Management of Acute Atrial Fibrillation and Atrial Flutter in Non-Pregnant Hospitalized Adults

**Patient Population.** Adult hospitalized patients with Atrial Fibrillation and Flutter. This guideline excludes pregnant women.

**Objectives.** The purpose of these inpatient care guidelines is to provide an evidence-based blueprint for the acute care of patients with atrial fibrillation (AF) and atrial flutter (AFL) at the University of Michigan Health System. It is hoped that this standardization of care will result in improved patient outcomes, shorter length of hospital stay, lower readmission rates, and overall cost savings for the system. This document will discuss evaluation of patients with new onset and recurrent AF/AFL, including indications for admission, rate vs. rhythm control strategies, and plan of care following patient’s discharge.

**Key Points**

**Clinical Presentation**
Patients presenting with palpitations, irregular pulse, chest pain, dyspnea, fatigue, lightheadedness, syncope, cardio-embolic disease and new or recurrent heart failure should be evaluated for AF/AFL. While AF may be asymptomatic and found incidentally, AFL is usually highly symptomatic.

**Diagnosis**
Electrocardiogram (ECG) is essential in the diagnosis of AF/AFL. The initial evaluation is summarized in Table 1 and should include:

- Physical exam
- Laboratory evaluation: CBC, basic metabolic profile, magnesium, thyroid-stimulating hormone, and cardiac enzymes as indicated
- Imaging: Chest X-ray, echocardiogram
- Continuous telemetry monitoring in the hospital

**Treatment**
Initial treatment of AF/AFL depends on hemodynamic stability:

Unstable AF/AFL (Figure 1)
- Begin resuscitation and consider other conditions contributing to instability
- If instability due to AF/AFL - immediate direct current cardioversion

Stable AF/AFL (Figure 2):
- For ED patients: Screen for early cardioversion in the Emergency Department (Figure 4)
- Administer rate controlling agents as indicated (Table 4) – [I, B]
  - EP consult for uncontrolled rate despite adequate trial of rate controlling agents
- Consider the appropriateness of a rhythm control strategy (Table 3) – [I, B]
  - If rhythm control strategy is appropriate/desired, consult EP and start immediate anticoagulation (Figure 3)
- Consider anticoagulation based on CHA2DS2-VASc score (Table 2, Figure 3) – [I, A]
  - The choice of anticoagulant will depend on the patient’s clinical circumstances and renal function (Figure 3)
  - Obtain Neurology consult prior to initiation of anticoagulation for patients with recent ischemic stroke within the prior two weeks
  - Patients with valvular disease and those requiring concomitant treatment with dual antiplatelet therapy should be anticoagulated with warfarin
  - Target-specific oral anticoagulants are preferred over warfarin in many cases

*Strength of recommendation:*
I = generally should be performed; II = may be reasonable to perform; III = generally should not be performed.

*Levels of evidence reflect the best available literature in support of an intervention or test:*
A=randomized controlled trials; B=controlled trials, no randomization; C=observational trials; D=opinion of expert panel.
Figure 1: Acute management of UNSTABLE atrial fibrillation and atrial flutter (AF/AFL)

**UNSTABLE AF**, defined as HR >120 at rest and signs of instability, including but not limited to:
- SBP < 90 with altered mental status, lightheadedness, chest pain, or shortness of breath
- Signs of acute cardiac ischemia [chest pain, ECG changes, troponin elevation]
- Signs of acute heart failure [acute respiratory distress, pulmonary edema]

**Begin resuscitation**
- IV access
- IV fluids, if appropriate
- Supplemental O2 and respiratory support, as needed
- Vital signs and telemetry monitoring
- Call for support (RRT or code), as needed
- Plan transfer to telemetry unit or higher level of care

**Consider other conditions that may be contributing to instability, including but not limited to:**
- Infection / sepsis
- Hypovolemia / bleeding
- Respiratory failure / hypoxemia
- ACS
- PE

**Begin initial evaluation**
- 12 lead ECG
- Labs: CBC, COMP, troponin, BNP, TSH
- If appropriate: UA/urine culture, blood cultures, CXR

Is instability secondary to another contributory condition?

- **Yes**
  - Treat underlying condition with consideration for DCCV if deemed appropriate.
  - If rate control needed as patient stabilizes, (see Figure 2)

- **No**
  - Immediate direct current cardioversion (DCCV)
    - Anterior / posterior defibrillation pad placement recommended
    - Call for support (in the hospital, call for RRT)
    - Consider sedation / analgesia before DCCV
    - Energy selection:
      - For A-fib, start with 200 J, synchronized, increased to 360 J
      - For A-flutter, start with 50 J, synchronized, increased to 200 J

**Successful DCCV?**
- **Yes**
  - Consult EP
  - Consider anticoagulation (see Table 2 and Figure 3)
  - Obtain TTE

- **No**
  - Escalate resuscitation (provide respiratory support, call code if needed)
  - Consult cardiology fellow stat for possible CCU transfer
  - Consider trial of amiodarone [150 mg over 10 minutes, then 1 mg/min IV infusion] if blood pressure tolerates (See Table 4 for ongoing dosing)
  - Consult EP

ACS: Acute coronary syndrome; BNP: Brain natriuretic peptide; CBC: complete blood count; CCU: Cardiac Care Unit; COMP: comprehensive metabolic panel; CXR: chest radiograph; ECG: electrocardiogram; EP: electrophysiology; HR: heart rate; IV: intravenous; J: Joules; PE: Pulmonary embolism; RRT: rapid response team; SBP: systolic blood pressure; TSH: thyroid stimulating hormone; UA: urinalysis.

Figure 2: Acute management of STABLE atrial fibrillation and atrial flutter with rapid ventricular response

1.
See Figure 2 notes on following page
Figure 2 Notes:

1 If BP does not tolerate these medications, see Table 4 (medications for rate control) and consider DCCV or EP/General Cardiology consult.

   Also, IV calcium channel blockers and IV beta blockers are not usually combined- if one is not effective, change to the other.

2 If patient spontaneously converts to normal sinus rhythm:
   • If in the ED, provide outpatient EP follow-up within 2 weeks
   • If inpatient, consider consulting EP in the hospital
   • If a postoperative patient, consult General Cardiology (if AF/AFL is sustained for > 24 hours)
   • Consider a rate control agent (Table 4), depending on the pre- and post-conversion heart rate
   • Consider the use of a 3 week event monitor after discharge to identify paroxysmal AF/AFL

   **In all cases, consider anticoagulation (see Table 2 and Figure 3)**

3 For patients with decompensated systolic heart failure: Consult cardiology, and consider digoxin or amiodarone for rate control.

Figure 3: Management of anticoagulation therapy in atrial fibrillation and atrial flutter

1 This algorithm assumes that there are no contraindications to anticoagulant therapy.
2 Enoxaparin can be substituted for IVUH in patients with CrCl > 30, if preferred.
3 For additional information about combining anticoagulation and antiplatelet agents, see text (Anticoagulation- Special Populations for Anticoagulation)
4 For CHA2DS2-VASC score of 1, any of the following may be appropriate: oral anticoagulation (as per the algorithm above), aspirin, or no treatment. Consider bleeding risk and patient preference.

IVUH = intravenous unfractionated heparin, TSOAC = target-specific oral anticoagulant, CrCl = creatinine clearance, AKI = acute kidney injury
Figure 4: Emergency Department screening for early cardioversion of atrial fibrillation and atrial flutter

Does the patient have any of these features?
- SBP < 90 + one of the following: altered mental status, lightheadedness, chest pain (CP), shortness of breath (SOB)
- signs of acute cardiac ischemia (CP, ECG changes, + troponin)
- signs of acute heart failure (pulmonary edema or acute respiratory distress)

Yes → See Figure 1 Unstable Patient algorithm

No → Does the patient have any of these features?
- chronically in AF/AFL, now rapid
- known onset AF/AFL > 48 hours (or unknown onset) and not anticoagulated for > 3 weeks
- age > 70
- history of Congestive Heart Failure (CHF)
- history of Coronary Artery Disease(CAD)
- ED attending preference for rate control

Yes → See Figure 2 Stable Patient algorithm

No → Does the patient meet these criteria?
No failed prior cardioversion attempts AND either:
- a known onset of < 48 hours
- a known onset of > 48 hours in a patient that has been effectively anticoagulated for > 3 weeks

Yes → See Figure 2 Stable Patient algorithm

No → Rhythm Control Algorithm

1. Consider pharmacologic cardioversion
   • procainamide* 1 gm IV over 60 minutes
   Hold for SBP < 90 mm Hg

   Yes → Success
   No → 2. Electrical cardioversion
   • procedural sedation per ED physician
   • 200 J synchronized cardioversion (AF)
   • 50 J synchronized cardioversion (AFL)
   • repeat with higher energy prn (360J for AF, 200 J for AFL)

   Yes → Success
   No → Monitor x 3 hours for recurrence
   Anticoagulate per CHA2DS2-VASc (Table 2, Figure 3)
   Referral for outpatient TTE (if none x 6 months)
   Referral for outpatient EP consult
   Discharge home
   Consider rate control agent, depending on pre-treatment heart rate

* Procainamide
Contraindications:
-- renal failure (CrCl <30)
-- hypotension
-- lupus
-- severe liver disease

CrCl: creatinine clearance; SBP: Systolic blood pressure.
Table 1: Diagnostic evaluation of atrial fibrillation and atrial flutter

- Current electrocardiogram (ECG)
- Complete physical exam
- Current Basic Metabolic Panel, Magnesium, complete blood count (CBC)
- Current thyroid stimulating hormone (TSH)
- Chest X-ray (CXR)
- Serial cardiac enzymes (Troponin, CK, CK-MB) as indicated
- Continuous telemetry monitoring
- Echo within the past 6 months to assess for the presence and severity of structural heart disease

