2. ATRIAL FIBRILLATION

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The quality indicators for atrial fibrillation were developed from recent reviews (Pritchett, 1992; Kudenchuk, 1996); results from the Framingham Study (Wolf, 1978; Kannel, 1982; Brand, 1985; Wolf 1991); and a consensus statement from the American Heart Association (Prystowsky, 1996). Additional sources were used for the anticoagulation quality indicators: guidelines from the Fourth American College of Chest Physicians (ACCP) Consensus Conference on Antithrombotic Therapy (Laupacis, 1995) and an analysis of pooled data from five randomized controlled trials of antithrombotic therapy in atrial fibrillation (Atrial Fibrillation Investigators, 1994). Where these core references cited studies to support individual indicators, we have referenced the original sources. We also performed narrow MEDLINE searches of the medical literature from 1985 to 1997 to supplement these references for particular indicators.

IMPORTANCE

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered in clinical practice, with a prevalence of approximately five percent in persons over the age of 65 in the United States (Halperin, 1988; Furberg, 1994; Prystowsky, 1996). Hospital stays are longer than for any other arrhythmia, and in 1990, atrial fibrillation was the primary diagnosis in approximately 180,000 hospital admissions (Bialy, 1992).

The incidence of atrial fibrillation increases markedly with age. Data from the Framingham Study suggest that the prevalence of atrial fibrillation is 0.5 percent in the 50 to 59 year old age group and rises to 8.8 percent among 80-89 year olds (Kannel, 1982; Wolf, 1991). In addition to increasing age, atrial fibrillation is also associated with the presence of structural heart disease (such as rheumatic heart disease, heart failure, and coronary heart disease) and non-cardiac disorders. Valvular atrial fibrillation arises in the presence of
valvular heart disease, most commonly rheumatic mitral stenosis. Nonvalvular atrial fibrillation occurs in the absence of mitral stenosis or valvular prostheses. With the decline of rheumatic valvular heart disease in this country, atrial fibrillation is most commonly found in patients with hypertensive and ischemic heart disease and heart failure.

Atrial fibrillation is associated with a significant increase in morbidity and mortality. There are a myriad of symptoms that may accompany atrial fibrillation, many of which can be disabling. In addition, the dysrhythmia is associated with a marked increase in risk of stroke, systemic emboli, and death. The rate of stroke in untreated patients with atrial fibrillation is approximately five times that of persons without atrial fibrillation. The attributable risk of stroke from atrial fibrillation is 1.5 percent for 50 to 59 year olds, rising to 30 percent for those aged 80 to 89 (Wolf, 1987). Risk factors for thromboembolism in persons with atrial fibrillation are discussed below.

SCREENING

There is no role for screening asymptomatic patients for atrial fibrillation.

DIAGNOSIS

Although many persons with atrial fibrillation may be asymptomatic, with the rhythm disturbance noted incidentally (Brand, 1985), others present with clear symptoms. Symptoms include fatigue, palpitations, dyspnea, chest discomfort, presyncope, and dizziness, but are often non-specific and may be difficult to attribute to atrial fibrillation. This is particularly true in persons with comorbid cardiac or pulmonary disease. While the symptoms of atrial fibrillation occur more frequently at heart rates over 150 beats per minute, some patients, particularly the elderly, may be symptomatic at normal heart rates because of a loss of organized atrial contractile activity.

Isolated atrial fibrillation tends to be transient and reversible and occurs in the presence of another event or illness, such as hyperthyroidism, acute alcohol intoxication, cholinergic intoxication, pulmonary conditions that produce hypoxemia (such as pulmonary embolus), and non-cardiac surgery. Atrial fibrillation is found in 9 to 22
percent of patients with thyrotoxicosis (Woeber, 1992). One study found that 13 percent of patients in atrial fibrillation with no obvious cardiovascular cause had thyrotoxicosis (Forfar, 1979). It is estimated that 35 percent of atrial fibrillation is due to alcoholism and binge drinking, especially in younger patients (Kostinen, 1990; Lowenstein, 1992). Stimulant drug use may also play an important role in atrial fibrillation in this age group.

