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Update on Current Management of Atrial Fibrillation

Introduction

Atrial fibrillation (AF) is an irregular, disorganized, electrical activity of the atria.[1] It is characterized by uncoordinated atrial activity that leads to inefficient atrial contraction and impaired ventricular filling, which leads to hemodynamic and mechanical functional impairments. The electrocardiographic (ECG) characteristics of AF include the absence of regular P wave activity that is replaced by rapid oscillations or fibrillatory waves that vary in size, shape, and timing. This results in an irregular ventricular response. The ventricular response in AF is dependent on a number of factors including the integrity of the atrioventricular (AV) node, autonomic function, as well as medications that affect the initiation and/or propagation of the action potential.

Classification

There are various systems of classification of AF. The one that is used most commonly employs the temporal nature of AF and the therapeutic response to treatment strategies. When AF is first detected, it is categorized as new-onset AF. When a patient experiences two or more episodes of AF, it is characterized as recurrent AF. Recurrent AF is further divided into three sub-categories: paroxysmal AF (PAF), persistent AF, and permanent AF. PAF is characterized by episodes of AF that self-terminate.[2] Most patients with PAF will terminate spontaneously within 7 days without treatment. Persistent AF is one that is sustained for more than 7 days. Consideration for pharmacologic or direct-current cardioversion is considered in symptomatic patients but does not change the designation. AF that is completely refractory to cardioversion or one where the option for cardioversion has been abandoned is termed as permanent AF. In permanent AF, the goal is rate control and anticoagulation to prevent thromboembolic events.

These classifications are not mutually exclusive; for instance, the arrhythmia may change from paroxysmal pattern to persistent or permanent (*see Table 1*).

Lone AF is characterized by an absence of recognizable structural heart disease or inciting events and usually occurs in individuals less than 60 years of age.[3]

Secondary AF is characterized by situations in which AF is attributable to a primary problem such as acute myocardial infarction, surgery, hyperthyroidism, or acute pulmonary disorder.

Nonvalvular AF is restricted to cases in which there is an absence of associated mitral valve disease, a prosthetic heart valve, or mitral valve repair.

Epidemiology

AF is the most common sustained cardiac arrhythmia. It is estimated that about 2.3 million people in the United States have the disease condition.[4] The

Executive Summary

Atrial fibrillation is the most common sustained arrhythmia, affects 2.3 million people in the United States and is a frequent management issue for primary care physicians.

- The most powerful predictors of atrial fibrillation include dilated cardiomyopathy, valvular heart disease, hypertension, history of myocardial infarction and advanced age.
- The three most important strategic objectives in management involve rate control, rhythm control and prevention of thromboembolism.
- Choices for oral anticoagulant therapy now include recently added direct thrombin inhibitors such as dabigatran, which does not require serial monitoring as required for warfarin.
- Non-pharmacologic options for therapy include surgical ablation, pacemaker therapy and implantable atrial debrillators.

increase in the prevalence of AF is partly a reflection of the increase in the elderly population as well as improved survival of patients with myocardial infarction and heart failure.[5] Hospital admissions with the principal diagnosis of AF have increased by about two-thirds during the last two decades[6] and account for one-third of hospitalizations for cardiac rhythm disorders. Other factors contributing to the rise in prevalence of AF include the corresponding increase in the prevalence of chronic heart disease as well as the widespread use and availability of non-invasive monitoring devices to diagnose AF. Although the prevalence of AF in the general population is estimated at 0.4% to 1%,[5] it increases to about 8% in those older than 80 years.[7] The median age of individuals with AF is 75 years of age.[8] The age-adjusted prevalence of AF among men has increased by 50% over the last two decades.[7] The incidence of AF across the various age groups is higher in men[9]; however, the absolute number of women with AF is higher in the age group above 75 years of age, because women comprise the majority of the group in this age demographic.[8] AF is a costly public health concern. Globally, the annual cost per patient is close to $3600.[10]

Etiology

Several etiological factors have been implicated in the genesis of AF. The underlying pathological process that is mostly noted in patients with AF is atrial fibrosis and loss of atrial muscle; these are normal age-related changes that may explain the increased prevalence of AF in the elderly.[11,12] Other inciting conditions such as valvular heart disease,

