



## Improving the Management of Atrial Fibrillation

### Learning Objectives

After completing this activity, participants should be better able to:

- Recognize presenting symptoms, patient complaints, and comorbidities commonly associated with atrial fibrillation (AF)
- Utilize evidence-based guidelines to identify appropriate patients for antiarrhythmic and/or antithrombotic therapy
- Develop initial and long-term management strategies for patients maintained on antiarrhythmic and antithrombotic therapy
- Educate patients and caregivers about long-term management of AF

### Introduction

AF is a supraventricular tachyarrhythmia typified by uncoordinated atrial activation that eventually causes a decline in atrial mechanical functioning.<sup>1</sup> It is the most widespread clinically significant cardiac arrhythmia, affecting approximately 2.3 million adults in the United States, and its prevalence is increasing.<sup>1,2</sup> The condition is more common in elderly persons: as many as 9% of those older than 80 years of age have AF (Figure 1).<sup>2</sup> The aging of the population, with a growing prevalence of chronic heart disease and more frequent AF diagnosis, has led to a 66% rise in hospitalizations due to AF over the past 20 years.<sup>1</sup>

Patients with AF are at 5-fold increased risk for stroke; at least 15% to 20% of all ischemic strokes are attributable to AF.<sup>3</sup> Such factors as rheumatic mitral valve disease or a prosthetic heart valve further increase the stroke risk in patients with AF.<sup>4</sup> AF also is an independent risk factor for mortality. In a cohort of patients from the Framingham Heart Study who developed AF over a 40-year follow-up, a 50% to 90% increase in the risk of death was seen after adjustment for coexisting cardiovascular (CV) conditions.<sup>5</sup>

In 2011, the American College of Cardiology (ACC) and the American Heart Association (AHA) updated their practice guidelines on the management of AF.<sup>1,6,7</sup> The update was prompted by new data regarding the relative benefits of strict versus lenient heart-rate control, the use of a new antiarrhythmic agent (dronedrone), and 2 new options for stroke prevention in the setting

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of AF (dual antiplatelet therapy and dabigatran). This chapter will review current strategies for evaluating and managing patients with AF based on these updated practice guidelines. Approaches to preventing thromboembolism, controlling rate, and correcting rhythm disturbances in the context of clinical care will also be discussed in the case study of a patient presenting with irregular tachypalpitation (see page 10).

## Clinical Considerations

### Risk Factors for AF

The most common risk factor for developing AF is hypertension, especially when it occurs in patients with left ventricular hypertrophy (LVH).<sup>1,4</sup> Other risk factors include hyperthyroidism (even subclinical hyperthyroidism), sleep apnea, and obesity.<sup>1,4,8,9</sup>

AF is also often comorbid with heart failure, coronary artery disease (CAD), valvular heart disease, and other cardiac diseases.<sup>1</sup> Additional factors that can predispose to AF include acute alcohol intoxication (ie, the “holiday heart syndrome”), cardiac surgery, pulmonary diseases, or metabolic disorders.<sup>1,4</sup>

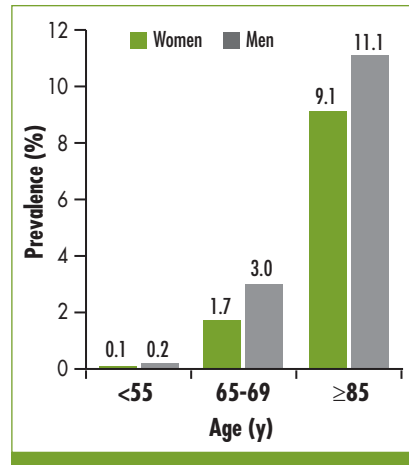
Although AF is frequently a manifestation of structural cardiac disease, as many as 30% of cases are observed in younger individuals without evidence of cardiopulmonary disease; such cases are referred to as *lone AF*.<sup>1,4,10</sup> Patients with lone AF have a lower risk of stroke than other AF patients.<sup>4</sup> Eventually, however, underlying heart malfunction may either emerge or develop with age, with a concomitant increase in the risk of stroke or death.<sup>1</sup>

