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Atrial Fibrillation: Management Strategies In The Emergency Department

Abstract

Atrial fibrillation and atrial flutter are the most common dysrhythmias seen in the emergency department. As the aging population continues to grow, atrial fibrillation and atrial flutter are expected to affect 6 million people by 2050. This will lead to an increase in emergency department visits for symptoms from the disease itself or its complications, such as heart failure or thromboembolic disease. This review examines the recent literature on the diagnosis and management of atrial fibrillation. Evidence-based recommendations are provided, including cost-effective strategies to evaluate new-onset arrhythmias and unstable patients with atrial fibrillation, rate control strategies, the use of medical and direct current cardioversion for new-onset atrial fibrillation/atrial flutter, whom and when to anticoagulate, and the use of the novel anticoagulation agents.

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CME Objectives

Upon completing the article, you should be able to:

1. Discuss the initial evaluation in patients with new-onset AF.
2. Understand the common rate and rhythm controlling agents.
3. Be familiar with the indication and contraindications for ED cardioconversion.
4. Differentiate among the different anticoagulation options and when it is appropriate to start anticoagulation in the ED.

Prior to beginning this activity, see the back page for faculty disclosures and CME accreditation information.

Case Presentations

You have just arrived to your morning shift in the ED, and as you are about to sit down for a cup of coffee, a 37-year-old female presents, complaining of palpitations that started this morning. She has no past medical history, is on no medications, and denies any drug use. On physical exam, she is slightly uncomfortable, has an irregular heart rate of 190 beats/min, and has a blood pressure of 115/75 mm Hg. Her ECG shows rapid atrial fibrillation with wide, bizarre QRS complexes. You wonder what the origin of the dysrhythmia is and whether you should rate control the patient with diltiazem or whether there is another intervention you are not thinking of . . .

Two beds down, the nurse tells you about an 85-year-old male from a nursing home who is febrile to 39.5°C, is tachycardic with a heart rate of 160 beats/min, and has a blood pressure of 98/57 mm Hg. He has a history of dementia, diabetes, and hypertension and is nonverbal at baseline. He is minimally responsive and unable to give you additional information. You begin fluid resuscitating him and administer acetaminophen, and you notice on the monitor that his heart rhythm is irregular. You wonder what the safest way to control the patient's rhythm is and whether and how he should be anticoagulated . . .

Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia.[1,2] Atrial flutter (AFL) is often associated with AF, and both of the conditions may occur in the same patient. The prevalence of AF/AFL has been increasing steadily over the past 20 years, and population estimates based on United States Census data estimate that 3 million cases of AF/AFL were documented in 2010 alone.[3]

Both AF and AFL are associated with increased thromboembolic events and stroke severity.[4,5] AF/AFL pose a tremendous healthcare and economic burden; a retrospective analysis of 2001 national healthcare databases found that AF/AFL accounted for approximately 350,000 hospitalizations, 5 million office visits, and 276,000 emergency department (ED) visits,[6] leading to over $6 billion in healthcare expense annually.[7]

Patients with AF/AFL may have presentations that range from asymptomatic to severe life-threatening episodes that include syncope, congestive heart failure, cardiogenic shock, stroke, and myocardial infarction. The emergency clinician must be alert to the diagnosis and understand the contributing factors and comorbidities. This issue of *Emergency Medicine Practice* provides an analysis of the best available evidence regarding the management of AF/AFL, including cardioversion, rate control, and anticoagulation.

Critical Appraisal Of The Literature

An Ovid MEDLINE® and a PubMed search were carried out for literature from 2002 through October 2012 using the search terms *atrial fibrillation, atrial flutter, management, treatment,* and *emergency.* The Cochrane Database of Systematic Reviews and the National Guidelines Clearinghouse (www.guidelines.gov) were also searched; 374 abstracts were identified, of which, 140 manuscripts were reviewed. In addition, the bibliographies of all reviewed articles were reviewed for additional publications. This process resulted in 10 practice guidelines, 10 systematic reviews, 69 prospective studies, and 35 retrospective studies.

The recommendations presented in this review were excerpted from the relevant guidelines of the American College of Cardiology (ACC), the American Heart Association (AHA), and the European Society of Cardiology's (ESC's) "2006 Guidelines for the Management of Patients with Atrial Fibrillation"[8] and the 2011 American College of Cardiology Foundation (ACCF), AHA, and Heart Rhythm Society's "Focused Update on the Management of Patients With Atrial Fibrillation."[9] These guidelines focus on both acute and long-term management of AF. The AHA levels and classes of evidence are expanded in **Table 1**. Additional guidelines that served as an important resource included the Canadian Cardiovascular Society's "Atrial Fibrillation Guidelines 2010: Management of Recent-Onset Atrial Fibrillation and Flutter in the Emergency Department,"[10] the "Focused 2012 Update of the Canadian Cardiovascular Society Atrial Fibrillation Guidelines: Recommendations for Stroke Prevention and Rate/Rhythm Control,"[11] and the ESC's Task Force for

Table 1. American Heart Association Classification Of Levels And Classes Of Evidence[9]

Levels of Evidence	
Level A	Multiple populations evaluated. Data derived from multiple randomized controlled trials or meta-analyses
Level B	Limited populations evaluated. Data derived from a single randomized trial or nonrandomized studies
Level C	Very limited populations evaluated; only consensus opinion of experts, case studies, or standard of care

Classes of Evidence	
Class I	Benefit >>> Risk: procedure SHOULD be performed/administered
Class IIa	Benefit >> Risk; IT IS REASONABLE to perform procedure/administer treatment
Class IIb	Benefit ≥ Risk; procedure/treatment MAY BE CONSIDERED
Class III	No proven benefit/harmful to patients

the Management of Atrial Fibrillation's "Guidelines for the Management of Atrial Fibrillation."[2]

Epidemiology

Estimates of AF/AFL prevalence have been reported to be 1% to 4% in the general population and 9% in patients over the age of 80.[12,13] The incidence of AF/AFL increases with age, and it is more common among males, with a median age of onset of 66.8 years for men and 74.6 years for women.[14] AFL has been studied much less as a clinically separate entity, but estimates suggest that the incidence of AFL is 88 per 100,000 person-years and that there are roughly 200,000 new cases of AFL in the United States annually.[15] The incidence rate for AF has shown to vary by age, from < 0.5 per 1000 person-years before age 50 to as high as 20.7 per 1000 person-years among those 80 to 84 years of age.[16-18] As the population ages, studies suggest that AF/AFL will affect 6 million people by 2050.[19]

Etiology And Risk Factor Stratification

Cardiac causes of AF/AFL, as described in the Framingham Heart Study, include mitral valve disease, myocardial disease, conduction system disorders, Wolff-Parkinson-White syndrome, and pericardial disease.[13] Conditions associated with AF include thyrotoxicosis, hypothermia, alcohol use, severe infection, hypoxia, pulmonary emboli and pneumonia, kidney disease, obesity, diabetes mellitus, digoxin toxicity, and electrolyte abnormalities.[20-22] Intrathoracic surgery, such as cardiac or pulmonary surgery, or invasive cardiac studies may also precipitate AF.[23]

AF is the most common cardiac complication of hyperthyroidism, and it is estimated to occur in up to 15% of hyperthyroidism patients;[24,25] however, hyperthyroidism accounts for < 1% of all patients with new-onset AF.[26]

In the first 24 hours of myocardial infarction, AF is common, and it carries a poor prognosis, with higher 30-day, 6-month, and 1-year mortality and stroke rates.[27-29] The predicted incidence of myocardial infarction in patients with AF on presentation to the ED is estimated to be as high as 5% to 15%.[30-33]

AF is categorized as follows:

- First detected episode
- Recurrent (after 2 or more episodes)
- Paroxysmal (if recurrent AF terminates spontaneously)
- Persistent (if sustained beyond 7 days)

Initial episodes of AF will often resolve spontaneously within 7 days, with most episodes self-terminating in < 24 hours. Persistent AF may require termination via medications or direct current electric cardioversion.[34] Lone AF applies to AF in individuals younger than 60 years of age without clinical or echocardiographic evidence of cardiopulmonary disease (including hypertension),[35] and it carries favorable risk profiles in regard to thromboembolism and mortality. Nonvalvular AF refers to AF in individuals without rheumatic mitral valve disease, prosthetic heart valves, or valve repair.

Pathophysiology

AF and AFL are supraventricular tachycardias that arise from disorganized or abnormal atrial depolarization. Atrial fibrosis and loss of atrial muscle mass are the most frequent histopathological changes in AF. Electrical remodeling occurs, resulting in multiple reentry circuits or rapidly firing atrial foci and shortening of atrial refractoriness and action potential, thus contributing to the maintenance of AF.[8] Triggers for AF include autonomic nervous system stimulation, bradycardia, atrial premature beats or tachycardia, accessory atrioventricular pathways, ectopic foci occurring in the sleeves of atrial tissues within the pulmonary veins or vena caval junctions, and atrial stretch.[36]

Decreased atrial contractile function and loss of synchronous atrial activity ("atrial kick") combined with rapid ventricular responses may have hemodynamic consequences with a markedly decreased cardiac output.[37,38] A persistently elevated ventricular rate during AF (usually > 120 beats/min) for prolonged time periods may also result in increased mitral regurgitation, eventually leading to a dilated ventricular cardiomyopathy (tachycardia-induced cardiomyopathy).[39,40]

Prolonged AF makes restoration and maintenance of sinus rhythm more difficult, as the adage "atrial fibrillation begets atrial fibrillation" suggests. Repeated or prolonged episodes of AF result in shortened effective refractory periods as electrophysiological remodeling occurs, which, combined with increased atrial mass volume and delayed conduction, favors sustained AF.[8] Prolonged AF also disturbs atrial contractile function, which may not recover for days or weeks following the restoration of sinus rhythm ("atrial stunning"), which has important implications for the duration of anticoagulation after cardioversion.

Differential Diagnosis

AF/AFL are supraventricular tachycardias with the distinguishing feature of unique P-wave morphologies. AF has low-amplitude fibrillatory waves that result in an irregularly irregular ventricular rhythm with an absence of well-defined P-waves. AFL is characterized by sawtooth-appearing P-waves with an atrial frequency around 300, often resulting in a regular ventricular frequency around 75 or 150 beats/min, depending

on the atrioventricular node conduction block. These distinguishing characteristics may help differentiate AF from other supraventricular tachycardias.

