LTC Information Series

Atrial Fibrillation

in the Long Term Care Setting

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Atrial Fibrillation

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Definitions

Atrial fibrillation (AF) is a supraventricular tachyarrhythmia characterized by rapid, disorganized atrial depolarizations that result in a loss of atrial contraction. An electrocardiogram will reveal replacement of regular P waves with oscillations or fibrillatory waves that vary in size, shape, and rate.

AF is characterized clinically according to its duration and frequency. **Paroxysmal** AF occurs intermittently and terminates spontaneously. **Recurrent** AF is paroxysmal AF that recurs three or more times. **Persistent** AF is AF that persists for more than 7 days. **Permanent** AF is persistent AF that cannot be cardioverted to sinus rhythm. **Secondary** AF is AF associated with an acute medical illness (e.g., hyperthyroidism, myocardial infarction, pericarditis, pneumonia, pulmonary embolus) or that occurs following cardiac, esophageal, or pulmonary surgery. **Lone** AF is AF that occurs in a patient under age 60 without evidence of hypertension, other cardiopulmonary diseases, or echocardiographic evidence of structural or functional abnormalities.[1,2]

Introduction

SCOPE OF THE PROBLEM

AF is a serious and increasingly prevalent health problem in an aging U.S. population. It is the most common arrhythmia requiring hospital admission and is associated with increasing health care costs. AF negatively impacts quality of life and increases both morbidity and mortality.

A 2001 study estimated that 2.5 million people in the United States had AF[3]; that number is expected to increase to 5.6 million by 2050.[4,5] The lifetime risk of developing AF is 1 in 4.[6] The prevalence of AF increases steadily with age, from 0.5% of those aged 50 to 59 to 8.8% of those aged 80 to 89.[6] Thirty-six percent of all patients with AF are aged over 80; 25% of patients with AF also have congestive heart failure (CHF).[5]

AF causes more hospitalizations than any other arrhythmia, accounting for nearly one-third of all hospitalizations for cardiac rhythm disturbances. The mean cost of an AF-related hospitalization exceeds $8,000; the mean length of stay is about 3.5 days.[7] One study estimated the annual cost to Medicare of treating patients with newly diagnosed AF at $15.7 billion.[8] Patients aged 65 to 74 account for 24% of all AF costs, while those 75 and older account for 53% of all AF costs.[9]

With a rapidly aging population, a high prevalence of predisposing risk factors for AF, and improved survival rates for patients with cardiovascular conditions, the prevalence of AF in the long-term care (LTC) setting can be expected to increase. Members of the LTC interdisciplinary team need to understand the importance of AF and contribute to an individualized management plan to reduce complications and optimize quality of life for patients with AF.

HEALTH CONSEQUENCES OF ATRIAL FIBRILLATION

AF can exacerbate chronic conditions, cause cardiac damage, and adversely impact quality of life. It doubles the risk for dementia,[10,11] triples the risk of CHF,[12] quintuples the risk of stroke,[12,13] and increases all-cause mortality by 50% in men and 90% in women.[12,14] The increase in mortality associated with AF is independent of pre-existing cardiac conditions.[14]

The loss of atrial contraction and the occurrence of rapid ventricular rates associated with AF can reduce cardiac output, which may then lead to symptoms such as angina, dizziness, edema, exercise intolerance, falls, fatigue, shortness of breath, and even loss of consciousness.

AF negatively affects quality of life. In a case-control study, patients without AF had a higher quality of life than those with asymptomatic AF, while those with symptomatic AF had the lowest quality-of-life scores.[15] Studies of both rate and rhythm control for AF have shown statistically similar improvements in

quality of life with treatment.[16] In some studies, patients who were in sinus rhythm when surveyed had the best quality of life.[16,17] When all studies are viewed together, however, no clear difference in overall quality of life emerges between groups assigned to rhythm or rate control strategies.[16,17] For patients with AF in the LTC setting, important treatment outcomes related to quality of life include avoiding hospitalization, controlling symptoms, optimizing functional independence, and remaining active.

For patients with chronic cardiac conditions such as CHF or ischemic heart disease, the loss of atrial contraction or the occurrence of a tachyarrhythmia may cause an exacerbation of previously controlled symptoms. If AF with a rapid ventricular rate (greater than or equal to 130) persists without controlling the heart rate or restoring sinus rhythm, a patient may develop tachycardia-induced cardiomyopathy. Myocardial damage may improve with ventricular rate control, but some damage may be irreversible.[1]

Ischemic stroke is perhaps the most serious possible consequence of AF and the one with the greatest impact on quality of life. AF increases the risk of stroke 4 to 5 fold and is responsible for 15% of ischemic strokes.[18] This is of particular concern in LTC settings because stroke risk increases with age, rising from 1.5% in people aged 50 to 59 to 24% in those aged 80 to 89.[18] The attributable risk for stroke from AF increases with age, from approximately 10% for patients aged 70 to 79 to approximately 23% for those aged 80 to 89.[19] After an initial diagnosis of AF, the incidence of a new stroke rises with advancing age from 0.7 per 100 patient-years at ages 40 to 59 to approximately 2 per 100 patient-years at ages 60 to 69 and 5.3 per 100 patient-years at age 80 or above.[20]

Recognition

Practitioners should suspect AF if they detect an irregular or rapid heart rate. AF may occur without symptoms or may be found while evaluating a patient with the symptoms or clinical presentations listed in Table 1. (Also see Appendix 1.) If a patient does not have a known diagnosis of AF, an electrocardiogram should confirm the clinical suspicion.

TABLE 1. Common Signs and Symptoms of Atrial Fibrillation

Chest pain or discomfort
Dizziness or lightheadedness
Feeling of overall weakness
Hypotension
Irregular heart rate
New or worsening edema
Palpitations
Rapid heart rate
Shortness of breath or difficulty breathing
Tiredness or fatigue

Adapted from: *Padanilam et al, 2008;*[21] *Fuster et al, 2006*[1]

CAUSES OF ATRIAL FIBRILLATION

Once AF has been recognized, it is important to determine the possible precipitating cause(s). AF may occur as a result of the long-term impact of known risk factors (Table 2). It may also be precipitated or

exacerbated by a number of acute conditions, uncontrolled chronic conditions (Table 3), or adverse medication effects (Table 4). Thus, an important element of the evaluation of a patient with newly diagnosed AF is to consider these precipitating factors and alleviate them if possible.

TABLE 2. Risk Factors for Atrial Fibrillation

- Advancing age
- Congenital heart disease
- Coronary artery disease
- Hyperglycemia
- Hypertension
- Male gender
- Obesity
- Obstructive sleep apnea
- Valvular heart disease

Adapted from: *Padanilam et al, 2008;*[21] *Fuster et al, 2006*[1]

TABLE 3. Conditions That May Precipitate or Exacerbate Atrial Fibrillation

Cardiac
Cardiomyopathy
Dilation or hypertrophy of atria or ventricles
Heart failure
Hypertension
Myocardial ischemia/infarction
Myocarditis
Pericarditis
Valvular heart disease

Pulmonary
Cor pulmonale
Pneumonia
Pulmonary embolism
Pulmonary hypertension
Sleep apnea

Systemic/metabolic
Electrolyte abnormalities
Fever
Hyperthyroidism
Obesity
Recent heavy alcohol use
Strenuous exercise
Systemic infection
Volume overload

Postoperative
Cardiac, pulmonary or esophageal surgery

Neurological
Increased parasympathetic tone
Increased sympathetic tone
Stroke or subarachnoid hemorrhage

Medications (see Table 4)

Adapted from: *Padanilam et al, 2008;*[21] *Fuster et al, 2006*[1]

TABLE 4. Medications Reported to Potentially Induce Atrial Fibrillation

DRUG CLASS	AGENTS	MECHANISM
Cardiovascular		
Vasodilators	Isosorbide mononitrate	Hypotension —> Adrenergic reflex
Antiarrhythmics	Verapamil, diltiazem, digoxin, atenolol	Changing atrial electrical properties
Diuretics	Thiazides	Hypokalemia
Respiratory System		
Alpha agonists	Pseudoephedrine	Adrenergic stimulation
Beta agonists	Albuterol	Adrenergic stimulation
Xanthines	Theophylline	Adrenergic stimulation
Corticosteroids	Methylprednisolone (high dose)	Local potassium efflux
Central Nervous System		
Cholinergics	Cholinesterase inhibitors	Vagal stimulation
Anticholinergics	Atropine, ipratropium	Adrenergic stimulation
Dopamine agonist	Ropinirole	Vagal activity
Antidepressants	Fluoxetine, trazodone	Serotonin
Antipsychotics	Risperidone, quetiapine	Not reported
Antimigraine	Sumatriptan	Not reported
Genitourinary		
Medications for erectile dysfunction	Sildenafil	Hypotension —> Adrenergic reflex
Miscellaneous		
Antithrombotic agents	Anagrelide, clopidogrel	Not reported
Bisphosphonates	Zoledronic acid	Not reported
Other	Niacin, calcium, nicotine, etanercept, azathioprine	Not reported

Adapted from: *van der Hooft et al*, 2004[22]

Special Considerations When Assessing and Treating Atrial Fibrillation in the Long-Term Care Setting

Practitioners should share decisions about diagnostic evaluations and therapeutic interventions with patients or their surrogate decision makers after they review the potential benefits and burdens of relevant tests and treatments. Patient characteristics to consider in the decision include functional status, presence of comorbid conditions, prior stated or recorded wishes about goals of care, and prognosis. Practitioners should avoid ordering testing that would not change the management course or recommending treatments that the patient would refuse. When a patient refuses testing or treatment, it is prudent to document the patient's reasoning and the decisions reached.

All patients in the LTC setting should be counseled about end-of-life planning and encouraged to designate a durable power of attorney for health care or a substitute decision maker. It is also important to help patients make informed decisions and clarify their preferences for cardiopulmonary resuscitation and other intensive or invasive treatments. Patients should consider completing a formal advance directive (e.g., living will, Physician Orders for Life-Sustaining Treatment form). Documentation of patients' goals of care and choices about interventions such as cardiopulmonary resuscitation, hospitalization, intensive care unit treatments, and tube feeding is intended to assure that their wishes are respected if they lose decisional capacity. Documentation of goals of care can provide helpful guidance for families and others involved in substitute decision making.