### Clinical Manifestations of AF

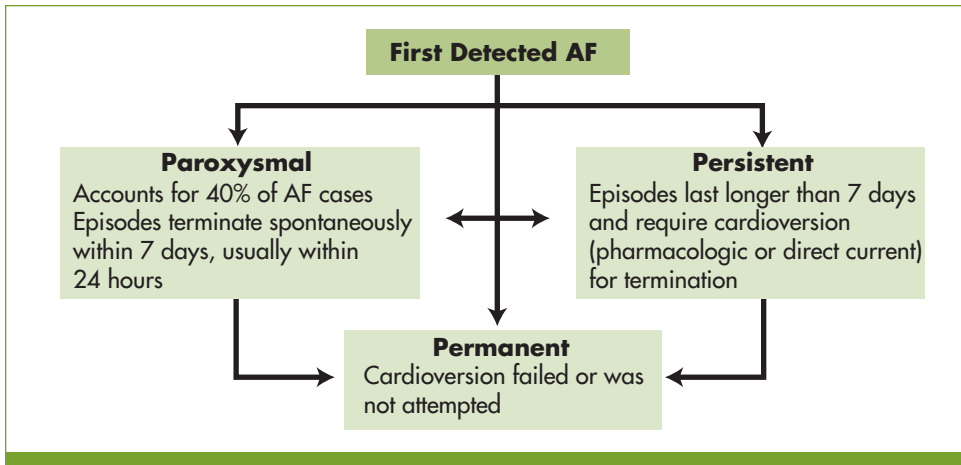
Symptoms of AF can vary from significant to minor. The condition usually presents as a rapid onset of irregular palpitations, coupled with fatigue.<sup>4</sup> Patients may also complain of light-headedness, chest pain, dyspnea, or syncope. However, many patients are asymptomatic, making AF difficult to recognize.<sup>1</sup> On the electrocardiogram (ECG), rapid oscillations or fibrillatory waves take the place of regular P waves and the ventricular response is erratic.<sup>1</sup>

### Classification of AF

Although several schemes for categorizing AF have been proposed, the ACC/AHA guidelines recommend a simplified classification system that takes into account both the pattern of AF presentation and the character of the arrhythmia at a particular instant (Figure 2).<sup>1</sup> After the first detected episode of AF, despite any uncertainty about the duration of the episode or any previously undetected episodes, the next episode is considered recurrent. Recurrent AF is classified either as paroxysmal if it stops spontaneously within 7 days or as persistent if it continues beyond 7 days. A patient may have either paroxysmal or persistent AF at any given time; the ACC/AHA guidelines suggest categorizing the arrhythmia by its most frequent presentation.<sup>1</sup> Paroxysmal AF typically occurs in younger patients and accounts for approximately 40% of all cases of the disease.<sup>4</sup> However, AF is a chronic disease, and once present, recurrences can



**Figure 1. AF prevalence increases with age.<sup>2</sup>**



**Figure 2.** The clinical classification of AF is based on the duration of episodes.<sup>1</sup>

be expected, regardless of the disease classification.<sup>1</sup> Paroxysmal AF may evolve to persistent AF, and either type may become permanent. AF is typically considered permanent when it has been present for many years and cannot be reliably terminated with cardioversion.<sup>1,4</sup>

### Evaluation of the Patient With AF

The minimum evaluation for suspected AF should include a history and physical examination; ECG; transthoracic echocardiogram; and blood tests of thyroid, renal, and hepatic function.<sup>1</sup> The history and physical findings can help clinicians to assess the severity of the patient's symptoms and responses to any previously prescribed medications and to classify the type of AF according to its most frequent presentation.<sup>1</sup> The presence of any underlying heart disease or reversible causes of AF should also be determined. The ECG confirms the presence of AF and can help detect LVH or other cardiac abnormalities. The transthoracic echocardiogram can measure left ventricular ejection fraction (LVEF), identify valvular heart disease, and help determine thromboembolic risk. Blood tests of thyroid, renal, and hepatic function can help determine whether any reversible causes of AF are present.<sup>1</sup>