Paroxysmal atrial fibrillation occurs intermittently and is unrelated to an acute event or illness. It may proceed to chronic atrial fibrillation, in which atrial fibrillation is the predominant rhythm. Primary (or “lone”) atrial fibrillation, which denotes atrial fibrillation that arises in the absence of other preexisting conditions and with normal left ventricular function by echocardiogram, is a diagnosis of exclusion (Prystowsky, 1996). The majority of this chapter will focus on chronic or paroxysmal nonvalvular atrial fibrillation.

There is controversy about the prognosis of lone atrial fibrillation. Data from Framingham revealed that the relative risk of stroke was 4.1 compared with control subjects (Brand, 1985); while data from Olmsted County, Minnesota (Kopecky, 1987) showed no increased risk of stroke. However, the two studies defined lone atrial fibrillation differently and the study populations were significantly different. In Framingham, lone atrial fibrillation was defined as atrial fibrillation occurring in adults in the absence of an acute precipitating factor, coronary heart disease, congestive heart failure, rheumatic heart disease, or hypertensive heart disease. In contrast, the Olmsted County participants were all under 60 years old, included children, and excluded patients with hypertension, diabetes mellitus, chronic obstructive pulmonary disease, and other patients who were “ill.” The Framingham patients were thus older and more likely to be chronically ill than those in the Minnesota study.

Because many conditions can precipitate atrial fibrillation, patients with new onset atrial fibrillation should be carefully evaluated for reversible causes. They should be closely questioned about stimulant drug use and alcohol use (Indicator 1). In addition, a laboratory evaluation for hyperthyroidism should be performed, including
a thyroid stimulating hormone (TSH) level (Forfar, 1979; Woeber, 1992; Prystowsky, 1996) (Indicator 2). A chest radiograph should be taken to evaluate the patient for cardiomegaly, left atrial enlargement, pulmonary edema, and other pulmonary processes that may predispose to atrial fibrillation (Indicator 3). Although echocardiograms have been recommended to evaluate the presence of structural disease when there is no evidence of underlying systemic or metabolic disease (Prystowsky, 1996), there are no studies on when echocardiogram is indicated in the management of atrial fibrillation.

**TREATMENT**

Physicians must consider three therapeutic goals for patients with new onset atrial fibrillation: 1) control of ventricular rate, 2) restoration and maintenance of sinus rhythm, and 3) prevention of thromboembolism. While rate control is the initial goal of management, the risks and benefits of therapy to restore sinus rhythm and prevent thromboembolism are largely determined individually. Therapy to maintain normal sinus rhythm remains particularly controversial. There is an ongoing NIH trial, Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM), that will evaluate the relative benefits and risks of rate control alone versus attempting to maintain sinus rhythm (Prystowsky, 1996). The prevention of thromboembolism will be discussed in the "Follow-up" section.

**Control of Ventricular Rate**

Emergent electrical cardioversion is indicated in patients with new onset atrial fibrillation and a rapid ventricular rate complicated by concomitant hypotension, pulmonary edema, or evidence of ongoing myocardial ischemia or infarction. Electrical cardioversion is the treatment of choice for new atrial fibrillation when emergent rate and rhythm control are required.

In hemodynamically stable patients with chest pain or mild heart failure secondary to a rapid ventricular response, intravenous medications are indicated. Intravenous calcium antagonists (such as diltiazem or verapamil) or beta blockers (such as esmolol, propranolol, or metoprolol) often result in rapid rate control. Intravenous digoxin
slows the heart rate at rest, but has a delayed onset of action, with onset at 60 minutes and full effect requiring up to six hours.

**Restoration and Maintenance of Sinus Rhythm**

There are limited data on how and whether sinus rhythm should be restored and maintained in treating new onset atrial fibrillation. Restoration of sinus rhythm may improve symptoms and hemodynamics, and may be associated with increased cerebral blood flow (Prystowsky, 1996). In patients with atrial fibrillation who have hypertrophic cardiomyopathy and other forms of severe diastolic dysfunction, restoring the atrial contribution to ventricular filling may alleviate the symptoms of heart failure and syncope, and reduce mortality (Kudenchuk, 1996). It is also assumed that maintaining sinus rhythm lessens the risk of stroke. However, the data for the efficacy of maintaining sinus rhythm on stroke prevention are limited.

