

# Atrial Fibrillation

**E**vidence for diagnosis and treatment of atrial fibrillation (AF) has expanded substantially since 2017, when In the Clinic last considered this subject. Direct oral anticoagulants have become the predominant therapy for thromboembolic disease, and antidotes for these drugs are now available. Device-based left atrial appendage occlusion is frequently used in patients who cannot tolerate systemic anticoagulation, and growing evidence suggests that early rhythm control improves outcomes. Catheter ablation is now frequently performed to prevent recurrent AF. Managing risk factors for AF, such as hypertension, diabetes, and obesity, remains paramount in prevention of this condition.

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Diagnosis

Treatment

Practice Improvement

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Atrial fibrillation (AF) is the most common clinically significant cardiac arrhythmia. It occurs when a diffuse and chaotic pattern of electrical activity in the atria suppresses or replaces the normal sinus mechanism. AF is a major cause of morbidity, mortality, and health care expenditure. In the United States, 2.3 million persons have AF, and this number is expected to increase to 5.6

million by 2050 (1). AF is associated with a 5-fold increase in risk for stroke and is estimated to cause 15% of all strokes (2). It is also associated with a 2-fold increase in risk for all-cause mortality, independent of comorbid conditions, and a 5-fold increase in risk for heart failure (3). AF has also been associated with cognitive decline, including dementia (4).

## Diagnosis

### Who is at risk for AF?

AF occurs in fewer than 1% of persons aged 60 to 65 years but in 8% to 10% of those older than 80 years. Prevalence is higher in men than in women and seems to be higher in White persons than in Black persons (1). Risk for AF increases with age and the presence and severity of underlying heart disease, particularly heart failure and valve disease. Of the 5.8 million adults with heart failure with reduced or preserved ejection fraction, up to 40% develop AF. Other conditions that are commonly associated with AF include sleep-disordered breathing, obesity, diabetes, hypertension, and heavy alcohol consumption (5).

### What symptoms and signs should cause clinicians to suspect AF?

Some patients with AF have prominent symptoms, including palpitations, shortness of breath, exercise intolerance, chest pain, and malaise (6). However, many persons, particularly older ones, have asymptomatic (silent) AF, including some with severe symptoms during other episodes of AF (7). Silent AF is often recognized during routine interrogation of pacemakers implanted for bradycardia and implantable cardioverter-defibrillators for prevention of sudden cardiac death in patients with no history of AF (8). Also, silent AF is increasingly being recognized by consumer wearables, such as wristwatches and fitness trackers (9). Symptoms result from elevation of the ventricular rate (either at rest or when exacerbated by exercise), and in patients with heart failure, the irregular ventricular rate and loss of

atrial contribution to cardiac output contribute to symptom severity.

Examination findings include a faster-than-expected heart rate, which varies greatly from patient to patient; an "irregularly irregular" time between heart sounds; and peripheral pulses that vary irregularly in both rate and amplitude.

### Is a single electrocardiogram sufficient to diagnose or exclude AF?

**Appendix Figure 1** (available at Annals.org) shows an electrocardiogram (ECG) of a patient with AF and illustrates that a single ECG is sufficient to diagnose AF. However, AF is often paroxysmal, so a normal ECG cannot rule it out. Monitoring for a longer duration is needed when AF is suspected but the initial ECG is normal. In patients with daily symptoms, 24- or 48-hour continuous Holter monitoring is usually sufficient for diagnosis. Newer patch monitors allow up to 30 days of continuous monitoring without attached leads and are a good alternative to traditional Holter monitors (10). The advent of consumer wearables and personal ECGs with 1 or more leads has made it easier to capture AF in patients with vague, infrequent, or no symptoms (9).

Implanted pacemakers and implantable cardioverter-defibrillators (generally with atrial leads) identify and record both symptomatic and asymptomatic AF. Subcutaneous implanted monitors are also increasingly used to identify AF, particularly in patients with cryptogenic stroke in whom identification of AF will result in initiation of anti-coagulation (11). Consumer wearables

can record symptomatic and asymptomatic AF; however, the accuracy of these modalities in diagnosing AF is unclear (9).

### What is the role of history and physical examination in the assessment of patients with AF?

Clinicians should seek historical and physical evidence of hypertension, heart failure, cardiac surgery, murmurs indicative of stenotic or regurgitant valve disease, and other indications of structural heart disease. They also should look for signs and symptoms of noncardiac causes of AF, including pulmonary disease, hyperthyroidism, use of adrenergic drugs (such as those used to treat pulmonary disease) or other stimulants, and use of alcohol. Other risk factors include diabetes, obesity, and sleep-disordered breathing. A family history might identify first-degree relatives with AF, which may have therapeutic implications in the future (12).

### What other electrocardiographic arrhythmias can be confused with AF?

Other arrhythmias that are commonly confused with AF include sinus rhythm with frequent premature atrial contractions, atrial flutter, and atrial tachycardia. The key electrocardiographic findings of AF are the absence of P waves and the presence of an irregular ventricular rhythm without a recurring pattern. When an irregular rhythm is present but the diagnosis of AF is uncertain, clinicians should examine long recordings from multiple leads to look for partially obscured P waves in deformed T waves and ST segments.

Appendix Figure 2 (available at Annals.org) shows an ECG of an irregular

rhythm that might be attributable to AF, but the presence of P waves and other features indicates sinus rhythm with frequent premature atrial contractions. The QRS is wide with the premature beat because of aberrant conduction. Appendix Figure 3 (available at Annals.org) shows an ECG of another irregular rhythm that might be attributable to AF, but the presence of "saw-tooth" P waves and a ventricular response that varies from 2:1 atrioventricular conduction to 4:1 atrioventricular conduction indicates atrial flutter.

### How should clinicians classify AF?

Although classification of AF is a subject of debate, the most accepted convention categorizes AF as paroxysmal, persistent, long-standing persistent, or permanent (13) (see the Box: Classification of AF). In paroxysmal AF, episodes terminate without intervention in less than 7 days (often within 24 hours). Persistent AF lasts longer than 7 days or requires an intervention, such as cardioversion, to restore sinus rhythm. Long-standing persistent AF is continuous AF lasting longer than 12 months. Permanent AF means that the arrhythmia is continuous, and interventions to restore sinus rhythm have failed, have not been attempted, or have been forgone. Patients may change categories over time, so clinicians should classify them according to their current pattern. Patients in all categories should be assessed for the need for anticoagulation independent of the type, frequency, or duration of AF episodes.

