Providing Best Practice in the Management of Atrial Fibrillation in the United States

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Background: Atrial fibrillation (AF) is a prevalent arrhythmia. Patients with AF may report a variety of symptoms and often describe compromised quality of life. Atrial fibrillation increases the risk of stroke, heart failure, and all-cause mortality. Purpose: The purpose of this review article was to provide an overview of AF management based on current guidelines and new data. Conclusions: The 2011 American College of Cardiology Foundation/American Heart Association/Heart Rhythm Society guideline update provides diagnostic and management recommendations for the patient with AF based on the current evidence. Clinical Implications: Nurses are integral to the care of patients with AF. It is essential for nurses to stay apprised of current guidelines and new evidence so that the assessment, management, and education of the AF patients and their families can be optimized.

KEY WORDS: atrial fibrillation, nurses, treatment guidelines

Atrial fibrillation (AF) is a supraventricular arrhythmia that is frequently encountered in the clinical setting. The earliest report of AF appears to be in the Yellow Emperor’s Classic of Internal Medicine, which dates from approximately 1696 to 2598 BC, although the invention of the electrocardiograph did not occur until 1900. Over the past century, AF was often viewed as a benign arrhythmia. However, over the past decade, extensive research has been performed that has provided new insight into the impact and treatment of AF.

The prevalence of AF in the United States is estimated to be 3.03 million and is projected to be 7.56 million in 2050. Given this continued increase in AF and expanding treatment options, the practice of evidence-based treatment is critical. Many management guidelines have been published, including the guidelines for the management of patients with AF from the American College of Cardiology (ACC), American Heart Association (AHA), and European Society of Cardiology (ESC) and the 2011 Focused Update on the Management of Patients With Atrial Fibrillation published by the American College of Cardiology Foundation (ACCF), AHA, and the Heart Rhythm Society (HRS) (some sections of the 2006 guidelines have not changed). Guidelines serve to provide a range of recommendations for treatment based on the current best evidence with the goal of improving patient care and outcomes. Although AF is common and comprehensive guidelines exist, management of this patient population remains challenging and complex.

Nurses in a wide variety of settings play an important role in the treatment and management of patients with AF. Therefore, it is important for nurses to be aware of the best evidence that supports current recommendations and treatment strategies to improve patient outcomes. The purpose of this review was to provide an overview of AF management based on the current AF guidelines, with a focus on patient outcomes, available pharmacologic and nonpharmacologic therapies, and new and emerging treatment options.

Significance of Atrial Fibrillation

Atrial fibrillation is an uncoordinated atrial systole characterized by an irregular ventricular response. The loss of atrial synchrony is reflected on the electrocardiogram (ECG) by the absence of consistent P waves (Figure 1). This irregular rhythm results in loss of the atrial contribution to cardiac output (with an overall reduction in cardiac output) and to symptoms of palpitations, fatigue, and dyspnea. This
progressive disease becomes more difficult to treat with increasing duration. In clinical practice, AF is the most commonly reported arrhythmia and accounts for approximately one-third of cardiac arrhythmia hospitalizations. The number of hospitalizations for AF has increased by 144% over the last 20 years, and patients with AF are often rehospitalized, with most readmissions occurring within 6 months of the initial hospitalization. Costs associated with inpatient management are a key contributor to the overall healthcare costs of AF. Data from the Framingham Heart Study revealed that AF was a predictor of mortality in both men and women; at age 40, the lifetime risk for developing AF is 26.0% for men and 23.0% for women. The presence of AF is an independent risk factor for ischemic stroke, which has an average rate of 5% per year in patients with AF.

Patients with AF also are reported to have a markedly impaired health-related quality of life (HRQOL). When compared with the HRQOL among other patient populations, HRQOL among AF patients was either significantly worse or as impaired as that of patients who were post–myocardial infarction or diagnosed with heart failure (ie, patient populations with a greater degree of structural heart disease than do patients with AF).

**Classification of Atrial Fibrillation**

According to the 2006 ACC/AHA/ESC guidelines, the 3 major clinical categories of AF are defined as paroxysmal, persistent, and permanent. Paroxysmal AF refers to episodes of less than 7 days’ duration that generally stop spontaneously. In persistent AF, episodes last more than 7 days and are not self-terminating but require cardioversion to restore sinus rhythm. Permanent AF is defined as sustained AF after failure of cardioversion. The categories are not mutually exclusive, and patients should be categorized according to their most frequent presentation.