Cardioversion may be achieved through electrical or chemical means. It is extremely difficult to compare methods and drugs for cardioversion based on the published literature. Recent-onset or paroxysmal atrial fibrillation often reverts spontaneously, and most patients with atrial fibrillation can be transiently cardioverted to normal sinus rhythm (Lown, 1967). However, after cardioversion, there are high rates of reversion back to atrial fibrillation, with less than 25 percent of patients maintaining sinus rhythm at one year (Suttorp, 1993). Electrical cardioversion has success rates of 70 to 85 percent, in selected populations, with declining rates as patient age and the duration of atrial fibrillation increases (Arnold, 1992; Van Gelder, 1991).

There are minimal data from randomized clinical trials comparing the efficacy of any one drug over the others for restoration and maintenance of sinus rhythm in new onset atrial fibrillation. Therefore, selection of an antiarrhythmic agent should be individualized and will depend in part on renal and hepatic function, concomitant illnesses and drugs, and cardiovascular function (Prystowsky, 1996). In addition, as discussed above, the efficacy of maintaining normal sinus
rhythm is unproven. For these reasons, our indicators will not focus on maintenance of sinus rhythm.

**FOLLOW-UP**

Chronic atrial fibrillation is defined as atrial fibrillation of greater than 48 hours known duration.

**Rate Control**

For long term rate control, there are several potential treatment options. Patients with depressed atrioventricular node function may have adequate ventricular rate control and have no need for further therapy. However, even among patients whose heart rate is controlled at rest, changes in autonomic tone may result in excessive rates during exercise and may contribute to a tachycardia-mediated cardiomyopathy.

While several studies have compared heart rates of patients on digoxin, beta blockers, and calcium antagonists, there are no data from randomized clinical trials comparing the efficacy of any one of these drugs over the others for controlling symptoms or maintaining quality of life (Prystowsky, 1996). Therefore, our indicators will not focus on methods for rate control in chronic atrial fibrillation.

**Prevention of Thromboembolism**

Thromboembolism is a common and potentially devastating complication of chronic atrial fibrillation. Seventy-five percent of the thromboembolic events associated with atrial fibrillation involve the brain (Kudenchuk, 1995). Stroke in the majority of patients with atrial fibrillation is believed to be due to embolization of stasis-induced thrombi formed in the left atrium, especially the left atrial appendage. However, large percentages of patients with atrial fibrillation also have hypertension and carotid artery stenosis, both of which are additional risk factors for stroke. As a result, 25 percent of strokes in this population are believed to be due to intrinsic cerebrovascular disease, other cardiac sources of embolism, and aortic arch atheroma. It is also noteworthy that there does not appear to be a difference in the stroke rates of atrial fibrillation patients with and without carotid stenosis. Thus, the literature does not support routine
carotid ultrasound for patients with atrial fibrillation (Prystowsky, 1996).

Studies show that two groups of patients should definitely receive anticoagulant therapy: patients with valvular atrial fibrillation and those undergoing elective cardioversion. Patients with atrial fibrillation secondary to mitral valve disease (particularly mitral valve stenosis) have a 17-fold higher risk of a thromboembolic complications than age-, sex-, and blood pressure-matched controls, with 30 to 75 percent of this group sustaining an embolic event in the absence of anticoagulation (Wolf, 1978; Sherman, 1986; Siegel, 1987). Patients with chronic atrial fibrillation undergoing elective cardioversion without anticoagulation have a seven times higher risk of thromboembolism than those on anticoagulant therapy (Bjerkelund, 1969).

