in the clinic

Atrial Fibrillation

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Atrial fibrillation, the most common clinically significant arrhythmia, occurs when a diffuse and chaotic pattern of electrical activity in the atria replaces the normal sinus mechanism, leading to deterioration of mechanical function. Atrial fibrillation is a major cause of morbidity, mortality, and health care expenditures, with a current prevalence of 2.3 million cases in the United States and an estimated increase in prevalence to 15.9 million by the year 2050 (1). Atrial fibrillation is associated with a 5-fold increased risk for stroke and is estimated to cause 15% of all strokes (2). Independent of coexisting diseases, the presence of atrial fibrillation confers a 2-fold increased risk for all-cause mortality (3).

Diagnosis

Who is at risk for atrial fibrillation?

Atrial fibrillation occurs in less than 1% of individuals age 60 to 65 years, but in 8% to 10% of those older than 80 years. Prevalence is higher in men than in women and higher in whites than in blacks. The risk for atrial fibrillation increases with the presence and severity of underlying heart failure and valvular disease.

What symptoms and signs should cause clinicians to suspect atrial fibrillation?

Many patients, particularly the elderly, are asymptomatic during atrial fibrillation. Other patients may have prominent symptoms, including palpitations, shortness of breath, exercise intolerance, or malaise. When present, symptoms are generally greatest at disease onset, when episodes are typically paroxysmal, and tend to diminish over time or when the pattern becomes persistent. Symptoms during atrial fibrillation result from elevation of ventricular rate (at rest and exaggerated with exercise), the irregular nature of the ventricular rate, and the loss of atrial contribution to cardiac output. Even patients with severe symptoms during atrial fibrillation episodes often have episodes of silent atrial fibrillation as well (4), which has important implications for therapeutic strategies, especially anticoagulation.

On physical examination, the signs of atrial fibrillation include a faster-than-expected heart rate (which is quite variable from patient to patient), an irregularly (irregular in timing) irregular (in terms of the amplitude of the pulse) peripheral pulse, as well as irregular heart sounds on auscultation.

Is a single electrocardiogram (ECG) sufficient to diagnose or exclude atrial fibrillation?

Figure 1 is an example of an ECG showing atrial fibrillation. A single ECG is sufficient to diagnose atrial fibrillation if it is recorded during an arrhythmia episode. However, atrial fibrillation is often paroxysmal, so a single ECG showing normal rhythm does not exclude the diagnosis. Longer-term monitoring can be helpful when atrial fibrillation is suspected but the ECG is normal. In patients with daily paroxysmal symptoms, 24- or 48-hour continuous Holter monitoring is usually sufficient to make the diagnosis. In patients with less-frequent symptoms, monitoring during longer periods with electrocardiographic loop recorders may be necessary for diagnosis. However, even monitoring for periods as long as a month can be nondiagnostic in patients with very infrequent episodes. In addition, because patients trigger loop recorders to record when symptoms occur, these recorders are not helpful in detecting episodes of asymptomatic arrhythmia. In some cases, a diagnosis of atrial fibrillation occurs only after several years of symptoms because of the nonspecific nature of symptoms

1. Miyasaka Y, Barnes ME, Gersh BJ, et al. Secular trends in incidence of atrial fibrillation in Olmsted County, Minnesota, 1980 to 2000, and implications on the projections for future prevalence. Circulation. 2006;114:119-25. [PMID: 16818816]
2. Hart RG, Benavente O, McBride R, Pearce LA. Antithrombotic therapy to prevent stroke in patients with atrial fibrillation: a meta-analysis. Ann Intern Med. 1999;131:492-501. [PMID: 10507957]

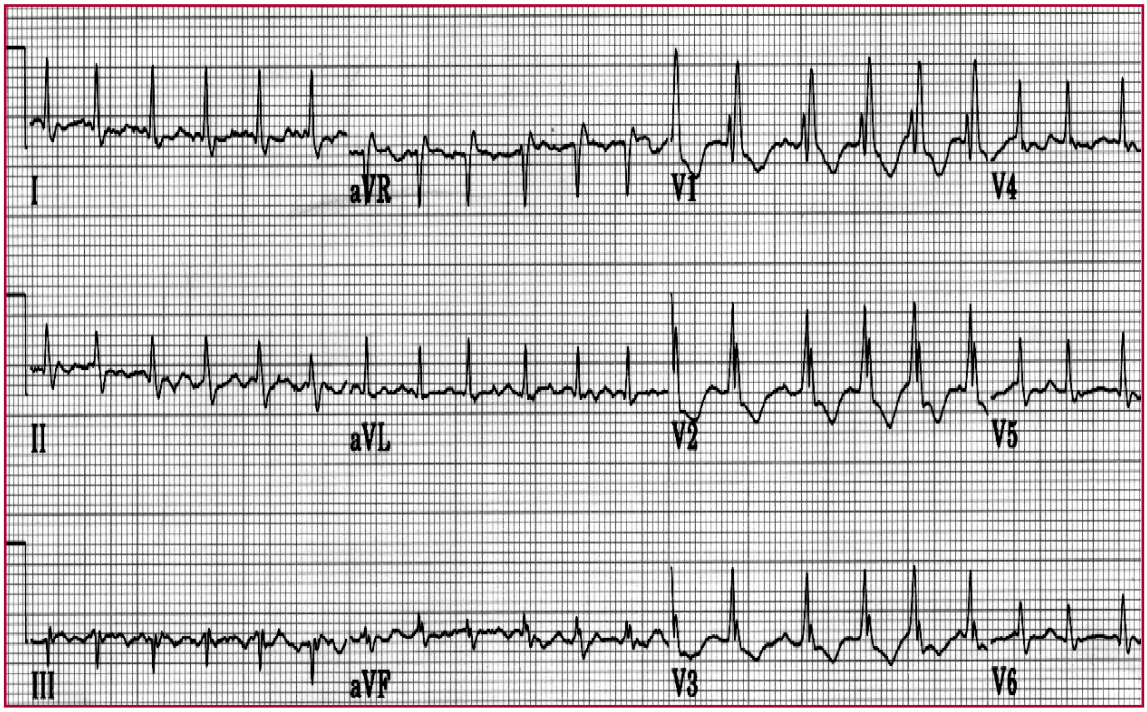

Figure 1. Electrocardiogram showing atrial fibrillation with rapid ventricular rate.

and the occasionally long periods between episodes in some patients at the beginning of the disease process.

What is the role of history and physical examination in patients with atrial fibrillation?

