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ACC Guidelines for Atrial Fibrillation: 2006 Update

Atrial fibrillation (AF) is the most common cardiac rhythm disturbance affecting an estimated 2.2 million people in the United States and often requires hospitalization (416,000 hospital discharges per year). Its growing prevalence in the aging population is associated with high rates of morbidity and mortality and is believed to be responsible for 15% to 20% of all strokes. With the increasing incidence of chronic heart disease and more frequent diagnosis of AF, this arrhythmia represents a substantial challenge to physicians.

Since the last guidelines for the management of atrial fibrillation (AF) in 2001 there has been a wealth of clinical trial data and technologic advances that necessitated an update. In August of this year, the American College of Cardiology (ACC), the American Heart Association (AHA), and the European Society of Cardiology (ESC) released the new guidelines.[1] Less then 10% of physicians will read the executive summary (only 53 pages).[2] Clearly, almost no one will read the 98 pages in it's entirety including myself. The purpose of this *Heartbeat* will be to boil these guidelines down into a user-friendly packet of information. These recommendations will assist physicians to optimize and improve the treatment of their patients. The most important differences and the new recommendations on AF management on which physicians should focus will be outlined.

Management of AF

Antithrombotic Therapy—Stroke Prevention: Anticoagulation is the paramount treatment issue in patients with AF and the new guideline changes are more clear regarding this often confusing matter. The most obvious change to the new recommendations is that the threshold for anticoagulation has been raised. Warfarin treatment has been eliminated in individuals whose stroke risk is marginal. Previously instituting warfarin in otherwise healthy patients between 65 and 75 years old would mean an enormous lifestyle change. The new guidelines will improve the overall care of patients and obviate the need for anticoagulation, unneeded labs and follow-up.

Avoiding stroke is critical in those with AF. Emphasis is now placed on identifying patients' risk factors for stroke ($CHADS_2$—Table 1.) and this will help guide optimal treatment plans.

Table 1. ACC/AHA/ESC 2006 Guidelines: Risk Factors for Stroke

Less Validated/Weaker Risk Factors	Moderate Risk Factors	Score	High Risk Factors	Score
Female gender	**C**ardiac failure (LVEF < 35%)	**C - 1**	Previous **S**troke, TIA, or embolism	**S 2**
Age 65-74 yrs	**H**ypertension	**H - 1**	Mitral stenosis	
Coronary artery disease	**A**ge ≥ 75 yrs	**A - 1**	Prosthetic heart valve	
Thyrotoxicosis	**D**iabetes mellitus	**D - 1**		

LVEF = left ventricular ejection fraction; TIA = transient ischemic attack

The recommendation of *81-325 mg of aspirin* has been made for patients with less validated or weaker risk factors. For those with one moderate risk factor, aspirin or warfarin is recommended. *Warfarin* is recommended for all those with previous TIA or stroke, systemic embolism, prosthetic valves and those with two or more moderate risk factors (Table 2.).

Table 2. ACC/AHA/ESC 2006 Guidelines: Recommended Therapies According to Stroke Risk

Risk Category	Recommended Therapy
No risk factors	Aspirin, 81-325 mg daily
One moderate risk factor	Aspirin, 81-325 mg daily, or warfarin (INR 2.0-3.0, target 2.5)
Any high risk factor or ≥ 1 moderate risk factor	Warfarin (INR 2.0-3.0, target 2.5)*

*If mechanical valve, target INR greater than 2.5 (2.6-3.5)

Obviously, there will still be some clinical decision-making (*aspirin vs. warfarin*) for the moderate-risk AF patients who are eligible for warfarin. Patient preference should always be part of the decision process. After the benefits and risks of warfarin are reviewed patients generally react in 1 of 2 ways. Some patients are incredibly frightened by the notion of a stroke, because a family member or friend has suffered a stroke. These patients tend not to have a problem with warfarin treatment and long-term follow-up because they're afraid of the devastating effects stroke can incur—even though stroke risk is low. Most patients, however, strongly dislike taking medications and would rather avoid repetitive phlebotomy for INR level checks. The majority of patients are likely to choose aspirin therapy and avoid warfarin if you tell them two important points: firstly, their stroke risk is fairly low and secondly, there isn't an enormous difference between the 2 treatment options in terms of outcomes. This decision should always be documented. When there is a preference—like the patient who is 70 in the moderate risk group—I lean toward treatment with warfarin earlier rather than later as risk increases with age.

