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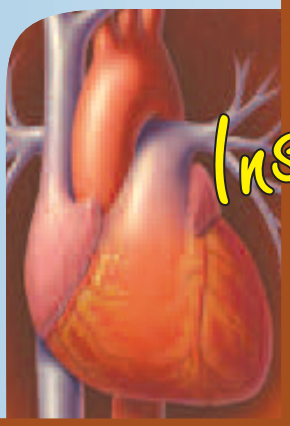

Insight HEART

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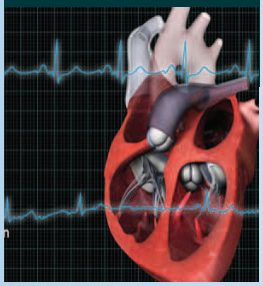
Atrial fibrillation in heart failure

Cardiology News



Atrial fibrillation in heart failure: drug therapies for rate and rhythm control

Introduction

Atrial fibrillation (AF) and heart failure (HF) are major worldwide epidemics that often coexist with complex bidirectional interactions. AF is an occasional cause, a common precipitant, and a frequent complication of HF. Conversely, structural and electrical remodeling changes associated with HF may predispose to AF. When AF occurs in the context of HF, it is associated with worse outcomes. Yet, it remains debated whether AF *per se* is an independent determinant of morbidity and mortality or merely a marker of disease severity.

Managing AF in the HF patients poses several therapeutic challenges. First, the pharmacological armamentarium is restricted by contraindications to drugs commonly used in AF. Second, the relationship between AF and acute decompensated HF may be unclear; AF could be the cause or a consequence of acute HF. In the former scenario, prompt optimal control of AF is advised; in the latter, initiation and optimization of HF treatment provide the best opportunity to improve outcomes. Third, HF patients may respond less favorably to pharmacological therapy for AF. Fourth, although treatment of AF in the absence of HF is primarily guided by symptoms, therapeutic goals in HF patients are less clear. While recent studies have demonstrated similar outcomes with rate- and rhythm-control strategies in patients with AF and HF, it still remains to be determined whether certain patients may benefit from one strategy over the other.

Recent clinical practice guidelines on HF and AF describe the main principles involved in managing AF in the setting of HF. The objective of this review is to provide a structured and practical action plan for managing AF in HF patients. Specifically, an emphasis is placed on the evidence-based selection of an appropriate treatment strategy, defining treatment objectives, and initiating and periodically assessing the impact of selected therapies.

Selecting the strategy: rate versus rhythm

Does routine rhythm control increase survival?

When managing a patient with AF and HF, the first task is to select an initial treatment strategy, bearing in mind that subsequent changes may be required. A rhythm-control strategy involves efforts to restore and maintain sinus rhythm, but it should also aim to control the ventricular response rate in the event of recurrent AF. In contrast, maintenance of sinus rhythm is not the objective of a rate-control strategy, which focuses exclusively on controlling the ventricular response rate. Importantly, both treatment strategies are subject to similar anticoagulation guidelines.

Several clinical trials that compared rate and rhythm strategies in unselected patients with non-permanent AF demonstrated equivalent clinical outcomes. Subgroup analyses limited to HF patients in AFFIRM and RACE trials also suggested that one strategy was not superior to the other. The AF-CHF trial specifically compared rate-control versus rhythm-control therapy in 1,376 patients with AF, HF, and a left

ventricular ejection fraction (LVEF) of 35 % or less. At a median follow-up of 47 months, no difference was observed in cardiovascular mortality (primary outcome). No clinical trial has specifically focused on patients with HF and preserved LVEF although observational data consistently suggest a lack of survival advantage with rhythm over rate control. As a result of these studies, current clinical guidelines do not recommend that rhythm control should be routinely favored over rate control for patients with HF and AF.

Does routine rhythm control affect other outcomes?

In the AF-CHF trial, rhythm control was associated with more frequent hospitalizations in the first year of follow-up compared to rate-control. Although worsening HF requiring hospitalization occurred at similar rates, rhythm control was associated with significantly higher rates of hospitalization for atrial fibrillation and bradyarrhythmias. Moreover, in the AF-CHF echocardiographic substudy, the two treatment strategies were associated with a similar increase in LVEF. While the smaller CAFE II trial reported greater improvement in LV function with rhythm control, ventricular function was assessed qualitatively.

