CLINICAL PRACTICE

Newly Diagnosed Atrial Fibrillation

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This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.*

A 77-year-old woman with a history of hypertension treated with metoprolol presents for her annual examination. She reports no new symptoms. The examination is remarkable only for the finding of an irregular heart rate. Electrocardiographic testing reveals atrial fibrillation at an average rate of 75 beats per minute. She has no history of arrhythmia, coronary disease, valvular disease, diabetes, alcohol abuse, transient ischemic attack, or stroke. For the past several months, she has exercised on a treadmill without difficulty, although she notes that the machine does not always measure her heart rate. What should her physician advise?

THE CLINICAL PROBLEM

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Atrial fibrillation is the most common arrhythmia that requires treatment, with an estimated prevalence in the United States of 2.3 million patients in 2001.[1] The prevalence increases with age — atrial fibrillation occurs in 3.8 percent of people 60 years of age and older and in 9.0 percent of those 80 years of age and older.[1]

RISK OF STROKE AND DEATH

The most devastating consequence of atrial fibrillation is stroke as a result of thromboembolism typically emanating from the left atrial appendage.[2] The rate of stroke varies but may range from 5.0 percent to 9.6 percent per year among patients at high risk who are taking aspirin (but not warfarin).[3,4]

Patients with paroxysmal (i.e., self-terminating) and persistent atrial fibrillation (i.e., that lasts more than seven days or requires cardioversion) appear to have a risk of stroke that is similar to that of patients with permanent atrial fibrillation.[5] In the Stroke Prevention in Atrial Fibrillation studies of patients with atrial fibrillation, the risk of stroke among those with sinus rhythm that had been documented within the 12 months before enrollment (3.2 percent per year) was similar to that among those with permanent atrial fibrillation (3.3 percent per year).[5] The duration of episodes of atrial fibrillation and the overall time spent in atrial fibrillation (i.e., burden) have not been established as determining the risk of stroke. Atrial fibrillation is associated with an increase in the relative risk of death ranging from 1.3 to twice that value, independent of other risk factors.[6,7] This risk may be greater for women than for men.[7]

ASSOCIATED DISEASES AND PREDISPOSING CONDITIONS

In most cases, atrial fibrillation is associated with cardiovascular disease, in particular hypertension, coronary artery disease, cardiomyopathy, and valvular disease (primarily mitral); it also occurs after cardiac surgery and in the presence of myocarditis or pericarditis. When atrial fibrillation complicates severe mitral regurgitation, valve repair or replacement is indicated.[8] In some cases, atrial fibrillation results from another supraventricular tachycardia. When it is associated with the Wolff–Parkinson–White syn-

drome, rapid conduction down the accessory pathway may result in hemodynamic collapse.[9]

Other predisposing conditions include excessive alcohol intake, hyperthyroidism, and pulmonary disorders, including pulmonary embolism. Obstructive sleep apnea may also be related, in which case the provision of continuous positive airway pressure reduces the risk of the recurrence of atrial fibrillation.[10] Both vagal and sympathetic mechanisms of paroxysmal atrial fibrillation have been described (neurogenic atrial fibrillation),[11] as have familial forms of the condition.[12] "Lone" atrial fibrillation (i.e., that occurring in the absence of a cardiac or other explanation) is common, particularly in patients with paroxysmal atrial fibrillation — up to 45 percent of such patients have no underlying cardiac disease.[13]

EVALUATION

The patient's history and the physical examination should focus on these potential causes of atrial fibrillation. The "minimum evaluation" recommended at diagnosis should include 12-lead electrocardiography, chest radiography, transthoracic echocardiography, and serologic tests of thyroid function.[11] Echocardiographic testing is used to assess valve function, chamber size, and the peak right ventricular pressure and to detect hypertrophy and pericardial disease. Additional tests may be warranted, including exercise testing to determine whether the patient has symptoms and to assess the heart rate with exercise, 24-hour ambulatory monitoring to evaluate heart-rate control, transesophageal echocardiography to screen for a left atrial thrombus and to guide cardioversion, and, rarely, an electrophysiological study to detect predisposing arrhythmias.[11]

