aging and atrial hypertrophy in response to hypertension and other cardiac disease may be an important promoting factor, although electrophysiologic changes to conduction and refractoriness occur as well in response to chronic tachycardia in the atrium.

Clinical consequences are related to rapid ventricular rates, loss of atrial contribution to ventricular filling, and predisposition to thrombus formation in the left atrial appendage with potential embolization. Presentations vary with the ventricular rate and underlying heart disease and comorbidities. Many patients are asymptomatic. Rapid rates may cause hemodynamic collapse or heart failure exacerbations particularly in patients with impaired cardiac function, hypertrophic cardiomyopathy, and heart failure with preserved systolic function. Exercise intolerance and easy fatigability are common. Occasionally, dizziness or syncope occurs due to pauses when AF terminates to sinus rhythm **(Fig. 276-13)**.

TREATMENT ATRIAL FIBRILLATION

Treatment for AF is primarily guided by patients' symptoms, the hemodynamic effect of AF, the duration of AF if there are persistent risk factors for stroke, and underlying heart disease. Oral anticoagulation in high-risk patients with AF includes vitamin K antagonists or the newer anticoagulants such as thrombin inhibitors (dabigatran) or factor Xa inhibitors (rivaroxaban, apixaban), but not antiplatelet agents (aspirin and clopidogrel), which have substantially less effect.

New-onset AF that produces severe hypotension, pulmonary edema, or angina should be electrically cardioverted starting with a QRS synchronous shock of 200 J, ideally after sedation or anesthesia is achieved. Greater shock energy and different electrode placements may be tried if the shock fails to terminate AF. If AF terminates and reinitiates, administration of an antiarrhythmic drug, such as ibutilide, and repeat cardioversion may be considered. If the patient is stable, immediate management involves rate control to alleviate or prevent symptoms, anticoagulation if appropriate, and cardioversion to restore sinus rhythm if AF is persistent. Anticoagulation strategies for new-onset AF are debated. In the absence of contraindications, it is usually appropriate to initiate systemic anticoagulation with heparin immediately, while evaluation and other therapies are implemented.

CARDIOVERSION AND ANTICOAGULATION

Cardioversion *within 48 h of the onset of AF* is common practice in patients who have not been anticoagulated, provided that they are not at high risk for stroke due to a prior history of embolic events, rheumatic mitral stenosis, or hypertrophic cardiomyopathy with marked left atrial enlargement. These patients are usually at risk of recurrence, such that initiation of anticoagulation is considered based on the patient's individual risk for stroke, commonly assessed from the CHA_2DS_2-VASc score.

If the duration of AF exceeds 48 h or is unknown, there is greater concern for thromboembolism with cardioversion, even in patients considered low risk for stroke. There are two approaches to mitigate the risk related to cardioversion. One option is to anticoagulate continuously for 3 weeks before and a minimum of 4 weeks after cardioversion. A second approach is to start anticoagulation and perform a transesophageal echocardiogram to determine if thrombus is present in the left atrial appendage. If thrombus is absent, cardioversion can be performed and anticoagulation continued for a minimum of 4 weeks because recovery of atrial mechanical function after electrical or pharmacologic cardioversion may be delayed and thrombus can form and embolize days after cardioversion. Some patients may merit ongoing anticoagulation after cardioversion, depending on stroke risk profile.

RATE CONTROL

Acute rate control can be achieved with beta blockers and/or the calcium channel blockers verapamil and diltiazem administered either intravenously or orally, as warranted by the urgency of the clinical situation. Digoxin may be added, particularly in heart failure

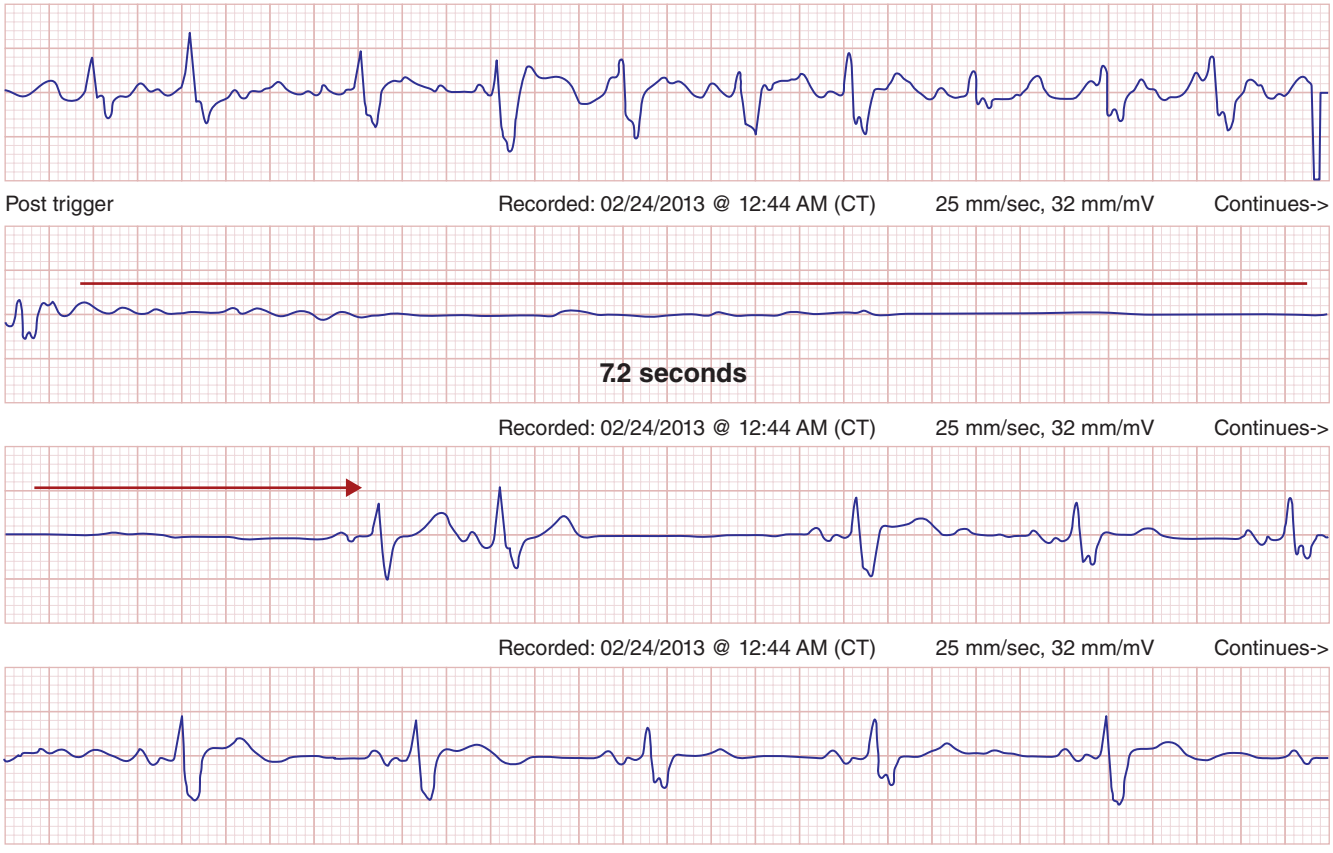

FIGURE 276-13 A continuous rhythm strip is shown. Atrial fibrillation is present at the top and abruptly terminates in the second tracing, with atrial and ventricular standstill for 7.2 s until resumption of sinus rhythm. The patient experienced syncope.