Published in final edited form as:

Thromb Res. 2010 August ; 126(2): e69–e77. doi:10.1016/j.thromres.2009.11.031.

Peri-procedural anticoagulation in patients undergoing ablation for atrial fibrillation

Sara R. Vazquez^{a,*}, Stacy A. Johnson^b, and Matthew T. Rondina^{c,d,e}

^aUniversity of Utah Thrombosis Service, University of Utah, Department of Pharmacy Services, 675 Arapen Drive, Suite 100, Salt Lake City, UT 84108, United States

^bDepartment of Internal Medicine, University of Utah School of Medicine, United States

^cDepartment of Internal Medicine, University of Utah Health Sciences Center, United States

^dUniversity Thrombosis Service, University of Utah Health Sciences Center, United States

^eTranslational Research, University of Utah Health Sciences Center, United States

Abstract

Radiofrequency catheter ablation is being used with increasing frequency as a strategy to manage atrial fibrillation. Patients undergoing this procedure are at increased short-term risk of thromboembolism for several days and up to 4 weeks or longer after their ablation, and anticoagulation management surrounding the ablation procedure remains controversial. Although no conclusive recommendations can be made, published guidelines and data support therapeutic anticoagulation with warfarin for 3 weeks prior and intravenous heparin during the ablation. Warfarin may either be continued through the ablation or stopped 2–5 days prior. If the latter approach is chosen, a pre-ablation bridging strategy of enoxaparin 1 mg/kg twice daily is reasonable in selected patients unless the patient's bleeding risk dictates using a lower dose regimen (0.5 mg/kg twice daily) or avoiding bridging altogether. Fewer data are available for post-ablation management strategies, and current practice patterns are based largely on single-center experiences in smaller, non-randomized studies. For lower risk patients (CHADS₂ 0–1), either warfarin or aspirin may be utilized without bridging. In higher thromboembolic risk patients (CHADS₂ ≥ 2), either enoxaparin (1 mg/kg twice daily) or heparin may be started within the first 12–24 h post-procedure. For patients with bleeding risk factors, enoxaparin may be subsequently reduced to 0.5 mg/kg until the INR is therapeutic, although the efficacy of this lower dosing regimen has not been well studied. In accordance with national guidelines, warfarin should be continued post-ablation for a minimum of 2 months and then indefinitely in patients with a CHADS₂ score ≥ 2.

Keywords

Atrial Fibrillation; Ablation; Anticoagulation; Low-molecular weight heparin; Unfractionated heparin; Warfarin; Peri-procedural; Bridging; Stroke; Transient Ischemic Attack; Thromboembolism

Atrial fibrillation (AF) is one of the most common, sustained arrhythmias and is associated with significant morbidity and mortality. Although there is still controversy regarding the best management approach to AF, most providers choose a strategy of either ventricular rate or rhythm control along with chronic anticoagulation to prevent thromboembolic (TE) complications [1]. More recently, in addition, or at times, as an alternative to these approaches, catheter-directed ablation is being used with increasing frequency in patients with symptomatic, refractory AF.

Regardless of what strategy is used, AF may result in TE - often from thrombus in the left atrium or atrial appendage - and accounts for nearly 15% of all strokes in the US [2], with an annual incidence of stroke of up to 6% or even higher in some patients [3]. The risk of stroke in AF is neither static nor uniform, may depend on both clinical and echocardiographic factors, and usually requires an individualized approach to assess stroke risk and determine appropriate AC management. Re-estimation of the estimated stroke risk is recommended as a patient's risk may change over time, and alternative therapy may be indicated.

Although recent meta-analysis data failed to identify one particular stroke risk assessment scoring system that is clearly superior to other validated systems[4], the CHADS₂ score is commonly used to assess baseline stroke-risk and guide antithrombotic therapy (Table 1) [5]. Most of the published reports of peri-ablation anticoagulation reviewed in this paper utilized the CHADS₂ scoring system and thus, we will also use this same risk assessment tool. In patients with a CHADS₂ score of ≥ 2 , warfarin effectively reduces the risk of stroke and is recommended for chronic anticoagulation [1]. In AF patients with a CHADS₂ score of 0–1 – where the risk of stroke may be lower – warfarin is more controversial, given its narrow therapeutic window and risk of major or life-threatening bleeding, and either warfarin or aspirin are recommended for chronic anticoagulation, according to patient and provider preferences [6,7]. Although widely used, several limitations exist with the classical CHADS₂ scoring system. Anticoagulant selection for the “intermediate risk” group (score 1–2), encompassing >60% of patients, is determined by clinician preference [8] and fear of warfarin-related bleeding complications may result in suboptimal TE prophylaxis. Additionally, the CHADS₂ scoring neglects other known AF-related stroke risk factors (female sex, age 65–74, peripheral arterial disease), and lacks validation in the peri-procedural period [8,9].

AF ablation, if successful long-term, may reduce the risk of future stroke and transient ischemic attack (TIA). However, somewhat paradoxically, AF patients are at increased short-term risk of TE for several days, and up to four weeks, after their ablation [10]. This pro-thrombotic period may result in a higher, albeit transient, TE risk in AF patients who were previously judged to be low-risk. Although the precise mechanism(s) by which ablation induces this pro-thrombotic effect are not clearly understood, several factors are implicated. Transseptal sheath placement can precipitate thrombus formation on the catheter or sheath during the procedure (although catheter thrombus may be minimized with adequate peri-procedural anticoagulation and an open-irrigated tip) or in the left atrial appendage (LAA) [11–13]. Delivery of radiofrequency energy during the ablation disrupts the endocardium, possibly exposing procoagulant proteins (e.g. tissue factor) and activating the clotting cascade [14]. “Char” (hard coagulum) from tissue heating and denaturation and aggregation of plasma proteins [15] may form and systemically embolize [11]. Additionally, the atrial tissue may be stunned for several weeks, or even months, post-procedure leading to impairment of normal contraction [16].

Reported rates of peri-procedural TE complications are 0.5–2.8% [11,17–19] and may be higher in patients with a CHADS₂ score ≥ 2 , patients with a history of stroke [10], patients

with persistent rather than paroxysmal AF [1], and patients with marked left atrial enlargement or a dilated LAA [20].