History and physical examination can help to determine the duration of symptoms and potential underlying causes. Clinicians should document history and physical examination evidence of hypertension, heart failure, murmurs indicating structural heart disease, or recent cardiac surgery. In addition, clinicians should look for signs and symptoms of noncardiac causes of atrial fibrillation, including pulmonary disease; hyperthyroidism; use of caffeine or other stimulants; or use of adrenergic drugs (such as those

3. Benjamin EJ, Wolf PA, D'Agostino RB, et al. Impact of atrial fibrillation on the risk of death: the Framingham Heart Study. Circulation. 1998;98:946-52. [PMID: 9737513]
4. Page RL, Wilkinson WE, Clair WK, et al. Asymptomatic arrhythmias in patients with symptomatic paroxysmal atrial fibrillation and paroxysmal supraventricular tachycardia. Circulation. 1994;89:224-7. [PMID: 8281651]

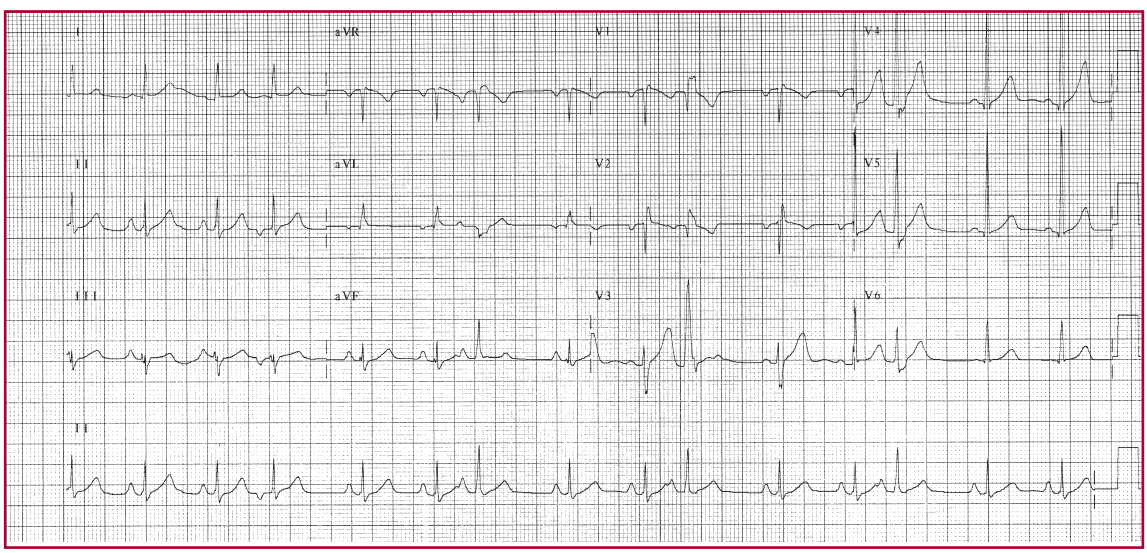

Figure 2. Electrocardiogram showing sinus rhythm with frequent PACs.

Classification of Atrial Fibrillation

Paroxysmal: Episodes spontaneously terminate within 7 days

Persistent: Episodes last >7 days and require intervention to restore sinus rhythm

Permanent: Interventions to restore sinus rhythm have either failed or have not been attempted

used to treat pulmonary disease) or alcohol.

What other electrocardiographic arrhythmias might clinicians confuse with atrial fibrillation?

Other arrhythmias that are commonly confused with atrial fibrillation include sinus rhythm with frequent premature atrial contractions (PACs), atrial tachycardia, and atrial flutter. The key electrocardiographic components of atrial fibrillation are the absence of discernable P waves and an irregular ventricular response without any pattern. When the diagnosis of atrial fibrillation is unclear, clinicians should obtain long rhythm strips with multiple lead combinations to evaluate for irregular rhythms and P waves. Clinicians should look for deformed T waves or ST segments for evidence of P waves.

Sinus rhythm with frequent PACs is an irregular rhythm, but P waves are present (Figure 2). Atrial tachycardia and atrial flutter have evident atrial activation, but may be conducted to the ventricles in an irregular matter; even in this circumstance, there is a pattern to the irregular conduction (Figure 3). This is an important distinction because catheter ablation is first-line therapy in many patients with atrial tachycardia and atrial flutter.

How should clinicians classify atrial fibrillation according to duration and frequency?

Although there is a great deal of confusion regarding this question, the convention is to describe atrial fibrillation by the qualifiers paroxysmal, persistent, or permanent (5). "Paroxysmal" means that atrial fibrillation episodes terminate without intervention within 7 days but often in less than 24 hours. "Persistent" means that episodes last longer than 7 days or require intervention (such as cardioversion) to restore sinus rhythm. "Permanent" means that interventions to restore sinus rhythm have either failed or have not been attempted. These qualifiers are not necessarily mutually exclusive in a given patient, so clinicians should characterize the current or most-usual pattern.

Traditionally, these distinctions have been used to predict response to therapy. The response

5. American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 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): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Circulation. 2006;114:e257-354. [PMID: 16908781]

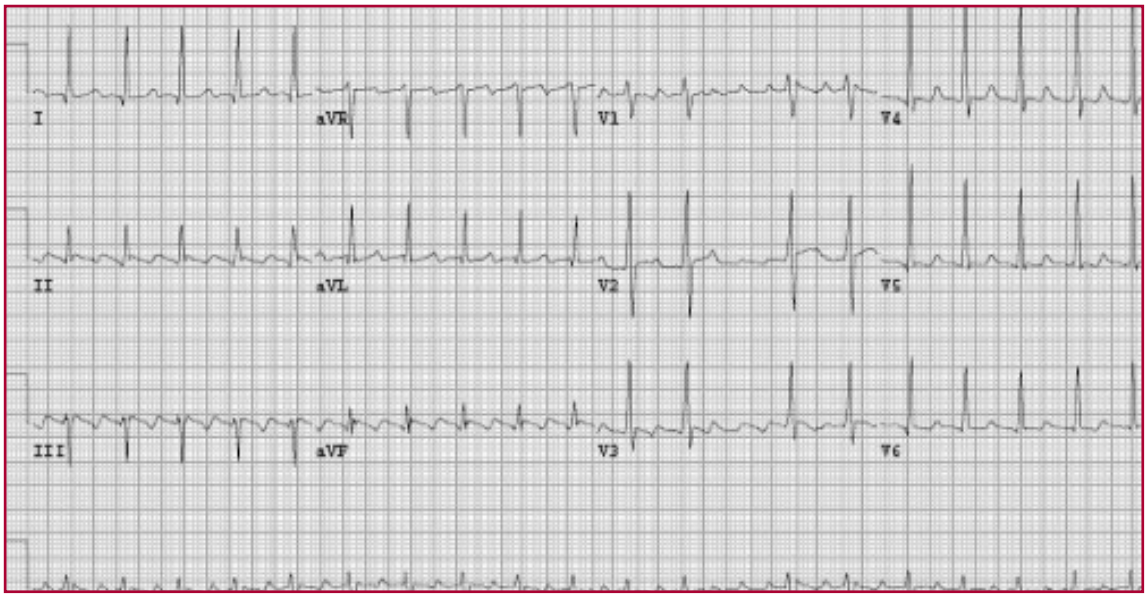

Figure 3. Atrial flutter. Classic "saw-tooth" flutter waves are seen in all 12 leads, and the ventricular response is mostly regular. (There is a transient change from 2:1 to 4:1 atrioventricular conduction following the 12th QRS complex.)

to antiarrhythmic drug therapy is less favorable as the pattern goes from paroxysmal to persistent to permanent. There is no difference in the need for anticoagulation on the basis of the pattern of atrial fibrillation.

What laboratory studies should clinicians obtain in patients with atrial fibrillation?

On initial presentation of atrial fibrillation, clinicians should measure sensitive thyroid-stimulating hormone to exclude hyperthyroidism, serum electrolytes, and renal and hepatic function to evaluate risk for toxicity to specific drugs. Transthoracic echocardiogram is generally helpful to evaluate the presence of structural heart disease, but is specifically able to evaluate left atrial size, valvular heart disease, pericardial disease, and left ventricular hypertrophy, which are associated with responsiveness to antiarrhythmic therapy. Transesophageal echocardiogram is indicated to exclude atrial clot when transthoracic images are inadequate or when cardioversion is planned in a patient who has been anticoagulated for less than 3 weeks. In selected patients, tests to evaluate specific disease processes that may be causative in acute episodes of atrial fibrillation, such as pulmonary embolism, acute myocardial infarction, or acute heart failure, may also be appropriate. Clinicians should test stool for occult blood before initiation of anticoagulation.