Table 2: CHA2DS2-VASc score and annual stroke risk

<table>
<thead>
<tr>
<th>C</th>
<th>Congestive heart failure (or Left ventricular systolic dysfunction)</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)</td>
<td>1</td>
</tr>
<tr>
<td>A2</td>
<td>Age ≥75 years</td>
<td>2</td>
</tr>
<tr>
<td>D</td>
<td>Diabetes Mellitus</td>
<td>1</td>
</tr>
<tr>
<td>S2</td>
<td>Prior Stroke or TIA or thromboembolism</td>
<td>2</td>
</tr>
<tr>
<td>V</td>
<td>Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>Age 65–74 years</td>
<td>1</td>
</tr>
<tr>
<td>Sc</td>
<td>Sex category (i.e. female gender)</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHA2DS2-VASc Score</th>
<th>Stroke Risk (%/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td>2</td>
<td>2.2</td>
</tr>
<tr>
<td>3</td>
<td>3.2</td>
</tr>
<tr>
<td>4</td>
<td>4.0</td>
</tr>
<tr>
<td>5</td>
<td>6.7</td>
</tr>
<tr>
<td>6</td>
<td>9.8</td>
</tr>
<tr>
<td>7</td>
<td>9.6</td>
</tr>
<tr>
<td>8</td>
<td>6.7</td>
</tr>
<tr>
<td>9</td>
<td>15.2</td>
</tr>
</tbody>
</table>

Table 3: When to consider a rhythm control strategy for atrial fibrillation/flutter

- First occurrence of symptomatic AF/AFL
- Occurrence or recurrence of AF due to reversible cause (e.g. hyperthyroidism, pulmonary embolism, postoperative state, pneumonia, acute coronary syndrome/acute myocardial infarction ACS/AMI)
- Hospital readmissions for AF/AFL or management of AF-related comorbidities
- Atrial Tachyarrhythmia-related symptoms despite adequate rate control, or inability to achieve adequate rate control
- Cardiomyopathy presumed to be secondary to tachycardia
- Younger patients (age < 65), even with minimally symptomatic or asymptomatic AF/AFL
Table 4: Pharmacologic agents useful for rate control in patients with AF/AFL

<table>
<thead>
<tr>
<th>Drug</th>
<th>When to consider use? [level of recommendation / level of evidence]</th>
<th>Warnings/Contraindications</th>
<th>Intravenous Dosing</th>
<th>Oral dosing/notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium Channel Blockers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diltiazem</td>
<td>See Figure 2 [Class I, LOE B]</td>
<td>Can cause hypotension and AV nodal block. Cl: Bradycardia Cl: Systolic heart failure</td>
<td>IV: 10-20 mg IVP bolus over 2 min. If HR remains &gt; 120, consider a second bolus over 2 minutes Then start infusion at 5 mg/hr, titrate by 2.5 mg/hr every 30 minutes to HR, maximum dose 15 mg/hr [onset time 2-7 min]</td>
<td>120 to 360 mg daily in divided doses; extended-release preparation is available [onset 2 to 4 hours]</td>
</tr>
<tr>
<td>Verapamil</td>
<td>Can be used as an alternative to diltiazem. [Class I, LOE B]</td>
<td>Can cause hypotension and AV nodal block. Cl: Bradycardia Cl: Systolic heart failure</td>
<td>IV: 2.5-5 mg IVP over 2 minutes; second dose of 5-10 mg (~0.15 mg/kg) may be given 15-30 minutes after the initial dose if patient tolerates, but does not respond to initial dose; maximum total dose: 20-30 mg. [onset time 3-5 min]</td>
<td>120 to 360 mg daily in divided doses; extended-release preparation is available [onset 1 to 3 hours]</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>See Figure 2 [Class I, LOE C]</td>
<td>Can cause hypotension and AV nodal block. Avoid in patients with bronchoconstriction or emphysema. Use with caution in patients with decompensated heart failure. Cl: Bradycardia.</td>
<td>IV: 5 mg IVP every 10-20 minutes x 3 [onset time 5 min]. If HR &lt; 120 results, start oral metoprolol (see dosing suggestions to right). If HR &gt; 120 after 15 mg IV metoprolol, consider an alternate agent such as diltiazem above.</td>
<td>Oral dosing: If HR &lt; 120 after 5 mg IV, consider oral dose of 25 mg PO BID. If HR &lt; 120 after 10 mg IV, consider oral dose of 50 mg PO BID. If HR &lt; 120 after 15 mg IV, consider oral dose of 75 mg PO BID. [onset 4-6 hours]</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Best for use in situations where there is an indication to use a beta-blocker, but the patient a.) cannot take oral medications, or b.) has a labile or tenuous blood pressure (esmolol has a short half-life, and its effect can be quickly halted by stopping the infusion). [Class I, LOE C]</td>
<td>Can cause hypotension and AV nodal block. Avoid in patients with acute or active airway obstruction/bronchoconstriction. Avoid in patients with decompensated heart failure. Relatively expensive Cl: Bradycardia.</td>
<td>IV: 50 mcg/kg/minute infusion for 4 minutes. Infusion may be continued at 50 mcg/kg/minute or, if the response is inadequate, titrated upward in 50 mcg/kg/minute increments (increased no more frequently than every 4 minutes) to a maximum of 200 mcg/kg/minute. [onset time &lt; 5 min]</td>
<td>N/A Loading doses are not necessary due to rapid onset of action</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Consider for use in thyrotoxicosis [Class I, LOE C]</td>
<td>Can cause hypotension and AV nodal block. Avoid in patients with acute or chronic airway obstruction/bronchoconstriction. Avoid in patients with decompensated heart failure. Cl: Bradycardia.</td>
<td>IV: 0.5-1 mg over 1 minute; may repeat, if necessary, up to a total maximum dose of 0.1 mg/kg [onset time 5 min]</td>
<td>Usual oral dose: 10-30 mg/dose every 6-8 hours</td>
</tr>
</tbody>
</table>
### Table 4: Pharmacologic agents useful for rate control in patients with AF/AFL, continued

<table>
<thead>
<tr>
<th>Drug</th>
<th>When to consider use? [level of recommendation / level of evidence]</th>
<th>Warnings/Contraindications</th>
<th>Intravenous Dosing</th>
<th>Oral dosing/ notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other agents</td>
<td></td>
<td></td>
<td><strong>Amiodarone</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Primarily functions as an antiarrhythmic (rhythm control) agent, as shown in Appendix A. Useful as a second line rate control agent when first line agents fail, usually in consultation with cardiology or EP. May be useful in patients with decompensated systolic heart failure. Also may be useful in situations when an accessory pathway is suspected. [Class IIa, LOE C]</td>
<td>Can cause hypotension (when given intravenously), and pulmonary toxicity (so patients with severe lung disease are poor candidates for long-term administration). Relatively expensive Note: This medication can result in cardioversion when used for rate control CI: Bradycardia.</td>
<td>IV: 150 mg IV over 10 minutes. Follow up with an infusion of 1 mg/min IV x 6 hours, then 0.5 mg/min IV x 18 hours. [onset time &lt; 20min]</td>
<td>EP or general cardiology consult should be requested if long-term amiodarone use is contemplated.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Digoxin</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Second line rate control agent, primarily because it tends to exert its rate controlling effect at rest, less so with exercise. Most useful in combination with a beta-blocker or a calcium channel blocker, especially for patients with systolic heart failure, or in cases where low blood pressure is a major issue (digoxin does not lower blood pressure). [Class I, LOE B]</td>
<td>Can cause AV nodal block, digoxin toxicity. CI: Bradycardia, renal failure</td>
<td><strong>Loading dose:</strong> 10-15 mcg/kg LBW – rounded to the nearest 0.125mg (most average sized patients should receive 1mg). Administer load in 3 divided doses as 50%, 25%, 25% of the total dose Q6 hours (i.e. total dose given over 12 hours). Reduce total calculated dose by 25% for IV loading. Consider reduction in loading dose for those with renal insufficiency. [onset time 30-180min]</td>
<td></td>
</tr>
</tbody>
</table>

CI: Contraindication
LOE: Level of Evidence
LBW: Lean Body Weight
Incidence

Atrial fibrillation (AF) and atrial flutter (AFL) are the most common sustained arrhythmias in the U.S., affecting 2.5 million adults with the majority of patients over the age of 65. AF/AFL is associated with numerous comorbidities including hypertension, coronary artery disease, heart failure, and valvular heart disease. The cost of direct care of patients with AF in the U.S. is an estimated $6.65 billion annually, the majority of which is attributed to hospitalizations due to rapid ventricular response, heart failure, and stroke. There are over 150 admissions to the UMHS annually with the principle diagnosis of new-onset AF, and there are many more than that for recurrent or chronic atrial fibrillation.

Rationale for Recommendations

AF/AFL is prevalent in the population of patients evaluated in the Emergency Department and admitted to the University Hospital. Given the vast spectrum of patient presentations and the breadth of treatment options, management of AF patients is inherently complex. Patients presenting with AF may receive inconsistent care. These guidelines have been developed to assure consistent care delivery for patients with AF across the inpatient services. These guidelines are applicable to all inpatients with notable exception of patients on the Cardiology, Cardiac Intensive Care or Cardiac Surgery Services.

Diagnosis

Electrocardiographic documentation is essential to establish the diagnosis of AF/AFL. AF is characterized by the replacement of consistent P waves by rapid oscillations or fibrillatory waves that vary in amplitude, shape, and timing, associated with an irregular, frequently rapid ventricular response. Patients in AF/AFL can rarely have regular cardiac cycles (R-R intervals), in the presence of AV block, ventricular tachycardia, or AV junctional tachycardia.

AF may be detected on a 12 lead surface ECG, Holter monitor (usually 24 or 48 hour recording), or event monitor (usually worn for up to 3 weeks). AF may or may not be associated with rapid ventricular response depending on intrinsic atrio-ventricular conduction system, autonomic tone, and medications.

