1. Establish an Accurate Diagnosis of AF

- AF is characterized by replacement of consistent P waves with fibrillatory waves, varying in amplitude, shape, and timing.
- The ventricular response is irregular and frequently rapid when AV nodal conduction is intact.
- In patients with pacemakers, diagnosis of AF may require temporary inhibition of the pacemaker to expose atrial activity.
- AF should be distinguished from 1) atrial flutter, which has regular organized atrial activity with a rate typically between 240 and 320 bpm, 2) multifocal atrial tachycardia, which has 3 or more distinct P waves of variable morphology, 3) regular supraventricular tachycardias, such as AV nodal reentry and 4) sinus rhythm (SR) with multiple premature atrial complexes.

2. Determine AF Pattern, Clinical History, and Symptoms

- Clinical type of AF can be classified as:
  - Paroxysmal: Recurrent AF (≥2 episodes) that terminates spontaneously within 7 days. Episodes of AF of ≤48 hours’ duration that are terminated with electrical or pharmacologic cardioversion should also be classified as paroxysmal AF.
  - Persistent: AF that is sustained >7 days. Episodes of AF terminated by electrical or pharmacologic cardioversion after ≥48 hours of continuous AF, but prior to 7 days, should also be classified as persistent AF episodes.
  - Longstanding persistent: Continuous AF of >1 year duration.
  - Permanent: AF for which a decision has been made, by the patient and the physician treating the AF, not to pursue restoration of SR by any means.

- Onset of the first symptomatic attack or date of discovery of AF.
- The onset of the current episode, if persistent.
- Presence and severity of symptoms associated with AF.
- Frequency, duration, precipitating factors, and modes of termination of AF.
- Presence of other symptoms that might indicate an etiology.
- History of prior evaluation and response to prior management.
- An event recorder may be useful to correlate symptoms with the rhythm and determine the classification of AF.
- Identification of thromboembolic and bleeding risks.

3. Assess for Structural Heart Disease

- Patients who initially present with AF should be evaluated for concomitant structural heart disease. The presence or absence of heart disease will help to individualize AF management.
- Coronary artery disease should be excluded in patients with risk factors but is rarely a reversible cause of AF.
- Severe left atrial dilation correlates with a low likelihood of maintenance of SR.

4. Identify Correctable Secondary Causes

- Rule out potentially correctable causes such as sleep apnea, hyperthyroidism, WPW, and drug or alcohol abuse.

5. Develop a Treatment Strategy

**Management Principles**

- A comprehensive treatment plan must address the three cornerstones of AF management: (1) rate control, (2) rhythm control, and (3) prevention of thromboembolism.
- Hospitalization should be considered in patients who are significantly symptomatic, hemodynamically unstable, or being started on an antiarrhythmic drug.
- Electrical cardioversion can be performed as an outpatient procedure.
- When the cause of AF is reversible, such as AF after cardiac surgery, no long-term therapy may be necessary.
- Patients being treated by a cardiologist who continue to be symptomatic or are difficult to manage should be referred to an electrophysiologist.

**Rate and Rhythm Control**

- The AFFIRM, RACE, and AF-CHF trials have shown no mortality benefit to a rhythm control strategy compared to a rate control strategy.
- Therefore, a rate control strategy, without attempts at restoration or maintenance of SR, is reasonable in some patients with AF, especially those who are elderly and asymptomatic.
- If rate control offers inadequate symptomatic relief, restoration of SR may become a long-term goal.
- Restoration and maintenance of SR continues to be a reasonable treatment approach in many patients with AF.
**Stroke Prevention**

