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Invasive therapies for patients with concomitant heart failure and atrial fibrillation

Wei Wei[1,2] · Michael Shehata[3] · Xunzhang Wang[1,3] · Fang Rao[1,2,4] · Xianzhan Zhan[1,2] · Huiming Guo[1] · Xianhong Fang[1,2] · Hongtao Liao[1,2] · Jian Liu[1] · Hai Deng[1,2] · Yang Liu[1,2] · Yumei Xue[1,2] · Shulin Wu[1,2]

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Abstract
Atrial fibrillation (AF) and heart failure (HF) are two clinical entities that can present either separately or concurrently. One entity can lead to the other and vice versa as AF can not only be the underlying etiology of HF but also exacerbate HF due to other cardiac diseases. Besides prevention of cerebral and systemic embolism and elimination of AF-related symptoms, restoration of sinus rhythm for AF patients helps to avoid or reduce HF, irrespective of their underlying heart disease. Successful rates of medical therapy for AF are low in persistent AF, and much lower in long-standing AF, while invasive procedures for AF yield promising results. In this review, the authors evaluate the value of invasive therapies for HF patients complicated with non-valvular AF. We examine this clinical problem by interpreting the relationships between these two entities: the mechanism of tachycardia-induced cardiomyopathy (TIC), past opinions about rhythm control and rate control of AF, discrimination of HF-related AF and AF-induced HF, how to identify the AF patients that could benefit from invasive therapies, and how to select invasive therapies for different AF patients and peri-operative treatments.

Keywords Heart failure · Atrial fibrillation · Invasive therapy · Ablation · Medication

Introduction

Heart failure (HF) is often the end-stage of many cardiac conditions. Atrial fibrillation (AF) is both an exacerbating factor for HF and also often a result of HF. Both non-valvular AF and valvular AF can cause tachycardia-induced cardiomyopathy (TIC) which leads to HF. The problem is the certainty of the degree of attribution to the valve versus other clinical situations. Cardiac decompensation elicited by TIC can become a major health hazard caused by AF due to thrombo-embolism, and either rate or rhythm control is essential to treat decompensated TIC [1, 2]. In a word, AF and HF interact as both cause and effect. According to evidence from the recently published clinical trial, Catheter Ablation versus Standard Conventional Therapy in Patients with Left Ventricular Dysfunction and Atrial Fibrillation (CASTLE-AF), AF patients with HF benefited from catheter ablation of AF, including reduced all-cause mortality, reduced hospitalization for worsening HF, and death from cardiovascular causes, compared with medical therapy [3]. How to know whether a patient with HF and AF suffers from TIC or primary cardiomyopathy, whether invasive therapies will benefit the patient, how to select patients with AF and HF for invasive therapies, and how to select proper invasive therapies are important issues that providers are challenged with regularity.

Wei Wei and Michael Shehata contributed equally to this work.

✉ Yumei Xue
xymgdci@163.com

✉ Shulin Wu
dr_shulinwu@126.com

[1] Department of Cardiology, Guangdong Cardiovascular Institute, Guangzhou, People's Republic of China

[2] Guangdong Key Laboratory of Clinical Pharmacology, Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, No. 96, Dongchuan Road, Guangzhou 510080, People's Republic of China

[3] Heart Institute Los Angeles, Cedars Sinai Medical Center, 8700 Beverly Boulevard, Los Angeles, CA 90048, USA

[4] Research Center of Medical Sciences, Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, Guangzhou, People's Republic of China

AF causing HF: a form of TIC

In patients with HF, there is evidence to support structural, neurohormonal, and electrical atrial remodeling—each of them promotes the development of AF [4–7]. The

development of AF in HF appears to be a multifactorial process, including atrial enlargement, conduction heterogeneity from intra-atrial fibrosis, ion channel dysregulation, and autonomic remodeling [8–11]. This causative relationship also works in the opposite direction: AF can induce electrical and hemodynamic deterioration and can cause TIC, resulting in HF. Mediated by a rapid ventricular response or altered diastolic ventricular function, AF can cause HF symptoms in patients with preserved left ventricular systolic function as well. AF prevalence increases as HF severity worsens.

