

# EDUCATIONAL ARTICLE

## Key Issues in the Management of Atrial Fibrillation - Protecting the Patient and Controlling the Arrhythmia

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### Abstract

Atrial fibrillation (AF) is the most common sustained tachyarrhythmia and its prevalence is increasing. It is an independent risk factor for stroke and is associated with significant morbidity and mortality. AF currently accounts for 1% of NHS expenditure. The management of AF has a broad evidence base and both the American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) and the National Institute for Clinical Excellence (NICE) have recently published guidelines. Some controversy persists regarding stroke risk stratification and appropriate anticoagulation regimes although a general consensus is now emerging. Rate and rhythm control strategies have been shown to be comparable in terms of clinical outcomes. Current anti-arrhythmic drugs have limited efficacy and significant side-effect profiles. Electrophysiological and surgical interventions have a role in both strategies. This article broadly reviews the evidence for different management strategies in AF and presents a practical approach to treatment in light of the recently published national and international guidelines.

### Introduction

Atrial fibrillation (AF) is the most common sustained tachyarrhythmia and is associated with significant morbidity and mortality.<sup>1</sup> It is particularly prevalent in the elderly, affecting 10% of those > 80 years.<sup>2</sup> AF currently accounts for 1% of NHS expenditure and this is likely to increase with our ageing population.<sup>2</sup> This article broadly reviews the evidence for different management strategies in AF

and presents a practical approach to treatment in light of the recently published national and international guidelines.<sup>1,3</sup>

### Clinical Assessment

All patients presenting with AF should have a formal clinical assessment. In addition to a history and physical examination we would recommend routine blood tests including thyroid function tests, electrocardiography, chest radiography and transthoracic echocardiography in all patients. In some patients ambulatory electrocardiogram (ECG) monitoring may be appropriate. These investigations may identify reversible precipitating factors for the AF. Moreover they will facilitate accurate stroke risk stratification and influence the decision to adopt either a rate control or rhythm control strategy.

### Thromboprophylaxis

#### Stroke Risk

AF is an independent risk factor for stroke irrespective of age or symptomatology.<sup>4,5,6</sup> The reported rate of ischaemic stroke in patients with non-valvular AF varies from 5% to 7% per year.<sup>4,7</sup> In AF associated with rheumatic heart disease the attributable stroke risk is five times greater than in non-valvular AF.<sup>4</sup> Paroxysmal atrial fibrillation (PAF) was traditionally considered to equate to a lower stroke risk than persistent AF<sup>8</sup> but this was not supported by multivariate analyses.<sup>9,10</sup> It is well recognised that when stroke occurs in association with AF there is higher mortality and morbidity.<sup>11</sup>

## **Aspirin**

A meta-analysis of six large placebo controlled trials demonstrated that aspirin, in doses varying from 50mg to 1200mg daily, reduced the incidence of stroke in AF by 22% (95% Confidence Interval (CI) 2% to 38%) with no significant increase in haemorrhage.<sup>12</sup> This translates as an absolute risk reduction of 1.5% per year for primary prevention and 2.5% per year for secondary prevention. This is similar to the stroke risk reduction with antiplatelet use in vascular disease and may therefore simply represent the general benefits of aspirin in these patients.<sup>13</sup> Interestingly, while the six randomized controlled trials (RCTs) all individually showed trends towards reduced stroke risk with aspirin, this was statistically significant in only one study.<sup>14</sup> This study reported the highest proportion of non-disabling stroke (52%), and the relative risk reduction with aspirin was highest for these patients.

There is some controversy regarding aspirin dosage in AF thromboprophylaxis. The recent large Antithrombotic Trialists' Collaboration meta-analysis found the proportional reduction in vascular events to be 19% with aspirin 500mg to 1500mg daily, 26% with 160mg to 325mg daily and 32% with 75mg to 150mg daily.<sup>15</sup> The American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) guidelines in 2001 recommended aspirin 325mg for low risk patients whereas the more recent guideline recommended a broader dose range from 81mg to 325mg.<sup>1,16</sup> We feel that aspirin 150mg represents a reasonable compromise and this is our current policy for thromboprophylaxis when aspirin is considered appropriate. If the patient is aspirin intolerant then we would recommend clopidogrel 75mg.