As the number of AF ablation procedures increases, especially in higher-risk and older patients, comprehensive and careful peri-procedural anticoagulation management based on best-available data and individualized for each patient is paramount. Although preprocedure warfarin (goal international normalized ratio [INR] 2–3) and intra-procedure intravenous unfractionated heparin (IV UFH) with a target activated clotting time (ACT) of ≥ 300 seconds are routinely used, practice patterns for other peri-procedural anticoagulation (e.g. post-procedure heparinoids, antiplatelet agents, and duration of warfarin) vary widely. These differences are due, in part, to a paucity of adequately powered studies and, in the studies published to date, heterogeneity in patient populations, type of bridging strategy employed, and outcomes. These limitations underscore the need for caution when interpreting published data and, in the authors' opinion, the need for an individualized peri-procedural anticoagulation management plan for most patients. A more detailed list of the studies referred to in this review can be found in Appendix A.

Should AF patients receive pre-procedural bridging therapy?

Current guidelines recommend anticoagulating AF patients with warfarin (INR goal 2–3) for a minimum of 3 weeks prior to either electrical or chemical cardioversion [1,21]. Given the risk of peri-procedural TE events, expert consensus statements extend these recommendations to include ablation procedures [22]. For patients either refusing or who are intolerant to oral anticoagulation, antiplatelet agents, although less effective than warfarin in preventing TE, are a recommended alternative, especially in lower TE risk patients (i.e. those with paroxysmal AF and/or a CHADS₂ score of 0) [10,23–25].

Warfarin may be stopped, at the clinician's preference, 2–5 days prior to the procedure [10,11,23,24,26–32]. Alternatively, and as is done at our center and others, warfarin may be continued during the procedure without interruption. In two studies of lower risk patients (mean INR $\pm$ standard deviation day of procedure 2.7 ± 0.5 and 2.8 ± 0.7), continuing warfarin during the procedure was not associated with an increased risk of peri-procedural bleeding [27,33].

In patients where warfarin is stopped several days pre-ablation, either IV UFH or low molecular weight heparins (LMWH) may be used as "bridge" therapy (e.g. between the time warfarin is discontinued and the procedure begins) [10,22–27,29,30,34–36]. Given their ease of use outside the hospital, the lack of monitoring required, and their potential economic savings, LMWHs are emerging as the preferred agents for peri-procedural bridging in many patients [37].

Most published studies employing LMWHs pre-procedure have used enoxaparin dosed at either 1 mg/kg twice daily or a lower dose of 0.5 mg/kg twice daily. Enoxaparin 0.5 mg/kg twice daily is being used with increasing frequency due to the lower rate of femoral vascular complications when this dosing regimen is employed [10,27,30,31]. The last dose of LMWH should be given at least 24 hours prior to the procedure to minimize bleeding events, as recommended in national guidelines [10,24,27,29,37].

How is anticoagulation managed intra-procedurally?

To prevent thrombus formation, a loading dose of UFH (100 U/kg) followed by a continuous infusion (10 U/kg/hr) is often utilized. The ACT is monitored until therapeutic or as needed [22]. In a trial of 3 different ACT targets (250–300s, 300–350s, and 350–400s), there was a lower incidence of TE (stroke and TIA) when the two higher ACT targets were

maintained [11]. Consistent with these data, current guidelines recommend a target ACT of at least 300–350s throughout the procedure [22,25], although additional TE risk factors (e.g. spontaneous echo contrast and marked atrial enlargement) may warrant higher ACT targets of 350–400s [34,38].

How can anticoagulation post-procedure be managed?

As with direct current cardioversion, the atria are often "stunned" following radiofrequency ablation [16] and anticoagulation may help prevent thrombus formation and, importantly, stroke and TIA. Many centers restart warfarin and/or heparinoids a few hours after the procedure or the evening of the procedure [10,11,18,22,25,27–29,31,32,39–41]. In patients with higher post-procedure bleeding-risk, the initial use of IV UFH until the morning after the procedure followed by a transition to LMWH [10,18,32,40,41] allows for easier and more rapid reversal of anticoagulation if an early bleeding complication occurs.

When transitioning to LMWH and restarting warfarin, enoxaparin may be initially used therapeutically (1 mg/kg twice daily in the first 24 hours post-procedure) and then decreased to 0.5 mg/kg twice daily until a therapeutic INR is achieved [31,39]. This approach was associated with a reduction in post-procedure femoral vascular complications compared to 5 days of therapeutic enoxaparin (5.7% vs 1.6%). Importantly, there were no procedure-related TE events in patients who received this lower-dose enoxaparin regimen and a very low incidence of other bleeding events such as hematomas and pseudoaneurysms [31]. Low rates of post-procedure ischemic stroke were also reported in a study of similar design and were higher in patients with a CHADS₂ score ≥ 2 (odds ratio [OR] 7.1 vs. CHADS₂=0) or a history of stroke (OR 9.5), although the absolute number of events was small (10/721 patients, 1.4%) [10].

How do we Estimate Individual Bleeding Risk?

Although antithrombotic therapy effectively reduces TE risk, this benefit comes at the risk of increasing bleeding, especially in higher-risk patients. Several bleeding risk-assessment scales [42–44] may help clinicians determine their patient's risk of bleeding, although none have been validated well enough to recommend the preferential use of one over another. One of these bleeding risk scales, called "HEMORR₂-HAGES" [45], was developed using a cohort of elderly AF patients prescribed warfarin. HEMORR₂HAGES also identifies subgroups at higher risk of bleeding, including those with liver disease, ethanol abuse, and reduced platelet count or function. Regardless of which risk assessment tool is employed, many of the unique bleeding risks associated with AF ablation (usually from accessing the vasculature - groin hematomas, retroperitoneal bleeding, hemopericardium, atrioesophageal fistula, etc) are not incorporated into any of these bleeding risk schemes. Rather than relying on just one of these bleeding risk assessment tools, we and others [44] recommend incorporating the presence of these bleeding risk factors (Table 2) into a patient's individual anticoagulation plan.

What is the role of antiplatelet agents pre- and post-ablation?

Aspirin (ASA) has been used in lower-risk AF patients undergoing ablation, although its precise role as monotherapy and as combination therapy with warfarin remains controversial. In 207 patients with symptomatic AF (85% had paroxysmal AF and 88% had a CHADS₂ score of 0–1), warfarin was discontinued 3 days prior to ablation and patients received ASA 325 mg daily. Warfarin was resumed on the day of procedure and both medications were continued for 6 weeks. TE events occurred in 2 patients (0.97%) within 8 days post-procedure, both with subtherapeutic INRs, and two patients had small groin hematomas post-procedure (0.97%) [28]. Thus, ASA may be an alternative to LMWH

bridging to warfarin in AF patients with a low CHADS₂ score and paroxysmal AF, although more data is clearly needed. ASA as monotherapy post-procedure may also be safe and effective in carefully selected low-risk AF patients (i.e. CHADS₂ score of 0–1 and paroxysmal AF) [41], although the temporarily higher TE risk post-ablation may warrant warfarin as the preferred agent.