**Goals of Therapy**

Atrial fibrillation is a complex arrhythmia to treat. Irrespective of the classification of AF, the management of patients should be guided by the patient’s symptoms and associated comorbidities. The general cardiovascular health of the patient needs to be addressed and managed. Although not recommended by the 2006 ACC/AHA/ESC guidelines, there is some clinical evidence to indicate that drugs modulating the renin-angiotensin-aldosterone system (angiotensin-converting enzyme inhibitors and angiotensin receptor blockers) may decrease the incidence of AF and that HMG CoA-reductase inhibitors (statins) may prevent
AF recurrence.7 Treatment of AF should meet 3 general goals: (1) rate control, (2) reduction of thromboembolic complications, and (3) establishment and maintenance of sinus rhythm.7 The initial decision in managing patients with AF is to determine whether to restore sinus rhythm or use a rate control strategy. The treatment strategy should take into consideration the stability of the patient, predisposing factors, AF duration, patient age, and comorbid medical conditions. Regardless of the treatment approach, anticoagulation is based on stroke risk and not on whether sinus rhythm is maintained.7

**Risk Stratification**

The presence of AF increases the risk for a thromboembolic event. Such an event is a devastating complication of AF and can be reduced with antithrombotic therapy. Several antithrombotic clinical trials have facilitated identification of risk factors that predispose patients with AF to a greater risk of ischemic stroke.18 Because stroke risk is dependent upon age and comorbid conditions, the Cardiac Failure, Hypertension, Age, Diabetes, Stroke (Doubled) (CHADS2) scoring system (Table 1) is frequently used to stratify the stroke risk of AF patients and aid in the decision to use antithrombotic therapy.7

The CHADS2 acronym represents identified risk factors. Each risk factor is assigned a score of 1, with the exception of prior stroke or transient ischemic attack, which is assigned a score of 2. Patients who score 2 or higher with either 1 high-risk factor or more than 1 moderate-risk factor are considered at high risk for stroke. Patients who have no risk factors are considered at low risk. Patients who have 1 moderate-risk factor have an intermediate risk for stroke (3%–5% per year).7 The 2010 ESC guidelines also recommend a risk factor–based approach for patients with nonvalvular AF, expressed as the acronym CHA2DS2-VASc (Congestive Heart Failure, Hypertension, Age ≥75 [Doubled], Diabetes, Stroke [Doubled], Vascular Disease, Age 65–74, and Sex Category [female]). This scheme is based on a point system in which 2 points are assigned for a history of stroke or transient ischemic attack or age 75 years or older and 1 point each is assigned for age 65 to 74 years, a history of hypertension, diabetes, recent cardiac failure, vascular disease, and female sex. This score defines patients and their risk of stroke. This acronym extends the CHADS2 scheme by considering additional stroke risk factors.19

**Thromboprophylaxis**

Stroke is the most common thromboembolic event in AF and occurs at a higher frequency among individuals with AF compared with those without AF.7 Antithrombotic therapy has been shown to be effective in reducing the incidence of stroke; the 2006 ACC/AHA/ESC guidelines highlight the need for initiation of thromboprophylaxis with minimal delay after establishing a diagnosis of AF.7 In general, anticoagulation is recommended whether the patient has paroxysmal, persistent, or permanent AF.7,20 Antithrombotic recommendations according to categories of risk are listed in Table 2.7 Unless contraindicated, patients who are determined to be at high risk for a thromboembolic event should be placed on warfarin. An international normalized ratio (INR) range of 2.0 to 3.0 should be maintained. A meta-analysis revealed that adjusted-dose warfarin is highly efficacious for prevention of all strokes (both hemorrhagic and ischemic), with a risk reduction of approximately 60% versus placebo in 6 clinical trials comprising 2900 participants.21 Among low-risk AF patients, aspirin is recommended. However, in moderate-risk patients, warfarin with maintenance of an INR between 2.0 and 3.0 or daily aspirin is recommended.7 Although warfarin is more efficacious for prevention, aspirin offers modest protection, with an approximately 20% reduction in stroke among AF patients compared with no treatment.20,21 Among patients considered to be unsuitable for oral anticoagulation with warfarin because of patient preference or physician assessment, the addition of clopidogrel to aspirin to reduce the risk of stroke may be considered.8,22,23 A careful balance between stroke prevention and bleeding complications is necessary so that the benefit of antithrombotic therapy offsets the occurrence of hemorrhage.21 Unfortunately, more than half of AF patients do not receive appropriate antithrombotic therapy despite guideline recommendations.24 Nonpharmacologic stroke prevention (ie, atrial appendage closure devices) in patients who cannot safely undergo antithrombotic therapy is a rapidly growing area of study.7