SYMPTOMS AND HEMODYNAMIC CONSEQUENCES

Patients with atrial fibrillation may have palpitations, dyspnea, fatigue, light-headedness, and syncope. These symptoms are usually related to the elevated heart rate and, in most patients, can be mitigated with the use of drugs to control the heart rate. Exceptions are due, presumably, to an irregular ventricular response or a reduction of cardiac output.

The hemodynamic consequences of atrial fibrillation are related to the loss of atrial mechanical function, irregularity of ventricular response, and high heart rate. These consequences are magnified in the presence of impaired diastolic ventricular filling, hypertension, mitral stenosis, left ventricular hypertrophy, and restrictive cardiomyopathy.[11] Irregularity of the cardiac cycle, especially when accompanied by short coupling intervals, and rapid heart rates in atrial fibrillation lead to a reduction in diastolic filling, stroke volume, and cardiac output. In a study of patients who were evaluated while in atrial fibrillation and again during ventricular pacing at the same overall heart rate, the irregular rhythm was associated with a lower cardiac output (4.4 vs. 5.2 liters per minute) and higher pulmonary-capillary wedge pressure (17 vs. 14 mm Hg).[14]

A chronically elevated heart rate of 130 beats per minute or more may result in secondary cardiomyopathy,[15] a type of left ventricular dysfunction that may largely be reversed when control of the ventricular rate is achieved.[15,16] A report in this issue of the *Journal*[17] indicates that, in patients with atrial fibrillation, heart-rate control and rhythm control with the use of radiofrequency catheter ablation improve left ventricular function in both those with and those without congestive heart failure.

ASYMPTOMATIC ATRIAL FIBRILLATION

Asymptomatic, or "silent," atrial fibrillation occurs frequently.[18] Among patients in the Canadian Registry of Atrial Fibrillation, 21 percent in whom the condition was newly diagnosed were asymptomatic.[19] The first presentation of asymptomatic atrial fibrillation may be catastrophic; in the Framingham Study, among patients with stroke that was associated with atrial fibrillation, the arrhythmia was newly diagnosed in 24 percent.[20] Even among patients with documented symptomatic atrial fibrillation, asymptomatic recurrences are common. In one study of patients with symptomatic paroxysmal atrial fibrillation, asymptomatic episodes were 12 times more common than symptomatic episodes.[21] In a recent trial,[22] among untreated patients, 17 percent had asymptomatic episodes before they noted symptoms, and the percentage was probably an underestimation, because the monitoring of these patients was intermittent. Some antiarrhythmic agents, by reducing conduction in the atrioventricular node, may increase the likelihood of the occurrence of asymptomatic atrial fibrillation. Both propafenone and propranolol have been associated with frequent asymptomatic atrial fibrillation,[23] and the risk may be similar with other agents that block atrioventricular nodal conduction.[24] Among patients with a pacemaker and a history of atrial fibrillation, one in six had silent recurrences lasting 48 hours or longer.[25]

STRATEGIES AND EVIDENCE

ANTICOAGULANT THERAPY

The need for anticoagulation to reduce the risk of stroke among patients with atrial fibrillation due to mitral stenosis is well recognized.[11] Several randomized, prospective trials involving patients with nonvalvular atrial fibrillation[26-32] have confirmed a significant reduction in the risk of stroke with warfarin. These studies defined the patients at greatest risk as the elderly, variably defined as those older than 60, 65, and 75 years of age, and those with a history of thromboembolism, diabetes mellitus, coronary artery disease, hypertension, heart failure, and thyrotoxicosis.[11,33] These trials have provided a basis for two important guidelines for the use of warfarin in such patients[11,33,34] (Table 1). Recently, an index based on the assignment of points for five risk factors (i.e., congestive heart failure, hypertension, age, diabetes, and transient ischemic attack or stroke) was reported to be accurate in predicting stroke when it was used to evaluate the risk among patients in the Medicare database[4]; it is the basis for yet another guideline for antithrombotic therapy in atrial fibrillation[35] (Table 1). In addition, complex aortic plaques detected by transesophageal echocardiography that are associated with an increased risk of stroke in patients with atrial fibrillation also warrant the institution of anticoagulant therapy.[36]

