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Review
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# The electrophysiologist's conundrum: catheter ablation of atrial fibrillation in hypertensive patients

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## Abstract

Hypertension (HTN) is a well-established major cardiovascular risk factor, with a growing prevalence in the population and underlies several other entities. It has been recognized as an independent predictor of atrial fibrillation (AF). HTN and AF, in conjunction, contribute to increased stroke risk. The emergence of catheter ablation in AF revolutionized the management of AF; however, recurrence rates have shadowed its merits. It was initially hypothesized that recurrence rates relate strictly to an electrophysiological substrate. However, further research revealed that several factors, including HTN, perpetuate an atrial substrate, especially when uncontrolled. In turn, this prompted the scientific community to advocate for rigorous evaluation before and after transcatheter ablation and aggressive control of blood pressure to ensure a higher rate of success and better long-term management.

**Keywords:** atrial fibrillation, hypertension, risk factors, catheter ablation, oral anticoagulation.

## Introduction

Hypertension (HTN) and atrial fibrillation (AF) are two important public health priorities. Their prevalence is increasing worldwide, and the two conditions often coexist in the same patient. Hypertension is the most common worldwide risk factor associated with the development of atrial fibrillation and contributes to the arrhythmogenic substrate.

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The conventional treatment approach to AF beyond anticoagulation includes either restoration and maintenance of the sinus rhythm or ventricular rate control [1]. AF catheter ablation is a well-established treatment for preventing AF recurrences [2, 3], the main clinical benefit being the reduction of arrhythmia-related symptoms and improved quality of life. Studies have shown that hypertension predicts AF recurrence after AF ablation; however, it is not well established whether, besides aggressive blood pressure (BP) control, other methods such as modulation of the autonomic system or inhibition of the renin-angiotensin-aldosterone system (RAAS) are useful in reducing AF recurrence in HTN patients undergoing AF ablation [4].

This review discusses specific epidemiological and periprocedural issues related to AF catheter ablation in HTN patients.

## Epidemiology and definitions

AF affects approximately two million patients in the United States of America and an equal number in Europe and increases morbidity and mortality in affected patients and populations worldwide [5]. The prevalence of AF is higher in men and increases with age. It is associated with hemodynamic impairment, reduced quality of life, and a high risk of thromboembolism.

Due to its high prevalence in the general population, HTN is the most significant population-attributable risk for AF; it has been estimated to be responsible for 14% of all AF cases [6]. HTN was present in >70% of AF patients in epidemiological studies [7, 8] and recent AF real-world registries [9–11] and in 49–90% of patients in randomized AF trials [12, 13]. HTN is the most potent predictor of mortality in both high and low-income countries [14].

## Pathogenesis of atrial fibrillation in hypertensive patients

Hemodynamic changes, neuroendocrine factors, atrial and ventricular structural remodeling (*i.e.*, myocardial fibrosis), and a proarrhythmogenic electrophysiologic phenotype of a hypertrophied left ventricle (LV) all contribute to the complex pathophysiology of arrhythmogenesis in hypertension [15].

Despite the well-established epidemiological association between HTN and AF, the pathogenetic mechanisms explaining the higher propensity of HTN patients to develop AF are still incompletely known [16]. It is unclear whether the increased risk of AF with BP is linear or based on a threshold value.

Several different mechanisms may be involved in the genesis of AF in HTN patients. A central role is expressed by the so-called atrial cardiomyopathy, defined as a complex of structural, architectural, contractile, and electrophysiological changes affecting the atria with the potential to produce clinically relevant manifestations [17], which may be induced by predominantly hemodynamic and non-hemodynamic mechanisms. The predominantly hemodynamic mechanisms include increased left ventricular (LV) wall thickness and/or stiffness and impaired LV diastolic function associated with hypertension. These processes may lead to a rise in left atrial (LA) pressure and stretch, with subsequent LA remodeling and dysfunction, ultimately predisposing to AF and stroke.

Several animal models have been developed to investigate the pathophysiological mechanisms underlying the greater propensity of hearts of humans with hypertension to develop AF. In general, experimental hypertension rapidly induced hypertrophy, fibrosis and inflammation of the LA [16, 18–21].