**TABLE 1** The Cardiac Failure, Hypertension, Age, Diabetes, Stroke (Doubled) Index

<table>
<thead>
<tr>
<th>Risk Criteria</th>
<th>Score</th>
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<tbody>
<tr>
<td>Prior stroke or TIA</td>
<td>2</td>
</tr>
<tr>
<td>Age &gt;75 years</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Heat failure</td>
<td>1</td>
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</table>

Dabigatran is a new oral direct thrombin inhibitor that recently received US Food and Drug Administration (FDA) approval (2010). This new anticoagulant is designed for patients with nonvalvular AF. Dabigatran is taken twice daily and has an elimination half-life of 12 to 17 hours. However, dabigatran does not require periodic adjustment or monitoring of the INR. Patients with AF receiving dabigatran 150 mg reported fewer major bleeds (3.1%) compared with those receiving warfarin (3.4%). Dyspepsia was reported more frequently with dabigatran. The rate of myocardial infarction was found to be higher with dabigatran compared with warfarin. The 2011 ACCF/AHA/HRS guideline update states that dabigatran is useful as an alternative to warfarin for the prevention of stroke and systemic thromboembolism in patients with paroxysmal to permanent AF and risk factors for stroke or systemic embolism who do not have a prosthetic heart valve or hemodynamically significant valve disease, severe renal failure, or advanced liver disease.

### Rate Control and Rhythm Control

The current established treatment options for patients with AF are rate control and rhythm control. Under the rate control strategy, the ventricular rate is controlled without restoration of sinus rhythm. The aim of rate control is to reduce the ventricular rate and reduce the symptoms of AF. The rhythm control strategy attempts to restore and maintain sinus rhythm, with attention to rate control. The treatment strategy selected should be tailored to the individual patient. Rate control may be a reasonable initial therapy in older patients with persistent AF or in patients with mild symptoms attributable to AF. Rhythm control is often considered in younger patients with a shorter duration of AF and in patients who can tolerate antiarrhythmic drugs. The management of AF with pharmacotherapy can be suboptimal because of the complexity of treatment. Initial rhythm control drug therapy in newly diagnosed patients is associated with a high rate of discontinuation, especially early in therapy (predictors of discontinuation of initial drug therapy for AF included cardiac arrest before or after the index date, history of coronary artery bypass graft surgery, valvular heart disease, ischemic heart disease, and severity of illness). Persistence with some antiarrhythmic agents (especially amiodarone) is poor, and patients who discontinue treatment are unlikely to restart therapy; lack of efficacy and drug-related adverse effects may be important reasons for the overall poor persistence. Clinical studies show no significant difference in major clinical outcomes including total mortality, thromboembolic events, and symptomatic improvement between rate and rhythm control strategies. In addition, several studies found no difference in reported HRQOL when rhythm control was compared with rate control.

### Pharmacologic Management of Rate Control

The goal of rate control is to control the ventricular rate during rest and exercise while avoiding excessive tachycardia as well as excessive bradycardia. Adequate rate control varies among clinical trials but is recommended to range between 60 and 80 beats/min at rest and between 90 and 115 beats/min with exercise. However, lenient rate control may be a reasonable strategy in some patients with permanent AF.

