Atrial Fibrillation and Heart Failure: A Review of the Intersection of Two Cardiac Epidemics

Kay Lee Park, MD and Elad Anter, MD
Beth Israel Deaconess Medical Center, Boston, Massachusetts

Abstract
Atrial fibrillation and heart failure are closely linked cardiac conditions that are both increasing in prevalence due to shared risk factors and common disease mechanisms. The presence of both disease entities portends an increase in morbidity and mortality. There are significant similarities in the treatment strategies of these conditions, and the adequate management of one disease may prevent the development of the other. To this date, a rhythm control strategy, even in the heart failure population, has not been proven to be superior to a rate control strategy. This may in large be due to study design coupled with deleterious effects of antiarrhythmic agents. There have been considerable advances over the past decade in catheter and device based management of atrial fibrillation and studies aimed to examine their long-term effect in patients with heart failure are underway.

Introduction
Epidemiology
Atrial fibrillation (AF) and congestive heart failure (CHF) have emerged as new cardiovascular epidemics over the last decade, and often manifest as coexistent conditions. The high prevalence and progressive nature of these two disease entities is a cause for significant morbidity and mortality. Currently, an estimated 6.6 million patients in the United States, or 2.8% of the population, are affected by heart failure, with >670,000 new diagnoses each year. Heart failure is the primary reason for 12 to 15 million office visits and 6.5 million hospital days yearly. According to the National Hospital Discharge Survey data the annual number of hospitalization for heart failure as a primary diagnosis has increased from 409,000 in 1979 to 1,166,000 in 2004. The steadily increasing number of patients with heart failure is in part due to increased "salvage" of patients with extensive myocardial infarction who previously would not have survived. As the most common hospital discharge diagnosis, heart failure presents a significant economic burden on our society with more Medicare dollars spent in the diagnosis and treatment of heart failure than for any other diagnosis. In 2007, the American Heart Association estimated than $33 billion was spent on heart failure alone.

Atrial fibrillation is also a common diagnosis, with an estimated prevalence of AF in the United States ranging from 2.7 to 6.1 million in the year 2010, with a projected increase in its prevalence. Population based studies based on the growing proportion of elderly individuals in the United States and the current rate of increase in AF incidence, propose a projected number ranging from 5.6 to 15.9 million persons with AF in the United States by 2050. AF is the most common arrhythmia in clinical practice, accounting for approximately one third of admissions resulting from cardiac rhythm disturbances. During the last 20 years, hospital admissions for AF have increased by 66% for a number of reasons, including the aging of the population, the rising prevalence of chronic heart disease, and more frequent diagnosis as a result of increased monitoring. An estimated 26 billion Medicare dollars was spent in the management of AF in the year 2008.

AF and Heart Failure
The association between AF and heart failure was appreciated almost a century ago, and in 1937, Paul Dudley White noted, "Since auricular fibrillation so often complicates very serious heart disease, its occurrence may precipitate heart failure or even death, unless successful therapy is quickly instituted." Modern heart failure series report a prevalence of AF ranging from 13 to 27%, and the prevalence of AF increases in parallel with the degree of heart failure present. Patients with mild heart failure and New

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Corresponding Author:
Elad Anter, MD, Beth Israel Deaconess Medical Center, Division of Cardiovascular Medicine, Harvard School of Medicine, 330, Brookline Ave, Boston, MA
York Heart Association (NYHA) functional class I have an AF prevalence of <5%,19-20 while those with severe heart failure and NYHA functional class IV symptoms have a prevalence of AF up to 50%.21 NYHA functional class II or III heart failure patients have an intermediate prevalence of AF.22-23 Heart failure and AF share common risk factors such as age, hypertension, diabetes, and obesity, along with ischemic, non-ischemic, and valvular heart disease. These factors are associated with myocardial cellular and extracellular alterations, electrophysiologic and neurohormonal changes that combine to create an environment that promotes the development of both heart failure and AF.24