Table 1: Classification of Atrial Fibrillation

Type of Atrial Fibrillation	Comments
Lone AF	No underlying structural heart disease or inciting event. Patients often are younger than 60 years and do not need anticoagulation.
Newly Diagnosed Atrial Fibrillation	First recognition of AF.
Paroxysmal Atrial Fibrillation	AF that spontaneously reverses to sinus rhythm, usually within a week.
Persistent Atrial Fibrillation	AF lasting longer than 7 days.
Permanent Atrial Fibrillation	AF that is refractory to cardioversion. The goal of treatment is rate control and consideration for anticoagulation.
Secondary Atrial Fibrillation	AF attributable to a specific cause.
Nonvalvular Atrial Fibrillation	AF without associated valvular heart disease.

hypertrophic cardiomyopathy,[13] ischemia, and hypertension may accelerate these histologic changes beyond that expected due to normal aging. Nonhomogeneity of conduction created by the juxtaposition of normal atrial fibers next to patchy areas of fibrosis serves as a nidus for the initiation and maintenance of AF.[14,15] The extent of fibrosis predicts a poor prognosis for the therapeutic efficacy of various treatment interventions and for the maintenance of sinus rhythm in patients with AF.[16] Atrial fibrillation in turn further remodels atrial tissue structurally by accelerating atrial fibrosis and electrophysiologically through the progressive shortening of effective refractory periods further perpetuating the arrhythmia.[17,18] Other factors potentially involved in the induction or maintenance of AF include inflammation, autonomic nervous system activity, atrial ischemia, atrial dilation, anisotropic conduction,[19] and structural changes associated with aging.[20-23]

A particularly dangerous setting for AF occurs in conduction across an accessory pathway, which exists in Wolf-Parkinson-White syndrome (WPW).[24] In WPW, a dangerously rapid ventricular rate can degenerate into ventricular fibrillation and lead to death.[25,26] AF commonly complicates cardiac surgery where the incidence after coronary artery bypass approaches 15% in patients younger than 65 years and 30% in patients older than 65 years of age.[27] The incidence of AF in patients after valve replacement surgery is about 60%.[27] The most powerful predictors of AF include dilated cardiomyopathy, valvular heart disease, hypertension, history of myocardial infarction, and advanced age.[28] The presence of left atrial enlargement and ventricular hypertrophy are associated with an increased incidence of AF.

The electrophysiologic basis for AF is somewhat complex and involves two prevailing mechanisms that are supported by currently available experimental data. These mechanisms may coexist in the same patient and are not mutually exclusive.

AF can originate from localized areas of atrial myocardium that have electrical properties which generate rapid spontaneous impulses and serve as a substrate for reentry and sustained AF. These tissues most commonly have been found in the distal pulmonary veins as they enter the left atrium, but they also have been identified in superior vena cava, ligament of Marshall, left posterior free wall, crista terminalis, and coronary sinus.[29-31] Utilizing catheter-based electroanatomic mapping and ablation of these foci allows AF to be extinguished successfully in some patients. Alternatively, AF may be dependent on a larger mass of tissue distributed throughout the atria that possesses non-uniform refractoriness and conduction. Such non-uniformity favors the development of multiple simultaneous daughter wavelets of depolarization as electrical activity propagates across the atria and provides a milieu for the maintenance of AF.

Reversible Causes of AF

AF can be caused by reversible conditions such as alcohol intake (holiday heart syndrome), surgery, electrocution, acute myocardial infarction, pericarditis, myocarditis, pulmonary embolism or other pulmonary disorders, hyperthyroidism, as well as other metabolic disorders. (*See Table 2.*) Treatment of the underlying conditions will result in correction of the disorder and maintenance of normal sinus rhythm. AF in the setting of acute myocardial infarction is associated with poor prognosis.[27] AF also may be associated with other arrhythmias such as atrial flutter, the WPW syndrome, or AV nodal re-entrant tachycardias. Treatment of the primary arrhythmias reduces or eliminates the incidence of recurrent AF.[32]

Symptoms and Manifestation of AF

Patients with AF have variable modes of presentation and may occur in the presence or absence of detectable heart disease. AF may be self-limited with minimal symptoms or may require urgent medical intervention due to symptomatic hemodynamic compromise manifesting as severe palpitations, chest pain, heart failure, dyspnea on exertion or at rest, and rarely with syncope. Some patients may be completely asymptomatic until they ominously present with a thromboembolic complication.