The lack of superiority of rhythm control over rate control also extends to quality-of-life (QoL) metrics. Overall, QoL and functional capacity are substantially impaired in patients with AF and CHF and improve to a similar extent with rate-control versus rhythm-control treatment. In addition, the two treatment strategies incur similar overall costs. Increased expenditures related to therapy for rhythm control (mainly cardioversions) were counterbalanced by other hospitalization costs.

Is maintaining sinus rhythm beneficial?

During follow-up, 58% of patients in the rhythm-control group of AF-CHF had at least 1 recurrence of AF. It has thus been postulated that the lack of benefit in rhythm control may be related to inefficacy of current anti-arrhythmic drugs. In the CHF-STAT trial, which randomized HF patients to amiodarone or placebo, patients with baseline AF who were converted to SR with amiodarone had lower mortality than those who remained in AF. In a similar subanalysis of the DIAMOND trial, sinus rhythm was associated with better survival, regardless of the treatment group (dofetilide vs. placebo). In a subanalysis of RACE, patients with heart failure had superior outcomes if sinus rhythm was actually maintained. Moreover, a higher proportion of time spent in sinus rhythm has been associated with a greater modest gain in health-related QoL, particularly mental health components.

Although the ability to maintain sinus rhythm appears to be a marker of a favorable prognosis, there is little evidence to support the hypothesis that underlying rhythm is independently associated with clinical outcomes. Indeed, in an extensive cardiac rhythm analysis from the AF-CHF trial, AF was not predictive of cardiovascular mortality, total mortality, or worsening HF. The “Rate Versus Catheter Ablation Rhythm Control in Patients With Heart Failure and High Burden Atrial Fibrillation” (RAFT-AF) trial is currently assessing whether a more aggressive rhythm-control strategy that includes catheter ablation may prove superior to rate control. In the interim, current evidence does not support the concept that maintaining sinus rhythm should be favored over rate control in most patients with LV systolic dysfunction.

Individualized care: who benefits from rhythm control?

Although the above-mentioned data apply to most HF patients with AF, rhythm control may be the preferred treatment strategy in selected patients according to their clinical profile (Table 1). For example, clinical trials comparing the two strategies excluded patients with very brief episodes of AF (<1 h), ventricular pre-excitation, highly symptomatic AF for which rate control could not be tolerated, and AF occurring in the setting of an acute myocardial infarction or post-operatively. In patients with new-onset AF and new-onset systolic HF, it may be

Table 1: Selecting the Strategy: Rate vs. Rhythm

Routine rhythm control is not recommended as an initial strategy
Situations in which rhythm control may be considered include:
New-onset rapid AF with newly diagnosed systolic ventricular dysfunction
New-onset AF with acute decompensation of previously stable chronic heart failure
Clinical situations in which rate control is unlikely to succeed, such as:
Rapid AF despite maximal tolerated dose of β-blockers
Paroxysmal rapid AF alternating with sinus bradycardia
High levels of anxiety sensitivity

reasonable to initially opt for a rhythm-control strategy and assess whether maintaining sinus rhythm reverses the cardiomyopathy. In patients with chronic HF acutely decompensated by new-onset AF, an initial rhythm-control strategy may be preferred if the ventricular rate is optimal or only mildly elevated. With such a scenario, it may be speculated that loss of atrial contraction is a more important contributor to decompensation than irregular or fast heart rates. In all situations, it is important to emphasize that whether or not a rhythm-control strategy is pursued, optimal rate control is necessary.

Interestingly, a subanalysis of AF-CHF provocatively suggests that personality traits may be helpful in tailoring therapy for patients with AF and HF. More specifically, anxiety sensitivity refers to the fear of sensations that occur in anxiety-provoking situations, such as palpitations or rapid heart rates. In the AF-CHF trial, a reduction in cardiovascular mortality was observed in patients with a high anxiety sensitivity level who were randomized to rhythm control. In contrast, no differences were observed between rhythm- and rate-control strategies in patients with lower anxiety sensitivity levels. Thus, rhythm control may be preferable for patients in whom episodes of AF provoke a high level of anxiety.