How long should patients remain anticoagulated after ablation?

The optimal duration of anticoagulation post-ablation ranges from one to six months depending on several variables, including stroke risk factors, CHADS₂ score, [22,25,35], type of pre-procedure AF (persistent or paroxysmal) [28], and AF recurrence post-procedure [11,28,41,46,47]. Based on the high rate of asymptomatic AF after ablation [48] as well as the increased failure rates in patients with long-standing AF and/or those who have undergone multiple procedures [32,35,46], the duration of anticoagulation should be based largely on a patient's TE risk factors (i.e. CHADS₂ score), as recommended in published guidelines [22,25]. The Venice Chart International Consensus Document on Atrial Fibrillation Ablation recommends that patients complete 3–6 months of warfarin. If the patient's CHADS₂ score is ≥ 2 , indefinite warfarin is recommended. For lower risk patients, either ASA or warfarin (CHADS₂=1) or ASA or no therapy (CHADS₂=0) are recommended [25]. Similarly, the Heart Rhythm Society/European Heart Rhythm Association/European Cardiac Arrhythmia Society guidelines recommend at least 2 months of warfarin (INR 2–3) post-ablation followed by a duration of anticoagulation based on the patient's stroke risk factors [22]. Both guidelines recommend continuing warfarin indefinitely in any patients with a CHADS₂ score ≥ 2 [22,25]. Interestingly, a recent study by Dagues and colleagues evaluated real-world adherence to these guidelines. The investigators found that AF recurrence was a more significant predictor for patients remaining on oral anticoagulation at 12 months post-ablation than the patient's CHADS₂ score, possibly leading to overtreatment of low-risk patients, and undertreatment of high-risk patients [49].

Conclusions

Until large, adequately powered, randomized-controlled trials are available, practitioners must use the best available data and recommendations from consensus guidelines to understand how to safely and effectively reduce TE risk in AF patients undergoing ablation (Tables 3 and 4). Although no conclusive recommendations can be made, published guidelines and data support therapeutic anticoagulation (INR 2–3) for 3 weeks prior to ablation and use of IV UFH during the ablation itself (target ACT of at least 300–350s). Warfarin may either be continued through the ablation or stopped 2–5 days prior to the ablation, depending on practitioner preference and institutional protocols. If the latter approach is chosen, the authors recommend a pre-ablation bridging strategy of enoxaparin 1 mg/kg twice daily unless the patient's bleeding risk dictates using the lower dose regimen (0.5 mg/kg twice daily).

Bridging management strategies post-ablation are even less well standardized or studied, and current practice patterns are based largely on single-center experiences in smaller, non-randomized studies. No single strategy (either drug or dosing) has been shown to be unequivocally superior and LMWHs, IV UFH, and/or antiplatelet agents may be used until patients are therapeutic on warfarin. For lower risk patients (CHADS₂ 0–1), either warfarin or ASA may be utilized without bridging with a heparinoid. In higher TE risk patients (i.e. CHADS₂ score ≥ 2), we recommend the use of therapeutic anticoagulation with either enoxaparin (1 mg/kg twice daily) or IV UFH for the first 12–24 hours. This recommendation places a larger emphasis on preventing TE events post-procedure and should be used thoughtfully, given the potential risk of bleeding. In patients with bleeding risk factors,

enoxaparin may be subsequently reduced to 0.5 mg/kg until the INR is therapeutic, although the efficacy and pharmacology of this lower dosing regimen in stroke prevention has not been well studied and should be considered unproven until adequately powered and designed studies have been published. In accordance with national guidelines, warfarin should be continued post-ablation for a minimum of 2 months and then indefinitely in patients with a CHADS₂ score ≥ 2 .

Appendix A

Table 1

Clinical Trials Evaluating Peri-Procedural Anticoagulation for Atrial Fibrillation Ablation.

Authors	Patients	Pre-Procedural AC	Intra-Procedural AC	Post-Procedural AC	Bleeding	Stroke/TIA
Schmidt, et al 2009 [33]	n = 194 Follow-up:	Warfarin or phenprocoumon either continued pre-procedure or started 3 days prior to procedure <i>Controls</i> (n = 107): INR <2 day of procedure <i>Cases</i> (n = 87): INR ≥ 2 day of procedure Procedure postponed if INR >3.5 day of procedure	ACT 350–450	<i>Controls</i> : enoxaparin 0.5 mg/kg BID started 6–8 hours post-procedure and continued until therapeutic INR achieved Warfarin or phenprocoumon restarted in all patients PM of procedure	Minor vascular complications ^a <i>Controls</i> : n = 7 (6.5%) <i>Cases</i> : n = 5 (5.8%) p = NS Major vascular complications ^b : <i>Controls</i> : n = 1 (0.93%) <i>Cases</i> : n = 1 (1.2%)	<i>Controls</i> : n = 0 <i>Cases</i> : n = 1 (1.2%)
Bunch, et al 2009 [41]	n = 630 CHADS ₂ score 0–1: 20% CHADS ₂ score ≥ 2 : 80% Mean follow-up: 327 $\pm$ 368 days	NR	NR	UFH 4 hours post-sheath removal and continuing for 24 hours If CHADS ₂ score 0–1 = ASA 325 mg daily (n = 123) If CHADS ₂ score 2: LMWH (dose NR) + warfarin (n = 507)	NR	ASA group = 0% Warfarin group = 0.4% p = NS
Prudente, et al 2009 [31]	n = 539 (603 procedures) Mean follow-up: 1 month	Warfarin D/C 4 days pre-procedure to achieve INR <2	ACT 300–350	Warfarin re-started 4–6 hours post-procedure enoxaparin 1 mg/kg at 4 hours post-procedure and the next AM <i>Protocol A</i> (n = 263): enoxaparin 1 mg/kg BID $\times$ 5 additional days <i>Protocol B</i> (n = 85): enoxaparin 1 mg/kg BID $\times$ 3 additional days <i>Protocol C</i> (n = 255): enoxaparin 0.5 mg/kg BID $\times$ 3 additional days	Femoral vascular complications: <i>Protocol A</i> : 5.7% <i>Protocol B</i> : 2.4% <i>Protocol C</i> : 1.6% p < 0.03 (comparing A to B + C or A to C)	<i>Protocol A</i> : n = 1 (0.38%) <i>Protocol B</i> : n = 0 <i>Protocol C</i> : n = 0
Scherr, et al 2009 [10]	n = 579 (721 procedures)	Warfarin (INR 2–3) for at	ACT 300–400	Warfarin + UFH initiated 6 hours post-sheath	Pericardial effusions n = 9/721 (1.2%)	Within ≤ 30 days post-

