# Atrial fibrillation and heart failure: A contemporary review of current management approaches

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Atrial fibrillation (AF) and heart failure (HF) frequently coexist and complicate the course of treatment of each other. AF with rapid ventricular conduction can lead to tachycardia-mediated cardiomyopathy, which is a reversible cause of cardiomyopathy. However, in most cases, AF is the manifestation of various underlying cardiomyopathies. Guideline-directed pharmacological and device therapy for HF is essential. The management options for AF and HF include pharmacological rhythm control, pharmacological rate control, and interventional approaches, which include catheter ablation for AF via pulmonary vein isolation and atrioventricular node ablation. This is a contemporary review to discuss the available evidence regarding the various management approaches in this specific patient group.

**KEYWORDS** Anti-arrhythmic agents; Atrial fibrillation; Heart failure; Catheter ablation; Rate control; Rhythm control

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## Introduction

Atrial fibrillation (AF) and heart failure (HF) are the 2 significant epidemics in cardiovascular medicine, leading to increased morbidity and mortality.[1] AF and HF frequently coexist and impact reciprocally on each other.[2] The management of patients with AF and HF is challenging, as many medications used for AF in the absence of HF are contraindicated in the presence of HF, especially with HF and reduced ejection fraction. There has also been increasing data on catheter ablation in patients with AF and HF. Here, we attempt to provide an updated contemporary review of the various management approaches for patients with AF and HF, both with preserved (HFpEF) and reduced ejection fraction (HFrEF).

## Pathophysiology

AF can contribute to HF by multiple mechanisms. Loss of atrial transport causes a fall in cardiac output.[3] Increase in heart rate and shortening of diastolic filling time can also reduce cardiac output.[4] Irregular ventricular response results in a 25% reduction in cardiac output, as filling during long cycles does not compensate for the reduced filling in short cycles.[4,5] AF with rapid ventricular rate and irregularity can lead to a tachycardia-mediated reversible cause of cardiomyopathy.[6]

HF also contributes to AF via multiple mechanisms. HF also increases atrial filling pressure and atrial dilatation, leading to atrial scarring and fibrosis, promoting ionic remodeling and AF.[7] Atrial tissue stretching in HF promotes AF by inducing triggered activity.[8] Left atrial dilatation has been shown in animal studies to be associated with significant shortening of the atrial refractory period, which has been shown to promote AF.[9,10]

There are some differences between HFrEF and HFpEF in atrial remodeling in AF. There is greater eccentric left atrial remodeling in HFrEF and increased left atrial stiffness in HFpEF.[11] AF is more likely to precede HF than to follow HF, especially in HFpEF.[2] The reasons for this may be owing to similar underlying mechanisms driving the development of AF and diastolic dysfunction, such as myocardial inflammation and fibrosis that also leads to atrial fibrosis.[12] It is also possible that AF is less well tolerated in patients predisposed to HFpEF and therefore triggers its clinical recognition.[13] There are some differences in patient outcomes based on the temporal relationship of AF and HF. Mortality is higher in patients with HFrEF who develop new AF compared with patients with HFpEF who develop new AF.[2] Preexisting AF, occurring before HF, is less associated with mortality.[2,14]

## Risk factors

The risk factors that predispose to AF are similar in HFpEF and HFrEF.[15] These risk factors include obesity, sleep-disordered bleeding, diabetes, and hypertension. The mechanistic pathways based on animal models on obesity and AF suggest that mechanisms include increased inflammatory infiltrates, atrial fibrosis, fat cell infiltration, and shortening of pulmonary vein effective refractory period.[16,17] Studies have shown that AF burden reduction can be achieved with

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**KEY FINDINGS**

- The treatment of atrial fibrillation (AF) in patients with heart failure (HF) begins with optimal guideline-directed management of HF.
- The management options for AF and HF include pharmacological rhythm control or rate control, as well as interventional approaches, which include catheter ablation for AF via pulmonary vein isolation and atrioventricular node ablation.
- Randomized trials have shown that rhythm control with catheter ablation via pulmonary vein isolation can improve symptoms, increase quality of life, and improve left ventricular ejection fraction.
- Catheter ablation should be used selectively, taking into consideration the patient's comorbidities and risk of complications.