TIC is defined as a condition characterized by atrial or ventricular myocardial dysfunction resulting solely from increased atrial or ventricular rates [12]. TIC can be caused by a variety of tachyarrhythmias, including AF, atrial flutter, incessant supraventricular tachycardia, ventricular tachycardia, and very frequent premature ventricular beats, but most commonly is a consequence of AF [13, 14]. Restoration of sinus rhythm, control of the ventricular response, or decrease in the frequency of premature contractions all result in an improvement in left ventricular function and clinical HF. TIC is generally a reversible cardiomyopathy if the tachycardia can be treated effectively, either with medications, surgery, or catheter ablation. TIC can also manifest in patients with baseline left ventricular dysfunction from underlying structural heart diseases that develop a worsening of their myocardial dysfunction in the setting of prolonged tachycardia, which can be reversed with control of the tachycardia. The diagnosis is usually made after demonstrating recovery of left ventricular function with normalization of heart rate in the absence of other identifiable etiologies [15]. Many reports have been published documenting cases of complete resolution of congestive HF after cardioversion of AF [16–18]. The most common presentation is dilated cardiomyopathy with AF. Given that this diagnosis represents a potentially reversible cause of HF, its recognition is critically important because it informs the treatment strategy which can be quite effective.

Myocardial changes in animal models of TIC by rapid ventricular pacing have demonstrated four aspects: hemodynamic changes, structural changes, cellular changes, and neurohormonal changes (see Table 1 [15]). With respect to hemodynamic changes, TIC can cause depressed left ventricular function, elevated left ventricular filling pressures, impaired ventricular contractile function, reduced cardiac output, elevated systemic vascular resistance, increased left ventricular wall stress, left ventricular diastolic dysfunction, and mitral regurgitation [19]. Regarding structural changes, TIC can cause dilation of left ventricular cavity, subendocardial fibrosis, reduced left ventricular wall thickness, and reduced myocardial blood flow. Cellular changes observed in TIC include myocyte elongation, increased oxidative stress, myocyte hypertrophy, extracellular matrix/basement membrane disruption, disruption of myofibril alignment, reduced myocardial energy stores, mitochondrial dysfunction, downregulation of β-adrenergic receptors, abnormal calcium handling, and increased myocyte apoptosis. Neurohormonal change in TIC include changes in the secretion of renin, aldosterone, angiotensin II, epinephrine, norepinephrine, atrial natriuretic peptide, brain natriuretic peptide, and endothelin-1 [20–23].

A former study assessing the time of improvement in left ventricular function following cardioversion of AF demonstrated that atrial systolic function improved 1 week after restoration of sinus rhythm, whereas left ventricular ejection fraction (LVEF) and peak oxygen consumption lagged behind and did not show improvement until 1 month following cardioversion. These results suggested the presence of an underlying ventricular myopathy causing HF rather than only the loss of atrial contractile function and atrioventricular synchrony [24].

Discrepancies in rhythm control and rate control of AF

In the EuroObservational Research Programme HF Long-Term Registry, AF was documented in 44% of patients hospitalized for acute HF and in 37.6% of patients with chronic HF [25]. Past research including Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM), AF-CHF, and RAte Control versus Electrical cardioversion (RACE) mainly focused on comparison between medical rhythm control and rate control. The main conclusions were neutral—medical rhythm control brought no benefit to HF and AF patients over rate control. They failed to demonstrate any benefit from successful sinus rhythm maintenance, and usage of antiarrhythmic drugs was still associated with increased mortality [26–28]. Such results had a profound influence on the treatment strategy of AF over a long period. Later analyses also found that antiarrhythmic drugs have an even lower success rate with side effects that cannot be neglected [27].

However, a major limitation of such trials is that the study populations consisted of patients with both paroxysmal and persistent AF. In the AFFIRM trial, 54% of patients were in sinus rhythm at enrollment and 35% of patients randomized to rate control arm were in sinus rhythm after 5 years. This may reduce the power of any study comparing rhythm control versus rate control of AF as this significant spontaneous "rhythm control" occurring in patients assigned to rate control arm may mask any anticipated benefit achieved by pharmacological rhythm control. Moreover, only 63% of patients randomized to the rhythm control arm in AFFIRM maintained sinus rhythm after 5-year follow-up with a high degree of crossover between groups (14.9% in the rate control group and 37.5% in the rhythm control group at 5 years). A post hoc analysis of the AFFIRM study showed that restoration and maintenance of sinus rhythm were associated with a lower mortality and that the application of antiarrhythmic drugs was associated with increased mortality [29]. Also, maintenance of sinus rhythm in patients with EF > 35% was associated with better