## **Warfarin**

The largest systematic review and meta-analysis of thromboprophylaxis trial data in AF demonstrated that adjusted-dose warfarin significantly reduced the risk of ischaemic stroke or systemic embolism compared to aspirin (Relative Risk (RR) 0.59; 95% CI 0.40 to 0.86).<sup>17</sup> While adjusted-dose warfarin reduced mortality compared to placebo (RR 0.69;

95% CI 0.53 to 0.89), there was no significant difference in mortality when compared to aspirin (RR 0.87; 95% CI 0.67 to 1.13). Predictably aspirin had a lower risk of major bleeding when compared to warfarin (RR 0.58; 95% CI 0.35 to 0.97). This meta-analysis confirmed results of previous trials indicating no benefit from fixed low dose warfarin. A sub-therapeutic International Normalised Ratio (INR), therefore, does not confer any protective effects.

It is essential to assess bleeding risk prior to implementing anticoagulation with warfarin. NICE suggest exercising caution in patients who: are aged > 75 years; are on concomitant antiplatelet or non-steroidal anti-inflammatory drugs; are on multiple drug treatments; have uncontrolled hypertension; have a history of bleeding; or have a history of poorly controlled anticoagulation.<sup>3</sup> Both the ACC/AHA/ESC and the NICE guidelines highlight the need for initiation of thromboprophylaxis with minimal delay after establishing a diagnosis of AF.<sup>1,3</sup> The benefits and potential risks of long-term anticoagulation clearly need to be discussed with the patient. The decision to commence warfarin should be individually tailored to the patient with consideration of various other factors in addition to stroke risk. These include the patient's co-morbidities and commitment to long-term anticoagulation. Discussion with the patient's general practitioner often provides valuable information.

## **Aspirin and Warfarin**

The addition of antiplatelet therapy to warfarin for patients with AF and concomitant coronary artery disease (CAD) or cerebrovascular disease is controversial. While there is no real evidence for this strategy it is common in clinical practice.<sup>1</sup> A recent meta-analysis of ten trials with a total of 4180 patients found no difference in the risk of arterial thromboembolism between AF patients on aspirin and oral anticoagulation vs oral anticoagulation alone (Odds Ratio (OR) 0.99; 95% CI 0.47 to 2.07).<sup>18</sup> Furthermore, there was no difference in all-cause mortality between the two groups. Of importance, major bleeding was significantly higher in patients

receiving both aspirin and oral anticoagulation compared with oral anticoagulation alone (OR 1.43; 95% CI 1.00 to 2.02). Similarly, in the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) trial the most important predictor of bleeding in anticoagulated patients was concomitant aspirin therapy.<sup>19</sup>

The ACC/AHA/ESC guidelines state that for most patients with AF and stable CAD, warfarin anticoagulation alone (INR 2.5) should provide satisfactory antithrombotic prophylaxis against both cerebral and myocardial events.<sup>1</sup> They do, however, recommend that clopidogrel be administered in addition to warfarin for 9 to 12 months following insertion of a bare metal or drug eluting stent into a coronary artery.<sup>1</sup> Our current policy is to give aspirin 75mg in addition to warfarin in patients with AF and previous coronary artery bypass grafting or angiographic evidence of CAD. We do, however, recognize the divergent views regarding this.

### **Risk Stratification**

Various risk stratification schemes have been generated aimed at identifying high and moderate risk patients with AF who are likely to benefit most from warfarin therapy. These schemes are based on multivariate analyses of prospective cohorts of AF patients prescribed aspirin or no antithrombotic therapy, yielding independent predictors of stroke.