- Antithrombotic therapy to prevent thromboembolism is recommended for all patients with AF regardless of whether a rhythm or rate control strategy is chosen, except those with lone AF or contraindications.
- The CHADS\(_2\) scoring system can be used to risk-stratify patients with nonvalvular AF to determine the need for anticoagulation therapy. The annual risk of stroke with a CHADS\(_2\) score of 0 is 1.9%, but the annual risk of stroke with a CHADS\(_2\) score of 6 is 18.2%.
- Long-term oral anticoagulation (warfarin, Factor Xa inhibitor, or direct thrombin inhibitor) is indicated in patients with a CHADS\(_2\) score of ≥ 2 and should be considered in patients with a CHADS\(_2\) score of 1. For patients at low risk of stroke (CHADS\(_2\)=0-1), the presence of additional risk factors for stroke such as age 65-74 years, female sex, and the presence of vascular disease (as used in the CHA\(_2\)DS\(_2\)-VASc scoring system) should also be considered.\(^3\)
- Aspirin plus clopidogrel is not a substitute for warfarin or the newer oral anticoagulants.
- Antithrombotic therapy is recommended for patients with atrial flutter as for those with AF.
- In patients with AF who do not have mechanical valves and are not at a particularly high risk of stroke (have not had recent stroke or TIA, rheumatic valve disease, cardioversion in the past month), it is reasonable to interrupt anticoagulation for up to 1 week without substituting heparin for procedures that carry a risk of bleeding.
- Patients with AF who have hypertrophic cardiomyopathy, mitral stenosis, or a mechanical valve should be treated with warfarin.

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<table>
<thead>
<tr>
<th>CHADS(_2) Risk Criteria</th>
<th>Score</th>
</tr>
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<tbody>
<tr>
<td>Congestive Heart Failure</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
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<tr>
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</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke or TIA in the past</td>
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</table>

<table>
<thead>
<tr>
<th>CHADS(_2) Score</th>
<th>Recommended Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Aspirin (81 to 325 mg daily) or no therapy*</td>
</tr>
<tr>
<td>1</td>
<td>Aspirin (81 to 325 mg daily) or oral anticoagulant**</td>
</tr>
<tr>
<td>≥2</td>
<td>Oral anticoagulant**</td>
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</table>

<table>
<thead>
<tr>
<th>CHA(_2)DS(_2)-VASc Risk Criteria(^3,4)</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive Heart Failure/LV Dysfunction</td>
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</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Age &gt; 75 Years</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Prior Stroke, TIA, thromboembolism</td>
<td>2</td>
</tr>
<tr>
<td>Peripheral Vascular Disease or Coronary Artery Disease</td>
<td>1</td>
</tr>
<tr>
<td>Age 65-74 Years</td>
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<tr>
<td>Sex Category (i.e., Female Sex)</td>
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</table>

<table>
<thead>
<tr>
<th>CHA(_2)DS(_2)-VASc Score</th>
<th>Recommended Therapy</th>
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<tr>
<td>0</td>
<td>No therapy preferred</td>
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<tr>
<td>1</td>
<td>Aspirin, 81 to 325 mg daily, or oral anticoagulant**</td>
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<tr>
<td>≥2</td>
<td>Oral anticoagulant**</td>
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<thead>
<tr>
<th>Score</th>
<th>Adjusted stroke rate (%/year) based on CHADS(_2) score(^6)</th>
<th>Adjusted stroke rate (%/year) based on CHA(_2)DS(_2)-VASc score(^4)</th>
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<tr>
<td>0</td>
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<td>1</td>
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<td>8</td>
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<td></td>
</tr>
<tr>
<td>9</td>
<td>15.2</td>
<td></td>
</tr>
</tbody>
</table>

* No therapy is acceptable for patients < 65 years old and no heart disease (lone AF).

** If warfarin is the oral anticoagulant used, INR should be 2.0 to 3.0, with a target of 2.5. INR < 2.0 is not effective at preventing strokes. If mechanical valve, target INR > 2.5.
**Warfarin alternatives**  
(Dosing must be adjusted for renal insufficiency):  
- Dabigatran, a direct thrombin inhibitor, is superior to warfarin for stroke prophylaxis in atrial fibrillation.  
- Rivaroxaban, an oral factor Xa inhibitor, is non-inferior to warfarin for the prevention of stroke or systemic embolism. If anticoagulation with rivaroxaban must be discontinued for a reason other than bleeding, consideration must be given to administering another anticoagulant (FDA boxed warning).  
- Apixaban, an oral factor Xa inhibitor, is superior to warfarin in preventing stroke or systemic embolism, and is associated with less bleeding and lower mortality. There is an increased risk of stroke following discontinuation of apixaban in patients with nonvalvular AF. If apixaban must be discontinued for a reason other than bleeding, coverage with another anticoagulant should be strongly considered (FDA boxed warning).