at least a 10% reduction in weight.[18] As for obstructive sleep apnea, analysis of ORBIT-AF (Outcomes Registry for Better Informed Treatment of AF) demonstrated that patients with sleep-disordered breathing who were using continuous positive airway pressure therapy were less likely to have progression of AF to permanent AF.[19] In those who underwent AF ablation, there is a lower risk of recurrence in patients with sleep-disordered breathing who used continuous positive airway pressure compared to those who did not.[20] Diabetes has been associated with a higher risk of AF and may predispose to atrial structural, electrical, and autonomic changes.[21] Glycemic control has also been associated with a reduced risk of AF.[22] The RACE 3 trial demonstrated that in patients with early persistent AF and HF, risk factor–driven pharmacological and lifestyle interventions improve maintenance of sinus rhythm.[23] Therefore, risk factor modification is an important part of AF management in patients with HF.

## Pharmacologic rhythm control

Pharmacologic therapy to maintain sinus rhythm is challenging in HF patients because many antiarrhythmic drugs may be proarrhythmic, have negative inotropic effects, or are associated with adverse events and toxicity. Class IC antiarrhythmic drugs are contraindicated in ischemic heart disease and reduced left ventricular ejection fraction (LVEF) owing to increased risk of proarrhythmia, based on data from the CAST trial.[24] Therefore, class IC antiarrhythmic drugs are generally recommended only in patients with AF and without any structural heart disease.

Table 1 summarizes the antiarrhythmic drugs used in patients with HF. The choices of pharmacologic rhythm control agents in patients with HFrEF are dofetilide and amiodarone.[25] Dofetilide, a class III antiarrhythmic drug, has its safety in patients with HF established by the DIAMOND-CHF trial, which showed that in patients with congestive HF and reduced left ventricular function, it was effective in converting AF and also reducing risk of hospitalization for worsening HF (Table 2).[26] However, because of the risk of torsades de pointes seen in 3% of HFrEF patients compared to 1% in those with normal left ventricular function, with the majority of them in the first 3 days, it is recommended that patients be hospitalized for 3 days for dofetilide initiation. It is contraindicated if baseline QTc $>$ 440 ms. Amiodarone's safety and efficacy in HF have been evaluated in the CHF-STAT trial, which showed that patients treated with amiodarone who convert to sinus rhythm have lower mortality compared to those who do not.[27]

Options of management in patients with HFpEF, in addition to dofetilide and amiodarone discussed above, also include sotalol and dronedarone.[28] There are a few reasons why sotalol is not preferred in patients with reduced ejection fraction. One reason is that patients with HFrEF are usually on beta blockers as part of their guideline-directed medical therapy. Sotalol also has a beta-blocker effect, and the addition of sotalol may lead to intolerance, especially at a dose needed to achieve class III antiarrhythmic dosing effect, which is at least 80 mg twice a day. Furthermore, there is also an increased risk of torsades de pointes in patients with low ejection fraction.[29] Dronedarone, a class III antiarrhythmic drug, is reasonable as an antiarrhythmic in patients with HFpEF but is contraindicated in patients with New York Heart Association class III or class IV HF or with left ventricular systolic dysfunction. The ANDROMEDA trial[30] showed increased mortality owing to worsening HF in patients with severe HF and left ventricular dysfunction treated with dronedarone. From that study, there have been varying extrapolations of the data, ranging from being only contraindicated in those with HFrEF but acceptable in HFpEF to being contraindicated in HFrEF or decompensated HFpEF, to being contraindicated in any HF. In a study of rhythm vs rate control in patients with HFpEF, which showed lower mortality with rhythm control, 11.4% of patients were on dronedarone.[31] A Swedish study on the safety of dronedarone in routine clinical care showed that HF patients on dronedarone had lower mortality than HF patients not on dronedarone, though it could be attributable to selection bias towards low-risk patients.[32] In the 2020 European Society of Cardiology AF guideline,[28] a class IA recommendation for dronedarone for rhythm control was given, even in patients with HFpEF or mildly reduced but stable left ventricular function.

