Contemporary Management of Atrial Fibrillation

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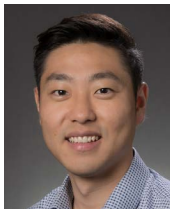

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Atrial fibrillation (AF) is the most common arrhythmia diagnosed in clinical practice.[1] It is estimated that 12.1 million people in the United States will have AF by the year 2030.[2] Currently, more than 450,000 hospitalizations with AF as the primary diagnosis occur each year.[2] AF contributes to about 158,000 deaths annually, and the rate from AF as a primary or contributing cause of death has been rising for more than two decades.[2] As these statistics suggest, most medical providers have encountered patients with some form of AF. This contemporary review will discuss management for patients with AF.

EVALUATION OF THE AF PATIENT

Symptoms of AF vary by individual. Symptoms may range from fatigue, shortness of breath, palpitations, chest discomfort, edema, exercise intolerance, hypotension, or even syncope. Conversely, some pateints may be asymptomatic with AF. Many times, AF is discovered in the presence of other underlying heart disease, which may be due to the consequence of the rhythm itself.[4] Suboptimal heart rate control and loss of atrioventricular synchrony can impact hemodynamics drastically and contributes to hypertension, hypotension, heart failure exacerbation, and/or stroke.[1,5]

RISK FACTORS

Risk factors for AF may include hypertension, ischemic heart disease, obesity, sleep apnea, diabetes, and heart failure.[6] These comorbid conditions can promote strain on the myocardium, which leads to atrial stretch and dilatation.[4] Atrial hypertrophy are associated with a lower success rates of treatment interventions including pharmacological or electrical cardioversion, or catheter ablation.[4] Many of these risk factors can be managed with lifestyle modification and/or treatment of morbidities; therefore, these comorbidities should be proactively managed to prevent atrial stretch, adverse atrial remodeling, and reduction of AF triggers.[4]

AF AND STROKE RISK

It is well known that AF increases stroke risk. AF is associated with an approximately a fivefold increased risk of ischemic stroke. Cardioembolic strokes have the highest morbidity and mortality burden of all stroke subtypes.[8] These strokes also have a high risk of recurrence if the appropriate use of antithrombotic therapy is not used.[8]

Anatomically, the left atrial appendage (LAA) is the major source of thrombus formation in patients with AF.[9] The LAA loses its contractile strength when a patient is in AF, causing blood to pool and stagnate within the structure. This pooling leads to thrombus formation.[10]

The CHA2DS2-VASc point system, which stratifies ischemic stroke risk among patients with nonvalvular AF, has become the standard for anticoagulation recommendations.[11] When a patient in AF needs assessment for stroke risk and anticoagulation, the risk of bleeding must also be reviewed. The HAS-BLED score is commonly used to identify patients that may be at risk for bleeding and require close observation for anticoagulation monitoring or require further workup for non-pharmacological stroke prevention, such as left atrial appendage management.[1] Therefore, all patients with documented AF should have both assessments **(Table 1)**.

Assessment using the CHA2DS2-VASc and HAS-BLED scoring systems is important for every AF patient **(Table 1)**. Fortunately, an increase in oral anticoagulation options for nonvalvular AF patients has allowed for better compliance and reduced the need for frequent monitoring.[12] Selection of oral antithrombotic therapy depends on clinical factors an in some cases financial consideration. For male AF patients with a CHA2DS2-VASc of 2 or greater, and females with a CHA2DS2-VASc of 3 or greater, oral anticoagulation is recommended **(Figure 1)**.[6]

LEFT ATRIAL APPENDAGE MANAGEMENT

Oral anticoagulation is essential for thromboembolic prevention for patients with atrial fibrillation. While many patients do well with oral anticoagulants, there are some who may not tolerate oral anticoagulation or have increased bleeding risk; fortunately, these patients have options for non-pharmacological thromboembolic prevention.

Table 1. Assessment of Stroke (CHA_2DS_2-VASc) and Bleeding Risk (HAS-BLED) in Atrial Fibrillation Patients. (Note: maximum score is 9 since age may contribute 0,1, or 2 points)

CHA_2DS_2-VASc[1]	Score	HAS-BLED[2]	Score
Congestive heart failure/LV dysfunction	1	**H:** Hypertension (systolic blood pressure >160 mm Hg)	1
Hypertension	1	**A:** Abnormal renal and liver function (1 point each)	1 or 2
Age ≥75 years	2	**S:** Stroke	1
Diabetes mellitus	1	**B:** Bleeding tendency or predisposition	1
Stroke/TIA/TE	2	**L:** Labile INRs	1
Vascular disease (prior MI, PAD, or aortic plaque)	1	**E:** Elderly (>65 years)	1
Aged 65 to 74 years	1	**D:** Drugs (concomitant aspirin or NSAIDs) or excess alcohol use (1 point each)	1 or 2
Sex Category	1		
Maximum Score	9		9