Recommendations for the majority of patients with chronic or paroxysmal atrial fibrillation, who do not fit into either of the above categories, are less clear cut. These patients can be stratified into those at low risk and those at high risk for thromboembolism. Pooled data from five randomized controlled trials suggest that the risk factors for stroke in patients with atrial fibrillation include prior stroke or transient ischemic attack (TIA), diabetes, a history of hypertension, age over 65, congestive heart failure, and coronary heart disease (Atrial Fibrillation Investigators, 1994; Stroke Prevention in Atrial Fibrillation Investigators, 1992a). In these patients, the annual risk of stroke is five percent or more. Age is a particularly important risk factor, and in patients over the age of 75, the risk of stroke without oral anticoagulation appears to outweigh the risk of intracranial hemorrhage on warfarin. In contrast, patients with atrial fibrillation under 65 years of age, who do not have risk factors for stroke, have an annual stroke risk of one percent or less. An intermediate group of patients, those between the ages of 65 and 75, have an annual risk of stroke of two to four percent. Additional echocardiographic predictors of stroke include left atrial enlargement and impaired left ventricular function (Stroke Prevention in Atrial Fibrillation Investigators, 1992b). The clinical factors and
transthoracic echocardiogram can be combined to identify patients in atrial fibrillation with high risk of thromboembolism.

Transesophageal echocardiogram provides better visualization of the left atrial appendage and may identify thrombi or areas of stasis in the left atrial or left atrial appendage. However, there are insufficient clinical data to recommend routine transesophageal echocardiography for risk stratification for patients with atrial fibrillation (Prystowsky, 1996), and our indicators do not specify any mandatory use for transesophageal echocardiogram.

Use of Anticoagulation

Several recent randomized controlled trials have evaluated the efficacy of oral anticoagulation and antiplatelet agents for stroke prevention in atrial fibrillation. An intention-to-treat analysis of five randomized controlled trials\(^1\) that evaluated warfarin compared to placebo showed a mean reduction in ischemic stroke of approximately 70 percent on therapy, using international normalized ratios (INRs) ranging from 1.8 to 4.2 (Atrial Fibrillation Investigators, 1994). There was an even greater reduction in the on-therapy analysis, and warfarin decreased the rate of death by 33 percent.

Warfarin can be difficult to take, requires frequent monitoring, and may require substantial dietary modification. However, the most significant risk of warfarin therapy is the risk of bleeding. Risk factors for bleeding on oral anticoagulants for atrial fibrillation vary from study to study, depending on the patient population (Fihn, 1993; Levine, 1995; Fihn, 1996; The Stroke Prevention in Atrial Fibrillation Investigators, 1996). Known risk factors for bleeding in patients on anticoagulant therapy include the intensity of the anticoagulation, the duration of therapy, and the use of other drugs that affect hemostasis (Levine, 1995; Fihn, 1996). There is some disagreement over the

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\(^1\) Atrial Fibrillation, Aspirin, And Anticoagulant Therapy Study (AFASAK), Boston Area Anticoagulation Trial for Atrial Fibrillation (BAATAF), Canadian Atrial Fibrillation Anticoagulation (CAFA), Stroke Prevention in Atrial Fibrillation (SPAF), Stroke Prevention in Nonrheumatic Atrial Fibrillation (SPINAF).
association between hemorrhage on warfarin and several other factors, among them increasing patient age, hypertension, prior gastrointestinal hemorrhage, renal disease, and a history of cerebrovascular disease (Levine, 1995; Fihn, 1993). In the five randomized controlled trials of warfarin and aspirin, a bleed was labeled major if it required a transfusion or hospitalization or if it involved a critical anatomic region, such as an intracranial or paraspinal hemorrhage (Laupacis, 1995). The combined risk of major bleeding for the patients in these five studies (mean age 65 years) was 1.0 percent in the control patients and 1.3 percent in the warfarin-treated patients. Among patients over 75, there was only one intracranial hemorrhage. However, other data suggest that older patients and those who are less closely monitored are more likely to have major bleeding. One study found that patients over 75 had a much higher risk of major hemorrhage during anticoagulation, using INRs of 2.0 to 4.5 (mean 2.7), than those younger than 75 (SPAF II, 1994).

The effectiveness of stroke prevention in atrial fibrillation is less clear for aspirin than for warfarin. Three clinical trials have evaluated aspirin versus placebo for stroke prevention. Using doses of aspirin ranging from 75 to 325 mg per day, there was an overall risk reduction of 25 percent (range 14 to 44 percent) (Petersen, 1989; SPAF, 1991; EAFT, 1993). However, direct comparisons have found aspirin to be significantly less effective than warfarin for preventing atrial fibrillation-related stroke (Petersen, 1989; SPAF, 1991; EAFT, 1993).