AF may occur alone or be associated with other arrhythmias, notably AFL and atrial tachycardia (AT). AFL in the typical form is characterized by a saw-tooth pattern of regular atrial activation called flutter waves on the ECG, particularly visible in leads II, III, aVF, and V1.

Definitions and Classification

AF and AFL are supraventricular tachyarrhythmias; AF is characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. Typical AFL is a macro-reentrant atrial arrhythmia utilizing the cavo-tricuspid isthmus. AFL may result from treatment to prevent recurrent AF.

Many of the recommendations in this document apply to both AF and AFL, especially as they relate to rate control and anticoagulation.

There are a number of types of AF/AFL:

- **First detected** - new onset
- **Paroxysmal** – self-terminating, episodes generally last 7 days or less (most less than 24 hours)
- **Persistent** – not self-terminating, episodes usually last longer than 7 days, or require cardioversion
- **Permanent** – cardioversion failed to acutely convert or was not attempted
- **Recurrent** – both paroxysmal AF/AFL and persistent AF/AFL may be recurrent
- **Post-operative** – Post-operative AF/AFL usually occurs within one week of the surgical procedure

Causes and Medical Conditions Associated with AF/AFL

Incidence of AF/AFL increases with increasing age. Many patients have structural heart disease such as coronary artery disease (CAD), hypertensive heart disease, valvular heart disease (especially mitral regurgitation or stenosis), or dilated or hypertrophic cardiomyopathy. Other comorbidities include obesity with or without sleep apnea, diabetes, and bronchopulmonary disease. AF/AFL may be caused by “reversible” causes such as post-operative state, pneumonia, pulmonary embolism, or hyperthyroidism.

Treatment

Clinical Approaches: Unstable and Stable AF/AFL

The approach to the evaluation and management of AF/AFL depends on the clinical circumstances of the arrhythmia. For instance, patients with unstable AF/AFL require immediate supportive care for stabilization, and often require emergent interventions to terminate the arrhythmia (rhythm control). On the other hand, patients with stable AF/AFL may not require emergent treatment, but might need measures to control their heart rate (rate control) and prevent strokes (anticoagulation). Stable patients may not always require interventions to terminate the arrhythmia (rhythm control), although that may be appropriate in some cases.
Because of the differences between these groups, this guideline will divide the discussion into 2 sections:

- Unstable AF/AFL
- Stable AF/AFL

There are also special considerations for populations presenting to the Emergency Department, or those who experience AF postoperatively (with special consideration for thoracic surgery).

**Unstable AF/AFL**

AF/AFL can be defined as unstable when it results in altered mental status, ischemic chest discomfort, acute heart failure, or other signs of shock or hemodynamic instability. Patients with unstable AF/AFL require rapid stabilization. Differentiating between hemodynamic instability that is a direct result of the arrhythmia and instability in the setting of AF/AFL but secondary to another cause (such as sepsis, hypovolemia, ACS or PE) will help to determine the focus of treatment efforts.

An overview of initial management of unstable AF is illustrated in Figure 1, and includes cardiac monitoring, supplying supplemental oxygen, obtaining large-bore IV access, and addressing intravascular volume depletion with IV fluid administration, provided the patient does not have an acute exacerbation of heart failure.

At the same time, a focused history and physical should be conducted, with special attention paid to the duration and nature of symptoms, comorbidities, and identifying reversible causes of AF/AFL. Underlying causes of hemodynamic instability other than the arrhythmia should be sought and treated, including early resuscitation for sepsis, blood products for severe anemia, cardiac catheterization for ACS, anticoagulation and possible thrombolysis for pulmonary embolism. Supportive treatment with vasoactive agents may also be necessary. Although cardioversion should be considered in such patients, it is important to recognize that many of the critically ill patients seen in the ED may have chronic AF or additional comorbidities that may lead to failed or short-lived effects of cardioversion. The main focus should be placed at treating the underlying condition causing the hemodynamic instability.

In patients who are hemodynamically unstable as a result of AF/AFL without other causes for shock or hypotension, immediate synchronized direct current cardioversion (DCCV) is first-line treatment. For AF, the recommended initial energy level is 200 joules (J), which can be increased to 360 J if lower energy levels are unsuccessful. For AFL, the recommended initial energy level is 50 J, which can be increased to 200 J if unsuccessful.

If cardioversion is repeatedly unsuccessful, a STAT CCU consult should be placed to help direct care. Resuscitative efforts should also be escalated to include consideration of respiratory support, additional vasoactive agents, and calling a code if needed. A trial of amiodarone with an intravenous loading dose of 150mg over 10 minutes followed by a 1mg/min IV infusion can also be considered.

If a patient with unstable AF/AFL is successfully cardioverted with resolution of clinical instability, post-cardioversion treatment should then transition to diagnostic evaluation as directed by Table 1 as well as considering anticoagulation (Figure 3) and placing an EP consult for assistance with long-term rhythm management of AF/AFL.

**Stable AF/AFL with RVR**

Stable AF/AFL with rapid ventricular response (RVR) can be defined as AF/AFL with HR > 110 at rest with no signs of instability listed above. Stable AF/AFL can be new, chronic, or paroxysmal, and differentiating between these classes can have important long-term management implications. In this section, we will focus on the evaluation and management of any type of AF with RVR.

**Evaluation of New-Onset AF/AFL.** The evaluation of AF/AFL is intended to discover the cause of the arrhythmia, and clinical features of the patient that might impact treatment (See Table 1).

**Management of New-Onset AF/AFL.** The management of stable, new-onset AF/AFL with RVR can be divided into these general categories:

- Treatment of underlying conditions
- Consideration of an accessory pathway
- Consideration of rhythm control
- Control of heart rate
- Anticoagulation

Often, several of these categories of treatment will be applied, in parallel. However, the main goals of AF/AFL with RVR management are symptomatic improvement (via rate/or rhythm control), and prevention of thromboembolic complications (with antiplatelet or anticoagulant medications). An overview of the management of stable AF/AFL is illustrated in Figure 2.

**Treatment of underlying conditions.** Patients with AF/AFL with RVR can have a number of underlying acute conditions (infectious, hypovolemia, anemia, etc.) that may be driving the tachycardic response. These should be suspected and appropriately evaluated early, as treatment of these underlying conditions is key to resolving the RVR in such patients.

**Consideration of an accessory pathway.** Some patients with AF/AFL will present with features suggestive of an accessory pathway associated with Wolff-Parkinson-White (WPW) Syndrome. The ECG of a patient with preexcitation during AF/AFL typically shows varying degrees of preexcitation, variable RR intervals and variable (bizarre) QRS morphologies. This represents a special circumstance
in the management of AF/AFL and necessitates an urgent EP consult.

Drugs contraindicated in patients with accessory pathways include digoxin, and non-dihydropyridine calcium channel antagonists (e.g. verapamil, diltiazem), which slow conduction across the AV node, and can result in paradoxical acceleration of the ventricular rate, hypotension, or ventricular fibrillation. Beta blockers are ineffective and may cause hypotension. When the arrhythmia is associated with hemodynamic compromise, early DCCV is indicated (see Figure 1). In hemodynamically stable patients with preexcited AF/AFL, procainamide is recommended to restore sinus rhythm. Further management should be guided by consultation with EP. Of note, any patient with preexcitation and syncope, with or without history of AF/AFL, warrants inpatient EP consultation.

**Control of heart rate.** Rate control medications slow conduction through the AV node, thus slowing ventricular rates in rapid AF/AFL. In the absence of pre-excitation syndromes, non-dihydropyridine calcium channel antagonists and beta-blockers are most commonly used, and can safely achieve rate control in most patients. For patients with AF/AFL and mildly elevated heart rates of 100-119 at rest (or those with resting heart rates that increase to ≥130 with activity), adjustment of oral rate controlling agents can safely be undertaken (Table 4). For rapid AF/AFL with elevated heart rates of ≥120 at rest, acute rate control is preferred and is best achieved with intravenous formulations of AV nodal blocking agents as illustrated in Figure 2.

Beta blockers should be considered early for the treatment of new-onset, rapid AF/AFL. Beta blockers are very effective for rate control, achieving the specified heart rate endpoints in 70% of patients compared with 54% with use of calcium channel antagonists in the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) trial. Beta blockers also have additional indications for comorbidities that are commonly found in patients with AF/AFL, such as hypertension, CAD, or heart failure, although they may not be well tolerated by patients with acute bronchospasm or severe emphysema. Metoprolol is usually administered as 5mg IV doses that can be re-dosed every 10-20 minutes for up to three doses. Metoprolol may cause hypotension and should not be used in the setting of a labile or tenuous blood pressures. In this setting, esmolol infusion is preferred due to its short onset of action and rapid clearance. Of note, propranolol is most useful in the setting of thyrotoxicosis.

Non-dihydropyridine calcium channel antagonists, such as diltiazem and verapamil, are another effective class of medications for heart rate control in AF/AFL. Calcium channel antagonists should be used in the setting of a contraindication to beta blockers such as an allergy or bronchoconstriction. Diltiazem, given its rapid onset, tends to be more popular, and has been shown in a randomized controlled trial to be more effective in controlling the ventricular rate than amiodarone or digoxin.

Should a patient with rapid AF/AFL not achieve adequate control of their heart rate following an initial trial of one of the IV AV nodal blockers, the initial medication should be stopped and the other class of IV AV node blockade should subsequently be attempted prior to adding adjunctive medications. If both IV calcium channel antagonists and IV beta blockers prove to be ineffective, consultation with EP is recommended. Co-administration of oral beta blockers and non-dihydropyridine calcium channel antagonists for long term heart rate control may be necessary, and is generally safe with the appropriate monitoring.

There are a few notable considerations for patients with systolic heart failure and AF/AFL. Non-dihydropyridine calcium channel blockers are best avoided altogether in this population. Also, in decompensated heart failure, both beta-blockers and non-dihydropyridine calcium channel antagonists may acutely exacerbate heart failure symptoms. Cardiology consultation is recommended for patients presenting with decompensated systolic heart failure and AF/AFL with RVR. Digoxin and amiodarone are both useful rate control agents in this population. Of note, amiodarone results in increased plasma digoxin levels.