Other tests that may be useful include a 6-minute walk test, exercise testing, Holter monitoring or event recording, a transesophageal echocardiogram, an electrophysiologic study, or a chest radiograph. These investigations can help characterize cardiac and other factors relevant for determining the cause of the AF, for further elucidating patient history, or for guiding treatment.<sup>1</sup> Observations need to be made over a long period of time to diagnose paroxysmal AF, in which palpitations are less consistent than in persistent AF.<sup>4</sup>

### AF Management

The main reasons for treating AF are to relieve symptoms and prevent stroke (Figure 3).<sup>1</sup> For patients with disabling symptoms, the clinician has the option of pursuing either a rhythm-control or a rate-control strategy. No clear benefit from pursuing one strategy over the other has been demonstrated.<sup>1,7</sup>

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## Rhythm Control Versus Rate Control

These 2 strategies for treating AF have been compared in a number of randomized trials. The largest of these was the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) study, which enrolled more than 4000 patients with AF and a high risk of stroke or death.<sup>11</sup>

Overall mortality, the primary end point, was similar among patients assigned to either strategy.<sup>11</sup>

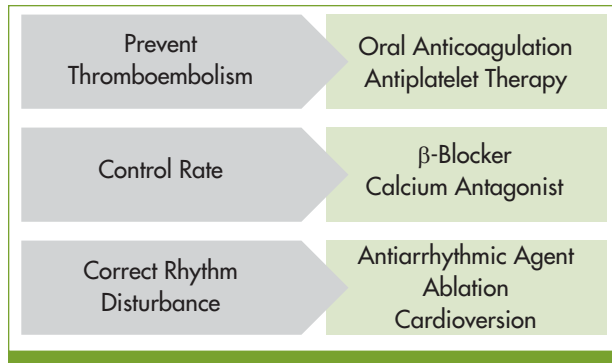
The Strategies of Treatment of Atrial Fibrillation (STAF) study showed no difference in the combined primary end point of death, cardiopulmonary resuscitation, cerebrovascular event, and systemic embolism. There were also no significant differences in any secondary end points except for hospitalization for CV reasons, which were more frequent in the rhythm-control group, although these were mostly for cardioversion and beginning antiarrhythmic therapy.<sup>12</sup> Similar results were observed in the Pharmacological Intervention in Atrial Fibrillation (PIAF) trial, which compared improvement in AF symptoms and quality of life after using the 2 strategies. Early termination due to adverse effects occurred in significantly more patients receiving amiodarone for controlling sinus rhythm than in those receiving diltiazem for controlling ventricular rate.<sup>13</sup>

Two other studies that found no significant differences between rate-control and rhythm-control strategies were the How to Treat Chronic Atrial Fibrillation (HOT CAFE) study and the Rate Control versus Electrical Cardioversion for Persistent Atrial Fibrillation (RACE) study, both of which had composite primary end points incorporating mortality, thromboembolisms, and bleeding; the RACE end point also included heart failure, implantation of a pacemaker, and severe adverse effects of medications.<sup>14,15</sup> In this latter trial, thromboembolic events occurred more frequently in the rhythm-control group, although the protocol permitted individuals in this group to end anticoagulant therapy after maintaining sinus rhythm for at least 1 month.<sup>15</sup>

## Rate-Control Agents

First-line agents for ventricular rate control in AF include calcium channel blockers and  $\beta$ -blockers. Digoxin is no longer a first-line therapy in AF but may be added, especially for patients with heart failure or left ventricular dysfunction. Adverse effects of these drugs include heart block, bradycardia, and hypotension. When pharmacotherapy is inadequate or the side effects are intolerable, atrioventricular junction ablation may be employed.<sup>1</sup>

In 2011, the ACC and AHA updated their recommendations for ventricular rate control for hemodynamically stable persons with AF based on the results of the RACE II trial. This study randomized patients with permanent AF to 1 of 2 rate-control strategies: a target heart rate  $\leq 80$  beats per minute at rest and  $\leq 100$  beats per minute after a 6-minute walk or a more