[1] TIA indicates transient ischemic attack; TE, thromboembolic; INR, international normalized ratio; MI, myocardial infarction; and PAD, peripheral artery disease. CHA_2DS_2-VASc score of 0: recommend no antithrombotic therapy. CHA_2DS_2-VASc score of 1: recommend antithrombotic therapy with oral anticoagulation or antiplatelet therapy but preferably oral anticoagulation. CHA_2DS_2-VASc score ≥2: recommend oral anticoagulation.[2] A HAS-BLED score of ≥3 indicates that caution is warranted when prescribing oral anticoagulation and regular review is recommended.[2]

[2] Abnormal renal function is classified as the presence of chronic dialysis, renal transplantation, or serum creatinine ≥200 mmol/L. Abnormal liver function is defined as chronic hepatic disease (eg, cirrhosis) or biochemical evidence of significant hepatic derangement (bilirubin 2 to 3 times the upper limit of normal, in association with aspartate aminotransferase/alanine aminotransferase/alkaline phosphatase 3 times the upper limit normal, etc), history of bleeding or predisposition (anemia), labile INR (ie, time in therapeutic range <60%), concomitant antiplatelets or nonsteroidal anti-inflammatory drugs, or excess alcohol. Used with permission from the American College of Chest Physicians. Chest 2010. Published by Elsevier.

Left atrial appendage occlusion (LAAO) devices are an increasing option for patients that are poor candidates for anticoagulation due to bleeding risk, fall risk, and/or non-compliance.[9,13] Using a percutaneous access sheath, these devices are deployed into the LAA **(Figure 2)**. The device compresses or hooks into the base of the LAA, occluding the LAA from the rest of the left atrium. The device usually endothelializes 45 to 60 days after implant.[9]

Surgical management of the LAA is widely practiced and is recommended during open heart surgeries by the Society of Thoracic Surgeons guidelines.[14] Management includes LAA clips or surgical closure by means of stapling, amputation, ligation, or sewing.

LAA CLIP

A minimally invasive surgical option for LAA management with a specialized clip has encouraging outcomes by not only providing structural isolation, but electrical isolation of the LAA from the left atrial body as well, thereby adding the benefit of eliminating an arrhythmic trigger for AF **(Figure 2)**.[15] The LAA clip procedure can be performed as a stand-alone thoracoscopic procedure or can be concomitant during other cardiac surgeries. In a recent trial, patients with AF who had undergone cardiac surgery with concomitant atrial appendage occlusion had a reduced risk of ischemic stroke or systemic embolism.[13,16]

AF CLASSIFICATION

The treatment of AF is largely dependent on the frequency and duration of episodes as well as severity of symptoms. Accurately characterizing AF is clinically relevant since successful outcomes are best achieved if treatments are implemented within the first six months of the first episode of AF.[18,19] Accurate classification will help the provider develop a tailored longitudinal plan for patient care **(Panel 1)**.

TREATMENT OPTIONS FOR AF: CURRENT GUIDELINES FOR RATE AND RHYTHM CONTROL

Rate Control Medications

Rate control recommendations have remained the first-line strategy for patients with a diagnosis of AF.[5] Controlling heart rate improves symptoms, reduces mortality, and reduces the risk of tachycardia-mediated cardiomyopathy.[5] According to the