Table 1 Myocardial changes in animal models of TIC

Aspects	Items
Hemodynamic changes	Depressed left ventricular function
	Elevated left ventricular filling pressures
	Impaired ventricular contractile function
	Reduced cardiac output
	Elevated systemic vascular resistance
	Increased left ventricular wall stress
	Left ventricular diastolic dysfunction
	Mitral regurgitation
Structural changes	Left ventricular cavity dilation
	Subendocardial fibrosis
	Normal or reduced left ventricular wall thickness
	Reduced myocardial blood flow
Cellular changes	Myocyte elongation
	Increased oxidative stress
	Myocyte hypertrophy
	Extracellular matrix/basement membrane disruption
	Myofibril alignment disruption
	Reduced myocardial energy stores
	Mitochondrial dysfunction
	Down regulation of beta adrenergic receptors
	Abnormal calcium handling
	Increased myocyte apoptosis
Neurohormonal changes	Increased renin, aldosterone, angiotensin II, epinephrine, norepinephrine, ANP, BNP, ET-1

ANP atrial natriuretic peptide, *BNP* brain natriuretic peptide, *ET-1* endothelin-1

survival as reflected in the DIAMOND trial [30]. In the RACE substudy, they also observed a higher mortality rate and increased major bleeding with rate control over rhythm control [31]. The CAFÉ-II study demonstrated that the patients assigned to rhythm control not only improved quality of life but also improved LV function and had lower NT-pro BNP levels at 1 year [32].

With the development of catheter ablation of AF, satisfactory effects in rhythm control were observed in persistent AF and even long-standing persistent AF, besides paroxysmal AF compared with medical therapy. In contrast to the AFFIRM study, sinus rhythm maintenance now showed benefit over rate control, with respect to both HF and thrombo-embolism. Compared with medical therapy controlling ventricular rate, catheter ablation significantly improved cardiac function of patients with AF and cardiac insufficiency [3, 32–37]. However, the risks of catheter ablation are higher in patients with HF than those with normal heart function and render it clinically important to identify which patients would substantially benefit from this invasive therapy. Complications include pericardial tamponade, stroke, pulmonary vein stenosis, phrenic nerve injury, vascular complications, exacerbation of HF, esophageal injury, and very rarely atrio-esophageal fistula. A worldwide survey on more than 16,000 patients undergoing mainly radiofrequency ablation reported a 4.5% risk of major complications with a 0.15% mortality rate [38].

How to know whether AF is the main cause of HF in an individual patient?

Clarifying whether AF or HF appears earlier helps to discriminate whether TIC is the essential problem or AF is only a concomitant issue to HF. The diagnosis is also directly related to the patient's prognosis. If the patient suffered from TIC caused by AF, restoration of sinus rhythm can significantly reverse HF. Otherwise, restoration of sinus rhythm may only be of limited benefit for reversing HF. It is often the fact that when patients seek medical care, congestive HF co-exists with AF and it may be difficult to find out which condition appears first. Whether AF is the cause or result of congestive HF is hard to tell only by symptoms if precise past medical records are not available. It becomes essential to know the patient's medical history before making the diagnosis of TIC. If persistent AF appeared earlier than symptoms of HF, AF may be the main reason for development of HF. Otherwise HF is likely a risk factor for AF. Clearly, it may be difficult to discriminate between the above two situations because symptoms of the

two are not specific to each other. Additionally, certain risk factors of HF besides AF, such as ischemic cardiomyopathy, valvular heart disease, poorly controlled hyperthyroidism, significant structural heart disease, chemotherapy for malignancies, and so on can also be found in some HF patients; HF is also a probable reason for AF. Besides echocardiography, other imaging screening methods including cardiac computed tomography (CT), magnetic resonance imaging (MRI), positive electron tomography (PET), and emission computed tomography (ECT) can also detect structural heart disease and primary myocardial lesions by showing myocardial dysplasia or abnormal myocardial metabolism. These ancillary examinations help to detect other etiologies of HF.