An international normalized ratio (INR) value in the range of 2.0 to 3.0 is recommended. The risk of stroke doubles when the INR falls to 1.7, although values up to 3.5 do not convey an increased risk of bleeding complications.[37] INR values of 2.0 or greater are associated with a reduced severity of stroke and, if stroke occurs, a lower likelihood that it will result in death.[38]

Certain patients are at relatively low risk for a thromboembolic event and do not require intensive anticoagulant therapy[11,33,35] (Table 1). Aspirin is often recommended for these patients, although their risk is so low that even aspirin may not be necessary. Alternative antiplatelet agents, such as clopidogrel, have not been tested adequately in this clinical situation.

The duration of atrial fibrillation becomes important when cardioversion (with the use of electric or pharmacologic means) is being considered. It is generally accepted that patients who have had an episode of atrial fibrillation lasting less than 48 hours may safely undergo cardioversion without anticoagulant therapy, although the data supporting this practice are scant.[11] For episodes lasting longer than 48 hours, adequate anticoagulant therapy is warranted, both before cardioversion and for four weeks afterward. A recent report concluded that a strategy of initiating anticoagulant therapy and ruling out left atrial thrombus with the use of transesophageal echocardiography was a possible alternative to the usual strategy of anticoagulant therapy for three weeks before cardioversion.[39]

RATE CONTROL

Current guidelines recommend a ventricular rate during atrial fibrillation of 60 to 80 beats per minute at rest and 90 to 115 beats per minute during exercise.[11] A number of pharmacologic agents are available to control the heart rate and rhythm (Tables 2 and 3). Digoxin has been replaced as first-line therapy for rate control by β-adrenergic blockers and calcium-channel blockers, largely owing to improved rate control during exertion with the use of these alternative agents.[11] In one study, during peak exercise, the mean heart rate was 175 beats per minute in patients receiving digoxin, as compared with 130 in those receiving a β-adrenergic blocker and 151 in those receiving a calcium-channel blocker.[40] Digoxin is useful in combination with other agents[40] or when β-adrenergic–blocking agents and calcium-channel blockers are not tolerated. In some patients, particularly the elderly, the ventricular rate during atrial fibrillation may be intrinsically controlled, so that no atrioventricular nodal–blocking agent is required. Among patients with a pause that causes symptoms after the spontaneous conversion of atrial fibrillation, or those whose symptoms are due to low heart rates in spite of their having high heart rates at other times, a pacemaker may be necessary to permit therapy with atrioventricular nodal–blocking agents (as in the "tachy-brady" or the sick sinus syndrome).

RHYTHM CONTROL

A number of agents may maintain sinus rhythm (Tables 2 and 3). The use of β-adrenergic agents may be effective in adrenergically mediated and paroxysmal atrial fibrillation[41] (although the effects may be related to the conversion of symptomatic atrial fibrillation into asymptomatic atrial fibrillation).[24] With the exception of the β-adrenergic–blocking agents, most antiarrhythmic drugs carry a risk of serious adverse effects. Antiarrhythmic therapy should be chosen on the basis of the patient's underlying

Table 1. Guidelines for Antithrombotic Therapy in Atrial Fibrillation.