In the RACE II study (Rate Control Efficacy in Permanent Atrial Fibrillation: A Comparison Between...
Lenient Versus Strict Rate Control II), lenient rate control (resting heart rate <110 beats/min) was not inferior to strict rate control (resting heart rate <80 beats/min and heart rate during moderate exercise <110 beats/min). Current medications recommended for rate control of AF (β-blockers, calcium channel blockers [CCBs], and digoxin), along with mechanisms of action and considerations for nurses, are presented in Table 3.7,32

The efficacy of β-blockers in controlling heart rate is well established in this patient population. A thorough patient assessment is necessary before implementation to ensure that there are no contraindications (eg, history of asthma). Common adverse effects include bradycardia, hypotension, and fatigue. Caution should be used when starting β-blockers in patients with low blood pressure and/or heart failure. The 2006 ACC/AHA/ESC guidelines recommend not only β-blockers for long-term rate control management of AF but also implementation of β-blocker therapy before cardiac surgery to prevent postoperative AF.7

Verapamil and diltiazem are nondihydropyridine CCBs commonly used to control rapid heart rate during an acute event and may also be used in long-term AF management. When given intravenously, nondihydropyridine CCBs have a rapid onset of action (1–5 minutes), and a continuous infusion is usually given because of the short half-life of the drugs.32 Calcium channel blockers may be preferred in patients with known respiratory disease but should be administered cautiously in heart failure patients.7 When initiating CCBs, patients may experience peripheral vasodilatation with associated hypotension and lightheadedness, as well as bradycardia.32 Careful monitoring of these patients is warranted. With long-term use of CCBs, patients may report lower extremity edema and constipation.32,33 Nondihydropyridine CCBs are the only agents that have been associated with an improvement in HRQOL and exercise tolerance in patients with AF.7

Digoxin is no longer considered a first-line therapy except in patients with left ventricular dysfunction because of controversial data concerning this drug as well as the availability of more effective agents.7 The most common adverse effects of digoxin are ventricular arrhythmias, atrioventricular (AV) block, and sinus pauses.7 Drug interactions and toxicity can be problematic with digoxin. Therefore, close follow-up and monitoring for signs of toxicity are necessary. Because digoxin is almost completely eliminated by the kidneys, changes in renal function and electrolytes can significantly contribute to toxicity. In addition, several drugs interact with digoxin, such as amiodarone, diltiazem, propafenone, spironolactone, and verapamil, leading to increased digoxin levels.32 Dronedarone also increases digoxin levels 1.7- to 2.5-fold.34 When digoxin is given in conjunction with these agents, a reduction in the digoxin maintenance dose and testing is necessary to ensure that a therapeutic dose is maintained and potential complications are avoided. The frequency of testing will depend upon the patient and drug given in conjunction with digoxin. A digoxin level should be considered within 1 week of initiation of therapy, when the dose of digoxin has been changed, or when the dose of drug given in conjunction with digoxin is changed.

**Pharmacologic Agents for Rhythm Control**

The decision to maintain sinus rhythm in the patient with AF must be based on patient history, reported symptoms, and the potential adverse effects of antiarrhythmic drugs. For patients who have recurrent paroxysmal or persistent AF, several drugs are available for the maintenance of sinus rhythm. However, determining which drug to use should be individualized.
and based upon whether the patient has underlying cardiovascular disease.7,8 Drugs such as flecainide, propafenone, or sotalol can be used in patients who do not have structural heart disease and in patients with hypertension but without left ventricular hypertrophy.7,33 The use of these drugs decreased the recurrence of AF (42%–67% vs 71%–84% for controls) but also increased the rate of adverse effects, including proarrhythmias.34 Guidelines recommend class IC and selected class III medications for rhythm control.8 Table 4 contains a list of rhythm control medications, mechanisms of action, pharmacokinetics, and nursing considerations.7,32,36,37 Recommended rhythm control medications for patients with paroxysmal or persistent AF include flecainide, propafenone, amiodarone, dronedarone, dofetilide, and sotalol (Figure 2).8

In patients with no structural heart disease, class IC agents (flecainide and propafenone) are considered first-line treatment based on the current guidelines.7,8 Both drugs can contribute to proarrhythmia effects. The more common adverse effects of flecainide include dizziness, nervousness, headache, fatigue, blurred vision, and nausea.32 The adverse effects of propafenone include metallic taste in the mouth, constipation, nausea, vomiting, dizziness, fatigue, and conduction abnormalities.32 Careful continued assessment for medications that may interact with the class IC drugs, as well as monitoring the 12-lead ECG and renal, pulmonary, and hepatic functions, will serve to reduce arrhythmia complications.