<table>
<thead>
<tr>
<th>Mechanism of Action</th>
<th>Direct thrombin inhibitor</th>
<th>Direct factor Xa inhibitor</th>
<th>Direct factor Xa inhibitor</th>
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<tr>
<td><strong>Pro-Drug</strong></td>
<td>Yes</td>
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<td>No</td>
</tr>
<tr>
<td><strong>Food Effect</strong></td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>Dosing (PO)</strong></td>
<td>75-150mg bid*</td>
<td>2.5-5mg bid*</td>
<td>15-20mg qd**</td>
</tr>
<tr>
<td>Renal Clearance</td>
<td>85%</td>
<td>~27%</td>
<td>~33%</td>
</tr>
<tr>
<td>Mean Half Life (t1/2)</td>
<td>14-17 hrs</td>
<td>~12 hrs</td>
<td>5-13 hrs</td>
</tr>
<tr>
<td>Time to peak effect</td>
<td>0.5-2 hrs</td>
<td>3-4 hrs</td>
<td>2-4 hrs</td>
</tr>
</tbody>
</table>

*150mg bid for patients with CrCl > 30mL/min; 75mg bid for patients with CrCl 15-30mL/min. Discontinue use in patients who develop acute renal failure. Do not use in patients with mechanical heart valves.

* 5mg bid is recommended dose. 2.5mg bid is recommended for patients with at least two of the following: age of 80 yrs or more, body weight of 60 kg or less, SCr of 1.5 mg/dL or more. Not recommended for use in patients with severe hepatic impairment.

** 20 mg with evening meal for patients with CrCl > 50mL/min; 15mg with evening meal for patients with CrCl 15-50mL/min. Do not use in patients with moderate and severe hepatic impairment or with hepatic disease associated with coagulopathy.

**SOURCE:** Data from individual drug package inserts.

**Ventricular Rate Control**

**Principles of Rate Control Strategy**

- Adequate control of the ventricular response during AF can significantly improve symptoms and is critical to avoid tachycardia-mediated cardiomyopathy.
- Most patients managed using a rhythm control strategy also require medications for rate control.
- Hospitalization is rarely required to control the ventricular response during AF, unless the patient is symptomatic.
- Rate control for atrial flutter tends to be more difficult than for AF.

**What is Adequate Rate Control?**

- Control of the ventricular rate during AF is important both at rest and with exertion.
- Criteria for adequate rate control vary:
  - For the AFFIRM trial, adequate control was defined as an average HR< 80 bpm at rest and either an average rate< 100 bpm during Holter monitoring with no rate above 100% of the maximum age-adjusted predicted exercise HR, or a maximum HR of 110 bpm during a 6-min walk test.
  - In the RACE II trial, lenient HR control (target< 110 bpm) was noninferior to strict HR control (resting rate< 80 bpm and rate during moderate exercise< 110 bpm).

**Drugs to Control the Ventricular Response**

**AV nodal blocking drugs** that can be used to control the ventricular response include:

**Beta Blockers, Calcium Channel Antagonists (nondihydropyridine), and Digoxin**

- Beta blockers are the most effective drug class for rate control.
- Digoxin provides relatively poor rate control during exertion and should be reserved for patients who are sedentary or those with systolic HF.
- Digoxin does not convert AF to SR and may perpetuate AF.
- A combination of a beta blocker and either a calcium channel antagonist or digoxin may be needed to control the HR.
- The choice of medication should be individualized and the dose modulated to avoid bradycardia.
- Beta blockers and calcium channel antagonists should be used cautiously in patients with HF.
- AV nodal blocking drugs at doses needed to control the ventricular response can cause symptomatic bradycardia requiring pacemaker therapy.
- Some antiarrhythmic drugs that are used to maintain sinus rhythm, such as sotalol, dronedarone, and amiodarone, also provide some control of the ventricular response when patients are in AF.
• Amiodarone should rarely be used for rate control because of its potential for toxicity.
• IV digoxin and nondihydropyridine calcium channel antagonists are contraindicated in patients with ventricular preexcitation during AF (WPW syndrome) because they may accelerate the ventricular response and precipitate VF.
• Doses for commonly used drugs are shown on the last page of this guide.