The following recommendations have been made by the American College of Chest Physicians (ACCP) Consensus Conference on Antithrombotic Therapy (Laupacis, 1995):

- All patients over 65 years old with atrial fibrillation and those patients under 65 years old with one or more risk factors for stroke (such as previous TIA or stroke; a history of hypertension, even if controlled; heart failure; diabetes; coronary heart disease; or thyrotoxicosis) should be strongly considered for oral anticoagulant therapy with a target INR of 2.0 to 3.0.
• Patients over 65 years old with atrial fibrillation and those patients under 65 years old with one or more risk factors for stroke who have contraindications to anticoagulation or who decline warfarin should receive aspirin at a dose of 325 mg per day.

• For patients in atrial fibrillation between 65 and 75 years old who do not have any risk factors for stroke, the decision between oral anticoagulants and aspirin should be based on patient and physician assessment of the potential benefits relative to the risk and inconvenience of therapy.

• Patients under 65 years of age with no risk factors for stroke can receive aspirin alone or no antithrombotic therapy.

Our proposed indicators on anticoagulation for atrial fibrillation stem from these recommendations (Indicators 4, 5, and 6).

Anticoagulation for Cardioversion

Both electrical and chemical cardioversion of atrial fibrillation can be complicated by systemic emboli. The risk of stroke in this situation is three to five percent. The likelihood of thromboembolism increases in older persons and in persons with a history of prior embolic events, coronary heart disease, heart failure, hypertension, and long duration atrial fibrillation (Bjerkelund, 1969; Prystowsky, 1996). No randomized controlled trials have evaluated the effectiveness of anticoagulation prior to cardioversion, however, as stated above, the rates of thromboembolism without anticoagulation range from 30 to 75 percent (Wolf, 1978; Sherman, 1986; Siegel, 1987).

Patients with atrial fibrillation of unknown duration or for more than 48 hours should receive at least three weeks of anticoagulation with warfarin prior to either electrical or pharmacological cardioversion and at least four weeks after cardioversion. Alternatively, immediate cardioversion preceded by intravenous heparin and followed by four weeks of warfarin may be used in those without thrombi on transesophageal echocardiography. Those with atrial thrombi should get warfarin if they are candidates for anticoagulation. If not, they should receive aspirin. The Assessment of Cardioversion Using
Transesophageal Echocardiography (ACUTE) Pilot Study suggested that transesophageal echocardiography (guided cardioversion with short term anticoagulation) was a safe means of providing cardioversion earlier than would be possible with conventional therapy (Klein, 1997). Although it is routinely suggested that persons with atrial fibrillation of recent onset (less than 48 hours) can be cardioverted without anticoagulation, emboli have been seen in patients cardioverted from atrial fibrillation of only a few days duration (Arnold, 1992). However, a recent study suggests that in patients who are clinically estimated to have had atrial fibrillation for less than 48 hours, there is a low likelihood of thromboembolism related to cardioversion (Weigner, 1997) (Indicators 7 and 8).

Monitoring anticoagulation

Patterns of monitoring warfarin are highly variable, and it is not known how frequently the INR must be monitored to attain stable control. Patients with atrial fibrillation newly started on warfarin should have an INR checked within one week (Errichetti, 1984). Most patients are followed at intervals ranging from two to five weeks. Patients with a stable INR can be followed at intervals of up to eight to ten weeks (Fihn, 1994). However, frequent monitoring may not always be necessary; in one study, 89 percent of patients required no change in warfarin dose on more than 50 percent of visits (Errichetti, 1984). Therefore, our indicators on monitoring anticoagulation focus on initiation of warfarin or changing warfarin in dose (Indicators 9 and 10).
REFERENCES


Prystowsky EN, et al. 1996. Management of patients with atrial fibrillation: a statement for healthcare professionals from the


## RECOMMENDED QUALITY INDICATORS FOR ATRIAL FIBRILLATION

These indicators apply to men and women age 18 and older who have atrial fibrillation.