### What laboratory studies should clinicians obtain in patients newly diagnosed with AF?

When patients are initially diagnosed with AF, clinicians should measure serum electrolyte and thyroid-stimulating

#### Classification of AF

Paroxysmal: Episodes spontaneously terminate in <7 d

Persistent: Episodes last >7 d or require intervention to restore sinus rhythm

Long-standing persistent: Continuous AF lasting >12 mo

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

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hormone levels to identify possible causes. They should perform blood tests of renal and hepatic function to guide selection of drug therapy, and they should check the complete blood count to look for anemia and should check the stool for occult blood before starting anticoagulation. Transthoracic echocardiography helps assess left atrial size, may reveal underlying structural heart disease that may not otherwise be recognized, and can identify tachycardia-induced cardiomyopathy, which may occur when AF has been present for an extended period. Transesophageal echocardiography is indicated to rule out atrial clot before cardioversion in patients who have received anticoagulation for less than 3 weeks or those who missed doses of their anticoagulant in the 3 weeks before cardioversion. In patients with appropriate clinical indications, additional tests may be warranted for pulmonary embolism, acute myocardial infarction, or acute heart failure.

### What underlying conditions should clinicians look for in patients presenting with AF?

Eighty percent of patients with AF have structural heart disease, particularly left ventricular hypertrophy due to hypertension and also coronary artery disease, valvular heart disease, or cardiomyopathy. Atrial fibrosis occurs frequently with structural heart disease and is considered to be central to the AF's pathogenesis (13). The commonly used term "nonvalvular AF" originally referred to AF in the absence of rheumatic heart disease but has now been generalized to AF in the absence of other forms of significant valve disease.

Some acute illnesses are associated with AF, including acute myocardial infarction, pulmonary embolism, and thyrotoxicosis. AF occurs in approximately 40% of patients after cardiac or thoracic surgery, but it may also occur after other types of major surgery (14) or during a severe illness. Obesity and sleep apnea are also associated with increased incidence of AF.

**Diagnosis...** AF is the most common and clinically significant cardiac arrhythmia, and its incidence increases with advancing age. Typical symptoms include palpitations, shortness of breath, and exercise intolerance. However, some patients report only general malaise, and many are asymptomatic. An ECG during an episode of AF is the main way to confirm the diagnosis. If the diagnosis is suspected and the ECG is normal, longer monitoring with a loop recorder, a patch continuous monitor, or consumer wearables may be helpful. The initial assessment should include laboratory tests for electrolytes, thyroid-stimulating hormone, renal function, and a complete blood count to rule out underlying disorders or contraindications to therapies. An echocardiogram should be done to look for structural heart disease and assess left atrial size.

## CLINICAL BOTTOM LINE

### Treatment

#### What are the complications of AF, and how can therapy decrease risk for them?

There are 3 reasons to treat AF: to improve symptoms, to prevent thromboembolism, and to prevent cardiomyopathy and heart failure.

The symptoms of AF can be disabling. They are usually caused by inappropriately rapid ventricular rates and/or

the irregularity of the ventricular response (15). The loss of atrial contribution to ventricular filling ("atrial kick") is well tolerated by most patients except those with ventricular hypertrophy from long-standing hypertension, aortic stenosis, hypertrophic obstructive cardiomyopathy, and heart failure with reduced or preserved ejection fraction.

Stroke is the most common form of clinically detectable arterial thromboembolism associated with AF. In patients with nonvalvular AF, the average annual risk for arterial thromboembolism, including stroke, is 5%, and this increases to 7% in patients with heart failure. The risk for stroke is particularly high in patients older than 75 years and those with a history of stroke or transient ischemic attack (13). Left atrial thrombi, mostly arising from the left atrial appendage, are believed to cause most strokes in patients with AF (16).

Treating the tachycardia of AF is important because tachycardia can lead to cardiomyopathy (17).

### When should clinicians consider immediate cardioversion?

Prompt cardioversion should be considered for new-onset AF when the patient is hemodynamically unstable, when the patient is experiencing angina or decompensated heart failure, or when the arrhythmia has been present for less than 48 hours. Most patients with AF do not require immediate cardioversion, but it may be appropriate in selected patients. Patients with AF and Wolff-Parkinson-White syndrome can have extremely rapid atrioventricular conduction mediated by the accessory pathway, which can be life-threatening and requires urgent cardioversion.

### Which patients with AF should clinicians consider hospitalizing?

Although AF is usually managed in an outpatient setting, clinicians should consider hospitalization when the patient is acutely ill and/or when management requires close monitoring for safety (see the Box: Situations in

Which Patients With AF May Require Hospitalization).

### Should clinicians attempt rate control or rhythm control?

Older trials have shown that compared with rate control, rhythm control generally does not improve mortality, frequency of stroke or hospitalization, or quality of life (18-20).

*The AFFIRM (Atrial Fibrillation Follow-up Investigation of Rhythm Management) trial included 4060 patients with AF 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 ( $P = 0.080$ ). Patients with apparently successful rhythm control in whom anticoagulation was stopped had increased risk for stroke, and patients who were able to maintain sinus rhythm had a survival advantage that was almost balanced by the risks associated with long-term use of antiarrhythmic medications (18).*

*A subsequent trial extended these observations to patients with severe heart failure by randomly assigning 1376 patients to rate control or rhythm control. Patients had AF, left ventricular ejection fraction of 35% or less, and heart failure symptoms. At 37 months, 25% of patients in the rate control group died of cardiovascular disease compared with 27% in the rhythm control group ( $P = 0.6$ ). There was no improvement in all-cause mortality, stroke, heart failure, or need for hospitalization in the rhythm control group (20).*

#### Situations in Which Patients With AF May Require Hospitalization

- Uncertain or unstable underlying arrhythmia
- Acute myocardial infarction, altered mental status, decompensated heart failure, or hypotension
- Intolerable symptoms despite hemodynamic stability
- After cardioversion (if the patient has a cardioversion-related complication)
- Need for telemetry monitoring during initiation of certain drugs
- Procedures, such as cardiac catheterization, electrophysiologic studies, and catheter or surgical ablation and placement of pacemakers or implantable cardioverter-defibrillators

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Reasons that rhythm control with medications may not be better than rate control include the fact that antiarrhythmic medications are not effective at maintaining normal rhythm, and these medications are proarrhythmic, leading to a new arrhythmia despite therapeutic (nontoxic) drug levels (21). However, newer studies have shown that early rhythm control improves patient outcomes.