Authors	Patients	Pre-Procedural AC	Intra-Procedural AC	Post-Procedural AC	Bleeding	Stroke/TIA
	CHADS ₂ score 0–1: 580/721 (79%) CHADS ₂ score ≥2: 152/721 (21%) Follow-up: 3 months	least 4 weeks pre-ablation Warfarin D/C 5 days pre-procedure, and bridged with enoxaparin 0.5–1 mg/kg BID ^g		removal UFH D/C and enoxaparin 0.5–1 mg/kg BID ^c started the following AM and continued until INR >2 Warfarin continued for at least 3 months	Femoral vascular complications n = 11/721 (1.5%)	procedure: n = 10/721 (1.4%) (9 were within 24 hours of procedure, 1 within 6 days of procedure) CHADS ₂ score 0: 0.3% CHADS ₂ score 1: 1.0% CHADS ₂ score ≥2: 4.7%
Mortada, et al 2008 [28]	n = 207 CHADS ₂ score 0–1: 88% CHADS ₂ score ≥2: 12% Mean follow-up: 24 ± 6 months	Warfarin D/C 3 days pre-procedure, then ASA 325 mg daily × 3 days Need INR ≤2	ACT 300–350	Warfarin (INR 2–3) and ASA restarted day of procedure and continued for 6 weeks Warfarin continued beyond 6 weeks only if pt had AF recurrence or prior history of persistent AF If no AF recurrence at 6 months, warfarin D/C ASA continued for at least 6 months in all patients	Groin hematoma n = 2 (0.97%) Pericardial effusion n = 2 (0.97%)	n = 2 (0.97%) CHADS ₂ scores 1, 2 Both events occurred within 8 days of procedure, both with subtherapeutic INR
Rossillo, et al 2008 [47]	n = 170 n = 85 undergoing PVAI (28% low-medium stroke risk ^f ; 72% high stroke risk ^f) n = 85 matched controls undergoing DCCV (24% low-medium stroke risk ^f ; 76% high stroke risk ^f) Mean follow-up: 15 ± 7 months	AC started 1 month pre-procedure	ACT 350–400	ASA 325 mg × 1 dose given at the end of procedure All patients discharged with warfarin × 3 months Warfarin was D/C after 3 months unless patient had one of the following: recurrence of AF >60% PV narrowing Poor atrial contractility Presence of other indications for AC	NR	PVAI group n = 1 (1.2%) (<30 days after procedure) Control group n = 5 (5.9%) (1 <30 days after procedure, 4 >30 days after procedure)
Nademanee, et al 2008 [29]	n = 635 (1,065 procedures) Age >65 or had at least 1 stroke risk factor Mean follow-up: 836 ± 605 days	Warfarin (INR 2–3) for at least 3 weeks pre-procedure Warfarin D/C 4 days pre-procedure If persistent or permanent AF, bridged with	NR	Warfarin + enoxaparin 1 mg/kg BID restarted immediately post-procedure Enoxaparin D/C 3 days later Warfarin D/C after 3 months if NSR maintained,	Hemopericardium n = 9/635 (1.4%) Femoral vascular complications n = 13/635 (2%)	n = 11/517 (2.1%) Patients who D/C warfarin n = 5/434 (1.2%) Patients who continued warfarin n = 6/83 (7.2%)

Authors	Patients	Pre-Procedural AC	Intra-Procedural AC	Post-Procedural AC	Bleeding	Stroke/TIA
		enoxaparin 1 mg/kg BID		then ASA or clopidogrel prescribed		4 with adequate INRs 3 months prior, 2 had no documented INR within 3 months of event
Seow, et al 2007 [32]	n = 56 (86 procedures) Mean follow-up: 21.6 ± 8.8 months	AC D/C 3–5 days pre-procedure	ACT 300–350	UFH 4 hours post-procedure and continued until the following AM, then changed to LMWH until INR >2 Warfarin re-started the following AM	Cardiac tamponade n = 1 (1.1%)	n = 2 (2.3%)
Wazni, et al 2007 [27]	n = 355 Mean follow-up: 3–4 months	<i>Groups 1 and 2:</i> warfarin D/C 2–3 days prior to procedure <i>Group 3:</i> warfarin continued without interruption (INR 2–3.5)	ACT 350–450	ASA 325 mg × 1 dose at the end of procedure Warfarin restarted PM of procedure <i>Group 1</i> (n = 105): enoxaparin 1 mg/kg BID until INR >2 <i>Group 2</i> (n = 100): enoxaparin 0.5 mg/kg BID until INR >2 <i>Group 3</i> (n = 150): continued warfarin (INR 2–3.5)	Minor bleeding ^d : <i>Group 1</i> : n = 23 (22%) <i>Group 2</i> : n = 19 (19%) <i>Group 3</i> : n = 8 (5.3%) p < 0.001 Major bleeding ^e : <i>Group 1</i> : n = 9 (8.6%) <i>Group 2</i> : n = 0 (0%) <i>Group 3</i> : n = 0 (0%) p < 0.001	<i>Group 1</i> : n = 1 (0.95%) <i>Group 2</i> : n = 2 (2%) <i>Group 3</i> : n = 0 p = NS
Oral, et al 2006 [30]	n = 755 No stroke risk factors ^c : 44% ≥1 stroke risk factors ^c : 56% Mean follow-up: 25 ± 8 months	Warfarin D/C 5 days pre-procedure	ACT 300–350	UFH 1000 units/hour within 3 hours of sheath removal, continued until the following AM, then changed to enoxaparin 0.5 mg/kg BID until INR ≥2 Warfarin restarted PM of procedure and continued for at least 3 months ASA 81–325 mg daily indefinitely after warfarin D/C	Cerebral hemorrhage n = 2 (0.3%)	≤30 days of ablation: n = 7 (0.9%) (1 intraprocedural, 3 on enoxaparin with INR <2, 3 off enoxaparin with INR ≤2) >30 days post-ablation: n = 2 (0.3%) (both on warfarin with INR 2.6–3.2)
Wazni, et al 2005 [11]	n = 785 Follow-up: 12 months	Warfarin (INR 2–3) D/C 48 hours pre-procedure	<i>Group 1:</i> (n = 194) ACT 250–300	ASA × 1 dose at end of procedure (dose NR) Warfarin restarted day of procedure	Groin hematoma: <i>Group 1</i> : n = 1 <i>Group 2</i> : n = 2 <i>Group 3</i> : n = 2	<i>Group 1</i> : n = 7 (3.6%) <i>Group 2</i> : n = 3 (1.7%)
Wazni, et al 2005 [11]			<i>Group 2:</i> (n = 180) ACT 300–350 + Eptifibatide	If history of persistent AF: enoxaparin 0.5 mg/kg	Pericardial effusion: <i>Group 1</i> : n = 1 <i>Group 2</i> : n = 1 <i>Group 3</i> : n = 0	<i>Group 3</i> : n = 2 (0.49%) All events were intra-procedural