What underlying cardiac and noncardiac conditions should clinicians look for in patients with atrial fibrillation?

Of patients with atrial fibrillation, 80% have some component of structural heart disease, particularly hypertensive heart disease, but also coronary disease, valvular heart disease, or cardiomyopathies of any cause. Because the incidence of atrial fibrillation is markedly affected by aging, age-related atrial fibrosis is considered central to the arrhythmia's pathogenesis. Some patients have atrial fibrillation in the absence of identifiable heart disease, which is referred to as lone atrial fibrillation, but the usefulness of this designation is a matter of some debate. Some experts believe that the designation "lone atrial fibrillation" should be restricted to patients younger than 60 years of age because it is difficult to exclude structural heart disease in older patients.

In 30 years of follow-up of 5209 participants in the Framingham Study, 193 men and 183 women developed atrial fibrillation. Of these, 32 men and 11 women had no evidence of underlying heart disease. Matched comparisons between patients with lone atrial fibrillation and control participants showed that individuals with atrial fibrillation had similar cardiac risk factors but significantly higher rates of preexisting nonspecific T- or ST-wave abnormalities, intraventricular block, and stroke (6).

In a population-based, longitudinal study of risk factors for coronary artery disease and stroke in 5201 men and women age ≥65 years, 4.8% of women and 6.2% of men had atrial fibrillation at baseline. Prevalence was 9.1% in patients with clinical cardiovascular disease, 4.6% in patients with evidence of subclinical but no clinical cardiovascular disease, and only 1.6% in patients with neither clinical nor subclinical cardiovascular disease. The low prevalence of atrial fibrillation in the absence of clinical and subclinical cardiovascular disease calls into question the clinical usefulness of the designation "lone atrial fibrillation," particularly in the elderly (7).

Acute illnesses that may be associated with new-onset atrial fibrillation include acute myocardial infarction, pulmonary embolism, and thyrotoxicosis. Atrial fibrillation is common after cardiac or thoracic surgery, but may also occur in reaction to another major surgery or severe illness. Atrial fibrillation has an increased incidence in sleep apnea and obesity; however, treatment of these conditions does not seem to affect the subsequent progression of arrhythmia.

6. Brand FN, Abbott RD, Kannel WB, Wolf PA. Characteristics and prognosis of lone atrial fibrillation. 30-year follow-up in the Framingham Study. JAMA. 1985;254:3449-53. [PMID: 4068186]
7. Furberg CD, Psaty BM, Manolio TA, et al. Prevalence of atrial fibrillation in elderly subjects (the Cardiovascular Health Study). Am J Cardiol. 1994;74:236-41. [PMID: 8037127]

Vagal forms of atrial fibrillation usually occur in men age 40 to 50 years who have no structural heart disease. Symptoms often occur at night, at rest, or after alcohol use. Adrenergic forms of atrial fibrillation occur during waking hours preceded by emotional stress or exercise. The mechanism of alcohol-precipitated atrial fibrillation is unclear, but may be related to increases in circulating catecholamines, changes in conduction time and refractory periods in the myocardium, and increases in vagal tone.

Diagnosis... Atrial fibrillation is the most common clinically significant arrhythmia, and prevalence increases with advancing age. Typical symptoms include palpitations, shortness of breath, and exercise intolerance. However, some patients report only general malaise, and many patients are asymptomatic. Electrocardiogram recordings during episodes are the only way to confirm the diagnosis. If the diagnosis is suspected and ECG is normal, longer-term monitoring with a Holter monitor or loop recorder can be helpful. Initial assessment should include laboratory tests (electrolytes, thyroid-stimulating hormone, and renal and hepatic function) to rule out underlying disorders or contraindications to therapies, and echocardiogram to look for structural heart disease.

CLINICAL BOTTOM LINE

Treatment

What are the complications of atrial fibrillation and how can therapy decrease the risk for these events?

There are 3 reasons to prescribe therapy for atrial fibrillation: to reduce symptoms, to prevent thromboembolism, and to reduce the risk for tachycardia-related myocardiopathy.

Although atrial fibrillation is not always symptomatic, when present it can be disabling for some patients. Symptoms are usually caused by inappropriately rapid ventricular rates or the irregularity of the ventricular response during atrial fibrillation (8). Rhythm control (restoring and maintaining sinus rhythm) and rate control (controlling heart rate response without attempts to restore and maintain sinus rhythm) can both help to reduce symptoms.

The average annual risk for arterial thromboembolism is 5% in patients with nonvalvular atrial fibrillation, and the risk for stroke is higher in patients older than age 75 (9). Both established risk factors for thromboembolism and specific features of atrial fibrillation modulate stroke risk (6). Left-atrial thrombi cause 75% of strokes in patients with atrial fibrillation (10). Antithrombotic therapy reduces stroke risk.

The other pathologic consequence of atrial fibrillation is tachycardia-related cardiomyopathy (11). Drug therapy to control rate or rhythm can reduce the risk for this complication.

What are the relative benefits of rate control versus rhythm control in patients with atrial fibrillation?

High-quality clinical trials suggest that rhythm control does not improve mortality, stroke rates, hospitalization rates, or quality of life compared with rate control for most patients with atrial fibrillation (12–14). Rate control is easier to accomplish and prevents exposure to potentially harmful antiarrhythmic agents. On the other hand, rhythm control may be useful in selected patients with severe atrial

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9. Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation. Analysis of pooled data from five randomized controlled trials. Arch Intern Med. 1994;154:1449-57. [PMID: 8018000]
10. Hart RG, Sherman DG, Easton JD, Cairns JA. Prevention of stroke in patients with nonvalvular atrial fibrillation. Neurology. 1998;51:674-81. [PMID: 9748009]
11. Redfield MM, Kay GN, Jenkins LS, et al. Tachycardia-related cardiomyopathy: a common cause of ventricular dysfunction in patients with atrial fibrillation referred for atrioventricular ablation. Mayo Clin Proc. 2000;75:790-5. [PMID: 10943231]
12. Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) Investigators. A comparison of rate control and rhythm control in patients with atrial fibrillation. N Engl J Med. 2002;347:1825-33. [PMID: 12466506]

fibrillation symptoms (before or after failure of rate control) or in younger patients without structural heart disease.

The AFFIRM (Atrial Fibrillation Follow-up Investigation of Rhythm Management) trial included 4060 patients with atrial fibrillation who had at least 1 risk factor for stroke. The mean age was 69 years, and structural heart disease, aside from hypertension, was unusual. All-cause mortality at 5 years was 25.9% in the rate-control group and 26.7% in the rhythm-control group (P = 0.080). Important observations from the trial are that patients with apparently successful rhythm control still need to continue anticoagulation because of persistent stroke risk, and that patients who were able to maintain sinus rhythm had a survival advantage that was almost balanced by the disadvantage imposed by antiarrhythmic drug therapy (15).