The most important atrial changes include the proliferation of fibroblasts, alterations of the extracellular matrix, and hypertrophy of myocytes [22]. The resulting disorders of interconnections between muscle bundles may lead to shortening of LA refractoriness, unidirectional blocks, and re-entry phenomena [22]. These processes may initiate AF eventually, triggered by ectopic stimuli originating from pulmonary veins or other sites [16]. Over time, tissue remodeling promotes and maintains atrial fibrillation by changing the fundamental properties of the atria [23].

Mechanical overload due to high BP may induce an abnormal expression of ion channels and/or junctional complexes, such as connexin 40 and connexin 43, which can enhance myocardium vulnerability by triggering focal ectopic and re-entry activity [16, 24]. Altered Ca2+ handling by the atrial myocytes has been identified as another mechanism potentially able to trigger AF. Pluteanu *et al.* demonstrated the existence of subcellular alterations in Ca2+ handling in SHRs, which were associated with an increased propensity of atrial myocytes to develop frequency-dependent, arrhythmogenic Ca2+ alternans [25].

The RAAS is also involved in the pathogenesis of atrial fibrillation. Several potential mechanisms have been described: the proliferation of fibroblasts, extracellular matrix, and hypertrophy of myocytes. Angiotensin II may also modulate some ion currents in myocytes, including the L and T type inward Ca2+ current [26, 27] and the potassium current [28], although further studies are required. In isolated cardiac myocytes from rats and mice, angiotensin II activated cAMP-dependent protein kinase A and Ca2+/calmodulin kinase II, representing a well-established proarrhythmogenic pathway in the setting of increased angiotensin II stimulation [29].

The complex relations between aldosterone and AF in hypertension have been recently reviewed. An impressive 12-fold higher risk of AF has been reported in patients with primary hyperaldosteronism when compared with patients with essential hypertension [30], which is in line with the known effect of aldosterone on cardiac inflammation, fibrosis, and hypertrophy [31–33].

The autonomic nervous system plays an essential part in the initiation and perpetuation of atrial fibrillation by changing the electrophysiological proprieties of the atrium. Increased central sympathetic outflow and efferent cardiac sympathetic nerve stimulation can promote the development of atrial fibrillation. Cardiac autonomic innervation is constantly remodeling, especially during disease states. Several studies showed that in cardiac diseases, neural remodeling might occur throughout the heart, potentially increasing nerve activities and, in this way, promoting the development of atrial arrhythmias [34].

We reiterate the fact that maintaining sinus rhythm (with catheter ablation of atrial fibrillation being the most effective strategy) on the one hand, in addition to adequate control of hypertension, on the other hand, may offer enhanced reverse-remodeling of both left heart chambers by addressing multiple mechanisms. This is often revealed early by cardiac imaging as LV/LA decreased dimensions and/or LA/LV improved contractility and filling. However, microscopic alterations reverse slowly or are irreversible [35].

## Management

Adequate management of hypertension is essential for AF prevention, rhythm control, heart failure, and stroke prevention. According to the 2018 ESC/ESH Guidelines, hypertension is defined as office SBP values ≥140 mmHg and/or diastolic BP (DBP) values ≥90 mmHg [36]. As a relevant remark, several studies observed that both pre-hypertension (SBP 120–129 mmHg) and hypertension confer a 1.8- and 2.6-fold increased risk of incident AF, respectively [37].

Thomas *et al.* showed in a case-control study of patients treated for hypertension a J-shaped relationship between BP and incident AF over a 12-year follow-up, with the lowest rates of incident AF at an SBP value of 120–130 mmHg and DBP of 60–69 mmHg, respectively, thus suggesting that optimal BP control might decrease AF burden in hypertensive patients [38].

Recent trials have investigated the effects of aggressive BP control in patients with AF [39, 40]. The Substrate Modification with Aggressive Blood Pressure Control (SMAC-AF) trial was a randomized control trial that investigated whether aggressive blood pressure control could impact AF recurrence, demonstrating similar results for the primary outcome (61.4% in the aggressive treatment group and 61.2% in the standard treatment group, with a hazard ratio [HR]=0.94, 95% confidence interval, 0.65–1.38; P=0.763) and a higher incidence of hypotension requiring treatment adjustment [40]. A possible benefit was remarked in patients over 61 years of age, with a lower primary outcome event rate in the aggressive BP control arm (HR=0.58, 95% confidence interval, 0.34–0.97; P=0.0013) that did not reach, however, statistical significance. The ARREST-AF trial concluded that, despite the fact that late recurrence of AF post-catheter ablation is certainly attributed to PV reconnection, atrial substrate progression secondary to suboptimal control of risk factors also has an important role [41].