Authors	Patients	Pre-Procedural AC	Intra-Procedural AC	Post-Procedural AC	Bleeding	Stroke/TIA
			Group 3: (n = 411) ACT 350–400	BID until therapeutic INR achieved Warfarin D/C after 4–6 months unless AF recurrence or >70% PV narrowing	Cardiac tamponade: Group 1: n = 1 Group 2: n = 1 Group 3: n = 0	

AC = anticoagulation; ACT = activated clotting time; AF = atrial fibrillation; AM = morning; ASA = aspirin; BID = twice daily; CHADS₂ = stroke risk stratification system; D/C = discontinued; DCCV = direct current cardioversion; INR = international normalized ratio; LMWH = low-molecular weight heparin; NR = not reported; NSR = normal sinus rhythm; PM = evening; PV = pulmonary vein; PVAI = pulmonary vein antrum isolation; TIA = transient ischemic attack; UFH = unfractionated heparin.

^a minor vascular complications defined as hematoma that did not require intervention;

^b major vascular complications defined as hematoma that required intervention or bleeding that required blood product transfusions;

^c congestive heart failure, hypertension, age >65, diabetes mellitus, prior stroke or transient ischemic attack;

^d hematoma not requiring intervention;

^e cardiac tamponade, hematoma requiring intervention, bleeding requiring transfusion;

^f low stroke risk defined as age <65, no hypertension, no diabetes, no congestive heart failure, no previous stroke; medium stroke risk defined as age >65, no hypertension, no diabetes, no congestive heart failure, no previous stroke; high stroke risk defined as age >65 plus hypertension or diabetes or congestive heart failure or previous stroke OR age >75 without other risk factors;

^g enoxaparin 1 mg/kg BID used from 2001–2004, but then was decreased to 0.5 mg/kg BID from 2005–2008 due to high incidence of vascular complications.

References

1. Fuster V, Rydén LE, Cannom DS, Crijns HJ, et al. European Heart Rhythm Association, Heart Rhythm Society. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation). *J Am Coll Cardiol*. 2006; 48:854–906. [PubMed: 16904574]
2. Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation. Analysis of pooled data from five randomized controlled trials. *Arch Intern Med*. 1994; 154:1449–1457. [PubMed: 8018000]
3. Bornstein N, Corea F, Gallai V, Parnetti L. Heart-brain relationship: atrial fibrillation and stroke. *Clin Exp Hypertens*. 2002; 24:493–499. [PubMed: 12450224]
4. Stroke Risk in Atrial Fibrillation Working Group. Comparison of 12 risk stratification schemes to predict stroke in patients with nonvalvular atrial fibrillation. *Stroke*. 2008; 39:1901–1910. [PubMed: 18420954]
5. Gage BF, Waterman AD, Shannon W, Boechler M, Rich MW, Radford MJ. Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrillation. *JAMA*. 2001; 285:2864–2870. [PubMed: 11401607]
6. Hart RG, Benavente O, McBride R, Pearce LA. Antithrombotic therapy to prevent stroke in patients with atrial fibrillation: a meta-analysis. *Ann Intern Med*. 1999; 131:492–501. [PubMed: 10507957]
7. Gage BF, van Walraven C, Pearce L, Hart RG, Koudstaal PJ, Boode BS, et al. Selecting patients with atrial fibrillation for anticoagulation: stroke risk stratification in patients taking aspirin. *Circulation*. 2004; 110:2287–2292. [PubMed: 15477396]

8. Lip G, Nieuwlaar R, Pisters R, Lane D, Crijns H. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor based approach: The Euro Heart Survey on Atrial Fibrillation. *Chest* Sep. 2009; 17 [Electronic publication ahead of print].
9. Rietbroek S, Heeley E, Plumb J, van Staa T. Chronic atrial fibrillation: Incidence, prevalence, and prediction of stroke using the Congestive heart failure, Hypertension, Age >75, Diabetes mellitus, and prior Stroke or transient ischemic attack (CHADS2) risk stratification scheme. *Am Heart J*. 2008; 156:57–64. [PubMed: 18585497]
10. Scherr D, Sharma K, Dalal D, Spragg D, Chilukuri K, Cheng A, et al. Incidence and Predictors of Periprocedural Cerebrovascular Accident in Patients Undergoing Catheter Ablation of Atrial Fibrillation. 2009; 20(12):1357–1363.
11. Wazni OM, Rossillo A, Marrouche NF, Saad EB, Martin DO, Bhargava M, et al. Embolic events and char formation during pulmonary vein isolation in patients with atrial fibrillation: impact of different anticoagulation regimens and importance of intracardiac echo imaging. *J Cardiovasc Electrophysiol*. 2005; 16:576–581. [PubMed: 15946352]
12. Dorwarth U, Fiek M, Remp T, Reithmann C, Dugas M, Steinbeck G, et al. Radiofrequency catheter ablation: different cooled and noncooled electrode systems induce specific lesion geometries and adverse effects profiles. *Pacing Clin Electrophysiol*. 2003; 26:1438–1445. [PubMed: 12914619]
13. Ren JF, Marchlinski FE, Callans DJ. Left atrial thrombus associated with ablation for atrial fibrillation: identification with intracardiac echocardiography. *J Am Coll Cardiol*. 2004; 43:1861–1867. [PubMed: 15145112]
14. Luther T, Mackman N. Tissue factor in the heart. Multiple roles in hemostasis, thrombosis, and inflammation. *Trends Cardiovasc Med*. 2001; 11:307–312. [PubMed: 11728878]
15. Demolin JM, Eick OJ, Münch K, Koullick E, Nakagawa H, Wittkampf FH. Soft thrombus formation in radiofrequency catheter ablation. *Pacing Clin Electrophysiol*. 2002; 25:1219–1222. [PubMed: 12358173]
16. Sparks PB, Jayaprakash S, Vohra JK, Mond HG, Yapanis AG, Grigg LE, et al. Left atrial "stunning" following radiofrequency catheter ablation of chronic atrial flutter. *J Am Coll Cardiol*. 1998; 32:468–475. [PubMed: 9708477]
17. Cappato R, Calkins H, Chen SA, Davies W, Iesaka Y, Kalman J, et al. Worldwide survey on the methods, efficacy, and safety of catheter ablation for human atrial fibrillation. *Circulation*. 2005; 111:1100–1105. [PubMed: 15723973]
18. Oral H, Chugh A, Ozaydin M, Good E, Fortino J, Sankaran S, et al. Risk of thromboembolic events after percutaneous left atrial radiofrequency ablation of atrial fibrillation. *Circulation*. 2006; 114:759–765. [PubMed: 16908760]
19. Bertaglia E, Zoppo F, Tondo C, Colella A, Mantovan R, Senatore G, et al. Early complications of pulmonary vein catheter ablation for atrial fibrillation: a multicenter prospective registry on procedural safety. *Heart Rhythm*. 2007; 1265–1271. [PubMed: 17905330]
20. Sakurai K, Hirai T, Nakagawa K, Kameyama T, Nozawa T, Asanoi H, et al. Left atrial appendage function and abnormal hypercoagulability in patients with atrial flutter. *Chest*. 2003; 124:1670–1674. [PubMed: 14605033]
21. Singer DE, Albers GW, Dalen JE, Fang MC, Go AS, Halperin JL, et al. Antithrombotic therapy in atrial fibrillation: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest*. 2008; 133:546S–592S. [PubMed: 18574273]
22. Calkins H, Brugada J, Packer DL, Cappato R, Chen SA, Crijns HJ, et al. HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for personnel, policy, procedures and follow-up. A report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation developed in partnership with the European Heart Rhythm Association (EHRA) and the European Cardiac Arrhythmia Society (ECAS); in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), and the Society of Thoracic Surgeons (STS). Endorsed and approved by the governing bodies of the American College of Cardiology, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, and the Heart Rhythm Society. *Europace*. 2007; 9:335–379. [PubMed: 17599941]