A patient's pattern of AF can be characterized by its number of episodes, duration, frequency, mode of onset, triggers, and response to therapy, but these features may be impossible to discern when AF is first encountered in an individual patient.

For most patients presenting with symptoms, the symptoms are non-specific.[33] Patients with paroxysmal AF have significant functional impairment comparable to those with severe cardiac conditions such as angioplasty.[34]

Patients who are treated with rate control medications, rhythm control, or even ablation of the AV node and implantation of the pacemaker do have improvement in the quality of life.[35-37]

Approach to a Patient with AF

Diagnosis of AF requires ECG confirmation, which is determined by irregular ventricular activity with the presence of fibrillatory wave pattern of atrial activity. Once the AF diagnosis has been made, the pertinent issues with regard to the work involve determining the cause of the arrhythmia; defining the associated cardiac and extra cardiac factors related to the etiology; and assessing the hemodynamic consequences of the arrhythmia, tolerability, and implications of therapeutic interventions on the individual. A careful history and physical examination is important. It is important to look for new onset angina or congestive heart failure as this may necessitate the need for early cardioversion. A careful documentation of the nature and type of symptoms associated with AF is important. Determination of the frequency, duration and precipitating factors, and mode of termination

Table 2: Causes of Atrial Fibrillation

Electrophysiological
- Enhanced automaticity (focal AF)
- Conduction abnormality (re-entry)

Atrial pressure elevation
- Mitral or tricuspid valve
- Myocardial disease
- Semilunar valvular abnormalities
- Systemic or pulmonary hypertension (especially with associated ventricular hypertrophy)
- Intracardiac tumors or thrombi

Atrial ischemia

Inflammatory or infiltrative atrial disease
- Pericarditis
- Amyloidosis
- Myocarditis
- Age-related fibrosis

Toxins
- Alcohol
- Caffeine

Endocrine Abnormalities
- Hyperthyroidism
- Pheochromocytoma

Autonomic tone changes
- Increased parasympathetic tone
- Increased sympathetic tone

Surgery
- Thoracic
- Pulmonary
- Esophageal

Primary or metastatic disease in or adjacent to the atrial wall

Congenital heart disease
- Atrial septal defect
- Epstein's anomaly

CNS
- Subarachnoid hemorrhage
- Nonhemorrhagic, major stroke

Idiopathic (lone AF)

Familial AF

of the arrhythmia is also important. Patients should be assessed for underlying heart disease and other reversible causes of the arrhythmia such as the use of alcohol, presence of thyroid diseases, or other metabolic disorders. Echocardiographic evaluation is important to determine structural abnormalities as well as functional assessment of the heart. Electrolyte and metabolic evaluation of reversible causes of AF is important. Three other key considerations in the assessment and management of the arrhythmia are as follows:

1. Physicians should assess the need for restoration to sinus rhythm.
2. Anticoagulation to prevent embolic stroke.
3. Appropriate control of ventricular rate while the patient is experiencing atrial fibrillation.

Investigations

Diagnosis of AF requires ECG documentation by at least a single lead recording during the arrhythmia. In patients with implanted pacemakers or defibrillators, the diagnosis of AF may be made from detection of the arrhythmia from retrievable memory functions.[38]

In evaluating the ECG in patients with AF, attention must be paid to assess the left ventricular hypertrophy, P wave duration and morphology and the presence of fibrillatory waves, presence of bundle branch blocks, or pre-excitation. Attention must be paid to evaluate for evidence of ischemic heart disease. It is also important to measure electrocardiographic intervals such as the R-R, QRS, and QTs for the purposes of determining and monitoring the therapeutic intervention. A chest x-ray is important to determine the presence of intrinsic pulmonary and vascular abnormalities.

A transthoracic echocardiogram is important to identify valvular heart disease, determination of chamber dimensions, and presence of pericardial disease, left ventricular hypertrophy, and left atrial thrombus.

Additional Testing

1. Six-minute walk test if there is question about the adequacy of rate control.
2. Exercise testing: a) if there is a question regarding the adequacy of rate control; b) to reproduce exercise-induced AF; and c) to exclude ischemia before treatment of selected patients with a type 1C antiarrhythmic drugs.
3. Holter monitoring or event recording. If the diagnosis of the type of an arrhythmia is

in question and as means of evaluating rate control.