A more recent trial extended these observations to outpatients with severe heart failure by randomly assigning 1376 patients with atrial fibrillation, left ventricular ejection fraction of ≤35%, and heart failure symptoms to rate control versus rhythm control. Over 37 months, 27% of the rhythm-control group and 25% of the rate-control group died from cardiovascular causes (P = 0.6). There was no improvement in all-cause mortality, stroke, heart failure, or need for hospitalization in the rhythm-control group (16).

What drugs should clinicians consider for rate control in patients with rapid atrial fibrillation?

Clinicians should consider drug therapy to control ventricular rate in all patients with atrial fibrillation. Criteria for rate control vary with patient age, but clinicians should generally target therapy to achieve target heart rates of 60 to 80 beats per minute at rest and between 90 to 115 beats per minute during moderate exercise (17). Agents that affect atrioventricular nodal conduction and are recommended as first-line therapy in this setting include β-blockers and nondihydropyridine calcium-channel antagonists (Table 1). Amiodarone and digoxin also block the atrioventricular node, but are not recommended as first-line monotherapy for rate control (17). Digitalis is not helpful in reducing heart rate with exercise. Additionally, monotherapy with digitalis is unlikely to control rate in patients with heart failure and high sympathetic activity.

Amiodarone is occasionally used to reduce ventricular response during continued atrial fibrillation if other agents have failed. This practice is more difficult to justify because of toxicities associated with amiodarone and the trial evidence that rhythm control is no better than rate control.

When should clinicians consider antiarrhythmic drugs in the treatment of atrial fibrillation?

Rhythm-control therapy is no longer the preferred strategy in most patients with atrial fibrillation. However, the major trials comparing rate control with rhythm control did not include younger patients or patients with highly symptomatic atrial fibrillation. Consequently, it is reasonable to consider rhythm control in these patient subgroups, either primarily or when symptoms persist despite rate control. Rhythm control is often favored for the first episode of symptomatic atrial fibrillation, particularly in young patients because many maintain sinus rhythm for a period after pharmacologic cardioversion without continued antiarrhythmic drug treatment. Antiarrhythmic drugs have been shown to have modest effects compared with placebo in prolonging time to recurrent atrial fibrillation (Table 1), but rather than the complete absence of atrial fibrillation episodes, antiarrhythmic drug therapy is generally judged to be effective if it reduces episodes and symptoms.

The Canadian Trial of Atrial Fibrillation randomly assigned 403 patients to amiodarone, sotalol, or propafenone and found that after mean follow-up of 16 months, recurrence of atrial fibrillation was 35% during amiodarone and 63% during sotalol or propafenone therapy (18).

13. STAF Investigators. Randomized trial of rate-control versus rhythm-control in persistent atrial fibrillation: the Strategies of Treatment of Atrial Fibrillation (STAF) study. J Am Coll Cardiol. 2003;41:1690-6. [PMID: 12767648]
14. Hohnloser SH, Kuck KH, Lilienthal J. Rhythm or rate control in atrial fibrillation—Pharmacological Intervention in Atrial Fibrillation (PIAF): a randomised trial. Lancet. 2000;356:1789-94. [PMID: 11117910]
15. AFFIRM Investigators. Relationships between sinus rhythm, treatment, and survival in the Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) Study. Circulation. 2004;109:1509-13. [PMID: 15007003]
16. Atrial Fibrillation and Congestive Heart Failure Investigators. Rhythm control versus rate control for atrial fibrillation and heart failure. N Engl J Med. 2008;358:2667-77. [PMID: 18565859]
17. American College of Cardiology/American Heart Association Task Force on Practice Guidelines. ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation. Circulation. 2006; 114:e257-354. [PMID: 16908781]
18. Roy D, Talajic M, Dorian P, et al. Amiodarone to prevent recurrence of atrial fibrillation. Canadian Trial of Atrial Fibrillation Investigators. N Engl J Med. 2000;342:913-20. [PMID: 10738049]

Table 1. Drug Therapy for Rate and Rhythm Control in Atrial Fibrillation

Agent	*Mechanism of Action*	*Dosage*	*Benefits*	*Side Effects*	*Notes*
Rate-Controlling Agents					
ß-Blockers					
Metoprolol	Selective $ß_1$-adrenergic–receptor blocking agent	5 mg IV every 5 min, up to 15 mg 50–100 mg PO twice daily	Convenient IV administration in NPO patients, rapid onset of action, dependable AV nodal blockade	Bradycardia, hypotension, heart block, bronchospasm (less frequently than nonselective ß-blockers), worsening of CHF	
Propranolol	Nonselective ß-adrenergic–receptor blocking agent	1–8 mg IV (1 mg every 2 min). 10–120 mg PO 3 times daily; long-acting preparation: 80–320 mg PO once daily	Inexpensive, commonly available	Bradycardia, hypotension, heart block, bronchospasm, worsening of CHF	
Esmolol	Short-acting IV $ß_1$ selective adrenergic receptor-blocking agent	0.05–0.2 mg/kg per min IV	Short-acting, titratable on or off with very rapid half-life	Bradycardia, hypotension, heart block, bronchospasm (less frequent)	Occasionally inconsistent effect in high-catecholamine states
Pindolol	Nonselective ß-adrenergic–receptor blocking agent with intrinsic sympathomimetic activity	2.5–20 mg PO 2 to 3 times daily	Less bradycardia, less bronchospasm	Bradycardia, hypotension, heart block	Less propensity for heart block than other ß-blockers
Atenolol	Selective $ß_1$-adrenergic–receptor blocking agent	5 mg IV over 5 min, repeat in 10 min. 25–100 mg PO once daily	Does not cross blood–brain barrier, fewer CNS side effects	Bradycardia, hypotension, heart block	
Nadolol	Nonselective ß-adrenergic–receptor blocking agent	20–120 mg once daily	Lower incidence of crossing of blood–brain barrier, fewer CNS side effects	Bradycardia, hypotension, heart block	Oral form only
Calcium-channel blockers					
Verapamil	Calcium-channel blocking agent	5–20 mg in 5-mg increments IV every 30 min, or 0.005 mg/kg per min infusion. 120–360 mg PO daily, in divided doses or in the slow-release form	Consistent AV nodal blockade	Hypotension, heart block, direct myocardial depression	Do not use in the Wolff–Parkinson–White syndrome
Diltiazem	Calcium-channel blocking agent	0.25–0.35 mg/kg IV followed by 5–15 mg/h. 120–360 mg PO daily as slow release	Consistent AV nodal blockade	Hypotension, heart block, less myocardial depression	Do not use in the Wolff–Parkinson–White syndrome
Cardiac glycoside					
Digoxin	Na+-K+ pump inhibitor, increases intracellular calcium	0.75–1.5 mg PO or IV in 3–4 divided doses over 12–24 h. Maintenance dose: 0.125 mg PO or IV to 0.5 mg daily	Particularly useful for rate control in CHF.	Heart block, digoxin-associated arrhythmias; dosage adjustment required in renal impairment	First-line therapy only in patients with decreased left-ventricular systolic function. Not useful for rate control with exercise. Not useful for conversion of AF or aflutter to NSR.
Antiarrhythmic agents					
Class Ia					
Procainamide	Prolongs conduction and slows repolarization by blocking inward Na+ flux	1–2 g q 12 h (shorter-acting oral preparations are no longer available)	Convenient IV dosing available with maintenance infusion, and conversion to PO tablets, very effective at converting AF to NSR	Not recommended because of frequent side effects, including hypotension, nausea, vomiting, lupus-like syndrome, QT prolongation, and arrhythmia	Need to follow drug levels and QT interval for toxicity, adjust dose in patients with renal insufficiency, Not for use in patients with severe LV dysfunction.
Quinidine gluconate	Prolongs conduction and slows repolarization. Blocks fast inward Na+ channel.	324–648 mg PO every 8–12 h	Relatively effective in converting AF to NSR but may take several days to achieve NSR because of PO dosing	Proarrhythmia, nausea, vomiting, diarrhea, QT prolongation	Not recommended because of frequent side effects. Follow drug levels and QT interval for toxicity. Adjust dose in patients with renal insufficiency. Oral agent only