Although antihypertensive drugs reduce the risk for AF mainly by lowering high blood pressure, specific regimens may additionally reduce the risk through other mechanisms [23]: blockade of the RAAS may prevent LA fibrosis, dysfunction, and slowing of conduction velocity, with some studies also indicating direct antiarrhythmic properties. Favorable effects of RAAS blockers on cardiac alterations, such as atrial enlargement and LV hypertrophy, may explain the reduction in new-onset AF [23].

RAAS blockers have been shown to reduce the first occurrence of AF, compared with beta-blockers or calcium-channel blockers [42, 43]. When it comes to secondary prevention, data are conflicting. In a meta-analysis by Schneider *et al.*, RAAS blockade reduced the odds of AF recurrence after cardioversion by 45% (0.34–0.89, P<0.01) and medical therapy by 63% (0.27–0.49, P<0.00001) [44]. On the other hand, other studies [45], [46] report that RAAS blockers do not prevent the recurrence of paroxysmal or persistent atrial fibrillation.

Considering all of the above, RAAS blockers may be considered part of the antihypertensive treatment strategy in hypertensive patients with a high risk of AF.

The priority in AF patients is stroke prevention. In a study by Verdecchia *et al.*, the annual incidence of stroke was significantly higher in hypertensive patients with intermittent or chronic AF (2.7 and 4.6%, respectively) than in those without AF (0.81%, P=0.0005) [47]. Therefore, in HTN patients, all efforts should be made to document AF. AF can rarely be ruled out as the underlying problem on clinical grounds alone, and the diagnosis of AF usually carries important implications, at least regarding anticoagulation [48, 49]. "Silent" AF is also associated with a significant risk for stroke [50, 51], which has led to the recommendation of "opportunistic screening" for AF using clinical examination, ECG, or mobile devices (especially with ECG capabilities).

Currently, the CHA2DS2-VASc score is widely used by most guidelines for stroke prevention in AF [52]. However, anticoagulants should be strongly considered in AF patients in whom HTN is the only additional stroke risk factor [52, 54, 56]. Additionally, AF patients with a longer HTN history or uncontrolled systolic BP values should be categorized as "high-risk", and strict control of BP, in addition to oral anticoagulation, is important to reduce the risk of ischemic stroke and intracerebral hemorrhage [53, 55, 56].

## Pre-procedural evaluation of hypertensive patients

All eligible patients for transcatheter ablation for atrial fibrillation should be rigorously evaluated prior to the procedure. There are many scores that try to predict the likelihood of arrhythmia recurrence, none of them being superior. Considering

all the aforementioned aspects, currently available literature suggests that pre-procedural evaluation of hypertensive patients with AF should comprise screening and adequate control of risk factors, 12 lead electrocardiogram (ECG), and multimodality imaging for anatomical and functional assessment for both left atrium and left ventricle [55].

Uncontrolled hypertensive patients have a higher rate of arrhythmic recurrence after transcatheter ablation; therefore, efforts should be made to achieve and maintain better BP control [36].

Age is a powerful driver of stroke risk, and most population cohorts show that the risk rises from age 65 years upwards; therefore, this population should be more carefully evaluated. However, several studies provide evidence that catheter ablation of AF has an acceptable safety and efficacy profile in selected older individuals [36].

ECG-derived information may also be useful. LVH criteria provide independent prognostic information, even after adjusting for other cardiovascular risks. Moreover, the presence of a "strain pattern" on the ECG is associated with an increased risk of AF recurrence [36]. The analysis of f-waves in time [39, 56], frequency domains [57, 58], or using more elaborate complexity indices [59] has been shown to correlate with CA outcome. Cuculich *et al.* studied continuous biatrial epicardial activation patterns in AF using non-invasive electrocardiographic mapping [60]. Utility of the system in panoramic 3D mapping and in describing the global cardiac activation patterns edges past 12-lead ECG. Rapid, reliable, and the single beat/cycle-based diagnostic ability of the system expresses its potential to reduce ablation, fluoroscopic and procedural times [61].