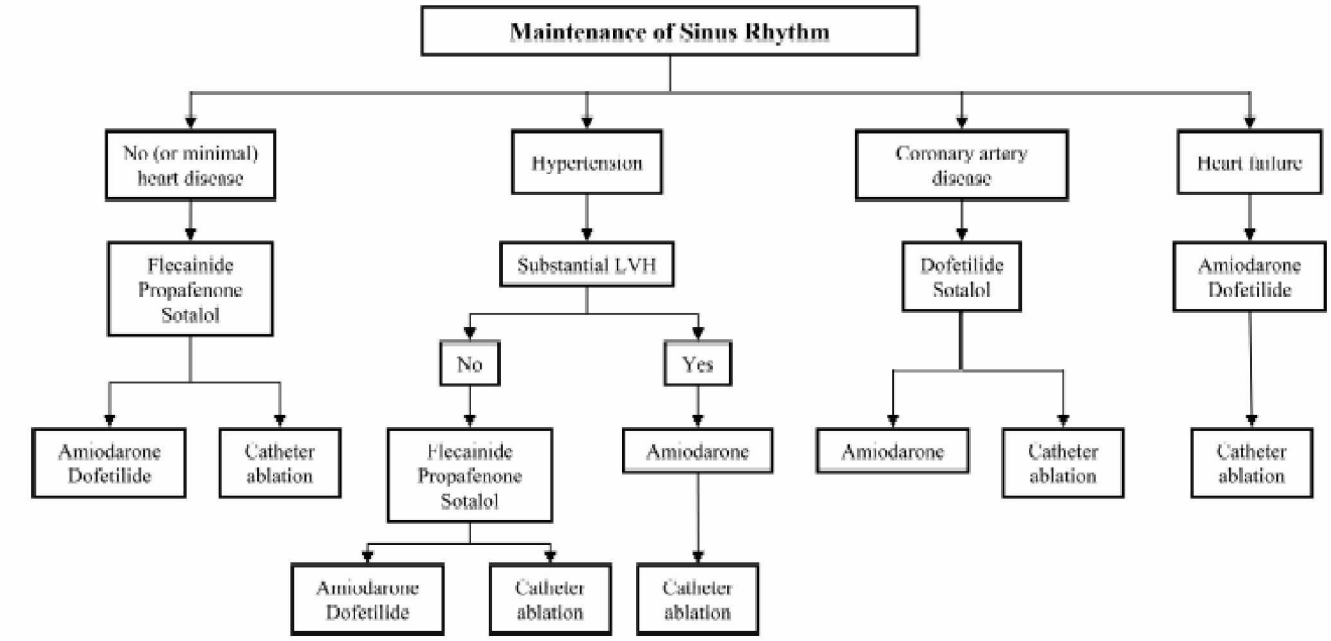
Finally, a key point that must always be remembered is that the antithrombotic therapy decision is separate and distinct from the rate and rhythm control decision. Utilization of anticoagulation therapy should be determined solely by stroke risk. In clinical terms, for example, the patient on rhythm-control strategy in normal sinus rhythm (NSR) will still need warfarin if they are determined to be high risk or have greater than one (two) moderate risk factors using the $CHADS_2$ scoring system.

Rate or Rhythm Control Strategy—Control Symptoms: The **A**trial **F**ibrillation **F**ollow-up **I**nvestigation of **R**hythm **M**anagement (**AFFIRM**)[3] and the **RA**te **C**ontrol versus **E**lectrical Cardioversion for Persistent Atrial Fibrillation (**RACE**)[4] trials were two landmark studies published in 2002. The investigators found that treating AF with a rhythm-control strategy involving cardioversion and class III antiarrhythmic drug (AAD) therapy offers no survival or clinical advantages over simpler rate-control therapy with medications such as calcium channel blockers and beta-blockers. In fact, the potential benefits of a rhythm-control strategy were largely offset by the adverse side effects associated with AAD therapy. These studies have led to the feeling that rate and rhythm control are both reasonable options.

The newer trial findings have resulted in physicians leaning away from the automatic expectation of immediate AF cardioversion or attempted restoration of NSR upon initial discovery of new AF—although still a viable option in this setting for certain patients (young and no structural heart disease so as to prevent atrial remodeling). It's not that AF is just as good as NSR—we all like NSR—but the key question is whether we can keep them in NSR safely. One of the side effects of antiarrhythmic drugs for rhythm control is the possibility of proarrhythmia, further aggravating AF.

Once the mortality risk has been addressed by tailoring the appropriate antithrombotic therapy to individual patients, the focus switches to the management of symptoms. There is no "one size fits all" therapy. Dealing with symptoms will drive the therapeutic decision and treatment should be targeted to improve the quality of life of each patient. If rate control is a priority and coronary artery disease is present, avoid AAD if possible and use beta-blockers. For the majority of our older patient population, depending on symptoms, rate control is reasonable in the face of hypertension or heart disease. The problem is with people who are very symptomatic. They don't want to hear that rate control is just as good. Following the algorithm provided in Figure 1 is a good guide to starting with the appropriate AAD rhythm control treatment. Always consider efficacy and safety of ADD in a given patient population depending on their associated heart disease. Flecainide, propafenone and amiodarone can be started as an outpatient. Due to possible serious adverse side effects initiating s*otalol* requires continuous monitoring and should be *started only as an inpatient.* Although no drugs have been eliminated in the new guidelines, quinidine and procainamide have been deemphasized because they are considered less effective or incompletely studied.