Pathophysiology of AF and Heart Failure
AF Begets AF
The pathophysiologic changes that occur in patients with AF and heart failure are complex and only partially understood, with each disease process creating an environment promoting the development of the other (Figure 1). AF may facilitate the development or progression of heart failure. The incidence of heart failure in individuals with AF in Framingham, Massachusetts17 and Olmsted County, Minnesota13 ranged from 3.3 to 4.4 per 100 person-years of follow up. Compared with patients in sinus rhythm, patients with severe HF and AF have a reduction in stroke volume, cardiac output, peak oxygen consumption, and peak workload.18 Cardiac output is decreased in patients with AF due to various mechanisms. Increase in resting heart rate and an exaggerated heart rate response to exercise results in shortening of diastolic filling time, with a resultant decrease in cardiac output. The loss of atrioventricular synchrony plays a significant role, by impairing diastolic filling, decreasing stroke volume, and increasing mean diastolic atrial pressure, resulting in an estimated 20% reduction in cardiac output.18 In addition, the irregularity of the ventricular response may adversely affect ventricular function and hemodynamic status, with decreased cardiac output, independent of heart rate.19,24

The relationship between AF and heart failure is most notable in the development of tachycardia-induced cardiomyopathy; in patients with poorly controlled ventricular rates during AF. AF is the most common cause of tachycardia-induced cardiomyopathy. The incidence of tachycardia-induced cardiomyopathy is unknown, as most reports have been small, retrospective series or case studies involving mostly patients with AF. Improvement in ejection fraction has been reported in patients who undergo radiofrequency ablation for AF or atrial flutter, and in this patient population the incidence of tachycardia-induced myopathy appears to be around 25-50%.26-29 The first experimental model for this condition was presented by Whipple et al.,30 who demonstrated that chronic rapid atrial pacing led to low-output heart failure. The mechanisms responsible for tachycardia-induced cardiomyopathy have not been fully elucidated. However, experiments in animal models suggest that potential mechanisms for tachycardia-induced cardiomyopathy include myocardial ischemia, myocardial energy depletion, and abnormalities in calcium regulation.31 Studies have confirmed that the elimination of these arrhythmias reverses the hemodynamic and clinical manifestations associated with this syndrome.32

HF Begets AF
Heart failure produces changes in the atrium which promote the development of AF. Various mechanisms including elevation of cardiac filling pressures, dysregulation of intracellular calcium, and autonomic and neuroendocrine dysfunction all play an important role (Figure 1). These changes result in decreased atrial refractory period, slowed atrial conduction, or increased heterogeneity of atrial repolarization, creating a substrate for the initiation and maintenance of AF.18 Atrial stretch, as a consequence of increased atrial volume and pressure, activates stretch-activated ionic currents which result in increased dispersion of refractoriness and alterations in anisotropic and conduction properties.33 Inhibition of these stretch-activated currents by gadolinium can reduce the susceptibility to AF in response to atrial pressure overload.34 Dysregulation of intracellular calcium is an important shared mechanism in the pathophysiology of heart failure and AF. The key regulators of intracellular calcium metabolism, the ryanodine receptor and the sarcoplasmic reticulum Ca2+-ATPase, are downregulated in AF.35-36 Furthermore, atrial ion channel remodeling has been demonstrated by an experimental HF model, with a notable increase in the Na+-Ca2+ exchanger current, which may cause delayed afterdepolarizations and triggered activity.27 Heart failure has been associated with increased interstitial fibrosis.38 Increased fibrosis in the atria leads to abnormal conduction and creates a substrate for AF in animal models.39-40 Lastly, the neurohormonal alterations that occur in HF also promotes structural remodeling and atrial fibrosis.38,41