Table 1. Drug Therapy for Rate and Rhythm Control in Atrial Fibrillation (continued)

Agent	*Mechanism of Action*	*Dosage*	*Benefits*	*Side Effects*	*Notes*
Disopyramide	Similar electrophysiologic properties to procainamide and quinidine	150 mg PO every 6–8 h, or 150–300 mg twice a day	Can be useful in patients with hypertension and normal LV function	QT prolongation (not PR or QRS), torsades de pointes, heart block	Rarely used in current era of antiarrhythmic therapy. Oral agent only, negative inotropic properties.
Class Ic					
Flecainide	Blocks Na+ channels (and fast Na+ current)	2 mg/kg, IV*. 50–150 mg PO every 12 h. Also, single loading doses of 300 mg are efficacious in conversion of recent onset AF.	Efficacy in paroxysmal AF with structurally normal hearts	Aflutter or atrial tachycardia with rapid ventricular response but not with acute single loading doses. VT and VF in diseased hearts	Not for use in patients with structurally abnormal hearts
Propafenone	Blocks myocardial Na+ channels	2 mg/kg, IV*. 150–300 mg PO every 8 h. Also, single loading doses of 600 mg are efficacious in conversion of recent onset AF.	Efficacy in paroxysmal and sustained AF	Aflutter or atrial tachycardia with rapid ventricular response, but not with acute single loading doses	Antiarrhythmic and weak calcium channel and ß-blocking properties. Not for use with structural heart disease.
C. Class III					
Ibutilide	Prolongs action potential duration (and atrial and ventricular refractoriness) by blocking rapid component of delayed rectifier potassium current	1 mg IV over 10 min. May be repeated once if necessary.	Efficacy in acute and rapid conversion of AF to NSR	Polymorphic VT (torsades de pointes) occurred in 8.3% of patients in a clinical trial (most with LV dysfunction), QT prolongation	In some centers, only used in the electrophysiology laboratory. May also be used to facilitate unsuccessful direct-current cardioversion.
Amiodarone	Blocks Na+ channels (affinity for inactivated channels). Noncompetitive ß- and ß-receptor inhibitor.	5–7 mg/kg IV up to 1500 mg per 24 h. 400–800 mg PO daily, for 3–4 wk, followed by 100–400 mg PO daily	Safest agent for use in patients with structural heart disease, good efficacy in maintaining NSR chronically	Bradycardia, QT prolongation, hyperthyroidism, lung toxicity, argyria (blue discoloration of skin) with chronic use	Can be used in the Wolff–Parkinson–White syndrome.
Sotalol	Nonselective $ß_1$- and $ß_2$-blocking agent, prolongs action potential duration	80–240 mg PO every 12 h	Similar efficacy to quinidine, but fewer adverse effects. Better rate control because of ß-blocking properties.	Fatigue, depression, bradycardia, torsades de pointes, CHF	ß-blocking properties, but some positive inotropic activity. Lethal arrhythmias possible. Adjust dose in patients with renal insufficiency. Initiate on telemetry.
Dofetilide	Blocks rapid component of the delayed rectifier potassium current (I_{Kr}), prolonging refractoriness without slowing conduction	500 µg twice daily	More effective than quinidine in conversion to and maintenance of NSR.	QT prolongation, torsades de pointes (2%–4% risk).	Must be strictly dosed according to renal function, body size, and age. Contra-indicated in patients with creatinine clearance <20 mL/min. Initiate on telemetry.

AF = atrial fibrillation; AV = atrioventricular; CHF = congestive heart failure; CNS = central nervous system; IV = intraventricular; LV = left ventricular; NPO = nil per os; NSR = normal sinus rhythm; PO = orally; VF = ventricular fibrillation; VT = ventricular tachycardia.

Antiarrhythmic drugs other than amiodarone are generally have equal efficacy, so susceptibility to side effects should guide choice from among them (Table 1). Drugs that block cardiac sodium channels (class I effect), such as flecainide and propafenone, are useful in patients without coronary heart disease or advanced left-ventricular dysfunction. Trial data do not support their use in patients with heart disease because of an increase in mortality (19). In patients without heart disease, the side effects are due to unwanted sodium-channel blockade in other organ systems, such as the gastrointestinal tract (resulting in anorexia or esophageal reflux) and the central nervous system. Other class I drugs, such as quinidine and procainamide, are no longer used because of their frequent noncardiac side effects.

19. The Cardiac Arrhythmia Suppression Trial (CAST) Investigators. Preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. N Engl J Med. 1989;321:406-12. [PMID: 2473403]

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26. The Boston Area Anticoagulation Trial for Atrial Fibrillation Investigators. The effect of low-dose warfarin on the risk of stroke in patients with nonrheumatic atrial fibrillation. N Engl J Med. 1990;323:1505-11. [PMID: 2233931]
27. Stroke Prevention in Atrial Fibrillation Investigators. Lessons from the Stroke Prevention in Atrial Fibrillation trials. Ann Intern Med. 2003;138:831-8. [PMID: 12755555]

Drugs that block potassium channels (class III effects), such as sotalol and dofetilide, have a potential to prolong the QT interval and cause torsades de pointes.

Amiodarone can be used in patients with advanced structural heart disease. However, amiodarone can cause permanent end organ toxicity (liver, lungs) that is dose- and duration-dependent. Other side effects include thyroid dysfunction (hypothyroidism, hyperthyroidism), sun sensitivity, and ocular symptoms.

Some nonantiarrhythmic drugs, such as angiotensin-converting enzyme inhibitors and statins, have been demonstrated (albeit primarily in patients with heart failure) to reduce the incidence of atrial fibrillation, presumably because of antifibrotic effects (20).

Before pharmacologic cardioversion of atrial fibrillation present for more than 48 hours, patients should first receive adequate therapy for rate control and anticoagulation. In addition, serum potassium level should be greater than 4.0 mmol/L, serum magnesium level should be greater than 1.0 mmol/L, and ionized calcium levels should be greater than 0.5 mmol/L [2.0 mg/dL]. In most cases, pharmacologic cardioversion should be performed in a monitored hospital setting to permit adequate assessment of the degree of rate control, bradycardia, proarrhythmic affects of antiarrhythmic agents, and other adverse effects (21).

When is anticoagulation indicated for patients with atrial fibrillation?

A meta-analysis (9) of 5 high-quality randomized trials (22–26) supports the use of antithrombotic therapy for appropriate patients with nonvalvular atrial fibrillation.

Anticoagulation is indicated for patients with atrial fibrillation when the risk for thromboembolism exceeds the risk for anticoagulation-associated bleeding (17). About one third of patients with atrial fibrillation have a low risk for thromboembolism, one third have a high risk, and one third have a moderate risk (27).