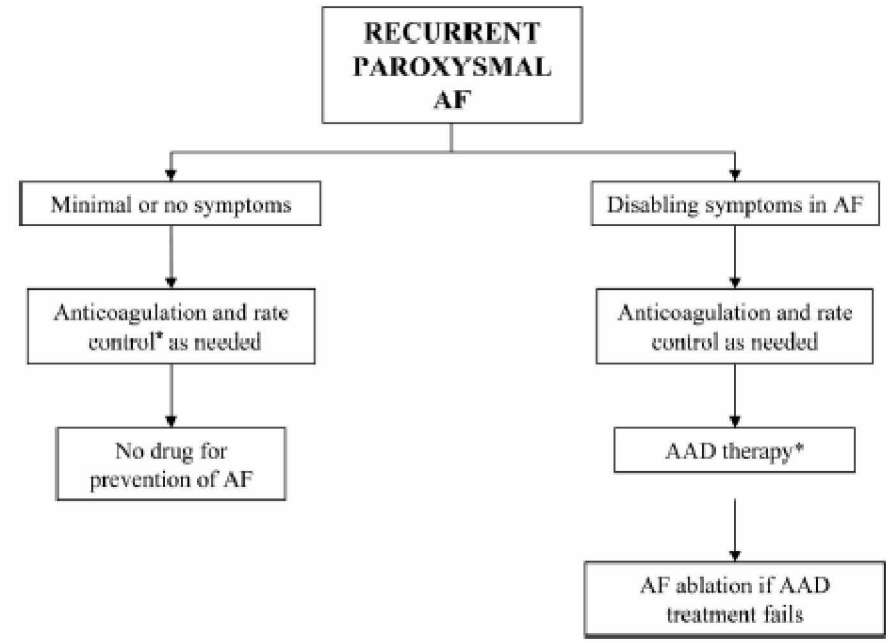
Figure 1. Antiarrhythmic drug therapy to maintain sinus rhythm in patients with recurrent paroxysmal or persistent atrial fibrillation. Within each box, drugs are listed alphabetically and not in order of suggested use. The vertical flow indicates order of preference under each condition. The seriousness of heart disease proceeds from left to right, and selection of therapy in patients with multiple conditions depends on the most serious condition present. LVH indicates left ventricular hypertrophy.



Younger patients without existing structural heart disease, particularly those with lone paroxysmal AF (PAF), may benefit from rhythm control. The new guidelines reflect the growing acceptance of the use of catheter ablation to treat AF (rhythm control) as well as rate control. Under the previous guidelines, patients were typically expected to fail amiodarone therapy before they were considered for ablation therapy—treatment of last resort. Now, in terms of options, amiodarone and ablation are considered equal. Since ablation is an invasive procedure, difficult and not guaranteed, most experts would try at least one good rhythm control medication before proceeding to ablation. If a patient fails at least one reasonable medication—flecainide, propafenone or sotalol—it is unlikely that switching medications is going to make any difference. So then the real question becomes do you go with amiodarone or catheter

ablation therapy for rhythm control in the symptomatic patient with recurrent PAF (Figure 2.)? A young or middle aged patient with PAF and little or no structural heart disease—no left atrial enlargement—should be a favorable candidate for ablation and the success rate is pretty good. If they had failed AAD treatment, they shouldn't be expected to undergo multiple drug trials. Moreover, consideration of long-term complications, especially with amiodarone, should be taken into account. Catheter ablation therapy would be a reasonable option in this setting.

Figure 2. Management of patients with Recurrent PAF.



Adjunctive Therapy: Recent studies have demonstrated some benefit of ACE-inhibitors, angiotensin receptor blockers, statins and omega III fatty acids in maintaining NSR. At this time the recommendation is that these medications should be encouraged for appropriate indications (hypertension, diabetes, heart failure and dyslipidemia) but not solely for AF prevention until further more conclusive study.

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[1] Fuster V et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation). *J Am Coll Cardiol* August 15 2006; 48: e149 - e246.

[2] ACC/AHA/ESC 2006 Guidelines for the Management of Patients With Atrial Fibrillation—Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation) *J Am Coll Cardiol* August 15 2006 48: 854-906.

[3] Wyse DG et al. The atrial fibrillation follow-up investigation of rhythm management (AFFIRM Investigators). A comparison of rate control and rhythm with atrial fibrillation. *N Engl J Med* 2002; 347: 1825-1833.

[4] Van Gelder IC et al. A comparison of rate control and rhythm control in patients with recurrent persistent atrial fibrillation (RACE). *N Engl J Med*. 2002; 347: 1834-1840.