left atrial cavity function was studied by Doppler interrogation of mitral inflow after cardioversion of atrial fibrillation, with greater and more prolonged depression of atrial function occurring in patients who had electrical versus chemical cardioversion. Because TEE was only performed before cardioversion in that study, the appraisal of SEC after cardioversion was not accomplished. In each of these studies, the causal relation of electric cardioversion to impaired atrial and LAA function after the procedure was largely circumstantial.

The results of the two case studies reported here demonstrate that worsening LAA function occurs after *spontaneous* conversion from atrial fibrillation and flutter to sinus rhythm, independent of electric or chemical therapeutic interventions. Moreover, SEC can intensify after spontaneous conversion from atrial fibrillation to sinus rhythm, again without the interference of electric or chemical intervention. Therefore, it appears as though the mere conversion to sinus rhythm may be sufficient to predispose the atrial appendage to thrombogenesis and thromboembolism. Increased vagal tone and/or an atrial myopathy may be underlying causes of this phenomenon, although a contributory role for electric and/or chemical effects cannot be excluded.

The clinical implications of these case studies are pertinent to the embolic risk associated with cardioversion of atrial arrhythmias and to the embolic risk attributed to valvular and nonvalvular atrial fibrillation. Electric cardioversion should not be abandoned for fear of causing LAA stunning, which may in turn place the patient at increased risk for stroke after the procedure. Additionally, it is unproven that pharmacologic cardioversion has any more or less of an effect on LAA function than electric cardioversion. Therefore, all patients undergoing cardioversion of atrial arrhythmias (whether chemical or electric) should be considered at equivalent risk for the development of postcardioversion thromboembolism, and similar anticoagulation strategies should be used regardless of the modality of cardioversion. Clinical studies support this contention because a similar incidence of thromboembolic complications has been reported after chemical and electric cardioversion.[3, 8] In this report we summarized the findings of two patients with atrial fibrillation and flutter who demonstrated worsening LAA function after spontaneous conversion to sinus rhythm while being monitored with Doppler TEE. In the patient with spontaneous conversion from atrial fibrillation into sinus rhythm, we also observed an increase in the intensity of left atrial SEC after the conversion. These findings demonstrate the phenomenon of LAA stunning after spontaneous conversion to sinus rhythm without use of direct-current countershock, therefore exonerating electric energy as the sole cause of postcardioversion thrombogenesis.

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Acute myopericarditis resulting from Lyme disease

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Lyme disease is a multisystemic infection caused by the spirochete *Borrelia burgdorferi*; it most commonly affects the skin, central nervous, musculoskeletal, and cardiovascular systems. Heart involvement has been reported to occur in approximately 8% of patients with Lyme disease. The most common manifestation of cardiac disease is atrioventricular node block.[1, 2] Chronic dilated cardiomyopathy with congestive heart failure resulting from Lyme carditis has also been described.[2, 3] Symptomatic acute myopericarditis is a rarely reported manifestation of acute Lyme disease.[1, 4, 5] We report here a case of acute symptomatic myopericarditis resulting from Lyme disease to increase health care professionals' awareness of this problem.

A 23-year-old man came to the emergency department on July 24, 1993, with a 4-day history of fever. His medical history included typhoid fever (April 1992) that was acquired and treated in Guatemala and malaria (July 1992) that was acquired and treated with chloroquine in Ecuador. He lived and worked in northern Westchester County, N. Y., and was frequently in areas with woods and

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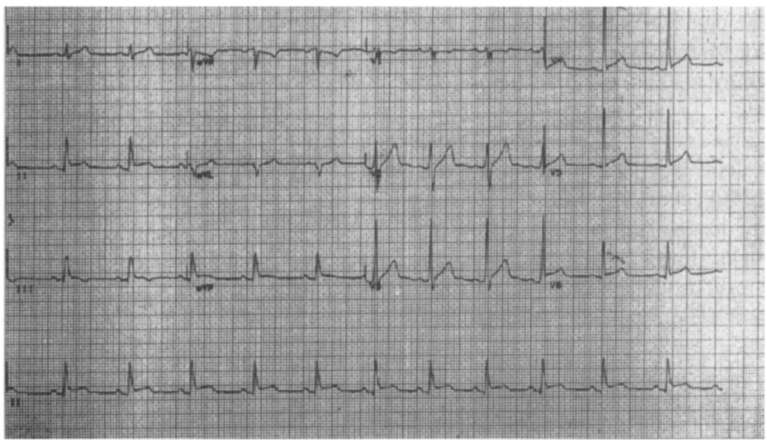