Echocardiography is the main tool in preprocedural evaluation. 2D echocardiography evaluates LA reservoir function and stiffness, while 3D echocardiography is the most reliable echocardiographic method for evaluating LA size/volumes. Functional imaging includes tissue Doppler imaging (TDI) and strain. Global strain in the four-chamber view likely offers three exciting parameters in the absence of segmental failures of deformation: maximum positive strain, late atrial strain, and peak negative diastolic strain. However, no single parameter actually predicts AF relapse after CA. The predictors of AF recurrence after CA confirmed by several groups were LA diameter >50–55 mm or LAVi >34 mL/m$^2$, E/e' >13–15, LA strain assessed by STE <20–25%, and total atrial conduction time measured by TDI >150 ms.

AF in the context of LVH is associated with worse outcomes. These patients are more susceptible to AF progression. This population also has a higher degree of LA fibrosis. Screening for LVH is convenient and cost-efficient. Still, more extensive randomized controlled trials are needed to demonstrate the independence of LVH as a predictor of the recurrence of AF.

In selected patients, transesophageal echocardiography can be used to evaluate valvular heart disease or left atrial appendage (LAA) thrombus. It can also provide additional information important in guiding the procedure by careful, multiplanar inspection of the LAA, the number of LAA lobes and may aid in identifying the best site for transseptal puncture (visualizing patent foramen ovale, IAS aneurysm, and overall anatomy). LAA emptying velocity measured during preprocedural TEE can serve as a predictor of AF recurrence in patients undergoing CA [62]. A recent study showed that LAA emptying velocity of ≥52.3 cm/s was associated with decreased AF recurrence post-ablation (odds ratio [OR]: 0.55; 95% confidence interval [CI]: 0.31–0.97; p=.03*). This notion may be useful in the optimization of treatment strategies and the care of patients with AF undergoing transcatheter ablation [63]. However, more prospective trials are needed to verify these findings in the future.

An accurate assessment of LA anatomy can be obtained by computer tomography (CT), which is essential for a safe and effective AF ablation procedure. Emerging data suggest that CT imaging can be valuable in detecting thrombi prior to the procedure. High-resolution CT can also be useful in measuring LA wall thickness [64]. Consequently, this may aid in selecting the most appropriate ablation strategy (high-energy radiofrequency application in patients with thicker atrial walls). Left atrial wall thickness (LAWT) on CT was greater in HTN subjects and had a positive correlation with LVH findings on TTE and no correlation with LA size or LV diastolic dysfunction. LAWT may be an important response in subjects with HTN and LVH [65].

Recently, the delayed enhancement on cMRI has been introduced for detecting, quantifying, and localizing atrial fibrosis, including defining the four categories of structural changes (Utah stages I-IV). The association of atrial tissue fibrosis and AF ablation outcomes was confirmed by the DECAAF 1 and 2 studies [66, 67], with more extensive fibrosis associated with a lower efficacy. Interestingly, when compared with atrial fibrosis, none of the traditional risk factors for AF recurrence (including HTN) were independent predictors of recurrence, except significant mitral valve disease [67]. Additionally, there was no consistent correlation between the amount of LA fibrosis and AF pattern (paroxysmal *vs.* persistent).

## Radiofrequency ablation

Regarding the procedure, radiofrequency catheter ablation has emerged as an essential therapy for AF; however, recurrence rates remain high. Hypertension represents an important pre-procedural predictor of recurrence. Berruezo *et al.* [68] showed how

 

high BP and LA diameter are the main predictors of arrhythmia recurrence after pulmonary vein antrum isolation (PVAI) and suggested the potential role of poor control of hypertension. Another predictor of a lower success rate of ablation is obstructive sleep apnea [69].

Currently, the 2020 guidelines of the European Society of Cardiology Guidelines recommend catheter ablation of AF with pulmonary vein isolation after initial failed or intolerable antiarrhythmic drug therapy in patients with paroxysmal AF or persistent AF, with or without major risk factors for AF recurrence, with a class I level of recommendation, as opposed to earlier guidelines that rendered catheter ablation to a class IIa. However, in patients with AF and heart failure with reduced ejection fraction (HFrEF) with a highly probable tachycardiomyopathy component, catheter ablation is leveled with antiarrhythmics as first-line therapy, with a class I level of recommendation, as well. Moreover, emphasis on patient options and shared decision-making were brought to the forefront [6].

Currently, the mandatory procedural endpoint in AF ablation is PV electrical disconnection (class I, LOE A) [4]. This is often done by radiofrequency (RF) point-by-point ablation, followed by cryoballoon ablation [4]. Some researchers consider that targeting non-PV triggers [70–74] would improve the success rate of AF catheter ablation; however, recent data have not supported this [75]. Moreover, previous studies have also considered vagal denervation of the pulmonary veins (peri-PV ganglionic plexus ablation) useful to reduce the recurrence [76, 77], but this is currently proved valid only in patients with vagally-mediated AF.