The CHADS2 risk stratification scheme has been well validated.<sup>20-22</sup> CHADS2 is an acronym for Congestive heart failure, Hypertension, Age > 75 years, Diabetes mellitus, and prior Stroke or transient ischaemic attack (TIA). Each risk factor is assigned 1 point except prior Stroke or TIA which is assigned 2 points. It has been demonstrated that adjusted stroke rate increases with increasing CHADS2 score (Table I).<sup>20,21,22</sup>

Despite the validation of CHADS2, therapeutic guidelines for intermediate scores have not yet been established. Furthermore, the CHADS2 scheme presents a significant contradiction. A patient with a previous stroke or TIA, if < 75 years of age with no other risk factors would have a CHADS2 score of 2.

This corresponds to only a moderate risk of stroke when clearly a patient with AF and previous stroke is at high risk and should be anticoagulated with warfarin.

Neither the ACC/AHA/ESC guidelines nor NICE specifically advocate the use of the CHADS2 scheme in AF.<sup>1,3</sup> Their recommendations for aspirin and warfarin thromboprophylaxis in AF are based on the presence or absence of well defined moderate or high risk factors and are summarised in tables II and III. The guidelines for thromboprophylaxis are the same irrespective of whether the patient has paroxysmal, persistent or permanent AF.

### **Rate or Rhythm Control**

A key decision in the management of AF is whether to accept the AF and adopt a rate control strategy or whether to attempt cardioversion to sinus rhythm (SR) and aim for rhythm control. Both rate control and rhythm control strategies aim to lessen morbidity as measured by a reduction in symptoms and hospital admissions and an improvement in exercise tolerance and quality of life. Rate control is achieved by administration of rate-limiting agents. Rhythm control involves either electrical or pharmacological cardioversion to restore sinus rhythm (SR) with or without maintenance anti-arrhythmic drugs (AADs). Electrophysiological and surgical interventions have a role in both strategies but particularly in rhythm control.

Traditionally rhythm control was the preferred approach in most patients due to a perceived lower risk of thromboembolism and postulated morbidity and mortality benefits. However, five large RCTs have demonstrated comparable outcomes irrespective of treatment strategy.<sup>23,24,25,26,27</sup> In the largest trial there was an almost significant trend towards reduced all cause mortality in the rate control group.<sup>23</sup> The excess mortality in the rhythm control group may have been due to the administration of AADs. Indeed, a secondary analysis of the AFFIRM trial found that the presence of SR did confer a survival advantage but only after adjustment for the use of AADs.<sup>28</sup>

The AFFIRM trial also demonstrated a trend towards increased risk of ischaemic stroke in the rhythm control group.<sup>28</sup> This was considered to be due to suboptimal anticoagulation which was the case in 72% of strokes during follow-up. No RCT has demonstrated an improvement in quality of life with either strategy.<sup>23,24,25,26,27</sup> Significant improvements in exercise tolerance and NYHA class in the rhythm control group were offset by increased hospitalisation in two studies.<sup>23,25</sup> The failure of rhythm control to confer any significant advantage may have been secondary to the limited efficacy of AADs. The percentage of patients in the rhythm control group actually maintained in SR at the end of follow-up was 63% at 5 years in AFFIRM,<sup>23</sup> 23% at 3 years in Strategies of Treatment of Atrial Fibrillation (STAF),<sup>26</sup> 39% at 2.3 years in Rate Control versus Electrical Cardioversion (RACE)<sup>24</sup> and 64% at 1.7 years in How to Treat Chronic Atrial Fibrillation (HOT CAFÉ).<sup>27</sup> The choice of treatment strategy is, therefore, dependent on the patient's age, symptoms, co-morbidities and preference (Table IV). Minimizing stroke risk with appropriate anticoagulation is imperative irrespective of treatment strategy.<sup>1,3</sup>