*EAST-AFNET 4 (Early Treatment of Atrial Fibrillation for Stroke Prevention Trial) randomly assigned 2789 patients with early AF (diagnosed  $\leq$  1 year before enrollment) and cardiovascular conditions to either early rhythm control or usual care (22). Early rhythm control included treatment with an antiarrhythmic medication or catheter ablation, and usual care limited the use of rhythm control to controlling symptoms believed to be due to AF. Early rhythm control resulted in a significant reduction in the primary composite outcome of death due to cardiovascular causes, stroke, or hospitalization for worsening heart failure or acute coronary event (249 patients in the early rhythm control group vs. 316 in the usual care group; hazard ratio [HR], 0.79 [95% CI, 0.66 to 0.94]; P = 0.005). There were no safety concerns related to early rhythm control (21).*

*Similarly, the EARLY-AF (Early Aggressive Invasive Intervention for Atrial Fibrillation) trial assigned 303 patients with symptomatic, paroxysmal, untreated AF to cryoablation or an antiarrhythmic medication for initial rhythm control (23). A cardiac monitor was implanted in all patients to detect atrial tachyarrhythmia during 12 months of follow-up. The primary outcome was the first documented recurrence of any atrial tachyarrhythmia between 91 and 365 days after receipt of the study intervention. This occurred in 66 of 154 patients (42.9%) in the cryoablation group and 101 of 149 (67.8%) in the antiarrhythmic medication group (HR, 0.48 [CI, 0.35 to 0.66]; P < 0.001). Serious complications occurred in 5 patients (3.2%) in the ablation group and*

*6 (4.0%) in the antiarrhythmic medication group (23).*

#### **Selecting patients for rate versus rhythm control**

Rhythm control is becoming the preferred strategy for managing many patients with AF, especially those with significant symptoms. Given emerging data on the benefits of early rhythm control (22, 23), the increasing use of rhythm control is expected to continue. Patients with recurrent episodes of AF and those with persistent AF should be referred to a cardiologist to discuss rate versus rhythm control. In general, a rhythm control strategy is preferred when 1) patients have symptoms related to AF despite adequate rate control; 2) cardiac remodeling, such as a severely enlarged left atrium, is absent; and 3) comorbid conditions that may lessen the effectiveness of rhythm control are minimal and/or well controlled.

#### **Drug therapy for rate control**

Although early rhythm control should be considered in patients with AF, clinicians should determine the need for drug therapy to control the ventricular rate in all patients with AF even if rhythm control is the goal. Although criteria for rate control vary with patient age, the traditional targets have been 60 to 80 beats/min at rest and 90 to 110 beats/min during moderate exercise. However, a study comparing a strategy of lenient rate control (resting heart rate  $\leq$  110 beats/min) versus strict rate control ( $\leq$  80 beats/min) found no advantage to the stricter strategy (24). Recommended first-line therapy to decrease atrioventricular nodal conduction includes  $\beta$ -blockers and nondihydropyridine calcium-channel antagonists.

Digitalis and amiodarone slow conduction through the atrioventricular node but are not recommended as first-line monotherapy for rate control (13). Digitalis does not reduce the tachycardia that occurs with exercise and is unlikely to control rate in patients with heart failure and high sympathetic activity. However, digitalis has the advantage

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of rate slowing without the potential for lowering of blood pressure. Amiodarone is occasionally used to reduce ventricular response if other agents have failed, but this practice is difficult to justify because of the drug's associated toxicities (25, 26).

#### **Strategies for rhythm control**

Patients can be converted to normal sinus rhythm with direct electrical current or with drugs. Electrical cardioversion is indicated when the patient is hemodynamically unstable.

Patients should receive therapy to achieve both rate control and adequate anticoagulation before elective direct current or pharmacologic cardioversion of AF lasting longer than 48 hours. In addition, serum potassium, serum magnesium, and ionized calcium levels should be above 4.0, 1.0, and 0.5 mg/dL, respectively. In most cases, cardioversion should be performed in a hospital setting to permit adequate monitoring for potential adverse effects, such as bradycardia and the proarrhythmic effects of antiarrhythmic drugs (21).

Several antiarrhythmic medications can be used to treat AF (Table 1). Medications that block cardiac sodium channels (class I effect), such as flecainide and propafenone, are useful in patients without coronary heart disease or left ventricular dysfunction. They should not be used in patients with significant structural heart disease because they have been associated with increased mortality in these patients due to ventricular tachycardia or their negative inotropic effects (27). Other class I drugs, such as quinidine, disopyramide, and procainamide, are used rarely because of noncardiac adverse effects and concerns about proarrhythmia. Drugs that block potassium channels (class III effects), such as sotalol and dofetilide, can prolong the QT interval and cause torsade de pointes.

Dronedarone is a multichannel-blocking drug similar in structure to amiodarone but without iodine, rendering it

safer. Unlike amiodarone, dronedarone does not appear to cause thyroid, hepatic, or pulmonary toxicities. A study of 4300 patients demonstrated its safety in patients who had AF without advanced heart failure (28). As a result, dronedarone is approved by the U.S. Food and Drug Administration (FDA) to reduce hospitalizations in patients with AF but is contraindicated in patients with heart failure. Another trial in patients with permanent AF found increased mortality associated with dronedarone compared with placebo, so it is also contraindicated in this group (29).

#### **When should clinicians use antiarrhythmic medications to prevent recurrence of AF?**

The effectiveness of antiarrhythmic medications varies by patient, chronicity of AF, and underlying structural heart disease (25) (Table 1). Antiarrhythmic therapy is generally considered effective if it reduces the frequency of episodes and symptoms.

Very few prospective studies have compared antiarrhythmic medications. The Canadian Trial of Atrial Fibrillation randomly assigned 403 patients to amiodarone, sotalol, or propafenone and found that after a mean follow-up of 16 months, recurrence of AF was 35% with amiodarone compared with 63% with sotalol or propafenone (30). However, due to the risk for adverse effects, which increases with increasing duration of treatment, amiodarone should be avoided in younger patients and should generally be considered much later in the treatment of many patients with AF.

#### **When is anticoagulation indicated?**

Patients with paroxysmal, persistent, and permanent AF and those with atrial flutter have the same indications for anticoagulation. Anticoagulation is indicated when the risk for thromboembolism exceeds that for serious bleeding associated with anticoagulation (13).

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

Agent	Mechanism of Action	Dosage	Benefits	Adverse Effects	Notes
<b>Rate-controlling agents</b>					
<b>β-Blockers</b>					
Metoprolol	Selective β <sub>1</sub> -adrenergic receptor blocker	5 mg IV every 5 min, up to 15 mg; 50–100 mg PO twice daily	Convenient IV administration in patients with NPO status, rapid onset of action, dependable AV nodal blockade	Bradycardia, hypotension, heart block, bronchospasm (less frequently than nonselective β-blockers), worsening of CHF	Do not use in patients with Wolff-Parkinson-White syndrome
Propranolol	Nonselective β-adrenergic receptor blocker	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	Do not use in patients with Wolff-Parkinson-White syndrome
Esmolol	Short-acting IV β <sub>1</sub> selective adrenergic receptor blocker	0.05–0.2 mg/kg/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 β-adrenergic receptor blocker 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 β-blockers
Atenolol	Selective β <sub>1</sub> -adrenergic receptor blocker	5 mg IV over 5 min, repeat in 10 min; 25–100 mg PO once daily	Does not cross blood-brain barrier; fewer CNS adverse effects	Bradycardia, hypotension, heart block	Do not use in patients with Wolff-Parkinson-White syndrome
Nadolol	Nonselective β-adrenergic receptor blocker	20–120 mg once daily	Lower incidence of crossing of blood-brain barrier; fewer CNS adverse effects	Bradycardia, hypotension, heart block	Oral form only
<b>Calcium-channel blockers</b>					
Verapamil	Calcium-channel blocker	5–20 mg in 5-mg increments IV every 30 min, or 0.005-mg/kg/min infusion; 120–360 mg PO daily, in divided doses or in slow-release form	Consistent AV nodal blockade	Hypotension, heart block, direct myocardial depression	Do not use in patients with Wolff-Parkinson-White syndrome
Diltiazem	Calcium-channel blocker	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 patients with Wolff-Parkinson-White syndrome
Digoxin (cardiac glycoside)	Na <sup>+</sup> -K <sup>+</sup> 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 (see Diagnosis section)	Do not use a loading dose; first-line therapy only in patients with decreased LV systolic function; dosage adjustment required in renal impairment; not useful for rate control with exercise; not useful for conversion of AF or atrial flutter to NSR
<b>Antiarrhythmic agents</b>					
Class Ia					
Procainamide	Prolongs conduction and slows repolarization by blocking inward Na <sup>+</sup> flux	1–2 g every 12 h (shorter-acting oral preparations are no longer available)	Convenient IV dosing available with maintenance infusion, conversion to oral tablets, very effective at converting AF to NSR	Hypotension common (slow rate of infusion), negative inotropic agent, nausea, vomiting, lupus-like syndrome, QT prolongation, proarrhythmia	Not recommended because of frequent adverse effects; need to follow drug levels and QT interval for toxicity, adjust dose in patients with renal insufficiency, and avoid in patients with more than mild renal function impairment; not for use in patients with severe LV dysfunction; can be used in patients with Wolff-Parkinson-White syndrome