Prognostic Significance of AF in Heart Failure
The prognostic significance of AF in patients with heart failure remains controversial due to a lack of consensus that AF is an independent risk factor of adverse outcome (Table 1). Several recent trials have identified the presence of AF as an important predictor of mortality. In a retrospective analysis of the Studies of Left Ventricular Dysfunction (SOLVD) trial which enrolled 6517 patients with LV ejection fraction (LVEF) < 35%, baseline AF was an independent predictor for all-cause mortality.19 The increased mortality in AF patients compared to those in sinus rhythm was largely due to an increase in pump failure (16.7 vs. 9.4%). In the DIG trial which enrolled 7788 patients, 11% developed a supraventricular tachycardia (including, but not limited to AF) over a 3 year follow up period.42 The development of supraventricular tachycardia independently increased the risk of total mortality (RR 2.45), stroke (RR 2.35), and hospitalization for worsening CHF (RR 3.00). In the Valsartan in Acute Myocardial Infarction (VALIANT) trial of 14 703 patients with acute myocardial infarction complicated by heart failure, AF was associated with a greater long-term morbidity and mortality.43 AF is also associated with increased mortality in patients with heart failure and preserved ejection fraction. In a study evaluating 300 elderly patients with prior myocardial infarction and HF with preserved LVEF, AF was associated with a significantly higher 6 month mortality rate compared to sinus rhythm (11% vs. 2%).44

Interestingly, AF appears to be a stronger predictor of negative outcomes in the subset of patients with mild to moderate heart failure compared with patients with severe heart failure, in whom the contribution of AF to further impairment in survival is limited. Middlekauf et al12 found that in patients with advanced heart failure with NYHA functional class III-IV, the presence of AF was predictive of decreased 1 year survival (44% vs. 83%) only in patients with a pulmonary capillary wedge pressure of less than 16mmHg on therapy, but not in patients with high pulmonary capillary wedge pressure. Corell et al45 reported a similar finding in outpatients with...
AF and heart failure in whom AF is a stronger predictor of adverse outcome in patients with better cardiac function (LVEF>35%). In the Trandolapril Cardiac Evaluation (TRACE) study, long term mortality was increased in all subgroups of patients with AF except those with the most advanced disease (LVEF<25%). These trials suggest that the independent effect of AF on mortality may be limited to patients with mild to moderate degrees of heart failure.

The timing and chronicity of AF has also been found to be an important prognostic factor. Many studies have found that new-onset AF carries a particularly grave prognosis in patients with heart failure. Almed and Perry studied 944 elderly patients hospitalized for heart failure and found that compared with patients with no past or current AF, those with new onset AF had a 57% higher risk of death. Past or chronic AF was not associated with a significant higher risk of death. New onset AF, but not baseline AF, remained an independent predictor of all-cause mortality in an analysis of COMET. The mortality risk is particularly elevated in the first few months after initial diagnosis. In a community-based cohort of patients newly diagnosed with AF, the mortality risk was substantially higher within the first 4 months, with a hazard ratio of 9.62 (95% CI, 8.93 to 10.32) compared with a hazard ratio of 1.66 (95% CI, 1.59 to 1.73) thereafter (Figure 3).

In summary, there is a large body of evidence to suggest that AF confers worse prognosis in patients with heart failure. This is especially relevant to patients with less advanced heart failure to patients with recent onset of arrhythmia.