## Rate Control

The purpose of rate control in AF is to prevent symptoms and haemodynamic instability while reducing the risk of developing tachycardia-associated cardiomyopathy. This requires heart rates to be controlled both at rest and during exercise. It is unclear what constitutes adequate rate control and no randomized clinical trials have validated target heart rates. Guidelines suggest a rate of 60 to 90 beats per minute at rest and < 115 beats per minute with moderate exercise.<sup>1,3</sup>

Beta-blockers or rate-limiting calcium-channel antagonists (CCAs) are first line therapy for rate control.<sup>1,3</sup> Both drugs have been extensively validated against placebo.<sup>29</sup> Bisoprolol or carvedilol are preferred if there is concomitant stable left ventricular failure. Digoxin is not recommended as monotherapy except in sedentary individuals or patients with decompensated heart failure due to its limited efficacy in exercise.<sup>1,3</sup> However, digoxin is

useful as an add-on treatment. In particular the combination of digoxin and atenolol produces a synergistic effect on the AV node and is considered more effective than a CCA with digoxin.<sup>30</sup> Evaluation of the efficacy of rate control treatment in the AFFIRM trial demonstrated that beta blockers both with and without digoxin provided better rate control than CCAs.<sup>31</sup>

The Class III AAD amiodarone is second line therapy for rate control.<sup>1,3</sup> It is not deemed appropriate for initial treatment due to its significant side effect profile. A maintenance dose of 100mg rather than 200mg may be effective, reducing the risk of adverse effects.<sup>32</sup> When rate control is sub-optimal it is important to exclude other factors that may be driving the rate e.g. cardiac failure, thyrotoxicosis or infection. If rate control is inadequate despite optimal pharmacological treatment then referral for an "ablate and pace strategy" may be warranted. This involves laser ablation of the AV node with permanent pacing and may result in significant symptomatic improvement.<sup>33</sup>

## Rhythm Control

Rhythm control also aims to alleviate symptoms and AF mediated morbidity. This may initially involve restoration of SR by cardioversion. This is achieved either electrically by Electrical (Direct Current) Cardioversion (DCV) or pharmacologically (PCV). Thereafter, maintenance AAD therapy is often appropriate. Chronic AAD therapy is frequently used in patients with PAF.

### **Restoration of SR in Acute AF**

Emergency DCV is the treatment of choice for patients with life-threatening haemodynamic instability due to AF.<sup>1,3</sup> This is true regardless of the duration of AF. These patients should be anticoagulated with heparin immediately prior to DCV.

In the less acute situation where the duration of AF is less than 48 hours then either PCV or DCV may be appropriate. It has been demonstrated that these cardioversion strategies are equally

effective.<sup>34</sup> However, based on clinical experience, it is accepted that PCV is preferable for AF of less than 48 hours.<sup>35</sup> Class Ic (propafenone and flecainide) and Class III (amiodarone and sotalol) AADs are commonly used for PCV. These drugs have been shown to have comparable efficacies after a 24 hour period.<sup>35,36,37</sup> Sotalol should be used with caution as it may significantly prolong the QT interval. Class Ic drugs are contraindicated in the presence of structural heart disease and accordingly guidelines advocate the use of amiodarone in such patients.<sup>1,3</sup> In patients without structural heart disease flecainide or propafenone is suggested as first line PCV therapy.<sup>1,3</sup> Patients undergoing cardioversion should be anticoagulated with heparin and oral warfarin should be commenced if SR is not achieved within 48 hours.<sup>1,3</sup> It should be recognised, however, that thromboembolism remains a risk even when cardioversion is successful within 48 hours.<sup>38</sup>

### **Restoration of SR in Persistent AF**

Electrical (Direct Current) Cardioversion (DCV) is the most appropriate strategy for cardioversion of AF of duration > 48 hours.<sup>35</sup> Successful cardioversion becomes less likely as duration of AF increases.<sup>39</sup> Early referral for this strategy is therefore beneficial.