Setting the objectives

The main objectives of managing AF in HF patients are to control symptoms, decrease the need for hospitalizations, improve/stabilize ventricular function, and prevent thromboembolic events. These objectives overlap with the goals of HF therapy, which also include prolonging survival.

Rate control: what is the target?

Mounting evidence suggests that slower heart rates in sinus rhythm are associated with superior long-term survival. For patients with HF and sinus rhythm, the SHIFT trial reported a correlation between higher heart rates and adverse outcomes. The I_f blocker ivabradine was associated with a reduction in adverse events, a benefit attributed to its negative chronotropic effects.

However, the relationship between faster heart rates in AF and poorer outcomes has not been established. The question of whether AF patients benefit from strict rate control was addressed by the RACE II trial. Patients with permanent AF were randomized to either strict rate control (<80 beats per minute bpm at rest and <110 bpm with moderate exercise) or lenient rate control (<110 bpm at rest). At a follow-up of 3 years, lenient rate control was non-inferior for the combined primary outcome of cardiovascular death, HF hospitalization, systemic embolism, major bleeding, and arrhythmic events. Importantly, at baseline, only 15% of patients had an LVEF <40% and only 10% were ever hospitalized for HF. Caution should, therefore, be exerted in generalizing these findings to patients with HF and/or LV systolic dysfunction.

Data on heart rate in AF and HF are primarily derived from retrospective studies or subgroup analysis of randomized trials. In a recent CHARM subanalysis, heart rate in patients with baseline AF was of no prognostic value, although it was a strong predictor of adverse outcomes in patients without AF ($p < 0.001$ for interaction) . Other studies have reported similar findings. Another recent study assessed the effect of increasing beta-blocker dose to achieve a heart rate below 70 bpm in AF. Patients did not improve their exercise tolerance, QoL, or BNP levels. Similarly, preliminary data from a combined AFFIRM and AF-CHF analysis show that higher baseline heart rates in sinus rhythm but not in AF are associated with increased mortality and hospitalization rates.

In the absence of robust clinical data, recent guidelines do not recommend a specific target ventricular rate for AF in patients with HF. We, therefore, target rates tested in clinical trials that demonstrated equivalent outcomes with rate versus rhythm control, that is, below 80 bpm at rest and 110 bpm during a 6-min walk test (6MWT). We generally perform 6MWTs if the patient remains symptomatic despite an optimal heart rate at rest. Moreover, in patients with non-permanent AF, we aim for 60 bpm or the lowest tolerated heart rate in sinus rhythm.

Rhythm control: what is the target?

Management decisions for rhythm control are often based on the objectives of controlling symptoms and decreasing the need for hospitalization. It is important to note that recurrence of AF despite anti-arrhythmic drug therapy (AAD) is to be expected and is not synonymous with treatment failure. AF may be considered well

controlled if recurrences are infrequent, especially if they are well tolerated, self-terminating, and do not prompt hospital visits. Changes to therapy are generally considered in the event of frequent symptomatic or poorly tolerated recurrences, especially if they result in repeated hospitalizations. In short, abolishing AF is not a realistic target of rhythm-control therapy. Rather, pharmacological therapy should aim to reduce the symptomatic AF burden.

Attaining the objectives

Rate control: pharmacological treatment

In HF patients with LV systolic dysfunction and AF, β-blockers and digitalis are the primary agents for rate control and are discussed below. Amiodarone may be occasionally helpful for this purpose, especially in acute decompensated HF where it is often better tolerated than β-blockers and more effective than digitalis. Since amiodarone can convert the patient to sinus rhythm, it should be reserved for patients with new-onset AF <48 h or those with therapeutic anticoagulation for at least 3 weeks. In other cases, a transesophageal echocardiogram (TEE) should be obtained to exclude an atrial thrombus prior to administering amiodarone. In patients with HF and preserved LVEF, non-dihydropyridine calcium channel blockers such as diltiazem and verapamil could also be considered. They should be avoided in patients with depressed LVEF because of their negative inotropic effects. Based on the ANDROMEDA and PALLAS trials, dronedarone should also be avoided in patients with HF and those with permanent AF.

