extremities were warm and well perfused without any cyanosis or edema. His lungs were clear to auscultation bilaterally without any adventitious sounds. His abdominal examination was remarkable for positive bowel sounds, mild right upper quadrant tenderness to palpation without organomegaly, guarding, or rebound tenderness.

1. On the basis of the patient’s history and physical examination findings, which one of the following is the best initial test to order?
   a. Complete blood count test
   b. Baseline troponin I test
   c. Twelve-lead electrocardiography
   d. Chest radiography
   e. D-dimer test

Although a complete blood count test would be part of any initial work-up of dyspnea to rule out anemia, it may not be the first test to order in a bradycardic patient with presyncopal symptoms. A troponin I test is a great second step to investigate acute coronary syndrome (ACS), but the latter is unlikely in an otherwise healthy 32-year-old patient. Twelve-lead electrocardiography has the highest yield in delineating the etiology of the bradyarrhythmia possibly underlying
the shortness of breath and presyncopal symptoms. It is also the initial test of choice to investigate ACS. Chest radiography would be an ideal first test if the suspicion for an urgent structural cardiopulmonary etiology, such as pneumothorax or cardiac tamponade, was high. Similarly, a D-dimer test would be appropriate to work up shortness of breath if the index of suspicion for pulmonary embolism was high in a patient with a low pretest probability. However, in this patient, without a clear history of coagulopathy, recent immobility or major/orthopedic surgery, pulmonary embolism is much less likely. Also, note that the normal respiratory rate and normal oxygenation argue against a pulmonary embolism diagnosis and therefore the immediate use of chest radiography or a D-dimer test.

His electrocardiogram revealed a ventricular rate of 41 beats/min (60 to 100 beats/min) with a complete atrioventricular (AV) dissociation consistent with sinus rhythm with complete heart block (CHB), left axis deviation, and a prolonged QRS complex at 184 milliseconds in a left bundle branch pattern. His work-up disclosed the following results (reference ranges within parentheses): potassium level, 4.9 mmol/L (3.6 to 5.2 mmol/L); sodium level, 139 mmol/L (135 to 145 mmol/L); magnesium level, 2 mg/dL (1.7 to 2.3 mg/dL); blood urea nitrogen level, 17 mg/dL (8 to 24 mg/dL); creatinine level, 1.31 mg/dL (0.74 to 1.35 mg/dL); glucose level, 96 mg/dL (70 to 140 mg/dL); hemoglobin level, 14.1 g/dL (13.2 to 16.6 g/dL); and leukocyte count, (6.8×10^9/L (3.4 to 9.6)×10^9/L). His serum lactate level, serum lipase level, and hepatic functional panel were unremarkable. The erythrocyte sedimentation rate was 12 mm/h (0 to 22 mm/h), and the C-reactive protein level was 14.7 mg/L (<8 mg/L). The ferritin level was normal at 128 μg/L (24 to 336 μg/L). The fifth-generation troponin T level was less than 6 ng/L (≤15 ng/L) at baseline and at 2 hours. His chest radiography revealed a cardiac silhouette at the upper limit of normal without pulmonary findings.

2. On the basis of the above findings, which one of the following tests would most likely yield a positive result?
   a. Serum tick-borne panel polymerase chain reaction
   b. *Trypanosoma cruzi* IgG level
   c. High Fe gene mutation
   d. Positive antistreptolysin O titers
   e. Lyme disease IgM

In a clinical setting, polymerase chain reaction can be used to detect *Borrelia burgdorferi* DNA in cerebrospinal and synovial fluids, but not in blood or urine. This is thought to be due to the transient blood-borne phase of the spirochete *B burgdorferi* and its low concentration. Patients with Chagas cardiomyopathy, caused by *T. cruzi* infection, can present with AV block. However, it is highly unlikely in a young Midwestern patient who has not traveled to a Chagas endemic region, such as South America. High Fe gene mutation can be seen in patients with hemochromatosis, which is unlikely to manifest in a young patient with a normal ferritin level. Antistreptolysin O titers can be positive in rheumatic heart disease caused by *Streptococcus pyogenes* infection, a disease now rarely encountered in a developed country and unlikely in a young man without an opening snap of thickened mitral valve leaflets auscultated on physical examination. In a patient who is active outdoors in a Lyme endemic region presenting with CHB, Lyme disease should be ruled out with serology.

The patient was admitted to the cardiac care unit for continued cardiac monitoring and further management and stabilization. Indeed, the patient’s Lyme disease IgG and IgM were both positive, consistent with an active *B burgdorferi* infection.