**Therapeutic Considerations**

**Rate versus Rhythm Control**

Heart failure patients who develop AF have an increased morbidity and mortality, which would suggest that the restoration and maintenance of sinus rhythm in these patients might improve their long-term outcomes. However, there is currently no data to support that pursuing a rhythm control strategy provides any benefit over rate control. The AF Follow-Up Investigation of Rhythm Management (AFFIRM) and the Rate Control Versus Electrical Cardioversion for Persistent AF (RACE) studies found no benefit for rhythm control strategy and actually showed a trend toward harm compared with rate control. Three other prospective randomized trials comparing rhythm to rate control including the How to Treat Chronic Atrial Fibrillation (HOT CAFE), Strategies of Treatment of Atrial Fibrillation (STAF), and Pharmacological Intervention...
in Atrial Fibrillation (PIAF) trials all showed equivalent outcomes in both arms. It should be noted, however, that only 23% to 64% of patients assigned to rhythm control in these studies actually remained in sinus rhythm. Furthermore, the applicability of these trial data to patients with heart failure is questionable, given the small proportion of patients with heart failure. In the AFFIRM trial, for example, 76% of patients had a normal LVEF, and only 9% had an NYHA functional class of II or greater. Subgroup analyses of large trials focusing on patients with heart failure suggest favorable outcomes for the maintenance of sinus rhythm. In the RACE study, subgroup analysis of heart failure patients indicated an improved outcome with the maintenance of sinus rhythm after cardioversion. Subgroup analyses of heart failure patients with AF who converted to sinus rhythm with amiodarone have demonstrated a survival benefit in the Congestive Heart Failure: Survival Trial of Antiarrhythmic Therapy (CHF-STAT) and a significant improvement in cardiac function and quality of life in the CAFE-II trial, when compared with a rate control strategy. The first prospective trial designed to examine AF therapy strategies in patients with heart failure was the AF Congestive Heart Failure (AF-CHF) trial. In this prospective and randomized study, rhythm control was not superior to a rate control strategy. A total of 1,376 patients with AF and systolic heart failure were randomized to rhythm control (typically with amiodarone) versus rate control. After a mean follow-up of 3 years, the investigators found that rhythm control did not improve mortality, hospitalization due to heart failure exacerbation or stroke when compared to rate control. This study confirmed the applicability of the AFFIRM and RACE trials also to patients with heart failure. However, caution is warranted in interpretation and acceptance of these data. First, patients assigned to rate control strategy were able to achieve adequate rate control at rest and at low-level exercise, which may not reflect “real-life” patients. Second, the benefit of sinus rhythm could have been counterbalanced by the harm of antiarrhythmic medications in a similar fashion to the AFFIRM study. Third, although the prevalence of sinus rhythm in the group assigned to rhythm control was as high as 80%, the actual percentage of patients free of AF following randomization may have been diluted due to significant cross over between the two groups, reflecting a more traditional success rate of amiodarone in the range of 60%. In addition, there is a wide variability in mechanisms of heart failure and underlying structural and hemodynamic abnormalities. Some patients, especially those with diastolic dysfunction, are highly symptomatic in AF and derive significant benefit in the restoration of sinus rhythm while other patients do not significantly benefit from AV synchrony.

Rate Control

Although optimal ventricular rate control in AF is a matter for debate, the guidelines advocates for ventricular rate of 60-80 beats per minutes at rest and 90-110 beats per minutes during moderate exertion. Therefore, adequate rate control should be determined with assessment of chronotropic response with exertion or with a 24-hour Holter monitor. Beta-blockers are the first line agent for rate control in patients with AF and chronic heart failure. In addition to controlling ventricular response, beta-blockers (in particular, bisoprolol, metoprolol succinate, and carvedilol) have shown to decrease mortality in heart failure. Nondihydropyridine calcium channel blockers (including verapamil and diltiazem) are also effective rate-controlling agents, but may not be tolerated in patients with a low LVEF due to their negative inotropic effect. Digoxin is a second line agent for rate control and can be used in conjunction with other rate modulating drugs, and has been shown to improve symptoms and decrease hospitalizations in patients with heart failure.

Rhythm Control

Antiarrhythmic drug options are limited in patients with heart failure. The use of class IC agents was associated with increased mortality in the Cardiac Arrhythmia Suppression Trial (CAST) 70 in patients with ventricular ectopy after myocardial infarction, and their use is not recommended in patients with structural heart disease. Antiarrhythmic drug choices in heart failure patients are limited to amiodarone, dofetilide and sotalol. Amiodarone, a class III agent, has been shown to be safe and effective, but is associated with an increase risk for symptomatic bradycardia in patients with advanced heart failure. Another class III agent, dofetilide, was found to be safe and effective in heart failure patients in the Danish Investigations of Arrhythmia and Mortality on Dofetilide (DIAMOND) study. Sotalol should be used with caution given its increased risk for torsades de pointes, especially in the setting of electrolyte abnormalities, LVEF ≤ 40%, acute onset or decompensated heart failure or renal failure.