23. Stabile G, Bertaglia E, Senatore G, De Simone A, Zoppo F, Donnici G, et al. Catheter ablation treatment in patients with drug-refractory atrial fibrillation: a prospective, multi-centre, randomized, controlled study (Catheter Ablation For The Cure Of Atrial Fibrillation Study). *Eur Heart J*. 2006; 27:216–221. [PubMed: 16214831]
24. Scherr D, Dalal D, Chilukuri K, Dong J, Spragg D, Henrikson CA, et al. Incidence and predictors of left atrial thrombus prior to catheter ablation of atrial fibrillation. *J Cardiovasc Electrophysiol*. 2009; 20:379–384. [PubMed: 19017348]
25. Natale A, Raviele A, Arentz T, Calkins H, Chen SA, Haïssaguerre M, et al. Venice Chart international consensus document on atrial fibrillation ablation. *J Cardiovasc Electrophysiol*. 2007; 18:560–580. [PubMed: 17456138]
26. Scheinman M, Calkins H, Gillette P, Klein R, Lerman BB, Morady F, et al. NASPE policy statement on catheter ablation: personnel, policy, procedures, and therapeutic recommendations. *Pacing Clin Electrophysiol*. 2003; 26:789–799. [PubMed: 12698688]
27. Wazni OM, Beheiry S, Fahmy T, Barrett C, Hao S, Patel D, et al. Atrial fibrillation ablation in patients with therapeutic international normalized ratio: comparison of strategies of anticoagulation management in the periprocedural period. *Circulation*. 2007; 116:2531–2534. [PubMed: 17998456]
28. Mortada ME, Chandrasekaran K, Nangia V, Dhala A, Blanck Z, Cooley R, et al. Periprocedural anticoagulation for atrial fibrillation ablation. *J Cardiovasc Electrophysiol*. 2008; 19:362–366. [PubMed: 18284509]
29. Nademanee K, Schwab MC, Kosar EM, Karwecki M, Moran MD, Visessook N, et al. Clinical outcomes of catheter substrate ablation for high-risk patients with atrial fibrillation. *J Am Coll Cardiol*. 2008; 51:843–849. [PubMed: 18294570]
30. Oral H, Chugh A, Ozaydin M, Good E, Fortino J, Sankaran S, et al. Risk of thromboembolic events after percutaneous left atrial radiofrequency ablation of atrial fibrillation. *Circulation*. 2006; 114:759–765. [PubMed: 16908760]
31. Prudente LA, Moorman JR, Lake D, Xiao Y, Greebaum H, Mangrum JM, et al. Femoral vascular complications following catheter ablation of atrial fibrillation. *J Interv Card Electrophysiol*. 2009; 26:59–64. [PubMed: 19387807]
32. Seow SC, Lim TW, Koay CH, Ross DL, Thomas SP. Efficacy and late recurrences with wide electrical pulmonary vein isolation for persistent and permanent atrial fibrillation. *Europace*. 2007; 9:1129–1133. [PubMed: 17923474]
33. Schmidt M, Segerson NM, Marschang H, Akoum N, Rittger H, Clifford SM, et al. Atrial fibrillation ablation in patients with therapeutic international normalized ratios. *Pacing Clin Electrophysiol*. 2009; 32:995–999. [PubMed: 19659617]
34. Ren JF, Marchlinski FE, Callans DJ, Gerstenfeld EP, Dixit S, Lin D, et al. Increased intensity of anticoagulation may reduce risk of thrombus during atrial fibrillation ablation procedures in patients with spontaneous echo contrast. *J Cardiovasc Electrophysiol*. 2005; 16:474–477. [PubMed: 15877616]
35. Shah AN, Mittal S, Sichrovsky TC, Cotiga D, Arshad A, Maleki K, et al. Long-term outcome following successful pulmonary vein isolation: pattern and prediction of very late recurrence. *J Cardiovasc Electrophysiol*. 2008; 19:661–667. [PubMed: 18284502]
36. He H, Kang J, Tao H, Zhen B, Zhang M, Long D, et al. Conventional oral anticoagulation may not replace prior transesophageal echocardiography for the patients with planned catheter ablation for atrial fibrillation. *J Interv Card Electrophysiol*. 2009; 24:19–26. [PubMed: 18982437]
37. Douketis JD, Berger PB, Dunn AS, Jaffer AK, Spyropoulos AC, Becker RC, et al. The perioperative management of antithrombotic therapy: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest*. 2008; 133:299S–339S. [PubMed: 18574269]
38. Pappone C, Santinelli V. The who, what, why, and how-to guide for circumferential pulmonary vein ablation. *J Cardiovasc Electrophysiol*. 2004; 15:1226–1230. [PubMed: 15485455]
39. Shea JB. Anticoagulation practice in cardiac electrophysiology. *Heart Rhythm*. 2006; 3:372–374. [PubMed: 16500316]