Digoxin has been largely replaced by more effective AV nodal blockers, but remains useful as an adjunct in patients with heart failure. Digoxin has little, if any effect on blood pressure, so it can also be useful in hypotensive patients. However, digoxin tends to exert its rate controlling effect at rest, less so with exercise, which may result in rate control at rest with continued tachycardia with exertion.

Amiodarone is known to slow ventricular rates during AF/AFL. It is generally indicated only when maintenance of sinus rhythm is desired given its significant toxicities, but can be used for rate control in some circumstances.

Table 4 provides a review of drugs used for control of ventricular rate in AF/AFL, their side effects, and contraindications. Long-term control of heart rate is important to control symptoms and to prevent the development of a tachycardia-induced cardiomyopathy, as a sustained, uncontrolled tachycardia may lead to deterioration of ventricular function. Fortunately, this tachycardia-induced cardiomyopathy tends to resolve within 6 months of rate or rhythm control.

For heart rate that cannot be controlled well with medications: consultation with EP should be obtained if the patient’s heart rate is not controlled after 24 hours of treatment with adequate IV AV nodal agents.

**Defining adequate rate control.** Based on the results of the RACE II trial, a resting heart rate <110 bpm indicates adequate rate control. Treatment to achieve strict rate control of heart rate (80 bpm at rest or 110 bpm during a 6-minute walk) is not beneficial compared to achieving a resting heart rate 110 bpm in patients with persistent AF/AFL who have stable ventricular function (left ventricular ejection fraction ≥40) and no or acceptable symptoms related to AF/AFL. Of
note, long-term (> 3 years) effects on ventricular function were not evaluated in this study, therefore periodic monitoring of LV function is recommended.

Consideration of rhythm control. Rhythm control should be considered in the following clinical scenarios (Table 3):

- First Occurrence of AF/AFL
- Occurrence or recurrence of AF/AFL due to reversible cause [e.g. post-operative state, pulmonary embolism, pneumonia, or acute coronary syndrome/acute myocardial infarction (ACS/AMI)]
- Hospital readmissions for AF/AFL
- AF/AFL-related symptoms despite adequate rate control, or failure to achieve rate control despite the use of appropriate medications
- Cardiomyopathy presumed to be secondary to tachycardia
- Younger patients (age <65), even with minimally symptomatic AF/AFL, in whom the effects of long term AF/AFL are unknown

If a rhythm control strategy is appropriate, consultation with EP is recommended in most cases. There are several antiarrhythmic medications that may be used for rhythm control (see appendix A). The clinical use of these medications is a complex endeavor, typically done in consultation with an electrophysiologist or cardiologist, although some of these agents may be given prior to consultation for unstable patients, or for patients on certain specialty services (e.g., Cardiac Surgery, Thoracic Surgery).

Elderly, minimally symptomatic or asymptomatic AF/AFL patients may not benefit from antiarrhythmic drugs in an attempt to revert to sinus rhythm. The AFFIRM trial found no difference in mortality or stroke rate between elderly patients with permanent AF assigned to one strategy or the other. The RACE (Rate Control vs. Electrical cardioversion for recurrent persistent AF) trial found rhythm control with cardioversion and antiarrhythmic medications not inferior to rate control for prevention of death and morbidity. The trial data do not necessarily apply to younger patients without heart disease or to patients whose dependency upon sinus rhythm is likely to change over time. Subsequent development of ventricular hypertrophy, systolic heart failure, or pulmonary disease will affect patient’s symptomatic status. Such a patient may not feel different in sinus rhythm when initially evaluated but if left in AF/AFL may face difficulties in the future with an increase in difficulty to restore sinus rhythm because of atrial remodeling.

Treating the patient that spontaneously converts to sinus rhythm. These patients represent a special challenge for the clinician, because it is often unclear whether the AF/AFL was a transient rhythm related to a specific stressor, or if it represents a paroxysm of AF/AFL that will recur in the future. For example, surgical stress that results in transient AF/AFL may indicate a predilection in such patients for future paroxysms of AF/AFL, therefore we recommend close follow-up and monitoring of these patients (see Special Considerations: Postoperative AF/AFL, below). In most cases, these patients may be treated as if they have paroxysmal AF/AFL, a condition that poses a stroke risk similar to chronic AF. For patients with AF/AFL that convert spontaneously, we recommend these considerations:

- For patients seen in the ED, provide outpatient consultation with EP
- For inpatients, consider consulting EP in the hospital
- Consider anticoagulation, as per the Anticoagulation section below (see Figure 3).
- Consider the use of an oral rate control agent, depending on the pre- and post-conversion heart rates.
- Consider the use of an outpatient 3 week event monitor, to assess for AF/AFL recurrences.
- Provide education to these patients about AF/AFL, including instructions about how to recognize the rhythm, and what to do if it recurs.

Ablative therapies. AF ablation with pulmonary vein isolation may be considered for symptomatic patients who have failed at least one antiarrhythmic agent and in those patients who do not wish life-long antiarrhythmic drug therapy. AF ablation is more efficacious and is associated with improved quality of life compared to antiarrhythmic medication. However, risks and benefits of an invasive procedure must be carefully weighed against the benefits of maintaining sinus rhythm.

AFL ablation of typical AFL is a safe, well-tolerated procedure with complete elimination of the arrhythmia in >95% of patients, and should be considered a first line treatment in appropriately selected patients.

AV nodal ablation does not result in conversion to sinus rhythm, and it is merely designed to control ventricular response to AF. AV nodal ablation should not be attempted without a prior trial of medication to control the ventricular rate. Catheter AV nodal ablation results in complete AV block and requires implantation of a permanent pacemaker. AV nodal ablation may be a viable option for elderly or frail patients in whom pharmacologic rate control is complicated by hypotensive episodes.

Anticoagulation

Due to the increased risk of stroke and systemic embolism with AF/AFL, anticoagulation is utilized in almost all patients undergoing acute management with electrical or pharmacologic cardioversion, and for long-term management based on additional risk factors. This section will present recommendations for anticoagulation of patients with AF/AFL in the following circumstances:

- Acute anticoagulation with cardioversion
- Acute anticoagulation of new-onset AF/AFL, without cardioversion
- Long-term anticoagulation
These recommendations are illustrated in the algorithm in Figure 3. For post-thoracic surgery patients presenting with AF/AFL, refer to Appendix C. In all cases, the risk of bleeding should be considered, and contraindications to anticoagulation should be recognized.

**Acute anticoagulation with cardioversion.** Anti-coagulation is especially important when attempts are made to convert AF/AFL to sinus rhythm. The following anticoagulation recommendations should be used regardless of cardioversion strategy (i.e., pharmacologic or electrical). In addition, AF/AFL that is determined to be less than 48 hours in duration may proceed to cardioversion because the likelihood of prior thrombus formation is low. Even in this clinical scenario, post-cardioversion anticoagulation is generally indicated. Patients in AF/AFL for longer than 48 hours (or if the duration in unknown) have an elevated risk of having developed an atrial thrombus. Therefore, these patients should undergo a TEE-guided cardioversion or, alternatively, be maintained on therapeutic anticoagulation for a minimum of three weeks prior to undergoing planned cardioversion. The following recommendations are summarized in Figure 3:

- If the patient has a history of ischemic stroke in the past 2 weeks, consult neurology for anticoagulation recommendations. If the patient has a history of intracranial hemorrhage, neurology and/or neurosurgery should be consulted for anticoagulation recommendations.
- If the patient has a history of rheumatic heart disease or a prosthetic heart valve, target-specific oral anticoagulants (TSOACs, currently also known as “Novel Oral Anticoagulants—”NOACs”) are not recommended and the type of acute anticoagulation depends on renal function with transition to warfarin therapy:
  - If the estimated creatinine clearance is < 30 ml/min, then IV unfractionated heparin with transition to warfarin therapy is preferred.
  - If the estimated creatinine clearance is > 30 ml/min, then anticoagulation with subcutaneous enoxaparin 1 mg/kg twice daily can be used in place of IV unfractionated heparin, with transition to warfarin therapy.
- If the patient does not have a history of rheumatic heart disease or prosthetic heart valves, target-specific oral anticoagulant (TSOAC) is recommended, but is also dependent on renal function:
  - If the estimated creatinine clearance < 30 ml/min, then IV unfractionated heparin with transition to warfarin therapy is preferred.
  - If the estimated creatinine clearance is > 30 ml/min, then a TSOAC is preferred (Appendix B) with transition to warfarin therapy can also be chosen.
- If the patient is on antiplatelet therapy, see discussion in the Special Populations section below.
- Once a patient has undergone cardioversion, all patients should be anticoagulated for at least four weeks after cardioversion, with subsequent long-term anticoagulation therapy based on risk factors, as outlined below. (See Long-term anticoagulation.)