The wide QRS complexes in AF with Wolff-Parkinson-White syndrome may give an appearance similar to ventricular tachycardia; however, AF with preexcitation can usually be distinguished by the very rapid ventricular rate and irregularly irregular rhythm. **Table 2** presents the differential diagnosis for patients suspected of having AF.

Prehospital Care

Prehospital care begins by assessing and stabilizing the airway, breathing, and circulation. In hemodynamically stable patients, a targeted history and physical examination should be performed to assess for underlying causes of the tachycardia. According to the Advanced Cardiovascular Life Support (ACLS) guidelines, cardioversion should be considered if the patient exhibits signs of hemodynamic compromise or poor coronary artery perfusion.[43] The prehospital provider must consider that the ACLS guidelines address patients with hemodynamic instability solely from acute AF/AFL. These guidelines do not take into account the patient with chronic AF/AFL who may be hemodynamically unstable due to shock from another cause (such as sepsis or hypovolemia), where interventions should target the acute process.

There are few studies of prehospital management of AF. One retrospective study of paramedic responses to 33 patients in rapid AF reported that optimal prehospital care can be safely achieved with symptomatic treatment alone using nitroglycerine, furosemide, aspirin, and morphine. No adverse events were reported; however, none of these patients were hemodynamically unstable.[44] A small retrospective study of 70 patients examining the safety of diltiazem in the prehospital setting, and a case report of the Cardizem Lyo-Ject® infusion for rapid AF found diltiazem to safely decrease ventricular response to AF without precipitating hypotension, endotracheal intubation, cardiac arrest, or unstable dysrhythmias.[45,46]

Currently, there is no universally accepted treatment protocol for prehospital management of AF. Initial management should be focused on providing supportive care for the patient, with consideration of crystalloid fluid boluses, intravenous (IV) diltiazem for rate control, or electrical cardioversion if the patient becomes acutely unstable from AF/AFL.

Table 2. Differential Diagnosis For Atrial Fibrillation

Rhythm	Atrial Frequency, beats/min	Ventricular Frequency, beats/min	P-wave
Sinus tachycardia	100-180	100-180	Precedes every QRS complex
Atrial fibrillation	400-600	60-190, irregularly irregular	Absent
Atrial flutter	250-350	75-150, regular, sometimes alternating block	Sawtooth
Atrioventricular nodal reentrant tachycardia	180-250	180-250	In QRS complex (R)
Atrial tachycardia	120-250	75-250	Precedes QRS; P-wave differs from sinus P-wave
Multifocal atrial tachycardia	> 100	> 100	3 or more different P-wave morphologies at different rates
Atrial fibrillation with Wolff-Parkinson-White syndrome	400-600	180-300, with wide, bizarre QRS complexes	Absent

Emergency Department Evaluation

History And Physical Examination

Presentation of AF/AFL may be related to the disease itself or to complications from associated conditions (eg, thromboembolism, heart failure, thyroid disease, or alcohol or drug toxicity). Clinical symptoms associated with AF/ AFL may include anxiety, palpitations, shortness of breath, dizziness, chest pain, or generalized fatigue. A careful history of medications and alcohol and drug use should be obtained.

The physical examination must be comprehensive (with a full set of vital signs, including oxygen saturation) and should include a careful evaluation for evidence of thyroid disease (eg, exophthalmos and enlarged thyroid) and for evidence of deep vein thrombosis/pulmonary embolus (eg, unilateral lower extremity swelling or tenderness). The cardiac evaluation should assess rate, rhythm, and the presence of heart murmurs.

Diagnostic Studies

Electrocardiogram

The diagnosis of AF/AFL requires documentation of an electrical heart tracing with at least a single lead recording during the arrhythmia.

Atrial Fibrillation Findings

Several characteristic electrocardiogram (ECG) changes define AF:

1. Presence of low-amplitude fibrillatory waves on ECG without defined P-waves
2. Irregularly irregular ventricular rhythm
3. Fibrillatory waves typically have a rate of > 300 beats per minute
4. Ventricular rate is typically between 100 and 160 beats per minute **(See Figure 1)**

Wolff-Parkinson-White syndrome (preexcitation syndrome) should be considered in any patient with bizarre, wide QRS complexes of different morphologies; in patients with a very rapid ventricular rate with an RR interval > 250; and in younger patients who present to the ED in AF. **(See Figure 2.)**

Atrial Flutter Findings

AFL is an atrial tachyarrhythmia secondary to a reentry mechanism, and is characterized by an atrial rate of 250 to 350 beats per minute. Ventricular rates in AFL are usually around 150 beats per minute secondary to 2:1 conduction through the atrioventricular node. Classic ECG pattern includes the presence of flutter waves. **(See Figure 3.)**

Laboratory Tests

Initial laboratory testing is tailored to the patient's presentation. Tests to consider include a complete blood cell count, metabolic panel, and hepatic function panel. Coagulation studies should be drawn on patients, especially those on warfarin. A thyroid panel is obtained for patients with clinical signs of hyperthyroidism and in patients older than 55 with new-onset AF,[24] as older patients may not present with classic signs and symptoms of hyperthyroidism.[47,48] Thyroid function studies are also suggested for the first episode of AF when the ventricular rate is difficult to control.

Cardiac Serum Markers

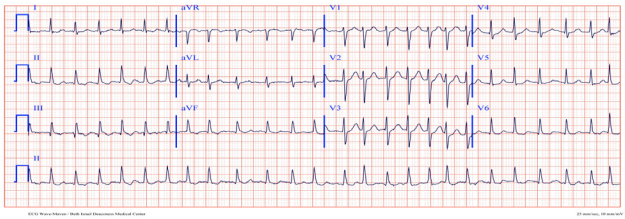
Cardiac serum markers may have some utility in select patients suspected to be at risk of acute coronary syndromes (ACS), including those with ECG changes suspicious for ischemia or underlying heart disease or significant risk factors for coronary artery disease.[31,49-51] Two studies that examined the incidence of myocardial infarction among patients admitted for AF found that 5% to 6% of patients have an ACS.[31,33] ST-segment elevation or depression > 2 mm on admission ECG were found to be associated with patients diagnosed with acute myocardial infarction.[31]

Additional Studies

Urine drug screens may be obtained on a case-by-case basis. Patients should be questioned about the use of herbal products and supplements (such as creatine monohydrate), especially young patients without structural heart disease.[52]

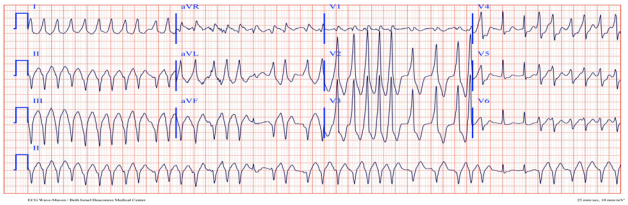
Pregnancy tests should be obtained on women of reproductive age, since pharmacological therapies should be selected based on trimester of pregnancy. Although AF is the most common dysrhythmia en-

Figure 1. Atrial Fibrillation With Rapid Ventricular Response Around 150 Beats Per Minute



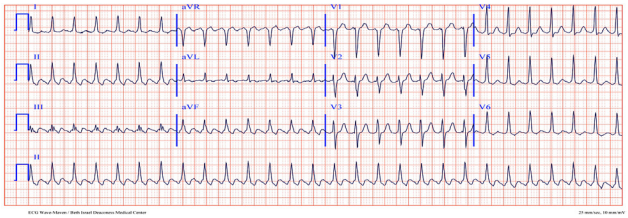
Note the irregularly irregular pattern with narrow-complex QRS complexes.

Figure 2. Atrial Fibrillation With Wolff-Parkinson-White Preexcitation And Conduction Through The Bypass Tract



This wide complex tachycardia with a rate of about 230 beats per minute looks similar to ventricular tachycardia; however, the "irregularly irregular" rhythm and extremely rapid rate leads towards atrial fibrillation with preexcitation.

Figure 3. Atrial Flutter With 2:1 Block



The atrial rate is 320 with a 2:1 atrioventricular node conduction block with a ventricular response of 160 beats per minute. The classic flutter "F" waves give a "sawtooth" appearance to the atrial activity.

Figures 1-3 are reprinted with permission from: Nathanson L A, McClennen S, Safran C, Goldberger AL. ECG Wave-Maven: Self-Assessment Program for Students and Clinicians. http://ecg.bidmc.harvard.edu.

countered in the ED, it is rare in pregnancy, and, when encountered, it is usually associated with maternal hyperthyroidism, congenital heart disease, or rheumatic heart disease.[53] In this population, initial evaluation should include ECG, basic laboratory tests, urine drug screen, thyroid panel, and echocardiogram.[54]

Tests for underlying pulmonary embolism have been suggested by some medical textbooks; however, most studies imply that patients who are otherwise not suspected of having pulmonary embolism are unlikely to have pulmonary embolism and do not require additional testing.[55]

Patients on digoxin (Lanoxin®, Cardoxin®, Digitek®) should have their digoxin level obtained, as noncompliance may result in rapid ventricular response in those with chronic AF. Digoxin toxicity may be associated with a variety of dysrhythmias, including AF with a slow ventricular response. Digoxin toxicity is a relative contraindication to electrical cardioversion.

While theophylline (Uniphyl®, Elixophyllin®, Theolair®) is rarely used today, toxicity is associated with AF, and theophylline levels should be obtained if the patient has been prescribed this drug.[56]

Imaging Studies

Chest radiography may be performed to evaluate lung parenchyma and pulmonary vasculature for significant findings such as pulmonary edema in heart failure, pulmonary masses, left atrial enlargement from mitral valve regurgitation, Hampton hump, or Westermark sign in pulmonary embolus.

Use of focused ultrasonography in hypotensive patients with AF/AFL may help identify additional causes of shock or hypotension.[57-59] Cardiac views can help to evaluate right heart strain that might indicate a diagnosis of pulmonary embolism[55] and the presence of a pericardial effusion and cardiac tamponade, and they may also assist in evaluating left ventricular function. Measurement of the inferior vena cava can assess intravascular volume depletion and guide resuscitation.[60,61] Additional views of the abdominal aorta and the Morison pouch can help eliminate causes of acute blood loss from occult aortic aneurysmal rupture or intraabdominal bleeding as a cause of hypotension.[58]

Routine transthoracic echocardiography in the ED is not recommended, but it may be performed in the inpatient or outpatient setting to further evaluate cardiac function and causative factors for AF.[12,62] Measurement of the left atrial size might also help identify patients who might be successfully converted and remain in normal sinus rhythm.[63] Transesophageal echocardiography may help guide acute cardioversion of patients with AF of unknown duration, with evaluation of the atrial appendage for clot visualization.[8,64-70]

Treatment Of Unstable Patients

Initial Approach To Management

The goals of AF management are focused on achieving hemodynamic stability, symptomatic treatment, and the prevention of complications (such as thromboembolism).[71,72] The initial management includes cardiac monitoring, supplying supplemental oxygen as needed, establishing IV access, and rapidly assessing the patient's hemodynamic status. A history and physical examination should be conducted, with the focus placed on the duration and nature of the symptoms, the comorbidities, and identifying reversible causes of AF.