3. Which one of the following is the most appropriate next step in managing this patient’s acute presentation?
   a. Cardiac biopsy
   b. Intravenous (IV) ceftriaxone
   c. Oral doxycycline
   d. Intravenous atropine
   e. Permanent pacing
Cardiac sarcoidosis can present with conduction abnormalities and is usually diagnosed first with cardiac magnetic resonance and/or 18F-fluorodeoxyglucose-pet emission tomography. Cardiac biopsy may be appropriate if the suspicion for sarcoidosis remains high with undiagnostic imaging. However, cardiac biopsy is quite invasive, and in a patient with CHB and a positive Lyme serology, disseminated Lyme carditis can be diagnosed without cardiac biopsy. For Lyme carditis patients with symptoms such as dyspnea on exertion or presyncope, first-degree AV block with PR interval greater than 300 milliseconds, or second- or third-degree AV block, IV antibiotics (such as ceftriaxone 2 g IV daily) are recommended as initial therapy. Oral antibiotics, such as doxycycline, amoxicillin, or cefuroxime axetil are appropriate options for asymptomatic patients, low-grade AV block, or PR interval less than 300 milliseconds. Note that there is no evidence that IV antibiotics are superior to oral antibiotics for cardiac manifestations of Lyme disease. Atropine is an anticholinergic agent used to block vagal stimulation of the sinoatrial node and slow down conduction through the AV node. Atropine can therefore be used for symptomatic sinus bradycardia or in cases of hemodynamic instability due to bradycardia, but its use in third-degree AV block may be dependent on the region of the blockage. According to the American College of Cardiology/American Heart Association, patients with reversible causes of AV block should first be treated medically before being evaluated for a permanent pacemaker.

The patient was initiated on ceftriaxone. The next morning, a repeat electrocardiogram revealed sinus rhythm with 2:1 AV block and left bundle branch block, reverting to CHB in the evening. Because of persistent bradycardia with a heart rate of 32 beats/min at rest, he received a temporary-permanent pacemaker to assist with backup pacing, which enabled him to ambulate and rehabilitate. Transthoracic echocardiography revealed borderline global hypokinesis and mild left and right ventricular enlargement with an intact left ventricular ejection fraction of 51%. Over the next 2 days, the patient’s heart rhythm converted to first-degree AV block with a PR interval of 234 milliseconds and right bundle branch block pattern. He then underwent a treadmill exercise test to assess AV conduction and tendency for AV block with exertion, which revealed no evidence of conduction abnormalities.

4. Which one of the following is the most likely course of disease in this patient?
   a. Complete AV block persistence for at least 6 months
   b. Conduction abnormalities resolution within 6 weeks
   c. Heart failure with reduced ejection fraction
   d. Intermittent attacks of frank arthritis starting a few years after treatment
   e. Sudden cardiac death is expected

High-grade AV block in Lyme disease tends to resolve within 7 days of appropriate treatment, which seems to be the case with our patient. Indeed, conduction abnormalities, including minor ones, usually resolve by 6 weeks. In Europe, cases have been reported in which chronic dilated cardiomyopathy has been associated with B. burgdorferi, but no such cases have been reported in the United States. Post-Lyme disease syndrome constitutes a constellation of nonspecific symptoms, such as headache, arthralgias, and fatigue, which usually resolve within 6 to 12 months after the initiation of treatment. According to a report by the Centers for Disease Control and Prevention in 2013, 3 deaths were attributed to Lyme carditis between November 2012 and July 2013, representing an extremely low overall carditis-related mortality.

Given the patient’s stability and the unremarkable nature of his exercise stress testing, the pacemaker was removed and the patient was dismissed from the hospital. The patient was switched to oral doxycycline 100 mg twice daily to complete a 21-day course.
5. Which **one** of the following is the **most appropriate** piece of advice you can give the patient to prevent another episode of a tick-borne illness?

- a. Wear clothing treated with 0.5% permethrin
- b. Check your body for ticks weekly
- c. Refrain from using picaridin-containing repellents
- d. Shower within 24 hours after hiking
- e. The “hot match” method is the best way to remove ticks without leaving the mouthparts in the host skin

According to the Centers for Disease Control and Prevention, permethrin-treated clothes, boots, and camping gear can help prevent tick bites and they remain protective after several washings. Rather than weekly, checking your body for ticks daily can help lower the likelihood of contracting a tick-borne illness. Insect repellents with picaridin, oil of lemon eucalyptus, para-methane-diol, 2-undecanone, ethyl butylacetylaminopropionate, or N,N-diethyl-m-toluamide that are Environmental Protection Agency-registered are effective at preventing tick bites. Taking a shower within 2 hours after coming indoors has been proven effective at reducing the risk of acquiring Lyme disease as it may help rinse off any unattached ticks. Among the 5 methods of tick removal—petroleum jelly, fingernail polish, 70% isopropyl alcohol, a hot kitchen match, or tweezers—pulling up firmly and gently without twisting or jerking was the most effective method of removal without leaving mouthparts on the skin, not the hot match method.