In addition, 3D navigation mapping systems assist in the electroanatomical reconstruction of the LA. Superposition with pre-registered imaging acquisitions (magnetic resonance or computer-tomography) offers superior anatomical accuracy and tailored ablation lesions, depending on patient anatomy and clinical characteristics (see preprocedural evaluation).

One important aspect to be taken into account during the procedure in HTN patients is the risk of sodium-volume overload [71, 72–75]. The overwhelming majority of RF-based ablations used saline to cool the tip of the ablation catheter, which might be acutely deleterious in HTN patients. Therefore, using micropores-irrigated tip catheters instead of a standard irrigated ablation catheter will reduce the flow of saline infusion (17 ml/min instead of 30 ml/min), thus mitigating the risk of sodium overload. Other alternatives would be to use a half-saline solution for irrigated catheters or the use of high-power/short-duration (very-high-power/very-short-duration) RF set-ups/technologies.

Additionally, some intraprocedural data might be used to tailor therapy, as well as predict recurrences for AF ablation: pre-existent left atrial scarring during catheter mapping [78], voltage abatement [79], the percentage of left atrium ablated [80], conduction slowing or block across the ablation lines [81, 82], AF inducibility after left atrial circumferential ablation [83], and after-segmental ostial ablation.

There are no statistical differences in AF recurrence rate following ablation between patients with controlled hypertension and no hypertension in terms of long-term follow-up. In contrast, pharmacologically uncontrolled hypertension confers higher AF recurrence risk and requires more extensive ablation [70]; as such, strict control of blood pressure is warranted in hypertensive patients with atrial fibrillation. Therefore, renal artery denervation has been studied as a potential more comprehensive interventional strategy in patients with refractory AF and resistant hypertension [84]. The ERADICATE-AF trial confirmed that renal sympathetic denervation, in conjunction with catheter ablation of PVs, resulted in a statistically significant proportion of patients arrhythmia-free at 12 months of follow-up [85–87].

## Postprocedural aspects

For further improvement of atrial fibrillation ablation outcome, it is crucial to consider the management of all risk factors, including BP, body weight, glycemic control, and lipid profile, as it was suggested by The ARREST-AF cohort study [41] and RACE studies [87]. In hypertensive patients, it has been observed that uncontrolled BP, in conjunction with the type of AF, could predict the progression of the atrial disease.

In addition, a rhythm control strategy, particularly transcatheter ablation, seems to significantly benefit BP in uncontrolled hypertensive patients with AF. There are several studies that sustain this hypothesis. The AF-FIRM trial [88] has shown that a rhythm control strategy was associated with decreased usage of antihypertensive drug therapy in patients with AF and hypertension, possibly due to avoidance of sympathetic activation produced by arrhythmia burden. Ramírez *et al.* [89] have also shown that successful CA in patients with AF and hypertension is associated with a decrease in systolic blood pressure when compared to an increase in patients with failed ablation. Restoring sinus rhythm could have an antihypertensive effect in this population; therefore, the follow-up of these patients should include repeated monitoring of BP.

AF has been shown to be associated with ventricular and atrial remodeling and deterioration in both left ventricular (LV) diastolic and systolic function. Whether those changes are the cause or consequence of the arrhythmia remains debatable [90]. The left atrium undergoes structural, metabolic,

neurohumoral, and electrical changes in response to chronic external stressors [91]. Animal experiments have shown that the mechanism for LA remodeling is different between atrial tachycardia-induced LA remodeling and LV pressure/volume overload-induced AF [92].

Post-ablation oral anticoagulation (OAC) is recommended for two months, with long-term OAC guided by CHA2DS2-VASc score ($^{3}$2 in men, $^{3}$3 in women), irrespective of the AF ablation outcome. Besides the CHA2DS2-VASc score, there are special populations with higher thromboembolic risk in whom long-term OAC should be considered (the elderly, uncontrolled hypertensive patients, dilated LA). In a recent meta-analysis, Liu *et al.* studied thromboembolic risk in patients on and off OAC after successful CA for AF, and no statistical difference was found. Moreover, patients on OAC after successful CA had a higher risk of major bleeding events [93]. Current data also suggest that LA size after successful catheter ablation seems to decrease, regardless of the imaging modality of evaluation, and this may have a significant impact on long-term stroke risk and/or OAC maintenance. Consequently, all patients after AF ablation should be systematically evaluated by imaging to assess anatomical and functional post-procedural improvement of the left heart.