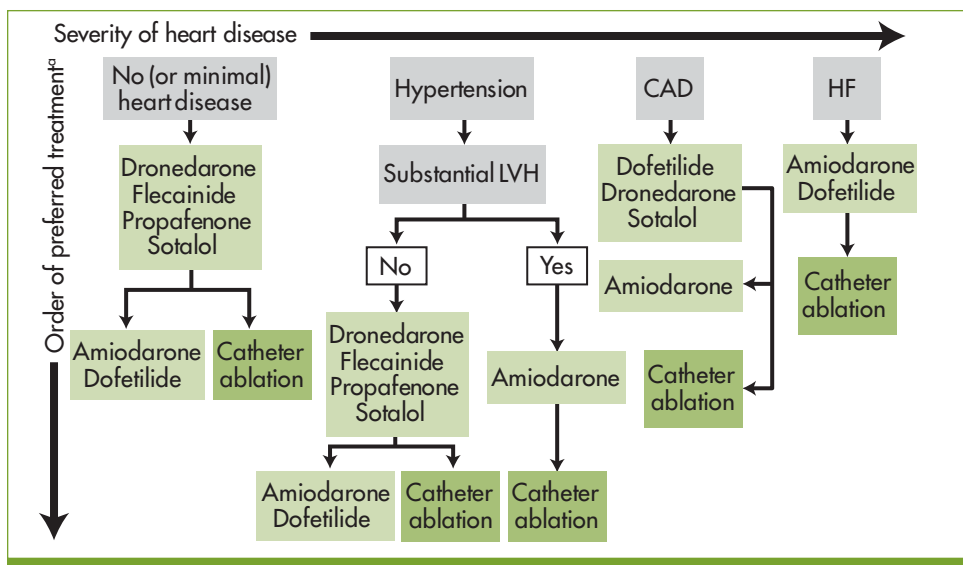
**Figure 3. The main reasons for treating AF are to relieve symptoms and prevent thromboembolism.<sup>1</sup>**

lenient target resting heart rate of <110 beats per minute. After 3 years, lenient rate control was found to be noninferior to strict rate control in preventing the composite primary outcome of death from CV causes, hospitalization for heart failure, and stroke, systemic embolism, bleeding, and life-threatening arrhythmic events. In addition, significantly more individuals in the lenient-control group achieved their target heart rate.<sup>16</sup> Thus, the updated ACC/AHA guidelines recommend lenient-rate control (resting heart rate <110 beats per minute) for patients with persistent AF who have stable ventricular function (LVEF >0.40).<sup>7</sup>

## Rhythm-Control Agents

Antiarrhythmic drugs (AADs) recommended in the ACC/AHA guidelines for maintaining sinus rhythm in patients with recurrent AF include amiodarone, dofetilide, dronedarone, flecainide, propafenone, and sotalol (Figure 4).<sup>1,7</sup> The choice of agent is based on the severity of underlying heart disease.

Dronedarone, a noniodinated amiodarone derivative, is the most recent addition to the consensus guidelines algorithm for maintaining sinus rhythm. The drug, which, unlike other AADs, can be started as outpatient therapy, is recommended in the ACC/AHA guidelines as an acceptable choice to reduce hospitalizations for CV events in patients with paroxysmal AF or after conversion of persistent AF.<sup>7</sup> This recommendation is based mainly on the results of the ATHENA trial (A Placebo-Controlled, Double-Blind, Parallel Arm Trial to Assess the Efficacy of Dronedarone 400 mg bid for the Prevention of Cardiovascular Hospitalization or Death from Any Cause in Patients with Atrial Fibrillation/Atrial Flutter), in which dronedarone significantly reduced the risk for CV hospitalization or death compared with placebo in patients



**Figure 4. Treatment recommendations for maintaining sinus rhythm in patients with recurrent AF are based on the severity of the underlying heart disease.**<sup>1,7</sup> <sup>a</sup>Within each category, drugs are listed alphabetically, not in order of suggested use. HF = heart failure. Reprinted with Permission. *Circulation*. 2011;123:104-123. ©2011 American Heart Association, Inc.