In addition to established risk factors for thromboembolism, specific features of atrial fibrillation and underlying disease also modulate thromboembolism risk. Patients with reversible causes of atrial fibrillation and those with structurally normal hearts are less likely to have persistent or recurrent episodes and may be at a lower risk for thromboembolism than patients without these features. However, clinicians should keep in mind that the rate of stroke in patients with nonvalvular atrial fibrillation and at least 1 risk factor exceeds that of hemorrhage from chronic anticoagulation.

Risk factors for thromboembolism have been identified in the Stroke Prevention in Atrial Fibrillation trial (9). Patients younger than age 65 years with nonvalvular atrial fibrillation and no risk factors have an annual stroke risk of about 0.5%, whereas patients older than 65 years with no risk factors have a risk of about 1%. This latter figure approximates the risk for major bleeding while on warfarin with an international normalized ratio (INR) of 2.0 to 3.0 (28, 29). The mean annual rate of major bleeding in the major anticoagulation trials on warfarin therapy was 1.2%.

Because of the delicate balance between risk and benefit, indices have been developed to assess which patients with atrial fibrillation are at sufficient risk for stroke to warrant anticoagulation therapy. The most popular of these is the $CHADS_2$ score (30, 31) (Table 2). Table 3 presents recommendations for therapy based on this score.

A 2007 meta-analysis of 29 trials including 28 044 participants characterized the efficacy and safety of antithrombotic agents for stroke prevention in patients who have atrial fibrillation. Compared with control participants, adjusted-dose warfarin (6 trials, 2900 participants) and antiplatelet agents (8 trials, 4876 participants) reduced stroke by 64% (95% CI, 49% to 74%) and 22% (CI, 6% to 35%), respectively. Adjusted-dose warfarin was substantially more efficacious than antiplatelet therapy (relative risk reduction, 39% [CI, 22% to 52%]) (12 trials, 12 963 participants). Absolute increases in major extracranial hemorrhage were small (0.3% per year) (32).

Some data indicate that risk factor–adjusted incidence of ischemic stroke and major bleeding is currently considerably less than that reflected in the previously described trials. The consensus is that this is related to improved therapy for hypertension (33). Less-encouraging recent data have indicated that the rate of major bleeding is high in the elderly (34).

What regimens should clinicians use to anticoagulate patients with atrial fibrillation?

Adjusted-dose warfarin to an INR of 2.0 to 3.0 is the first choice for anticoagulation of patients with atrial fibrillation. Certain patients with prosthetic valves in addition to atrial fibrillation should have warfarin titrated to an INR of 2.5 to 3.5. In patients without additional stroke risk factors (previous stroke or transient ischemic attack, age >75 years, hypertension, diabetes, heart failure) or who have contraindications to full anticoagulation, aspirin 325 mg/d can be used as alternative thromboembolism prophylaxis.

The effect of aspirin is controversial, but is probably present and less dramatic than that of warfarin (35). A recent trial demonstrated that aspirin plus clopidogrel was

Table 2. Stroke Risk in Patients with Nonvalvular Atrial Fibrillation Not Treated with Anticoagulation According to $CHADS_2$ Index*

$CHADS_2$ Risk Criteria	*Score*
Past stroke or TIA	2
Age >75 y	1
Hypertension	1
Diabetes mellitus	1
Heart failure	1

Patients (n= 1733)	*Adjusted Stroke Rate (%/y)† (95% CI)*	*$CHADS_2$ Score*
120	1.9 (1.2 to 2.0)	0
463	2.8 (2.0 to 3.8)	1
523	4.0 (3.1 to 5.1)	2
337	5.9 (4.6 to 7.3)	3
220	8.5 (6.3 to 11.1)	4
65	12.5 (8.2 to 17.5)	5
5	18.2 (10.5 to 27.4)	6

$CHADS_2$ = Cardiac Failure, Hypertension, Age, Diabetes, and Stroke (Doubled); TIA = transient ischemic attack.
** Reproduced from reference 5 with permission from the American Heart Association.*
† The adjusted stroke rate was derived from multivariate analysis assuming no aspirin usage. Data from from references 30, 31.

Table 3. Antithrombotic Therapy for Patients with Atrial Fibrillation*

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

Less-Validated or Weaker Risk Factors	*Moderate Risk Factors*	*High Risk Factors*
Female sex	Age ≥75 y	Previous stroke, TIA, or embolism
Age 65–74 y	Hypertension	Mitral stenosis
Coronary artery disease	Heart failure	Prosthetic heart valve†
Thyrotoxicosis	LV ejection fraction 35% or less, diabetes mellitus	

INR = international normalized ratio; LV = left ventricular; TIA = transient ischemic attack.
** Reproduced from reference 5 with permission from the American Heart Association.*
† If mechanical valve, target INR >2.5.

28. Jung F, DiMarco JP. Treatment strategies for atrial fibrillation. Am J Med. 1998;104:272-86. [PMID: 9552091]
29. Zabalgoitia M, Halperin JL, Pearce LA, et al. Transesophageal echocardiographic correlates of clinical risk of thromboembolism in nonvalvular atrial fibrillation. Stroke Prevention in Atrial Fibrillation III Investigators. J Am Coll Cardiol. 1998;31:1622-6. [PMID: 9626843]
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31. van Walraven C, Hart RG, Wells GA, et al. A clinical prediction rule to identify patients with atrial fibrillation and a low risk for stroke while taking aspirin. Arch Intern Med. 2003;163:936-43. [PMID: 12719203]
32. Hart RG, Pearce LA, Aguilar MI. Meta-analysis: antithrombotic therapy to prevent stroke in patients who have nonvalvular atrial fibrillation. Ann Intern Med. 2007;146:857-67. [PMID: 17577005]

clearly inferior to adjusted-dose warfarin (36).

Institution of warfarin without loading doses or concurrent heparin is sufficient in lower-risk patients, whereas patients at high risk for thromboembolism should be hospitalized for immediate anticoagulation with unfractionated heparin before target levels of oral anticoagulation are reached. There are limited data on the use of low-molecular-weight heparin in this setting.

Patients with atrial fibrillation lasting more than 48 hours or those with intracardiac thrombus should receive anticoagulation with warfarin before cardioversion and for at least 4 weeks afterward.

Clinicians should consider chronic anticoagulation in patients who are at high risk for recurrent atrial fibrillation, have asymptomatic atrial fibrillation, have evidence of intracardiac thrombus, or have known risk factors for thromboembolism (age >65 years, recent heart failure, left-ventricular dysfunction on echocardiogram, past thromboembolism, diabetes mellitus, hypertension, or left atrial enlargement).

Warfarin has a narrow therapeutic window, and its metabolism is affected by many drug and dietary interactions, requiring frequent INR monitoring and dosage adjustment. These limitations of warfarin have prompted a search for alternative anticoagulants. A trial showed ximelagatran not to be a suitable alternative to warfarin (37). Investigational studies to develop other oral anticoagulants (direct thrombin inhibitors, factor Xa inhibitors) are in progress, but there are no current alternatives to warfarin.

When should clinicians consider immediate cardioversion in patients with atrial fibrillation?

Prompt cardioversion should be considered for new-onset atrial fibrillation when it is clear that the duration of the arrhythmia is less than 48 hours (as might be the case when atrial fibrillation onset occurs in a hospitalized patient on cardiac monitoring) and the patient is not at high risk for stroke. Immediate cardioversion, if successful, could obviate the need for anticoagulation.