AV Nodal Ablation
• Ablation of the AV conduction system and permanent pacing (the “ablate and pace” strategy) is an option for patients who have rapid ventricular rates despite maximum medical therapy and often yields remarkable symptomatic relief.
• There is growing concern about the negative effects of long-term RV pacing.
• Biventricular pacing may overcome many of the adverse hemodynamic effects associated with RV pacing and should be considered in selected patients after AV node ablation.
• Catheter ablation of the AV node should only be considered after rate-control strategies have been exhausted.

Anticoagulation Considerations with Cardioversion
• For all patients with AF for > 48 hours, or when AF duration is unknown, 3 weeks of therapeutic anticoagulation is required prior to cardioversion (CV).
• Transesophageal echocardiography (TEE) to exclude the presence of LA thrombus can be used as an alternative to 3 weeks of anticoagulation prior to CV. For patients starting warfarin and at high risk for thromboembolism, heparin or low molecular weight heparin should be initiated and continued until a therapeutic level of warfarin has been established.
• Anticoagulation must be continued for at least 4 weeks after CV regardless of the use of TEE before CV. Anticoagulation after 4 weeks is dependent upon the patient’s risk of stroke regardless of the perceived effectiveness of rhythm control.

Direct Current Cardioversion
• Shocks should be delivered synchronous to the R-wave.
• The use of a biventricular defibrillator should be considered with 150-200 joules as the initial energy setting.
• When a rapid ventricular response does not respond promptly to pharmacological measures for AF patients with ongoing myocardial ischemia, symptomatic hypotension, angina, or HF, immediate CV is recommended.
• In case of early relapse of AF after CV, repeated direct-current CV attempts may be made following administration of antiarrhythmic medication.
• Electrical CV is contraindicated in patients with digitalis toxicity or hypokalemia.

Pharmacological Cardioversion
• IV ibutilide is an effective drug available to convert AF.
  – Due to its risk of torsades de pointes, ibutilide should be avoided in patients with severe systolic dysfunction (EF <20%) or a prolonged QTc (> 480 ms).
  – More effective for conversion of atrial flutter than of AF; more effective in cases of more recent onset.
  – Can also be used to facilitate electrical CV when it is unsuccessful, or when there is an immediate recurrence of AF after initially successful CV.
  – Consider IV magnesium (2 grams) prior to giving ibutilide to reduce risk of torsades de pointes.
  – Electrocardiographic (ECG) monitoring must be performed for 4 hours after administration.
• Flecainide and Propafenone
  – Both flecainide and propafenone have been studied for their use as a “pill-in-the-pocket” approach to cardioverting AF.
  – Generally, a beta blocker or a calcium channel blocker should be taken an hour prior to taking the antiarrhythmic drug when trying to convert AF to SR. For a person > 70 Kg, 300 mg of flecainide or 600 mg of propafenone should be administered. For < 70 Kg, the dose for flecainide and propafenone is 200 mg and 450 mg, respectively. After administration of the drug, heart rhythm must be monitored for at least 4-8 hours.