Amiodarone is a class III antiarrhythmic drug. It reduces the automaticity of the sinus atrial node, prolongs repolarization, increases conduction time and the refractory period of the AV node, and reduces the conduction in the Purkinje fibers.32 Although amiodarone is not FDA approved for AF in the United States, in a recent Cochrane review, it was concluded that amiodarone is the most effective agent for reducing the recurrence of AF.35

Amiodarone is a benzofuran derivative containing iodine. Because amiodarone is lipophilic, it has an extremely long half-life (average of 58 days). When orally administered, amiodarone also has a highly variable absorption.32 Chronic use of amiodarone is limited by adverse effects including bradycardia, photosensitivity, pulmonary toxicity, thyroid dysfunction, liver toxicity, and visual disturbances; in addition, amiodarone is associated with prolongation of the QT interval.32 Close follow-up of patients receiving amiodarone is warranted. Nurses following this patient population should obtain liver, thyroid, and pulmonary function tests at baseline. A baseline ophthalmologic evaluation should also be obtained in any patient who has significant visual abnormalities. The follow-up evaluation should include a yearly ECG and chest x-ray film and, semiannually, a thyroid profile and profile of liver enzymes.39 Regardless of the effectiveness of amiodarone, the potential adverse effects make it a second-line choice for AF patients who do not have contraindications to other antiarrhythmic drugs.39