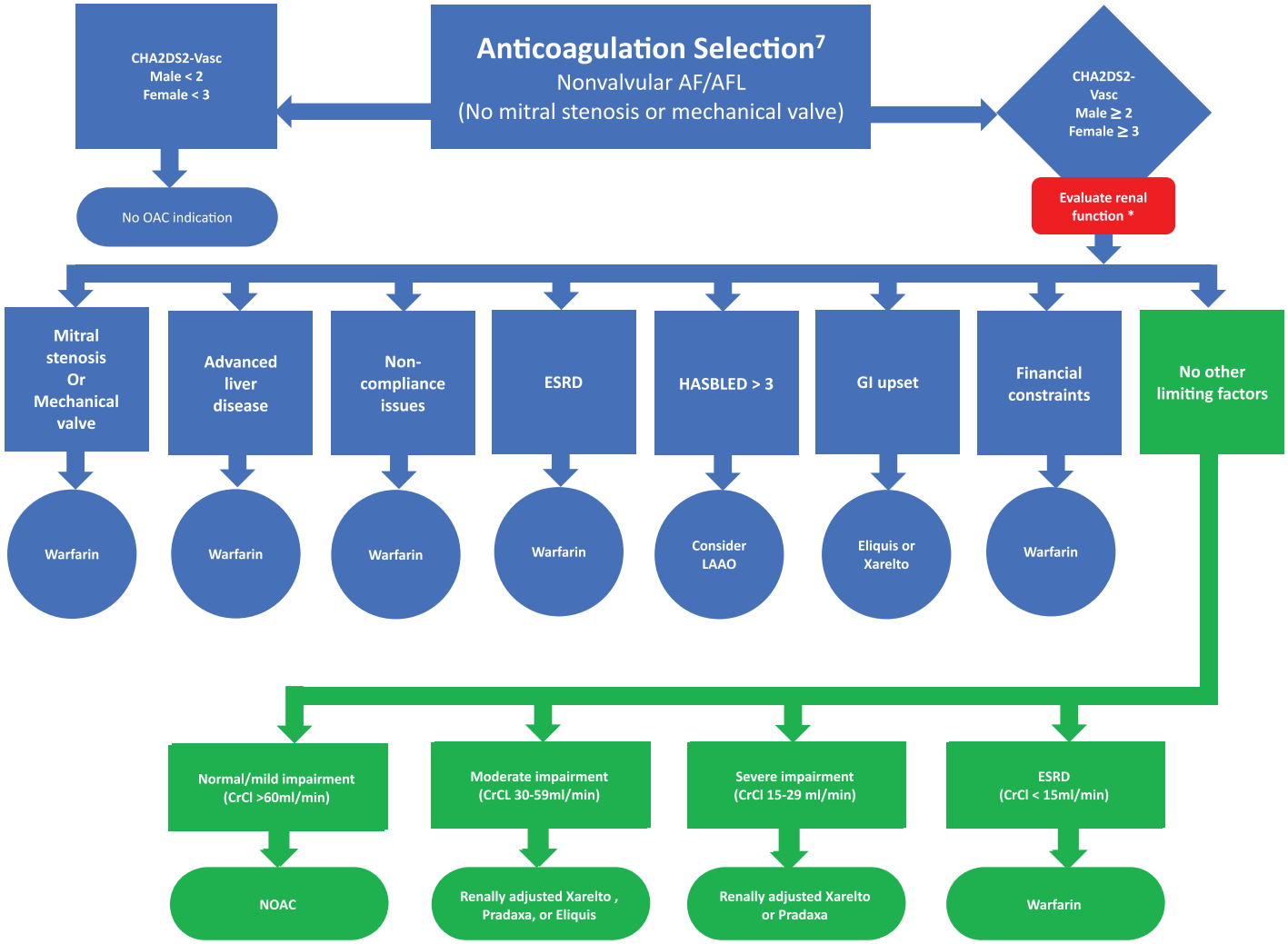

Figure 1: *Anticoagulation Selection.*
**Renal function should be evaluated on all patients prior to initiating anticoagulation.*

2014 AHA/ACC/HRS Guidelines for the Management of Patients with Atrial Fibrillation, the first line medications for rate control are beta blockers or nondihydropyridine calcium channel antagonists. **Panel 2** lists class I, IIa, and IIb recommendations.[5]

Rhythm Control — When Rate Control Is Not Enough

There is increasing evidence that rate control strategies for newly detected AF patients may not be as effective as attempting rhythm control.[20] Maintaining sinus rhythm may improve symptoms reduce the risk of atrial remodeling, AF-related death, heart failure, and strokes in high-risk patients.[20]
A recent analysis from the Get With The Guidelines-Atrial Fibrillation® registry showed that patients admitted to the hospital with first-detected AF had a shorter length of stay when a plan for rhythm control was in place.[21] The analysis also showed that a higher number of patients with a rhythm control plan in place were more likely to discharge home versus a facility, thereby reducing the overall burden on healthcare systems.[21] Another recent review of AF trials evaluating the use of early rhythm control showed that patients with a new diagnosis of AF (<1 year) had a lower rate of cardiovascular death and stroke. Patients with AF lasting more than one year showed no significant difference in cardiovascular death or stroke.[22]

There are an increasing number of options for rhythm control, and the technological advancements for procedural options continue to improve over time. Some patients may benefit from less invasive first line treatment options such as antiarrhythmic medications or cardioversion in order to assess their symptoms and/or comorbid conditions, such as heart failure, while in normal sinus rhythm.[20,23]

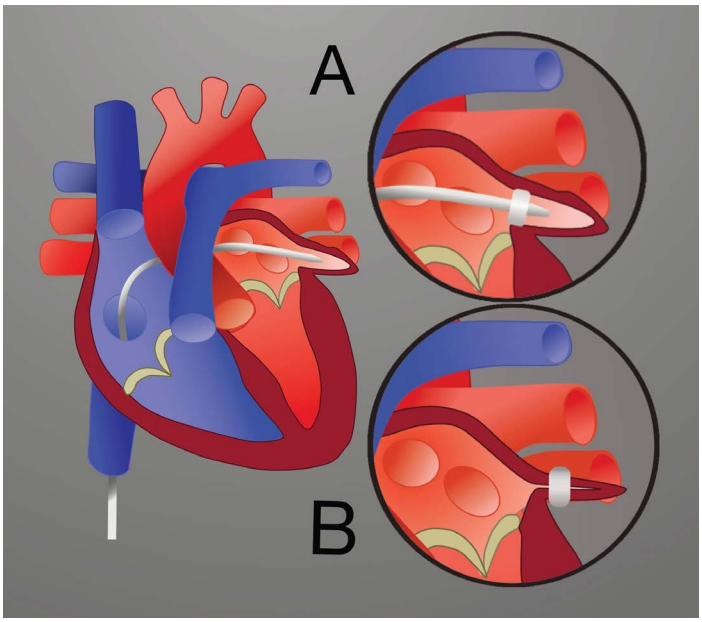

Figure 2: *LAAO&LAA Clip Illustration. A. Left atrial appendage occlusion devices are an option for patients that are poor candidates for anticoagulation. Using a percutaneous access sheath, the device is deployed into the LAA. The device compresses or hooks into the base of the LAA, occluding the LAA. The device usually endothelializes within 45 to 60 days. B. The LAA implantable clip is applied epicardially, isolating the LAA from left atrial body structurally and electrically.*