The ACC/AHA/ESC and NICE guidelines both recommend a period of anticoagulation of at least 3 weeks prior to elective DCV for persistent AF.<sup>1,3</sup> Thereafter they advocate continuing anticoagulation for a minimum of 4 weeks. This recommendation is based on studies demonstrating that atrial mechanical function may not normalise for several weeks.<sup>40</sup> Long term anticoagulation should be considered in patients at high risk of AF recurrence.<sup>1,3</sup> Such patients may have AF of longer than 12 months duration, mitral valve disease, left ventricular systolic dysfunction, an enlarged left atrium or a previous failed cardioversion.

There is evidence that amiodarone therapy prior to DCV increases the rate of restoration to SR.<sup>41</sup> In some cases it allows pharmacological cardioversion (PCV), hence negating the need for DCV.<sup>42</sup> Furthermore, administration of amiodarone for up to 12 months after successful DCV reduces the rate of

AF recurrence.<sup>42</sup> Published guidelines recommend pre and post DCV treatment with amiodarone only for patients with a high risk of DCV failure or AF recurrence.<sup>1,3</sup>

It is our policy to routinely pre-treat with amiodarone once INR is therapeutic to ensure a high probability of success at the first attempt. Following successful cardioversion we would recommend continuing amiodarone and warfarin for 12 months in most patients. Studies suggest that while around 50% of failed cardioversions manifest within 2 weeks,<sup>43</sup> a further 25% of patients may revert to AF by 12 months.<sup>44</sup> Warfarin can be discontinued after 12 months provided ambulatory ECG monitoring confirms SR. If the patient is at high risk of AF recurrence then we would recommend continuing amiodarone. The dose of amiodarone can often be reduced to 100mg to minimise adverse effects.<sup>32</sup>

### **Maintenance of SR in Paroxysmal AF**

Patients with PAF can be highly symptomatic. Management may involve suppression of arrhythmia, rate control of paroxysms or both. Patients should be anticoagulated according to their overall stroke risk.<sup>1,3</sup> In young patients without structural heart disease the Class Ic antiarrhythmic drugs (AADs) such as flecainide and propafenone are most effective. Flecainide is regarded by many as the drug of choice and if paroxysms are rare a "pill in the pocket" approach may be appropriate.<sup>45</sup> Class Ic AADs are contraindicated in structural heart disease and amiodarone is regularly used as an alternative. In line with the NICE guidelines it is our policy to attempt maintenance of SR with a standard beta-blocker such as atenolol or bisoprolol and reserve the higher risk AADs for patients where beta-blockers are ineffective.<sup>3</sup>

### **Radiofrequency Ablation**

In the last decade radiofrequency ablation (RFA) has emerged as a treatment for symptomatic AF refractory to AAD therapy. The success rate of this procedure may be as high as 90% in PAF.<sup>46</sup> More recently studies have evaluated the efficacy of RFA

in patients with persistent and permanent AF and reported success rates up to 75%.<sup>47,48,49</sup> However, many of these patients require multiple procedures and some require chronic administration of AADs. Furthermore, follow-up studies of RFA where symptoms are used as a marker of AF may overestimate success. One recent study demonstrated that following RFA, AF recurrences were asymptomatic in 37% of patients.<sup>50</sup> Moreover, RFA is associated with several significant complications including thromboembolic events, pulmonary vein stenosis and cardiac tamponade. A recent worldwide survey comprising information on 8745 RFA procedures reported the occurrence of major complications in 6%.<sup>51</sup> The ACC/AHA/ESC guidelines consider RFA to be an appropriate strategy in symptomatic patients with little or no left atrial enlargement. This is, however, a Class IIa recommendation based on Level C evidence.<sup>1</sup> NICE suggests referral for consideration of RFA in patients with AF refractory to AADs or in patients with lone AF.<sup>3</sup>