**Acute anticoagulation of new-onset AF/AFL without cardioversion.** The decision for acute anticoagulation of AF/AFL patients who are not selected for initial conversion to sinus rhythm should be based on individual stroke risk, as assessed in non-valvular AF/AFL by the CHA2DS2-VASc score (Table 2). Generally, patients with a high risk for stroke should be initiated on therapeutic anticoagulation acutely while those with a lower stroke risk do not require immediate therapeutic anticoagulation. Of note, the newer TSOAC are felt to have a rapid onset of action and do not require bridging with other agents. An overview of the management of anticoagulation in this setting is illustrated in Figure 3, with the specifics depending on patient characteristics, as follows:

- If the patient has a history of ischemic stroke in the past 2 weeks, consult neurology for anticoagulation recommendations. If the patient has a history of intracranial hemorrhage, neurology and/or neurosurgery should be consulted for anticoagulation recommendations.
- If the patient has a history of rheumatic heart disease or prosthetic heart valves, they are at increased risk for stroke irrespective of their CHA2DS2-VASc score and should be acutely anticoagulated with agents determined by renal function:
  - If the estimated creatinine clearance < 30 ml/min, then IV unfractionated heparin with transition to warfarin therapy is preferred.
  - If the estimated creatinine clearance is > 30 ml/min, then anticoagulation with subcutaneous enoxaparin 1 mg/kg twice daily can be used in place of IV unfractionated heparin, with transition to warfarin therapy.
  - TSOACs are not recommended in this patient population.
- If the patient does not have a history of rheumatic heart disease or prosthetic heart valves, but does have a history of ischemic stroke (> 2 weeks ago) or a CHA2DS2-VASc score ≥ 6, acute anticoagulation is recommended with agents determined by renal function:
  - If the estimated creatinine clearance is < 30 ml/min then initiate IV unfractionated heparin with transition to warfarin therapy.
  - If the estimated creatinine clearance is > 30 ml/min then a TSOAC is preferred (Appendix B) or subcutaneous enoxaparin 1 mg/kg twice daily with transition to warfarin.
- If the patient does not have a history of rheumatic heart disease or prosthetic heart valves but has a CHA2DS2-VASc score of 5, then consider acute therapeutic anticoagulation if it is felt that the reduction in stroke
outweighs the risk of bleeding complications. When acute anticoagulation is selected the agent is determined by renal function:
- If the estimated creatinine clearance is < 30 ml/min then initiate IV unfractionated heparin with transition to warfarin therapy.
- If the estimated creatinine clearance is > 30 ml/min initiate a TSOAC or subcutaneous enoxaparin 1 mg/kg twice daily with transition to warfarin.
- If the patient does not have a history of rheumatic heart disease or prosthetic heart valves, but has a CHA2DS2-VASc score of 0 to 4:
  - Acute therapeutic anticoagulation is not necessary in the absence of planned cardioversion.
  - Recommendations for long-term anticoagulation therapy in these patients are outlined below.
- If the patient is on antiplatelet therapy, see the discussion in the Special Populations section below.

**Long-term anticoagulation.** Anticoagulation should be continued indefinitely, or until the patient develops a contraindication, even if antiarrhythmic agents appear to maintain sinus rhythm. The need for long-term anticoagulation for prevention of stroke/TIA and systemic embolism is determined based on the patient’s thrombotic risk assessment. Derived from the CHADSV2, the CHA2DS2-VASc is a well-studied risk stratification scheme used in the 2012 European Society of Cardiology AF guidelines. The CHA2DS2-VASc accounts for additional risk factors not included in the CHADS2 and appears to present the anticoagulation for prevention of stroke/TIA and systemic embolism with a similar rate of major bleeding. When-life-threatening and intracranial bleeding were significantly lower with dabigatran, whereas more patients suffered gastrointestinal bleeding with high-dose dabigatran. Although rare, the rate of myocardial infarction was higher in dabigatran-treated patients.

The TSOACs have demonstrated similar to increased efficacy in stroke prevention and a similar to reduced rate of major bleeding when compared to adjusted-dose warfarin therapy in phase 3 clinical trials.

**Dabigatran.** The RE-LY trial compared two doses of dabigatran (150 and 110 mg twice daily) to open-label, adjusted-dose warfarin. The majority of patients had a CHADSV2 score greater than 2, dabigatran 150 mg twice daily was found to be superior to warfarin in reduction of stroke or systemic embolism with a similar rate of major bleeding. Life-threatening and intracranial bleeding were significantly lower with dabigatran, whereas more patients suffered gastrointestinal bleeding with high-dose dabigatran. Although rare, the rate of myocardial infarction was higher in dabigatran-treated patients.

**Rivaroxaban.** Rivaroxaban was compared with adjusted-dose warfarin in the ROCKET-AF trial. Patients included had an average CHADSV2 score of 3.5. Rivaroxaban was found to be noninferior to warfarin for prevention of stroke or systemic embolism, and the composite of major and non-major clinically relevant bleeding events (primary safety endpoint) was similar. Critical, intracranial and fatal bleeding was significantly lower in the rivaroxaban group, while gastrointestinal bleeding was significantly higher. Within 30 days of stopping the trial, an increase in stroke and systemic embolism occurred in the rivaroxaban group leading to the recommendation for avoidance of abrupt cessation of rivaroxaban in the absence of adequate anticoagulation (an effect also seen with apixaban).

**Apixaban.** Apixaban was compared to adjusted-dose warfarin in the ARISTOTLE trial. The stroke risk of patients was similar to that seen in the RE-LY trial. The trial found a significant reduction in stroke or systemic embolism in the apixaban group compared to warfarin and a reduction in all bleeding indices. The rates of gastrointestinal bleeding and myocardial infarction were not increased. Apixaban was also compared to aspirin 81-324 mg daily in patients at high risk for stroke (average CHADSV2 score of 2) but who were
deemed “unsuitable” vitamin K antagonist candidates. The most frequent reasons for “unsuitability” were patient refusal and low likelihood of INR measurement at requested intervals, and few patients were included for bleeding history or bleeding risk. The trial was terminated early due to significant reduction in stroke or systemic embolism in the apixaban group with no difference in major bleeding events, although minor bleeding was more common in the apixaban group.

Pharmacokinetic and other relevant parameters of the TSOACs are compared in Appendix B. While all of the agents undergo renal and hepatic metabolism, dabigatran is primarily eliminated renally and rivaroxaban and apixaban undergo more extensive hepatic metabolism. Although these agents have fewer drug-drug interactions than warfarin therapy, there are still clinically relevant interactions to be aware of including P-gp interactions with all of the agents and CYP3A4 interactions with rivaroxaban and apixaban.

TSOACs are preferred over adjusted-dose warfarin therapy, given the outcome data and the reduction in monitoring requirements, dietary interactions, and amount of drug interaction. A few exceptions exist due to a lack of data; including patients with stage 4 or 5 chronic kidney disease, advanced liver disease, concomitant high dose aspirin or dual-antiplatelet therapy use, and valvular AF/AFL. In addition, a trial comparing dabigatran to warfarin in patients with mechanical heart valves was terminated early due to increased risk of thromboembolic and bleeding complications with dabigatran therapy. Warfarin is preferred in these patient populations until more data is available. (See below- Special Populations)

When prescribing TSOAC’s, clinicians must be aware that these novel agents can be quite costly, and that they are not covered uniformly by all insurance companies. Therefore, when these agents are prescribed, it is critical that clinicians assure that the patient will be able to obtain the medication, and that cost (or insurance coverage issues) is not a barrier to medical compliance after discharge. Determination of an individual’s co-pay for TSOACs is done in conjunction with pharmacy and should be done prior to discharge.

Special Populations for anticoagulation. Some circumstances require special consideration of anticoagulants.

- If the patient has valvular AF (rheumatic mitral valve disease, mitral stenosis, prosthetic valves, or mitral valve repair), then oral anticoagulation with adjusted-dose warfarin is strongly preferred over other antithrombotic strategies
- If the patient has AF/AFL and an ischemic stroke within the previous 2 weeks, then consultation with the Stroke Team is recommended for anticoagulation decision.
- If the patient has an estimated creatinine clearance < 30 ml/min, then when oral anticoagulation is selected, adjusted-dose warfarin therapy is preferred over other antithrombotic strategies due to the limited data with TSOACs in this patient population. When parenteral anticoagulation is desired, unfractionated heparin is preferred over low molecular weight heparin.
- If a patient with AF/AFL also has an indication for antiplatelet therapy, the management will depend on the precise indication for the antiplatelet agent(s):
  a. If the patient has AF/AFL, and the indication for antiplatelet therapy is primary or secondary prevention, and the patient has not had ACS within the previous year, and has not had coronary stent placement, then oral anticoagulation alone is sufficient (no antiplatelet agent is needed in addition to the oral anticoagulant).
  b. If the patient has AF/AFL and ACS within the previous year, without stent placement, then when oral anticoagulation is selected, oral anticoagulation plus single antiplatelet therapy is preferred over triple therapy
  c. If the patient has AF/AFL and ACS with stent placement, then when oral anticoagulation is selected, triple therapy (warfarin + clopidogrel + low dose aspirin) is preferred over other strategies for the first 1 month after bare-metal stent placement or first 3-6 months after drug-eluting stent placement. When used in combination with dual antiplatelet agents in this way, warfarin is the oral anticoagulant of choice, given lack of data/experience with the TSOAC’s. After this time period single antiplatelet therapy plus oral anticoagulation is preferred. Consultation with general cardiology should be considered in such patients.
  d. ACC and AHA recommend targeting an INR of 2 to 2.5 for patients receiving triple therapy, but the effectiveness and safety of this approach compared with the conventional INR range of 2 to 3 remains unproven.
  e. Low dose aspirin is often recommended, in addition to anticoagulation therapy, in patients with mechanical heart valves, or those with bioprosthetic heart valves and additional risk factors.

- Recommendations for postoperative AF are discussed in a separate section below.

Transient AF/AFL that resolves and was caused by “reversible” causes such as post-operative state, pneumonia, pulmonary embolism, or hyperthyroidism in otherwise low risk patients may not require long term anticoagulation. Patients at higher risk of stroke may warrant periodic screening for asymptomatic AF/AFL with ECG or event monitor to detect asymptomatic AF/AFL.

Special Considerations:
Emergency Department (ED) Management

The acute ED management of atrial AF/AFL is variable, and few specific treatment recommendations exist.
Traditionally, management strategies have focused on rate control of rapid AF/AFL, and admission to the hospital for continued administration of rate control agents and decisions for anticoagulation, and these remain reasonable in many patient populations. Recent studies suggest that for some category of low-risk patients, management via rhythm control is also a reasonable strategy. This section will detail recommendations specific to the ED patient with AF/AFL.

**Unstable ED Patient**

The evaluation and management of unstable patients with AF/AFL are covered in Figure 1 and the accompanying text above.