Overall, the data suggest that about half of the patients with HF and AF who receive pharmacologic rhythm control maintained normal sinus rhythm at 1 year. The AF-CHF trial evaluated pharmacologic rhythm control over rate control in patients with HF.[33] Patients with symptomatic HFrEF and AF (33% paroxysmal and 67% persistent) were randomized to rhythm control, where 82% received amiodarone, vs rate control.[33] There was no difference in all-cause mortality, cardiovascular mortality, or worsening HF. However, on follow-up, 58% of patients in the rhythm control group had a recurrence of AF, and 30% of patients in the rate control group were in sinus rhythm, suggesting the less optimal

**Table 1** Antiarrhythmic drugs used in heart failure patients

| | Antiarrhythmic drug | Channels blocked | Dose | Metabolism | Proarrhythmia | Side effects | Selected drug interactions |
|---|---|---|---|---|---|---|---|
| HFrEF or HFpEF | Dofetilide | $I_{Kr}$ | 250–500 mcg b.i.d. | Renal (80%) and hepatic (20%) | Torsades de pointes | Generally well tolerated | Verapamil, hydrochlorothiazide |
| | Amiodarone | $I_{Kr}$, $I_{Na}$, $I_{Ca}$, β, α, Ach | Oral load, then 200 mg/d | Hepatic | Bradycardia | Pulmonary, hepatic, thyroid, neurologic, ocular, photosensitivity. | QT-prolonging drugs, warfarin, digoxin |
| HFpEF only | Sotalol | $I_{Kr}$, β | 80–160 mg b.i.d. | Renal | Bradycardia, torsades de pointes | Fatigue, worsening heart failure | QT-prolonging drugs, beta blockers |
| | Dronedarone | $I_{Kr}$, $I_{Na}$, $I_{Ca}$, β, α, Ach | 400 mg b.i.d. | Hepatic | Bradycardia | Hepatotoxicity | Verapamil, statins, beta blockers, digoxin, dabigatran |

b.i.d. = twice a day; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction.

**Table 2** Randomized trials of antiarrhythmics for atrial fibrillation in patients with heart failure

| Trial | Population | Intervention | Comparator | Results |
|---|---|---|---|---|
| Torp-Pedersen et al, 1999[26] | Symptomatic CHF and severe LV failure | Dofetilide | Placebo | Dofetilide was effective in converting AF, decreasing its recurrence, and decreasing HF hospitalization without increasing mortality. |
| Deedwania et al, 1998 [CHF-STAT][27] | CHF and AF | Amiodarone | Placebo | Amiodarone was effective in converting AF and patients who convert had lower mortality. |
| Køber et al, 2008 [ANDROMEDA][30] | CHF and severe LV failure | Dronedarone | Placebo | Dronedarone was associated with increased early mortality. |
| Roy et al, 2008 [AF-CHF][33] | AF and HFrEF | Rhythm control (antiarrhythmics) | Rate control | No differences in mortality. |

AF = atrial fibrillation; CHF = congestive heart failure; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; LV = left ventricle.

outcomes of pharmacological rhythm control. The RACE 3 trial,[34] which included patients with HFpEF and HFrEF with early persistent AF randomized to targeted therapy of underlying conditions plus rhythm control or routine rhythm control therapy, showed that antiarrhythmic therapy was effective in half of the patients at 1 year. Amiodarone was the most effective drug and maintained normal sinus rhythm in 58% of patients, compared to 32% with flecainide and 23% with sotalol or dronedarone.

## Pharmacologic rate control

The options for atrioventricular (AV) nodal blockers to obtain rate control include beta blockers, nondihydropyridine calcium channel blockers (diltiazem and verapamil), and cardiac glycosides (digoxin).

The nondihydropyridine calcium channel blockers (diltiazem and verapamil) are contraindicated in patients with reduced ejection fraction owing to increased mortality.[25] Therefore, beta blocker can be considered as first-line therapy for rate control in patients with HFrEF. Digoxin is not as effective as beta blockers or calcium channel blockers for rate control and therefore should not be used as a first-line drug. As it slows ventricular rate primarily by vagotonic inhibition of AV nodal conduction, it is more effective in a resting state and less effective when vagal tone is low and sympathetic tone is high, such as during exercise.[35]

Data from a large meta-analysis in patients with HF demonstrated that beta blockers led to a significant reduction in all-cause mortality in patients with sinus rhythm but not in patients with AF.[36] It is unclear if the findings suggest that beta blockers have no benefit in patients with AF and HF, or patients with sinus rhythm have better outcomes. An analysis of the AF-CHF trial showed that beta blockers are associated with lower mortality in patients with HFrEF.[37] Therefore, beta blockers remain an important medication to be used in patients with AF and HFrEF.