Characteristic	Therapy Recommended by the ACC–AHA and ESC	Differences in ACCP Guidelines
Age		
<60 yr, no heart disease	Aspirin at a dose of 325 mg per day, or no therapy	Aspirin at a dose of 325 mg for patients <65 yr of age with no risk factor†
<60 yr, with heart disease but no risk factors*	Aspirin at a dose of 325 mg per day	No divergence
≥60–75 yr, no risk factors*	Aspirin at a dose of 325 mg per day	Option of aspirin at a dose of 325 mg per day or warfarin (INR, 2.0–3.0) for patients 65–75 yr of age
≥60 yr, with diabetes mellitus or coronary artery disease	Warfarin (INR, 2.0–3.0), aspirin optional in addition (at a dose of 81–162 mg per day)	Option of aspirin at a dose of 325 mg per day or warfarin (INR, 2.0–3.0) for patients with diabetes alone or coronary artery disease alone who are <65 yr of age
>75 yr, especially among women	Warfarin (INR, approximately 2.0; target INR, 1.6–2.5)	Warfarin (INR, 2.0–3.0), but no recommendation for INR value <2.0
Heart failure, left ventricular ejection fraction ≤0.35, thyrotoxicosis, and hypertension	Warfarin (INR, 2.0–3.0)	No divergence
Rheumatic heart disease (mitral stenosis)	Warfarin (INR, 2.5–3.5 or higher) may be appropriate	Other than for patients with mechanical valves, no INR recommended above target, 2.5 (range, 2.0–3.0)
Previous thromboembolism	Warfarin (INR, 2.5–3.5 or higher) may be appropriate	Other than for patients with mechanical valves, no INR recommended above target, 2.5 (range, 2.0–3.0)
Persistent atrial thrombus on transesophageal echocardiography	Warfarin (INR, 2.5–3.5 or higher) may be appropriate	Other than for patients with mechanical valves, no INR recommended above target, 2.5 (range, 2.0–3.0)
Prosthetic heart valves	Warfarin (INR, 2.5–3.5 or higher) may be appropriate	Depending on the type of prosthetic valve, warfarin (INR, 2.5 [range, 2.0–3.0] or INR, 3.0 [range, 2.5 to 3.5]) with or without additional aspirin, at a dose of 80 to 100 mg[34]
Warfarin recommended but contraindicated or refused	Aspirin at a dose of 325 mg per day	No divergence

* According to the guidelines of the American College of Cardiology and American Heart Association (ACC–AHA) Task Force on Practice and the European Society of Cardiology (ESC) Committee for Practice, the risk factors for thromboembolism include heart failure, a left ventricular ejection fraction of less than 35 percent, and a history of hypertension.[11] INR denotes international normalized ratio.
† According to the American College of Chest Physicians (ACCP), moderate risk factors include an age of 65 to 75 years, diabetes mellitus, and coronary artery disease with preserved left ventricular function; high risk factors include previous stroke, transient ischemic attack, or systemic embolus; a history of hypertension; poor left ventricular systolic function; an age of 75 years or older; rheumatic mitral-valve disease; and the presence of a prosthetic heart valve.[33]

cardiac condition (Table 3).[11] Antiarrhythmic agents classified according to the Vaughn Williams system as class IC are reserved to treat patients without a structural cardiac abnormality, and as described elsewhere in this issue of the *Journal*,[42] may be prescribed for outpatients with acute conversion of paroxysmal atrial fibrillation (i.e., the so-called pill-in-the-pocket approach). Agents in classes IA and III should be avoided by patients with prolongation of the QT interval or left ventricular hypertrophy be-

cause of the potential for torsades de pointes. On the one hand, amiodarone, which has a low risk of proarrhythmia (less than 1 percent per year),[43] causes substantial noncardiac toxic effects and is therefore generally reserved for second-line therapy except in the treatment of patients with severe cardiomyopathy. On the other hand, it is the most effective antifibrillatory agent; in one trial, 65 percent of patients treated with amiodarone were free from recurrence after 16 months of therapy (as compared with 37