Panel 1. Definitions of Atrial Fibrillation[5]

Paroxysmal AF
AF that terminates spontaneously or with intervention within 7 days of onset
Episodes may recur with variable frequency
Persistent AF
Continuous AF that is sustained >7 days
Long-standing persistent
Continuous AF >12 months in duration
Permanent AF
The term "permanent" is used when the patient and provider make a joint decision to no longer pursue rhythm control. AF is treated therapeutically
Acceptance of AF may change as symptoms, efficacy of therapeutic interventions, and patient and provider preferences evolve
Nonvalvular AF
AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or a mitral valve repair[6]

Antiarrhythmic medications (AAD)

Antiarrhythmic drug therapy is used to reduce the frequency and duration of atrial fibrillation.[5,24] While these medications are less invasive than procedural management for AF such as catheter ablations, they can be proarrhythmic, toxic, and ineffective over both short and long term periods. Before the initiation of an AAD, it is important to consider risks of cardiac and noncardiovascular side effects.[5]

Antiarrhythmic drugs are categorized according to the Vaughan-Williams classification system. The system classifies the medications into four classes according to the main mechanism of action.[25]

Vaughan-Williams Classification System

Class I: Voltage-gated Na+ Channel Blockers

- Class Ia: Causes moderate degree blockage of fast sodium channels. Drugs include quinidine, procainamide, and disopyramide. These are the most pro-arrhythmic of the sodium channel blockers due to prolonged QTc interval; use is limited due to this proarrhythmic potential.[25–27]
- Class Ib: Causes mild degree blockage of sodium channels. Drugs include lidocaine and mexiletine. These drugs shorten the QTc interval, are used for ventricular arrhythmias only, especially post-myocardial infarction (not effective in treating AF).[25,28]
- Class Ic: Causes marked degree of sodium blockage and no significant effect on QT interval. Drugs include flecainide or propafenone. These drugs are reasonable for ongoing management in patients without structural heart disease or ischemic heart disease who have symptomatic AF and prefer not to undergo catheter ablation.[5,25]

Class II: Autonomic Inhibitors/Activators

Beta-blockers (BB) are indicated for rate control in patients with paroxysmal, persistent, or permanent AF and atrial flutter. Oral beta-blockers are used for ongoing management in patients with symptomatic supraventricular tachycardia (SVT) and AF.[28,29]

Class III: K+ Channel Blockers/Openers

Potassium channel blockers decrease potassium efflux out of the cell and prolong the QTc interval (amiodarone, dofetilide, sotalol, ibutilide, dronedarone).

- Amiodarone exerts sympatholytic, sodium, and calcium antagonistic properties that decrease AV and sinus node conduction. This drug is recommended in patients with AF to maintain sinus rhythm, especially in patients with left ventricular systolic dysfunction. It is also a reasonable

Panel 2. Treatment Options for AF: Rate Control Medications

Medications
Rate control recommendations have remained the first-line strategy for patients with a diagnosis of AF.[5] Controlling heart rate improves symptoms, reduces mortality, and reduces the risk of tachycardia-mediated cardiomyopathy.[5] According to the 2014 AHA/ACC/HRS Guidelines for the Management of Patients with Atrial Fibrillation, the first line medications for rate control are beta blockers or nondihydropyridine calcium channel antagonists. The class I, IIa, and IIb recommendations are as follows:[5]
Class I
1. Control of the ventricular rate using a beta blocker or nondihydropyridine calcium channel antagonist is recommended for patients with paroxysmal, persistent, or permanent AF. 2. Intravenous administration of a beta blocker or nondihydropyridine calcium channel blocker is recommended to slow the ventricular heart rate in the acute setting in patients without pre-excitation. In hemodynamically unstable patients, electrical cardioversion is indicated. 3. In patients who experience AF-related symptoms during activity, the adequacy of heart rate control should be assessed during exertion, adjusting pharmacological treatment as necessary to keep the ventricular rate within the physiological range.
Class IIa
1. A heart rate control (resting heart rate <80 bpm) strategy is reasonable for symptomatic management of AF. 2. Intravenous amiodarone can be useful for rate control in critically ill patients without pre-excitation. 3. AV nodal ablation with permanent ventricular pacing is reasonable to control heart rate when pharmacological therapy is inadequate and rhythm control is not achievable.
Class IIb
1. lenient rate-control strategy (resting heart rate <110 bpm) may be reasonable as long as patients remain asymptomatic and LV systolic function is preserved. 2. Oral amiodarone may be useful for ventricular rate control when other measures are unsuccessful or contraindicated.[5]

option in pharmacological cardioversion.[5,28] Amiodarone is typically well tolerated, but its lipid affinity can lead to adverse effects. Amiodarone can concentrate in tissues, leading to toxicity. Surveillance for liver, lung, ocular and thyroid toxicity is required.[29]