AV nodal Ablation and Pacemaker Implantation

Pharmacologic therapy is often ineffective or associated with significant side effects. In patients with symptomatic AF radiofrequency atrioventricular (AV) nodal ablation with subsequent pacemaker placement may be an attractive therapeutic option. In addition to providing symptomatic relief, ablate and pace strategy has been shown to improve cardiac performance. Over a follow up period of 2 years, patients who underwent an AV nodal ablation with pacemaker placement had an improvement in NYHA functional class and decreased hospitalizations. In addition, the LVEF improved from a mean of 42±16% to 50±14%, with the greatest improvement seen in patients with baseline depressed LVEF with an increase from a mean of 35±9% to 46±8%.

The long-term outcomes of the “ablate and pace” strategy is less clear. In a study comparing AV node ablation with AF ablation in elderly patients with pharmacologically refractory AF, AV nodal ablation with pacing with an increased incidence of new heart failure (53% vs. 24%), lower LVEF (44±8% versus 51±10%), and a higher NYHA functional class (1.7±0.9 versus 1.4±0.7). A growing body of evidence underscores the harmful effects of long-term right ventricular pacing. This was evidenced by the DAVID trial which found that in patients with a LVEF ≤40% with an indication for ICD implantation but no indication for antibradycardia pacing, there was trend towards increased mortality and HF hospitalization in patients with chronic RV pacing. Mechanical ventricular dyssynchrony is an established contributor to heart failure and the LV dyssynchrony imposed by right ventricular apical pacing can lead to LV remodeling with dilatation and decreases in LVEF.

Cardiac resynchronization therapy (CRT) may be a preferable pacing method in these patients; however, there is insufficient data at this point to support its routine use. Small randomized studies comparing CRT versus RV pacing in patients undergoing AV
nodal ablation for refractory AF have yielded conflicting results with some studies showing a benefit of CRT\cite{79,85} with significant improvements in 6 minute walk test,\cite{79} reduction in exacerbation of HF and hospitalization,\cite{80} and prevention of the reverse remodeling of the left atrium and left ventricle,\cite{81} while another study failed to show any additional benefit of CRT beyond that conferred by rate regularization.\cite{82} A meta-analysis of five randomized clinical trials of patients with AF undergoing AV nodal ablation found no significant reduction in mortality with CRT.\cite{83}

The Block-HF study that was recently published showed that biventricular pacing was superior to conventional RV pacing in patients with AV block and heart failure. Although this study was not designed to examine the effect of pacing solely in patients with refractory AF, about 50% of all participants had AF.\cite{84}

The theoretical benefit of CRT in conjunction with AV nodal ablation needs to be further evaluated in large-scale, multicenter, randomized controlled trials which are more adequately powered to detect major clinical outcomes, including mortality.

### AF Ablation

Our ever expanding understanding of the mechanisms of atrial fibrillation and rapidly advancing technologies have made catheter-based ablation of atrial fibrillation an increasingly effective and safe modality of treating patients with atrial fibrillation. Despite studies suggesting an equivalent outcome for pharmacologic rhythm or rate control, many patients derive much symptomatic benefit from the maintenance of sinus rhythm.\cite{85-87} The benefit of rhythm control may be counterbalanced by the lack of effective antiarrhythmic drugs, coupled with their significant adverse effects. Catheter-based ablation for AF offers the unique opportunity to retain the benefits of rhythm control without the detrimental effects of antiarrhythmic drugs.\cite{84-87,88-91}

In a prospective study of 58 patients with systolic heart failure, AF ablation resulted in significant improvement in LV function, exercise capacity, symptoms, and quality of life with the majority of patients (78%) remained in sinus rhythm after a mean follow-up of 1 year.\cite{84}