β-Blockers should be considered the mainstay of rate-control therapy in patients with AF and HF. Beyond their efficacy in rate control, beneficial effects on long-term survival are well established in patients with LV systolic dysfunction. Clinical guidelines thus recommend that most patients with an LVEF <40% should be prescribed a β-blocker, regardless of underlying rhythm. Because of variable pharmacodynamics of different β-blockers, one of the three agents with demonstrated survival benefits should be preferred, that is, carvedilol, bisoprolol, or metoprolol succinate. When selecting the β-blocker, it should be noted that carvedilol may have less potent rate-slowing effects, especially with certain genetic polymorphisms. Nevertheless, a subanalysis of the US Carvedilol Heart Failure Trials limited to patients with AF reported that carvedilol was associated with an increased LVEF and trend toward decreased mortality. Two other subanalyses of non-placebo-controlled β-blocker trials suggested similar improvements of LVEF. In contrast, a subanalysis of the CIBIS II trial found that bisoprolol compared with placebo did not improve survival in patients with AF, unlike those with sinus rhythm. Similar results were reported with nebivolol in the SENIORS trial. A subanalysis of MERIT-HF did not show a mortality benefit of metoprolol in patients with AF, possibly related to lack of statistical power. Whether or not β-blockers do indeed significantly alter the course of HF in patients with coexisting AF, their known safety and efficacy renders them the first choice for rate control in this population.

Digitalis has been used for centuries in patients with HF. More recently, the DIG trial demonstrated reduced hospitalizations with digoxin in patients with depressed LVEF and sinus rhythm but no mortality reduction. In AF, ventricular rate slowing is due to a parasympathetic effect. Thus, it is of limited efficacy during exercise when used alone, but exerts a synergistic effect with β-blockers. In patients with both HF and AF, the CAFE trial suggests that the combination of digoxin and carvedilol reduces symptoms, improves ventricular function, and leads to better ventricular rate control than either agent alone. Yet, in a large cohort of patients with AF and HF, digoxin either alone or with β-blockers did not affect mortality. More alarmingly, a recent multivariate analysis of the AFFIRM trial showed increased all-cause mortality with digoxin regardless of the presence or absence of HF. Based on such concerns, it appears reasonable to discourage routine digoxin use as first-line rate-controlling agent in patients with AF and HF. It may be considered as a second-line agent when rate control with β-blockers is suboptimal or to decrease hospitalizations from HF.

Rhythm control: pharmacological treatment

Amiodarone is the most common pharmacological agent used for rhythm control in patients with coexisting AF and HF. In the AF-CHF trial, 82 % of patients randomized to rhythm control were on amiodarone at 12 months. The safety of amiodarone in HF is well established. It is also the most efficacious AAD for maintaining sinus rhythm. Preliminary data from a combined AFFIRM and AF-CHF analysis suggest that amiodarone's efficacy in

maintaining sinus rhythm is independent of LVEF, with recurrence rates that are no higher in the setting of HF. While the rate of conversion to sinus rhythm also appears to be independent of left ventricular function, amiodarone is not the most potent agent for acute pharmacological conversion. Unlike class IC AADs that act within the first few hours, it may take up to 24 h to achieve amiodarone's maximum efficacy for cardioversion. Importantly, amiodarone's numerous multiorgan adverse effects are well known, which limit long-term therapy and contribute to the 15% rate of discontinuation.

Dofetilide is an alternative AAD for rhythm control in patients with HF. Its safety was demonstrated in the DIAMOND trial, designed to assess the effect of dofetilide on mortality in patients with HF and LV systolic dysfunction with or without AF. In a subanalysis restricted to patients with AF, dofetilide was associated with a higher rate of conversion to and maintenance of sinus rhythm. It was also associated with a reduction in the hospitalization rate of patients with both AF and HF. Mortality was not increased in the overall cohort or in the subgroup of patients with AF. The feared complication with dofetilide is QT prolongation with subsequent torsades de pointes, which has been reported in about 3% patients with HF and 75% of episodes occurring within the first 3 days. Initiation of therapy should, therefore, be performed in a hospital setting by a physician experienced in prescribing dofetilide. Doses should be adjusted according to the creatinine clearance, and the QT interval should be monitored closely.