Table 2. Pharmacologic Agents to Control Heart Rate and Rhythm.*

Drug (Class)†	Purpose	Usual Maintenance Dose	Adverse Effects	Cautions and Contraindications
Metoprolol (II)	Rate control (rhythm in some cases)	50–200 mg daily, divided doses or sustained-release formulation	Hypotension, heart block, bradycardia, asthma, congestive heart failure	—
Propranolol (II)	Rate control (rhythm in some cases)	80–240 mg daily, divided doses or sustained-release formulation	Hypotension, heart block, bradycardia, asthma, congestive heart failure	—
Diltiazem (IV)	Rate control	120–360 mg daily, divided doses or sustained-release formulation	Hypotension, heart block, congestive heart failure	—
Verapamil (IV)	Rate control	120–360 mg daily, divided doses or sustained-release formulation	Hypotension, heart block, congestive heart failure, interaction with digoxin	—
Digoxin	Rate control	0.125–0.375 mg daily	Toxic effects of digitalis, heart block, bradycardia	—
Amiodarone (III)	Rhythm control (rate in some cases)	100–400 mg daily	Pulmonary toxic effects, skin discoloration, hypothyroidism, gastrointestinal upset, hepatic toxic effects, corneal deposits, optic neuropathy, interaction with warfarin, torsades de pointes (rare)	—
Quinidine (IA)	Rhythm control	600–1500 mg daily, divided doses	Torsades de pointes, gastrointestinal upset, enhanced atrioventricular nodal conduction	Prolongs QT interval; avoid with left ventricular wall thickness ≥1.4 cm
Procainamide (IA)	Rhythm control	1000–4000 mg daily, divided doses	Torsades de pointes, lupus-like syndrome, gastrointestinal symptoms	Prolongs QT interval; avoid with left ventricular wall thickness ≥1.4 cm
Disopyramide (IA)	Rhythm control	400–750 mg daily, divided doses	Torsades de pointes, congestive heart failure, glaucoma, urinary retention, dry mouth	Prolongs QT interval; avoid with left ventricular wall thickness ≥1.4 cm
Flecainide (IC)	Rhythm control	200–300 mg daily, divided doses	Ventricular tachycardia, congestive heart failure, enhanced atrioventricular nodal conduction (conversion to atrial flutter)	Contraindicated in patients with ischemic and structural heart disease
Propafenone (IC)	Rhythm control	450–900 mg daily, divided doses	Ventricular tachycardia, congestive heart failure, enhanced atrioventricular nodal conduction (conversion to atrial flutter)	Contraindicated in patients with ischemic and structural heart disease
Sotalol (III)	Rhythm control	240–320 mg daily, divided doses	Torsades de pointes, congestive heart failure, bradycardia, exacerbation of chronic obstructive or bronchospastic lung disease	Prolongs QT interval; avoid with left ventricular wall thickness ≥1.4 cm
Dofetilide (III)	Rhythm control	500–1000 μg daily, divided doses	Torsades de pointes	Prolongs QT interval; avoid with left ventricular wall thickness ≥1.4 cm

* The information in the table is adapted from Fuster et al.[11]
† The Vaughn Williams class of antiarrhythmic drugs is given for those classified. Digoxin is not classified in this system.

Table 3. Choice of Antiarrhythmic Agent According to the Underlying Cardiac Disorder.*