Emergent Stabilization Of Critically Ill Patients

The 2010 ACLS guidelines suggest immediate direct current cardioversion (DCC) for patients with altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of shock or hemodynamic instability.[43] Other patients who may benefit from immediate DCC include those with wide complex AF/AFL that may signify an accessory pathway with very rapid ventricular rates or hemodynamic instability. These guidelines, however, do not address many of the AF/AFL patients encountered in the ED who are hemodynamically unstable due to other disease processes (including sepsis, hypovolemia, massive pulmonary embolism, or pericardial tamponade). Additionally, while the recommendations state that cardioversion should be attempted in unstable patients, the critically ill patient may have chronic AF/AFL or additional comorbidities and illnesses that may lead to failed or short-lived effects of DCC, and alternative methods of stabilization must be considered early on. **(See Table 3.)**

For patients with AF/AFL who are hemodynamically unstable, initial actions to stabilize the patient include obtaining large-bore intravascular access and addressing intravascular volume depletion with rapid infusions of 20- to 40-mL/kg crystalloid bolus challenges.[57] Concurrent evaluation for myocardial infarction, signs of infection, or blood loss as a primary cause of hypotension should be conducted, and initial therapies (including revascularization, early goal-directed therapy, blood transfusion, and vasoactive agents, as warranted) should be targeted to the underlying cause of hypotension or shock.

Electrical cardioversion may be the fastest method to obtain rate control in AF/AFL patients because it converts the patient back to sinus rhythm; however, it requires procedural sedation and carries a risk of embolic events and cardiac arrhythmias. In unstable patients without other causes for shock or hypotension and patients with preexcitation syndromes with very rapid ventricular response, the use of DCC may be life-saving, and it is the first-line treatment. For

these patients, emergent DCC should not be withheld due to concerns for thromboembolism.

Based on the new ACLS guidelines, the starting energy for patients with AFL should be between 50 J and 100 J biphasic waveform synchronized cardioversion (or monophasic equivalent) and 100 J for AF; however, a multicenter trial found that only 60% of patients cardioverted with 100 J biphasic, while 90% converted with 200 J.[73] Thus, starting at a higher energy may be beneficial in an unstable patient.[73-75] **(See Table 4.)**

Urgent Stabilization Of Hemodynamically Unstable Patients

Rate-control medications will cause further hypotension in patients if they are given in the standard recommended doses, but they may be necessary to rate control a hemodynamically unstable patient or a patient who fails emergent DCC (especially if the AF/AFL is chronic). One strategy advocated to reduce further hypotension includes pretreatment with push-dose phenylephrine to a goal diastolic blood pressure > 60 mm Hg prior to slow amiodarone or diltiazem infusion.[76]

Amiodarone lacks significant inotropic effects and may have the added benefit of restoring sinus rhythm.[77] Low-dose diltiazem (< 0.2 mg/kg) was shown in 1 small study to have less of a hypotensive effect on patients than standard-dose diltiazem; however, none of the participants in this study were hypotensive at baseline.[78]

Calcium, an inopressor, may have some beneficial effect as a pretreatment agent. Some studies have shown that pretreatment with calcium may reduce or reverse the hypotensive effects of verapamil.[79-82] This was not shown in a trial with diltiazem;[83] however, none of the patients became hypotensive in either group.

Pretreatment Doses:

Phenylephrine	50-200 mcg every 1-2 minutes
Calcium	5-10 mL of calcium gluconate or 1-3 mL of calcium chloride

Rate-Control Agent Doses:

Amiodarone	150-mg bolus, then drip or repeat bolus
Diltiazem	2.5 mg/min continuous drip until heart rate < 100 beats/min (or 50-mg total dose)

Table 3. Factors Associated With Failed Cardioversion[21]

- Underlying illness (eg, congestive heart failure, thyrotoxicosis, valvular disease)
- Dilated left atrium
- Longer duration of atrial fibrillation
- Too-low energy
- Technique
- Other patient factors

Table 4. Strategies To Improve Direct Current Cardioversion In The Unstable Patient

- 360 J monophasic or 200 J biphasic synchronized shock
- Time shock delivery during patient's full expiration for optimal energy delivery
- Apex-anterior, apex-posterior, and anterior-posterior pad placement are all effective, although anterior-posterior may be more useful with biphasic defibrillators

Treatment Of Stable Patients

Treatment of hemodynamically stable patients focuses on symptom relief, maintenance of rate control with consideration of cardioversion to sinus rhythm, and the prevention of complications, such as thromboembolism.[71,72]

Selecting A Rate Control Versus A Rhythm Control Strategy For Atrial Fibrillation/Atrial Flutter

The acute ED management of AF/AFL is variable, and few recommendations exist. The 2006 ACC/AHA/ESC guidelines make no distinction between ventricular rate and rhythm control management. Multiple studies have shown no difference in all-cause mortality, cardiovascular mortality, or stroke rate among patients treated with rate or rhythm control;[84-90] however, these studies did not investigate the optimal management choice for new-onset AF/AFL patients presenting to the ED.

Traditionally, most patients with new-onset AF were rate-controlled with a beta blocker or calcium channel blocker, admitted to the hospital, and subsequently converted to sinus rhythm or discharged with rate control.[91-93] Given the rising number of ED visits for new-onset AF/AFL as well as increasing pressures to decrease hospital admissions and total hospital length of stay in order to reduce medical costs and help alleviate ED crowding, there is growing interest in cardioversion of patients with new-onset AF/AFL of < 48 hours in the ED.[94-99]

Spontaneous conversion within the first 48 hours of the initial onset of AF may occur in nearly 50% of patients, and one strategy advocated by the Canadian Cardiovascular Society for patients presenting immediately after onset of symptoms is to rate control the patient while awaiting spontaneous cardioversion. In this strategy, select patients may be started on a rate control agent and instructed to return the following day for reevaluation and possible cardioversion if they do not spontaneously return to sinus rhythm.

Recent studies of elective ED cardioversion (EDCV) have suggested a high rate of success with few complications in low-risk patients. In numerous studies, EDCV has been shown to be safe and to decrease hospital length of stay. A few studies have shown it may allow for safe discharge of select patients from the ED.[93-95,100-109] Choosing appropriate patients for EDCV is of some concern, as multiple studies have shown that patients may not be able to correctly identify the time of onset of AF based on their symptoms.[23,36,44]

Some controversy exists regarding rate control prior to EDCV. Only 2 large studies have examined this. Blecher et al found that administering a rate control agent prior to electrical cardioversion decreased success, with an odds ratio (OR) of 0.39 (95% confidence interval [CI], 0.21-0.74). There was no difference in successful chemical cardioversion.[110] Scheuermeyer et al found no difference in success rates of cardioversion after initial rate control.[111] Neither study found an increase in adverse events among patients treated with rate control prior to cardioversion.

Selecting A Rate Control Agent

In rapid AF/AFL, conduction of disorganized atrial contractions occurs through the atrioventricular node, and most rate control strategies use medications that prolong the atrioventricular refractory periods, thus slowing atrioventricular nodal conduction.[112] **(See Table 5.)** In the absence

Table 5. Intravenously Administered Pharmacological Agents For Rate Control Of Atrial Fibrillation Without An Accessory Pathway[8]

Drug	Class of Recommendation / Level of Evidence	Initial Loading Dosage	Maintenance Dosage	Onset Time	Potential Adverse Effects / Comments
Beta Blockers					
Esmolol	Class I, LOE C	0.5 mg/kg over 1 min	0.06-0.2 mg/kg/min*	< 5 min	• Bradycardia, peripheral vascular insufficiency, hypotension, heart failure, atrioventricular block, dyspnea, bronchospasm • Avoid in patients with obstructive pulmonary disease • Propranolol useful in thyrotoxicosis
Metoprolol	Class I, LOE C	2.5- to 5-mg bolus over 2 min, up to 3 doses	NA	5 min	
Propranolol	Class I, LOE C	0.15 mg/kg	NA	5 min	
Nondihydropyridine Calcium Channel Blockers					
Diltiazem	Class I, LOE B	0.25 mg/kg/dose over 2 min; may give a second dose at 0.35 mg/kg/dose	5-15 mg/kg for < 24 h	2-7 min	• Hypotension, heart failure, compromised ventricular function • First-line medication for patients with obstructive pulmonary disease • Verapamil has more potent negative inotropic and vasodilator effects than diltiazem. May also increase digoxin concentration if used in combination
Verapamil	Class I, LOE B	0.075-0.15 mg/kg over 2 min	NA	3-5 min	
Cardiac Glycoside					
Digoxin	Class I, LOE B	0.25 mg IV every 2 h, up to 1.5 mg	0.125-0.375 mg daily IV or orally	30-180 min	• Digitalis toxicity, heart block • Most useful in combination with a beta blocker or calcium channel blocker for patients with congestive heart failure
Class III Antiarrhythmic Agent					
Amiodarone	Class IIa, LOE C	150 mg over 10 min	0.5 to 1 mg/min IV	< 20 min	• Hypotension, prolonged QT, bradyarrhythmias • Useful as a second-line agent or in patients with congestive heart failure
Adjunctive Therapies					
Magnesium	NA	2 g over 15 min	NA	< 5 min	• Hypotension, respiratory muscle fatigue, cardiac pauses at high doses • Can accumulate rapidly in patients with renal failure

*Very short half-life; needs careful monitoring and titration of dose.

Abbreviations: IV, intravenous; LOE, level of evidence; NA, not applicable.

Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibril executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to revise the 2001 guidelines for the management of patients with atrial fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Eur Heart J.* 2006;27(16):1979-2030. By permission of the European Society of Cardiology.

of preexcitation syndromes, beta blockers and nondihydropyridine calcium channel blockers such as diltiazem and verapamil are effective atrioventricular nodal blocking agents and can safely achieve rate control in most patients.[113-121] These agents work quickly, by slowing the atrioventricular conduction and prolonging refractoriness in the atrioventricular node, thus slowing the ventricular response to AF/AFL.[122] Digoxin is a weak atrioventricular nodal blocking agent, achieves rate control via its vagal tonic effect, and is more effective when the patient is at rest or in combination with another atrioventricular nodal blocking agent.