The optimal target for rate control in patients with AF and HF is unclear. The post hoc analysis of RACE II on patients with AF and HF (both HFrEF and HFpEF) showed that strict rate control had no effect on outcomes.[38] Data from the Swedish Heart Failure Registry specific to patients with HFrEF showed that a heart rate of greater than 100 beats per minute (bpm) in AF was associated with increased mortality.[39] The 2009 American College of Cardiology and American Heart Association HF guidelines recommended a target ventricular rate of $<$80–90 bpm at rest and $<$110–130 bpm with moderate exercise.[40] The 2020 European Society of Cardiology guidelines[28] recommended lenient rate control as an acceptable initial approach regardless of HF status. Stricter rate control is reserved for patients with tachycardia-induced cardiomyopathy, when patients remain symptomatic, or when there is biventricular pacing in order to achieve higher biventricular pacing percentage.[28] Therefore, the decision on rate control target should be individualized, based on patients' symptoms, suspicion for tachycardia-mediated cardiomyopathy, and activity level. The level of rate control can be assessed with an ambulatory monitor such as a Holter monitor.

## Interventional management: Rhythm control with catheter ablation

There has been increasing evidence in catheter ablation for patients with AF and HFrEF. So far, there have been 8 published randomized trials assessing the efficacy of AF ablation in patients with HFrEF (Table 3). There were variabilities in the ejection fraction inclusion, ranging from $\leq$35% to $<$50%. Most of the studies included only persistent AF. There were also variabilities in trial design with the control group ranging from medical rate control, amiodarone, AV node ablation and biventricular pacing, or best medical therapy with either rate or rhythm control.

Four studies compared catheter ablation with medical rate control in patients with reduced ejection fraction. The ARC-Heart Failure trial[41] showed that catheter ablation improved quality of life and exercise capacity. The CAMTAF trial[42] showed a sustained improvement in LVEF and improved functional capacity with catheter ablation of persistent AF in patients with HFrEF. The CAMERA MRI trial[43] showed significant improvement in LVEF in patients with HFrEF compared to rate control in patients with persistent AF. However, MacDonald and colleagues[44] did not observe any improvement in LVEF.

The AATAC trial,[45] which compared catheter ablation to amiodarone, showed improved functional capacity, quality of life, reduced hospitalization, and all-cause mortality. There were 2 published trials that compared catheter ablation to best medical therapy, either with rate or rhythm control: the CASTLE-AF trial and the AMICA trial.[46,47] In the CASTLE-AF trial,[46] about 60%–70% of enrolled patients had persistent AF, and 30% were on antiarrhythmic drugs. The CASTLE-AF trial showed a reduction of all-cause mortality and HF hospitalization, as well as sustained improvement in LVEF, improved exercise capacity, and reduction in AF burden. This was the first study prospectively designed and powered to evaluate hard cardiovascular outcomes. However, a small procedural risk should be considered, including a 0.5%–1% risk of stroke and a 1%–2% risk of tamponade. A study looking into the generalizability of data from CASTLE-AF showed that for most patients with AF and HF, catheter ablation was associated with a lower risk of all-cause mortality and HF hospitalization, but the risk reduction was more modest than that observed in CASTLE-AF.[48] The AMICA trial[47] showed no significant difference in improvement in ejection fraction when comparing catheter ablation to best medical therapy. Although ejection fraction improved by 8.8% in the catheter ablation group, which is similar to other trials (7.0% in CASTLE-AF and 9.6% in AATAC-AF), ejection fraction in the medical therapy group also improved by 7.3%—and therefore there was no significant difference in improvement in ejection fraction.

In the CABANA trial,[49] which randomized patients to catheter ablation vs drug therapy, 15% of patients had

**Table 3** Randomized trials of catheter ablation of atrial fibrillation in patients with heart failure and reduced ejection fraction