The patient was seen by his primary care physician 1 week after dismissal from the hospital, and he was found to have no symptoms with a benign physical examination.

**DISCUSSION**

Although Lyme carditis may not be common, CHB with resultant dyspnea and presyncope can certainly be encountered by internists. Therefore, keeping CHB on the list of differential diagnoses of dyspnea can be helpful.

Complete heart block, or third-degree (complete) AV block, is a condition in which the atrial impulse does not travel to the ventricle because of an anatomical or functional block in the AV node, in the bundle of His, or below in the Purkinje system. The hallmark of CHB is complete AV dissociation. Therefore, the electrical activity of the atria is controlled by the sinoatrial node or an ectopic focus while the electrical activity of the ventricles is controlled by a focus located usually just below the area of the block.

Overall, conduction system fibrosis and/or sclerosis (Lenègre disease) or calcification (Lev disease) accounts for more than 30% of all degrees of acquired AV block. In terms of CHB, the etiology can be divided into pathophysiologic and iatrogenic causes. The AV conduction system contains cholinergic receptors, which makes it susceptible to increased vagal tone. Coronary arterial disease, whether acute or chronic, can result in infarcted conduction system with CHB. In fact, the prevalence of CHB in acute myocardial infarcts is 7%. Viral and bacterial infections, such as with *B burgdorferi,* may also present with conduction abnormalities. Almost 5% of patients with sarcoidosis present with cardiac involvement alone. Additional causes include other infiltrative diseases, such as amyloidosis and hemochromatosis; metabolic abnormalities, such as hyperkalemia and thyroid disease; and congenital heart disease, which is usually irreversible. Interestingly, almost 90% of isolated congenital CHB cases occur in neonates of mothers with anti-Sjögren-syndrome-related antigen A or anti-Sjögren-syndrome-related antigen B antibodies. Iatrogenic etiologies include the use of agents with AV blocking properties, such as β-blockers, nondihydropyridine calcium channel blockers, adenosine, or digoxin; postcardiac surgery; or catheter-directed interventions. Nevertheless, despite characterizing a number of etiologies for CHB, more than 50% are deemed idiopathic.

The slow ventricular escape rhythm (usually ≤40 beats/min) leads to decreased cardiac output, which causes the classic...
symptoms of fatigue, dyspnea, presyncope/syncope, chest pain, or sudden cardiac death. The first step in evaluation should include a thorough history, delineating the patient’s comorbidities, such as coronary artery disease, recent exposures to ticks in endemic regions, medications, procedures, congenital heart disease, or thyroid disease. On physical examination, most patients can present with bradycardia. Other findings include appearing pale, with cold and diaphoretic skin due to decreased cardiac output.

The main diagnostic tool for diagnosing CHB is a 12-lead electrocardiogram, in which the atrial P wave and the ventricular QRS wave are completely dissociated. As a rule of thumb, the more distal the blockage in the conduction pathway, the wider the QRS wave.

The management of patients with third-degree AV block involves hospitalizing them and determining whether a patient is hemodynamically stable. If unstable, atropine with transcutaneous pacing is usually used. Transvenous pacing is more comfortable to patients, and it should be pursued when available. In the setting of associated hypotension or heart failure, dopamine or dobutamine should be administered, respectively. In hemodynamically stable patients, the initial effort is to look for reversible causes while ensuring placement of transcutaneous pads in case of clinical deterioration. If CHB persists, as it did in our patient, a temporary/permanent pacemaker may be needed. If ACS is thought to be the underlying etiology, reperfusion may reverse AV block. Atrioventricular-slowing medications should be held. In addition, in cases of hyperkalemia or hypothyroidism, hyperkalemia management and thyroid hormone replacement should be implemented, respectively. In cases of Lyme carditis, IV antibiotics should be administered.

In summary, CHB is a cause of dyspnea, fatigue, presyncope, or sudden cardiac death. The underlying etiology can be reversible, such as infectious or electrolyte abnormalities, or irreversible, such as infiltrative diseases. Diagnosis heavily relies on the electrocardiogram, and management involves admitting the patient for cardiac monitoring while searching for reversible etiologies. At a minimum, transcutaneous pacing pads should be in place while the work-up is completed. If no reversible cause is found, a permanent pacemaker should be implemented.

Potential Competing Interests: Dr Chen reports being a cofounder of Zumbro Discovery, receiving grants from Scios, and receiving royalties from UpToDate and that his institution has patents for designer natriuretic peptides. The other authors report no competing interests.

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CORRECT ANSWERS: 1. c. 2. e. 3. b. 4. b. 5. a