*Continued on following page*

Table 1—Continued

Agent	Mechanism of Action	Dosage	Benefits	Adverse Effects	Notes
Quinidine gluconate	Prolongs conduction and slows repolarization; blocks fast inward Na <sup>+</sup> 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 oral dosing	Proarrhythmia, nausea, vomiting, diarrhea, QT prolongation	Not recommended because of frequent adverse effects; follow drug levels and QT interval for toxicity; adjust dose in patients with renal insufficiency; oral agent only
Disopyramide	Electrophysiologic properties similar to those of 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; useful in patients with hypertrophic obstruction cardiomyopathy	QT prolongation (not PR or QRS), torsade de pointes, heart block	Rarely used in current era of antiarrhythmic therapy; oral agent only, negative inotropic properties; potent anticholinergic properties can cause urine retention or exacerbation of narrow-angle glaucoma
<b>Class Ic</b>					
Flecainide	Blocks Na <sup>+</sup> channels (and fast Na <sup>+</sup> current)	50–150 mg PO every 12 h; single loading doses of 300 mg are also efficacious in conversion of recent-onset AF	Efficacy in paroxysmal AF with structurally normal hearts	Atrial flutter or atrial tachycardia with rapid ventricular response; VT and VF in patients with heart disease	Not for use in patients with structurally abnormal hearts
Propafenone	Blocks myocardial Na <sup>+</sup> channels	225–400 mg PO every 8 h; single loading doses of 600 mg are also efficacious in conversion of recent-onset AF	Efficacy in paroxysmal and sustained AF	Atrial flutter or atrial tachycardia with rapid ventricular response	Antiarrhythmic and weak calcium channel and β-blocking properties; not for use in patients with structurally abnormal hearts
<b>Class III</b>					
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 (torsade de pointes) occurred in 8.3% of patients in a clinical trial (most with LV dysfunction); QT prolongation	In some centers, only used in electrophysiology laboratory; may also be used to facilitate unsuccessful direct-current cardioversion; IV form only
Amiodarone*	Blocks Na <sup>+</sup> channels (affinity for inactivated channels); blocks calcium channels; noncompetitive α <sub>1</sub> - and β-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 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 patients with Wolff-Parkinson-White syndrome; β-blocking properties
Sotalol	Nonselective β <sub>1</sub> and β <sub>2</sub> blocker; prolongs action potential duration	80–240 mg PO every 12 h	Helpful for rate control because of β-blocking properties	Fatigue, depression, bradycardia, torsade de pointes, CHF	β-blocking properties, but some negative inotropic activity; lethal arrhythmias possible; adjust dose in patients with renal insufficiency; initiate on telemetry Do not use in patients with an ejection fraction of < 20%
Dofetilide	Blocks rapid component of the delayed rectifier potassium current (I <sub>Kr</sub> ), prolonging refractoriness without slowing conduction	125–500 mcg twice daily	Can be used for conversion to and maintenance of NSR; well tolerated	QT prolongation, torsade de pointes (2%–4% risk); greatest risk in patients with baseline prolonged QT, patients with hypokalemia, patients taking other repolarization-prolonging agents, and after conversion to NSR	Must be strictly dosed according to renal function, body size, and age; contraindicated in patients with creatinine clearance < 20 mL/min; risk–benefit ratio determination in progress per larger clinical experience; no known significant drug interactions; initiate on telemetry

Continued on following page

Table 1—Continued

Agent	Mechanism of Action	Dosage	Benefits	Adverse Effects	Notes
Dronedarone	Blocks Na <sup>+</sup> channels (affinity for inactivated channels); blocks calcium channels; noncompetitive $\alpha$ - and $\beta$ -receptor inhibitor	400 mcg twice daily	Modest efficacy; shown to reduce hospitalizations and cardiovascular mortality in patients with non-permanent AF	Gastrointestinal intolerance	Contraindicated in patients with permanent AF or decompensated CHF

AF = atrial fibrillation; AV = atrioventricular; CHF = congestive heart failure; CNS = central nervous system; IV = intravenous (or intravenously); LV = left ventricular; NPO = nothing by mouth (*nil per os*); NSR = normal sinus rhythm; PO = orally; VF = ventricular fibrillation; VT = ventricular tachycardia.

\* Amiodarone can cause permanent liver and lung toxicities that are dose- and duration-dependent (25, 26). Liver toxicity causes hepatitis that can progress to cirrhosis. Pulmonary toxicity can develop within 6 weeks or after years of therapy and most often manifests as cough and dyspnea. Pulmonary imaging (generally a chest computed tomography scan) can show a broad range of findings, including segmental or diffuse infiltrates. Other adverse effects include thyroid dysfunction (hypothyroidism or hyperthyroidism), sun sensitivity, and tremors (26).