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Quality of Evidence</th>
<th>Literature</th>
<th>Benefits</th>
<th>Comments</th>
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<tr>
<td><strong>Diagnosis</strong></td>
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<tr>
<td>1. Patients presenting with new-onset atrial fibrillation or atrial fibrillation of unknown duration should have documentation of the following in the medical record at the time of presentation: a. alcohol use; and b. stimulant drug use.</td>
<td>III</td>
<td>Kostinen, 1990; Lowenstein, 1992; Prystowsky, 1996</td>
<td>Guide management; decrease symptoms.</td>
<td>No controlled trials directly evaluate the elements of quality in the history and physical for persons in atrial fibrillation. Identification of causes of isolated atrial fibrillation may eliminate the need for further evaluation. There is no evidence in support of the suggested time period.</td>
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<td>2. Patients presenting with new-onset atrial fibrillation or atrial fibrillation of unknown duration should have a thyroid stimulating hormone (TSH) level checked within the first week of presentation.</td>
<td>III</td>
<td>Forfar, 1979; Woeber, 1992; Prystowsky, 1996</td>
<td>Guide management; decrease symptoms.</td>
<td>Atrial fibrillation is found in 9-22% of patients with thyrotoxicosis, a potentially transient and reversible cause of atrial fibrillation. There is no evidence in support of the suggested time period.</td>
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<tr>
<td>3. Patients presenting with new-onset atrial fibrillation or atrial fibrillation of unknown duration should have a chest radiograph performed in the first 24 hours after presentation.</td>
<td>III</td>
<td>Prystowsky, 1996</td>
<td>Guide management; decrease symptoms.</td>
<td>Chest x-ray assists further evaluation and treatment. No clinical trials directly assess the role of the chest radiograph in the evaluation of new onset atrial fibrillation. There is no evidence in support of the suggested time period.</td>
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<tr>
<td>Treatment</td>
<td>Indicator</td>
<td>Quality of Evidence</td>
<td>Literature</td>
<td>Benefits</td>
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<td>4.</td>
<td>Patients with atrial fibrillation of greater than 48 hours duration who do not have contraindications to warfarin&lt;sup&gt;3&lt;/sup&gt; should receive warfarin if they are:</td>
<td>I, II-1, II-2, III</td>
<td>Petersen, 1989; BATAAF, 1990; Connolly, 1991; SPAF, 1991; Wolf, 1991; Ezekowitz, 1992; SPAF, 1992a; SPAF, 1992b; EAFT, 1993; Atrial Fibrillation Investigators, 1994; SPAF II, 1994; Laupacis, 1995; SPAF, 1996</td>
<td>Prevent stroke.</td>
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<td></td>
<td>a. under 65 with one or more other risk factors for stroke;&lt;sup&gt;2&lt;/sup&gt; or;</td>
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<td>b. 65 years of age or older.</td>
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<td>5.</td>
<td>Patients with chronic atrial fibrillation&lt;sup&gt;4&lt;/sup&gt; who have contraindications to warfarin or have declined warfarin therapy should receive aspirin if they are:</td>
<td>I, II-1, III</td>
<td>Petersen, 1989; Atrial Fibrillation Investigators, 1994; SPAF II, 1994; Laupacis, 1995</td>
<td>Prevent stroke.</td>
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<tr>
<td></td>
<td>a. under 65 with one or more other risk factors for stroke;&lt;sup&gt;2&lt;/sup&gt; or,</td>
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<td>b. age 65 years or older.</td>
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<td>6.</td>
<td>Patients with atrial fibrillation who do not have contraindications to warfarin should be started on warfarin within one month of presenting with either of the following:</td>
<td>I</td>
<td>SPAF, 1992b; EAFT, 1993; Atrial Fibrillation Investigators, 1994;</td>
<td>Prevent stroke.</td>
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<td></td>
<td>a. new onset ischemic or embolic stroke; or</td>
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<td></td>
<td>b. new onset transient ischemic attack.</td>
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<td>7.</td>
<td>Patients with atrial fibrillation of greater than 48 hours duration who are undergoing elective electrical or chemical cardioversion should receive anticoagulation for at least 3 weeks prior to cardioversion.</td>
<td>II-2, III</td>
<td>Bjerkelund, 1969; Laupacis, 1995</td>
<td>Prevent stroke.</td>
</tr>
<tr>
<td>Indicator</td>
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<td>8. All patients with atrial fibrillation should receive anticoagulation for at least 4 weeks after cardioversion unless there are contraindications to anticoagulation.(^2)</td>
<td>III</td>
<td>Bjerkelund, 1969; Laupacis, 1995</td>
<td>Prevent stroke.</td>
<td>In the postcardioversion period, forceful atrial and atrial appendage contractions may not resume until 2-3 weeks after sinus rhythm has returned. Anticoagulation prevents the formation of fresh thrombus in the left atrial appendage after cardioversion if the resumption of atrial contraction is delayed and/or if atrial fibrillation recurs after successful cardioversion.</td>
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<td>Follow-up</td>
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<td>9. Patients with atrial fibrillation started on warfarin should have an INR checked within 1 week of the first dose.</td>
<td>III</td>
<td>Errichetti, 1984; Fihn, 1994</td>
<td>Prevent stroke; prevent hemorrhage.</td>
<td>There is no evidence in support of the suggested time period. Timing of follow-up of INRs is rather arbitrary. Studies suggest that frequent monitoring is not always necessary, and that many visits and INR measurements were not associated with a change in warfarin dosage.</td>
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<tr>
<td>10. Patients on warfarin should have an INR checked a minimum of every three months.</td>
<td>III</td>
<td>Errichetti, 1984; Fihn, 1994</td>
<td>Prevent stroke; prevent hemorrhage.</td>
<td>There is no evidence from RCTs in support of the suggested time period, but there is some expert consensus.</td>
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</tbody>
</table>