Since cardiac decompensation elicited by TIC caused by AF can lead to major adverse events, either rate control or rhythm control is essential to treat decompensated TIC [39–41]. Theoretically, TIC can be diagnosed for the first time after observing improvement in ventricular function following rate or rhythm control. Heart rate control during AF without sinus restoration may result in an incomplete cure of TIC, suggesting the advantages of rhythm control with ablation in patients with TIC. Even if the patient does not suffer from TIC, but congestive HF complicated with AF, restoration of sinus rhythm can still help to improve prognosis [34].

Which HF patients will benefit from invasive therapies for AF?

AF and HF share several common risk factors and usually coexist [42–46]. It is likely that patients with HF who go on to develop AF have a worse prognosis than those who maintain sinus rhythm [47], though this has been controversial [48]. It is now generally accepted that the onset of AF in patients with chronic HF complicates management greatly and often leads to repeat hospitalizations because of frequent acute decompensation. Of patients admitted to the hospital with acute decompensated HF, 20–35% will be in AF. In one-third of these cases, AF will be of recent onset [48]. The 2016 European Society of Cardiology guidelines on the management of AF states that the indications for catheter ablation in HF patients with reduced ejection fraction (HFrEF) should be carefully balanced and recommended that procedures are performed in experienced centers [49]. The guidelines also recognized that AF ablation could be more complex in this patient cohort compared with those without HF. However, there is no clear consensus on which patients with HF should be offered catheter ablation or the optimal ablation strategy in this setting.

Jedrzej Kosiuk and colleagues studied 73 patients with an implanted ICD due to ischemic cardiomyopathy ($n = 30$) or dilated cardiomyopathy ($n = 43$). They reported that catheter ablation of AF was associated with reduction of inappropriate and appropriate ICD therapies and improvement of LVEF [50]. A Japanese group did a retrospective study of AF in patients with severely impaired left ventricular systolic function (LVEF < 35%) due to multiple structural heart diseases including dilated cardiomyopathy, hypertensive cardiomyopathy, and coronary arterial disease and found that catheter ablation improved these patients' LVEF and NYHA class and decreased hospitalizations for HF. A repeat ablation procedure and antiarrhythmic drugs were often necessary to obtain a favorable outcome. The improvement in the HF status depended on the cardiac rhythm status after the procedure [51]. Haisaguerre's group, which first reported pulmonary vein isolation as a mainstay of AF ablation, also recommended AF ablation for patients with HF due to structural heart disease and drug refractory AF [52].

Prabhu Sandeep and colleagues compared the AF catheter ablation results of known heart diseases (including hypertrophic cardiomyopathy, valvular diseases, ischemic cardiomyopathy, totaling 24 patients) and idiopathic dilated cardiomyopathy (77 patients). They found that the latter group had much better long-term outcome than the former group (> 15% LVEF improvement post-ablation in two groups, 6% vs. 94%) [53]. And LVEF improvement was not statistically significant post-ablation in the group with known heart diseases ($P = 0.25$). Thus, they concluded that the presence of known heart diseases contributing to left ventricular dysfunction was associated with limited maintenance of sinus rhythm post-ablation, functional improvement, and improvement in LVEF. Conversely, the absence of known heart diseases identified a HF population who received greater benefit from catheter ablation for AF and should be strongly considered. Nedios and colleagues also reported that the presence of structural heart disease was not a significant multivariate predictor of LVEF improvement post-ablation, particularly in those with regional wall motion abnormalities [54].

Besides systolic HF, AF ablation also improves diastolic HF. Hideharu Okamatsu and colleagues enrolled 22 hypertrophic cardiomyopathy patients with paroxysmal ($n = 5$; 23%) or persistent ($n = 17$; 77%) AF. Left ventricular (LV) diastolic function was evaluated according to the ratio of the mitral inflow early filling velocity to the velocity of the early medial mitral annular ascent (E/e′) measured on pulsed wave and tissue Doppler assessments. During a follow-up of 21 ± 12 months, sinus rhythm was maintained in 13 patients (59%). E/e′ was significantly higher in the patients with AF recurrence than in those without (18 ± 7 vs. 11 ± 3; $P < 0.01$). The prevalence of AF recurrence was significantly higher in patients with E/e′ ≥ 15 ($n = 6$) than in those with E/e′ < 15. They concluded that LV diastolic dysfunction was linked to the possibility of rhythm control after RFCA in the patients with HCM and AF. Patients with mild or moderate LV diastolic dysfunction (E/e′ < 15) might be good candidates for RFCA in those with HCM and AF [55].