Restoration of Sinus Rhythm

Principles of Cardioversion
• CV may be achieved by means of a drug or an electrical shock.
• Direct-current CV is more effective than pharmacological CV.
• The more recent the onset of AF, the more effective is pharmacological CV.
• The primary disadvantage of electrical CV is that it requires sedation or anesthesia.
• The primary disadvantage of pharmacological CV is the risk of ventricular proarrhythmia.
• The risk of thromboembolism or stroke does not differ between pharmacological and electrical CV.
• Be prepared for significant bradycardia after CV in patients on high-dose AV nodal blocking drugs.
• Antiarrhythmic drug therapy may be administered prior to CV to facilitate long-term success and maintenance of normal sinus rhythm.
Principles of Antiarrhythmic Drug Therapy

- Pharmacological therapy to maintain SR is indicated in patients who have troublesome symptoms related to paroxysmal AF or recurrent AF after CV who can tolerate antiarrhythmic drugs and have a good chance of remaining in SR.
- Antiarrhythmic drug choice is based on side effect profiles and the presence or absence of structural heart disease, HF, and hypertension (see flow diagram).
- Drug choice should be individualized and must account for underlying renal and hepatic function.
- Goals of drug therapy are to decrease the frequency and duration of episodes, and to improve symptoms. AF recurrence while taking an antiarrhythmic drug is not indicative of treatment failure and does not necessitate a change in antiarrhythmic therapy.
- An antiarrhythmic drug should be abandoned when it does not result in symptomatic improvement or causes adverse effects.
- Ensure normal electrolyte status and appropriate anticoagulation prior to starting antiarrhythmic drug therapy.
- Initiate AV nodal blockade prior to use of antiarrhythmics (e.g., flecainide) that do not provide substantial AV node blockade.
- Initiate therapy at low dose and titrate up as needed and after evaluating drug effects on ECG parameters.

Specific Antiarrhythmic Drugs

- Flecainide and propafenone are class IC drugs that delay conduction by blocking sodium channels. Propafenone also exerts mild beta-blocking effects. These drugs have been shown to prolong the time to first recurrence of AF, but should not be used in patients with ischemic heart disease or LV dysfunction due to the high risk of proarrhythmia.
- These drugs can also be used for acute pharmacological conversion in a monitored setting.
- Class IC drugs can slow the atrial rhythm during AF resulting in acceleration of the ventricular response. Therefore, these agents should be combined with AV nodal blocking drugs to maintain rate control when AF recurs.
- Outpatient initiation may be considered in patients in sinus rhythm in the absence of structural heart disease or sinus or AV node dysfunction.

Sotalol

- Sotalol is a nonselective beta-blocking drug with class III antiarrhythmic activity that prolongs repolarization. It is not effective for conversion of AF to sinus rhythm, but may be used to prevent AF. Sotalol should be avoided in patients with asthma, HF, renal insufficiency, or QT interval prolongation and should be used with caution in those at risk for torsades de pointes (e.g., female, age > 65 yr, taking diuretics).
Dofetilide

- Dofetilide is a pure class III drug that prolongs repolarization by blocking the rapid component of the delayed rectifier potassium current.
- Dofetilide was shown in the SAFIRE-D trial to be effective in maintaining sinus rhythm. To reduce the risk of early torsades de pointes, dofetilide must be initiated in the hospital at a dose titrated to renal function and the QT interval. Dofetilide is safe to use in patients with coronary artery disease or CHF. The FDA mandates prescriber registration and inpatient loading for initiation of this medication due to its proarrhythmic potential.

Amiodarone

- Amiodarone is the most effective antiarrhythmic drug, but is associated with relatively high toxicity, making it a second-line or last-resort agent in many cases.
- Amiodarone is an appropriate initial choice in patients with LVH, HF, or CAD, because it is associated with a low risk of proarrhythmia.
- Outpatient initiation may be considered in the absence of other risk factors for torsades de pointes and sinus or AV node dysfunction. Patients taking amiodarone should be monitored at least annually for thyroid, hepatic, and pulmonary toxicity.
- Low-dose amiodarone (≤ 200 mg daily) is associated with fewer side effects than higher-dose regimens.