4. QRS complex tachycardia. To identify the predisposing arrhythmia such as atrial flutter or paroxysmal supraventricular tachycardia. It is also used to map out the sites for curative ablation.

Approach to Patients with Newly Diagnosed AF

In patients with minimal or no symptoms, it is often difficult to determine whether the initial episode of AF is really the first episode or whether it's part of the spectrum of recurrent AF. Patients who are hemodynamically stable with self-limited episodes of AF may not need an anti-arrhythmic therapy. Newly diagnosed AF patients who have hemodynamic instability, such as angina, pulmonary edema, or with concomitant pre-excitation, will benefit from urgent cardioversion. Patients with AF and concomitant pre-excitation have unpredictable response to antiarrhythmic therapy.[39] In AF patients who are hemodynamically stable, rate control with intravenous diltiazem, beta-blocker, or digoxin may be indicated if there is spontaneous conversion. Then the patient may be followed up for recurrence and assessment of the etiology. If the AF onset or duration is deemed to be less than 48 hours in the setting where the patient is free of significant left ventricular dysfunction, mitral-valve disease, or previous embolism, then restoration to sinus rhythm may be attempted with either pharmacological means (intravenous ibutilide, oral propafenone, flecainide or oral quinidine) vs direct current shock.

Spontaneous conversion to sinus rhythm within 24 hours after the onset of AF is common, occurring in up to two-thirds of patients.[3] If the AF has been present for 7 days or more, the chance for spontaneous conversion is rare.[40,41]

For patients with duration of symptoms longer than 48 hours, the options include assessing for presence of left atrial thrombus with a transesophageal echo, and if there is no evidence of a thrombus on imaging then proceeding with cardioversion is important. Another acceptable option would be to fully anticoagulant for 4 weeks before attempting cardioversion. Patients who have been successfully cardioverted to sinus rhythm need to be on oral anticoagulation for 6-12 weeks to prevent thromboembolic events. For those patients with failed cardioversion or early recurrence, rate control with long-term anticoagulation is one reasonable strategy with the option for repeat cardioversion.

Management

There are three strategic objectives in the management of AF.

These objectives involve rate control, rhythm control, and prevention of thromboembolism. These three objectives can be achieved by pharmacological as well as non-pharmacological means.

Heart Rate Control vs Rhythm Control

Patients with symptomatic AF that has been present for some time must be given medication for rate control and anticoagulation to prevent embolic events with the long-term goal of conversion to sinus rhythm. If rate control does not offer symptomatic relief, then restoration of sinus rhythm becomes a therapeutic goal.

Randomized trials comparing outcomes of rhythm vs rate-control treatment strategies in patients with AF have found that there is no difference in mortality or stroke rate between patients with rate-controlled strategies vs rhythm control. The AFIRM trial (Atrial Fibrillation Follow up Investigation of Rhythm Management) found rate control to be non-inferior to rhythm control for the prevention of death and morbidity.[42] There are conflicting data regarding the quality of life on the two treatment strategies.

Rate control may be a reasonable treatment strategy in elderly patients with persistent AF. In younger patients, particularly those with PAF, rhythm control may be a better alternative. What constitutes adequate rate control is not clearly defined; however, heart rate between 60-80 beats per minute (bpm) at rest while heart rate between 90-115 bpm during moderate exercise is considered an acceptable goal. A more recent study, RACE II (Rate Control Efficacy in Permanent Atrial Fibrillation), evaluated clinical outcomes between more lenient controls of heart rate in patients with AF vs strict heart rate control.[43] Strict heart rate control was defined as resting heart rate of < 80 bpm and a heart rate of < 110 bpm with moderate exercise. Lenient control was resting heart rate of < 110 bpm. In this study at 3 years, the primary endpoints were death from cardiovascular causes, hospitalization for heart failure, stroke, systemic embolization, bleeding, and life-threatening arrhythmias. At 3 years, the cumulative incidence of primary outcomes was statistically significant with 12.9% in the lenient group vs 14.9% in the strict heart rate control group. Based on these findings, the most recent updated management guidelines by the American College of Cardiology advocate a more lenient heart rate control with a caution that patients with normal left ventricular function in whom a lenient heart control strategy is adopted should have LV function monitored for deterioration.