| Trial | Population | Comparator | Follow-up, months | Primary outcome | Results |
|---|---|---|---|---|---|
| Khan et al, 2008 [PABA-CHF][62] | EF ≤40% Persistent AF (n = 81) | AVN ablation + BiV pacing | 12 | Composite of EF change, 6MWD, MLWHF score | EF improved 8% ± 8% in ablation vs -1% ± 4% in control; $P < .001$. Improved 6MWD and MLWHF score. |
| MacDonald et al, 2011[44] | EF ≤35% Persistent AF (n = 41) | Medical rate control | 6 | EF change by CMR | No difference in EF change in ablation vs control. |
| Jones et al, 2013 [ARC-HF][41] | EF ≤35% Persistent AF (n = 52) | Medical rate control | 12 | Change in peak $VO_2$ consumption | Peak $VO_2$ increased by 2.1 in ablation compared with decrease -0.04 in control; $P = .018$. |
| Hunter et al, 2014 [CAMTAF][42] | EF <50% Persistent AF (n = 50) | Medical rate control | 12 | EF change | EF improved 8.1% (CI, 3.0% to 13.1%) in ablation vs -3.6% (CI, -7.7% to 0.5%) in rate control ($P < .001$). Improved $VO_2$ max and MLWHF score. |
| Di Biase et al, 2016 [AATAC][45] | EF <40% Persistent AF (n = 203) | Amiodarone | 24 | AF recurrence | Greater freedom from AF recurrence in ablation group (70%; 95% CI: 6%–78%) compared to amiodarone (34%; 95% CI: 25%–44%); $P < .001$. |
| Prabhu et al, 2017 [CAMERA-MRI][43] | EF ≤45% Persistent AF (n = 68) | Medical rate control | 6 | EF change | EF improved 18.3% in ablation vs 4.4% in control; $P < .0001$. |
| Marrouche et al, 2018 [CASTLE AF][46] | EF ≤35% Paroxysmal or persistent AF (n = 363) | Medical rate or rhythm control | 60 | Composite of mortality and HF admissions | Lower composite endpoint in ablation group with hazard ratio of 0.62 (95% CI 0.43-0.87; P=0.007). Lower mortality in ablation group (13.4% vs. 25.0%; P=0.01). |
| Kuck et al, 2019 [AMICA][47] Subgroup analysis | EF ≤35% Persistent AF (n = 202) | Medical rate or rhythm control | 12 | EF change | No significant difference in EF change. +8.8% in ablation vs +7.3% in BMT. |
| Packer et al, 2021 [CABANA][50] | Subgroup analysis of HF and AF | Medical rate or rhythm control | 60 | Death, stroke, serious bleeding, or cardiac arrest. | Relative reduction in all-cause mortality of 43% in ablation vs medical therapy in AF and HF. |
| Rillig et al, 2021 [EAST-AFNET 4][52] | Subgroup analysis of HF and AF | Usual care[†] | 60 | Death, stroke, HF hospitalization, ACS | Early rhythm control in HF reduces composite of cardiovascular death, stroke, HF hospitalization, or ACS compared to usual care (5.7 vs. 7.9 per 100 patient-years). |

6MWD = 6-minute walk distance; ACS = acute coronary syndrome; AF = atrial fibrillation; AVN = atrioventricular node; BiV = biventricular; BMT = best medical therapy; CI = confidence interval; CMR = cardiac magnetic resonance; EF = ejection fraction; HF = heart failure; MLWHF = Minnesota Living With Heart Failure.

[†]Trial compared early rhythm control using a combination of antiarrhythmic drugs and atrial fibrillation ablation with usual care, where majority received rate control.