Circumstances may exist where selection of specific rate or rhythm strategies may benefit special populations such as pregnant women and those with underlying pulmonary disease, congestive heart failure, acute myocardial infarction, and hyperthyroidism. **(See Table 6, pages 9 and 10.)**

Beta Blockers

In the absence of preexcitation syndromes, beta blockers should be the first drug of choice in patients with congestive heart failure or left ventricular dysfunction, hypertension, and acute coronary syndromes. Beta blockers may be beneficial for postoperative patients who may have new-onset AF/AFL secondary to adrenergic surge.[123,124] Propranolol may be especially beneficial in patients with underlying hyperthyroidism or thyrotoxicosis. Beta blockers should be used with caution in patients with hypotension or acutely decompensated heart failure.

Nondihydropyridine Calcium Channel Blockers

Nondihydropyridine calcium channel blockers (such

Table 6. Evidence-Based Recommendations: Special Circumstances For Pharmacological And Electrical Rate And Rhythm Control Strategies For Atrial Fibrillation[8] (Continued on page 10)

Class of Evidence	Recommendation	Indication (Level of Evidence)
Acute Myocardial Infarction		
Class I Recommendations	DCC	Severe hemodynamic compromise, intractable ischemia, adequate rate control cannot be achieved with pharmacological agents **(Level of Evidence C)**
	IV beta blockers, IV nondihydropyridine calcium channel antagonists	To slow rapid ventricular response in patients who do not display chronic LV dysfunction, bronchospasm, or AV block **(Level of Evidence C)**
	IV amiodarone	To slow rapid ventricular response and improve LV function **(Level of Evidence C)**
Class IIa Recommendations	IV digoxin	Patients with severe LV dysfunction and heart failure **(Level of Evidence C)**
Class III Recommendations	Propafenone, flecainide	Contraindicated **(Level of Evidence C)**
Wolff-Parkinson-White Preexcitation Syndrome		
Class I Recommendations	DCC	Prevent ventricular fibrillation in patients with short anterograde bypass tract refractory period if AF occurs with rapid ventricular response associated with hemodynamic instability **(Level of Evidence B)**
	IV procainamide, IV ibutilide	Rapid AF without hemodynamic instability in association with wide QRS ≥ 120 ms or rapid preexcited ventricular response **(Level of Evidence C)**
Class IIa Recommendations	DCC	Rapid ventricular rates involving conduction over an accessory pathway **(Level of Evidence B)**
	IV flecainide	
Class IIb Recommendations	IV quinidine, procainamide, ibutilide, or amiodarone	Hemodynamically stable patients with AF involving conduction over an accessory pathway **(Level of Evidence B)**
Class III Recommendations	IV digitalis glycosides, beta blockers, or nondihydropyridine calcium channel antagonists	Contraindicated **(Level of Evidence C)**
Hyperthyroidism		
Class I Recommendations	Beta blocker	Control ventricular response rate in patients with AF complicating thyrotoxicosis, unless contraindicated **(Level of Evidence B)**
	Calcium channel blocker	If beta blocker cannot be used **(Level of Evidence B)**
	Oral anticoagulation	Prevent thromboembolism as recommended for AF patients with other stroke risk factors **(Level of Evidence C)**. Once euthyroid state is restored, recommendations for antithrombotic prophylaxis are the same as for patients without hyperthyroidism **(Level of Evidence C)**

Abbreviations: AF, atrial fibrillation; AV, atrioventricular; DCC, direct current cardioversion; IV, intravenous; LV, left ventricular.

as diltiazem and verapamil) are another first-line medication for the treatment of acute AF. They can be useful when there are contraindications to the use of beta blockers for patients with obstructive pulmonary disease or can be used as a second-line choice for thyrotoxicosis when beta blockers cannot be used.

Diltiazem tends to be more popular than verapamil for acute rate control, as verapamil has more potent negative inotropic and vasodilator effects that may lead to hypotension.[120] Aside from esmolol, diltiazem has a faster time of onset than beta blockers[115,124] and has been shown in a randomized controlled trial to be more effective in controlling the ventricular rate than IV amiodarone or digoxin.[125]

Digoxin

Digoxin was once the medication of choice for AF, but it has largely been replaced by more potent atrioventricular nodal blockers.[126] Digoxin has both negative chronotropic and positive inotropic effects, which is particularly useful in patients with congestive heart failure, but the onset of action may take up to 3 hours, and the full effect of digoxin may not be felt for up to 6 hours. It can also be especially useful in hypotensive patients due to its lack of effect on systemic blood pressure.[127,128]

When used in combination with beta blockers and calcium channel blockers, digoxin has a synergistic effect, with improved rate control and expanded use for patients with congestive heart fail-

Table 6. Evidence-Based Recommendations: Special Circumstances For Pharmacological And Electrical Rate And Rhythm Control Strategies For Atrial Fibrillation[8] (Continued from page 9)

Class of Evidence	Recommendation	Indication (Level of Evidence)
Pregnancy		
Class I Recommendations	DCC	Can be performed safely at all stages of pregnancy and is recommended in patients who are hemodynamically unstable and whenever the risk of ongoing AF is considered high for the mother or for the fetus **(Level of Evidence C)**
	Anticoagulation	Administration of an oral vitamin K antagonist is recommended from the second trimester until 1 month before expected delivery **(Level of Evidence B)** Subcutaneous administration of LMWH in weight-adjusted therapeutic doses is recommended during the first trimester and the last month of pregnancy. Alternatively, UFH may be given to prolong the activated PTT to 1.5 times the control **(Level of Evidence B)**
Class IIa Recommendations	Beta blocker, nondihydropyridine calcium channel antagonist	If rate control is necessary, a beta blocker or nondihydropyridine calcium channel antagonist should be considered. During the first trimester of pregnancy, the use of a beta blocker must be weighed against the potential risk of negative fetal effects **(Level of Evidence C)**
Class IIb Recommendations	Digoxin	If rate control is necessary and a beta blocker or nondihydropyridine calcium channel antagonist is contraindicated, digoxin may be considered **(Level of Evidence C)**
	IV flecainide, IV ibutilide	In hemodynamically stable patients with structurally normal hearts, flecainide or ibutilide may be considered to terminate recent-onset AF if conversion is mandatory and DCC is inappropriate **(Level of Evidence C)**
Pulmonary Disease		
Class I Recommendations	Correct hypoxemia and acidosis	Primary therapeutic measure for acute pulmonary illness or exacerbation of chronic pulmonary disease **(Level of Evidence C)**
	Nondihydropyridine calcium channel antagonist	To control the ventricular rate in patients with obstructive pulmonary disease **(Level of Evidence C)**
	DCC	Attempt in patients with pulmonary disease who become hemodynamically unstable **(Level of Evidence C)**
Class III Recommendations	Theophylline and beta-adrenergic agonists	Contraindicated in patients with bronchospastic lung disease with AF **(Level of Evidence C)**
	Beta blocker, sotalol, propafenone, and adenosine	Contraindicated in patients with obstructive lung disease with AF **(Level of Evidence C)**

Abbreviations: AF, atrial fibrillation; AV, atrioventricular; DCC, direct current cardioversion; IV, intravenous; LMWH, low-molecular-weight heparin; LV, left ventricular; PTT, partial thromboplastin time; UFH, unfractionated heparin.

Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibril executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to revise the 2001 guidelines for the management of patients with atrial fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Eur Heart J.* 2006;27(16):1979-2030. By permission of the European Society of Cardiology.

ure.[129-131] The combination of atenolol and digoxin may have the best rate controlling effect; however, this combination can precipitate severe bradycardia and should be used with caution.[125] Verapamil may increase the concentration of digoxin despite its synergist effects.[132]

Amiodarone

Amiodarone, an antiarrhythmic drug, is widely used as a rate-controlling agent, and IV administration can lower the ventricular response in patients with acute-onset AF through atrioventricular nodal blockade via indirect sympatholytic action.[91,133,134] It is a second-line agent due to its slower onset and greater potential for adverse side effects.[125,135] Amiodarone has fewer negative inotropic effects, and it may be a useful alternative agent for those who cannot tolerate beta blockers or calcium channel blockers (such as patients with decompensated congestive heart failure).[77] Amiodarone is a Class III antiarrhythmic agent, and it should be used with caution in those who are not anticoagulated or in those who are at high risk for thrombotic events, as it can promote cardioversion.

Magnesium

Magnesium decreases conduction through the atrioventricular node, and it has been shown in multiple small studies to have some effect in decreasing the ventricular response to AF;[136-141] however, its use is most often recommended as an adjunctive therapy. Magnesium has few negative inotropic effects and is generally well tolerated with few side effects other than flushing, warmth, and tingling. Rapid infusion and large doses may be associated with respiratory muscle fatigue, hypotension, and cardiac pauses.[136] Magnesium may also promote conversion to sinus rhythm, with some studies showing 50% to 60% of patients converted to sinus rhythm.[136,137,139]

Rhythm Control Strategies: Electrical And Pharmacological Cardioversion

The development of new drugs for pharmacological conversion has increased its popularity; however, DCC with biphasic shocks remains more effective. Elective DCC is painful, and it requires procedural sedation or anesthesia. The Ottawa Aggressive Protocol is a rapid ED strategy that has been in use in Canadian EDs for several years. **(See Table 7.)** A prospective review of 660 patient visits using this protocol found successful conversion of 85% of patients with new-onset AF as well as a decreased ED length of stay, few side effects, and low ED recidivism rates with repeat episodes of AF.[94] Additional studies have shown a shorter ED length of stay among patients who are electrically cardioverted compared to those treated with oral or IV antiarrhythmic medications.[106,107]

Table 7. Details Of The Ottawa Aggressive Protocol For Emergency Department Patients With Recent-Onset Atrial Fibrillation[94]

1. Assessment
   - Stable without ischemia, hypotension, or acute CHF?
   - Onset clear and < 48 h?
   - Severity of symptoms?
   - Previous episodes and treatments?
   - Anticoagulated with warfarin and INR therapeutic?