Assessment

When a patient is found to have AF, the practitioner must first determine whether the patient shows any evidence of hemodynamic instability (e.g., hypotension, signs or symptoms of shock, symptomatic tachycardia). Unstable patients should generally receive emergent evaluation and treatment in a hospital setting. They may be appropriate candidates for intravenous medications or electrical cardioversion to control heart rhythm and rate.[21]

If the patient is hemodynamically stable, the practitioner should next review the patient's history and medications to search for medical conditions or medications that could have precipitated or exacerbated AF. In general, detection of precipitating factors should focus acute treatment on amelioration of those factors, followed by reassessment to determine whether AF terminates or persists.[21] The physical examination should focus on signs of underlying heart disease or complications related to heart disease and arrhythmia.

LABORATORY EVALUATION

Next, the practitioner can decide on laboratory testing and cardiac evaluations. Obviously, an electrocardiogram can confirm the presence of AF, quantify the heart rate, detect acute ischemia or infarction, and document the presence of conduction abnormalities or left ventricular hypertrophy. A chest x-ray can help to detect pulmonary disease, quantify cardiac size and assess pulmonary congestion. An echocardiogram will quantify left ventricular function and may detect valvular abnormalities, ventricular hypertrophy, or evidence of pericardial effusion. Blood tests that may be helpful for identifying precipitating factors for AF include thyroid-stimulating hormone to detect hyperthyroidism or hypothyroidism, a complete blood count (CBC) to detect anemia or polycythemia, and a comprehensive metabolic panel to search for electrolyte abnormalities or evidence of renal or hepatic dysfunction.[1]

In selected individuals, more extensive evaluation with a cardiac stress test or cardiac consultation may be appropriate. Consider cardiac consultation when the diagnosis is unclear, when patients remain symptomatic despite rate control, when patients have complex arrhythmias, or when multiple comorbid conditions complicate treatment.

Treatment

In the LTC setting, patients with paroxysmal AF are treated identically to patients with persistent AF: precipitating factors should be alleviated when possible and tachycardia should be controlled. For recurrent paroxysmal AF and persistent AF, the practitioner must determine the most appropriate management goals (Table 5). These will be influenced by the patient's comorbid conditions, prognosis and overall goals of care, symptom severity, and treatment preferences.[1,21]

TABLE 5. Goals for Management of Atrial Fibrillation

The patient and practitioner may decide to pursue one or all of these goals. Goals may be pursued simultaneously as appropriate.

- Prevention of stroke and systemic thromboembolism
- Prevention of tachycardia-induced cardiomyopathy
- Control of symptoms
- Restoration and maintenance of sinus rhythm

Adapted from: *Padanilam et al, 2008;*[21] *Fuster et al, 2006*[1]

STROKE PREVENTION

For patients with AF, anticoagulation with warfarin reduces the occurrence of stroke[18] and decreases the likelihood of severe disability among those who suffer stroke while taking warfarin.[23] Decisions about a patient's need for anticoagulation should be based on his or her stroke risk and not on whether sinus rhythm has been temporarily restored.[1] Anticoagulation with a vitamin K antagonist (warfarin) to reduce the risk of stroke should be considered regardless of whether AF is persistent or paroxysmal or whether a rhythm-control or rate-control strategy is pursued.[1]

Warfarin therapy is the current standard antithrombotic treatment for AF. Research data are evolving, however, for new anticoagulants such as direct thrombin inhibitors (e.g., dabigatran, argatroban) and direct Factor Xa inhibitors (e.g., rivaroxaban, apixaban). Because these new agents do not eliminate the risk of serious bleeding events, practitioners and patients will continue to be challenged with weighing the risks and benefits of anticoagulation for stroke prevention.

Shared Decision Making About Anticoagulant Therapy

The therapeutic dilemma for practitioners in the LTC setting is that the patients who have the highest risk for stroke obtain the most benefit from warfarin therapy but also suffer the most complications from it.[18] Practitioners often hesitate to prescribe warfarin to patients of advanced age and frailty.[24] Studies suggest that practitioners' decisions are influenced more heavily by concerns about warfarin complications than by the potential benefits of preventing stroke or venous thromboembolism (VTE).[24] When patients are included in risk-benefit discussions about warfarin use, they may reach a decision that differs from that of the practitioner. For this reason, it is prudent to inform patients about the benefits and burdens of warfarin therapy and reach a shared decision about this medication's use.

All recommendations for prophylactic warfarin use in patients in the LTC setting assume that the practitioner has access to a system to monitor the patient's international normalization ratio (INR) and maintain it in the ideal range. These recommendations also assume that the practitioner has assessed the patient's bleeding risk, functional status, and prognosis. Examples of decision guides for assessing the benefits and

risks of warfarin therapy, flow sheets for INR monitoring, and policies and algorithms for warfarin initiation and INR-based dosage adjustment can be found in Appendixes 2–6.

Risk/Benefit Assessment

It is helpful to use a stepwise approach to determine the risks and benefits of warfarin for individual patients. Algorithmic, evidence-based approaches help to avoid the influence of common errors of reasoning caused by cognitive biases and the inability of simple heuristics to capture important elements of complex decisions.[25]

The Antithrombotic Baseline Risk and Benefit Assessment Tool (see Appendix 2) can provide meaningful risk and benefit estimates for such a decision. The patient and practitioner can refer to these data when they discuss the relative value of reducing the risk of VTE or cardioembolic stroke with warfarin versus increasing the risk of bleeding complications.

1. Assess the likelihood of meaningful benefit from anticoagulation to reduce stroke risk.

Practitioners should first determine whether anticoagulation can improve the quality or length of life for a patient with AF. If the patient has advanced disease or a poor short-term prognosis, anticoagulant therapy may not provide a meaningful benefit. For those receiving anticoagulant therapy, it is prudent to reassess the benefits and risks periodically as well as any time a major change in clinical status occurs.

2. Estimate the patient's risk of stroke without anticoagulant therapy.

The CHADS-2 Instrument (Table 6) is a practical tool for estimating stroke risk for patients with persistent or paroxysmal AF, thus identifying those patients for whom the benefits of anticoagulation to reduce stroke risk are likely to outweigh the risks of treatment with anticoagulants.[26] Note that even if sinus rhythm is restored, the need for long-term anticoagulant therapy is based on stroke risk factors.[27,28] The American College of Chest Physicians (ACCP) guideline recommends warfarin therapy on the basis of CHADS-2 score.[18] The American College of Cardiology uses clinical characteristics, rather than the CHADS-2, to stratify risk and make recommendations for warfarin therapy (Table 7).[1]

TABLE 6. Risk of Stroke for Patients With Atrial Fibrillation: CHADS-2 Instrument

CHADS-2 Score	Adjusted stroke rate per 100 person-years [95% confidence interval]	Comments
0	1.9 [1.2–3.0]	
1	2.8 [2.0–3.8]	
2	4.0 [3.1–5.1]	The adjusted stroke rate is the expected stroke rate per 100 person-years. The model assumes that patients did not take aspirin.
3	5.9 [4.6–7.3]	
4	8.5 [6.3–11.1]	
5	12.5 [8.2–17.5]	
6	18.2 [10.5–27.4]	

Calculation of CHADS-2 score:

- Add 1 point for each of the following conditions:·
  - Age 75 or older
  - Diabetes mellitus
  - Hypertension
  - Moderately or severely impaired LV systolic function and/or CHF
- Add 2 points for a prior stroke or TIA

CHF: congestive heart failure; TIA: transient ischemic attack

Adapted from: *Gage et al,* 2001[26]

TABLE 7. Antithrombotic Therapy for Patients With Atrial Fibrillation: American College of Cardiology Recommendations

Risk category	Recommended therapy
No risk factors	Aspirin 81–325 mg/d
One moderate-risk factor	Aspirin 81–325 mg/d, or warfarin
Any high-risk factor or more than one moderate-risk factor	Warfarin

Less-validated or weak risk factors	*Moderate-risk factors*	*High-risk factors*
• Age 65 to 74 years • Coronary artery disease • Female gender • Thyrotoxicosis	• Age 75 or older • Diabetes mellitus • Heart failure • Hypertension • Left ventricular ejection fraction 35% or less	• Mitral stenosis • Previous stroke, TIA or embolism • Prosthetic heart valve

Source: *Fuster et al,* 2006[1]

3. Estimate the reduction in stroke risk with anticoagulant therapy.

Anticoagulation with an oral vitamin K antagonist (VKA) such as warfarin decreases by approximately 65% the risk of stroke caused by AF.[18] This relative risk reduction produces an increasing absolute reduction in stroke occurrence as baseline stroke risk increases. For example, a patient aged 75 with a history of transient ischemic attack (TIA) or stroke (CHADS-2 score of 3) has an annual stroke risk of 6% or more. For such patients, the number needed to treat (NNT) is 25; that is, treating 25 similar patients with warfarin can be expected to prevent one stroke.[1] By comparison, a patient with a CHADS-2 score of 0 (i.e., aged over 75 with no history of diabetes, hypertension, stroke, TIA, or ventricular dysfunction) has an annual stroke risk of 2% and the NNT rises to 100.[1] Even if a stroke occurs despite warfarin therapy, if the INR is in the therapeutic range at the time, the stroke is likely to be less severe.[23]

4. Assess absolute and relative contraindications to anticoagulant therapy.[24]

Absolute contraindications to warfarin include

- Blood pressure readings consistently greater than 160/90
- Current active bleeding
- Platelet count below 50,000
- Nonadherence with medication administration or INR monitoring

Relative contraindications include

- Consumption of 2 or more ounces of alcohol per day
- Extreme functional disability
- Poor short-term prognosis or advanced chronic disease
- Use of a nonsteroidal anti-inflammatory drug (NSAID) or aspirin without gastric cytoprotection

Data do not support the common practice of withholding warfarin from elderly patients with frequent falls or with risk factors for falling.[29,30] It is true that 70% of elderly patients with subdural hematoma (SDH) have a history of falls; the net increase in SDH, however, is one or two occurrences per 10,000 patients who fall.[30]

5. Assess active or suspected bleeding and risk factors for bleeding.

The Antithrombotic Baseline Risk and Benefit Assessment Tool (see Appendix 2) lists baseline rates of serious bleeding events with and without aspirin or warfarin. These data highlight the fact that serious bleeding can and does occur in the absence of antithrombotic therapy.