- Dronedarone is designed to resemble amiodarone, but with fewer noncardiovascular side effects due to the absence of the iodine moiety and the presence of methylsulfonamide group, which reduces fat solubility.[24] Dronedarone should not be used in patients with AF that cannot be converted to normal sinus rhythm (permanent AF). According to an FDA review, it doubles the rate of cardiovascular death, stroke, and heart failure in such patients.[5,28]
- Dofetilide is used for atrial arrhythmias only. Oral dofetilide is useful for acute pharmacological cardioversion in atrial fibrillation or atrial flutter patients.[5,28] It is renally cleared and dosed according to creatinine clearance. As with other potassium channel blockers, the risk of torsades de pointe increases with higher doses.[24] It requires a 3-day hospital admission for loading. It is also an option for heart failure patients.[30]
- Sotalol shares the effects of class II and class II, non-cardioselective beta-blocker, and potassium channel blockers. Therefore, clinicians can use it to treat both ventricular and supraventricular arrhythmias. It is not effective for converting AF to sinus rhythm but may be used to prevent recurrent AF.[5,28]

Class IV: Ca2+ handling modulators (diltiazem, verapamil)

Non-dihydropyridine calcium channel blockers decrease conduction velocity and slow conduction through the AV node. They are useful for ventricular rate control in acute and chronic AF and atrial flutter. Diltiazem and verapamil are options in the acute treatment of hemodynamically stable patients with SVT, AF, focal, and multifocal atrial tachycardias.[5]

Drug selection is guided primarily by safety concerns related to absolute or relative contraindications such as renal function, QT prolongation, hepatic dysfunction, structural heart disease, coronary disease, or left ventricular hypertrophy **(Figure 3)**.[5] Some AADs can impact bradyarrhythmias or AV conduction.[5] Patients with coronary disease, left ventricular hypertrophy, and heart failure have fewer options for AADs than a patient without structural heart disease.[5] The table below is a reference for dosage and safety considerations for the maintenance of sinus rhythm with AADs for patients in AF **(Table 2)**.