The efficacy and safety of sotalol in maintaining sinus rhythm are well demonstrated in the SAFE-T trial, which mostly enrolled patients with normal LVEF. The d-sotalol isomer increases mortality in patients with ischemic cardiomyopathy and depressed LVEF, as reported in the SWORD trial. This isomer lacks the β-blocker effect known to be beneficial in HF patients, while only retaining potassium channel-blocking properties. The racemic *d,l*-sotalol (the one currently on the market) might be beneficial in patients with HF and normal LVEF and some patients with low LVEF and an implantable cardioverter-defibrillator (ICD). In fact, a study of *d,l*-sotalol vs. placebo in patients with an ICD for secondary prevention showed that it was tolerated and that it reduced appropriate and inappropriate shocks without impacting mortality, even in patients with LVEF <30%. It remains to be determined whether additional benefit may be obtained by adding sotalol to another β-blocker with a proven mortality reduction in patients with HF. While in the aforementioned trial, addition of another β-blocker to sotalol did not affect the combined outcome of death or ICD shock, it was underpowered to detect differences in survival. If sotalol is used, renal function should be closely monitored, as HF patients tend

Table 2 Drugs commonly used for rate and rhythm control in AF and HF

	Loading dose	Maintenance dose	Comments
Rate control			
First-line treatment: β-blockers with demonstrated survival benefit			
Carvedilol	–	3.125 to 50 mg bid.	Start at minimal dose for patients with significant systolic dysfunction
Bisoprolol	–	1.25 to 10 mg qd.	
Metoprolol Succinate	–	25 to 200 mg qd.	Avoid initiating β-blockers during acute decompensation with systolic dysfunction
Second-line treatment: Digoxin			
Digoxin	1 mg in divided doses	0.0625 to 0.25 mg qd.	Renal dose adjustment required Avoid digoxin levels above 1 ng/mL Limited efficacy for rate control on exertion
Rhythm control			
Amiodarone	10 g over several weeks	100 to 200 mg qd.	Also useful for rate control Monitor for systemic toxicity Assess risk of embolism prior to initiation

to have a variable glomerular filtration rate, which might result in sotalol accumulation with the associated risk of torsades.

In practice, when considering pharmacological (or electrical) conversion to sinus rhythm, thromboembolic risk should first be assessed unless hemodynamic instability justifies immediate cardioversion. Factors associated with a low stroke risk include AF of known duration <48 h and absence of a mechanical valve, rheumatic heart disease, and recent stroke or transient ischemic attack. Patients with AF of unknown duration or >48 h or any of these high-risk features should either have therapeutic anticoagulation for at least 3 weeks or no demonstrated thrombus on TEE prior to cardioversion. AADs should not be initiated in patients with thromboembolic contraindications to cardioversion. Following cardioversion, amiodarone is the drug of choice for rhythm control in patients with HF. Dofetilide and sotalol are generally reserved for special circumstances such as amiodarone intolerance or failure, while class IC AAD should generally be avoided. When initiating amiodarone in a patient on warfarin, it is important to closely monitor the INR and reduce the dose of warfarin if need to be. Monitoring for bradyarrhythmias as well as thyroid, liver, and lung toxicity is also advised. Drugs used in arrhythmia management in AF are summarized in Table 2.

Upstream therapies

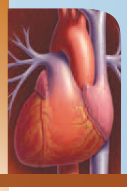
In a European survey, patients with AF and HF with systolic dysfunction had a lower prescription rate of recommended drugs, such as β-blockers and ACE inhibitors (ACEI) or angiotensin receptor blockers (ARBs). This may reflect the paucity of studies specifically designed for patients with AF and HF. Nonetheless, observational data and subanalyses of randomized trials support the benefits of HF therapy in patients with coexisting AF. Boldt et al. reported that the success of cardioversion was dependent on HF therapies. Other studies have validated that ACEI and ARBs have similar benefits in AF and non-AF patients. A meta-analysis also suggested that ACEI and ARBs prevent new and recurrent AF, an effect also supported by animal studies. However, two recent, large, randomized trials, GISSI-AF and ACTIVE-I, did not demonstrate a decreased recurrence rate of AF with ARBs. The question of whether ACEI and ARBs prevent AF recurrences remains unresolved and is the subject of ongoing trials. These drugs, however, are beneficial in HF patients, especially those with LV dysfunction, and should, therefore, be prescribed to patients with AF and HF.