## Future Directions in AF

### **Rhythm Control**

The indications for RFA for achieving and maintaining SR in patients with AF are likely to broaden as experience with the technique increases. Refinements in catheter technology along with improvements in anatomic and electrophysiologic mapping are expected to make future RFA procedures both faster and more effective. There is now some evidence that RFA may be feasible as a first line treatment strategy in symptomatic AF.<sup>52</sup>

Future AADs need to be more efficacious with better side-effect profiles. Some recent research has focused on the use of other pharmacological agents in promoting SR. As electrical and structural remodelling in the atrium may initiate or perpetuate AF, there is considerable interest in drugs that may inhibit this process.<sup>53</sup> The anti-oxidant and anti-inflammatory properties of statins may reduce electrical remodelling.<sup>54</sup> A recent large prospective cohort study found statins significantly reduced the

development of AF in patients with coronary artery disease.<sup>55</sup> Activation of the renin-angiotensin-aldosterone system (RAAS) is thought to contribute to both electrical and structural remodelling.<sup>56</sup> A recent meta-analysis of eleven RCTs comprising 56308 patients demonstrated that RAAS blockade reduced the risk of AF by 28% (95% CI 15% to 40%).<sup>57</sup> This was true irrespective of whether an ACE inhibitor or an Angiotensin II Receptor Blocker (ARB) was administered.

### **Thromboprophylaxis**

There is much interest in the use of oral direct thrombin inhibitors and oral Factor Xa inhibitors as an alternative to warfarin. These drugs have fewer drug and alcohol interactions and do not require dose adjustment or monitoring. The Stroke Prevention with the Oral Direct Thrombin Inhibitor Ximelegatran Compared with Warfarin in Patients with Non-Valvular Atrial Fibrillation (SPORTIF) III and V trials demonstrated non-inferiority of the oral direct thrombin inhibitor ximelegatran to adjusted-dose warfarin in moderate to high risk patients with non-valvular AF.<sup>58,59</sup> A recent meta-analysis confirmed ximelegatran was as effective as adjusted-dose warfarin in the prevention of ischaemic strokes or systemic emboli.<sup>17</sup> However, subsequent reports of significant adverse liver enzyme effects in the ximelegatran group prevented its introduction into clinical practice. Trials of subcutaneous and oral Factor Xa inhibitors in comparison to adjusted-dose warfarin are on-going. Future thromboprophylaxis strategies may also include a more invasive approach where the left atrial appendage is closed percutaneously. A recent feasibility study has produced encouraging results but further evaluation and follow-up studies are required.<sup>60</sup>

### **Conclusion**

AF is one of the most common disorders in cardiology practice. It impacts significantly on primary, secondary and tertiary care. There are numerous causes and precipitating factors that must be promptly elucidated as they will impact on effective management. The patient is likely to derive significant benefit from introduction of appropriate

thromboprophylactic treatment and the correct choice of a rate or rhythm control strategy. While there have been improvements in the management of this ever increasing condition there is no room for complacency with recent evidence demonstrating under-utilisation of treatment, particularly in women and in the elderly, in whom AF is so common.<sup>61</sup>

**Table I** Validation of CHADS<sub>2</sub><sup>20</sup>

CHADS <sub>2</sub> Score	Adjusted Stroke Rate %/yr (95% CI)
0	1.9 (1.2 to 3.0)
1	2.8 (2.0 to 3.8)
2	4.0 (3.1 to 5.1)
3	5.9 (4.6 to 7.3)
4	8.5 (6.3 to 11.1)
5	12.5 (8.2 to 17.5)
6	8.2 (10.5 to 27.4)