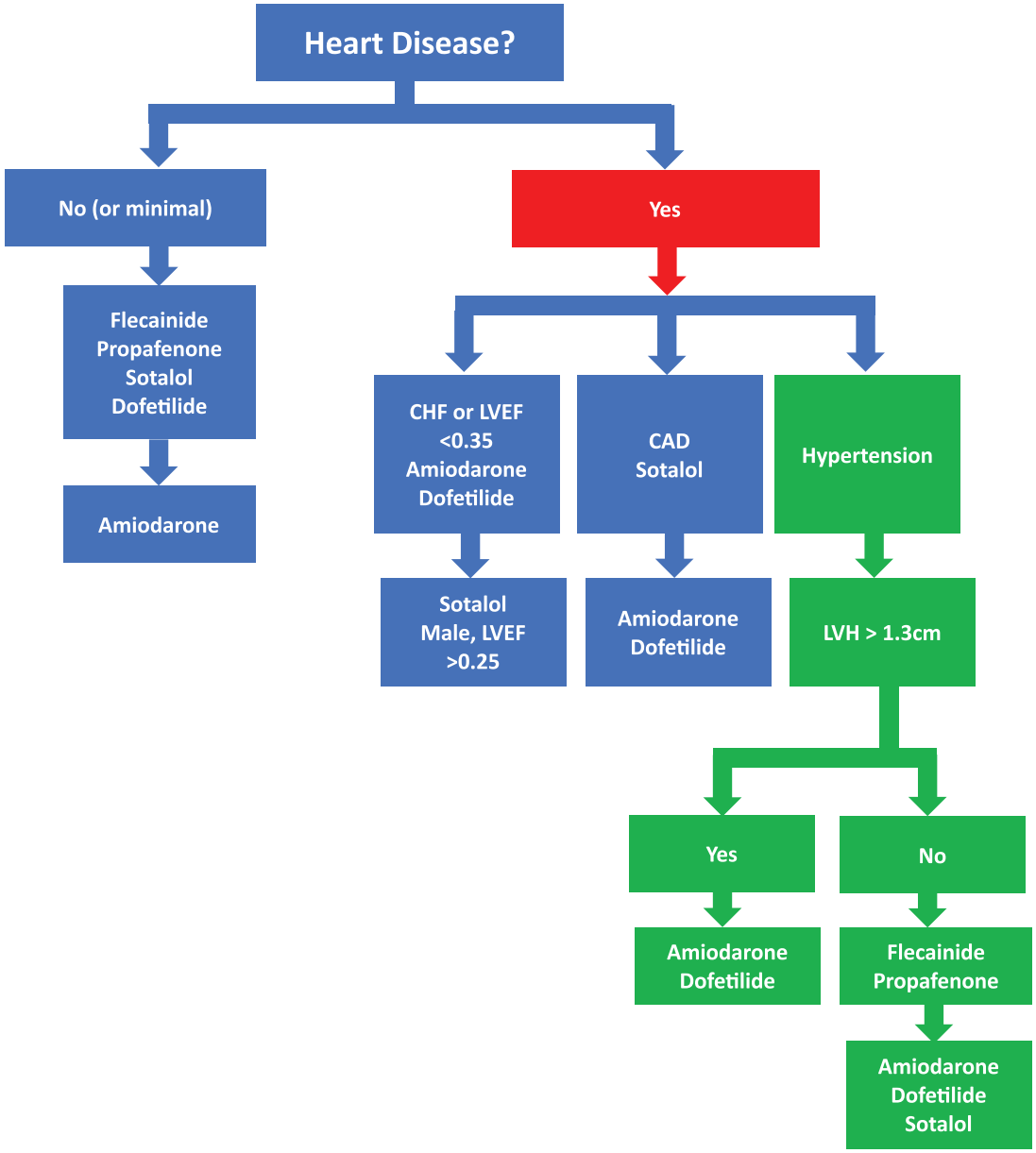

FIGURE 3: *AAD Drug Selection Algorithm.*

CARDIOVERSION

Cardioversions, whether they be electrical or pharmacological, are widely used for patients with AF and are often the first line of treatment in rhythm control management.[31] A pharmacological cardioversion is most effective when an AAD is administered within 7 days of AF onset, before the patient is considered to be in a persistent AF episode.[5] Electrical cardioversion by direct current is recommended to restore sinus rhythm, especially when the patient has a rapid ventricular response to AF, does not respond to pharmacological therapies, or is hemodynamically unstable.[5]

A pharmacological cardioversion converts new onset or paroxysmal AF to normal sinus rhythm in 50–70% of cases within a few hours, while electrical cardioversion converts 90% of patients to sinus rhythm.[31] Occasionally, a patient may only maintain sinus rhythm for a short period of time after undergoing cardioversion, prompting the need for other treatments to be considered.

As with all AF patients, the need for anticoagulation should be assessed prior to cardioversion. Cardioversions are generally considered safe, but the risk of thromboembolic events are increased if a plan for anticoagulation is not in

Table 2. Dosage and Safety Considerations for Maintenance of Sinus Rhythm in AF[7]

Drug	Usual Doses	Exclude/Use with Caution	Major Pharmacokinetic Drug Interaction
Disopyramide	• 100–200 mg once every 6 h • Extended release: 200–400 mg once every 12 h	• HF • Prolonged QT interval • Prostatism, glaucoma • Avoid other QT interval-prolonging drugs	• Metabolized by *CYP3A4*: caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., rifampin, phenobarbital, phenytoin)
Quinidine	• 324–648 mg every 8 h	• 324–648 mg every 8 h	• Inhibits *CYP2D6*: • ↑ concentrations of tricyclic antidepressants, metoprolol, antipsychotics; ↓ efficacy of codeine • Inhibits P-glycoprotein: ↑ digoxin concentration
Vaughan Williams Class IC			
Flecainide	• 50–200 mg once every 12 h	• Sinus or AV node dysfunction • HF • CAD • Atrial flutter • Infranodal conduction disease • Brugada syndrome • Renal or liver disease	• Metabolized by CYP2D6 (inhibitors include quinidine, fluoxetine, tricyclics; also genetically absent in 7%–10% of population) and renal excretion (dual impairment can ↑↑ plasma concentration)
Propafenone	• Immediate release: 150–300 mg once every 8 h • Extended release: 225–425 mg once every 12 h	• Sinus or AV node dysfunction • HF • CAD • Atrial flutter • Infranodal conduction disease • Brugada syndrome • Liver disease • Asthma	• Metabolized by CYP2D6 (inhibitors include quinidine, fluoxetine, tricyclics; also genetically absent in 7%–10% of population)—poor metabolizers have ↑ beta blockade • Inhibits P-glycoprotein: ↑ digoxin concentration • Inhibits CYP2C9: ↑ warfarin concentration (↑ INR 25%)
Vaughan Williams Class III			
Amiodarone	• Oral: 400–600 mg daily in divided doses for 2–4 wk; maintenance typically 100–200 mg QD • IV: 150 mg over 10 min; then 1 mg/min for 6 h; then 0.5 mg/min for 18 h or change to oral dosing after 24 h, consider decreasing dose to 0.25 mg/min	• Sinus or AV node dysfunction • Infranodal conduction disease • Lung disease • Prolonged QT interval	• Inhibits most CYPs to cause drug interaction: ↑ concentrations of warfarin (↑ INR 0%–200%), statins, many other drugs • Inhbits P-glycoprotein: ↑ digoxin concentration

...Continued

Table 2. Dosage and Safety Considerations for Maintenance of Sinus Rhythm in AF[7] (*cont.*)

Drug	Usual Doses	Exclude/Use with Caution	Major Pharmacokinetic Drug Interaction
Dofetilide	• 125–500 mcg once every 12 h	• Prolonged QT interval • Renal disease • Hypokalemia • Diuretic therapy • Avoid other QT interval prolonging drugs	• Metabolized by *CYP3A*: verapamil, HCTZ, cimetidine, ketoconazole, trimethoprim, prochlorperazine, and megestrol are contraindicated; discontinue amiodarone at least 3 mo before initiation
Dronedarone	• 400 mg once every 12 h	• Bradycardia • HF • Long-standing persistent AF/flutter • Liver disease • Prolonged QT interval	• Metabolized by *CYP3A*: caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., rifampin, phenobarbital, phenytoin) • Inhibits *CYP3A*, *CYP2D6*, P-glycoprotein: ↑ concentrations of some statins, sirolimus, tacrolimus, beta blockers, digoxin
Sotalol	• 40–160 mg once every 12 h	• Prolonged QT interval • Renal disease • Hypokalemia • Diuretic therapy • Avoid other QT interval prolonging drugs • Sinus or AV nodal dysfunction • HF • Asthma	• None (renal excretion)