Underlying Disorder	Rate Control†	Rhythm Control		
		First Choice	Second Choice	Third Choice
Minimal or no heart disease	β-adrenergic blocker or calcium-channel blocker	Flecainide, propafenone, sotalol	Amiodarone, dofetilide	Disopyramide, procainamide, quinidine (or nonpharmacologic options)
Adrenergic atrial fibrillation with minimal or no heart disease	β-adrenergic blocker	β-adrenergic blocker or sotalol	Amiodarone, dofetilide	—
Heart failure	β-adrenergic blocker, if tolerated; digoxin	Amiodarone, dofetilide	—	—
Coronary artery disease	β-adrenergic blocker	Sotalol	Amiodarone, dofetilide	Disopyramide, procainamide, quinidine
Hypertension with LVH but wall thickness <1.4 cm	β-adrenergic blocker or calcium-channel blocker	Flecainide, propafenone	Amiodarone, dofetilide, sotalol	Disopyramide, procainamide, quinidine
Hypertension with LVH and wall thickness ≥1.4 cm	β-adrenergic blocker or calcium-channel blocker	Amiodarone	—	—

* LVH denotes left ventricular hypertrophy. The information in this table is adapted from Fuster et al.[11]
† β-adrenergic blockers include metoprolol and propranolol; calcium-channel blockers includes diltiazem and verapamil.

percent of those who were treated with propafenone or sotalol).[44]

RATE CONTROL VERSUS RHYTHM CONTROL

In three recent randomized studies, rate control was compared with rhythm control in patients with persistent atrial fibrillation.[45-47] The Pharmacological Intervention in Atrial Fibrillation trial found no difference between the treatment groups in the primary end point of the quality of life, although a secondary analysis showed improvement in the distance walked in six minutes among patients in the rhythm-control group.[45] The Rate Control versus Electrical Cardioversion for Persistent Atrial Fibrillation (RACE) trial found that rate control was not inferior to rhythm control in the effects on a composite end point (consisting of death from cardiovascular causes, heart failure, thromboembolic complications, bleeding, implantation of a pacemaker, and serious adverse effects of drugs) over a period of 2.3 years (rate control, 17.2 percent, vs. rhythm control, 22.6 percent).[46] The largest of these trials, the Atrial Fibrillation Follow-up Investigation of Rhythm Management trial, which was designed to assess mortality, found no significant difference in this end point between the groups at five years

(rhythm control, 23.8 percent, vs. rate control, 21.3 percent).[47] Thus, the evidence suggests that the strategy used to treat atrial fibrillation — rate control versus rhythm control — does not have a substantial effect on the quality of life or on cardiovascular end points, including death.

Nonetheless, some questions remain. All three of these trials compared strategies with the use of an intention-to-treat analysis. The success rate for maintaining sinus rhythm was as low as 39 percent after 2.3 years of treatment[46] and as high as 73 percent at 3 years.[47] A secondary analysis of the data from the RACE trial showed that for patients with symptoms related to atrial fibrillation and those who were in sinus mechanism at the end of the follow-up period, regardless of the treatment randomly assigned, the quality of life had improved.[48] All three studies enrolled only patients for whom rhythm control was considered to be an option by both the patient and the physician; in highly symptomatic patients, rhythm control may still be preferable. For patients who have minimal symptoms or when sinus rhythm cannot be maintained, however, a strategy of rate control is safe and appropriate. Anticoagulant therapy should be continued, irrespective of the strategy used.

ABLATION

In the past decade, ablation for atrial fibrillation has become a therapeutic option. The initial efforts involved the creation of radiofrequency lines of conduction block, rather than surgical incisions.[49] The subsequent discovery that paroxysmal atrial fibrillation primarily emanates from the pulmonary veins[50] led to the use of focal-vein ablation and then to techniques to isolate the firing foci with the use of circumferential or segmental ablation near the ostia of the pulmonary veins.[51]

Recently, the use of anatomical ablation with lesions placed circumferentially around the right and left veins, with or without additional left atrial linear lesions, has been successful in patients with paroxysmal atrial fibrillation and those with persistent atrial fibrillation. In an observational study of 1171 patients, those who underwent ablation had significantly lower rates of recurrence after one year (16 percent) than those receiving antiarrhythmic drugs (39 percent)[52]; among the patients who underwent ablation, mortality and morbidity also were lower and the quality of life was better.