**Table II** Aspirin Thromboprophylaxis in AF - ACC/AHA/ESC<sup>1</sup> and NICE<sup>3</sup> Guidelines 2006

ACC/AHA/ESC guidelines 2006	NICE guidelines 2006
Aspirin 81mg - 325mg No Risk Factors	Aspirin 75mg - 300mg Age < 65 + No Risk Factors
Aspirin 81mg - 325mg or Warfarin INR 2.5 One Moderate Risk Factor: <ul style="list-style-type: none"> <li>● Age ≥ 75</li> <li>● Hypertension</li> <li>● Diabetes Mellitus</li> <li>● Heart Failure</li> <li>● LV Ejection Fraction &lt; 35%</li> </ul>	Aspirin 75mg - 300mg or Warfarin INR 2.5 Age 65 - 74 + No Risk Factors Age < 75 + One Moderate Risk Factor: <ul style="list-style-type: none"> <li>● Hypertension</li> <li>● Diabetes Mellitus</li> <li>● Vascular Disease</li> </ul>

**Table III** Warfarin Thromboprophylaxis in AF - ACC/AHA/ESC<sup>1</sup> and NICE<sup>3</sup> Guidelines 2006

ACC/AHA/ESC guidelines 2006 - Warfarin INR 2.5	NICE guidelines 2006 - Warfarin INR 2.5
Aspirin 81mg - 325mg No Risk Factors	Aspirin 75mg - 300mg Age < 65 + No Risk Factors
Any High Risk Factor <ul style="list-style-type: none"> <li>● Previous CVA, TIA or Embolism</li> <li>● Mitral Stenosis</li> <li>● Prosthetic Heart Valve</li> </ul> > One Moderate Risk Factor	Any High Risk Factor <ul style="list-style-type: none"> <li>● Previous CVA, TIA or Embolism</li> <li>● Age " 75 + HTN, DM or Vascular Disease</li> <li>● Clinical evidence of Valve Disease</li> <li>● Clinical HF or Impaired LV Function on Echo</li> </ul>

CVA, Cerebrovascular accident; TIA, Transient ischaemic attack; HTN, Hypertension; DM, Diabetes mellitus; LV, Left ventricle.

**Table IV** Initial Strategy Choice - NICE Guidelines 2006<sup>3</sup>

Rate Control Strategy	Rhythm Control Strategy
Age > 65 Coronary Artery Disease CIs to AADs CIs to Cardioversion No Heart Failure	Younger Patients Symptomatic 1st Episode of Lone AF AF 2° Treated Precipitant Heart Failure

CIs, Contraindications; AADs, Anti-arrhythmic drugs

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## Best of Five Questions

1. *An 82 year old woman was referred to the outpatient clinic with recent onset breathlessness on exertion. Clinical examination was consistent with atrial fibrillation and this was confirmed by ECG with a ventricular rate of 100 beats/min. The patient was referred to the anticoagulant clinic and Warfarin was commenced.*

Which of the following additional drugs would be the most appropriate to prescribe next?

- A Amiodarone
- B Bisoprolol
- C Digoxin
- D Flecainide
- E Sotalol

2. *A 59 year old man presented to the TIA clinic following an episode of left arm weakness and dysphasia lasting ten minutes. There was no past history of hypertension, diabetes or any cardiac disorder. Clinical examination was normal. Resting ECG revealed sinus rhythm and was normal as was transthoracic echocardiography. A 48hr ambulatory ECG monitor revealed a single run of atrial fibrillation lasting 20 beats.*

Which of the following drugs would be most appropriate?

- A Aspirin
- B Clopidogrel
- C Dipyridamole
- D Warfarin
- E Warfarin plus low dose Aspirin

3. *A 37 year old man with paroxysmal AF had attended the cardiology clinic for 9 months. An initial attempt to maintain SR with bisoprolol had failed. As his heart was structurally normal he was commenced on flecainide. Despite recent increases in his dose of flecainide he continued to be troubled with frequent symptomatic paroxysms of AF with an uncontrolled ventricular rate. Resting ECG in clinic showed SR with a rate of 64 beats/min.*

Which of the following would be most appropriate at this stage?

- A Amiodarone
- B Sotalol
- C AV node ablation and permanent pacing
- D Radiofrequency ablation
- E Diltiazem