Dronedarone was approved by the US FDA in 2009 for reducing the risk of cardiovascular hospitalization in patients with paroxysmal or persistent AF with a recent episode of AF/atrial flutter and associated cardiovascular risk factors who are in sinus rhythm or who will be cardioverted.8 Dronedarone has pharmacological properties similar to those of amiodarone but lacks the iodine moiety, which is thought to be responsible for amiodarone’s pulmonary, thyroid, hepatic, and ocular toxicity.39 Dronedarone inhibits potassium, sodium, and calcium channels and has α- and β-adrenergic-blocking activities.39 The half-life of dronedarone is 13 to 19 hours, with no significant accumulation in the tissue.37 It has demonstrated an improved safety profile over amiodarone.40 In a study that compared the efficacy of dronedarone with that of amiodarone, dronedarone was less effective than amiodarone in decreasing AF recurrence.41 In 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), dronedarone was shown to reduce hospitalizations due to cardiovascular events or death from any cause by 24% compared with placebo.42 A post hoc analysis of the ATHENA trial showed that there was a significant reduction in the risk of stroke with dronedarone compared with placebo.43 Given the favorable benefit-to-risk profile of dronedarone, it may be considered for treatment of AF. However, the ANDROMEDA trial (Antiarrhythmic Trial With Dronedarone in Moderate to Severe CHF Evaluating Morbidity Decrease), which included high-risk patients with heart failure, was discontinued early after an interim safety analysis revealed an excess risk of death in patients on active treatment.44 Therefore, dronedarone is contraindicated in patients with New York Heart Association class IV heart failure and in patients with New York Heart Association class II or III with a recent decompensation requiring hospitalization or referral to a specialized heart failure clinic.37 The 2011 ACCF/AHA/HRS guideline update does not recommend the use of dronedarone in patients with heart failure.45 Several cases of liver injury and hepatic failure in patients receiving dronedarone have been reported in the postmarketing setting, including 2 reports of acute hepatic failure requiring transplantation and new-onset or worsening heart failure.45,46
<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Pharmacokinetics</th>
<th>Nursing Considerations</th>
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<tbody>
<tr>
<td>Class IC</td>
<td></td>
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<tr>
<td>Flecainide</td>
<td>Blocks inward movement of sodium: depresses</td>
<td>Complete oral absorption, moderately bound to plasma proteins, biotransformed in</td>
<td>Assess for contraindications</td>
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<td></td>
<td>action potential phase 0, slows conduction</td>
<td>liver, excreted in urine</td>
<td>Monitor vital signs</td>
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<td></td>
<td></td>
<td></td>
<td>Withdraw previous antiarrhythmic therapy</td>
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<td></td>
<td></td>
<td></td>
<td>Monitor ECG rhythm, PR and QRS intervals</td>
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<td>Correct electrolytes</td>
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<td></td>
<td></td>
<td>Avoid medications that are known to interact with class IC drugs, such as digoxin and</td>
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<td></td>
<td>cimetidine</td>
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<td></td>
<td></td>
<td></td>
<td>Monitor QT/QTc intervals</td>
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<tr>
<td>Propafenone</td>
<td>Mechanism of action similar to that of</td>
<td>Complete oral absorption, bioavailability reduced</td>
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<td></td>
<td>flecainide with weak β-blocking activity</td>
<td>because of first-pass metabolism</td>
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<td>Class III</td>
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<tr>
<td>Amiodarone</td>
<td>Potent sodium and calcium channel blocker;</td>
<td>Variable absorption, biotransformed in liver, excreted in bile</td>
<td>Monitor for adverse effects</td>
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<tr>
<td></td>
<td>prolongs repolarization and increases conduction</td>
<td></td>
<td>Baseline liver, thyroid, and PFTs</td>
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<td></td>
<td>time; weak α- and β-blocking activity</td>
<td></td>
<td>Baseline ophthalmologic evaluation (patients with significant visual abnormalities)</td>
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<td>Follow-up evaluation: yearly ECG and chest x-ray film and, semianually, a thyroid</td>
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<td>profile and profile of liver enzymes</td>
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<td>Monitor for increased effects of warfarin and digoxin</td>
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<td>Monitor QT/QTc intervals</td>
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<td>Review and follow manufacturer's ECG monitoring requirements</td>
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<td>Monitor QT/QTc intervals</td>
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<td>Calculate and monitor creatinine clearance</td>
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<td>Assess and treat potassium and magnesium levels</td>
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<td>Multiple drug interactions</td>
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<td>Monitor for adverse effects</td>
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<td></td>
<td>Monitor for development of or worsening heart failure</td>
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<td></td>
<td>Baseline and periodically assess BMP and LFT</td>
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<td></td>
<td>Monitor QT/QTc intervals and ECG rhythm</td>
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<td></td>
<td></td>
<td>Maintain potassium and magnesium levels within reference range</td>
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<tr>
<td>Dofetilide</td>
<td>Selective potassium channel blocker; increases</td>
<td>Well absorbed orally, biotransformed in liver, excreted in urine and feces</td>
<td>Monitor INR level after initiating dronedarone in patients taking warfarin</td>
</tr>
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<td></td>
<td>the action potential duration</td>
<td></td>
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<tr>
<td>Dronedarone</td>
<td>Inhibits potassium, sodium, and calcium channels</td>
<td>Low systemic bioavailability, extensive first-pass hepatic metabolism, excreted</td>
<td>Monitor for adverse effects</td>
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<td></td>
<td>and has α- and β-blocking activities</td>
<td>mostly in feces</td>
<td>Monitor development of or worsening heart failure</td>
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<td>Baseline and periodically assess BMP and LFT</td>
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<td>Monitor QT/QTc intervals and ECG rhythm</td>
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<td>Maintain potassium and magnesium levels within reference range</td>
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<tr>
<td>Sotalol</td>
<td>Blocks β1 and β2 adrenergic receptors,</td>
<td>Rapidly absorbed, biotransformed in liver, excreted in urine</td>
<td>Assess for contraindications</td>
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<tr>
<td></td>
<td>prolonging the action potential</td>
<td></td>
<td>Monitor renal function and electrolytes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Monitor QT/QTc intervals</td>
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</table>

Abbreviations: BMP, basic metabolic panel; ECG, electrocardiogram; INR, international normalized ratio; LFT, liver function test; PFT, pulmonary function test.

*aUpdated prescribing information for Multaq can be found at http://www.fda.gov/Drugs/DrugSafety/ucm264059.htm.
updated prescribing information recommends obtaining periodic hepatic serum enzymes, particularly during the first 6 months of treatment.37 Postmarketing cases of increased INR with or without bleeding events have also been reported in patients on warfarin initiated on dronedarone.37,47

Dofetilide is a class III antiarrhythmic drug. In the SAFIRE-D study (Symptomatic Atrial Fibrillation Investigative Research on Dofetilide), dofetilide demonstrated a 58% probability of maintaining sinus rhythm 1 year after cardioversion compared with 25% for placebo.48 Dofetilide can contribute to prolongation of the QT interval and lead to ventricular arrhythmias. Therefore, it is currently recommended that initiation of dofetilide occur in the hospital with doses titrated to renal function and QT interval.48 Monitoring of the corrected QT interval, electrolytes, and creatinine clearance in follow-up can serve to further reduce arrhythmia risk.