31. January CT, Wann LS, Calkins H, et al. 2019 AHA/ACC/HRS focused update of the 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society in collaboration with the Society of Thoracic Surgeons. Circulation. 2019;140:e125-e151. [ PMID: 30686041 ]

32. Zabalgoitia M, Halperin JL, Pearce LA, et al. Transthoracic echocardiographic correlates of clinical risk of thromboembolism in nonvalvular atrial fibrillation. Stroke Prevention in Atrial Fibrillation III Investigators. J Am Coll Cardiol. 1998;31:1622-1626. [ PMID: 9626843 ]

33. Gage BF, Waterman AD, Shannon W, et al. Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrillation. JAMA. 2001;285:2864-2870. [ PMID: 11401607 ]

34. Connolly SJ, Ezekowitz MD, Yusuf S, et al.; RE-LY Steering Committee and Investigators. Dabigatran versus warfarin in patients with atrial fibrillation. N Engl J Med. 2009;361:1139-1151. [ PMID: 19717844 ]

35. Patel MR, Mahaffey KW, Garg J, et al.; ROCKET AF Investigators. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. N Engl J Med. 2011;365:883-891. [ PMID: 21830957 ]

The decision to initiate anticoagulation is driven by risk scores, the most popular of which (and the one recommended by professional guidelines) is the CHA<sub>2</sub>DS<sub>2</sub>-VASc score (2, 13, 31-33) (Table 2). Female sex is important to consider in the CHA<sub>2</sub>DS<sub>2</sub>-VASc score in patients 65 years of age or older and those with 2 or more stroke risk factors not related to sex (31). Table 3 presents recommendations for using this score to determine the need for anticoagulation. Current guidelines recommend anticoagulation for all patients with documented AF (symptomatic or asymptomatic) and 2 or more of the CHA<sub>2</sub>DS<sub>2</sub>-VASc risk factors. Anticoagulation is considered reasonable but not mandatory when 1 risk factor is present (13, 31).

### What anticoagulation regimens should clinicians use?

For decades, warfarin was the mainstay of therapy for anticoagulation in patients with AF. Although warfarin reduces risk for stroke by 65% (2), it has a narrow therapeutic window, and its metabolism is affected by many drug and dietary interactions, necessitating frequent international normalized ratio (INR) monitoring and dosage adjustments. These limitations have led to the development of several non-vitamin K-dependent oral anticoagulants, 4 of which have been approved by the FDA for prevention of thromboembolism in patients with AF who do not have a mechanical heart valve or mitral

stenosis (34-37) (Table 4). These drugs have several advantages, including rapid onset of action, no requirement for INR monitoring, and minimal potential for drug-drug interactions. They also are not influenced by diet and are cleared to varying degrees by the kidneys, with guidelines for renal dose adjustment. Based on these advantages and the strong evidence of efficacy and safety, current professional guidelines recommend non-vitamin K-dependent anticoagulants over warfarin for AF except in patients with mitral stenosis and mechanical heart valves, who should continue using warfarin (31, 38).

Because the onset of action and clearance of non-vitamin K-dependent oral anticoagulants are more rapid than those of warfarin, management is easier when anticoagulation is temporarily discontinued. Antidotes for the non-vitamin K-dependent oral anticoagulants have also been developed. Idarucizumab is a humanized antibody fragment that is approved for reversal of life-threatening bleeding associated with the direct thrombin inhibitor dabigatran (39). Andexanet- $\alpha$  is a modified recombinant derivative of factor Xa that acts as a decoy receptor to reverse the effects of rivaroxaban, apixaban, and edoxaban (40).

Before cardioversion, non-vitamin K-dependent oral anticoagulants or warfarin should be used (to achieve an INR

**Table 2. CHA<sub>2</sub>DS<sub>2</sub>-VASc Score**

Characteristic	Points
Congestive heart failure	1
Hypertension	1
Age ≥75 y	2
Diabetes mellitus	1
Stroke/transient ischemic attack	2
Vascular disease	1
Age 65-74 y	1
Sex category (female sex)	1

of 2.0 to 3.0 when warfarin is used) for at least 3 to 4 consecutive weeks in patients with AF of undetermined duration or AF lasting more than 48 hours. All of these anticoagulants should be continued for at least 4 weeks after cardioversion (31). Patients with a CHA<sub>2</sub>DS<sub>2</sub>-VASc score of 2 or higher should continue anticoagulation indefinitely unless they develop serious bleeding (31). Adherence to the non-vitamin K-dependent oral anticoagulants is more difficult to assess because blood tests are not used to assess anticoagulation, so patients need to be educated about not missing doses.

An alternative to 3 to 4 weeks of adequate anticoagulation before cardioversion is to perform transesophageal echocardiography. If a left atrial clot is not present and anticoagulation has been started, the patient can undergo cardioversion. It is critical that therapeutic anticoagulation be present at the time of cardioversion and continue uninterrupted for at least 4 weeks (31). Patients with a thrombus in the left atrial appendage must receive anticoagulation for 4 weeks before cardioversion regardless of the duration of AF,

and many clinicians repeat transesophageal echocardiography before cardioversion to confirm that the thrombus has resolved.

### When should clinicians consider nondrug therapies?

Nondrug therapies for AF are usually considered after failure of drug therapy. These include catheter ablation of the atrioventricular node and permanent pacing, catheter or surgical ablation of parts of the atrium where AF begins, and occlusion of the left atrial appendage for stroke prevention.

Atrioventricular node catheter ablation is used when pharmacologic rate control cannot be achieved, usually because of intolerance of medications. This situation is common in older patients. Atrioventricular node ablation is highly effective for control of excessive tachycardia, but it requires pacemaker insertion and often leads to pacemaker dependence. As such, it could cause progressive left ventricular dysfunction due to continuous right ventricular pacing. Cardiac resynchronization therapy and conduction system pacing are effective at preventing

36. Granger CB, Alexander JH, McMurray JJ, et al.; ARISTOTLE Committee and Investigators. Apixaban versus warfarin in patients with atrial fibrillation. *N Engl J Med.* 2011;365:981-992. [PMID: 21870978]
37. Giugliano RP, Ruff CT, Braunwald E, et al.; ENGAGE AF-TIMI 48 Investigators. Edoxaban versus warfarin in patients with atrial fibrillation. *N Engl J Med.* 2013;369:2093-2104. [PMID: 24251359]
38. Connolly SJ, Brueckmann M, et al.; RE ALIGN Investigators. Dabigatran versus warfarin in patients with mechanical heart valves. *N Engl J Med.* 2013;369:1206-1214. [PMID: 23991661]
39. Pollack CV Jr, Reilly PA, Eikelboom J, et al. Idarucizumab for dabigatran reversal. *N Engl J Med.* 2015;373:511-520. [PMID: 26095746]
40. Connolly SJ, Milling TJ Jr, Eikelboom JW, et al.; ANNEXA-4 Investigators. Andexanet alfa for acute major bleeding associated with factor Xa inhibitors. *N Engl J Med.* 2016;375:1131-1141. [PMID: 27573206]

**Table 3. Guidelines for Thromboembolic Prophylaxis According to CHA<sub>2</sub>DS<sub>2</sub>-VASc Score Recommendation**

Score	Recommendation
0	No therapy required
1	No therapy required, but treatment with an anticoagulant (dabigatran, rivaroxaban, apixaban, or edoxaban over warfarin unless the patient has mitral stenosis or a mechanical heart valve) is also reasonable
≥2	Anticoagulation with dabigatran, rivaroxaban, apixaban, or edoxaban (warfarin should be used in patients with mitral stenosis and those with a mechanical heart valve)

**Table 4. Non-Vitamin K-Dependent Anticoagulants for Atrial Fibrillation\***

Medication	Mechanism	Dose	Renal Dose Adjustment	Contraindications
Dabigatran	Direct thrombin inhibitor	150 mg twice daily	75 mg twice daily in patients with CrCl of 15-30 mL/min	CrCl <15 mL/min Mechanical heart valve
Rivaroxaban	Factor Xa inhibitor	20 mg once daily with food	15 mg once daily in patients with CrCl of 15-50 mL/min	CrCl <15 mL/min Mechanical heart valve
Apixaban	Factor Xa inhibitor	5 mg twice daily	2.5 mg twice daily in patients with ≥2 of the following: age >80 y, body weight ≤60 kg, creatinine level ≥1.5 mg/dL	CrCl <15 mL/min Mechanical heart valve
Edoxaban	Factor Xa inhibitor	60 mg once daily in patients with CrCl >50 and ≤95 mL/min	30 mg once daily in patients with CrCl of 15-50 mL/min	CrCl >95 mL/min Mechanical heart valve

CrCl = creatinine clearance.