Dronedarone

- Dronedarone is an analog of amiodarone with far lower risk of organ toxicity.
- Outpatient initiation may be considered in the absence of other risk factors for torsades de pointes, and sinus or AV node dysfunction.
- Dronedarone is indicated to reduce the risk of cardiovascular hospitalization in patients with paroxysmal or persistent AF/AFL, with a recent episode of AF/AFL and associated cardiovascular risk factors, who are in sinus rhythm or who will be cardioverted.
- Dronedarone is contraindicated in patients with decompensated congestive heart failure. It should be avoided in patients with advanced CHF. It is also contraindicated in patients with permanent AF (patients in whom sinus rhythm will not or cannot be restored) and for the sole purpose of rate control.
- There is a very small risk of liver toxicity with dronedarone and, therefore, liver function testing is recommended after drug initiation.
# DOUGLAS GUIDE FOR DRUGS COMMONLY USED TO TREAT AF

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOsing</th>
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</thead>
<tbody>
<tr>
<td><strong>HEART RATE CONTROL</strong></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>IV: 0.25mg q2hrs (up to 1.5mg), then 0.125-0.375mg daily  &lt;br&gt; PO: 0.125-0.375mg daily</td>
</tr>
<tr>
<td><strong>Beta Blockers</strong></td>
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</tr>
<tr>
<td>Atenolol</td>
<td>PO: 25-100mg daily</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>PO: 2.5mg daily; can be titrated to 20mg daily</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>PO: 3.125-25mg every 12 hrs (up to 50mg every 12 hrs for patients &gt; 85kg), may use carvedilol sustained release 10-80mg daily</td>
</tr>
<tr>
<td>Esmolol</td>
<td>IV: 500 mcg/kg over 1 min, then 50-200 mcg/kg/min</td>
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<tr>
<td>Metoprolol</td>
<td>IV: 2.5-5mg bolus over 2 min (up to 3 doses)  &lt;br&gt; PO: 25-100mg bid, may use metoprolol succinate ER 25-200mg daily</td>
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<tr>
<td><strong>Calcium Channel Blockers</strong></td>
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<tr>
<td>Diltiazem</td>
<td>IV: 0.25mg/kg (avg 20mg) over 2 min (2nd bolus can be given if HR &gt;100bpm), then 5-15mg/hr  &lt;br&gt; PO: 120-360mg daily (slow release preferred)</td>
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<tr>
<td>Verapamil</td>
<td>IV: 0.075-0.15mg/kg over 2 min  &lt;br&gt; PO: 120-360mg daily (slow release preferred)</td>
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<tr>
<td><strong>HEART RHYTHM CONTROL</strong></td>
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</tr>
<tr>
<td><strong>Vaughan Williams Class I</strong></td>
<td></td>
</tr>
<tr>
<td>Flecainide</td>
<td>PO: 50-150mg every 12 hrs</td>
</tr>
<tr>
<td>Propafenone</td>
<td>PO: 150-300mg every 8 hrs, or sustained release 225-425mg every 12 hrs</td>
</tr>
<tr>
<td><strong>Vaughan Williams Class III</strong></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>IV: 150mg over 10 min, then 0.5-1mg/min  &lt;br&gt; PO: 200mg TID x 2 wks, 200mg BID x 2 wks, then 200mg daily. Take with meals.</td>
</tr>
<tr>
<td>Dofetilide</td>
<td>PO: 125-500mcg every 12 hrs, based on renal function and QTc; must be initiated in the hospital</td>
</tr>
<tr>
<td>Dronedarone</td>
<td>PO: 400mg twice daily with meals</td>
</tr>
<tr>
<td>Ibutilide</td>
<td>IV: ≥ 60kg – 1mg over 10 min; &lt;60kg – 0.01mg/kg over 10 min while observing for QTc prolongation and ventricular proarrhythmia. Dose can be repeated after 10 min but the risk of proarrhythmia increases. Pre-treatment with MgSO4 1-2 gm IV may reduce the risk of TdP.</td>
</tr>
<tr>
<td>Sotalol</td>
<td>PO: 80mg BID, to a maximum of 240-320mg/day, based on renal function and QTc</td>
</tr>
</tbody>
</table>

**References**


This pocket guide was supported in part by Sanofi and Biosense Webster, a Johnson and Johnson Company