40. Oral H, Pappone C, Chugh A, Good E, Bogun F, Pelosi F Jr, et al. Circumferential pulmonary-vein ablation for chronic atrial fibrillation. *N Engl J Med*. 2006; 354:934–941. [PubMed: 16510747]
41. Bunch TJ, Crandall BG, Weiss JP, May HT, Bair TL, Osborn JS, et al. Warfarin Is Not Needed in Low-Risk Patients Following Atrial Fibrillation Ablation Procedures. *J Cardiovasc Electrophysiol*. 2009; 20:988–993. [PubMed: 19473299]
42. Wells PS, Forgie MA, Simms M, Greene A, Touchie D, Lewis G, et al. The outpatient bleeding risk index: validation of a tool for predicting bleeding rates in patients treated for deep venous thrombosis and pulmonary embolism. *Arch Intern Med*. 2003; 163:917–920. [PubMed: 12719200]
43. Kuijter PM, Hutten BA, Prins MH, Büller HR. Prediction of the risk of bleeding during anticoagulant treatment for venous thromboembolism. *Arch Intern Med*. 1999; 159:457–460. [PubMed: 10074953]
44. Kearon C, Ginsberg JS, Kovacs MJ, Anderson DR, Wells P, Julian JA, et al. Comparison of low-intensity warfarin therapy with conventional-intensity warfarin therapy for long-term prevention of recurrent venous thromboembolism. *N Engl J Med*. 2003; 349:631–639. [PubMed: 12917299]
45. Gage BF, Yan Y, Milligan PE, Waterman AD, Culverhouse R, Rich MW, et al. Clinical classification schemes for predicting hemorrhage: results from the National Registry of Atrial Fibrillation (NRAF). *Am Heart J*. 2006; 151:713–719. [PubMed: 16504638]
46. Katritsis D, Wood MA, Giazitzoglou E, Shepard RK, Kourlaba G, Ellenbogen KA. Long-term follow-up after radiofrequency catheter ablation for atrial fibrillation. *Europace*. 2008; 10:419–424. [PubMed: 18287087]
47. Rossillo A, Bonso A, Themistoclakis S, Riccio G, Madalosso M, Corrado A, et al. Role of anticoagulation therapy after pulmonary vein antrum isolation for atrial fibrillation treatment. *J Cardiovasc Med (Hagerstown)*. 2008; 9:51–55. [PubMed: 18268419]
48. Hindricks G, Piorkowski C, Tanner H, Kobza R, Gerdts-Li JH, Carbucicchio C, et al. Perception of atrial fibrillation before and after radiofrequency catheter ablation: relevance of asymptomatic arrhythmia recurrence. *Circulation*. 2005; 112:307–313. [PubMed: 16009793]
49. Dages N, Hindricks G, Kottkamp H, Sommer P, Gaspar T, Bode K, et al. Real-life anticoagulation treatment of atrial fibrillation after catheter ablation: Possible overtreatment of low-risk patients. *Thromb Haemost*. 2009; 102:754–758. [PubMed: 19806262]

Table 1 The CHADS₂ Scoring System.

A. Independent stroke risk factors comprising the CHADS ₂ score. [5]		
	Independent Stroke Risk Factors	Points
C	<u>C</u> ongestive Heart Failure	1
H	<u>H</u> ypertension	1
A	<u>A</u> ge > 75 years	1
D	<u>D</u> iabetes Mellitus	1
S ₂	<u>S</u> troke or TIA	2
B. CHADS ₂ score with corresponding risk and prophylactic anticoagulation recommendations. [8]		
CHADS ₂ Score	Stroke Risk	Recommended Anticoagulation
0	Low	ASA 81–325 mg daily
1	Intermediate	ASA 81–325 mg daily <i>or</i> warfarin INR 2–3 (dependent upon patient preference and risk/benefit ratio)
≥2	High	Warfarin INR 2–3 (unless contraindicated)

ASA=aspirin; INR=international normalized ratio; TIA=transient ischemic attack.

Table 2

Major risk factors associated with hemorrhage on anticoagulant therapy [42–44].

Increased age (>65 years)	Medication non-adherence, poor clinical follow-up
Female gender	Prior stroke or intracerebral hemorrhage
Diabetes mellitus	Gastrointestinal bleeding or peptic ulcer disease
Active malignancy	Bleeding diathesis (thrombocytopenia, coagulation defects)
Hypertension, uncontrolled	Concurrent aspirin or NSAID administration
Liver disease	INR > 3.0 and INR instability
Chronic kidney disease	Pretreatment INR > 1.2
Anemia	Prior major hemorrhage on therapeutic warfarin (INR 2–3)

INR=international normalized ratio; NSAID=non-steroidal anti-inflammatory drugs.

Table 3**Summary of Peri-Procedural Anticoagulation Recommendations for Atrial Fibrillation Ablation.**

A. Pre-Procedural Recommendations
Warfarin (with documented INR 2.0–3.0) for at least 3 weeks prior to ablation [1,22,25]
Discontinue warfarin 2–5 days prior to procedure and begin LMWH (enoxaparin 1 mg/kg twice daily, with last dose 12 hours before procedure) [22,25]
<i>Consider enoxaparin 0.5 mg/kg twice daily if at increased bleeding risk</i>
OR
Continue warfarin without interruption, maintaining INR 2.0–3.5 [27,33]
B. Intra-Procedural Recommendations
UFH loading dose (100–140 U/kg) prior to, or immediately upon, transeptal puncture [22,25]
UFH continuous infusion (10–18 U/kg/hr) titrated to target ACT [22,25]
• Maintain ACT target of at least 300–350 seconds   • Consider higher ACT target of 350–400 seconds if spontaneous echo contrast or significant atrial enlargement [22,33,34]
Monitor ACT at 10–15 minute intervals until therapeutic, then at 30 minute intervals
Discontinue UFH infusion once all catheters are removed from left atrium [25]
C. Post-Procedural Recommendations
Re-start warfarin evening of procedure or the following morning [22,25]
Re-start LMWH (enoxaparin 0.5–1 mg/kg twice daily) beginning the evening of procedure, and continuing until INR ≥ 2.0 [22,25,33]
<i>Consider using enoxaparin 1 mg/kg for the first 1 or 2 doses and then decreasing to 0.5 mg/kg twice daily until therapeutic INR achieved</i> [22,31,39]
OR
If warfarin was not interrupted, continue warfarin maintaining INR 2.0–3.5 [27,33]
Continue warfarin for at least 2–3 months post-ablation. Beyond that, continuation of warfarin depends on the patient's stroke risk factors (CHADS score ≥ 2 should continue warfarin indefinitely) [22,25]

INR=international normalized ratio; LMWH=low-molecular weight heparin; UFH=Unfractionated Heparin; ACT=Activated Clotting Time.