Aldosterone antagonists, spironolactone and eplerenone, are increasingly prescribed for patients with HF based on the RALES and EMPHASIS-HF trials. A subanalysis of EMPHASIS-HF supports a similar benefit in patients with and without baseline AF and a reduction in new-onset AF with eplerenone. The impact of spironolactone on recurrent AF remains controversial. Importantly, a recent subanalysis of AF-CHF found that spironolactone was not associated with a reduction in AF and, more concerning, with increased mortality (driven by an increased rate of sudden death). For the time being, these drugs should be prescribed according to HF guidelines, regardless of AF. If prescribed, close monitoring of creatinine and potassium levels is warranted to ensure safety.

Statins are beneficial in patients with ischemic heart disease. The GISSI-HF and CORONA studies, however, failed to show clinical benefit in HF patients. In a subanalysis of GISSI-HF, rosuvastatin decreased the incidence of new-onset AF only after adjustment for clinical variables. In a recent meta-analysis including patients with and without HF, statins decreased new and recurrent AF. However, a growing body of literature casts doubt on any clinically relevant beneficial effect of statins on AF. In a subanalysis of AF-CHF, statins did not prevent AF recurrences or decrease the overall AF burden in HF patients. In sum, statins are not indicated in patients with AF and HF for the purpose of protecting against recurrence of AF.

Supplementation of n-3 polyunsaturated fatty acids (PUFA) has been suggested to be beneficial in both AF and HF. The GISSI-HF trial also included randomization of PUFA vs. placebo in patients with HF and low LVEF. In this study, PUFA decreased both mortality and cardiovascular hospitalizations. While this study supports PUFA supplementation in patients with AF and HF, benefits remain controversial and disputed by recent trials. In sum, PUFA supplementation may be considered in HF patients but their effect on AF prevention and recurrence remains contentious.

Interestingly, a combined subanalysis of AF-CHF and



AFFIRM suggests that systolic blood pressure is a predictor of AF recurrence. Preliminary data show that in patients with LVEF <40%, a systolic blood pressure >140 mmHg is associated with an increased risk of AF recurrence and increased AF burden. By contrast, systolic blood pressure is not a determinant of AF recurrence or burden in patients with LVEF >40 %. These findings highlight the importance of appropriate blood pressure control in patients with AF and HF.

Assessing outcomes

As a general guiding principal, if the objectives of rhythm- or rate-control therapy are achieved, the treatment regimen is pursued. Therapy should be reassessed in patients who remain symptomatic and/or with suboptimal control. This may require optimizing the current strategy or changing approaches altogether. Maximization of HF drugs to recommended doses should also be done.

Differentiating AF from HF symptoms

An important challenge in managing coexisting AF and HF is differentiating AF- from HF-related symptoms. While palpitations are more likely attributable to AF, dyspnea or fatigue could reflect AF or HF and may be multifactorial. Evidence of hypervolemia suggests that symptoms are related to decompensated HF and should prompt increases in diuretics, titration of HF therapy, and possible optimization of AF treatment. If symptoms persist in a euvolemic patient, electrocardiographic monitoring could be useful in assessing the potential contributing role of AF. In a patient with a pacemaker or ICD, AF burden and rate control can be readily estimated in most devices. Alternatively, Holter monitoring may prove helpful. If symptoms occur on exertion, a 6MWT or formal exercise testing may assist in assessing the adequacy of rate- or rhythm-control strategies.

Persistent symptoms in the rate-control patient

In a patient managed with a non-stringent rate-control strategy who remains symptomatic, lower target levels at rest, such as 60 bpm or less, should be considered. If symptoms persist despite an optimal resting heart rate, rate control during exertion should be assessed and optimized. If drug therapy fails to adequately control heart rate despite combinations of rate-slowing agents, AV node ablation and implantation of a biventricular pacemaker may be considered. Alternatively, a trial of rhythm control may be contemplated if not previously attempted. After all, sinus rhythm offers the best rate control.

Persistent symptoms in the rhythm-control patient

One approach to managing the patient who presents with recurrent symptomatic AF episodes despite AAD therapy is to first optimize rate control. If rate control results in symptomatic relief, the AAD may be discontinued and the patient crossed over to a rate control strategy. If rate control insufficiently relieves symptoms, repeated cardioversions with AAD dose adjustments may be attempted. Amiodarone should be considered if not already attempted. Otherwise, AF ablation remains a potential option. Of note, symptoms may gradually abate in patients who initially report discomfort when crossed over from rhythm- to rate-control strategies.