Most patients with atrial fibrillation do not require immediate pharmacologic or electrical cardioversion, but it may be appropriate in selected patients with decompensated heart failure, severe angina or acute infarction, hypotension, or high risk for acute stroke. Patients with atrial fibrillation and the Wolff–Parkinson–White syndrome can have extremely rapid atrioventricular conduction during atrial fibrillation mediated by the accessory pathway, which is a potentially life-threatening condition that requires urgent cardioversion.

When should clinicians consider atrioventricular nodal catheter ablation, device therapy, or surgical techniques in patients with atrial fibrillation?

Nonpharmacologic therapy for atrial fibrillation should be considered after failure or intolerance to rate-control or rhythm-control therapy. Nonpharmacologic therapeutic options include catheter or surgical atrioventricular nodal ablation and pacing.

Atrioventricular nodal catheter ablation is used in situations where pharmacologic rate control cannot be achieved, usually because of intolerance to medications. This occurs most frequently in patients with advanced heart failure or obstructive pulmonary disease (limiting β-blocker usage) or elderly patients. Atrioventricular nodal ablation is highly effective (38) but requires pacemaker insertion, which introduces the risk for producing progressive left ventricular dysfunction secondary to

33. Hart RG, Tonarelli SB, Pearce LA. Avoiding central nervous system bleeding during antithrombotic therapy: recent data and ideas. Stroke. 2005;36:1588-93. [PMID: 15947271]
34. Hylek EM, Evans-Molina C, Shea C, et al. Major hemorrhage and tolerability of warfarin in the first year of therapy among elderly patients with atrial fibrillation. Circulation. 2007;115:2689-96. [PMID: 17515465]
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pacing. Catheter ablation of atrial fibrillation has been shown to be effective in preventing recurrent symptomatic atrial fibrillation in highly selected patients (39). Recent guideline statements have acknowledged that it may be reasonable to provide this therapy for highly symptomatic patients with paroxysmal atrial fibrillation in whom an attempt at antiarrhythmic drug therapy has failed. This relatively aggressive approach may prevent progressive atrial fibrillation–related morbidity (residual risk for stroke, risk for medication side effects), but the impact of this therapeutic strategy on mortality has not been demonstrated. Innovative minimally invasive surgical ablation of atrial fibrillation is also available at highly specialized centers.

Pacing therapy without atrioventricular nodal ablation has very little effect on atrial fibrillation burden, but may be helpful in patients with paroxysmal atrial fibrillation who have symptomatic bradycardia (often caused by side effects of atrial fibrillation pharmacologic therapy).

How should clinicians monitor patients with atrial fibrillation?

Although there are few studies to inform the appropriate frequency of follow-up for patients with atrial fibrillation, clinical consensus is that patients with atrial fibrillation should have regular follow-up to assess symptoms and clinical effectiveness of therapy. For many patients, anticoagulation monitoring drives the frequency of follow-up. Clinicians should assess rate control by asking about such symptoms as palpitations, easy fatigability, and dyspnea on exertion. Examination should assess resting and exercise heart rates for targets of 60 to 80 beats per minute and 90 to 115 beats per minute, respectively. Patients in whom rhythm control is chosen should be monitored for symptoms suggestive of atrial fibrillation. Sporadic asymptomatic episodes of atrial fibrillation on therapy are not important and do not need to be monitored for. However, patients who do not improve on rhythm-control drugs should be changed to less-toxic rate-control agents. Routine blood tests to evaluate for side effects of antiarrhythmic therapy are not essential, except in the case of amiodarone, which requires liver and thyroid function studies every 6 months and chest radiography every year.

Which patients with newly diagnosed atrial fibrillation should clinicians consider hospitalizing?

Although atrial fibrillation is usually managed in outpatient settings, clinicians should consider hospitalizing patients with atrial fibrillation when management requires close monitoring for safety (see Box).

Situations in Which Patients with Atrial Fibrillation May Require Hospitalization

- Uncertain or unstable underlying arrhythmia
- Acute myocardial infarction, altered mental status, decompensated heart failure, or hypotension
- Intolerable symptoms despite hemodynamic stability
- Elective cardioversion (if monitored outpatient setting is not available)
- Acute anticoagulation if very-high risk for stroke
- Telemetry monitoring during initiation of certain drugs
- Procedures such as cardiac catheterization, electrophysiologic studies, pacemakers, implantable defibrillators, or catheter or surgical ablation

Treatment... Treatment goals for atrial fibrillation include preventing stroke, reducing symptoms, and preventing tachycardia-related cardiomyopathy. The use of anticoagulants (aspirin or warfarin) is guided by risk classifications, such as the $CHADS_2$ score. Several randomized trials have demonstrated no general advantage to rhythm control over rate control. Rate control with calcium-channel antagonists or β-blockers to keep heart rate at 60 to 80 beats per minute at rest and 90 to 115 beats per minute during exercise should be the first-line therapy. Rhythm control, which has greater adverse effects than rate control, may be reasonable in individual patients who do not respond to rate control. Atrial and atrioventricular nodal ablation therapy may be appropriate for selected patients with highly symptomatic atrial fibrillation despite pharmacologic therapy.

CLINICAL BOTTOM LINE

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Practice Improvement

Do U.S. stakeholders consider management of patients with atrial fibrillation when evaluating the quality of care physicians deliver?

The Centers for Medicare & Medicaid Services (CMS) has issued specifications for 74 measures that make up the 2008 Physician Quality Reporting Initiative (PQRI). Of these 74 measures, none directly measures the quality of atrial fibrillation therapy. However, one of the stroke measures does relate to atrial fibrillation. This measure examines the percentage of patients age 18 years or older with a diagnosis of ischemic stroke or transient ischemic attack and documented permanent, persistent, or paroxysmal atrial fibrillation who were prescribed an anticoagulant at discharge.

What do professional organizations recommend with regard to the management of patients with atrial fibrillation?

The material presented in this review is consistent with the 2006 guidelines developed by a consensus panel of the American Heart Association, American College of Cardiology, and the European Society of Cardiology (17). In 2003, the American College of Physicians and the American Academy of Family Physicians released a guideline on atrial fibrillation management (40). Both guideline statements stress anticoagulation in appropriately selected patients and the nonsuperiority of the rhythm-control strategy. The AHA/ACC/ESC guideline emphasizes the cardiology perspective, whereas the ACP/AAFP focuses on the primary care perspective.

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in the clinic

Tool Kit

Atrial Fibrillation

PIER Modules

www.pier.acponline.org

Access PIER module on atrial fibrillation for updated, evidence-based information designed for rapid access at the point of care.

Quality Measures

pier.acponline.org/qualitym/prv.html

Access the PIER Quality Measure Tool, which links newly developed quality measures issued by the Ambulatory Quality Alliance and the Physician Quality Improvement QA Alliance and CMS's Physician Quality Reporting Initiative program to administrative criteria for each measure and provides clinical guidance to help implement the measures and improve quality of care.

Patient Information

www.annals.intheclinic/tools

Download copies of the Patient Information sheet that appears on the following page for duplication and distribution to your patients.

Anticoagulation Flow Sheet

www.acponline.org/running_practice/quality_improvement/projects/cfpi/doc_anticoag.pdf

Download a copy of a flow sheet to help manage patients on warfarin.