The increase in bleeding risk with warfarin therapy is relative and may be outweighed by the reduction in stroke risk. For example, among patients with AF, the rate of hemorrhage requiring hospitalization or transfusion is approximately 13 per 1,000 person-years for patients taking warfarin, compared with 7 per 1,000 person-years for those on placebo.[31] The reduction in stroke risk as a result of warfarin therapy, however, far outweighs the increased risk of bleeding.

6. Perform a baseline laboratory assessment.

Laboratory testing can identify many risk factors for bleeding and signs of occult bleeding. It is prudent to obtain the following tests as part of the initial assessment to determine the patient's suitability for antithrombotic therapy:

- Blood glucose values
- Complete blood count (CBC)
- Fecal occult blood
- International Normalization Ratio (INR)
- Renal and hepatic function
- Urinalysis

Although many risk factors for bleeding (e.g., age, chronic renal disease, diabetes) cannot be modified, practitioners may decide that high-risk patients should be monitored more closely (e.g., fecal occult blood, hemoglobin) while receiving antithrombotic therapy.

7. Assess for modifiable risk factors for bleeding complications and create a plan to reduce modifiable risks.

The practitioner should review the patient's medications and medical conditions to seek opportunities to reduce the risk of bleeding. Some evidence suggests that the risks of bleeding while taking warfarin may be reduced by controlling blood pressure, limiting alcohol consumption, prescribing a medication for gastric cytoprotection (i.e., misoprostol or a proton pump inhibitor) in patients who are taking aspirin or an NSAID, and treating *Helicobacter pylori* infection.[24]

Clinicians' and patients' greatest fears regarding the potential adverse effects of warfarin are intracerebral hemorrhage and subdural hematoma (SDH). Intracerebral bleeding can cause severe disability and death. The rate of intracerebral bleeding among patients with AF who are receiving placebo is 1 per 1,000 patients per year, compared with 3 per 1,000 patients per year among those receiving warfarin, an absolute increase of 2 patients per 1,000.[31] Keeping blood pressure below 160/90 and the INR below 3 can reduce the risk of intracerebral bleeding. A comprehensive evaluation for fall risk factors, with appropriate interventions, may reduce the occurrence of falls.

As the Antithrombotic Baseline Risk and Benefit Assessment Tool (see Appendix 2) shows, SDH occurs in community-dwelling elderly patients who are not taking warfarin at a rate of 4 per 10,000 patient-years. For patients taking aspirin, the rate increases to approximately 8 per 10,000 patient-years. Warfarin increases the SDH rate to 12 per 10,000 patient-years. Seventy percent of SDHs are related to head trauma and 50% of head trauma incidents are caused by falls. Thus, 35% of all SDHs in community-dwelling elderly patients can be directly attributed to falls.[30]

A decision analysis estimating the risks and benefits of warfarin for stroke prevention in elderly patients with AF concluded that falls increase the risk of SDH by 1 to 2 additional cases per 10,000 patients. The authors calculated that elderly patients with AF who took warfarin would have to fall 295 times in 1 year before the quality-of-life benefits of warfarin therapy failed to exceed those of aspirin or no therapy. This analysis, which used the best available evidence, shows that the common practice of withholding warfarin from patients who fall relatively infrequently may deprive them of quality and length of life. Thus, falls alone should not be a major factor in the decision to withhold warfarin from elderly patients with AF.[30] In addition, it is possible to reduce the risk of falling by optimizing physical and cognitive function and by reducing or eliminating medications associated with falls.

8. Review the expected benefits and potential adverse effects of antithrombotic therapy.

The practitioner should review with the patient the data obtained from the patient's history, the risk assessment tool, and laboratory testing, as well as plans for reducing the risk of bleeding and of falls. The practitioner can then respond to the patient's questions and discuss the patient's perspective on the risks and benefits of choosing or foregoing warfarin therapy.

9. Consult guidelines to identify the optimal antithrombotic therapy based on the patient's stroke risk.

Patients who have not had a peripheral embolism, stroke, or TIA but who have AF and a CHADS-2 score of 2 or more should receive lifelong VKA therapy (1A).[18] (See Appendix 7 for an explanation of grades of recommendations.) Patients with AF and only one risk factor may choose either lifelong VKA (1A) or aspirin 75-325 mg/d (1B), but the ACCP recommends VKA over aspirin (2A).[18] Patients with paroxysmal or persistent AF and a CHADS-2 score of 0 are at low risk of stroke and should take lifelong aspirin 75-325 mg/d (1B).[18] The ACCP recommends aspirin 75-100 mg/d because this dose range provides good efficacy with reasonable safety.[18] The American College of Cardiology makes similar recommendations based on clinical criteria for assessing stroke risk (see Table 7).[1]

The target INR for stroke prevention is 2.5 (range 2.0–3.0). Studies show that the preventive efficacy of warfarin is ideal when the INR is 2.0; no further risk reduction is seen with INRs above 2.0. Conversely, an INR of 2.0 does not offer a decrease in bleeding risk compared with INRs of 2.1 to 3.0,[32] but stroke risk rises dramatically when the INR is less than or equal to 1.8.[33]

Aspirin, although offered as an alternative for patients with AF and low stroke risk, may have little or no demonstrable effect at reducing stroke caused by AF.[18] Meta-analyses of studies comparing aspirin to placebo report wide confidence intervals that include the possibility of no benefit from aspirin compared with placebo.[18] On the basis of data from studies comparing warfarin to aspirin, the NNT to prevent one stroke using warfarin rather than aspirin is estimated at 23.[18]

Safe and Effective Anticoagulant Therapy

1. Assure access to a system of care that optimizes INR control.

Use of warfarin assumes that the patient will be monitored closely so that the INR can be kept in the therapeutic range. This may involve the use of an anticoagulation management service, consultation with an experienced practitioner, or the use of evidence-based guides to adjust INR (see Appendix 6). Keeping INR values within the range of 2.0 to 3.0 may reduce bleeding risk; most bleeding events are associated with INR values above 3.0. Frequent INR monitoring (e.g., 2 to 3 times weekly) is especially important when initiating warfarin therapy because bleeding risk varies over time. The risk of major bleeding is 3% during the first month of therapy, decreases to 0.8% per month in the subsequent 11 months, and stabilizes at 0.3% per month thereafter.[31]

2. Implement a plan to monitor for bleeding.

Monitoring for adverse effects of warfarin may include periodic testing of urine and stool for occult bleeding and checking hemoglobin for blood loss.

3. Utilize facility policies for safe and effective anticoagulant administration.

Facilities should consider implementing a policy that requires specific information to be documented before warfarin therapy is initiated. At a minimum, the practitioner should specify, and the nurse or practitioner record, the following information:

- An appropriate diagnosis for warfarin use
- INR goal and range (e.g., INR goal 2.5, range 2.0–3.0)
- Duration of warfarin therapy, with a specific stop date, if appropriate
- INR monitoring frequency

- INR notification parameters (e.g., notify practitioner if INR is below 2.0 or above 3.0)
- Baseline laboratory assessment as appropriate (e.g., CBC, comprehensive metabolic panel, INR, stool guaiac, urinalysis)
- Ongoing laboratory monitoring as appropriate (e.g., monthly stool guaiac, CBC)

For optimal stroke risk reduction, warfarin's benefits should be combined with smoking cessation, blood pressure control, and lipid-lowering therapy because 20% of ischemic strokes are caused by atherothrombosis rather than by cardioembolism due to AF. (See Appendix 8, Modifiable Stroke Risk Factors: Interventions, Treatment Goals, and Strategies for Monitoring Adverse Drug Effects.)

For a summary of steps in the safe and appropriate management of anticoagulant therapy in elderly patients, see Table 8. Elements of a quality assurance program for managing AF are presented in Appendix 9. Appendix 10 provides suggested information about AF to be shared with patients and family members.

TABLE 8. Steps in the Safe and Appropriate Management of Anticoagulant Therapy in Elderly Patients

Step	Task(s)
	Quantifying Risks and Benefits of Anticoagulant Therapy
1	Assess whether a patient is likely to receive meaningful benefit from the stroke risk reduction due to anticoagulant therapy. Review continued appropriateness of anticoagulant therapy periodically and with major changes in clinical status.
2	Estimate the patient's risk of a stroke without anticoagulant treatment.*
3	Estimate the reduction in the risk of stroke with effective anticoagulant treatment.*
4	Review the patient's history for absolute and relative contraindications to therapy.*
5	Assess the patient for active or suspected bleeding and risk factors for bleeding.
6	Perform a baseline laboratory evaluation if one has not recently been completed (e.g., CBC, creatinine, glucose, INR, fecal occult blood test, urinalysis for blood; consider optional stool test for *Helicobacter pylori* antigen).
7	Assess the patient for modifiable risk factors that increase the risk of bleeding during antithrombotic therapy (e.g., NSAIDs, antiplatelet therapy, *H. pylori*-associated ulcer).
8	Create a plan to reduce modifiable risk factors (e.g., stop NSAIDs, treat *H. pylori* infection, prescribe proton pump inhibitor to reduce the risk of gastrointestinal bleeding caused by antiplatelet medication).
9	Review the benefits and potential adverse effects of antithrombotic therapy with the patient and family or surrogate decision maker and decide whether to begin therapy.
10	Consult appropriate guidelines to identify optimal antithrombotic therapy based on the patient's stroke risk.
	Safe and Effective Anticoagulant Therapy
1	Assure access to a system of care to optimize INR control.
2	Monitor the patient for bleeding complications of anticoagulant therapy (e.g., bleeding signs or symptoms, regular hemoglobin and/or fecal occult blood testing).
3	Utilize facility polices and procedures for safe and effective anticoagulant therapy.