Patients with tachycardia that is allowed to run unabated for sometime may have deterioration in left ventricular function. The cardiomyopathy that ensues following a sustained period of tachycardia is referred to as tachycardia-induced cardiomyopathy. This type of cardiomyopathy usually resolves within 6 months of achieving adequate control of heart rate.[44]

In patients with systolic dysfunction, defined as LV function less than 35%, there was no difference in clinical outcomes between patients treated for rhythm control vs those treated for rate control.[45]

Anticoagulation in AF

Thromboembolism is the most important complication of AF.[46] Non-rheumatic AF accounts for about one-third of all strokes per year in patients older than 65 years.[47] Even in patients with AF who do not have clinical evidence of stroke, about 6-25% of patients will have evidence of intra-atrial thrombus on TEE.[48] The risk of stroke in patients without heart disease and younger than 60 years is 0.5% per year.[3] The risk of stroke increases with age and is about 4.2% for those aged between 70-79 years and 5.1 between 80-89 years.[49] Known risk factors that increase the risk for stroke include advanced age, female gender, hypertension, left ventricular dysfunction, diabetes mellitus, and previous history of stroke. In the Framingham Heart Study, there was a 3.4-fold increase of stroke in patients with hypertension while there was a 2.4-fold increase stroke in patients with coronary artery disease. Heart failure can cause up to a 4.3-fold increase in stroke.[50] In patients with non-valvular AF, prior stroke or TIA is the strongest independent risk factor for stroke. There is no difference in the stroke rates between patients with PAF and those with permanent AF. In the stroke prevention in Atrial Fibrillation III trial, the annual rate of ischemic stroke in those with PAF was 3.2% while those with permanent AF was 3.3%.[51]

Echocardiography and Embolic Risk Stratification

Echocardiography is a valuable tool for risk stratification of AF patients who are at an increased risk for developing thromboembolic events. Patients with impaired LV dysfunction, enlarged left atrial dimensions, and clot in the left atrial appendage are at increased risk. Other features on echocardiography that are indicative for high risk for embolic potential include spontaneous echo contrast indicating low velocity flow in the atrium and complex atheromatous plaque in the thoracic aorta. There are various clinical criteria that have been used to identify patients at risk for developing thromboembolic events. One of the most clinically used clinical criteria for categorizing the risk of patients for thromboembolic disease is CHADS2 (Cardiac Failure, Hypertension, Age, Diabetes, Stroke [doubled]).[52] (*See Table 3.*) The scoring system attributes one point to age above 75 years, presence of hypertension, heart failure, and diabetes. Two points are ascribed to a past history of stroke or TIA. Patients with two or more points on the CHADS2 score are generally considered to be candidates for anticoagulation.

Table 3: Assessment of Stroke Risk in Non-valvular AF[52]

CHADS2 RISK SCORE	SCORE
Prior stroke or TIA	2
Age above 75 years	1
Hypertension	1
Diabetes mellitus	1
Heart failure	1

Oral Anticoagulant Therapy

Patients with AF with at least one risk factor should be considered for anticoagulation therapy to prevent thromboembolism. Aspirin therapy has modest protection against stroke in patients with AF.[53-55] Oral anticoagulation with warfarin is much more effective in the prevention of stroke in patients with AF than aspirin alone.[56] The goal of warfarin treatment is the INR range of 2.0 and 3.0.[57] At this level of INR, the balance between thromboembolic protection and adverse risk of bleeding is most favorable.

Recent studies looking at the role of combination therapy of antiplatelet agents have been published. A combination of aspirin and clopidogrel in AF did not show improvement in clinical outcomes when compared with warfarin.[58,59] However, a combination of aspirin and clopidogrel can be considered in patients who cannot tolerate other oral anticoagulants. This combination has a slight increase in the bleeding risk. A combination of aspirin, warfarin, and clopidogrel is acceptable combination therapy in patients with AF, prosthetic valves, and recently placed drug-eluting stents.[60] A new antithrombotic, dabigatran, recently was approved by the FDA for thromboembolic protection in patients with AF. This drug has the advantage of not requiring serial monitoring of the degree of anticoagulation like warfarin. Dabigatran at 150 mg twice-daily dosage was associated with lower rates of strokes and systemic embolization when compared to warfarin and was also associated with a comparable risk of bleeding.[61]

Antiarrhythmic Drugs for Maintenance of Sinus Rhythm

Therapy for the restoration of sinus rhythm can be considered in patients who have newly diagnosed AF if it can be adequately ascertained that the arrhythmia has been present for less than 48 hours or in those who have been adequately anticoagulated. Antiarrhythmic medications enhance the chances of successful restoration to sinus rhythm by about 90% if therapy is initiated early and in adequate doses.[62] Table 4 lists the most commonly used antiarrhythmic medications.