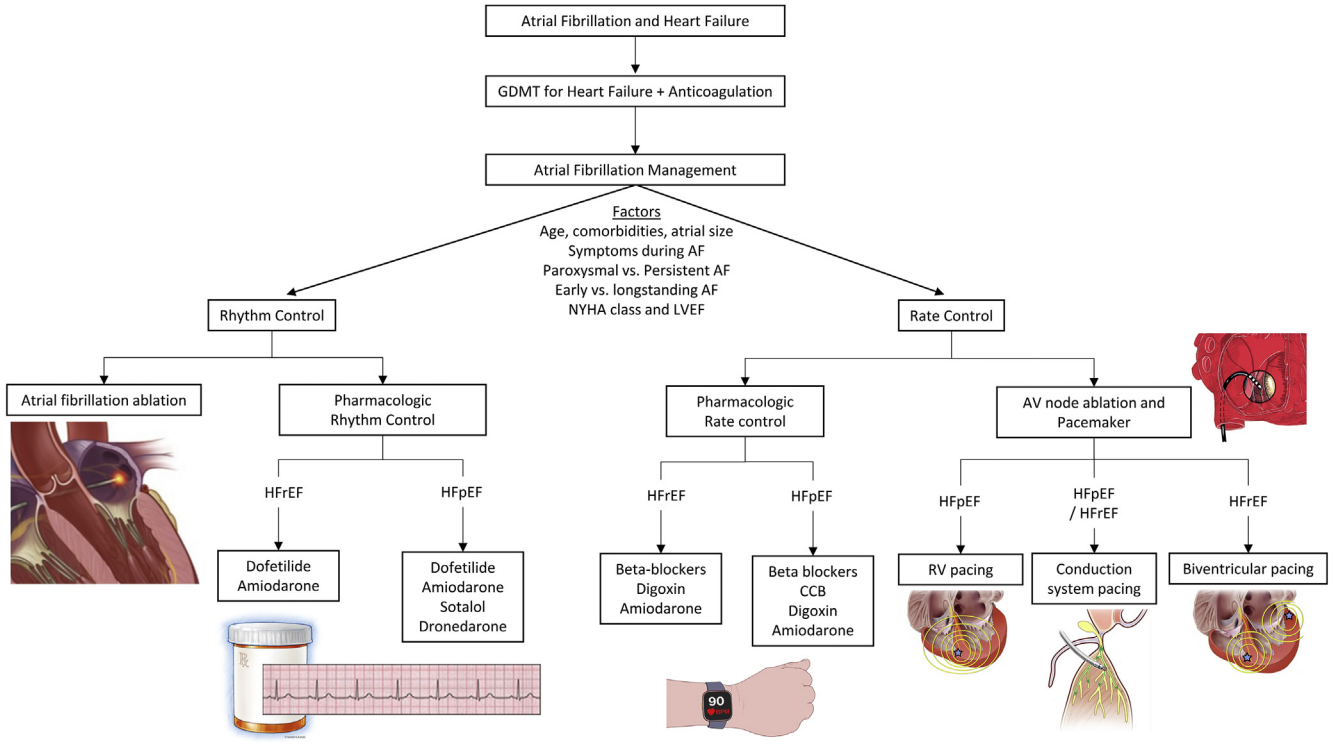


**Figure 1** Management approaches in patients with atrial fibrillation and heart failure. AF = atrial fibrillation; AV = atrioventricular; CCB = calcium channel blockers; GDMT = guideline-directed medical therapy; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; LVEF = left ventricular ejection fraction; RV = right ventricular.

congestive HF. The subgroup analysis from the CABANA trial of patients with HF showed that catheter ablation produced clinically important improvement in survival, freedom from AF recurrence, and quality of life relative to drug therapy.[50] This includes a mix of patients with HFpEF and HFrEF, with 79% of the patients having EF ≥50%. There is inadequate data in patients with reduced ejection fraction to reliably estimate a treatment effect on mortality. In the EAST-AFNET 4 trial,[51] which randomized patients to early rhythm control vs usual care, 28% of patients had HF. The study did find that early rhythm-control therapy was associated with a lower risk of adverse cardiovascular outcomes. The subanalysis of EAST-AFNET 4 showed that early rhythm control in patients with HF led to improved outcomes.[52] The subgroup analysis included a mix of patients with HFPEF and HFrEF, with 56% of patients having EF ≥50%. In the recently presented RAFT-AF trial,[53] which randomized 411 patients to catheter ablation or rate control, there was no difference between the rhythm-control group (via AF ablation) and the rate control group for cardiovascular outcomes at 5 years. The study was terminated early for futility, although secondary outcomes pointed towards improvements in LVEF, 6-minute walk distance, and quality of life in the ablation arm.

Overall, the outcomes of these trials suggest that there may be a benefit for AF ablation in patients with AF, especially in the improvement of LVEF, quality of life, and freedom from AF. Data on the improvement of hard endpoints such as mortality and hospitalization are limited to the CASTLE-AF trial.[46] Notably, in the 2019 AHA/ACC/HRS AF guideline update, catheter ablation for AF in HFrEF was given a class IIB indication.[54] In the 2020 European Society of Cardiology AF guideline,[28] first-line catheter ablation of AF in patients with HFrEF and HFpEF was a class IIA recommendation.

## Interventional management: Atrioventricular node ablation for definitive rate control

AV node ablation with right ventricular pacing is a strategy used for many years and is reasonable to be performed when pharmacological therapy is insufficient or not tolerated.[25] In a meta-analysis of a total of 1181 patients to evaluate clinical outcomes and survival after AV node ablation and right ventricular pacing in patients with medically refractory AF, the study demonstrated significant improvements in a range of measures, including quality of life, exercise duration, and healthcare use.[55] The APAF-CRT trial[56] showed that cardiac resynchronization therapy (CRT) and AV node ablation is superior to pharmacological rate control in patients with symptomatic permanent AF and narrow QRS.