CBC: complete blood count; INR: international normalized ratio;
NSAID: nonsteroidal anti-inflammatory drug
**See Appendix 2, Antithrombotic Baseline Risk and Benefit Assessment Tool*

PREVENTION OF TACHYCARDIA-INDUCED CARDIOMYOPATHY

AF may result in a rapid ventricular rate. When the ventricular rate is persistently at or above 130 beats per minute, the patient may develop tachycardia-induced cardiomyopathy. This dilated cardiomyopathy can be associated with a 50% decrease in ejection fraction. Control of the ventricular rate will usually produce a gradual return to the patient's baseline left ventricular function.[1]

Because patients may experience a rapid heart rate with activities of daily living, it is important to monitor heart rate both at rest and with exercise. Recommendations concerning ideal heart rate targets vary, but it is reasonable to maintain a resting heart rate of 60 to 80 beats per minute and a rate of 90 to 110 beats per minute with exercise.[1]

Studies comparing strict and more-lenient rate-control strategies found no differences in morbidity or mortality between patients with different levels of heart-rate control.[34-36] In general, these studies show that practitioners may accept heart rates of approximately 80 beats per minute at rest and less than 110 beats per minute after walking.[37]

The preferred agents for rate control are beta blockers (e.g., metoprolol, propranolol) and nondihydropyridine calcium-channel antagonists (e.g., diltiazem, verapamil). These agents may be used as monotherapy or combined to achieve the desired effect of rate control without excessive bradycardia. Beta blockers are superior to calcium-channel blockers for heart rate control in AF.[1] All beta blockers are effective at controlling ventricular rate. If a patient has inadequate rate control despite treatment with a beta blocker or nondihydropyridine calcium-channel antagonist, the addition of digoxin may exert a beneficial effect on ventricular rate. When combination therapy with a beta blocker, calcium-channel antagonist, and digoxin does not control rate, amiodarone is an effective alternative.

One beta blocker, sotalol, deserves special mention because of its potentially fatal proarrhythmic properties. The FDA black-box warning for sotalol requires that, for initiation, reinitiation, or titration of this agent, patients must undergo continuous electrocardiographic monitoring in a facility that can provide cardiac resuscitation until they have been on a maintenance dose for a minimum of 3 days.

The nondihydropyridine calcium-channel blockers verapamil and diltiazem control heart rate in patients with AF with equal efficacy.[1] Digoxin therapy is not a first-line monotherapy for rate control because of its potential toxicity in elderly patients and because it controls heart rate at rest but not with exercise.[1] Because digoxin exerts some beneficial effect on left ventricular function, it may be a reasonable choice for patients with left ventricular dysfunction or heart failure. For patients with chronic kidney disease, demonstrated by an estimated glomerular filtration rate (GFR) of less than 60, the maintenance dose of digoxin should be decreased by 25% to 75%. Coadministration of verapamil or amiodarone may raise digoxin levels and result in digoxin toxicity. Adverse cardiovascular effects of digoxin include ventricular arrhythmias, heart block and bradycardia. Noncardiac manifestations of digoxin toxicity in older adults include anorexia, confusion, diarrhea, nausea, and visual disturbances.

SYMPTOM CONTROL

Patients with AF may experience symptoms such as bradycardia, chest pain, dizziness, fatigue, palpitations, and tachycardia. In general, the initial approach to symptom control in older adults is to control the ventricular rate (Table 9).[1]

TABLE 9. Agents Used to Control Ventricular Rate

Drug Class/Agent	Maintenance Dose Range	Adverse Effects and Cautions
Beta blockers		
Metoprolol*	25–200 mg/d PO	Bradycardia, heart block, hypotension, exacerbation of asthma or heart failure
Propranolol	80–240 mg/d PO	
Nondihydropyridine calcium-channel blockers		
Diltiazem	120–360 mg/d PO	Hypotension, bradycardia, heart block, exacerbation of heart failure
Verapamil	120–360 mg/d PO	Hypotension, bradycardia, heart block, exacerbation of heart failure, interaction with digoxin
Other agents		
Digoxin	0.0625–0.375 mg/d PO	Digitalis toxicity, bradycardia, heart block. Reduce dose 25% to 75% if estimated GFR is less than 60. Exercise caution with coadministration of amiodarone and verapamil
Amiodarone	100–200 mg/d PO	Hypotension, bradycardia heart block, hyperthyroidism, hypothyroidism, pulmonary toxicity, skin discoloration, anorexia, nausea, fatigue, tremor, interaction with digoxin and warfarin

* Other beta blockers exert similar effects

GFR: glomerular filtration rate

HEART-RATE CONTROL VERSUS RHYTHM CONTROL

A treatment course that employs the use of cardioversion and antiarrhythmic drugs to restore and maintain sinus rhythm is referred to as a rhythm-control strategy. In contrast, a rate-control strategy does not try to restore sinus rhythm, but rather focuses on controlling ventricular rate at rest and with exercise.[38]

Five randomized controlled trials have found no important differences in outcome between rate control and pharmacological rhythm control.[27,28,39-41] The largest of these trials were the AFFIRM (Atrial Fibrillation Follow-up Investigation of Rhythm Management)[27,34] and RACE (Rate Control vs. Electrical Cardioversion for Persistent Atrial Fibrillation)[28] trials.

The AFFIRM trial[27,34] enrolled 4,060 subjects with paroxysmal and persistent AF. Subjects were randomized to receive rate control or antiarrhythmic drug therapy for rhythm control. All subjects were initially

anticoagulated but subjects in the rhythm-control group who were in sinus rhythm for at least 3 months did not have to continue warfarin. No differences in mortality or stroke rate were seen between subjects who underwent rhythm versus rate control. A trend was observed toward higher risk for ischemic stroke in the rhythm-control group, primarily in patients who did not receive adequate anticoagulation.

The RACE trial randomized 522 subjects who had persistent AF, despite previous electrical cardioversion, into rate- and rhythm-control groups.[28] All subjects received anticoagulation. After 2.3 years of follow-up, rate control was not inferior to rhythm control for prevention of death and morbidity.

AF management may be individualized depending on the severity of the patient's symptoms and underlying heart disease. The results from the AFFIRM and RACE trials are most applicable to elderly patients who have few or no AF symptoms. In this group of patients, anticoagulation and rate control may be the most appropriate approach. For younger, symptomatic patients who do not have underlying heart disease, restoration of sinus rhythm may be the best approach.[42] If rate control offers inadequate symptomatic relief, restoration of sinus rhythm should be considered.[1]

Rhythm Control

Restoring sinus rhythm improves cardiac hemodynamics and exercise tolerance. Symptoms of heart failure and overall quality of life may improve when an atrial contribution to cardiac output is maintained. Because AF contributes to pathologic atrial and ventricular remodeling, restoration of sinus rhythm may slow, and in some cases reverse, atrial dilatation and left-ventricular dysfunction.

Rhythm control can be achieved pharmacologically (chemical cardioversion; Table 10), electrically (electrical cardioversion), or surgically. Patient stability, the duration of AF, and patient preferences are important factors to consider when deciding between chemical and electrical cardioversion. Electrical cardioversion is usually preferred in unstable patients (e.g., those with hypotension, myocardial infarction, or pulmonary edema). Electrical cardioversion is also preferred in patients who have had an AF episode that persisted for more than 7 days, as chemical cardioversion is less effective in this situation.

TABLE 10. Pharmacologic Agents With Proven Efficacy for Cardioversion of Atrial Fibrillation

Amiodarone
Dofetilide
Flecainide
Ibutilide
Propafenone

Either chemical or electrical cardioversion may be utilized for rhythm control in stable patients with recent onset of AF. Direct-current cardioversion restores sinus rhythm in 75% to 93% of cases. Administration of antiarrhythmic drugs before electrical cardioversion has been shown to improve success rates. Success rates of pharmacological agents used alone vary, but average about 50% after 1 to 5 hours.[43]

Recurrence rates are high after successful cardioversion. In untreated patients, relapse rates range from 71% to 84% at 1 year. Utilizing rhythm-control medications may reduce the relapse rate by 30% to 50%.[44] After successful cardioversion, the benefit of maintaining sinus rhythm must be balanced against the side-effect profile of the antiarrhythmic agent. Table 11 lists agents with proven efficacy at maintaining sinus rhythm and preventing AF recurrence.

TABLE 11. Agents With Proven Efficacy at Maintaining Sinus Rhythm and Preventing Recurrence of Atrial Fibrillation

Amiodarone
Beta blockers
Disopyramide
Dofetilide
Dronedarone
Flecainide
Propafenone
Sotalol

AMIODARONE VS. DRONEDARONE FOR RHYTHM CONTROL

Amiodarone. Oral amiodarone is indicated for the treatment of recurrent, hemodynamically unstable ventricular tachycardia and recurrent ventricular fibrillation that is unresponsive to adequate doses of other antiarrhythmic medications or when the patient cannot tolerate alternative agents. Intravenous amiodarone is indicated for the initiation of treatment and for prophylaxis of frequently recurring ventricular fibrillation and hemodynamically unstable ventricular tachycardia in patients whose condition is refractory to other therapy.[45]

Amiodarone is considered a broad-spectrum antiarrhythmic agent because of its multiple, complex effects on the heart's electrical activity. Amiodarone also causes vasodilation, which may result in a drop in blood pressure.

Amiodarone has several potentially fatal toxicities; the most important is pulmonary toxicity (hypersensitivity pneumonitis or interstitial/alveolar pneumonitis), which can be fatal in about 10% of cases. Hepatic injury is common with amiodarone but is usually mild and evidenced only by abnormal levels of liver enzymes. Overt liver disease can occur, however, and has been fatal in a few cases.[46]

Exacerbation of ventricular arrhythmia and significant heart block or sinus bradycardia have been seen in 2% to 5% of patients treated with amiodarone.[46] Although the frequency of such proarrhythmic events does not appear to be greater with amiodarone than with other antiarrhythmic agents, these effects are prolonged because amiodarone is very slowly metabolized and excreted.