Fig. 1. Twelve-lead ECG during episode of pain.

high grass. Although he had noted no embedded ticks, 3 weeks before admission he noted a tick walking on his abdomen. The day before admission he had been treated at a nearby clinic, at which time he noted temperomandibular joint pain, posterior neck pain, and slight photophobia. A full body skin examination revealed no erythema migrans lesions. He was given 1 gm of azithromycin. Three hours later severe chills, increased fever, and headaches developed. During the day of admission he had fevers to 104.5° F and a pulsating headache. The patient returned to the local health clinic, where a malaria thick smear test result was negative, and he was referred to the emergency room. On admission he had a temperature of 99.8° F and otherwise normal vital signs. His physical examination was notable for mild neck flexion rigidity, normal cardiac and neurologic function, and lack of rashes. The results of his complete blood count and blood chemistry studies were all normal, as was a chest roentgenogram. A lumbar puncture revealed cerebral spinal fluid, with 1 cell/mm^3 and normal glucose and protein levels.

Approximately 6 hours after admission the patient had acute precordial chest pain that was not pleuritic or positional in nature. No cardiac or pleural rubs were heard. An electrocardiogram (ECG) revealed ST-segment elevations in the inferior and precordial leads (Fig. 1). Creatine phosphokinase was 511 U/L (6% MB fraction and lactate dehydrogenase levels of 265 U/L with an inverted fraction 1 and 2 were later reported). A diagnosis of acute myocardial infarction was initially considered. Urgent two-dimensional echocardiography did not show segmental wall motion abnormalities. A diagnosis of acute myocardial infarction was then thought unlikely. Mild diffuse left ventricular hypokinesis was seen, with no definite pericardial effusion. Acute myopericarditis was suspected on the basis of initial examination, reexamination of ECG, and echocardiographic findings. The next morning an evolving annular erythema migrans rash approximately 12 by 10 cm in size was found on the medial upper aspect of the patient's left calf. Ceftriaxone 1 gm intravenously every 12 hours was initiated. By the third day of admission the headache and temperomandibular joint pain had resolved, and the erythema migrans rash measured only 2.5 cm^2. On the fourth day of hospitalization, repeat echocardiogram was unchanged. ECG showed partial resolution of the ST-segment abnormalities. Creatine phosphokinase had returned to normal. The patient was discharged on the fifth day after admission and given oral amoxicillin. Cultures for *B. burgdorferi* of the CSF and blood before ceftriaxone grown on Barbour-Stoenner-Kelly for 6 weeks were negative. Cerebrospinal fluid viral cultures were negative, as were enteroviral stool cultures. Echovirus and Coxsackie A and B titers were normal. Lyme serologic tests with Western blot analysis were considered indeterminate on admission, with 41 kd IgG and 25 kd IgM immunoglobulin (Ig) bands identified. Four weeks later, a Western blot study was positive for antibodies to *B. burgdorferi*, with IgG bands of 41 and 58 kd and IgM bands of 41, 39, 25, 37, and 30 kd.

Acute symptomatic Lyme myopericarditis has rarely been reported.[4, 5] Although chest pain was reported in two of the initial 20 patients, of Steere et al.,[1] ECG findings suggestive of acute pericarditis occurred in 11 patients. However, clinical details concerning these patients were not reported. Pericarditis has been noted to occur far less frequently in the United States than in Europe.[2]

Treatment of acute myopericarditis is frequently frustrated by lack of effective antimicrobial agents for most infections that cause carditis. Lyme disease may represent one of the few treatable infectious agents that causes acute myopericarditis. In Lyme-endemic areas, Lyme disease must be suspected in patients with acute myopericarditis with or without erythema migrans rash so that appropriate diagnostic studies can be obtained. Acute and convalescent antibody enzyme-linked immunosorbent assay studies (with Western blot confirmation if positive) should be performed. Acute serologic tests may be negative or indeterminent as they were in this case. Seroconversion usually occurs by 6 weeks. Blood cultures for *B. burgdorferi* on Barbour-Stoenner-Kelly medium can be taken, but these tests are not routinely performed in most clinical microbiology laboratories. Moreover, during acute infec-

tion, blood cultures will be positive only 21% of the time.[6] Endomyocardial biopsy for histopathologic evaluation and culture of *B. burgdorferi* may also be obtained to make the diagnosis of Lyme carditis.