**Stable ED Patient**

Selected, stable patients with AF/AFL can sometimes receive all of their treatment in the ED, avoiding hospital admission (Figure 4).

**Rate Control.** For patient with stable rapid AF/AFL, decisions to pursue rate vs. rhythm control strategies have been defined. Patients who are high-risk, or unlikely to have success from rhythm control strategies include those who are chronically in AF/AFL, and now have a rapid rate, as they are unlikely to have sustained cardioversion. Patients who are not anticoagulated and have AF/AFL for a duration that is either unknown or > 48 hours are higher risk for thromboembolic complications, and rate control strategies should be utilized. Strong consideration for rate control should also be given to patients with a history of CHF or severe CAD. For these patient groups, the algorithm for stable AF/AFL (Figure 2) should be utilized, and the patient should be admitted to the hospital after rate control strategies have been initiated.

**Rhythm Control.** For the remainder of stable patients with rapid AF/AFL, rhythm control strategies may be considered in ED management (Figure 4). These patients would be generally healthy, with known onset of AF/AFL in the last 48 hours or with known anticoagulation for greater than 3 weeks. They should not have failed prior cardioversion attempt for AF/AFL.

Initial rhythm control attempt may be pharmacologic. The best-studied agent in ED populations is procainamide, 1gm IV over 1 hour. Studies show that this results in an approximately 60% cardioversion rate. Procainamide should be held for SBP < 90mmHg, and is contraindicated in patient with renal failure (CrCl<30), severe liver disease, lupus, or baseline hypotension. If cardioversion does not occur with procainamide, electrical cardioversion with procedural sedation may be administered. For AF, 200J synchronized cardioversion is recommended, and for AFL, 50J synchronized cardioversion is recommended. Anterior-posterior pad placement is recommended. If there is no success with the first attempt, higher energy levels (360J for AF 200J for AFL) may be attempted. For patients who fail cardioversion after these attempts, a rate control strategy should then be utilized, illustrated in Figure 2, with subsequent admission to the hospital.

Patients with successful cardioversion should be monitored for 3 hours for reoccurrence. Anticoagulation should be initiated per CHA2DS2-VASc score (Table 2, Figure 3). Referrals should be placed for outpatient EP consult, with a preceding transthoracic echocardiogram if they have not had one in the prior 6 months, and the patient may be discharged home.

**Criteria for Hospitalization**

Inpatient hospitalization is indicated for patients with highly symptomatic recurrent or new onset AF/AFL, especially if it is poorly tolerated and either rate control or rhythm control are urgently needed. AF/AFL may complicate or exacerbate an acute illness, which would also indicate the need for hospitalization. Well tolerated AF/AFL with controlled rates, whether new onset or recurrent, does not in itself automatically necessitate inpatient admission. Many patients may be treated in the ED with outpatient follow-up with the Electrophysiology Service.

Factors favoring discharge from the Emergency Department:
- Patients <60 years of age with lone AF
- Other patients with no structural heart disease and tolerable symptoms
- Well controlled ventricular rate during AF/AFL
- Established EP follow-up, living close

Factors favoring inpatient hospitalization:
- Patients with history of coronary artery disease, cardiomyopathy, or heart failure symptoms
- Patients with chest pain or dyspnea, if the cardiac cause of these complaints cannot be definitively ruled out in the ED.
- Inability or difficulty achieving rate control despite appropriate AV nodal agents
- Hypotension upon attempting to rate control
- Embolic event or high risk of thromboembolism (valvular AF/AFL)
- Intolerable symptoms requiring cardioversion and antiarrhythmic drug initiation, unless patient is a candidate for outpatient initiation of antiarrhythmic medication.
- Patients with non-cardiac causes of AF/AFL (hyperthyroidism, pulmonary embolism, pneumonia)
- Lack of efficacy using the current strategy (Figure 4)
- Pre-excitation on ECG or syncope

**Approach to Refractory Paroxysmal AF/AFL**

The goal of management of a patient with AF/AFL, especially in the setting of left atrial enlargement or long history of AF/AFL duration, is to reduce the recurrence rate or delay recurrence as much as possible. Thus, not all AF/AFL recurrences should be considered a therapeutic failure and prompt a change in strategy. When recurrences
occur, one should ask whether the patient is overall satisfied with the current AF/AFL management or if a change in the approach is indicated. This might involve changing antiarrhythmic agents, considering catheter ablation, concurrently using multiple treatment modalities, or abandoning the goal of rhythm control altogether. Patients should be educated on the pros and cons of ED visits vs. outpatient management based on patient’s individual risk profile and disease pattern. In addition to traditional risk factors for AF/AFL, obstructive sleep apnea has emerged as a significant predictor of AF/AFL recurrence. Patients at risk for sleep apnea (snoring, large BMI, large neck size, daytime sleepiness or fatigue) should be referred for sleep study and those already diagnosed with OSA – encouraged to adhere to their CPAP.

Approach to Refractory Persistent/Permanent AF/AFL

Not infrequently, patients with AF/AFL who were previously well rate controlled may present with worsening symptoms. Possible explanations for worsened clinical status may include noncompliance with medication or dietary indiscretion, which may trigger heart failure symptoms with fluid retention, hypoxia, and tachycardia. Patients should be questioned about caffeine use, recent changes to medications, and use of over the counter cold remedies, herbal and nutritional supplements. Thyroid function may need to be reassessed, especially in patients on amiodarone. Patients compliant with oral beta-blockers should be considered for adding oral calcium channel antagonists and vice versa (see Table 4). In cases of recurrent or persistently increased heart rate, AV nodal ablation with a pacemaker should be considered.

Hospital Follow-Up

Patients with new onset AF/AFL should be given appropriate follow-up upon discharge from the hospital or ED. If patient is seen in consultation by the Cardiology Service or the EP Service, they will determine the optimal clinic and timing of follow-up. If patient is discharged without inpatient cardiology or EP consult, the patient should be referred to the EP Clinic to be scheduled as a “next available” new patient appointment.

- In cases where there is concern about the adequacy of the rate control, patients may be offered a Holter monitor (48 hours) to assess the degree of rate control throughout the day. This can be helpful to the follow up physician.
- In cases where a patient is in sinus rhythm but there is a concern for recurrence of AF/AFL, an event monitor (3 weeks) may be offered.

Special Considerations: Postoperative AF/AFL

AF/AFL that occurs in the postoperative setting requires special consideration. Possible etiologies for postoperative AF/AFL should be considered based on the type of operation performed and potential perioperative events/complications. Many patients with postoperative AF/AFL will spontaneously convert back to sinus rhythm, and many of those patients will never have a recurrence of atrial fibrillation again. Therefore, postoperative AF/AFL is often treated with a rhythm control strategy. However, a subgroup of patients who suffer from postop AF/AFL will be found to have underlying heart disease, and go on to have recurrences. The optimal clinical approach to a patient with postoperative AF/AFL is not certain, but general recommendations will be provided in this section.

Approach to the Patient with Postoperative AF/AFL

For the purposes of this discussion, we will divide postop AF/AFL into categories based on the type of surgery with which it is associated.

Post-cardiac surgery. AF/AFL is an extremely common complication of cardiac surgery. For these patients, the approach is often one of aggressive rhythm control, and this population is outside of the scope of this guideline.

Post-non-cardiac, thoracic surgery. Similarly, AF/AFL is a common complication of non-cardiac, thoracic surgery. AF occurs in approximately 3% of this population, with the highest incidence after pneumonectomy and esophagectomy (21%). Because AF/AFL is so common in this patient population, it is often managed directly by the surgical service, and the management focuses on acute restoration of sinus rhythm. Appendix C outlines our institutional approach to these patients.

Amiodarone is often used for rhythm control in the thoracic surgery protocol. Amiodarone was effective in cardioverting 76-86% of patients postoperatively, and can be given IV, followed by oral dosing. Amiodarone should be avoided in patients with severe lung disease or after a pneumonectomy. If the patient requires an IV drip, a central venous catheter (e.g. PICC line) should be considered to avoid the risk of IV infiltration and subsequent skin necrosis.

Post non-cardiac, non-thoracic surgery. Lastly, there is a large population of non-cardiac, non-thoracic surgery patients that will suffer from postoperative AF/AFL. The remainder of this section will present an approach to these patients.

As with non-postoperative AF/AFL, the underlying cause of the rhythm should be sought. In addition to the standard evaluation (Table 1), postoperative patients are at risk for rapid fluid shifts, bleeding, thromboembolic disease, and other causes.

Patients with unstable postoperative AF/AFL should be treated as shown in Figure 1. For stable patients, treatment should first focus on controlling the heart rate as in Figure 2.
for the first 24 hours. The approach for patients who remain in AF/AFL for > 24 hours post-op includes cardiology consultation for consideration of rhythm control in the majority of patients (see more below).

Patients who spontaneously convert back to sinus rhythm within a 24-hour period may be treated somewhat differently. In these patients, it is unclear if the AF/AFL represents an isolated event, or if it marks the patient as being at risk for future AF/AFL. For these patients, the focus of treatment should be on assessing the risk for future AF/AFL. One approach to these patients might be to monitor them on telemetry while they are in the hospital, and to provide them with a 3-week event monitor at discharge to look for AF/AFL recurrences. It is useful to provide education to these patients about AF/AFL, including instructions about how to recognize the rhythm, and what to do if it recurs. These patients should be given non-urgent follow up with their PCP (or a cardiologist) after hospital discharge with the intention of specifically following up on possible recurrence of AF/AFL. Ideally, this problem should be added to the discharge summary problem list to facilitate adequate follow-up. For patients with transient postoperative AF/AFL anticoagulation is often withheld (see more below).