Concomitant use of simvastatin with amiodarone can in rare cases cause rhabdomyolysis, a condition of muscle injury that can result in kidney failure or death. This risk is dose related and increases when the patient receives more than 20 mg/d of simvastatin concomitantly with amiodarone. Predisposing risk factors for rhabdomyolysis include advanced age (over 65 years), renal impairment, and uncontrolled hypothyroidism.[47] Table 12 lists drug interactions that may occur with amiodarone.

TABLE 12. Drug Interactions With Amiodarone

Drug	Interactions with Amiodarone	Recommendations
Beta blockers (e.g., atenolol, metoprolol, propranolol) Certain calcium-channel blockers (e.g., diltiazem, verapamil)	Excessively slow heart rate or a block in electrical-impulse conduction through the heart	Monitor heart rate when initiating or titrating these medications
Digoxin	Increased blood levels of digoxin	Reduce digoxin dose by 50% when initiating amiodarone therapy
Flecainide	Up to 50% increase in blood concentrations	Monitor flecainide levels in blood
Procainamide Quinidine	Up to 30% to 50% increase in blood concentrations during the first week of amiodarone therapy Additive electrical effects, possibly leading to, worsening arrhythmias	Consider reducing the doses of these drugs when starting amiodarone
Phenytoin	Increased toxicity resulting from the 2- or 3-fold increase in blood concentrations of phenytoin caused by amiodarone. Symptoms of phenytoin toxicity include unsteady eye movement (temporary and reversible), tiredness, and unsteady gait.	Monitor clinically and measure phenytoin levels more frequently when initiating or titrating amiodarone
Ritonavir	Can inhibit the enzyme responsible for amiodarone metabolism. No clinical problems have been identified as a result of this interaction.	Avoid this combination to prevent amiodarone toxicity
Tricyclic antidepressants (e.g., amitriptyline) Phenothiazines (e.g., chlorpromazine)	Serious arrhythmias, QT prolongation	Avoid this combination
Warfarin	Increased risk of bleeding that can be serious or even fatal as early as 4-6 days or as late as a few weeks after starting the drug combination	Monitor INR frequently when initiating or titrating amiodarone
Statins (e.g., atorvastatin, lovastatin, simvastatin)	Increased risk of severe muscle breakdown and kidney failure or liver disease. This interaction is dose related, so lower statin doses are safer than higher doses when used with amiodarone.	Consider using an alternative statin, pravastatin, that does not interact in the same way and is safer in patients taking amiodarone
Dextromethorphan	Amiodarone inhibits dextromethorphan's metabolism. The significance of this interaction is unknown.	Instruct patients to avoid taking dextromethorphan and amiodarone together if possible

INR: international normalization ratio

Dronedarone. Dronedarone is a novel antiarrhythmic agent that was approved by the FDA in July 2009 to assist with the maintenance of normal heart rhythm in patients with a history of AF or atrial flutter. Dronedarone was approved for use in patients whose hearts have returned to normal rhythm or who will undergo chemical or electrical cardioversion to restore normal heart rhythm. Although dronedarone was not tested in patients in the LTC setting, its approval provides a welcome addition to the list of agents available for the management of AF in this setting and offers a safer alternative to amiodarone for maintenance of sinus rhythm.

Dronedarone structurally resembles amiodarone and has a similar electropharmacologic profile but different relative effects on ion channels. Dronedarone has a shorter half-life (approximately 24 hours) than amiodarone, resulting in reduced accumulation of the drug in tissue, which in turn produces lower risks of thyroid-related and pulmonary disease than those associated with amiodarone.[48]

Two randomized controlled trials involving 1,237 patients with AF or atrial flutter showed that dronedarone is more effective than placebo in maintaining sinus rhythm and controlling ventricular rate during AF recurrences, without significantly more side effects than were seen with the placebo.[49] A third study, however, in patients with advanced symptomatic CHF but without AF, was prematurely terminated because of an excess number of deaths among patients taking dronedarone. Dronedarone should not be used in patients with severe heart failure.[50]

Most recently, in the ATHENA[1*] trial, which enrolled 4,628 patients aged 70 or older in 37 countries, dronedarone significantly reduced the risk of death or hospitalization caused by cardiovascular events in patients with paroxysmal or persistent AF or atrial flutter.[48] After a mean follow-up of 21 months, 54.5% of patients receiving dronedarone died from any cause or suffered a cardiovascular event, compared with 71.7% of patients receiving a placebo.

Approximately 30% of patients in both the dronedarone and placebo arms discontinued the study drug prematurely. Bradycardia, QT-interval prolongation, diarrhea, nausea, rash, and an increase in serum creatinine levels occurred significantly more frequently in patients receiving dronedarone than in those on placebo.

Patients were excluded from participation in ATHENA if they had permanent AF, an unstable hemodynamic condition (e.g., decompensated heart failure within the previous 4 weeks), New York Heart Association class IV CHF, planned major surgery, acute myocarditis, bradycardia with a heart rate of less than 50 beats per minute or a PR interval of more than 0.28 seconds, or previous clinically significant sinus-node disease if the patient did not currently have a pacemaker. Other exclusion criteria included a baseline GFR of less than 10 ml/min, a potassium level of less than 3.5 mmol/L that was not being treated, and a requirement for prohibited concomitant medication (i.e., other class I or III antiarrhythmic agents).

INVASIVE AND SURGICAL PROCEDURES TO RESTORE CARDIAC RHYTHM

Invasive or surgical procedures such as those described below may be indicated in patients in whom pharmacologic rhythm-control therapy is ineffective and in those who are undergoing heart surgery for another condition. In general, such procedures are rarely appropriate for patients with AF in the LTC setting.

Catheter ablation. This minimally invasive procedure involves compartmentalization of the atria with continuous ablation lines. Ablation tends to take several hours and its success rate is only about 50% to 60%.[51] A newer form of ablation involves ablating in three or four areas within the left atrium near the openings of the four pulmonary veins. This is technically easier than traditional linear ablation, although the procedure still takes several hours.

Atrioventricular node ablation and insertion of a permanent pacemaker. This procedure may offer a treatment alternative for patients with chronic AF and an uncontrolled ventricular response despite aggressive medical therapy. Catheter ablation of the atrioventricular (AV) juncture permanently interrupts conduction from the atria to the ventricles but, because the AV block is permanent, a pacemaker is required. AF may still be present, but the pacemaker controls the ventricular response. A biventricular device may be appropriate for patients who have significant ventricular dysfunction and permanent ventricular pacing.

Implantable cardioverter defibrillator. This option is used for patients at risk for recurrent, sustained ventricular tachycardia or fibrillation. The device is connected to leads inside the heart or on its surface. The leads are connected to a pulse generator implanted beneath the skin of the chest or abdomen; they deliver electrical shocks, sense the cardiac rhythm, and sometimes pace the heart. When an implantable defibrillator detects ventricular tachycardia or fibrillation, it shocks the heart to restore normal rhythm. These devices have been proven useful in preventing sudden death in patients with known sustained ventricular tachycardia or fibrillation.[52-54]

Maze procedure. Surgical compartmentalization of the atria, or the Maze procedure, has the potential to resolve AF. In this procedure, the atria are transected and resutured to reduce the critical mass required to maintain AF. Atrial transport is restored postoperatively and long-term anticoagulation is not necessary. Although they are open-chest procedures, thoracoscopic procedures may reduce hospitalization and recovery times. The Maze procedure may be used for patients with AF who are undergoing concomitant mitral valve procedures; it has not been proven useful, however, as a primary therapy for AF.

Percutaneous closure of the left atrial appendage. This is a surgical option for treating AF. It may not be appropriate for frail elderly patients, but it may be a suitable alternative to chronic warfarin therapy for stroke prophylaxis in patients with nonvalvular AF.

Summary

AF is a serious health problem that is the most common arrhythmia requiring hospital admission and is associated with an increase in mortality. Because the prevalence of AF increases steadily with age, the number of patients in the LTC setting who have or are at risk for AF can be expected to rise in the near future. An urgent need therefore exists for LTC interdisciplinary teams to understand AF and to be able to address it promptly and effectively.

The treatment of AF involves choosing treatment goals and strategies and deciding whether to pursue certain medical or surgical treatments. When discussing goals and treatment options with the patient, family, or advocate, the practitioner should carefully explain the pros and cons of each option and its potential impact on survival and quality of life. Patient characteristics to consider when setting treatment goals may include functional status, presence of comorbid conditions, prior stated or recorded wishes about goals of care, and prognosis.

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APPENDIX 1.

Be an Atrial Fibrillation Detective

One or more of the following clues may indicate the presence of atrial fibrillation (AF) (although no single clue indicates with certainty that AF is present):

- Anxiety
- Chest pain
- Drop in blood pressure
- Lack of energy
- Light-headedness or faintness
- Palpitations (some people may describe an irregular or fluttering sensation in the chest)
- Shortness of breath
- Weakness

In patients with moderate-to-severe communication problems (e.g., aphasia, cognitive impairment, language barriers), it is important to observe and document these nonspecific clues and seek further assessment for possible AF. Symptoms may become more severe over time if AF is not recognized and addressed promptly. An absence of symptoms does not necessarily rule out AF because some patients with AF are asymptomatic. A thorough history and physical is necessary to confirm or rule out a diagnosis of AF.

APPENDIX 2.