Sequelae of Lyme carditis include dilated cardiomyopathy. *B. burgdorferi* has been cultured from the myocardium of patients with dilated cardiomyopathy,[3, 7] and resolution of dilated cardiomyopathy has been reported after intravenous penicillin therapy for *B. burgdorferi*.[7] Whether early diagnosis and antibiotic therapy for early Lyme myopericarditis prevents the development of cardiomyopathy needs further study.

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Diastolic intraventricular pressure gradient in symmetric left ventricular hypertrophy

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Systolic Doppler flow velocity patterns characteristic of dynamic left ventricular outflow tract obstruction are common in patients with hypertrophic cardiomyopathy and have also been recognized in the setting of concentric left ventricular hypertrophy.[1, 2] Recently an unusual pattern of paradoxic diastolic flow from left ventricular apex to base has been detected by Doppler in patients with the apical variant of hypertrophic cardiomyopathy.[3, 4] This diastolic intraventricular flow reversal has been attributed to a dysfunctional apical cavity and identified as a marker for increased risk of adverse clinical events.[4] Although previous studies suggest that this Doppler profile is associated solely with apical hypertrophic cardiomyopathy, we recently observed a similar diastolic Doppler flow pattern in a patient with a subaortic membrane and a hyperdynamic, concentrically hypertrophied ventricle that resulted in left ventricular midcavity obstruction. To our knowledge, this has not previously been reported. The prognostic significance of diastolic apex-to-base flow in patients without apical hypertrophic cardiomyopathy is unclear.

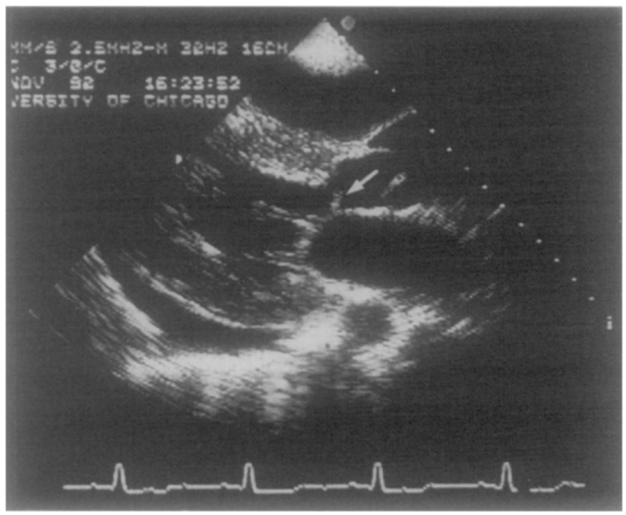

Fig. 1. Parasternal long-axis view demonstrates subaortic membrane *(arrow)* in left ventricular outflow tract. Concentric left ventricular hypertrophy and moderate pericardial effusion are also noted.

A 51-year-old woman with exertional dyspnea was referred for echocardiographic examination. She had previously been diagnosed with hypertrophic cardiomyopathy and was being treated with β-adrenergic antagonists and calcium channel blockers. On physical examination there was a IV/VI systolic ejection murmur that did not vary with the Valsalva maneuver or after spontaneously occurring premature beats. A II/VI early diastolic murmur was also audible. The electrocardiogram showed left-axis deviation with left bundle branch block. Two-dimensional echocardiography revealed marked concentric left ventricular hypertrophy with hyperdynamic systolic performance. A discrete membranous structure spanning the left ventricular outflow tract was visualized almost 2 cm proximal to a trileaflet aortic valve. No systolic anterior motion of the mitral valve apparatus was noted. A moderate pericardial effusion was present (Fig. 1). Continuous wave Doppler evaluation demonstrated marked flow acceleration at the level of the subaortic membrane with a peak systolic flow velocity of 6 m/sec. Moderate aortic regurgitation and mild

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