AF indicates atrial fibrillation; AV, atrioventricular; CAD, coronary artery disease; HCTZ, hydrochlorothiazide; HF, Heart Failure; INR, international normalized ratio; IV, intravenous; and QD, once daily. January CT, Wann LS, Calkins H, et al. Circulation. 2019 Jul 9;140(2):e125-e151. Used with permission from the American Heart Administration.

place. According to the 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guidelines for the Management of Patients with Atrial Fibrillation, the class I recommendation for patients with AF or atrial flutter with a duration of 48 hours or more or when time of onset cannot be determined, anticoagulation is recommended for at least 3 weeks prior to and 4 weeks after cardioversion, regardless of the CHA2DS2-VASc score. This applies for electrical cardioversions, pharmacological cardioversions, and cardiac ablations.[7] For patients with episodes over 48 hours or of unknown duration that are hemodynamically unstable and require immediate cardioversion, anticoagulation should be initiated as soon as possible and continued for at least 4 weeks, unless contraindicated.[7]

CATHETER ABLATION

If sinus rhythm is not maintained with AADs or with an electrical or pharmacological cardioversion, a cardiac ablation should be considered and is sometimes preferred as a first line therapy for rhythm management. Cardiac ablations are performed by cardiac electrophysiologists and have a low incidence of procedural complications. Patients are generally discharged on the same day of the ablation.[32] Anticoagulation guidelines remain unchanged for cardiac ablations.

Catheter ablations have historically been reserved for paroxysmal or persistent AF patients that are considered healthier and have failed at least one AAD.[7] There is increasing evidence suggesting that early rhythm control intervention for newly diagnosed AF patients by use of catheter ablation reduces

recurrent arrhythmias and rates of hospitalizations when compared to AADs.[22] Early intervention with rhythm management may delay the progression of AF and prevent irreversible atrial damage, thereby reducing symptoms, preventing heart failure, strokes, and AF-related deaths.[20] The choice for catheter ablation is dependent on many factors such as the type of AF, patient symptoms, structural heart disease, and patient preference.[7] The 2014 AHA/ACC/HRS class I recommendation for ablation states that ablation is useful for symptomatic paroxysmal AF that is refractory or intolerant to at least one class I or class III AAD.[5]

Catheter ablation is an important treatment option for AF patients, especially for those that have paroxysmal or early persistent AF. Patients have a risk for adverse atrial remodeling the longer they are in AF, which is thought to be a proarrhythmic factor for arrhythmias.[33] Risk factors such as heart failure, hypertension, obstructive sleep apnea, and valvular disease can contribute to increased atrial filling pressures and lead to adverse atrial remodeling and stretch.[33] A reduction in atrial stretch is considered antiarrhythmic, which is why it is important to consider risk factor modifications for all AF patients to reduce developing adverse atrial remodeling and dilatation.[33]

An important patient population to consider for rhythm control are those with a diagnosis of heart failure. The CASTLE-AF(Catheter Ablation compared with Pharmacological Therapy for Atrial Fibrillation) trial studied the effectiveness of catheter ablation versus medical treatment and randomized patients with heart failure that were not responsive to AADs to rate control therapy or catheter ablation.[34] The patients that underwent catheter ablation had a significantly reduced overall mortality rate and reduced hospitalization rate for worsening heart failure.[34] The 2019 AHA/ACC/HRS focused update on AF now has a class IIb