Sotalol is a class III antiarrhythmic drug that seems to be equally as effective as propafenone for maintenance of sinus rhythm.49 The most common adverse effects of sotalol include bradycardia and palpitations.32 Based on the current guidelines, sotalol can be initiated on an outpatient basis.7,8 According to the 2011 guideline update, sotalol can be beneficial in outpatients in sinus rhythm with little or no heart disease and prone to paroxysmal AF if the baseline uncorrected QT interval is less than 460 milliseconds, serum electrolytes are normal, and risk factors associated with class III drug-related proarrhythmia are not present.8 If the patient receives sotalol as an outpatient, continued periodic monitoring of the QT interval, electrolytes, and renal function is warranted.

Atrial fibrillation is usually a chronic arrhythmia, and many patients will eventually experience a recurrence. According to guidelines, well-tolerated recurrence of AF is reasonable as a successful outcome of antiarrhythmic drug treatment.8 Careful follow-up and correction of modifiable risk factors are necessary along with periodic assessment to prevent potential adverse effects. When a medication does not result in symptom improvement or results in adverse effects, the medication should be discontinued and an alternative treatment should be selected according to guidelines.7

**FIGURE 2.** Pharmacologic maintenance of sinus rhythm.7 *Recommended first-line medications. Drugs are listed alphabetically and not in order of suggested use. Please refer to the 2011 American College of Cardiology Foundation/American Heart Association/Heart Rhythm Society guideline update for a more detailed discussion.5 LVH indicates left ventricular hypertrophy. Reprinted from Journal of the American College of Cardiology, vol 57, no 11, Fuster V, Ryden LE, Cannom DS, et al. 2011 ACCF/AHA/HRS focused updates incorporated into the ACC/AHA/ESC 2006 guidelines for the management of patients with a trial fibrillation: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines Developed in partnership with the European Society of Cardiology and in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society, e101–e198, 2011, with permission from Elsevier.
New Pharmacologic Agents for the Management of Atrial Fibrillation

Phase II clinical trials examining the maintenance of sinus rhythm with celivarone (which is structurally related to amiodarone) were recently completed. This drug was compared with placebo in the maintenance of sinus rhythm with an incidence of 90-day recurrence at 52% compared with 67% for placebo. Celivarone is also under investigation with amiodarone for the prevention of implantable cardioverter defibrillator interventions or death. Another new investigational drug is vernakalant, a sodium and potassium channel blocker with a very short half-life (2–3 hours). Intravenous vernakalant has recently been approved (September 2010) in the European Union, Iceland, and Norway for the rapid conversion of recent-onset AF to sinus rhythm in adult non-surgery patients with AF of 7 or fewer days’ duration and for adult post–cardiac surgery patients with AF of 3 or fewer days’ duration. Vernakalant blocks sodium and ultrarapid potassium currents in the myocardium. Phase 2 placebo-controlled studies demonstrated that oral vernakalant successfully maintained sinus rhythm. Common adverse reactions included dysgeusia, paresthesia, sneezing, and nausea.

Nonpharmacologic Management of Atrial Fibrillation

Direct-Current Cardioversion

Direct-current cardioversion is a well-established form of treatment to restore sinus rhythm. The goal of cardioversion is to depolarize the atrial cells and prevent reentry (convert the heart from AF to normal sinus rhythm). Patients who present as hemodynamically compromised with a rapid ventricular response not responsive to pharmacologic measures or who are symptomatic are recommended for treatment with cardioversion. The effectiveness of direct-current cardioversion can be influenced by several factors, including the duration of AF, atrial size, and the presence of other cardiac conditions. Patients may be treated with antiarrhythmic medications before and after cardioversion to enhance the success of the procedure. Devices that use biphasic waveforms require less energy and are recommended compared with the monophasic-waveform devices; devices with biphasic waveforms have a median successful energy level of 100 joules versus 200 joules for devices with monophasic waveforms. Paddle position with an anterior-posterior configuration also enhances the success and reduces the required energy.