Before the initiation of antiarrhythmic drugs, reversible causes of AF should be identified and corrected. Selection of an appropriate

Table 4: Commonly Used Antiarrythmics for AF

CLASS
Type IA
• Disopyramide
• Procainamide
• Quinidine
Type IB
• Dronedarone
• Dofetilide
• Amiodarone
Type IC
• Flecainide
• Propafenone
Type II
• Beta-blockers
Type III
• Dronedarone
• Amiodarone
• Bretylium
• Dofetilide
• Ibutilide
• Sotalol
Type IV
• Non-dihydropyridine calcium channel antagonists

agent is based on patient safety. Patients should be assessed for the presence or absence of underlying heart disease.[63]

In patients with lone AF, dronedarone or a beta-blocker can be tried as initial therapy. Flecainide, propafenone, and sotalol are as effective. Amiodarone and dofetilide are reasonable alternatives. Patients with vagally mediated AF may benefit from disopyramide, which has anticholinergic properties. Flecainide and amiodarone may be alternatives. Propafenone has weak intrinsic beta-blocking properties that may worsen the condition. Patients taking antiarrhythmic medication must be aware of the drug–drug interactions and potential adverse effects. Patients should be monitored for QRS and QT prolongation.

Selection of Antiarrhythmic Drugs in Special Situations

Heart failure: Patients with heart failure are prone to ventricular arrhythmias. Amiodarone or dofetilide are acceptable options in these patients.[64,65]

Coronary artery disease: In patients with stable CAD, beta-blockers are the drug of choice. Sotalol is the drug of choice.

Hypertensive heart disease: Patients with left ventricular hypertrophy are prone to torsades. Dronedarone is an antiarrhythmic medication that has been recently approved by the FDA for maintenance of sinus rhythm. It is similar to amiodarone but lacks an iodine moiety and therefore lacks iodine-related toxicities associated with the use of amiodarone. Dronedarone was found to increase the time to recurrence of AF.[66] It also slows the ventricular rate by 11-13 bpm.[67] Dronedarone is less efficacious than amiodarone but has a better tolerability profile. It is contraindicated in patients with recent decompensated heart failure or left ventricular systolic dysfunction.[68] More recently there have been reports of dronedarone causing hepatic failure.[69] Serial monitoring of liver function tests are indicated in patients on this medication.

Non-pharmacological Therapy for AF

Non-pharmacologic alternatives for the treatment of AF have arisen due to the growing recognition of toxicities and limited efficacy of drug therapy.

Surgical ablation: Surgical ablation is based on the understanding that re-entry is the underlying mechanism contributing to the development and maintenance of AF.[70] This led to the development of the concept that barriers to conduction could be created by strategically placed incisions in the atrial wall that would prevent the propagation of AF. Thromboembolic episodes complicating AF are further reduced by the surgical resection or obliteration of the left atrial appendage. This surgical procedure is called the Maze procedure,[71] recognizing its similarity to a maze that thwarts a mouse's ability to escape. The procedure is performed in such a way that several incisions create dead ends for the re-entrant circuits. This procedure's limitation is that it requires cardiopulmonary bypass. The Maze procedure can be performed in AF patients requiring other cardiac procedures, particularly those involving the mitral valve. One of the potential complications of the procedure is that there may be atrial dysfunction with deterioration in the hemodynamic of the heart, and damage to the sinus node, requiring pacemaker implantation in 9% of the patients.[72] There also are newer techniques that use minimally invasive surgery and cryoablation as well as catheter-based epicardial techniques.[73]

Catheter ablation: Catheter-based radiofrequency ablation of the pulmonary vein is another approach to treatment of the AF that is incessant to medical management. Electrical isolation of the pulmonary veins is where most of the ectopic foci that lead to re-entry arise. There have been some modifications of the technique of isolating the pulmonary vein from isolating individual ectopic foci to circumferential electrical isolation of the entire pulmonary vein musculature that have resulted in higher cure rates.[74]

Catheter ablation is now recommended in patients with PAF who are symptomatic on at least one antiarrhythmic drug.[75]