Functional impairment precedes structural changes to the left atrium. Markers of LA remodeling, such as LA size, function, and late gadolinium enhancement, have long been associated with stroke risk in individuals in SR. The prognostic value of LA volumes and function in lone AF has been shown in the setting of cardiovascular events (including stroke) [94], [95]. Pagola *et al.* [96] reported the presence of silent AF in 86% of cryptogenic stroke patients with normal LA size but decreased LA strain. There are studies that have shown a reduction in LA functional metrics between a healthy cohort and a young cryptogenic stroke population, despite similar profiles within groups [97].

LA enlargement has proven to be associated with recurrent AF after cardioversion in the AFFIRM study [98] and portends higher stroke risk. Zaca *et al.* [99] have shown a direct correlation between increased LA size at baseline and progressive LA enlargement during follow-up, as well as the number of arrhythmic recurrences.

Multiple studies have reported an abnormal LA flow profile in patients with both paroxysmal and persistent AF [100] and demonstrated that this is related to clinical stroke risk. Aging and long-term exposure to cardiovascular risk factors lead to a subtle atrial and ventricular cardiomyopathic phenotype, disrupting LA flow characteristics. Altered LA flow parameters were observed in all high-risk patients in SR regardless of a history of AF [100].

LAA geometric parameters should be considered, coupled with the morphological characteristics, for a comprehensive evaluation of stroke risk. LAA geometric characteristics have an impact on the hemodynamic pattern within the LAA [101]. Not only complex LAA morphologies are characterized by low velocities, low vorticity, and consequently, a higher thrombogenic risk. Simple morphologies can also have a thrombogenic risk equal to or even higher, and therefore geometric features of LAA could play a key role in defining thromboembolic risk [102]. Masci *et al.* [102], on the other hand, consider that the complexity of the LAA shape alone does not correlate with clot formation, and additional parameters should be considered.

Atrial cardiomyopathy, as quantified by LA LGE severity, might be the physiological trigger associated with adverse AF sequelae [103]. In a recent meta-analysis, Kheirkhahan *et al.* showed that the risk of AF recurrence is higher in patients with new fibrosis after catheter ablation (new fibrosis >21%; HR 37% *vs.* 62%, p=0.01) [104]. Daccarett *et al.* [105] showed that AF patients with stroke had higher LA fibrosis as compared to those who did not (24.4±12.4% *vs.* 16.2±9.9%, p=0.01); similar results were reported by Akoum *et al.* [106]. Van Gelder *et al.* [107] noted that some patients with a rhythm control strategy remain at risk for cardiovascular events, even when sinus rhythm is maintained.

Severe LA scarring after ablation predisposes to AF recurrences, which seems to result from reconduction between the LA to pulmonary veins (PVs) [108]. Pre-existent LA scarring concomitant with dilated LA may reduce the success rate after ablation. The extent of ablation and re-ablation do not appreciably increase the success rate of patients with large LA scars [109].

Based on multiple characteristics of atrial myopathy like those described above, Marrouche proposed a score to predict the stroke risk for long-term OAC maintenance in patients in whom catheter ablation successfully eliminated AF (Table 1) [110].

**Table 1.** The score proposed by Marrouche for assessing the stroke risk in patients taking long-term OAC [110].

| | | |
|---|---|---|
| LA structure | Fibrosis >25% | 1 point |
| LA function | LAEF <40% | 1 point |
| LA shape and size | Dilated and spherical LA | 1 point |
| LA appendage characteristics | LAA curvature<br>Ostium>4 cm<br>Flow <40 cm/s | 1 point |
| LA flow | Poor LA flow | 1 point |
| Ablation induced scar | Extensive scar | 1 point |

LA – left atrium; LAEF – atrial ejection fraction.

## Conclusions

Hypertension is a significant risk factor for AF development, and the incidence of AF is increased in patients with hypertension. Extensive published data reinforce the idea that better BP control leads to a lower incidence of AF and a reduced risk of recurrence after transcatheter ablation. Additionally, several studies have shown that successful maintenance of sinus rhythm by AF catheter ablation allows for superior HTN control and/or a reduced need for antihypertensive drugs.

## Conflict of interest

The authors confirm that there are no conflicts of interest.

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