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75 years of age and older with AF or age 70 to 75 years with AF plus 1 or more CV risk factors (N = 4628) (Figure 5).<sup>17</sup>

However, the ACC/AHA guidelines advise that dronedarone should not be given to patients who have class IV heart failure or who had decompensated heart failure during the previous 4 weeks, particularly if they have reduced left ventricular function.<sup>7</sup> This recommendation is based on the results of ANDROMEDA (Antiarrhythmic Trial with Dronedarone in Moderate to Severe CHF Evaluating Morbidity Decrease).<sup>18</sup> In this study of patients with symptomatic heart failure and severe left ventricular systolic dysfunction, those in the dronedarone group had significantly higher mortality, largely due to worsening of heart failure, and the trial was ended prematurely for safety reasons.<sup>18</sup>

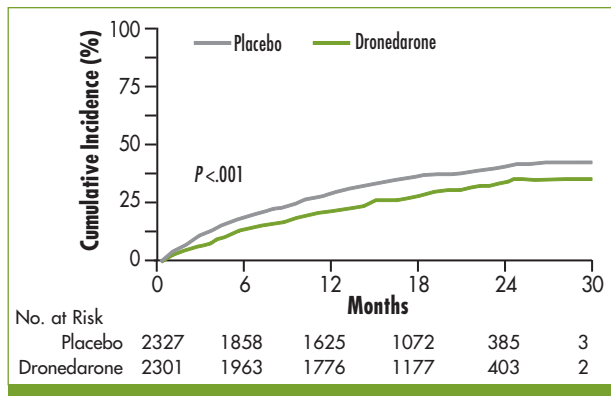
Dronedarone also should not be used in patients with permanent AF. The Food and Drug Administration (FDA) recently issued an alert based on preliminary findings from the Permanent Atrial Fibrillation Outcome Study Using Dronedarone on Top of Standard Therapy (PALLAS).<sup>19</sup> This trial was stopped early because data monitoring showed a 2-fold increase in death as well as 2-fold increases in stroke and hospitalization for heart failure in patients given dronedarone compared with those given placebo. The FDA review of the data is ongoing, and updates will be issued as warranted.

The first-line AADs for AF patients with little or no heart disease (dronedarone, flecainide, propafenone, and sotalol) are effective and generally well tolerated, and they can be taken on an as-needed basis. Although amiodarone maintains sinus rhythm over the long term more effectively than many of the other agents, it can cause potentially severe toxic side effects.<sup>1,20,21</sup> Thus, it is included among the second- or third-line choices, along with dofetilide.<sup>1,7</sup> Dronedarone is generally less effective than amiodarone but is better tolerated.<sup>22-24</sup>

For patients with AF and a history of hypertension with no LVH, initial treatment should be with medications that are known to be well tolerated, such as dronedarone, or that do not prolong repolarization or the QT interval, such as flecainide and propafenone.<sup>1,7</sup> Amiodarone, dofetilide, and sotalol are suitable second choices. For those with substantial LVH, amiodarone is considered to be the safest choice.<sup>1,7</sup>

Sotalol is often considered initially for maintaining sinus rhythm in AF patients with CAD or for adrenergic AF, with amiodarone, dofetilide, and dronedarone receiving secondary consideration. For patients with heart failure, amiodarone and dofetilide are usually preferred options.<sup>1,7</sup>

For all patient groups, if AF is not successfully controlled with any of these drugs, catheter ablation may be considered.<sup>1,7</sup> Patients should understand, however, that occasional recurrences of tolerable AF symptoms can occur while antiarrhythmic treatment is still considered successful.



**Figure 5. Dronedarone reduces risk for CV hospitalization or death.**<sup>17</sup> ©2009 Massachusetts Medical Society.