Pacemaker Therapy

Atrial pacing is a novel technique for treating AF. This is based on the concept that inhomogeneous conduction of electrical conduction or intra-atrial conduction delays facilitate the development of AF. Atrial pacing prevents AF by bradycardia-induced dispersion of atrial repolarization and premature atrial contractions. This is more important in patients with sinus node dysfunction and normal AV conduction.[76]

Though atrial-based pacing is associated with a lower risk of AF and stroke than ventricular-based pacing in patients requiring pacemakers for bradyarrhythmias, its value as a primary therapy for prevention of recurrent AF has not been proven. A number of other techniques to terminate AF by pacing are also under investigation, but indications may be limited to atrial tachycardia and atrial flutter.

Implantable Atrial Defibrillators

There is still uncertainty about the role of implantable atrial defibrillators. These devices are reserved for patients with infrequent, poorly tolerated episodes of AF who also meet criteria for implantable ventricular defibrillators due to their LV dysfunction. Such patients typically have failed medical therapy and have demonstrated benefit from the maintenance of sinus rhythm. Patient discomfort from the electrical shocks is the main disadvantage of the use of these devices,[77] so shocks typically are delivered while the patient is asleep after taking a sedative. With the increasing use of catheter ablation strategies, the role for atrial defibrillators has further dwindled.

Summary

AF is the most common sustained arrhythmia seen in clinical practice. The prevalence of AF is increasing due to the aging of the population and the increased survival of patients with chronic heart disease. Hemodynamic impairment and thromboembolic events related to AF result in significant morbidity, mortality, and cost. The main goals of management of patients with AF are threefold: control of symptoms through the maintenance of sinus rhythm, prevention of tachycardia-mediated cardiomyopathy through adequate rate control, and protection from thromboembolic complications by appropriate anticoagulation in patients who are at increased risk. Early referral to a cardiologist for specialized therapies is an important element in the management of AF patients who are refractory to routine medical management.

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Primary Care Reports CME Objectives

Upon completion of this activity, participants should be able to:

1. Summarize recent, significant studies related to the practice of primary care medicine;
2. Evaluate the credibility of published data and recommendations related to primary care medicine;
3. Discuss the advantages and disadvantages of new diagnostic and therapeutic procedures in the primary care setting.

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To earn credit for this activity, please follow these instructions.

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CME Questions

1. A patient comes to the office with palpitations for 6 hours that he reports for the first time. He has history of hypertension and diabetes, which are both well controlled. On his EKG, he is noted to be in atrial fibrillation with a heart rate of 110 bpm. How would you classify his AF?
   a. Paroxysmal AF
   b. Newly diagnosed AF
   c. Lone AF
   d. Permanent AF
2. An 80-year-old male patient comes to the office with known history of AF. He has well-controlled hypertension and diabetes. He had a CT scan of the head last week, which showed an old ischemic infarct in the frontal area. His BP is 110/60 and heart rate is 105 bpm. He is taking ASA 81 mg daily, lisinopril 5 mg daily and metoprolol succinate 50 mg daily. What would you recommend for this patient?
   a. Add a calcium channel blocker for adequate rate control
   b. Add warfarin with the INR goal of 2-3
   c. Increase the dosage of ASA to 325mg daily
   d. Add digoxin to his drug regimen
3. A 60-year-old patient with known history of PAF comes to your office with debilitating symptoms of palpitations. He has been on warfarin for 6 months with INR of 2.5. You elect to perform an electrical cardioversion for which he is successfully converted to sinus rhythm. You then decide to maintain him on Dronedarone therapy. What labs test should you be monitoring with this medication?
   a. Thyroid function test
   b. Fasting blood sugar
   c. Liver function test
   d. Renal function test
4. Which of the following is a known risk factor for thromboembolism in patients with AF?
   a. Reduced left ventricular ejection fraction
   b. Recent surgery
   c. Excessive alcohol use
   d. Current use of antiarrhythmic medications
5. Which of the following statements is true?
   a. Dronedarone is contraindication in patients with recent history of congestive heart failure
   b. Dabigatran is not indicated as an anticoagulant in patients with permanent AF
   c. Dabigatran requires drug monitoring for effective anticoagulation
   d. Hyperthyroidism in a non reversible cause of AF

In Future Issues

Sickle Cell Disease

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