2. Rate control
   - If highly symptomatic or not planning to convert
   - Diltiazem IV (0.25 mg/kg over 10 min; repeat at 0.35 mg/kg)
   - Metoprolol IV (5-mg doses q15min)

3. Pharmacologic cardioversion
   - Procainamide IV (1 g IV over 60 min; hold if SBP < 100 mg Hg)

4. Electrical cardioversion
   - Consider keeping patient NPO x 6 h
   - Procedural sedation and analgesia given by emergency physician (propofol IV and fentanyl IV)
   - Start at 150-200 J biphasic synchronized*
   - Use anterior-posterior pads, especially if not responding

5. Anticoagulation
   - Usually no heparin or warfarin for most patients if onset clearly < 48 h or if therapeutic INR for > 3 wk

6. Disposition
   - Home within 1 h after cardioversion
   - Usually no antiarrhythmic prophylaxis or anticoagulation given
   - Arrange outpatient echocardiography if first episode
   - Cardiology follow-up if first episode or frequent episodes

7. Patients not treated with cardioversion
   - Achieve rate control with diltiazem IV (target heart rate < 100 bpm)
   - Discharge home on diltiazem (or metoprolol)
   - Discharge home on warfarin and arrange INR monitoring
   - Arrange outpatient echocardiography
   - Follow up with cardiology at 4 wk for elective cardioversion

8. Recommend additions to protocol
   - Consider TEE if onset unclear
   - Alternate rhythm-control drugs: propafenone, amiodarone
   - If TEE-guided cardioversion > 48 h, start warfarin
   - If CHADS score ≥ 1, consider warfarin and arrange early follow-up

*Most patients treated with electrical cardioversion in the current study were managed with monophasic cardioversion.

Abbreviations: bpm, beats per minute; CHF, congestive heart failure; INR, international normalized ratio; IV, intravenous; NPO, nil per os (nothing by mouth); SBP, systolic blood pressure; TEE, transesophageal echocardiography.

Stiell IG, Clement CM, Perry JJ, et al. Association of the Ottawa Aggressive Protocol with rapid discharge of emergency department patients with recent-onset atrial fibrillation or flutter. *CJEM*. 2010;12(3):181-191. Reprinted with permission of Decker Publishing, Inc.

Pharmacological Enhancement Of Direct Current Cardioversion

Pretreatment with an antiarrhythmic agent such as amiodarone, flecainide, ibutilide, propafenone, or sotalol may increase the success of DCC and should be considered in patients for whom electrical cardioversion initially fails (Class IIa, LOE C). Alternatively, failure of an antiarrhythmic agent followed by an observation period may be followed with electrical cardioversion in the ED, as demonstrated by the Ottawa Aggressive Protocol.

Selecting Agents for Pharmacological Cardioversion Of AF/AFL

Prior to selecting a pharmacological agent to cardiovert a patient with AF, an assessment of the patient's proarrhythmia risk factors should be conducted. Patients who receive pharmacologic cardioversion should have normal electrolytes and a normal QTc interval. Depressed left ventricular function or underlying structural heart disease may preclude some patients from certain agents. **(See Tables 8 and 9.)**

Since the release of the 2006 ACC/AHA/ECS guidelines, procainamide has been studied for use in cardioversion of new-onset AF/AFL patients in the ED, with successful conversion in 52% to 58% of cases and a low rate of adverse events and no deaths. In a Canadian series, the most common side effect of procainamide administration was temporary hypotension (6.7%-8.5%), and most patients were discharged home from the ED with no episodes of torsades de pointes, stroke, or death.[94,107]

Table 8. Intravenously And Orally Administered Pharmacological Agents For Cardioversion Of Atrial Fibrillation[8]

Drug	Class of Recommendation / Level of Evidence			Route of Administration	Initial Loading Dosage		Potential Adverse Effects
	AF of up to 7-day duration	AF present for > 7 days	AF with Pre-excitation				
Flecainide	Class Ia, LOE A	Class IIb, LOE B	Class IIa, LOE B	Oral	200-300 mg		Hypotension, atrial flutter with high ventricular rate
				IV	1.5-3.5 mg/kg over 10-20 min		
Ibutilide	Class Ia, LOE A	Class IIa, LOE A	Class I, LOE C	IV	1 mg over 10 min; repeat 1 mg when necessary		QT prolongation, torsades de pointes
Propafenone	Class Ia, LOE A	Class IIb, LOE B	NA	Oral	600 mg		Hypotension, atrial flutter with high ventricular rate
				IV	1.5-2.0 mg/kg over 10-20 min		
Dofetilide	Class Ia, LOE A	Class Ia, LOE A	NA	Oral	Creatinine clearance (mL/min)	Dosage (mcg bid)	QT prolongation, torsades de pointes; adjust dose for renal function, body size, and age
					> 60	500	
					40-60	250	
					20-40	125	
Amiodarone	Class IIa, LOE A	Class IIa, LOE A	Class IIb, LOE B	Oral	1.2-1.8 g/d in divided doses until 10 g total		Hypotension, bradycardia, QT prolongation, torsades de pointes, GI upset, constipation, phlebitis (IV)
				IV	5-7 mg/kg over 30-60 min, then 1.2-1.8 g/day continuous IV or divided oral doses until 10 g total		
Procainamide	Class IIb, LOE B	Class IIb, LOE C	Class I, LOE C	IV	1 g IV over 60 min		Hypotension, QT prolongation, torsades de pointes
Quinidine	Class IIb, LOE B	Class IIb, LOE B	Class IIb, LOE B	Oral	0.75-1.5 g in divided doses over 6-12 h, usually with a rate-slowing drug		QT prolongation, torsades de pointes, GI upset, hypotension

Abbreviations: AF, atrial fibrillation; bid, two times per day; GI, gastrointestinal; IV, intravenous; LOE, level of evidence; NA, not applicable.

Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to revise the 2001 guidelines for the management of patients with atrial fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Eur Heart J.* 2006;27(16):1979-2030. By permission of the European Society of Cardiology.

Preexcitation syndromes such as Wolff-Parkinson-White syndrome may be particularly challenging to manage in patients with AF, as the short refractory period of the accessory pathway can result in extremely fast ventricular rates, and use of atrioventricular nodal blocking agents such as beta blockers, calcium channel blockers, and digoxin may induce ventricular fibrillation and are contraindicated.

Synchronized electrical cardioversion is the primary treatment for unstable patients with an accessory pathway or those who present with a very rapid heart rate, even if stable. Class Ia drugs such as procainamide and quinidine, or class Ic drugs such as flecainide, slow conduction through the accessory pathway and prolong the refractory period in the bypass tract, and they can be safely used in patients in rapid AF with Wolff-Parkinson-White syndrome.

Amiodarone may also be used, although it has not been shown to be safer or more effective than procainamide among patients with Wolff-Parkinson-White preexcitation syndrome.[142]

Reducing Stroke Risk

Prevention Of Postconversion Thromboembolism

AF and AFL are associated with an increased long-term risk of stroke and an increased risk of thromboembolism in the postconversion period.[42] Stagnant blood flow in the dysfunctional atria can lead to clot formation in the atria or atrial appendage, which can subsequently lead to embolization and cerebral vascular occlusion prior to or after cardioversion.[42,146] Cardioversion to sinus rhythm may result in atrial "stunning," which is further mechanical dysfunction of the atria that may last up to several weeks, further increasing the risk of thromboembolism even in patients with a negative transesophageal echocardiography prior to cardioversion.[143]

Thromboembolic events in all patients who are cardioverted appear to be as high as 5% to 7% without anticoagulation but can decrease to < 1.6% if cardioversion occurs after 2 to 4 weeks of anticoagulation or shorter-term anticoagulation and a negative screening transesophageal echocardiogram.[64,69,144] The rate of embolic events among patients with spontaneous or active cardioversion within the first 48 hours of AF onset appears to be very similar to the reported incidence of embolism after anticoagulation for 3 to 4 weeks.[144] Some studies, however, have shown that patients may not be able to correctly identify the time of onset of AF based on their symptoms,[23,36,44] and the use of transesophageal echocardiography has shown that a clot may be present in the atrium up to 13% of the time in patients with AF < 72 hours duration; thus, it is recommended that patients be anticoagulated prior to cardioversion and anticoagulated for 3 to 4 weeks after cardioversion unless they are low risk or it is contraindicated.

The following recommendations are excerpted with permission by the European Society of Cardiology from: "ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation-Executive Summary: a Report of the American College Of Cardiology/American Heart Association Task Force On Practice Guidelines and the European Society Of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients with Atrial Fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society:"

"Class I Recommendations For Immediate Cardioversion: For patients with AF of more than 48 hours duration requiring immediate cardioversion

Table 9. Intravenously Administered Pharmacological Agents For Rate And Rhythm Control Of Atrial Fibrillation With An Accessory Pathway[8]

Drug	Class of Recommendation / Level of Evidence	Initial Loading Dosage	Potential Adverse Effects
Procainamide	Class I, LOE C	1 g IV over 60 min	Hypotension, QT prolongation, torsades de pointes
Ibutilide	Class I, LOE C	1 mg over 10 min; repeat 1 mg when necessary	QT prolongation, torsades de pointes
Flecainide	Class IIa, LOE B	1.5-3.5 mg/kg over 10-20 min	Hypotension, atrial flutter with high ventricular rate
Amiodarone	Class IIa, LOE C	150 mg over 10 min	Hypotension, QT prolongation, bradyarrhythmias

Abbreviation: LOE, level of evidence.

Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to revise the 2001 guidelines for the management of patients with atrial fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Eur Heart J.* 2006;27(16):1979-2030. By permission of the European Society of Cardiology.

because of hemodynamic instability, heparin should be administered concurrently (unless contraindicated) by an initial IV bolus injection followed by a continuous infusion in a dose adjusted to prolong the activated partial thromboplastin time to 1.5 to 2 times the reference control value. Thereafter, oral anticoagulation (INR 2.0-3.0) should be provided for at least 4 weeks, as for patients undergoing electrical cardioversion. Limited data support subcutaneous administration of low-molecular-weight heparin in this indication (Level of Evidence C)."

"For patients with AF of 48 hours duration or longer, or when the duration of AF is unknown, anticoagulation (INR 2.0-3.0) is recommended for at least 3 weeks prior to and 4 weeks after cardioversion, regardless of the method (electrical or pharmacological) used to restores sinus rhythm (Level of Evidence B)."