\* An important advantage of the non-vitamin K-dependent oral anticoagulants is their significantly lower risk for intracranial hemorrhage compared with warfarin. The non-vitamin K-dependent oral anticoagulants are contraindicated in patients with mechanical heart valves (31, 38), but they can be used in patients with native valve disease, except for mitral stenosis (31).

cardiomyopathy induced by right ventricular pacing (41, 42). Pacing therapy without atrioventricular node ablation has little effect on the burden of AF but may be helpful in patients with AF and symptomatic bradycardia, which is often a side effect of antiarrhythmic medications.

Ablation of parts of the left atrium where fibrillation begins has been shown to be effective in preventing recurrent symptomatic AF in highly selected patients (13, 31). The ideal patient has paroxysmal AF, is young and otherwise healthy, and has no or mild structural heart disease; however, ablation has also shown effectiveness in other patients, including those with persistent AF, those with structural heart disease, and those with heart failure.

In the CASTLE-AF (Catheter Ablation versus Standard Conventional Therapy in Patients with Left Ventricular Dysfunction and Atrial Fibrillation) trial, patients with symptomatic paroxysmal or persistent AF were randomly assigned to either catheter ablation (n = 179) or rate- or rhythm-controlling medications (n = 184). All patients had a left ventricular ejection fraction of 35% or less; New York Heart Association class II, III, or IV heart failure; and an implantable cardioverter-defibrillator. During a median follow-up of 37.8 months, the primary composite outcome of death due to any cause or hospitalization for

worsening heart failure occurred in fewer patients who underwent ablation than in those who received medications (51 [28.5%] vs. 82 [44.6%] patients; HR, 0.62 [CI, 0.43 to 0.87]; P = 0.007). Significantly fewer patients in the ablation group died from any cause, were hospitalized for worsening heart failure, or died from cardiovascular causes (43).

In the CABANA (Catheter Ablation vs Antiarrhythmic Drug Therapy for Atrial Fibrillation) trial, 2204 symptomatic patients with AF who either were aged 65 years or older or were younger than 65 years and had 1 or more risk factors for stroke were randomly assigned to catheter ablation (n = 1108) or pharmacologic therapy (n = 1096). The primary end point was a composite of death, disabling stroke, cardiac arrest, or serious bleeding. In the intention-to-treat analysis, during a median follow-up of 48.5 months, the primary end point occurred in 8.0% (n = 89) of patients in the ablation group versus 9.2% (n = 101) in the pharmacologic therapy group (HR, 0.86 [CI, 0.65 to 1.15]; P = 0.30). However, in the 1240 patients using the study ECG event recording system, time to first recurrence of AF in the intention-to-treat analysis was reduced by 48% with catheter ablation versus pharmacologic therapy (adjusted HR, 0.52 [CI, 0.45 to 0.60]; P < 0.001) (44). In the quality-of-life substudy of CABANA, catheter ablation resulted in clinically important and

41. Khurshid S, Obeng-Gyimah E, Supple GE, et al. Reversal of pacing-induced cardiomyopathy following cardiac resynchronization therapy. *JACC Clin Electrophysiol*. 2018;4:168-177. [PMID: 29749933]

42. Sharma PS, Vijayaraman P. Conduction system pacing for cardiac resynchronization. *Arrhythm Electrophysiol Rev*. 2021;10:51-58. [PMID: 33936744]

43. Marrouche NF, Brachmann J, Andresen D, et al.; CASTLE-AF Investigators. Catheter ablation for atrial fibrillation with heart failure. *N Engl J Med*. 2018;378:417-427. [PMID: 29385358]

44. Packer DL, Mark DB, Robb RA, et al.; CABANA Investigators. Effect of catheter ablation vs antiarrhythmic drug therapy on mortality, stroke, bleeding, and cardiac arrest among patients with atrial fibrillation: the CABANA randomized clinical trial. *JAMA*. 2019;321:1261-1274. [PMID: 30874766]

*significant improvements in quality of life at 12 months compared with drug therapy (45). Also, in another CABANA substudy focused on patients with heart failure (mostly with preserved ejection fraction), catheter ablation led to clinically important improvements in survival, freedom from AF recurrence, and quality of life compared with pharmacologic therapy (46).*

Recent guidelines recommend catheter ablation for symptomatic patients with paroxysmal or persistent AF if an attempt at antiarrhythmic drug therapy has failed or as first-line therapy (47). Catheter ablation is associated with a likelihood of improving symptoms of approximately 70%, but patients may require a second and even a third procedure to achieve this level of success. Major complications occur in fewer than 1% of cases and include cardiac perforation; bleeding; access site issues, such as pseudoaneurysm formation; and stroke. In the CABANA trial, the most common serious complication in the catheter ablation group was cardiac tamponade (0.8%). Other complications in the ablation group included minor hematomas (2.3%) and pseudoaneurysms (1.1%) (44). A minimally invasive surgical ablation (Maze procedure) is also available at specialized centers. It is important to emphasize that a patient's decision to undergo ablation should not be based on the expectation of avoiding anticoagulation, and there is currently no evidence that any rhythm control therapy is reliably associated with reduced risk for thromboembolism.

A significant number of strokes in patients with AF are believed to be caused by emboli originating in the left atrial appendage (16). The FDA has approved the Watchman device for occlusion of the appendage when a nonpharmacologic alternative to warfarin is sought, with consideration of the risks of this device compared with the bleeding risks associated with warfarin. Major risks include cardiac perforation,

embolization of the device, and device-related thrombosis (48, 49). The Amplatzer Amulet (Abbott), another left atrial appendage occlusion device, is now available for clinical use in the United States. This device was found to be noninferior with regard to safety and effectiveness of stroke prevention for nonvalvular AF compared with the Watchman device and superior for left atrial appendage occlusion (50).