In the more recent the Pulmonary-Vein Isolation for AF in Patients With Heart Failure (PABA-CHF) pulmonary vein isolation was superior to AV nodal ablation combined with biventricular pacing in patients with heart failure.\cite{92} A more recent study randomized patients with advanced heart failure and severe left ventricular dysfunction to a rhythm control with pulmonary vein isolation and rate control strategy.\cite{93} Rhythm control strategy with AF ablation yielded less favorable results without improvement in left ventricular function or 6 minute walk. In addition, only 50% of patients in the ablation group maintained sinus rhythm at 6 months of follow up. Furthermore, this patient population showed increased procedural complication rate of 15%. Patients assigned to ablation in this study were older (mean age 62), had more severe systolic dysfunction (LVEF 16%), and had long-standing and persistent AF (mean 44 months), all predictors of decreased ablation success.\cite{94,95} This study underscores the importance of patient selection for rhythm control strategies, including ablative approach.

### AF Prevention

Once AF develops in patients with heart failure, this is usually accompanied by progressive and irreversible structural changes leading to disease progression.\cite{91} Hence, an ideal strategy in the management of heart failure patients should involve treatments aimed at prevention of AF. In addition to optimal heart failure therapy aimed at restriction and potentially reverse structural abnormalities, several other non-antiarrhythmic therapies have been shown to be effective in reducing the incidence and recurrence rates of AF in both the general population and those with heart failure. Clinical studies have shown that the inhibition of the renin-angiotensin-aldosterone system can decrease the incidence and recurrence of AF in select patients groups with heart failure.\cite{92-95}

In the Candesartan in Heart Failure: Assessment of Reduction in Mortality and Morbidity (CHARM) study angiotensin receptor-1 blockers were shown to decrease the incidence of AF in a broad spectrum of >7500 patients with heart failure.\cite{95} A recent study analyzing the EMPHASIS-HF database, found that aldosterone antagonism with eplerenone in NYHA functional class II patients with systolic heart failure reduced the new onset of AF.\cite{96}

Beta-blocker therapy is also associated with a decreased risk for AF. A meta-analysis of 7 randomized, placebo-controlled trials which included 11 952 patients with heart failure already on angiotensin-converting enzyme inhibitors found that beta-blockers reduced the incidence of new AF from 39 to 28 per 1000 patient-years, with a

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**Table 1: Prognostic Significance of Atrial Fibrillation in Patients with Heart Failure**

<table>
<thead>
<tr>
<th>Author/Substudy</th>
<th>Year</th>
<th>NYHA Class</th>
<th>Patients, n</th>
<th>AF, %</th>
<th>Follow-up, y</th>
<th>Patients in SR, n</th>
<th>Patients with AF, n</th>
<th>P</th>
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<td>1991</td>
<td>III-IV</td>
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<td>II-III</td>
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relative risk reduction of 27%. Lastly, statin therapy has been shown to reduce the incidence and recurrence of AF in heart failure patients. A recent meta-analysis of 6 randomized trials with statins including 3 557 patients showed that their use was associated with a significant decreased risk of AF compared with controls subjects (odds ratio, 0.39; 95% CI 0.18-0.85, p = 0.02), with a more marked benefit in the secondary prevention of AF (odds ratio 0.33) than for new onset or postoperative AF (odds ratio, 0.60). 

Conclusions:
AF and heart failure are common cardiac conditions which often coexist, due to common risk factors and a complex interplay of the pathophysiology of these two disease entities. Their joint association correlates with adverse outcomes. AF and heart failure share common disease mechanisms and treatment strategies. Optimal medical management of heart failure may protect against the occurrence of AF and therapies targeting AF may prevent the development of congestive heart failure. The debate between a rate control and rhythm control strategy is now fueled with new studies comparing rate control with catheter ablation, potentially increasing the efficacy of rate control while minimizing drug-related side effects. Our choice of antiarrhythmic agents remains limited in this sick population due to their deleterious effects. Further data is needed to guide our decision making in the appropriate use of catheter ablation in this patient population. In the meantime, it remains critical for us as caregivers to take into account the unique complexities of our patients in determining their optimal treatment strategy.

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