The optimal approach to postoperative AF/AFL is uncertain, and that is especially true when considering rhythm control for non-cardiac, non-thoracic surgery patients. Some experts feel that the majority of patients who develop AF/AFL after surgery should be treated with a rhythm control strategy. This is because postoperative AF/AFL is a potentially reversible condition. That is, many of these patients will respond to treatment, and be cured of their AF/AFL. However, rhythm control should be considered an urgent matter if the patient is unstable, highly symptomatic, or difficult to rate control with medications. Also, there are several reasons that a rhythm control strategy might be delayed in the postoperative setting. For example, attempts at cardioversion require systemic anticoagulation therapy to prevent embolic disease, and some postoperative patients may not be good candidates for immediate anticoagulation. In those cases, attempts at rhythm control might be delayed. We recommend a cardiology consult for most patients with postoperative AF/AFL that lasts > 24 hours after surgery. The consultant will be able to assist in this decision-making as it pertains to the value and timing of a possible rhythm control strategy.

Management of Anticoagulation for Postoperative AF/AFL

For patients in AF/AFL for more than 48 hours after surgery, anticoagulation should be considered, as discussed in the Anticoagulation section of this document. However, in postoperative patients, anticoagulation should always be discussed with the surgical service, considering the risk of bleeding in the postoperative patient. In some cases where the patient is at high risk for recurrent AF/AFL, or high risk for stroke with AF/AFL, it may be appropriate to provide anticoagulation therapy, even if the duration of the postoperative AF/AFL is < 48 hours.

Special Considerations: Management of Preoperative Anticoagulation in AF/AFL

Preoperative Considerations for Warfarin Patients

Because warfarin has a long half-life, it requires discontinuation several days prior to procedures where normal hemostasis is desired. The decision to maintain therapeutic anticoagulation up until the time of the procedure (“bridging”) should be based upon the risk for development of stroke or systemic embolism, and adapted based on individual patient circumstances. General recommendations are as follows:

- **Patients at low risk for thromboembolism (<5% per year):** no-bridging strategy is preferred over bridging with UFH or LMWH. Low risk patient populations include: CHA2DS2-VASc score of 0 to 4 (assuming no prior history of stroke or TIA).
- **Patients at moderate risk for thromboembolism (5-10% per year):** bridging or no-bridging strategy with UFH or LMWH is determined based on individual patient- and surgery-related factors. Moderate risk patient populations include: CHA2DS2-VASc score of 5.
- **Patients at high risk for thromboembolism (>10% per year):** Bridging with UFH or LMWH is preferred over no anticoagulation during interruption of warfarin therapy. High-risk patient populations include: CHA2DS2-VASc score of 6 or more, any previous stroke or TIA, or rheumatic valvular heart disease or prosthetic heart valves.

Special Considerations: Recent Stroke/CVA

In patients with recent ischemic stroke (< 2 weeks prior), Stroke Team consultation should be considered before initiating anticoagulation. Multiple factors such as size of stroke, time since the event, other bleeding risk, presence of intracardiac thrombus on echo, neurologic impairment, petechial hemorrhage in the infarct will impact on the recommendations.

Literature, Guidelines and Performance Measures

Strategy for Literature Search

The literature search for this guideline was conducted prospectively using the major keywords of “atrial fibrillation” or “atrial flutter.” Results were limited to humans, and published in the English language, for dates ranging from January 2011 to March 2013 on Medline. There were no limits on age groups. Results were limited to Guidelines, Clinical Trials, and Cohort Studies.
Additional key words included: rate control, rhythm control, procedures, established drug therapies, novel drug therapies, clinical classification systems/risk calculators, and post-operative.

The search was conducted in components each keyed to a specific causal link in a formal problem structure (available upon request). The search was supplemented with very recent clinical trials known to expert members of the panel. The search was a single cycle. Conclusions were based on prospective randomized clinical trials if available, to the exclusion of other data; if randomized controlled trials were not available, observational studies were admitted to consideration. If no such data were available for a given link in the problem formulation, expert opinion was used to estimate effect size.

Within the Cochrane Systematic Reviews, 35 reviews were found for the terms “atrial fibrillation” and “atrial flutter.”

Related National Guidelines

Within the National Guidelines Clearinghouse, these terms returned in 13 guidelines.

Related National Performance Measures

National programs that have clinical performance measures of atrial fibrillation and atrial flutter, primarily related to ambulatory care, include the following.

- Centers for Medicare & Medicaid Services, CMS179, ADE Prevention and Monitoring: Warfarin Time in Therapeutic Range, for adult patients with atrial fibrillation who are on chronic warfarin therapy. The measure includes Average percentage of time that patients in the measure population have INR results within the therapeutic range (i.e., TTR).
- National Quality Forum (NQF) Warfarin Therapy for Patients with Atrial Fibrillation. (NQF# 0084). Percent of all patients ≥ 18 years old with a diagnosis of heart failure and paroxysmal or chronic atrial fibrillation who were prescribed warfarin therapy. NQF. Ischemic stroke – Anticoagulation for A-fib/flutter. (NQF# 0436). Ischemic stroke patients with atrial fibrillation/flutter who are prescribed anticoagulation therapy at hospital discharge

Regional (Michigan) programs that have clinical performance measures of Atrial fibrillation or flutter include the following.

- Blue Cross Blue Shield of Michigan, EBCR.
  - CAD: Atrial Fibrillation/Atrial Flutter: Chronic Anticoagulation Therapy. Patients 18-64 years of age as of the end of the measurement period with medical and pharmacy coverage measuring the percent of calendar months during the measurement year in which at least 1 INR measurement was made.
- Blue Cross Blue Shield of Michigan, Physician Group Incentive Program clinical performance measures (PGIP).
  - #19 Atrial Fibrillation/Atrial Flutter--Chronic Anticoagulation Therapy. Percentage of patients aged 18 years and older with diagnosis of non-valvular atrial fibrillation (AF) or atrial flutter at high risk for thromboembolism who were prescribed warfarin
  - #20 Atrial Fibrillation or Atrial Flutter. Assessment of INR at least once monthly for patients with nonvalvular AF or atrial flutter receiving anticoagulation therapy with warfarin.

Disclosures

The University of Michigan Health System endorses the Guidelines of the Association of American Medical Colleges and the Standards of the Accreditation Council for Continuing Medical Education that the individuals who present educational activities disclose significant relationships with commercial companies whose products or services are discussed. Disclosure of a relationship is not intended to suggest bias in the information presented, but is made to provide readers with information that might be of potential importance to their evaluation of the information.

No team member reported a conflict of interest.

Review and Endorsement

Drafts of this guideline were reviewed in clinical conferences and by distribution for comment within departments and divisions of the University of Michigan Health System to which the content is most relevant: Emergency Medicine, General Medicine, Infectious Disease, Neurosurgery, Cardiology, Cardiac Surgery, Stroke, Pharmacy Services, and Thoracic Surgery. Medication recommendations were reviewed by the Pharmacy and Therapeutics Committee. The final version was endorsed by the Clinical Practice Committee of the University of Michigan Faculty Group Practice and the Executive Committee for Clinical Affairs of the University of Michigan Hospitals and Health Centers.

References

Initial AF/AFL Management

Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial


Ongoing AF/AFL management


Thromboembolic Risk Stratification


Anticoagulation


Patel et al. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. NEJM 2011;365:883-891

Pradaxa [package insert]. Ridgefield, CT: Boehringer Ingelheim Pharmaceuticals, Inc.; October 2010


Post Operative AF/AFL


## Appendix A. Commonly used antiarrhythmic drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Daily Dose</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Flecainide (Class IC) | 200 to 300 mg in two divided doses  | • A good first choice due to ease of administration and relative safety (lack of organ toxicity and low estimated incidence of proarrhythmia) - generally does not require admission for monitoring  
• Contraindicated in structural heart disease i.e. hypertrophy, cardiomyopathy and CAD  
• May convert AF to atrial flutter with 1:1 atrioventricular conduction, thus use in conjunction with AV nodal agents  
• Contraindicated in patients with Brugada Syndrome |
| Propafenone (Class IC) | 450 to 900 mg in two or three divided doses depending on formulation  | • A good first choice due to ease of administration and relative safety (lack of organ toxicity and low estimated incidence of proarrhythmia) - generally does not require admission for monitoring  
• Contraindicated in structural heart disease i.e. hypertrophy, cardiomyopathy and CAD  
• May convert AF to atrial flutter with 1:1 atrioventricular conduction, thus use in conjunction with AV nodal agents  
• Contraindicated in patients with Brugada Syndrome |
| Sotalol (Class III) | 160 to 320 mg in two divided doses  | • Generally should be initiated as an inpatient on telemetry  
• May cause QT prolongation and Torsades de pointes (proper monitoring essential for safe use)  
• Dosing should be adjusted to renal function  
• May exacerbate bradycardia and bronchospastic disease due to beta blocking activity |
| Dofetilide (Class III) | 250 to 1000 mg in two divided doses  | • Dofetilide must be initiated as an inpatient  
• Dofetilide is renally cleared and has multiple critical drug-drug interactions  
• May cause QT prolongation Torsades de pointes (proper monitoring essential for safe use)  
• Multiple critical drug-drug interactions  
• Dofetilide reserved to certified prescribers only |
| Amiodarone (Class III) | Loading dose:  | • The most effective antiarrhythmic medication for AF (FDA approved for ventricular arrhythmia management only)  
• May result in pulmonary and hepatic toxicity, hypothyroidism, and ocular side effects  
• Other side effects include photosensitivity, GI upset, and polyneuropathy  
• Doses of digoxin and warfarin should be reduced upon initiation of amiodarone in anticipation of the rises in serum digoxin levels and INR that typically occur.  
• In patients who are taking both simvastatin and amiodarone, the dose of simvastatin should not exceed 20 mg per day due to increased incidence of myopathy |
|               | Loading dose: (~10 gm total) 200 mg TID x 14 days, then 200 mg BID x 14 days. A higher daily dose over a shorter time period may be used if tolerated. Maintenance dose: 200mg daily |  
IV may be substituted for first 24 hours of therapy: 150mg IV bolus x 1, followed by IV infusion of 1mg/min x 6 hours, then 0.5mg/min for 18 hours. |
<table>
<thead>
<tr>
<th>Drug</th>
<th>Daily Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dronedarone (Class III)</td>
<td>400 mg Q12H</td>
<td>• Structurally similar to amiodarone except iodine moiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Less effective than amiodarone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Does not appear to cause pulmonary toxicity but severe hepatic failure requiring transplantation has been reported</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Contraindicated in patients with heart failure and permanent AF (increased mortality)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Multiple critical drug-drug interactions</td>
</tr>
<tr>
<td>Ibutilide (Class III)</td>
<td>≥ 60 kg: 1 mg IVPB over 10 minutes</td>
<td>• Potential for QT prolongation/Torsade de pointes – must be given on continuous telemetry and in the presence of the cardiologist or cardiology fellow. Proper resuscitation equipment should be available. Continuous telemetry monitoring should continue for 4 hours post infusion</td>
</tr>
<tr>
<td></td>
<td>&lt; 60 kg: 0.01 mg/kg IVPB over 10 minutes</td>
<td>• May consider infusion of magnesium (2 grams) immediately prior to ibutilide administration to reduce QT interval prolongation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Use not recommended in heart failure due to increased risk for proarrrhythmia</td>
</tr>
<tr>
<td></td>
<td>If arrhythmia doesn’t terminate within 10 minutes after end of initial infusion, may repeat dose over 10 minutes</td>
<td></td>
</tr>
<tr>
<td>Disopyramide (Class IA)</td>
<td>400 to 750 mg Q6h for immediate release formulation and Q12h for controlled release</td>
<td>• May be useful especially for vagally mediated AF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May cause glaucoma, urinary retention, dry mouth (anticholinergic)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Negative inotrope (may exacerbate heart failure)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May cause Torsade de pointes</td>
</tr>
<tr>
<td>Procainamide (Class IA)</td>
<td>1 gm IV over 1 hour for cardioversion</td>
<td>• Hepatic metabolism to active metabolite (NAPA) and renal elimination of parent drug and metabolites. Use with caution and reduce dose in hepatic or renal dysfunction</td>
</tr>
<tr>
<td></td>
<td>Maintenance infusion available but usually reserved for life-threatening arrhythmias</td>
<td>• Contraindicated in SLE</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May cause hypotension, Torsade de pointes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Negative inotrope (may exacerbate heart failure)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Procainamide and NAPA levels recommended with prolonged infusion</td>
</tr>
</tbody>
</table>
# Appendix B. Target-specific anticoagulants used for stroke prophylaxis in atrial fibrillation and atrial flutter