Antithrombotic Baseline Risk and Benefit Assessment Tool

Patient Name _____ Date_____

Height	_____	Weight	_____	Creatinine	_____
Estimated GFR	_____	INR	_____	Stool guaiac	_____
Urine dip for blood	_____	Hemoglobin	_____	Platelet count	_____
Stool *Helicobacter pylori* antigen (optional)	_____	Blood glucose	_____		

Estimated Stroke Risk for Patients With Atrial Fibrillation (Adapted from Gage et al, 2001)

CHADS-2 Score	Strokes/10,000 patients/year
0	190
1	280
2	400
3	590
4	850
5	1,250
6	1,820

1 point for
CHF or ejection fraction less than 50%
Hypertension
Age 75 or older
Diabetes

2 points for
Stroke or TIA

Benefits of Warfarin Therapy (Adapted from Singer et al, 2004)

☐ Risk of stroke decreased 65% ☐ Stroke less likely to cause death or severe disability if taking warfarin

Absolute Contraindications to Warfarin (Adapted from Man-Son-Hing, Laupacis, 2003)

☐ Current active bleeding ☐ Platelet count less than 50,000
☐ Blood pressure consistently more than 160/90 ☐ Noncompliance with medication or monitoring

Relative Contraindications to Warfarin (Adapted from Man-Son-Hing, Laupacis, 2003)

☐ Ethanol at or more than 2 oz/d[4] ☐ Nonselective NSAID without gastric cytoprotection (e.g., PPI, misoprostol)[4]
☐ Extreme functional disability ☐ Poor short-term prognosis due to malignancy, advanced chronic disease

Intracerebral Bleeding Risk for Outpatients With Atrial Fibrillation (per 10,000 patients/year) (Adapted from Levine et al, 2004)

No therapy: 10 ICH; 4 will die Warfarin: 75 ICH; 43 will die
Aspirin: 20 ICH; 5 will die

Subdural Hematoma Risk in Outpatient Elderly (per 10,000 people/y) (Adapted from Man-Son-Hing et al, 1999)

No therapy: 4 SDH; 2 will die Aspirin: 8 SDH; 4 will die
Warfarin: 12 SDH; 4 will die

Risk of Hospitalization for Central Nervous System Bleeding Among Nursing Home Stroke Survivors (Adapted from Quilliam et al, 2001)

Aspirin: 19/10,000 people/y Warfarin; 33/10,000 people/year

Relative Risks of Significant Gastrointestinal Bleeding (Adapted from Man-Son-Hing and Laupacis, 2003; 2002)

☐ History of active peptic or duodenal ulcer bleeding[3]	30% chance of rebleeding in 5 year; 13.5 times excess risk compared to those with negative PMH
	No increased risk if treated for *H. pylori*
☐ Taking NO warfarin, aspirin, NSAID[7]	RR 1; 117 upper-GI bleed/10,000 people/ year (16 people will die)
☐ Taking warfarin[7]	RR 2.4; 280 upper-GI bleed/10,000 people/ year (42 people will die)
☐ Taking aspirin[7]	RR 1.2; 140 upper-GI bleed/10,000 people/ year (7 people will die)
☐ Taking nonselective NSAID[7]	RR 3.8; 450 upper-GI bleed/10,000 people/ year
☐ Taking COX-2 selective NSAID[7]	RR 1.9; 320 upper-GI bleed/10,000 people/ year
☐ Taking PPI or misoprostol with NSAID[7,8]	RR 1.9; 320 upper-GI bleed/10,000 people/ year

Conditions That May Increase Bleeding Risk and Require More Frequent Monitoring[5,9]
(Adapted from Levine et al, 2004[5]; Beyth et al, 1998[9])

- ☐ Prior stroke[4]
- ☐ Liver disease[4]
- ☐ Serum creatinine more than 1.5 mg/dL[8]
- ☐ Age 65 or older[8]
- ☐ Malignancy[4]
- ☐ Malnutrition[4]
- ☐ Diabetes[8]
- ☐ Recent MI[8]
- ☐ GFR less than 30[4]
- ☐ Hematocrit less than 30%[8]

COX-2: cyclooxygenase 2 inhibitor; GFR: glomerular filtration rate; GI: gastrointestinal; ICH: intracerebral hemorrhage; INR: international normalized ratio; MI: Myocardial infarction; NSAID: nonsteroidal anti-inflammatory drug; PMH: past medical history; PPI: proton pump inhibitor; RR: relative risk; SDH: subdural hematoma; TIA: transient ischemic attack.

References

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Previously published in: American Medical Directors Association LTC Information Series Kit. Antithrombotic Therapy in the Long-Term Care Setting. Columbia, MD.

APPENDIX 3.

4 mg Warfarin Initiation and Titration Algorithm

(Recommended dose for starting warfarin in elderly patients)

Initiation Algorithm: Using 4 mg Warfarin Doses

1. Give warfarin dose at 6 p.m.
2. Give unfractionated heparin or low-molecular-weight heparin concomitantly, if indicated, during warfarin titration.
3. Measure international normalized ratio (INR) in a.m.
4. Measure INR according to the schedule in Dosing Algorithm 1 (below) until an estimated weekly dose is determined. Clinicians may use Appendix 5.7 (INR-Based Guide for Warfarin Monitoring and Dose Adjustment) to determine the frequency of subsequent INR measurements and dosing adjustments.

Dosing Algorithm

1. On days 0, 1, and 2, administer 4-mg warfarin.
2. Do not check INR on days 0, 1, 2.
3. Measure INR on day 3 to determine predicted daily warfarin dose.
4. Administer predicted warfarin dose daily, measuring INR at least every 2 days until maintenance dose is determined.
5. Maintenance dose is the dose that achieves an INR less than 2.0 and greater than 3.0 on two determinations 48–72 hours apart, with no change in dose for at least 4 days.
6. Once weekly maintenance dose is determined and INR is stable, monitor INR 2 times per week for 2–4 weeks.
7. Schedule subsequent monitoring every 2–4 weeks, or more frequently if clinically indicated.

Warfarin Initiation for Elderly Patients

Day	*INR Value*	*Warfarin Dose*
0	Do not measure	4 mg
1	Do not measure	4 mg
2	Do not measure	4 mg
		Predicted Daily Warfarin Dose
3	INR less than 1.3	5 mg
	1.3 equal to or less than INR less than1.5	4 mg
	1.5 equal to or less than INR less than1.7	3 mg
	1.7 equal to or less than INR less than1.9	2 mg
	1.9 equal to or less than INR less than2.5	1 mg
	INR greater than 2.5	Measure INR daily until INR less than 2.5, then give 1 mg

References

Siguret V, Gouin I, Debray M, et al. Initiation of warfarin therapy in elderly medical inpatients; a safe and accurate regimen. Am J Med 2005; 118(2): 137-142.

Previously published in: American Medical Directors Association LTC Information Series Kit. Antithrombotic Therapy in the Long-Term Care Setting. Columbia, MD.

APPENDIX 4.

5 mg Warfarin Initiation and Titration Algorithm

Initiation Algorithm: Using 5 mg Warfarin Doses

1. Give warfarin dose at 6 p.m.
2. Give unfractionated heparin (UFH) or low-molecular-weight heparin (LMWH) concomitantly, if indicated, during warfarin titration.
3. Measure INR in a.m.
4. Monitor INR daily and adjust dose accordingly.
5. Once weekly maintenance dose is determined and INR is stable, monitor INR 2 times per week for 2–4 weeks.
6. Schedule subsequent monitoring every 2–4 weeks, or more frequently if clinically indicated.

Dosing Algorithm

Day 1
Warfarin 5 mg

Day 2
INR less than 1.5: warfarin 5 mg
INR 1.5–1.9: warfarin 2.5 mg
INR 2–2.5: warfarin 1–2.5 mg
INR greater than 2.5: no warfarin

Day 3
INR less than 1.5: warfarin 5–10 mg
INR 1.5–1.9: warfarin 2.5–5 mg
INR 2–3: warfarin 0–2.5 mg
INR greater than 3: no warfarin

Day 4
INR less than 1.5: warfarin 10 mg
INR 1.5–1.9: warfarin 5–7.5 mg
INR 2–3: warfarin 0–5 mg
INR greater than 3: no warfarin

Day 5
INR less than 1.5: warfarin 10 mg
INR 1.5–1.9: warfarin 7.5–10 mg
INR 2–3: warfarin 0–5 mg
INR greater than 3: no warfarin

Day 6
INR less than 1.5: warfarin 7.5–12.5 mg
INR 1.5–1.9: warfarin 5–10 mg
INR 2–3: warfarin 0–7.5 mg
INR greater than 3: no warfarin

References

Carnahan W, Bracikowski J. Warfarin: Less may be better. Ann Intern Med 1997; 127(4): 332-333.

Previously published in: American Medical Directors Association LTC Information Series Kit. Antithrombotic Therapy in the Long-Term Care Setting. Columbia, MD.

APPENDIX 5.

Warfarin (Coumadin) Flow Sheet

(Printed with permission from William D. Smucker MD, CMD, Medical Director, Altenheim Nursing Home, Strongsville, Ohio)

☐ Manage patient per Facility Warfarin Protocol
☐ Manage patient per Attending Physician _____

Patient: _____ Attending Physician: _____ Attending Phone #: _____

DIAGNOSIS	ICD-9	INR GOAL/RANGE	TREATMENT DURATION	STOP DATE

Date	Current INR	Current Total Weekly Dose (TWD)	Recent Monitoring Guaiac Hemoglobin result (date)	ACTION Hold warfarin until _____ Give Vit K Change dose No change	New TWD (amount & start date)	Next INR Check Date	Nurse

Use a new row for each action (e.g., holding a dose, every other day dose, Vitamin K) or each phone call to physician. Record physician action. If new dose is ordered, record new total weekly dose and date to start new TWD.

APPENDIX 6.

INR-Based Guide for Warfarin Monitoring and Dose Adjustment

(Reformatted and printed with permission from William D. Smucker MD, CMD, Family Medicine Center of Akron, Summa Health System, Akron, Ohio)

Dose Adjustment to Maintain Target International Normalized Ratio (INR) 2–3

INR less than 2

Check Warfarin Flow Sheet (Appendix 5) to determine if patient suffered an acute DVT or PE within past 3 months

Scenario A

If patient has had DVT or PE within 3 months AND INR less than 1.5

- Contact attending physician to determine if patient requires heparin or LMWH until INR is therapeutic

Scenario B

If no DVT or PE within 3 months, choose Option 1 or Option 2 below, or choose patient specific plan for dosing and monitoring

Option 1

If

- No DVT or PE within 3 months, ***and***
- Last 3 INRs between 2-3, ***and***
- NO recent missed dose, acute illness, dietary change, new medicine or medicine dose

Continue current dose and recheck INR in 3 days

Option 2

If

- No DVT or PE within 3 months, ***and***
- NO recent missed dose, acute illness, dietary change, new medicine or medicine dose

Increase total weekly dose (TWD) by

- 2.5 mg/wk if TWD is between 17.5 and 45 mg
- 1 mg/wk if TWD is between 4 and 17 mg

Check INR 1 week after starting new TWD

INR 2–3

No change. Recheck INR in 3-4 weeks.