**Definitions and Examples**

1. New onset atrial fibrillation is defined as the first presentation of atrial fibrillation of known duration less than 48 hours.
2. High risk for stroke includes one or more of the following risk factors (Atrial Fibrillation Investigators, 1994):
   a. Prior stroke
   b. Diabetes mellitus
   c. Hypertension - any of the following:
      - At least three measurements on different days with a mean SBP>140 mm Hg and/or a mean DBP>90 mm Hg documented in the medical record
      - A diagnosis of hypertension mentioned in the chart
      - Documentation of chronic antihypertensive therapy
   d. Age >65 years old
   e. Heart failure — any charted diagnosis of heart failure
   f. Clinical coronary heart disease (angina or myocardial infarction mentioned in the chart)
   g. Mitral stenosis
   h. Prosthetic heart valves
   i. Echo criteria:
      - Left atrial enlargement (>4.5 cm)
   j. Impaired left ventricular function (ejection fraction <50% or LV dyskinesis, hypokinesis, or akinesis)
3 Potential contraindications to warfarin: These may be either specifically documented in the medical record or may use clinician assessment of the contraindication:
   a. Previous hemorrhagic stroke at any time or non-hemorrhagic stroke within 1 month
   b. Known intracranial neoplasm, mass, or other intracerebral pathology (e.g. aneurysm, abscess)
   c. Bleeding within the past 4 weeks (including gastrointestinal bleeding, melena, epistaxis, any bleeding requiring transfusion; excluding menses and occult hemoglobin in stools)
   d. Suspected aortic dissection
   e. Bleeding diathesis (e.g. dysfunctional platelets, von Willebrand’s disease, thrombocytopenia, clotting factor deficiency, hemophilia)
   f. Pregnancy
   g. Allergy/hypersensitivity to warfarin
   h. Notation of frequent falls in the medical record
   i. Concern about the patient’s mental status and ability to adhere to the regimen and follow-up
   j. Any mention of contraindications to anticoagulation in the medical record

4 Chronic atrial fibrillation: Atrial fibrillation of greater than 48 hour duration or of unknown duration.

Quality of Evidence Codes

I Randomized Controlled Trial (RCT)
II-1 Nonrandomized controlled trials
II-2 Cohort or case analysis
II-3 Multiple time series
III Opinions or descriptive studies