Based on the 2012 American College of Cardiology Foundation/American Heart Association/Heart Rhythm Society Focused Update of the 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities,[57] CRT can be beneficial in patients with HFrEF with LVEF ≤35% on

guideline-directed medical therapy undergoing AV node ablation, as they would require 100% ventricular pacing. In patients with HFrEF with LVEF between 36% and 50% undergoing AV node ablation, it would also be reasonable to consider biventricular pacing or conduction system pacing.[58] In the BLOCK HF trial, patients with AV block and reduced ejection fraction of <50% had improved outcomes when undergoing biventricular pacing compared to right ventricular–only pacing.[59] In a systematic review of nonrandomized data, AV node ablation in patients with CRT and AF was associated with a reduction in all-cause mortality, cardiovascular mortality, and improvement in New York Heart Association functional class compared with medical therapy.[60] Furthermore, a randomized trial showed CRT to be superior to right apical pacing in reducing HF events in patients with permanent AF undergoing AV nodal ablation.[61]

The downside of the AV node ablation and pacing strategy is that there is no restoration of sinus rhythm. The PABA-CHF trial[62] highlights the benefits of normal sinus rhythm, especially when comparing the ablate-and-pace approach vs catheter ablation. In the PABA-CHF trial, pulmonary vein isolation was superior, compared to the AV node ablation with biventricular pacing, in improving ejection fraction, exercise capacity, and patient-reported quality of life.

## General management approach

In patients with AF and HF, the primary management is optimizing HF management with guideline-directed medical therapy and device therapy such as implantable cardioverter-defibrillator with or without CRT based on guideline recommendations. Following that, the decision to rhythm control over rate control, or vice versa, requires consideration of multiple factors (Figure 1). The primary factor is the presence of symptoms of AF, especially despite adequate rate control. However, it may be hard to identify symptoms of AF in a patient with HF, as there are overlapping symptoms of AF and HF, such as shortness of breath and decreased exercise capacity. Practically, it would be reasonable to consider a patient with HF as having symptomatic AF when there is an improvement of symptoms in normal sinus rhythm compared to when in AF. This may require an attempt at cardioversion and assessment of symptoms following cardioversion. In some other patients, a correlation can be established where the onset of AF precedes HF decompensation. In those patients, consideration can be made for rhythm control strategy.

The other factor to consider is the likelihood of achieving successful rhythm control with the lowest possible risk to the patient. This includes considering the patient's age, comorbidities, renal function, left atrial size, type and duration of AF, type and duration of AF, NYHA class, and ejection fraction.[63] The third factor is shared decision-making with the patient. This involves a detailed discussion about the pros and cons of various management options. Patients should be aware that more than 1 AF ablation procedure may be needed in up to a third of all ablation patients with HF. As for the decision to pursue antiarrhythmics, there should also be a discussion on the short- and long-term side effects of antiarrhythmics.

## Future directions

There has been a growing body of evidence on catheter ablation of AF in patients with HFrEF. The ongoing RACE-8-HF trial (ClinicalTrials.gov: NCT04342832) compares cryoablation with standard medical therapy in patients with paroxysmal or persistent AF and HFrEF (LVEF <40%). However, there is still limited data on optimal management of AF in patients with preserved ejection fraction. The ongoing TAP-CHF trial (ClinicalTrials.gov: NCT04160000) will evaluate catheter ablation vs medical therapy in patients with AF and preserved ejection fraction. There are other knowledge gaps that may be potential areas for future research. More studies are needed to look into improving patient selection and timing of ablative therapy in HFrEF. Patients with more advanced HF, lower LVEF, and larger atrial size may benefit less from catheter ablation.[64] The role of the hybrid convergent procedure compared to standard catheter ablation in patients with AF and HF also will need to be studied.

## Conclusion

The treatment of AF in patients with HF begins with optimal guideline-directed management of HF. Randomized trials in patients with AF and HF have shown that rhythm control with catheter ablation via pulmonary vein isolation can improve symptoms, increase quality of life, and improve LVEF. There is increasing data showing improvement of hard endpoints such as mortality and HF hospitalization with rhythm control, especially with early rhythm control and with catheter ablation via pulmonary vein isolation. Catheter ablation should still be used selectively, taking into consideration the patient's comorbidities and risk of complications. More studies are needed to improve the efficacy of AF ablation in HF patients.

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## Authorship

All authors attest they meet the current ICMJE criteria for authorship.

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