INR 3.1–3.5

Hold current dose and check INR daily until INR less than 3, then choose Option A or Option B below

Option A

If last 3 INRs between 2-3 for last 3 checks

- Resume current TWD ***and***
- Check INR in 1 week

Option B

Decrease TWD by

– 2.5 mg/wk if TWD is between 17.5 and 45 mg

– 1 mg/wk if TWD is between 4 and 17 mg

Recheck INR in 3 days, then weekly for 2 weeks

INR 3.6–5

Withhold warfarin and check INR Daily until INR less than 3.

Decrease TWD by

- 2.5 mg/wk if TWD is between 17.5 and 45 mg
- 1 mg/wk if TWD is between 4 and 17 mg

Recheck INR in 3 days, then weekly for 2 weeks

Recommendations for Actions if INR Greater Than 5

Obtain vital signs

Evaluate patient for signs or symptoms of bleeding.

If INR Greater Than 5 and Patient Is Bleeding

1. Contact physician immediately if bleeding is seen or reported.
2. Consider immediate hospital transfer if signs of **life-threatening hemorrhage** are seen.
   - Systolic blood pressure less than100
   - Heart rate greater than100
   - Brisk bleeding
   - Decreased level of consciousness
   - Respiratory distress
3. If bleeding is not life-threatening and patient is stable, monitor vital signs and clinical condition until INR normalizes. Administer oral vitamin K per guide below.

If INR Greater Than 5 and Patient Is Not Bleeding

INR is above 5, below 9 choose Option A or Option B below

OPTION A

1. Hold warfarin and check INR daily until less than 3
2. When INR is less than 3, resume TWD ***and*** decrease by
   • 5 mg/wk if TWD is between 17.5 and 45 mg
   • 2 mg/wk if TWD is between 4 and 17 mg
3. Check INR 3-7 days after starting new TWD

OPTION B

1. Give 2.5 mg oral vitamin K (1/2 of 5 mg vitamin K tablet)
2. Hold warfarin and check INR daily until INR less than 3
3. If INR is 4 or above after 24 hours, repeat 2.5 mg dose of vitamin K
4. When INR is 3 or below, decrease TWD by
   • 5 mg/wk if TWD is between 17.5 and 45 mg
   • 2 mg/wk if TWD is between 4 and 17 mg
5. Check INR 3-7 days after starting new TWD

INR is above 9

1. Give 5 mg oral vitamin K
2. Hold warfarin and check INR daily until INR less than 3
3. If INR is 4 or above, give 5 mg vitamin K
4. When INR is 3 or below, decrease TWD by
   - 5 mg/wk if TWD is between 17.5 and 45 mg
   - 2 mg/wk if TWD is between 4 and 17 mg
5. Check INR 3-7 days after starting new TWD

References

Levine MN, Raskob G, Beyth RJ, et al. Hemorrhagic complications of anticoagulant treatment: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004; 126:287S-310S.

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Guide for Warfarin Monitoring and Dose Adjustment

TWD using 5-mg tablets

Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	TWD
0.5 tab	0.5 tab	0.5 tab	0.5 tab	0.5 tab	0.5 tab	0.5 tab	17.5 mg
0.5 tab	0.5 tab	0.5 tab	0.5 tab	0.5 tab	0.5 tab	1 tab	20 mg
0.5 tab	0.5 tab	0.5 tab	1 tab	0.5 tab	0.5 tab	1 tab	22.5 mg
0.5 tab	1 tab	0.5 tab	1 tab	0.5 tab	0.5 tab	1 tab	25 mg
0.5 tab	1 tab	0.5 tab	1 tab	0.5 tab	1 tab	1 tab	27.5 mg
0.5 tab	1 tab	1 tab	1 tab	0.5 tab	1 tab	1 tab	30 mg
0.5 tab	1 tab	1 tab	1 tab	1 tab	1 tab	1 tab	32.5 mg
1 tab	1 tab	1 tab	1 tab	1 tab	1 tab	1 tab	35 mg
1.5 tab	1 tab	1 tab	1 tab	1 tab	1 tab	1 tab	37.5 mg
1.5 tab	1 tab	1 tab	1 tab	1.5 tab	1 tab	1 tab	40 mg
1.5 tab	1 tab	1.5 tab	1 tab	1.5 tab	1 tab	1 tab	42.5 mg
1.5 tab	1 tab	1.5 tab	1 tab	1.5 tab	1.5 tab	1 tab	45 mg
1.5 tab	1.5 tab	1.5 tab	1 tab	1.5 tab	1.5 tab	1 tab	47.5 mg
1.5 tab	1.5 tab	1.5 tab	1.5 tab	1.5 tab	1.5 tab	1 tab	50 mg
1.5 tab	1.5 tab	1.5 tab	1.5 tab	1.5 tab	1.5 tab	1.5 tab	52.5 mg

TWD: total weekly dose

Adapted from Horton JD, Bushwick BM. Warfarin therapy: Evolving strategies in anticoagulation. Am Fam Physician 1999; 59(3): 635-646. Review. Erratum in : Am Fam Physician 1999; 60(5): 1333; Am Fam Physician 2002; 65(2): 172; Am Fam Physician 2006; 73(6): 974.

Dosage Adjustments *(see above)*

INR less than 2
Increase TWD by
- 2.5 mg/wk if current TWD is between 17.5 and 45 mg
- 1 mg/wk if TWD is between 4 and 17 mg

INR 3.1–3.5
Decrease TWD by
- 2.5 mg/wk if current TWD is between 17.5 and 45 mg
- 1 mg/wk if current TWD is between 4 and 17 mg

INR 3.6–5
Decrease TWD by
- 2.5 mg/wk if TWD is between 17.5 and 45 mg
- 1 mg/wk if TWD between 4 and 17 mg

INR greater than 5
See *Recommendations for Actions if INR Greater Than 5* on previous page.

Guide for Warfarin Monitoring and Dose Adjustment for Patients Requiring Less Than 17.5 mg/wk

TWD using 3, 2, and 1mg tablets

Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	TWD
0	1 mg	1 mg	0	1 mg	0	1 mg	4 mg
0	1 mg	1 mg	0	1 mg	1 mg	1 mg	5 mg
0	1 mg	1 mg	1 mg	1 mg	1 mg	1 mg	6 mg
1 mg	1 mg	1 mg	1 mg	1 mg	1 mg	1 mg	7 mg
1 mg	1 mg	1 mg	2 mg	1 mg	1 mg	1 mg	8 mg
1 mg	2 mg	1 mg	2 mg	1 mg	1 mg	1 mg	9 mg
1 mg	2 mg	1 mg	2 mg	1 mg	2 mg	1 mg	10 mg
1 mg	2 mg	1 mg	2 mg	1 mg	2 mg	2 mg	11 mg
1 mg	2 mg	2 mg	2 mg	1 mg	2 mg	2 mg	12 mg
1 mg	2 mg	2 mg	2 mg	2 mg	2 mg	2 mg	13 mg
2 mg	2 mg	2 mg	2 mg	2 mg	2 mg	2 mg	14 mg
3 mg	2 mg	2 mg	2 mg	2 mg	2 mg	2 mg	15 mg
3 mg	2 mg	2 mg	2 mg	3 mg	2 mg	2 mg	16 mg
3 mg	2 mg	2 mg	2 mg	3 mg	2 mg	3 mg	17 mg

Reference

Adapted from Horton JD, Bushwick BM. Warfarin therapy: Evolving strategies in anticoagulation. Am Fam Physician 1999; 59(3): 635-646. Review. Erratum in : Am Fam Physician 1999; 60(5): 1333; Am Fam Physician 2002; 65(2): 172; Am Fam Physician 2006; 73(6): 974.

APPENDIX 7.

American College of Chest Physicians Grades of Recommendation for Antithrombotic Agents

Grade of Recommendation*	Benefit vs Risk and Burdens	Methodologic Quality of Supporting Evidence	Implications
Strong recommendation, high-quality evidence, Grade 1A	Desirable effects clearly outweigh undesirable effects, or *vice versa*	Consistent evidence from RCTs without important limitations or exceptionally strong evidence from observational studies	Recommendation can apply to most patients in most circumstances; further research is very unlikely to change our confidence in the estimate of effect
Strong recommendation, moderate-quality evidence, Grade 1B	Desirable effects clearly outweigh undesirable effects, or *vice versa*	Evidence from RCTs with important limitations (inconsistent results, methodologic flaws, indirect or imprecise), or very strong evidence from observational studies	Recommendation can apply to most patients in most circumstances; higher quality research may well have an important impact on our confidence in the estimate of effect and may change the estimate
Strong recommendation, low or very low-quality evidence, Grade 1C	Desirable effects clearly outweigh undesirable effects, or *vice versa*	Evidence for at least one critical outcome from observational studies, case studies, or from RCTs with serious flaws or indirect evidence	Recommendation can apply to most patients in many circumstances; higher-quality research is likely to have an important impact on our confidence in the estimate of effect and may well change the estimate
Weak recommendation, high-quality evidence, Grade 2A	Desirable effects closely balanced with undesirable effects	Consistent evidence from RCTs without important limitations or exceptionally strong evidence from observational studies	The best action may differ depending on circumstances or patient or society values; further research is very unlikely to change our confidence in the estimate of effect
Weak recommendation, moderate-quality evidence, Grade 2B	Desirable effects closely balanced with undesirable effects	Evidence from RCTs with important limitations (inconsistent results, methodologic flaws, indirect or imprecise), or very strong evidence from observational studies	Best action may differ depending on circumstances or patient or society values; higher-quality research may well have an important impact on our confidence in the estimate of effect and may change the estimate
Weak recommendation, low or very low-quality evidence, Grade 2C	Desirable effects closely balanced with undesirable effects	Evidence for at least one critical outcome from observational studies, case series, or from RCTs with serious flaws or indirect evidence	Other alternatives may be equally reasonable; higher-quality research is likely to have an important impact on our confidence in the estimate of effect and may well change the estimate

Reference

*Guyatt GH, Cook DJ, Jaeschke R, et al. Grades of recommendation for antithrombotic agents: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest 2008; 133: 123S-131S.