Guidelines

www.americanheart.org/downloadable/heart/222_ja20017993p_1.pdf

Access the American Heart Association, American College of Cardiology, and European Society of Cardiology joint 2006 guidelines for the management of patients with atrial fibrillation.

www.annals.org/cgi/reprint/139/12/1009.pdf

Access the American College of Physicians/American Academy of Family Physicians 2003 guidelines for the management of newly detected atrial fibrillation.

WHAT YOU SHOULD KNOW ABOUT ATRIAL FIBRILLATION

In the Clinic
Annals of Internal Medicine
annals.org

Atrial fibrillation is an irregular and sometimes very fast heart beat. Atrial fibrillation can come and go or be constant. It is more common in older people than in younger people and in people with heart conditions.

Atrial fibrillation can lead to 3 bad health outcomes:

- Symptoms that can make a person unable to do their usual activities.
- Over the long term, a very fast heart beat can damage heart muscle.
- Atrial fibrillation can cause stroke when blood clots form in the heart and travel to the brain.

How would I know if I have atrial fibrillation?

- Many people with atrial fibrillation have no symptoms and don't know that they have it.
- When people have symptoms, they include palpitations (pounding in the chest), shortness of breath, or tiredness.
- Your doctor may see atrial fibrillation on an electrocardiogram (ECG) if an episode occurs during the test.
- If you have symptoms that could be atrial fibrillation but your ECG is normal, your doctor may send you for a test that records your heartbeat while you go about your usual activities.
- If you have atrial fibrillation, your doctor may do an echocardiogram to look for heart problems. Echocardiograms use sound waves to take pictures of the heart.

What is the treatment?

- Many patients with atrial fibrillation need to be on drugs to prevent stroke. Some people need only aspirin. Others need to take the blood thinner warfarin.
- Treatment also sometimes includes drugs to slow the heart rate down or make it more regular.
- Less often, treatment with catheters, surgery, and pacemakers is needed.
- Atrial fibrillation treatment can have dangerous side effects. It is important to follow instructions and see your doctor regularly.

Web Sites with Good Information

MedlinePlus: www.nlm.nih.gov/medlineplus/tutorials/atrialfibrillation/htm/_no_50_no_0.htm

Heart Rhythm Society: www.hrspatients.org/patients/heart_disorders/atrial_fibrillation/default.asp

American Heart Association: circ.ahajournals.org/cgi/content/full/117/20/e340

Patient Information

CME Questions

MKSAP
MEDICAL KNOWLEDGE Self-Assessment Program

1. A 70-year-old woman with type 2 diabetes mellitus is evaluated for dyspnea and fatigue. She has a history of atrial fibrillation that has resulted in these symptoms in the past. She has had successful cardioversions, most recently about 2 years ago. She has hypertension controlled with medication. She also has mild left-ventricular dysfunction related to coronary artery disease and history of myocardial infarction. Her current medications include atenolol, lisinopril, aspirin, atorvastatin, and insulin.

   Physical examination demonstrates an irregularly irregular rhythm with a heart rate of 78 beats per minute. Blood pressure is 130/80 mm Hg. The cardiovascular and pulmonary examinations are otherwise unremarkable.

   What medication should this patient receive before cardioversion?

   A. Aspirin and dipyridamole
   B. Warfarin
   C. Clopidogrel
   D. No additional medication is needed

2. A 70-year-old woman reports mild effort intolerance over the last several months but has continued her usual activities. She has a history of hypertension that is well controlled with diuretic therapy. Findings on physical examination and electrocardiography are consistent with atrial fibrillation, with a ventricular rate of 100 beats per minute, but were otherwise normal. Echocardiogram shows a left-ventricular ejection fraction of 55%, with left atrial enlargement, mild mitral annular calcification, and mild mitral regurgitation. She has no history of stroke or ulcer disease.

   Which of the following would be the most appropriate therapy for this patient?

   A. Aspirin and a β-blocker
   B. Aspirin and amiodarone
   C. Aspirin and digoxin
   D. Warfarin and a β-blocker
   E. Warfarin and amlodipine

3. A 61-year-old man with a history of myocardial infarction, paroxysmal atrial fibrillation, ischemic cardiomyopathy, and heart failure presents for further management. He underwent percutaneous revascularization 4 years ago. He has not had chest pain and he had undergone an exercise scintigraphic study 2 months ago without stress-induced ischemia. His left-ventricular ejection fraction was estimated to be 35%. He has had NYHA class II symptoms with mild dyspnea on moderate exertion. He had multiple episodes of atrial fibrillation starting 4 years ago. His last recurrence of atrial fibrillation was 4 months ago. At that time he was started on anticoagulation with warfarin and amiodarone. He has been tolerating his medications well. He has had no palpitations in the past 4 months. He is being treated with lisinopril, 20 mg/d, carvedilol, 25 mg/d, digoxin, 0.125 mg/d, atorvastatin, 10 mg/d, amiodarone, 200 mg/d, and warfarin, 5 mg/d. His INR has ranged from 2.1 to 2.7. His electrocardiogram on this visit shows normal sinus rhythm and evidence of a past anterior myocardial infarction. A 24-hour Holter monitor performed last week showed no evidence of atrial fibrillation and occasional premature ventricular contractions.

   Which of the following treatment options would you recommend?

   A. Discontinue warfarin, continue amiodarone
   B. Discontinue amiodarone, start procainamide
   C. Discontinue amiodarone and warfarin
   D. Continue amiodarone and warfarin
   E. Refer patient for atrial fibrillation ablation

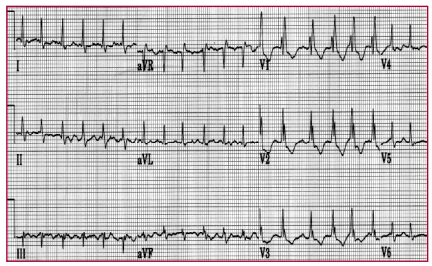
4. Which one of the following statements about atrial fibrillation is correct?

   A. Lone atrial fibrillation is a common cause of atrial fibrillation.
   B. Atrial fibrillation is more common in older women than in older men.
   C. Anticoagulation is not indicated in patients who have nonrheumatic heart disease and atrial fibrillation.
   D. Many patients who have atrial fibrillation do not require antiarrhythmic therapy.
   E. Atrial fibrillation is a serious and common problem in patients who have the Wolff–Parkinson–White syndrome.

5. A 67-year-old woman is admitted to the emergency room because of sudden onset of chest pain and rapid pulse. She has no history of similar occurrences. Physical examination reveals a pale diaphoretic woman in moderate respiratory distress. Her blood pressure is palpable at 75 mm Hg systolic. Lungs show bibasilar crackles. There is no jugular venous distention and heart sounds are distant with a variable S1. A 12-lead electrocardiogram is shown.

   What is the appropriate immediate therapy?

   A. Rapid infusion of 250 mL of normal saline
   B. Diltiazem, 20 mg intravenously, followed by 10 mg/h infusion
   C. Digoxin, 0.50 mg intravenously
   D. Direct-current cardioversion
   E. Procainamide, 500 mg intravenously over 20 min



Questions are largely from the ACP's Medical Knowledge Self-Assessment Program (MKSAP). Go to www.annals.org/intheclinic/ to obtain up to 1.5 CME credits, to view explanations for correct answers, or to purchase the complete MKSAP program.