## How should clinicians monitor patients?

Patients with AF should receive regular follow-up to determine the effectiveness and safety of therapy. For some patients, monitoring warfarin anticoagulation drives the frequency of follow-up. During these visits, clinicians determine whether symptoms are adequately controlled. In patients with persistent or permanent AF, resting and exercise heart rates should be assessed to determine the adequacy of rate control. Patients who have not improved or cannot tolerate antiarrhythmic medications should be considered for catheter ablation. Amiodarone requires liver and thyroid function studies at least every 6 months and pulmonary function tests with assessment of diffusion capacity for carbon monoxide (DLCO) every year or if pulmonary toxicity is suspected. Patients receiving dofetilide and sotalol should have an ECG performed to assess the QTc interval and renal function, potassium, and magnesium tested every 3 to 6 months. Patients receiving dabigatran, rivaroxaban, apixaban, and edoxaban should have renal function tested at least annually to determine the need for dose adjustment.

## What is new in this update?

Since In the Clinic last considered management of AF in March 2017 (51), the non-vitamin K-dependent oral anticoagulants have been recommended over warfarin for thromboembolic prophylaxis, and reversal agents for all non-vitamin K-dependent oral anticoagulants in clinical use have become

45. Mark DB, Anstrom KJ, Sheng S, et al.; CABANA Investigators. Effect of catheter ablation vs medical therapy on quality of life among patients with atrial fibrillation: the CABANA randomized clinical trial. *JAMA*. 2019;321:1275-1285. [PMID: 30874716]
46. Packer DL, Piccini JP, Monahan KH, et al.; CABANA Investigators. Ablation versus drug therapy for atrial fibrillation in heart failure: results from the CABANA trial. *Circulation*. 2021;143:1377-1390. [PMID: 33554614]
47. Calkins H, Hindricks G, Cappato R, et al. 2017 HRS/EHRA/ECAS/AFPS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation. *Heart Rhythm*. 2017;14:e275-e444. [PMID: 28506916]
48. Reddy VY, Doshi SK, Sievert H, et al.; PROTECT AF Investigators. Percutaneous left atrial appendage closure for stroke prophylaxis in patients with atrial fibrillation: 2.3-year follow-up of the PROTECT AF (Watchman Left Atrial Appendage System for Embolic Protection in Patients with Atrial Fibrillation) trial. *Circulation*. 2013;127:720-729. [PMID: 23325525]
49. Dukkipati SR, Kar S, Holmes DR, et al. Device-related thrombus after left atrial appendage closure: incidence, predictors, and outcomes. *Circulation*. 2018;138:874-885. [PMID: 29752398]
50. Lakshminrushti D, Thaler D, Ellis CR, et al. Amplatzer Amulet left atrial appendage occluder versus Watchman device for stroke prophylaxis (Amulet IDE): a randomized, controlled trial. *Circulation*. 2021;144:1543-1552. [PMID: 34459659]
51. Zimetbaum P. In the Clinic: Atrial fibrillation. *Ann Intern Med*. 2010;153:ITC61-15, quiz ITC616. [PMID: 21135291]

available. Early rhythm control has been shown to be superior to rate control. Catheter ablation has become more widely accepted for prevention of recurrent AF, even as first-line therapy. Closure of the left atrial appendage using an atrial occlusion device

has been approved for patients at risk for stroke who are unable to take systemic anticoagulation. Currently, 2 left atrial appendage occlusion devices are approved by the FDA and are available for clinical use in the United States, with promising results.

**Treatment...** Treatment goals for AF include improving symptoms, preventing stroke, preventing tachycardia-related cardiomyopathy and heart failure, and reducing risk for cognitive decline. Patients who should receive anticoagulation (largely with non-vitamin K-dependent oral anticoagulants) should be chosen using the CHA<sub>2</sub>DS<sub>2</sub>-VASc score. Treatment should prioritize early rhythm control in symptomatic patients. Catheter ablation has been shown to significantly reduce recurrence of AF and improve quality of life. Data also suggest improved survival and reduced hospitalization for heart failure with catheter ablation in patients with AF and heart failure. Atrioventricular nodal ablation therapy may be appropriate for select patients with highly symptomatic disease for whom other therapeutic modalities are not successful. Closure of the left atrial appendage is an alternative for thromboembolic protection in patients at risk for stroke who are not candidates for anticoagulation.

## CLINICAL BOTTOM LINE

## Practice Improvement

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

In 2020, the American College of Cardiology and the American Heart Association updated the 2016 clinical performance and quality measures for treatment of AF and atrial flutter. The updated version included 5 performance measures related to stroke risk

assessment and anticoagulation for stroke prevention (52).

### What do professional organizations recommend with regard to management of patients with AF?

The material presented in this review has been updated and is consistent with the 2019 guidelines from the American Heart Association, the American College of Cardiology, and the Heart Rhythm Society (31).

52. Heidenreich PA, Estes NAM 3rd, Fonarow GC, et al. 2020 update to the 2016 ACC/AHA clinical performance and quality measures for adults with atrial fibrillation or atrial flutter: a report of the American College of Cardiology/American Heart Association Task Force on Performance Measures. *Circ Cardiovasc Qual Outcomes*. 2021;14:e00100. [PMID: 33284642]

# In the Clinic Tool Kit

## Atrial Fibrillation

### Patient Information

<https://medlineplus.gov/atrialfibrillation.html>

<https://medlineplus.gov/languages/atrialfibrillation.html>

Information and handouts in English and other languages from the National Institutes of Health's MedlinePlus.

[www.heart.org/en/health-topics/atrial-fibrillation](http://www.heart.org/en/health-topics/atrial-fibrillation)

Resources from the American Heart Association.

[www.nhlbi.nih.gov/health/atrial-fibrillation](http://www.nhlbi.nih.gov/health/atrial-fibrillation)

[www.nhlbi.nih.gov/es/salud/fibrilacion-auricular](http://www.nhlbi.nih.gov/es/salud/fibrilacion-auricular)

Information in English and Spanish from the National Heart, Lung, and Blood Institute.

### Information for Health Professionals

[www.ahajournals.org/doi/10.1161/CIR.0000000000000665](http://www.ahajournals.org/doi/10.1161/CIR.0000000000000665)

2019 focused update of the 2014 guideline for the management of patients with atrial fibrillation from the American College of Cardiology, the American Heart Association, and the Heart Rhythm Society in collaboration with the Society of Thoracic Surgeons.

[www.ahajournals.org/doi/10.1161/HCQ.000000000000100](http://www.ahajournals.org/doi/10.1161/HCQ.000000000000100)

2020 update of the 2016 clinical performance and quality measures for adults with atrial fibrillation or atrial flutter from the American College of Cardiology and the American Heart Association.

[www.heartrhythmjournal.com/article/S1547-5271\(17\)30590-8/fulltext](http://www.heartrhythmjournal.com/article/S1547-5271(17)30590-8/fulltext)

2017 expert consensus statement on catheter and surgical ablation of atrial fibrillation from the Heart Rhythm Society, the European Heart Rhythm Association, the European Cardiac Arrhythmia Society, the Asia Pacific Heart Rhythm Society, and the Latin American Society of Cardiac Stimulation and Electrophysiology.