APPENDIX 8.

Modifiable Stroke Risk Factors: Interventions, Treatment Goals, and Strategies for Monitoring Adverse Drug Effects

Risk Factor	Intervention	Treatment Goal	Monitoring
Hypertension	• Encourage lifestyle modification (1C) • Antihypertensive treatment for those with hypertension (1A) • Consider antihypertensive treatment if not hypertensive (1B) • Combined diuretic and ACE inhibitor treatment is preferred (1A)	• Individualized BP goals based on patient characteristics and comorbidities • Benefit is seen with reductions of 10/5 mmHg (1B) • Normal blood pressure is 120/70 (1B)	• Check for symptoms of postural hypotension • Measure postural blood pressure, if appropriate • Perform appropriate laboratory testing as indicated (e.g., electrolytes, creatine, BUN)
Thrombotic stroke	• Prescribe antiplatelet therapy (1A)	• Prevent recurrent thrombotic strokes	• Check for signs or symptoms of bleeding tendency (e.g., bruising, bleeding, petechiae) • Monitor laboratory tests as indicated (e.g., fecal occult blood testing, hemoglobin, platelet count)
Thrombotic stroke and/or hyperlipidemia	• Recommend low-fat, low-cholesterol diet • Statin therapy to reach NCEP III goals (1A) • Consider statin therapy even if cholesterol levels are normal (IB) • Consider niacin or gemfibrozil if HDL is low (2B)	• LDL cholesterol less than 100 mg/dL (1A) • LDL less than 70mg/dL for very high risk persons (1A)	• Check for muscle pain • Measure liver enzymes periodically • Monitor lipid profile periodically
AF or cardioembolic stroke	• Consider long-term anticoagulation with warfarin (weigh benefits and risks on the basis of risk factors and comorbid conditions)	• INR 2-3	• Check for signs or bleeding • Test periodically for fecal occult blood and hemoglobin • Monitor adequacy of anticoagulation
Artificial cardiac valve	• Consider long-term anticoagulation with warfarin (weigh benefits and risks on the basis of risk factors and comorbid conditions)	• INR 2.5 - 3.5 • Intensity depends on valve type and location	• Check for signs or bleeding • Test periodically for fecal occult blood and hemoglobin • Monitor adequacy of anticoagulation
Diabetes	• Encourage increased activity. • Use oral medications and insulin as indicated • Consider rigorous glucose of blood pressure and lipids (1B)	• Base goals for glucose control on comorbid conditions, presence of diabetic complications, and patient preferences (see AMDA clinical practice guidelines on diabetes[b])	• Monitor according to medications used and goals of therapy

Risk Factor	Intervention	Treatment Goal	Monitoring
Smoking	• Provide counseling on the benefits of smoking cessation (1C)	• Smoking cessation	• Monitor smoking status hypotension
Alcohol consumption	• Recommend avoidance of heavy alcohol consumption (more than 5 drinks/day)	• Limit alcohol consumption to 2 drinks/day (men); 1 drink/day (women) (2C)	• Monitor alcohol consumption

AF: atrial fibrillation; BUN: blood urea nitrogen; INR: international normalized ratio; LDL: low-density lipoprotein.

[a] American Medical Directors Association. Stroke Management and Prevention in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.
[b] American Medical Directors Association. Managing Diabetes in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

References

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Sacco RL, Adams R, Albers G, et al. Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack: A statement for healthcare professional from the American Heart Association/ American Stroke Association Council on Stroke: co-sponsored by the Council of Cardiovascular Radiology and Intervention: The American Academy of Neurology affirms the value of this guideline. Stroke 2006; 37: 577-617.

APPENDIX 9.

Quality Assurance Program for Managing Atrial Fibrillation

LTC facilities should consider developing a quality assurance (QA) program for atrial fibrillation that considers the following steps in the care process:

- **Recognition:** Identifying a history of AF, risk factors for AF, or signs and symptoms suggestive of AF
- **Assessment:** Clarifying the nature, causes, and impact of AF on the patient
- **Treatment:** Selecting appropriate interventions for the patient and implementing them
- **Monitoring:** Reviewing the patient's response to treatment and deciding whether to continue, change, or stop interventions

As part of a good QA program, it is important to educate all staff about the signs, symptoms, causes, consequences, and treatment of AF as well as the potential adverse effects of medications used to treat AF. Nursing staff should be alert for signs and symptoms of AF and should know when to initiate an assessment by notifying the attending physician or other health care provider that a patient is exhibiting signs or symptoms that suggest AF.

Quality Measures

In 2008 a consensus panel of experts identified the following performance criteria for the management of adults with nonvalvular AF or atrial flutter:

- Assessment of stroke risk factors
- Anticoagulation with vitamin K antagonists (VKAs) for those with a high risk factor or more than one moderate risk factor, to achieve an INR of 2.0 to 3.0
- Warfarin or aspirin for those with one moderate risk factor
- Aspirin 81 to 325 mg/d for those with no risk factors

The panel made the following recommendations:

- All patients with AF except those with lone AF or contraindications should receive antithrombotic therapy to prevent thromboembolism. (1A)
- The selection of antithrombotic agent should be based on the absolute risks of stroke and bleeding and the relative risks and benefits for a given patient. (1A)
- Patients with more than one moderate risk factor should receive anticoagulation with a VKA. Moderate risk factors include age 75 or older, hypertension, heart failure, impaired left ventricular systolic function (ejection fraction 35% or less or fractional shortening less than 25%), and diabetes mellitus. (1A)
- Patients without mechanical heart valves who are at high risk of stroke should receive chronic oral anticoagulant therapy with a VKA in a dose adjusted to achieve the target intensity INR of 2.0 to 3.0, unless contraindicated. Factors associated with high risk for stroke in patients with AF are prior thromboembolism (stroke, TIA, or systemic embolism) and rheumatic mitral stenosis. (1A)
- The INR should be measured at least weekly during initiation of therapy and monthly once anticoagulation is stable. (1A)
- Aspirin, 81 to 325 mg daily, is recommended as an alternative to VKAs in low-risk patients and those with contraindications to anticoagulation. (1A)

- Antithrombotic therapy is recommended for patients with atrial flutter in a manner similar to that for those with AF. (1C)

Outcome Measures

Goals for AF treatment should be established and documented in the patient's record. Outcome measures should take into consideration the patient's age, cognitive level, comorbidities, function, and other factors. For instance, permanent resolution of AF with restoration of sinus rhythm is not achievable for most LTC patients. Still, it is possible to improve quality of life for patients with atrial fibrillation. The following are examples of useful outcome measures:

- Achievement of heart rate goals (e.g., resting rate of 60 to 80 beats per minute, ambulatory rate of 90 to 110 beats per minute)
- Increased exercise tolerance
- Increased involvement in activities
- Reduction in AF symptoms

Reference

Estes NA III, Halperin JL, Calkins H, et al. ACC/AHA/Physician Consortium 2008 clinical performance measures for adults with nonvalvular atrial fibrillation or atrial flutter: A report of the American College of Cardiology/American Heart Association Task Force on Performance Measures and the Physician Consortium for Performance Improvement (Writing Committee to Develop Performance Measures for Atrial Fibrillation) developed in collaboration with the Heart Rhythm Society. J Am Coll Cardiol 2008; 51: 865-884.

APPENDIX 10.

Information about Atrial Fibrillation to Share with Patients and Family Members

What is Atrial Fibrillation?

Atrial fibrillation (AF) is an irregular and often rapid heart rhythm. This irregular rhythm, or arrhythmia, happens when electrical impulses in the heart don't work normally. AF may be continuous, or it can come and go. The risk of AF increases with age, so your elderly family member or friend is at greater risk than younger family members and friends for this condition.

How Can You Tell if Someone Has AF?

Whether a person has AF isn't always obvious. Although some people may have such symptoms as heart palpitations (a fluttering feeling in the chest), light-headedness, weakness, fatigue, shortness of breath, or chest pain, others have no outward signs of AF.

Why is Finding Out if Your Family Member or Friend Has AF Important?

AF itself usually isn't life threatening. However, someone with AF has a higher than normal risk of stroke. AF also may contribute to death from heart disease or heart failure. In addition, AF hurts a person's quality of life because it increases their risk of falling and can make them too tired or uncomfortable to enjoy their usual activities.

It is important to work with your doctor to find out if your family member or friend has AF. If he or she does have it, you can work with the doctor and the patient to choose the best treatment and realistic goals of care that match the patient's preferences.

Questions to Ask

- Is my family member or friend at risk for AF?
- What effect does this condition have on my family member or friend's quality of life?
- What are the pros and cons of various treatments? Is medication enough, or will surgery be necessary?
- What is likely to happen if the AF isn't treated?
- Could any lifestyle changes help prevent AF in my family member or friend?
- What can my family member or friend do to prevent an AF-related stroke?

What You Can Do

- Make sure that your family member or friend's doctor takes a complete medical history of your family member or friend that includes past occurrences or symptoms of AF, heart disease or other heart problems, presence of diabetes, and history of smoking and other lifestyle issues that might contribute to heart disease.
- Reduce your family member or friend's risk of falling by making sure that he or she has well-fitting shoes, glasses that he or she can see well with, well-lit rooms, and no throw rugs or other items that might be a tripping hazard. Talk to the doctor about the value of a cane or other walking aid for your friend or family member.
- Make sure that your family member or friend has a current advance directive or living will that describes his or her wishes about such issues as the use of cardiopulmonary resuscitation (CPR) or tube feeding.
- Talk to your family member or friend about how he or she feels. Report any signs of AF to a nurse or doctor.

Additional Resources

Atrial Fibrillation
http://www.emedicinehealth.com/atrial_fibrillation/article_em.htm

Atrial Fibrillation
http://www.mayoclinic.com/health/atrial-fibrillation/DS00291

What Is Atrial Fibrillation?
http://my.clevelandclinic.org/heart/atrial_fibrillation/afib.aspx

New ACCP Guidelines Update Strategies for Antithrombotic/Thrombolytic Therapy
http://www.caringfortheages.com/article/S1526-4114%2808%2960301-7/fulltext

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