**Table 1.****Adverse Effects of Agents Used for Rhythm Control in Patients With AF<sup>1,7, 17, 25</sup>**

Agent	Potential Adverse Effects
Amiodarone	Photosensitivity, pulmonary toxicity, polyneuropathy, gastrointestinal upset, bradycardia, torsades de pointes (rare), hepatic toxicity, thyroid dysfunction, eye complications
Dofetilide	Torsades de pointes
Dronedarone	QT prolongation, gastrointestinal upset, bradycardia, photosensitivity or skin discoloration, increased serum creatinine levels
Flecainide	Ventricular tachycardia, heart failure, conversion to atrial flutter with rapid conduction through the atrioventricular node
Propafenone	Ventricular tachycardia, heart failure, conversion to atrial flutter with rapid conduction through the atrioventricular node
Sotalol	Torsades de pointes, heart failure, bradycardia, exacerbation of chronic obstructive or bronchospastic lung disease

Before prescribing AADs, the treating clinician should assess the benefits and risks of treatment for the individual patient, based on underlying heart disease and the pattern of AF.<sup>1</sup> One of the main concerns with AAD therapy is proarrhythmia in patients with structural heart disease.<sup>4</sup> Proarrhythmic effects differ according to the electrophysiologic properties of the medication.<sup>1</sup> Ventricular proarrhythmia effects include monomorphic ventricular tachycardia (seen with sodium channel blockers, such as flecainide or propafenone), polymorphic ventricular tachycardia (seen with potassium channel blockers, such as dofetilide or sotalol), and torsades de pointes. Atrial proarrhythmias include AF recurrence, an organization of AF to atrial flutter, and a rise in the defibrillation threshold. AADs may also cause sinus node dysfunction.<sup>1</sup> Potential adverse effects of AADs are summarized in Table 1.<sup>1,7,17,25</sup>

### Thromboembolism Prevention

Stroke is one of the most devastating consequences of AF. To prevent thromboembolism, the ACC/AHA guidelines on AF management recommend antithrombotic therapy for all patients with AF, except those with lone AF or contraindications.<sup>1,6</sup> Agent selection is based on each patient's stroke and bleeding risks; however, many patients with AF are undertreated, receiving no antithrombotic treatment or treatment inadequate for their stroke risk.<sup>26</sup>

Stroke risk in patients with nonvalvular AF can be estimated using the CHADS<sub>2</sub> scoring system (Table 2).<sup>27</sup> The calculated adjusted annual stroke rate ranges from 1.9 per 100 patient-years for patients with a CHADS<sub>2</sub> score of 0 to 18.2 per 100 patient-years for patients with a score of 6.<sup>27</sup> However, the simplest method of estimating stroke risk is to total the patient's CHADS<sub>2</sub> point score. If the patient's total CHADS<sub>2</sub> score is 0 or 1, the risk of stroke is low; a score of 2 or 3 indicates moderate stroke risk; and a score of  $\geq 4$  means the stroke risk is high.<sup>27</sup>

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**Table 2.**

## **CHADS<sub>2</sub> Scoring System for Assessing Stroke Risk in Patients With AF<sup>27</sup>**

CHADS <sub>2</sub> Risk Factor	No. of Points	Total CHADS <sub>2</sub> Score	Stroke Risk Classification
Cardiac failure (recent CHF)	1	0 or 1	Low
Hypertension (history of)	1	2 or 3	Moderate
Age ≥75 years	1	4, 5, or 6	High
Diabetes mellitus	1		
Stroke or TIA (history of)	2		

TIA = transient ischemic attack.

Warfarin, a vitamin K antagonist, and aspirin, a thromboxane inhibitor, have been the mainstay of thromboprophylaxis for patients with AF.<sup>1</sup> The efficacy of warfarin for reducing stroke in patients with AF has been well characterized. In a meta-analysis of 16 randomized trials, adjusted-dose warfarin was found to decrease the relative risk of stroke by 62% compared with placebo. Aspirin, by contrast, reduced the relative risk by 22%.<sup>28</sup>