"Class IIa Recommendations: As an alternative to anticoagulation prior to cardioversion of AF, it is reasonable to perform transesophageal echocardiography in search of thrombus in the left atrium or left atrial appendage (Level of Evidence B). For patients with no identifiable thrombus, cardioversion is reasonable immediately after anticoagulation with unfractionated heparin (initial IV bolus injection followed by a continuous infusion in a dose adjusted to prolong the activated partial thromboplastin time to 1.5 to 2 times the reference control value until oral anticoagulation has been established) (Level of Evidence B). Limited data support subcutaneous administration of low-molecular-weight heparin in this indication (Level of Evidence C). For patients in whom thrombus is identified by transesophageal echocardiography, oral anticoagulation (INR 2.0-3.0) is reasonable for at least 3 weeks prior to and 4 weeks after restoration of sinus rhythm and a long period of anticoagulation may be appropriate even after apparently successful cardioversion because the risk of thromboembolism often remains elevated in such cases (Level of Evidence C)."

Decreasing Thromboembolic Risk

Multiple studies have been conducted to further risk stratify patients with AF/AFL to detect who may benefit from anticoagulation therapy to decrease stroke, transient ischemic attack, and systemic thromboembolism. Patients at low risk (defined as < 2% stroke risk per 100 patient-years with aspirin as antithrombotic therapy) gain little benefit with oral anticoagulation with vitamin K antagonists (warfarin), and the risk of bleeding from vitamin K antagonists outweighs the potential benefit of stroke reduction.[145] For patients at high risk (> 4% stroke risk per 100 patient-years), vitamin K antagonists have consistently been shown to improve quality-adjusted survival over aspirin with an acceptable bleed rate.[145,146] Vitamin K antagonists decrease stroke risk by 66%, whereas antiplatelet therapy with aspirin 81 mg to 325 mg decreases stroke risk by only 22%.[147]

For those who cannot take vitamin K antagonists, adding clopidogrel to aspirin as an alternative to vitamin K antagonists provides some additional stroke risk reduction compared to aspirin alone (6.8% vs 7.6%/y); however, there is a higher rate of major bleeding in patients receiving combination therapy compared to those with aspirin alone (2.0% vs 1.3%/y).[148] This combination therapy is not as effective to reduce vascular events as vitamin K antagonist use (5.60% vs 3.93% annual risk);[149] however, there is an increased risk of intracranial hemorrhage among warfarin users (0.4%).[150]

The Novel Oral Anticoagulants

Prior to the United States Food and Drug Administration (FDA) approval of the direct thrombin inhibitor (DTI) dabigatran (Pradaxa®) in 2010, vitamin K antagonists were the only available oral anticoagulant options. Since then, rivaroxaban (Xarelto®), a factor Xa inhibitor, received FDA approval in 2011, apixaban (Eliquis®) received approval in 2012, and edoxaban (Lixiana®) is currently undergoing phase III clinical trials. Dabigatran, rivaroxaban, and apixaban have been shown to be noninferior to warfarin with regards to stroke and systemic embolism, with favorable results in safety outcomes (including major bleeding).[151-156]

Dabigatran was reviewed in the 2011 ACC/AHA/ESC update, receiving a Class I, Level of Evidence B recommendation as a useful alternative to warfarin to prevent thromboembolic events. Subgroup analysis of the RE-LY (Randomized Evaluation of Long-term anticoagulant therapY) trial examining patients who were cardioverted showed that, at 30 days, dabigatran was noninferior to warfarin in preventing thromboembolic events in patients anticoagulated with dabigatran prior to cardioversion.[157] Decision analysis models suggest that dabigatran may be a cost-effective strategy to reduce thromboembolic events for people with AF at a high risk of stroke or hemorrhage unless INR control with warfarin was excellent.[158]

Stroke Risk Stratification For Preventing Thromboembolism

In 2001, the CHADS2 (Congestive heart failure, Hypertension, Age, Diabetes mellitus, prior Stroke, transient ischemic attack, or thromboembolism) score was derived by expert consensus to simplify the determination of stroke risk by combining high-risk patient factors that had been previously identified into a simple scoring mechanism to predict thromboembolic risk and need for anticoagulation therapy.[159]

The revised CHADS2 defines low risk as a score of 0, moderate risk as a score of 1, and high risk as a score ≥ 2. Multiple validation studies have been

published for CHADS2; however; the high level of heterogeneity among groups in the limited studies included led the authors to conclude that the results should be used cautiously, and further studies should be performed to guide antithrombotic therapy. One such risk-factor-based approach includes the CHADS2-VASc risk stratification scheme. This scoring system was created by refining the 2006 Birmingham/NICE[160] criteria and adding some of the less well-validated risk factors into a scoring system.[161] **(See Table 10.)**

Multiple studies have validated the CHADS2-VASc scoring system, finding that use of this scoring system significantly improved the predictive value of the CHADS2 scoring system and improved classification of patients at very low, low, and intermediate risk of stroke.[163-166] The CHADS2-VASc scoring system has been incorporated into the ESC and Canadian Cardiovascular Society guidelines, but it has not been adopted into the ACC/AHA guidelines for stroke prevention. **(See Table 11.)** The ESC guidelines suggest using CHADS2 as an initial risk

Table 10. CHADS2/CHADS2-VASc Scoring[162]

Risk Factor	Score
Congestive heart failure/left ventricular dysfunction	1
Hypertension	1
Age > 75 years	2
Diabetes mellitus	1
Stroke/transient ischemic attack/thromboembolism	2
Vascular disease (prior myocardial infarction, peripheral artery disease, or aortic plaque)	1
Age 65-74 years	1
Sex **c**ategory (female gender)	1

Low risk, 0; moderate risk, 1; high risk, ≥ 2.

Reproduced with permission from the American College of Chest Physicians. Lip GY, Nieuwlaat R, Pisters R, et al. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the Euro heart survey on atrial fibrillation. *Chest*. 2010;137(2):263-272.

Table 11. Evidence-Based Recommendations: ACC/AHA/ESC Risk Stratification Recommendations For Preventing Thromboembolism[8]

Class of Evidence	Recommendation	Indication
Class I	No anticoagulation	Lone AF or contraindications to anticoagulation (LOE A)
	Vitamin K antagonist*†	• Rheumatic mitral stenosis (LOE A) • Prior thromboembolism (stroke, TIA, or systemic embolism) (LOE A) • 2 or more moderate risk factors (LOE A): ▪ Congestive heart failure or ejection fraction < 35% ▪ Hypertension ▪ Age ≥ 75 years ▪ Diabetes • AF with mechanical valves; target of intensity should be based on type of prosthesis, maintaining an INR of at least 2.5 (LOE B)
Class IIa	Aspirin or vitamin K antagonist*†	• Nonvalvular AF with 1 moderate risk factor (LOE A): ▪ Hypertension ▪ Age ≥ 75 years ▪ Diabetes ▪ Heart failure or impaired left ventricular function • Nonvalvular AF with 1 or more less well-validated risk factor (LOE B): ▪ Age 65-74 years ▪ Female gender ▪ Coronary artery disease

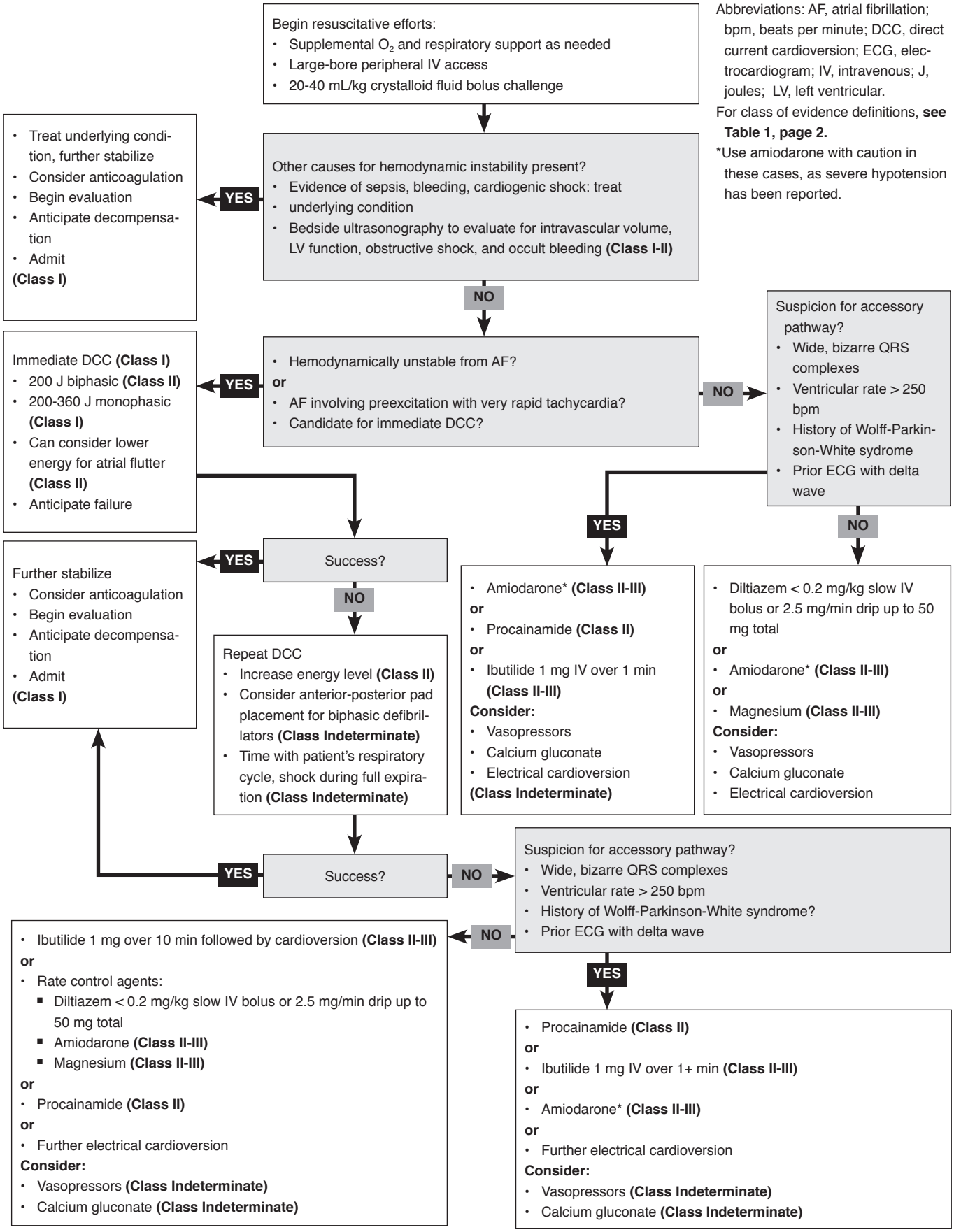
*Dabigatran may be used in place of vitamin K antagonist (Class I, LOE B). Recently FDA-approved rivaroxaban may be considered in place of vitamin K antagonist, but it was not included in the 2011 ACC/AHA/ESC update.