Strategy crossovers

In AF-CHF, 21% of patients in the rhythm-control group and 10% of patients in the rate-control group crossed over to the alternative strategy. A preliminary analysis revealed that those who crossed over had superior outcomes, likely reflecting the value of sound clinical judgment in managing patients with AF and HF.

Concluding remarks

In the last years, much effort has been directed toward improving the management of AF and HF as separate disease entities. In contrast, despite their frequent coexistence, few randomized trials have directly addressed management aspects in such challenging patients. Neither data from HF patients without AF nor data from AF patients without HF are sufficient to guide therapy in patients with concomitant AF and HF. In the absence of clear evidence-based guidelines, management considerations discussed in this review reflect the insights gained from the results of trials, such as AF-CHF and its numerous substudies, in addition to clinical experience and sensible judgment.

Ref.: Atrial fibrillation in heart failure: drug therapies for rate and rhythm control. Rafik Tadros, Paul Khairy, Jean L. Rouleau, Mario Talajic, Peter G. Guerra and Denis Roy. Heart Fail Rev (2014) 19: 315- 324.

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Cardiology News

Severe Sleep Apnea Linked to Resistant Hypertension

Severe obstructive sleep apnea may interfere with blood pressure (BP)–lowering treatment in patients at high cardiovascular disease risk or with established cardiovascular disease, results of a multicenter clinical trial suggest. Patients with severe obstructive sleep apnea had a 4-fold higher odds of resistant elevated BP despite receiving an aggressive antihypertensive medication regimen, even after consideration of well-recognized hypertension risk factors, including age, sex, race, body mass index, smoking, diabetes mellitus, and cardiovascular disease. Further, their group found that increased dietary sodium correlated with sleep apnea severity in patients with resistant hypertension and hyperaldosteronism.

J Clin Sleep Med. 2014;10:835-843.

AFFORD: Fish Oil Does Not Prevent AF Recurrence in Low-Risk Patients

For low-risk patients with symptomatic paroxysmal or persistent atrial fibrillation who are not currently taking antiarrhythmic medication, a high-dose fish oil does not prevent the recurrence of AF, nor does it appear to reduce inflammation or oxidative stress. These are the primary results of the Multicenter Study to Evaluate the Effect of n-3 Fatty Acids on Arrhythmia Recurrence in Atrial Fibrillation (AFFORD), a Canadian study. In total, 337 patients with symptomatic paroxysmal or persistent AF (mean duration 2.5 years since first AF diagnosis in the fish-oil arm) were randomized to 4 g of fish oil per day or to placebo. After an average follow-up of 271 days, the AF-recurrence rate was 64.1% in the fish-oil arm and 63.2% in the placebo arm, a difference that was not statistically significant.

J Am Coll Cardiol 2014; 64:1441-1448.

Living Near Major Roadways Increases Likelihood of Hypertension

Living close to major roadways, such as freeways, freeway ramps, or heavily trafficked arterial roads, is associated with higher blood pressure, according to a new analysis. For example, individuals who lived within 100 m of any major roadway were 22% more likely to have hypertension than those who lived more than 1000 m from the road. The association was adjusted for multiple variables, including potential causes of hypertension such as body-mass index (BMI), physical-activity levels, and smoking status. This is an analysis of the Women's Health Initiative and includes 5401 postmenopausal women living in the San Diego area. More than 40% had hypertension and were equally divided by BMI, with roughly one-third normal weight, overweight, or obese. The median distance from a major roadway was 836 m. The researchers did not assess pollution levels, but higher levels of traffic-related air pollution is associated with proximity to major roads. Noise levels are also higher. While both of these might increase the risk of hypertension, the researchers note that mechanisms are incompletely understood at this stage.

J Am Heart Assoc 2014

Editorial Note

Dear Doctor,

We are happy to present the 35th issue of "Insight Heart". It is a small endeavor to provde you compiled & updated information on cardiovascular diseases and its management. This issue is focused on ***"Drug treatment in Atrial Fibrillation and Heart Failure"***. We will appreciate your thoughtful comments.

Thanks and regards.