Unfortunately, warfarin may be inappropriate for some AF patients because of drug-drug interactions, increased bleeding risk, or an inability to comply with frequent international normalized ratio (INR) monitoring. Such patients have traditionally received aspirin instead (despite its modest efficacy for stroke prevention) or no therapy at all. One study estimates that half of AF patients receive no antithrombotic treatment, regardless of their stroke risk level.<sup>29</sup> In 2011, the ACC and AHA updated their recommendations for antithrombotic therapy, adding 2 alternatives to warfarin: dual antiplatelet therapy with clopidogrel plus aspirin and dabigatran, a direct thrombin inhibitor (Table 3).<sup>1,6,7</sup>

The ACC/AHA recommendation for dual antiplatelet therapy in patients for whom warfarin is deemed unsuitable is based largely on the results of the ACTIVE-A trial (Atrial Fibrillation Clopidogrel Trial with Irbesartan for Prevention of Vascular Events: Effect of Clopidogrel Added to Aspirin in Patients with Atrial Fibrillation).<sup>30</sup> When clopidogrel was added to aspirin in patients with AF, the risk of a major vascular event (stroke, non-central nervous system systemic embolism, myocardial infarction, or death from vascular causes) was significantly lower after 3.5 years of follow-up than with aspirin alone (relative risk [RR], 0.89; 95% confidence interval [CI], 0.81-0.98;  $P = .01$ ).<sup>30</sup> This was chiefly due to a significant decrease in annual stroke incidence (2.4% with clopidogrel plus aspirin vs 3.3% with aspirin alone,  $P < .001$ ). The incidence of major bleeding events was higher with dual antiplatelet therapy than with aspirin monotherapy, 2.0 versus 1.3% per year, respectively.<sup>30</sup> The guidelines note, however, that warfarin remains the preferred antithrombotic option, having been shown to be superior to clopidogrel plus aspirin for preventing vascular events in AF patients in the ACTIVE-W trial.<sup>31</sup>

The ACC/AHA guidelines recommend dabigatran as a useful alternative to warfarin for prevention of stroke and systemic thromboembolism in patients with paroxysmal to permanent AF who have no contraindications. This recommendation is based largely on the results of the Randomized Evaluation of Long-Term Anticoagulation Therapy (RE-LY) trial.<sup>32</sup> In this noninferiority trial, patients with AF received dabigatran (110 mg or 150 mg, twice daily) or

adjusted-dose warfarin (target INR 2.0-3.0). Dabigatran at 150 mg bid was superior to warfarin for the primary outcome of preventing stroke or systemic embolism (RR, 0.66; 95% CI, 0.53-0.82;  $P < .001$ ); rates of major hemorrhage were similar in the 2 treatment groups. Dabigatran at 110 mg was noninferior to warfarin for the primary efficacy outcome (RR, 0.91; 95% CI, 0.74-1.11;  $P < .001$ ), and the rate of major bleeding was significantly lower.<sup>32</sup>

A recent report of 2 cases, 1 fatal, of major bleeding with dabigatran suggests that caution is warranted when using the drug in the elderly.<sup>33</sup> The risk of major overdosage may be increased in this population because of renal function impairment, low body weight, and drug interactions. Currently no antagonist is available.

### Patient Education

Primary care clinicians need to educate their patients about the risks associated with AF (particularly the risk for stroke), its treatments, and how to manage the condition over the long term.<sup>1,4</sup> Patients should understand that the primary goals of therapy for AF are to relieve symptoms and prevent thromboembolism and that their treatment regimen will be individualized based on the severity of symptoms and underlying heart disease. Patients should recognize their own level of stroke risk as well as the warning signs of stroke. In addition to antithrombotic therapy, the AF treatment plan may include medication to control the patient's heart rate or rhythm. Thus, clinicians should teach their patients not only about the proarrhythmic effects and other risks of AADs but also about the benefits of being in sinus rhythm, mainly, symptom relief, so that patients will be more likely to adhere to treatment. Patients should also be aware that catheter ablation may be an option if pharmacotherapy for their AF is unsuccessful. Finally, patients should be taught how to monitor their own heart rate and rhythm and to report any changes in their condition.