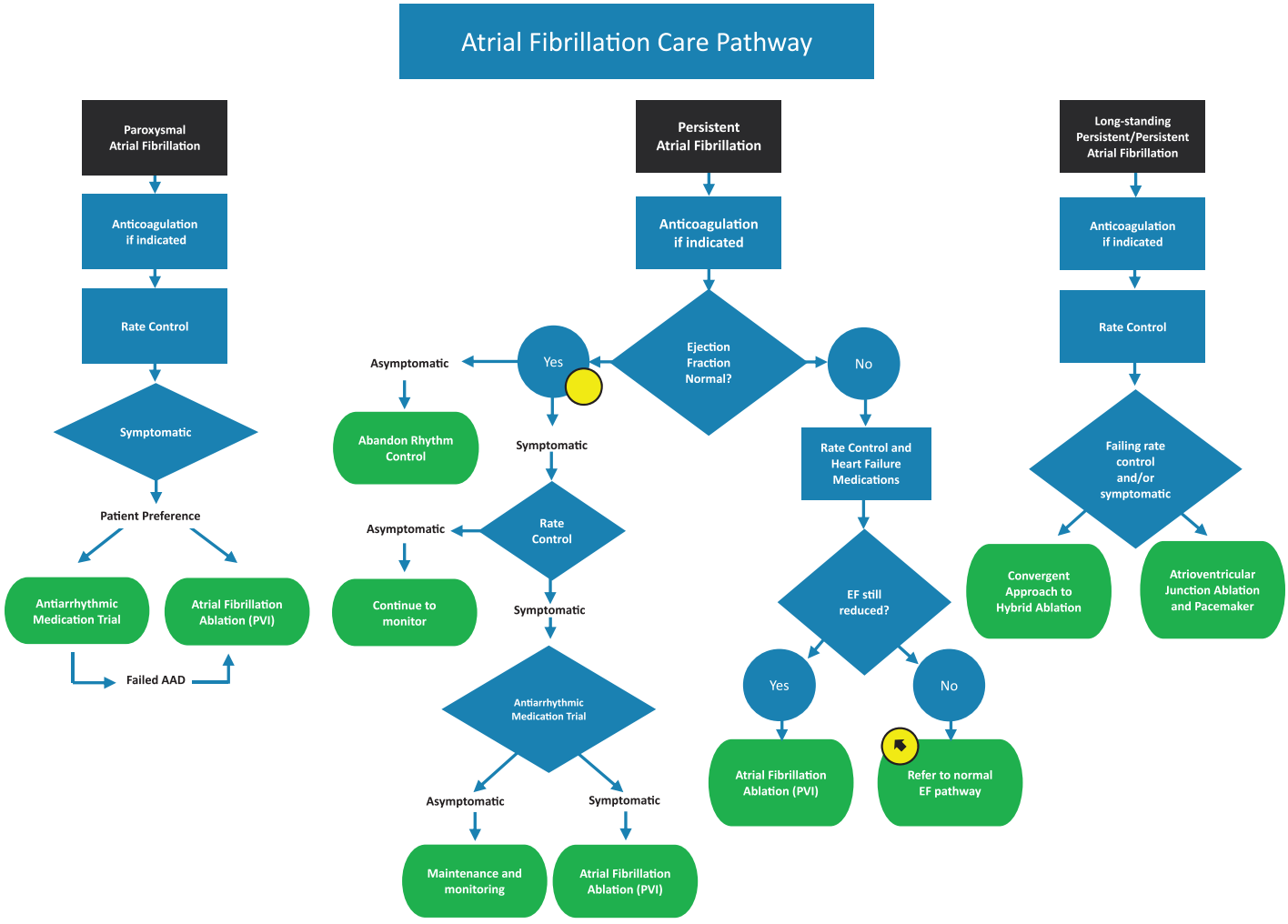

Figure 4: *Atrial Fibrillation Care Pathway. Infographic by Christine Van de Walker Handy, RN, CCDS. 2023. Virginia Mason Franciscan Health.*

recommendation in which catheter ablation may be reasonable for patients in AF and heart failure with a reduced ejection fraction.[7]

Surgical ablation

Surgical interventions for atrial fibrillation patients undergoing concomitant open heart cardiac surgery does not pose additional risk of operative mortality or major morbidity.[14] The Cox-Maze IV procedure for AF involves the creation of scar in the right and left atrium that blocks AF conduction and perpetuation. The Cox-Maze IV surgery has been considered the "gold standard" for surgical AF management for patients with a history of AF undergoing open heart surgery for concomitant open or closed cardiac surgeries, such as mitral valve, coronary artery bypass grafting, and aortic valve surgeries.[35] The 2017 HRS/EHRA/ECAS/SOLAECE Expert Consensus Statement on Catheter and Surgical Ablation of AF recommends surgical ablation for paroxysmal, persistent, and long-standing persistent AF patients in these settings.[35]

There has been increasing interest in stand-alone Cox-Maze IV intervention for long-standing persistent AF, especially for patients that have recurrent symptomatic AF episodes after catheter ablation and/or are intolerant or refractory to AADs. Stand-alone Cox-Maze IV surgery can be considered for these paroxysmal, persistent, or long- standing persistent AF patients.[35] The Cox-Maze procedures have seen high success rates with the maintenance of sinus rhythm in 80–90% of patients off AADs.[36] However, these patients may be candidates for a less invasive hybrid surgical AF ablation procedure.

Hybrid ablation therapy is a minimally invasive cardiac ablation procedure for AF. It is performed in two stages by a cardiac surgeon and a cardiac electrophysiologist, respectively. Hybrid ablation aligns itself with traditional catheter ablation rather than a complex surgical procedure. It is a two-part procedure that creates lesions endocardially and epicardially to form a more robust transmural lesion set in the left atrium, thereby isolating AF arrhythmic triggers.[36] In one study up to 87% of patients remained free from AF at 20 months.[36]

Catheter and surgical technologies to treat AF continue to expand, and decisions about the correct care pathway for the AF patient can appear complex. Below is a generalized care pathway for the AF patient based on the current guidelines **(Figure 4)**.

CONCLUSION

Treatment of AF continues to evolve and be refined over time. Although AF management can seem algorithmic, it must be tailored to each individual patient. Assessing stroke risk, rate and/or rhythm control strategy, and mitigating risk factors can lead to a reduction in AF occurrence and improved management of AF as a whole. Strategies to maintain long-term sinus rhythm continue to develop and early catheter ablation has become a cornerstone therapy. AF should be viewed as a progressive atrial myopathic disease rather than an arrhythmia and early identification and treatment offers the best chance of mitigating the negative consequences.

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