** Note: These recommendations are not a consensus statement and are limited by the lack of data from randomized, controlled, adequately-powered studies.

Table 4

Summary of the Consensus Guidelines Statements on Peri-Procedural Anticoagulation in Atrial Fibrillation Ablation.

	ACC/AHA/ESC Guidelines for the Management of Patients with Atrial Fibrillation (2006) [1]	Venice Chart International Consensus Document on Atrial Fibrillation Ablation (2007) [25]	HRS/EHRA/ECAS Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation: Recommendations for Personnel, Policy, Procedures and Follow-Up (2007) [22]
Atrial Fibrillation Stroke Risk Stratification Scheme Used	No risk factors ^a = ASA 81–325 mg daily 1 moderate risk factor=ASA 81–325 mg daily or warfarin (INR 2–3) Any high risk factor or > 1 moderate risk factor = warfarin (INR 2–3)	CHADS ₂ scoring system	CHADS ₂ scoring system
Pre-ablation Recommendations	No specific recommendations regarding AF ablation, but recommend anticoagulation for 3 weeks prior to cardioversion for patients with unknown duration or AF or with AF longer than 48 hours	CHADS ₂ score ≥1 or CHADS ₂ score 0 with persistent AF should receive warfarin (documented INR 2–3) for 3 weeks pre-ablation CHADS ₂ score 0 with paroxysmal AF may receive either ASA 75–325 mg daily or warfarin Recommend bridging with IV UFH or SC LMWH (doses not specified)	Recommend adherence to the ACC/AHA/ESC 2006 guidelines regarding anticoagulation for cardioversion “Some Task Force members” recommend bridging with enoxaparin 0.5–1 mg/kg BID until the evening prior to ablation
Intra-ablation Recommendations	“Higher intensity” anticoagulation may reduce intra-procedural thromboembolism	Immediately after left atrial access is established give IV UFH bolus 100–140 units/kg followed by an infusion of 15–18 units/kg/hour ACT target at least 250 s and up to 350–400 s Discontinue UFH infusion after all catheters have been removed from the left atrium	Prior to or immediately on transseptal puncture give IV UFH bolus 100 units/kg followed by an infusion of 10 units/kg/hour ACT target 300–350 s May target ACT 350–400 s if significant atrial enlargement or spontaneous echo contrast is present Discontinue UFH and remove sheaths when ACT < 200 s or reverse UFH with protamine
Post-ablation Recommendations	No specific recommendations regarding AF ablation, but recommend anticoagulation for at least 4 weeks post-cardioversion	Re-start warfarin the evening of procedure or the following morning in all patients May use IV UFH or SC LMWH for bridging until INR ≥2 Continue warfarin for at least 3–6 months	Re-start anticoagulation within 4–6 hours post-sheath removal Use “therapeutic loading doses” of IV UFH or SC enoxaparin and continue until therapeutic INR “Many of the members of this Task Force empirically and independently arrived at a dose of enoxaparin 0.5 mg/kg BID since an unacceptable incidence of bleeding complications has been observed at a dose of 1 mg/kg BID.” Continue warfarin for at least 2 months Determine duration of anticoagulation with warfarin on the patient's risk factors and not on the presence or type of AF

	ACC/AHA/ESC Guidelines for the Management of Patients with Atrial Fibrillation (2006) [1]	Venice Chart International Consensus Document on Atrial Fibrillation Ablation (2007) [25]	HRS/EHRA/ECAS Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation: Recommendations for Personnel, Policy, Procedures and Follow-Up (2007) [22]
		CHADS ₂ score ≥ 2 suggest continue long-term warfarin (INR 2–3). May consider discontinuing warfarin in select patients without evidence of recurrent AF. If warfarin discontinued, recommend ASA 75–325 mg daily unless contraindicated. For others without evidence of recurrent AF consider: CHADS ₂ score 1=ASA or warfarin CHADS ₂ score 0=no treatment	CHADS ₂ score ≥ 2 continue warfarin indefinitely
Other	“The long-term efficacy of catheter ablation to prevent recurrent AF requires further study...it remains uncertain whether apparent cures represent elimination of AF or transformation into an asymptomatic form of paroxysmal AF. The distinction has important implications for the duration of anticoagulation therapy in patients with risk factors for stroke associated with AF.”	“An overlying concern is the absence of long-term data in large numbers of patients following catheter ablation of AF. We need a prospective study not only on the incidence of recurrence of AF beyond the ‘blanking period,’ but also on the incidence of stroke in the absence of anticoagulation therapy, especially in patients with risk factors for stroke. Data from relatively small studies suggest that the stroke incidence is low, but the incidence of AF recurrence, both manifest and asymptomatic, is uncertain. Future well-conducted randomized trials have to establish the best anticoagulation protocol after ablation of AF, according to the clinical characteristics of the patients, especially their thromboembolic risk.”	“...the Task Force recognizes that patients who have undergone catheter ablation of AF represent a new and previously unstudied population of patients. Clinical trials are therefore needed to define the stroke risk of this patient population and to determine whether the risk factors identified in CHADS or other scoring systems apply to these patients.”

ACC=American College of Cardiology; ACT=activated clotting time; AF=atrial fibrillation; AHA=American Heart Association, ASA=aspirin; BID=twice daily; CAD=coronary artery disease; DM=diabetes mellitus; ECAS=European Cardiac Arrhythmia Society; EHRA=European Heart Rhythm Association; ESC=European Society of Cardiology; HF=heart failure; HRS=Heart Rhythm Society; HTN=hypertension; INR=international normalized ratio; IV UFH=intravenous unfractionated heparin; LVEF=left ventricular ejection fraction; s=seconds; SC LMWH=subcutaneous low molecular weight heparin; TIA=transient ischemic attack.

^a *Less validated or weaker risk factors:* female gender, age 65–74, CAD, thyrotoxicosis; *Moderate risk factors:* age ≥ 75 , HTN, HF, LVEF $\leq 35\%$, DM; *High risk factors:* previous stroke, TIA, or embolism, mitral stenosis, prosthetic heart valve.