†Combination therapy with clopidogrel and aspirin may be considered in patients with AF in whom oral anticoagulation with warfarin is considered unsuitable (due to patient preference or ability to sustain anticoagulation) (Class IIa, LOE B).

Abbreviations: ACC, American College of Cardiology; AF, atrial fibrillation; AHA, American Heart Association; ESC, European Society of Cardiology; INR, international normalized ratio; LOE, level of evidence; TIA, transient ischemic attack.

Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to revise the 2001 guidelines for the management of patients with atrial fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Eur Heart J.* 2006;27(16):1979-2030. By permission of the European Society of Cardiology.

Clinical Pathway For Initial Approach To The Hemodynamically Unstable Rapid Atrial Fibrillation Patient

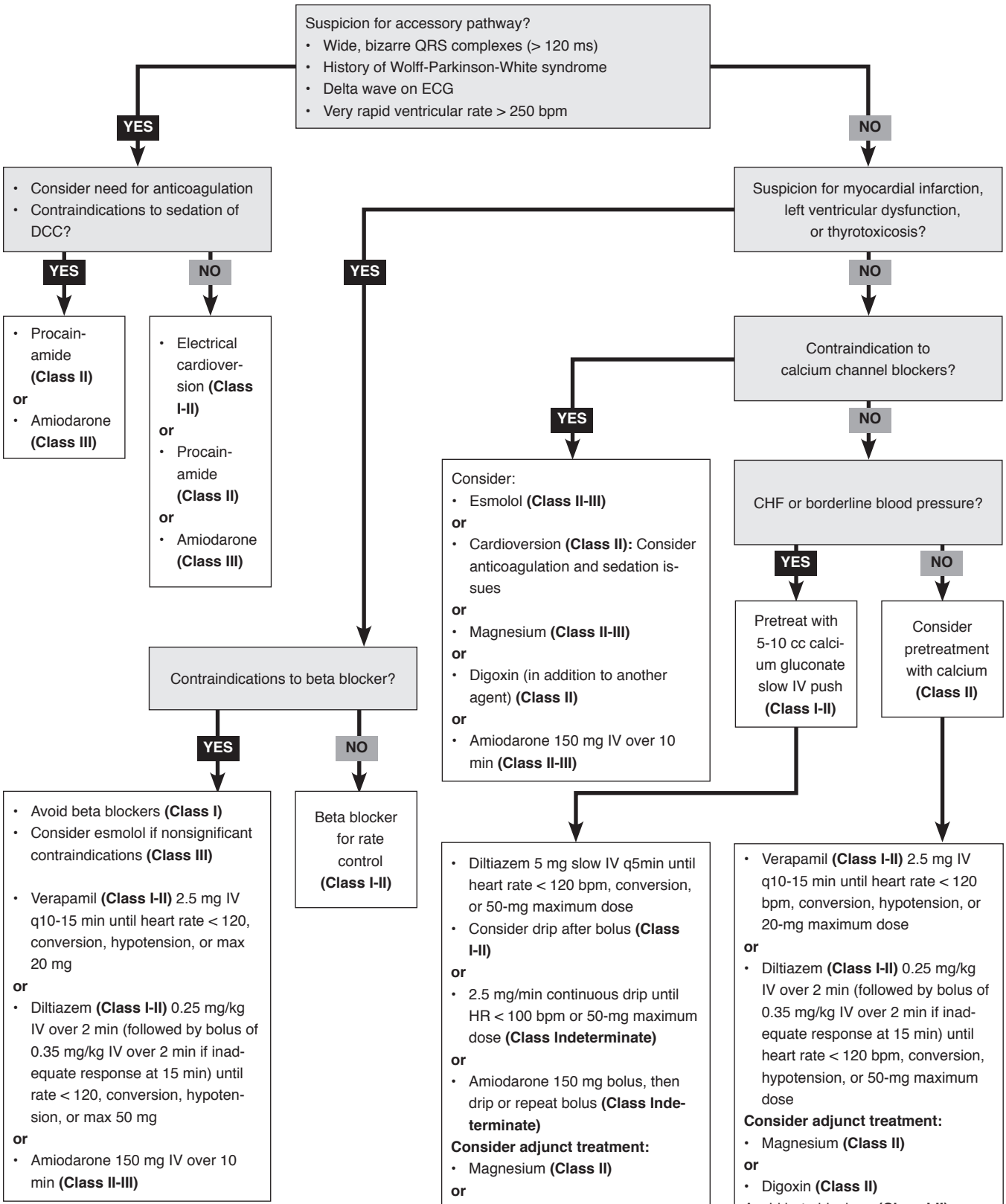


Abbreviations: AF, atrial fibrillation; bpm, beats per minute; DCC, direct current cardioversion; ECG, electrocardiogram; IV, intravenous; J, joules; LV, left ventricular.

For class of evidence definitions, **see Table 1, page 2.**

*Use amiodarone with caution in these cases, as severe hypotension has been reported.

Clinical Pathway For Rate Control For Stable Patients With New-Onset Atrial Fibrillation With Rapid Ventricular Response



Abbreviations: bpm, beats per minute; CHF, congestive heart failure; DCC, direct current cardioversion; ECG, electrocardiogram; IV, intravenous.

For class of evidence definitions, **see Table 1, page 2.**

stratification tool to identify high-risk individuals; those with CHADS2 scores ≥ 2 should be placed on chronic oral anticoagulation. In patients with a CHADS2 score of 0-1, or when a more detailed stroke risk assessment should be conducted, the CHADS2-VASc score is recommended in order to include additional risk factors as part of the criteria.

Bleeding Risk In Anticoagulation Therapy

While anticoagulation has been shown to decrease the risk of ischemic stroke, patients are more likely to experience major bleeding. A systematic review of physicians' attitudes regarding anticoagulation found that physicians were reluctant to prescribe warfarin for elderly patients due to bleeding concerns, despite the increased benefit in these patients compared to younger patients[167-170] and some evidence of safe use in the elderly on vitamin K antagonists.[161,171-173]

To provide objective risk stratification to assess bleeding risk, various tools have been developed (eg, HAS-BLED). HAS-BLED was created by multivariate analysis of risk factors associated with bleeding among patients with AF/AFL undergoing antithrombotic therapy with vitamin K antagonists[174] and has been validated by multiple studies and shown to have better predictive value than other tools.[175-179]

Analyses of the HAS-BLED score have shown that for the risk of bleeding to outweigh the benefit of anticoagulation, the HAS-BLED score should exceed the stroke risk calculated by CHADS2. **(See Table 12.)** For example, for the majority of high-risk patients who should be anticoagulated (CHADS2 ≥ 2), the HAS-BLED score must also exceed 2 for the potential harm of bleeding to outweigh the potential benefit of stroke prevention.The use of HAS-BLED is recommended by the Canadian Cardiovascular Society and the ESC but has not been adopted into the ACC/AHA guidelines.

Disposition

Strategies to determine which patients who present to the ED in new-onset AF/AFL can be safely discharged home are becoming increasingly important. Multiple studies have found the total direct cost per patient with AF to be much higher than patients without AF, at roughly $20,670 each year, in contrast to the average healthcare cost of $11,965 among patients without AF.[7] Each documented recurrence of AF increases annual healthcare costs by approximately $1600.[180] The level of acute care can vary among individuals, but the principal cost driver was found to be inpatient service charge.[181] A study done in Canada showed the average length of hospitalization for an AF patient was roughly 5.7 days.[182] The total monetary burden is expected to increase over the next few decades, especially since the anticipated number of older adults with AF is expected to double over the next 3 decades.[183]

Few studies of EDCV and discharge have been performed. They are mostly retrospective, but all have had favorable results. EDCV has a success rate of 86% to 92%, with decreased overall hospital length of stay, few complications, and high patient satisfaction, with only a 3% to 17% recidivism rate for relapsed AF.[94,95,101,102,105]

ED disposition is addressed only in the Canadian Cardiovascular Society guidelines, which recommend admission of new-onset AF only for patients with decompensated heart failure or myocardial ischemia or for patients who are highly symptomatic and in whom adequate rate control cannot be achieved. It is recommended by the Canadian Cardiovascular Society that other patients should be discharged after rate or rhythm control with outpatient cardiology consultation.

It is suggested that truly low-risk patients may be able to go home safely if they meet the following criteria:

- < 60 years of age
- No significant comorbid disease
- No clinical suspicion for pulmonary embolism or myocardial infarction
- Conversion in ED or rate control

Urgent cardiology follow-up is mandatory for all patients with new-onset AF who are being discharged.

Table 12. HAS-BLED Scoring[176]

HAS-BLED Acronym	Score	HAS-BLED Score*	Bleeds per 100 patient-years
Hypertension	1	1	1.13
Abnormal renal and liver function (1 point each)	1 or 2	2	1.02
Stroke	1	3	1.88
Bleeding history or predisposition	1	4	3.74
Labile INRs	1		8.70
Elderly (> 65 years of age)	1		
Drugs or alcohol concomitantly (1 point each)	1 or 2		

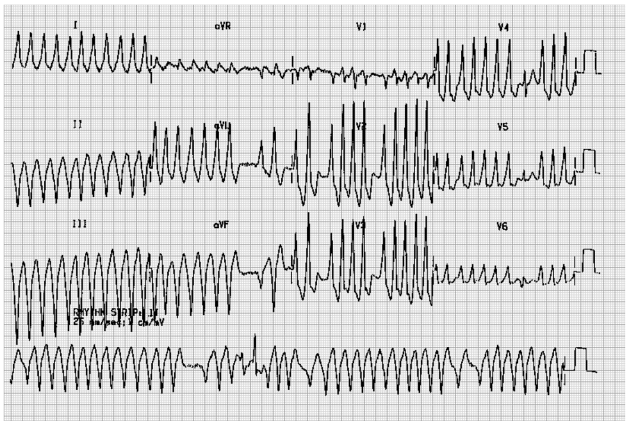
*Insufficient data for analysis of bleeds with HAS-BLED score ≥ 5.
Abbreviation: INR, international normalized ratio.
Reproduced with permission from the American College of Chest Physicians. Pisters R, Lane DA, Nieuwlaat R, et al. A novel user-friendly score (HAS-BLED) to assess 1-year risk of major bleeding in patients with atrial fibrillation: the Euro heart survey. *Chest.* 2010;138(5):1093-1100.