<table>
<thead>
<tr>
<th></th>
<th>Dabigatran</th>
<th>Rivaroxaban</th>
<th>Apixaban</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mechanism of action</strong></td>
<td>Direct thrombin inhibitor</td>
<td>Direct factor Xa inhibitor</td>
<td>Direct factor Xa inhibitor</td>
</tr>
<tr>
<td><strong>Tmax (hours)</strong></td>
<td>1-3</td>
<td>2-4</td>
<td>3-4</td>
</tr>
<tr>
<td><strong>Metabolism</strong></td>
<td>Phase II – glucoronidation</td>
<td>CYP3A4, CYP3A5, CYP2J2</td>
<td>CYP3A4, CYP3A5</td>
</tr>
<tr>
<td><strong>Excretion</strong></td>
<td>80% renal</td>
<td>2/3 liver, 1/3 renal</td>
<td>25% renal, 75% fecal</td>
</tr>
<tr>
<td><strong>Elimination half-life (hours)</strong></td>
<td>12-17</td>
<td>5-9</td>
<td>9-14</td>
</tr>
<tr>
<td><strong>Dose</strong></td>
<td>150mg PO BID</td>
<td>20mg PO daily</td>
<td>5mg PO BID</td>
</tr>
<tr>
<td><strong>Dose – Renal Impairment</strong></td>
<td>CrCl 15-30: 75mg BID</td>
<td>CrCl 15-50: 15mg daily</td>
<td>Any two: SCr 1.5-2.5; age ≥80 or weight &lt;60kg: 2.5mg BID</td>
</tr>
<tr>
<td><strong>Dose – Hepatic Impairment</strong></td>
<td>Moderate-severe: not recommended;</td>
<td>Child-Pugh B or C, or coagulopathy: not recommended;</td>
<td>Use caution</td>
</tr>
<tr>
<td><strong>Drug Interactions</strong></td>
<td>Ketoconazole or dronedarone &amp; CrCl 30-50 ml/min: 75mg BID; P-gp inhibitor &amp; CrCl 15-30 ml/min: not recommended; P-gp inducer: avoid</td>
<td>Strong dual- inhibitors or inducers of P-gp and CYP3A4: avoid; Combined P-gp and weak-moderate inhibitors of CYP3A4, &amp; CrCl 15-50ml/min: use caution</td>
<td>Strong dual-inhibitors of P-gp and CYP3A4: reduce dose to 2.5mg BID or avoid if 2.5mg BID is already indicated based on above factors; Strong dual-inducers: avoid</td>
</tr>
</tbody>
</table>
Appendix C. Management of acute atrial fibrillation/flutter after thoracic surgery.

**Unstable AF**
- Prepare for immediate DCCV.
- Call for support (RRT).
- Consider sedation/analgesia prior to DCCV.
- Energy selection: Afib 200 J, if unsuccessful increase to 360 J.
- A-flutter 50 J, if unsuccessful increase to 200 J.

Did the patient convert to sinus rhythm?
- Yes
  - Consider PICC, Repeat amiodarone 150 mg bolus IV every 20 minutes. Monitor blood pressure closely during boluses.

Did the patient have severe CPDU or a history of other severe lung disease?
- Yes
  - Amiodarone 150 mg IV over 10 min
  - Yes or unsure
    - Flecainide 200 mg PO/D tube (if >70 kg give 300 mg), then Flecainide 50 mg PO/D tube q12 hrs. Note: An AV nodal rate control drug (e.g., beta blocker or calcium channel blocker) must be coadministered with Flecainide see Table 4.
  - TTE if not done in the past 6 months.
  - Consult cardiology for cardioversion
  - Consider anticoagulation with warfarin input.
  - Consider need for rate control (Table 4)

  - Persistent or recurrent AF >24 hours?
    - Yes
      - Convert to amiodarone 0.5 mg/min IV (PICC), OR 200 mg po/D tube every 2 weeks, then 200 mg po/D tube BID for two weeks. Once in SR 24 hours okay to discharge.
    - No
      - Consult cardiology for cardioversion +/- antiarrhythmic.

  - Yes
    - Flecainide 50 mg po q12 hours x 31 days.
    - Note: An AV nodal rate control drug (e.g., beta blocker or calcium channel blocker) must be coadministered with flecainide see Table 4
    - Patient remains in AF: Go to A.
    - Patient has converted to sinus rhythm:
      - Transition to PO equivalent of above medications to complete a 3 week course (see Table 4)

  - Persistent or recurrent AF >24 hours?
    - Yes
      - Consider anticoagulation (Table 2, Figure 3)
    - No
      - Follow up with cardiology after discharge

- No
  - Amiodarone drip 1 mg/min x 6 hours then 0.5 mg/min x 18 hours

  - Convert to amiodarone 0.5 mg/min IV (PICC), OR 200 mg po/D tube every 2 weeks, then 200 mg po/D tube BID for two weeks. Once in SR 24 hours okay to discharge.

**Stable AF**
- Do you suspect an accessory pathway? (e.g.: Wide/bizarre/polyphoric QRS, History of WPW, or Delta wave on prior ECG)
- Yes
  - Defibrillator at the bedside
  - Procainamide 1g IV x1 over 60 min
  - Avoid AV nodal agents
  - Call STAT EP consult
  - Admit for clinical stability

- No
  - Amiodarone 150 mg IV over 10 min
  - Does the patient have structural heart disease? (e.g., LVH, RHF, EF<40%, need to severe valvular disease or CAD)
  - Yes
    - Flecainide 200 mg PO/D tube (if >70 kg give 300 mg), then Flecainide 50 mg PO/D tube q12 hrs. Note: An AV nodal rate control drug (e.g., beta blocker or calcium channel blocker) must be coadministered with Flecainide see Table 4.
  - TTE if not done in the past 6 months.
  - Consult cardiology for cardioversion
  - Consider anticoagulation with warfarin input.
  - Consider need for rate control (Table 4)

  - Persistent or recurrent AF >24 hours?
    - Yes
      - Convert to amiodarone 0.5 mg/min IV (PICC), OR 200 mg po/D tube every 2 weeks, then 200 mg po/D tube BID for two weeks. Once in SR 24 hours okay to discharge.
    - No
      - Consult cardiology for cardioversion +/- antiarrhythmic.

  - Yes
    - Flecainide 50 mg po q12 hours x 31 days.
    - Note: An AV nodal rate control drug (e.g., beta blocker or calcium channel blocker) must be coadministered with flecainide see Table 4
    - Patient remains in AF: Go to A.
    - Patient has converted to sinus rhythm:
      - Transition to PO equivalent of above medications to complete a 3 week course (see Table 4)

  - Persistent or recurrent AF >24 hours?
    - Yes
      - Consider anticoagulation (Table 2, Figure 3)
    - No
      - Follow up with cardiology after discharge

**Rate Control**: See Table 4 for dosing and additional information
- Start with a 1st line agent such as a BB (metoprolol or esmolol) or a CCB (diltiazem).
- If the patient has a strong indication for a BB (e.g., CAD, tachyarrhythmias, or left systolic CHF) or an allergy or intolerance to a CCB, then a BB may be preferred.
- If the patient has an allergy or intolerance to a BB, or is having acute bronchospasm, then a CCB may be preferred.
- Esmolol is the BB of choice for patients with tachycardia or tachyarrhythmia, given its short half-life.
- If one of the 1st line drugs does not achieve rate control, try a drug from the other class (BB, CCB) of 1st line agents.

---

BB = beta blocker
CCB = calcium channel blocker