# WHAT YOU SHOULD KNOW ABOUT ATRIAL FIBRILLATION

In the Clinic  
*Annals of Internal Medicine*

## What Is Atrial Fibrillation?

Atrial fibrillation, or Afib, is a heart rhythm problem where your heart beats very fast or abnormally. Over time, this irregular beat can damage your heart muscle. Because blood can clot when it doesn't move smoothly, Afib can also lead to formation of blood clots in the heart that travel to the brain and cause stroke. Afib can come and go, or you can have it all the time. It is more common in people with heart conditions and in older people. You are at higher risk for stroke from Afib if you:

- Are older than 65 years
- Have a history of stroke or mini-stroke
- Have heart failure
- Have high blood pressure
- Have diabetes
- Have coronary artery disease or peripheral artery disease
- Have sleep apnea



if you have it during the test. If you have symptoms that could indicate Afib but your ECG is normal, your doctor may ask you to wear a monitor that tracks your heart's activity while you go about your day.

## How Is It Treated?

- Afib should be treated to reduce symptoms, prevent stroke, and prevent the heart from becoming too large and weak.
- Your doctor may prescribe medicines called blood thinners or medicines that slow the heartbeat and make it more regular.
- If medicines do not work, your doctor may recommend a procedure called "ablation," which helps to stop abnormal heart signals.
- In some cases, a pacemaker can be implanted in the chest with wires in the heart to treat a slow heart rate.

Talk to your doctor about the best treatment plan for you.

## Questions for My Doctor

- How long will I need to take medicines for Afib?
- What are the side effects of my medicines?
- Should I worry about other medicines I'm taking?
- Can I still do all the things I like to do?
- How can I reduce my risk for stroke?
- Can I exercise with Afib?
- When should I go to the emergency room?

## How Is It Diagnosed?

Your doctor may order an electrocardiogram (ECG), which is a painless test that tracks your heartbeats. Your doctor may see Afib on an ECG

## For More Information



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### American College of Physicians

[www.acponline.org/online-learning-center/cardiology](http://www.acponline.org/online-learning-center/cardiology)

### MedlinePlus

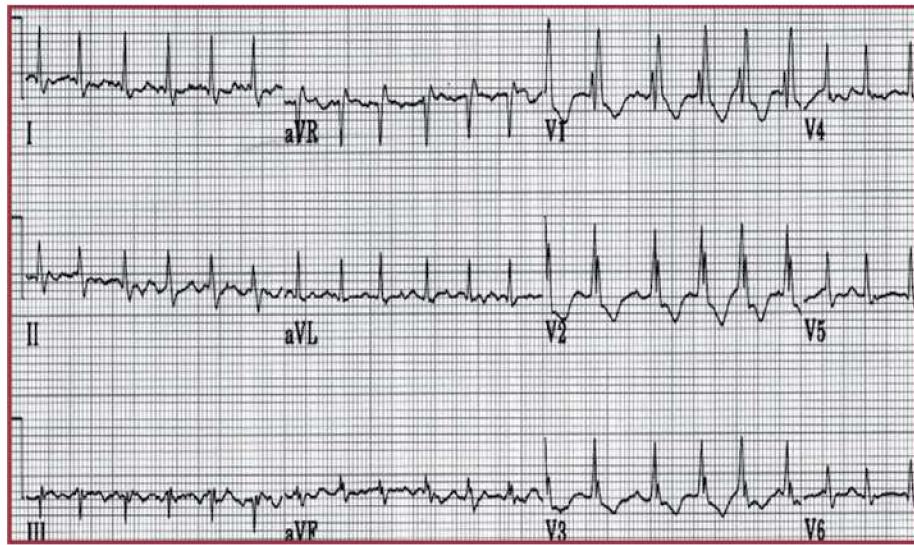
<https://medlineplus.gov/atrialfibrillation.html>

### Heart Rhythm Society

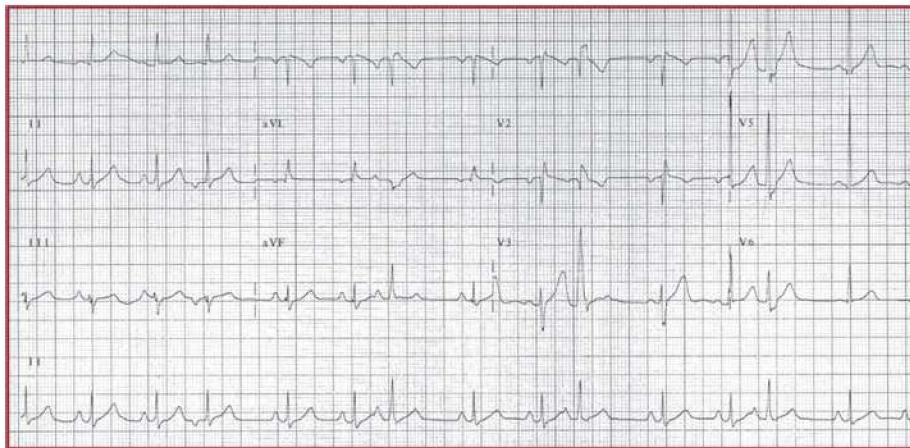
[www.hrsonline.org/Patient-Resources/Heart-Diseases-Disorders/Atrial-Fibrillation-Afib](http://www.hrsonline.org/Patient-Resources/Heart-Diseases-Disorders/Atrial-Fibrillation-Afib)

Patient Information

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



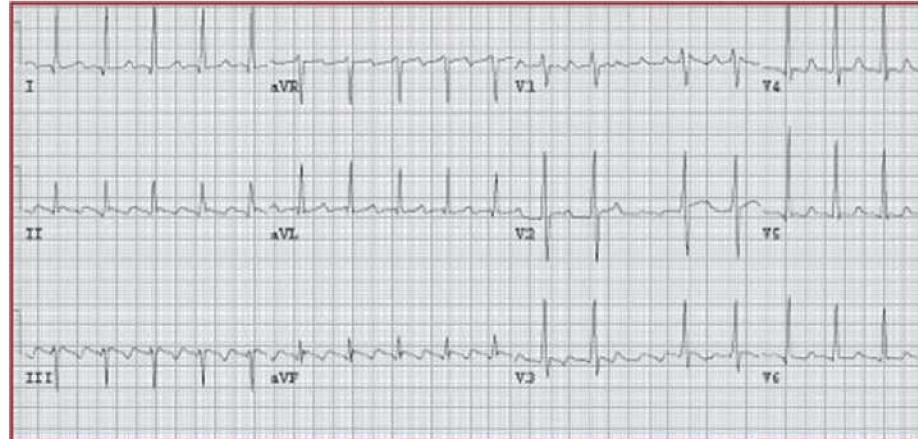
**Appendix Figure 2. Electrocardiogram showing sinus rhythm with frequent premature atrial contractions.**



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**Appendix Figure 3. Atrial flutter.**

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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).

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