Summary

AF/AFL are the most common cardiac arrhythmias encountered. As the United States population ages, we will likely encounter more cases of AF/AFL, and complications from these cardiac arrhythmias (such as thromboembolic events). Emergency clinicians must possess evidence-based knowledge of multiple approaches to rate or rhythm control in order to manage the AF/AFL patient, whom and when to anticoagulate, and cost-effective strategies to evaluate new-onset AF/AFL.

Case Conclusions

The first patient's ECG (shown below) shows AF with preexcitation consistent with Wolff-Parkinson-White syndrome. Because the patient was hemodynamically stable, you obtained 2 large-bore peripheral IV lines and began an infusion of procainamide, coadministering a normal saline bolus. She converted to normal sinus rhythm and felt much improved, with normal repeat vital signs. Her repeat ECG showed a short PR interval with delta waves. She had no prior history of this, no past medical history, and a CHADS2 score of 0. You consulted cardiology for an electrophysiology study, and she was successfully ablated and discharged home.



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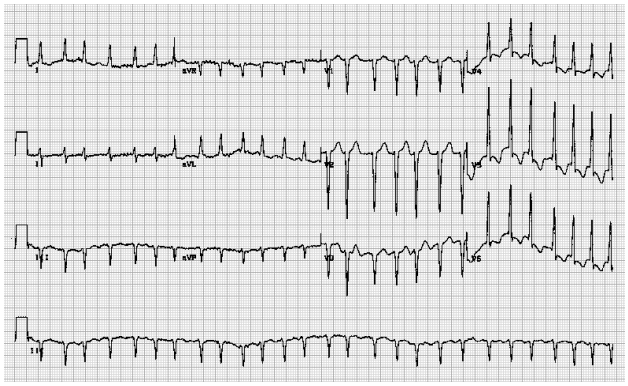
You thought the second patient's hypotension might have been due to sepsis; however, the irregular rhythm on the monitor appeared to be rapid AF, and the loss of atrial kick and decreased ejection fraction may have contributed to his hypotension. While the ECG was being performed, you "pressure-bagged" 2 500-cc normal saline boluses through 18-g peripheral IVs and obtained slightly improved hemodynamics, with a heart rate of 140 beats/min per minute and a blood pressure of 102/64 mm Hg but no improvement in his mental status. You performed a bedside ultrasound that showed a 2.5-cm inferior vena cava without respiratory variation, no pericardial effusion, a normal LV:RV ratio, no free fluid in the abdomen, and a normal-appearing aorta. The ECG (shown below) confirmed AF, and you placed the defibrillator pads on the patient in an anterior-posterior position. You direct current cardioverted him with a biphasic cardioverter using 200 J. The patient then became more awake, he was able to converse with you, and his blood pressure rose to 125/73 mm Hg. He had a CHADS2 score of 3 and no contraindications to anticoagulation, so you began a heparin drip and admitted him to a monitored unit.



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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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Risk Management Pitfalls For Atrial Fibrillation (continued on page 21)

1. **"The patient denied any chest pain, so I sent her home after she spontaneously cardioverted."**
   Unfortunately, the patient was a 62-year-old female with diabetes who had a prior history of myocardial infarction. Had you compared her prior ECG, you would have noted new ischemic changes. Although she spontaneously converted, ECG changes and significant cardiac risk factors should have prompted an admission to further evaluate for ischemia.

2. **"The ibutilide worked great. The patient felt much better and wanted to go home immediately."**
   While ibutilide works to convert AF/AFL approximately 40% to 50% of the time, it has significant risks—most notably an 8% risk of torsades de pointes and other ventricular tachyarrhythmias, which may be mitigated by pretreatment with IV magnesium sulfate. Use of this drug requires a 4-hour period of monitoring after administration.

3. **"The QRS complexes looked a little bizarre, but I figured she had an underlying bundle branch block. I didn't think a 20-mg diltiazem bolus would cause her to go into cardiac arrest."**
   Wide, bizarre QRS complexes with very rapid ventricular rates up to 300 beats per minute should lead you to suspect preexcitation such as Wolff-Parkinson-White syndrome, as should prior ECGs with delta waves, history of an accessory pathway, or very young patients with new-onset AF. Urgent electrical cardioversion should be performed for patients who are hemodynamically unstable with AF/AFL involving conduction over an accessory pathway, while IV procainamide, ibutilide, or amiodarone may be considered for hemodynamically stable patients.

4. **"She was hypotensive, so I gave calcium gluconate before giving the commonly quoted starting dose of diltiazem: a 20-mg bolus. It is unfortunate that she became profoundly hypotensive and went into cardiac arrest, but I did nothing wrong."**
   Pretreatment with calcium may potentially help blunt the hypotensive effects of diltiazem; however, had you started with a lower dose and titrated it slowly, you may have been able to prevent the hypotension and cardiac arrest. You can also consider using vasopressors, cardioversion, or an amiodarone drip to minimize hypotension and prevent decompensation.

5. **"When the diltiazem didn't give a good response, I decided to try metoprolol. I believe her complete heart block was from the acute coronary syndrome she was having, not what I did."**
   Combining IV beta blockers and calcium channel blockers can result in hypotension and can precipitate dysrhythmia and complete atrioventricular nodal blockade. It is safe to give 1 of these 2 classes of drugs intravenously—cautiously—if the patient is on an oral version of the other class, but giving both intravenously in a short time period could potentially lead to decompensation.

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Risk Management Pitfalls For Atrial Fibrillation (continued from page 20)

6. "I thought the patient might be having acute coronary syndrome, so I used a beta blocker for its beneficial effects. I was not expecting the patient to decompensate around that same time."

While there are advantages to using a beta blocker in new-onset AF in the setting of acute coronary syndromes or thyrotoxicosis, it is important to remember any contraindications to specific drug classes. Had you asked the patient about a history of asthma and her recent increased use of home nebulizers you might have considered a short-acting beta blocker such as esmolol or a calcium channel blocker instead.

7. "Sure, her AF was 1 week old, but I obtained a transesophageal echocardiogram that showed no left atrial clot, so I cardioverted the patient and sent her home. She was just one of those unfortunate people who had a thromboembolic event."

Although there is some suggestion in the literature that the method you used might be reasonable, the data suggest that anticoagulation would be required even with a negative transesophageal echocardiogram in this situation. If someone has been in AF for more than 48 hours, transesophageal echocardiogram may not show a clot, but there may still be as high as a 2% incidence of thromboembolism after conversion due to atrial stunning and dysfunction after cardioversion.

8. "She didn't look bug-eyed to me."

In the elderly, thyrotoxicosis can present very atypically, without the common findings that usually occur in younger patients. A thyroid-stimulating hormone screening is a reasonable test in patients > 55 years of age with new-onset AF.

9. "The patient was hypotensive, so I tried cardioversion. I couldn't get him to cardiovert after multiple attempts with 200 J, so I gave IV metoprolol to slow the heart rate down in hopes that the decreased rate would improve ventricular filling and increase his blood pressure. I couldn't believe that it worsened his blood pressure and he ended up going into cardiac arrest."

Failed cardioversion may occur in patients with long-standing AF/AFL, and pretreatment with an antiarrhythmic such as amiodarone may decrease the defibrillation threshold and improve success of cardioversion. Atrioventricular nodal blocking agents may slow the rate down, but this does not increase the "atrial kick" contribution to ventricular filling; thus, atrioventricular nodal blocking agents will likely only exacerbate the hypotension.

10. "She was altered and couldn't tell me how long she had been in AF. I didn't want to cardiovert her and cause a stroke, so I gave diltiazem."

The patient was showing evidence of poor perfusion with altered mental status, cool, clammy skin, and hypotension, and she needed immediate electrical cardioversion. Heparin should be started as soon as possible after cardioversion unless there is a significant contraindication.

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CME Questions

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1. **In AF, the P-wave:**
   a. Is absent
   b. Is buried in the QRS complex
   c. Precedes every QRS complex
   d. Has a sawtooth pattern

2. **AFL is characterized by:**
   a. Irregularly irregular ventricular rhythm
   b. Atrial rate of 250-350 beats/min
   c. Absent P-wave
   d. Ventricular rate between 180 and 250 beats/min

3. **All patients who are hemodynamically unstable and in AF should be immediately cardioverted.**
   a. True
   b. False

4. **Strategies to rate control hypotensive patients in rapid AF include all of the following EXCEPT:**
   a. Pretreatment with calcium gluconate
   b. 20 mL/kg crystalloid bolus infusion
   c. Diltiazem 2.5 mg/min continuous drip until heart rate < 100 beats/min or 50 mg total dose
   d. Diltiazem 3 mg/kg IV push

5. **Nondihydropyridine calcium channel blockers such as diltiazem and verapamil are first-line rate control medications for patients with which of the following conditions?**
   a. Thyrotoxicosis
   b. Asthma exacerbation
   c. Congestive heart failure
   d. Contraindication to beta blockers

6. **For a patient with Wolff-Parkinson-White pre-excitation syndrome, which of the following medication is safest to use?**
   a. Esmolol
   b. Diltiazem
   c. Magnesium
   d. Procainamide

7. **Routine transthoracic echocardiography should be performed on every patient who presents to the ED with new-onset AF.**
   a. True
   b. False

8. **Failed electrical cardioversion is associated with:**
   a. Thyrotoxicosis
   b. Long-standing AF
   c. Dilated left atrium
   d. All of the above

9. **Which of these agents has shown to be the fastest at achieving rate control?**
   a. Amiodarone
   b. Esmolol
   c. Digoxin
   d. Metoprolol

10. **Which of the following is the most likely explanation for a thromboembolic event after cardioversion?**
    a. Failure to detect a left atrial clot on transesophageal echocardiogram performed before cardioversion
    b. Atrial "stunning" and mechanical dysfunction following electrical cardioversion
    c. Dabigatran, which is inferior to warfarin in preventing thromboembolic events, was given prior to cardioversion

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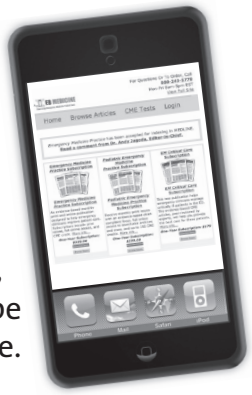

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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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