However, data are needed from a randomized trial to establish whether these differences are attributable to the therapy or to other factors. Early series primarily enrolled patients with normal left ventricular function, but in a recent study of 377 patients, one quarter had an ejection fraction below 40 percent,[53] and 73 percent of this group had no recurrence during a follow-up period of 14 months (as compared with 87 percent of the patients with a left ventricular ejection fraction of 40 percent or greater).[53]

Although new techniques and increased experience are associated with lower complication rates, concern persists about potential stroke and tamponade (events that are estimated to occur in 1 percent of cases among experienced physicians).[54] Furthermore, pulmonary-vein stenosis may occur in 5 to 6 percent of patients,[55,56] even when techniques to minimize the risk are used.[57] When a pulmonary-vein stenosis occurs, conservative management may be appropriate, but dilation with or without stenting may be necessary.[55,56]

AREAS OF UNCERTAINTY

Approaches to prevent the development of atrial fibrillation warrant further attention. Recent randomized trials involving patients with left ventricular dysfunction suggest that angiotensin-converting–enzyme inhibitors reduce the risk of atrial fibrillation.[58,59] These data emphasize the importance of treatment for hypertension and cardiovascular disease in such patients.

The role of ablation, as compared with antiarrhythmic therapy, remains uncertain; its use may increase as tools and techniques are improved. The role of new oral anticoagulant agents that are currently in development, which might obviate the need for dose adjustment and the measurement of INR values, needs to be determined. The direct thrombin inhibitor ximelagatran appears to be as effective as warfarin in the prevention of stroke and systemic embolism in patients with atrial fibrillation.[60] However, clinical use of ximelagatran may be limited by its hepatic toxicity; the elevation of levels of alanine aminotransferase to more than three times the upper limit of normal occurred in 6 percent of the patients taking ximelagatran, as compared with 1 percent of those taking warfarin, and hepatic failure leading to death has been reported with the use of ximelagatran.[61]

GUIDELINES

The American College of Cardiology and American Heart Association (ACC–AHA) Task Force on Practice and the European Society of Cardiology (ESC) Committee for Practice have published guidelines for the management of atrial fibrillation[11] that recommend the "minimum evaluation" of newly discovered atrial fibrillation, mentioned earlier, and advise on the use of antiarrhythmic agents (Tables 2 and 3).[11] These guidelines suggest that there is "no clear advantage"[11] to a strategy of rate control as compared with rhythm control. Their recommendations for antithrombotic therapy are similar to, but not identical with, those published by the American College of Chest Physicians (ACCP)[33,34] (Table 1). A third set of guidelines, proposed by the American Academy of Family Physicians (AAFP) and the American College of Physicians (ACP),[35] recommend less aggresssive anticoagulant therapy with warfarin. This set of guidelines defines patients who have no history of stroke or transient ischemic attack and have only a single risk factor for stroke (e.g., an age of 75 years or older, congestive heart failure, hypertension, or diabetes)[35] as at low risk (i.e., not in need of warfarin therapy).

RECOMMENDATIONS

The patient described in the vignette presented with atrial fibrillation that was asymptomatic and

may have been present for months (as suggested by the failure of the treadmill monitor to measure her heart rate). The evaluation should include testing with electrocardiography, echocardiography, and chest radiography and measurement of the serum thyrotropin level. On the basis of data from randomized trials, her survival would not be improved by the use of strategies aimed at conversion and the maintenance of sinus rhythm, and no strategy could improve her symptoms since she has none. Thus, I would continue heart-rate–control therapy with the use of her current β-adrenergic–blocking agent. Her age and hypertension place her at elevated risk for thromboembolism, and anticoagulant therapy with warfarin is indicated, with a target INR of 2.0 to 3.0. Because atrial fibrillation represents a marker of risk for atherosclerotic disease and stroke,[62] I would also assess the patient for and aggressively treat other risk factors for cardiovascular disease, including her hypertension.

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