In the CASTLE-AF trial, pursuing rhythm control with catheter ablation provided significant benefit. In the ablation group, 63% of patients were in sinus rhythm at 60 months versus 22% in the medical therapy group, which suggests that maintenance of sinus rhythm is beneficial when achieved without the use of antiarrhythmic drugs [3].

Based on the above reports, we know that AF ablation in the setting of HF due to many underlying cardiac diseases is beneficial, but we do not yet know the lower threshold for AF ablation. Arora and colleagues utilized American National Readmission Data to follow-up 1,128,372 AF patients from Jan 2010 to Sept 2014, of which 37,360 (3.3%) underwent catheter ablation. They found that diabetes, chronic obstructive pulmonary disease (COPD), or alcohol abuse was significantly relevant to 90-day readmission [56]. The authors consider that AF ablation should be avoided in the following situations: (1) Severe HF patients that cannot tolerate anesthesia of surgical ablation or saline irrigation of catheter ablation or a prolonged supine position during catheter ablation; (2) AF related to irreversible primary heart diseases with end-stage HF, such as severe pulmonary hypertension, severe COPD, restrictive myocardiopathy, severe congenital heart diseases, hypertrophic obstructive cardiomyopathy, and severe valvular stenosis due to rheumatic heart disease that are unamendable to correction; (3) significant structural heart diseases awaiting corrective cardiac surgeries such as valvular heart disease, congenital heart disease, and coronary arterial disease requiring CABG (these patients can undergo simultaneous open MAZE ablation during surgery); (4) acute phase of myocardial injuries, such as acute/subacute myocarditis, uncontrolled infectious endocarditis, and acute/subacute myocardial infarction; (5) concomitant unstable status or end-stage disease of other organ systems, such as uncontrolled hyperthyroidism and end-stage renal failure/liver failure/hematonosis. For other patients with AF and HF, one can cautiously consider ablation for severe refractory symptoms, keeping in mind potential complications and health care resources.

How to select invasive therapies for AF complicated by HF?

There are mainly four invasive therapies for AF: catheter ablation, surgical ablation, combined surgical and catheter ablation, and atrio-ventricular (A-V) node ablation with permanent pacing.

Catheter ablation based on pulmonary vein isolation has long been established. The detailed additional ablation targets for persistent AF include linear ablation of the left atrial roof, linear ablation of the mitral isthmus, linear ablation of the tricuspid isthmus [57], ablation on the endocardial aspect of the coronary sinus, ablation within the coronary sinus, ablation of complex fractionated atrial electrograms (CFAE), isolation of superior vena cava, box isolation of the left atrial posterior wall [58], isolation of the left atrial appendage [59], and ablation of triggers including the coronary sinus orifice and ligament of Marshall. The above sites are selectively targeted according to individual differences.

Based on the fact that surgical ablation produces deeper and transmural atrial lesions, trans-thoracoscopic surgical ablation of long-standing persistent AF has been demonstrated to yield better maintenance of sinus rhythm than catheter ablation [60]. In addition to a potentially more durable lesion set, other advantages of a surgical approach include access to epicardial structures such as the ligament of Marshall and ganglionated plexi, exclusion of the left atrial appendage, and avoidance of damaging collateral structures, such as the phrenic nerves and esophagus. Some randomized prospective trials have compared a thoracoscopic surgical approach to percutaneous endocardial catheter ablation for the treatment of patients with paroxysmal and non-paroxysmal AF, most of whom had failed an initial catheter ablation [61, 62]. Their results showed that surgical ablation was superior to catheter ablation in these patients. A meta-analysis of these and other observational studies demonstrated a significant improvement of arrhythmia-free survival for the surgical procedure (78.4 vs. 53%; RR 1.54; 95% CI 1.50–2.14; $I^2 = 0\%$; $P < 0.001$), with a clearer benefit for patients with persistent AF [63]. Complications were three times more frequent in the surgical group, mostly due to pneumothorax and pleural effusion. Surgical procedures can also provide a reasonable outcome for patients with large atria and long-standing persistent AF [64, 65].