Patients with AF lasting longer than 48 hours or those at increased risk for thromboembolism should receive 3 weeks of oral anticoagulation (target INR of 2.0–3.0) before cardioversion is recommended, and anticoagulation should be maintained for 4 weeks post-cardioversion. However, transesophageal echocardiography may be used to exclude thrombi and allow for immediate cardioversion with intravenous heparin. Patients must also be given warfarin for a period of time after transesophageal echocardiography with cardioversion.

Atrial Pacing and Defibrillation

Studies have shown atrial pacing to be associated with a reduced risk of AF and stroke when compared with ventricular pacing. Atrial pacing can decrease ectopic beats, which may trigger AF. Although atrial pacing shows promise, its use as a primary treatment in preventing recurrent AF has not been established. Large-scale studies are warranted to further examine this treatment approach.

Currently, atrial defibrillators have limited utility in the treatment of AF except for patients with left ventricular dysfunction who are candidates for an implantable ventricular defibrillator. Several devices are available that combine both atrial cardioversion and ventricular defibrillation capabilities and have been designed to treat both atrial and ventricular arrhythmias by pacing before delivering a shock.

Catheter Ablation

Ablation is an invasive procedure in which a catheter is placed in the heart and targeted sites are destroyed by radiofrequency. This technique has evolved in recent years from linear scars in the atrium to targeting ectopic foci in the pulmonary vein. Patients undergoing this procedure have reported improvement in their symptoms and HRQOL. Studies have shown atrial pacing to be associated with a reduced risk of AF and stroke when compared with ventricular pacing. However, there are insufficient data to support the long-term efficacy of catheter ablation to prevent recurrent AF. The ThermaCool trial reported significantly fewer episodes of recurrent AF after catheter ablation compared with additional antiarrhythmic drugs. Therefore, catheter ablation may be a reasonable treatment option for patients with symptomatic persistent or paroxysmal AF and have failed treatment with 1 or more antiarrhythmic drugs. The success and benefit of catheter ablation compared with additional antiarrhythmic drugs.

Ablation of the AV node with implantation of a permanent pacemaker, however, is a well-established rate control treatment in select patients. Patients with refractory AF who are symptomatic and unable to tolerate pharmacologic treatment are most likely to benefit from an AV node ablation and pacemaker. In a meta-analysis of 21 studies involving 1181 patients, significant improvements in HRQOL and clinical outcomes were recorded after AV node ablation and
Treatment of atrial fibrillation (AF) should meet 3 general goals: (1) rate control, (2) reduction of thromboembolic complications, and (3) establishment and maintenance of sinus rhythm. The 2011 American College of Cardiology Foundation (ACCF)/American Heart Association (AHA)/Heart Rhythm Society (HRS) guideline update \(^7\) (update to the 2006 ACC/AHA/ESC guidelines \(^8\)) discusses recommendations for strict versus lenient heart rate control, combined use of antplatelet and anticoagulant therapy, use of dronedarone and dabigatran, and catheter ablation. It is essential for nurses to stay apprised of current guidelines and new evidence so that the assessment, management, and education of AF patients and their families are optimized.

**Surgical Maze Procedure**

The goal of this surgical procedure is to reestablish or maintain sinus rhythm. The surgical approach was based on the hypothesis that reentry is the primary mechanism responsible for the development and maintenance of AF; as such, atrial incisions at critical locations would create barriers to conduction and prevent sustained AF. The maze procedure involves encircling the pulmonary veins and linear atrial incisions over both atria with radiofrequency, cryoablation, or laser. This procedure forces atrial conduction through designated atrial areas to the AV node. The reported success rates range from 70% to greater than 97%. \(^7,6,2\) Despite its high success rate, the maze procedure requires the use of cardiopulmonary bypass, which is the reason it has not been widely adopted except in patients undergoing other cardiac surgical procedures. \(^7,6,2\)

**Conclusions**

Atrial fibrillation has a profound negative impact on patients’ perceived HRQOL, mortality, and healthcare costs. Each patient presents with an individual collection of symptoms and comorbid disease states that must be taken into consideration when choosing a management strategy. Established evidence-based guidelines provide direction for treatment and management of the AF patient with the primary goals of stroke prevention and amelioration of symptoms. As the number of patients with AF increases, nurses must be prepared to play a critical role in the care and treatment of this patient population. It is essential for nurses to stay apprised of current guidelines and new evidence that can serve to optimize the assessment, management, and education of AF patients and their families.

**REFERENCES**