**Table 3.**

### Recommendations for Antithrombotic Therapy in Patients With AF<sup>1,6,7</sup>

Agent (Dose)	Recommendation
Aspirin (81-325 mg/d)	For patients with no risk factors or as an alternative to oral anticoagulation in patients with 1 moderate-risk factor or those with contraindications to oral anticoagulation
Warfarin (INR 2.0-3.0, target 2.5) <sup>a</sup>	For patients with $\geq 1$ moderate-risk factors or any high-risk factor
Clopidogrel (75 mg/d)	As addition to aspirin therapy if oral anticoagulation with warfarin is considered unsuitable because of patient preference or clinician-assessed ability to sustain anticoagulation safely
Dabigatran (150 mg twice a day) <sup>b</sup>	As an alternative to warfarin in patients with paroxysmal to permanent AF who do not have a prosthetic heart valve or hemodynamically significant valve disease, severe renal failure, or advanced liver disease

<sup>a</sup>If the patient has a mechanical valve, target INR  $>2.5$ .

<sup>b</sup>For patients with a creatinine clearance of 15-30 mL/min, a dose of 75 mg twice a day is recommended.

## CASE: 47-Year-Old Woman With Irregular Tachypalpitation



### History and Presentation

Diane is a 47-year-old woman, who is married and has 2 teenaged children. She presents to the emergency department with symptomatic, irregular tachypalpitations. Her medical history includes hypertension and mild renal insufficiency.

### Physical Findings

- Height: 5 ft 6 in
- Weight: 130 lb
- Body mass index: 21.0 kg/m<sup>2</sup>
- Blood pressure: 110/60 mm Hg
- Heart rate: 130 bpm
- Mild dyspnea
- ECG: no discernible P waves and an irregular ventricular response

### Laboratory and Imaging Test Results

- Thyroid-stimulating hormone: 2.2 mIU/L
- Serum creatinine: 1.9 mg/dL
- Liver function tests: normal
- Transthoracic echocardiogram: LVEF 0.60; mild LVH (consistent with her history of hypertension)

### Clinical Decision Point

*Based on her symptoms and ECG results, Diane is diagnosed with an initial presentation of AF. What are the treatment goals at this point?*

- Control symptoms
- Prevent myocardial infarction
- Maintain sinus rhythm
- Prolong survival

### Comment

The best answer is control symptoms. The primary treatment goal at this point is to control Diane's rapid heart rate. Diane's symptoms may improve if her heart rate is controlled. Maintaining sinus rhythm is not essential now but should be addressed if symptoms continue to be bothersome.

### Clinical Decision Point

*How should Diane's AF be treated initially?*

- Catheter ablation
- Digoxin and clopidogrel
- Flecainide and warfarin
- $\beta$ -blocker and aspirin

### Comment

The best answer is  $\beta$ -blocker and aspirin. The ACC/AHA guidelines recommend that treatment for AF begin with controlling the ventricular rate and preventing thromboembolism.<sup>1</sup>  $\beta$ -blockers and calcium channel blockers are first-line therapy

for ventricular rate control in AF. AAD therapy and catheter ablation are not indicated at this point. Diane's CHADS<sub>2</sub> score is 1; she has 1 moderate-risk factor for stroke: a history of hypertension. The guidelines recommend stroke prevention with aspirin for patients with only 1 moderate-risk factor.<sup>1</sup>

### Clinical Course

Diane's AF converted to normal sinus rhythm with  $\beta$ -blocker therapy. At discharge, the clinician instructs her to continue  $\beta$ -blocker therapy and to take 325 mg/d of aspirin. Diane returns home and resumes her normal activities. After 1 week, she begins to experience dizzy spells.

### Clinical Decision Point

#### *What could be the cause of Diane's symptoms?*

- $\beta$ -blocker-induced bradycardia
- Recurrent AF
- Side effect of medication
- Any of the above

### Comment

The best answer is any of the above. Each of these options could be causing Diane's symptoms. The most likely cause, however, is recurrent AF. Therapeutic options include changing the dose of the  $\beta$ -blocker; adding a calcium channel blocker to her regimen; and considering the addition of an AAD. In anticipation of the possibility cardioversion will be needed, switching from aspirin to warfarin or dabigatran also should be considered.

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