Hybrid ablation combining surgical and catheter ablation (one-stage or two-stage) may lead to better outcomes on long-standing AF than surgical ablation alone because catheter ablation can make supplementary lesions to cover any gaps following surgical ablation [66]. But the full scope of the synergistic effects of epi- and endocardial ablation can only be realized when the endocardial stage is delayed long enough (at least several weeks apart in a two-stage procedure) to ensure that the epicardial lesions are fully healed with fibrous tissue and that neither edema nor mechanical trauma is contributing to electrical conduction block. The maturation of epicardial "burns" allows easier identification of non-isolated substrate boundaries and of target points [67]. Less bleeding, lower infection risks, and shorter general anesthesia time also favors the two-stage approach [68]. The interval between surgical ablation and catheter ablation is usually 1 to 4 months [69]. One potential risk factor for AF patients with HF to undergo surgical ablation is the risk of general anesthesia which needs to be individualized.

If the patient's atrial condition is relatively poor with a very long history of long-standing persistent AF or diffuse lesions within the atria due to previous cardiac surgeries/ablations and importantly if almost no "f" waves can be identified on each

lead of the surface ECG (V1 showing the most prominent f waves usually), the patient may be a non-responder to all the above ablation strategies. If the ventricular rate of such patients suffering from permanent AF cannot be properly controlled by medicine, they may be better candidates for A-V node ablation and permanent ventricular pacing or cardiac resynchronization therapy (CRT) [70]. Due to the development of physiological pacing concepts nowadays, His bundle pacing or proximal left bundle pacing may be preferred over bi-ventricular pacing, activating sequential conduction of His-Purkinje system with the lowest pacing threshold [71]. His bundle or proximal left bundle pacing can avoid heart failure due to desynchronized ventricular activation caused by ventricular pacing and may prolong battery life of pacemakers.

Improvement of heart function and general physical status before and after procedures

Patients' ventricular function should be improved as much as possible by optimal medical treatment according to the latest guidelines before undergoing any procedure [72], especially for patients with acute HF. In patients presenting acutely with AF and HF, the guidelines recommend focusing on normalizing fluid balance, aiming for an initial heart rate < 110 bpm, application of anticoagulants, inhibition of the renin–angiotensin–aldosterone system, and early consideration of rhythm control [59]. Catheter ablation requires hours in a supine position and saline infusion due to the application of irrigated ablation catheters. General anesthesia also requires relatively compensated cardiac function. Diuretics are usually important since they not only improve heart function by reducing circulating fluid but also allow procedures by providing tolerance for saline irrigation. Application of irrigated catheters with lower irrigation flow rates can also avoid exacerbating HF. Reducing fluid load can also decrease bi-atrial size which are closely related to post-procedure recurrence of AF [73]. But whether reducing atrial size pre-procedurely can lead to immediate higher success rates of sinus rhythm restoration and long-term maintenance needs further studies. Functions of other organ systems also need to be optimized, such as renal failure due to reduced cardiac output (cardio-renal syndrome [74]). Sometimes, dialysis is required if the patient's cardiac function or renal function deteriorates without proper response to medical therapy. Basic medical treatments also include angiotensin converting enzyme inhibitors (ACEI)/ angiotensin II receptor blockers (ARB)/ angiotensin receptor-neprilysin inhibitors (ARNI), β-receptor blockers, spironolactone, natriuretic peptides, calcium sensitizers, phosphodiesterase inhibitors, cardiotonics, and others as suggested by present HF guidelines if the patient's physical status can endure, before and after procedures [72]. Other pre-ablation preparation including full anticoagulation, imaging screening for intra-atrial thrombosis, and so on are the same as in ordinary AF ablation.

Since AF is a chronic disease associated with multiple risk factors, invasive therapies are far from enough. Lifetime treatment and control of AF risk factors and general physical status are important. Hypertension is closely related to development of AF [73]. Hyperuricemia has also been demonstrated to be related to development of AF and AF recurrence after catheter ablation and also increases left atrial spontaneous echo contrast and left atrial stasis [75–78]. Besides the heart, long-term care of other organ systems is also important to avoid AF recurrence.

Conclusions

HF patients presenting as idiopathic dilated cardiomyopathy with AF can benefit from AF ablation (either catheter ablation or surgical ablation) with LVEF and NYHA heart function improvement and improved long-term prognosis. Further studies of larger sample sizes are necessary to demonstrate whether patients with known structural heart disease that are not cardiac surgical candidates would still benefit from AF ablation.

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