The management of atrial fibrillation is often complex, and can involve a variety of drugs as well as surgery to reduce the risks associated with it, especially stroke.

**Atrial Fibrillation: Part 2**

# Treating and managing atrial fibrillation

## In this article...
- Investigations to assess for treatment
- Efficacy of treatments
- Treatment to control symptoms

## Authors
Shona Holding is arrhythmia nurse specialist at Leeds Community Health Care Trust; Craig Russell and Keith Tyndall are arrhythmia nurses at Leeds Teaching Hospitals Trust.

## Abstract

The management of atrial fibrillation is complex and is influenced by the type of AF, the severity of symptoms, underlying disease and patient choice. The aim of treatment is to alleviate symptoms, prevent strokes and reduce other complications, such as heart failure.

The incidence of AF is increasing due to an ageing population and most health professionals will encounter patients with AF during their career. A widespread knowledge of AF management among the nursing profession is important to ensure that appropriate treatment and patient support are provided.

This article is the second in a two-part series on AF. Part one discussed the importance of detecting and treating AF and screening strategies. This second part discusses the management of AF and treatment options, using recent guidelines.

Atrial fibrillation is becoming more common in today’s ageing population due to its age-related prevalence (Sumeet et al, 2001). The impact of AF on mortality and morbidity is significant and it accounts for 15% of thromboembolic strokes and approximately 1% of NHS expenditure (Miller et al, 2005). It is therefore critical that all nurses have a good understanding of its management and treatment options.

AF management is often complex and determined by the type of AF (Table 1), severity of symptoms, underlying disease and patient choice (Camm et al, 2010; National Collaborating Centre for Chronic Conditions, 2006). The aim of treatment is to maximise thromboprophylaxis, reduce other complications such as heart failure, and alleviate symptoms by restoring sinus rhythm or controlling the ventricular rate.

There are a number of treatments, and guidance on AF management is provided by the National Institute for Health and Clinical Excellence (2013) and the European Society of Cardiologists (Camm et al, 2010; 2012). Guidelines are regularly updated as new drugs such as oral anticoagulants and anti-arrhythmics become available.

## Initial investigations
Before decisions can be made on treatment, patients must have a 12-lead electrocardiogram (ECG) to confirm evidence of the arrhythmia (Lip et al, 2011). The ECG may also detect evidence of coronary heart disease, left ventricular enlargement or a pre-excitation syndrome, such as Wolf-Parkinson-White syndrome.

Sometimes ambulatory monitoring is needed to capture the arrhythmia, particularly if symptoms are intermittent. A portable, battery-operated recorder can be worn on a belt attached to three electrodes on the patient’s chest. This is worn for between 24 hours and seven days.

**5 key points**

1. Atrial fibrillation management is complex and depends on AF type, symptoms and stroke risk
2. All patients with AF should be risk stratified for stroke and considered for anticoagulation therapy
3. There is little difference in bleed rates between warfarin and low-dose aspirin but warfarin is more effective in stroke prevention
4. Symptoms can be controlled with drugs, cardioversion or surgery
5. Electro-physiology procedures are considered in patients who have poor symptom control despite medication

---

**ECG showing atrial fibrillation**

---

Keywords: Atrial fibrillation/Treatment/Controlling symptoms/Arrhythmia

● This article has been double-blind peer reviewed
Most patients with new-onset AF will have a transthoracic echocardiogram. This is a non-invasive ultrasound imaging of the heart to determine left atrial size and left ventricular (LV) dimensions, such as LV wall thickness, LV function and movement. It also establishes the presence of valvular disease or clot formation.

Biochemical testing is used to identify abnormalities in blood count, renal, liver and thyroid function. A chest X-ray may also be needed if clinically indicated (NCC-CC, 2006).

**Optimising thromboprophylaxis**

Once AF is diagnosed, it is essential to complete a stroke risk assessment before considering treatment. Risk assessment using the CHADS2 score and more recent CHA2DS2VASc schemes, as explained in part 1, are recommended (Camm et al, 2010).

Oral anticoagulation therapy (OAC) is widely recognised as the most effective treatment for stroke prevention in non-valvular AF (absence of mitral valve disease or a mechanical valve) but it remains underused (Cowan et al, 2013; NCC-CC, 2006). OAC is three to four times more effective at reducing AF-related strokes than aspirin (Hart et al, 2007). Despite this, health professionals are often wary of prescribing warfarin due to perceived bleeding risk, particularly in older people. However, evidence from the Birmingham Atrial Fibrillation Treatment of the Aged (BAFTA) trial suggests aspirin is no less likely than warfarin to cause major bleeding and warfarin is far more effective in stroke prevention (Mant et al, 2007).

Recent guidelines advocate people at low risk (under 65 with no risk factors) should be managed with no antithrombotic treatment and those with one or more risk factors be considered for OAC (Camm et al, 2010). However, aspirin is still commonly used in place of OACs (Cowan et al, 2013). Reasons for underuse of warfarin include inconvenience to patients due to regular blood tests and variable doses, and concurrent indication for anti-platelets such as ischaemic heart disease. Maintaining a therapeutic INR can be an ongoing challenge for a variety of reasons and, unless time in therapeutic range (TTR) of 60-70% is maintained, the benefits of warfarin are greatly reduced (Hylek et al, 2007).

Novel oral anticoagulants (NOAC) for stroke prevention in non-valvular AF, which do not require regular monitoring, are therefore welcome. These new agents fall into two categories: oral direct thrombin inhibitors, such as dabigatran, and oral direct factor Xa inhibitors, for example rivaroxaban and apixaban. All three have NICE approval and are recommended as an “option for the prevention of stroke and systemic embolism in those with non-valvular atrial fibrillation with one or more risk factors such as congestive heart failure, hypertension, aged 75 years or older, diabetes mellitus and prior stroke or transient ischaemic attack” (NICE, 2013).

The main advantages of NOACs are that they do not require regular monitoring, have fewer food and drug interactions than warfarin and are given as a daily or twice-daily fixed dose, which should help with adherence. They have all been shown to be equivalent to warfarin in terms of stroke reduction (Camm et al, 2012). The variability in NOACs means more guidance is needed to help prescribers and patients choose the agent best suited to the individual. A NOAC may be considered over warfarin when the patient has:

- Poor INR control;
- Physical limitations that interfere with blood monitoring;
- Allergy to warfarin;
- Needle phobia;
- Difficult venepuncture;
- Decided to use a NOAC rather than warfarin.

Disadvantages of NOACs include cost and unknown long-term effects. At present, there is no licensed direct reversal agent, although these are in development. Bleeding associated with these drugs is currently treated with adjunctive supportive therapy such as fresh frozen plasma and transfusion.

It is hoped that educational events on stroke prevention and AF, including NOACs and the input of arrhythmia nurses, will help prescribers to become more confident about anticoagulants.

**Bleeding risk**

Bleeding is a major concern when using any OAC and is often the reason why they are not being used. Recent guidelines recommend the HAS-BLED score (Table 2).

This user-friendly tool identifies risk factors that can be actively managed to reduce the risk of bleeding, such as concomitant medications, hypertension and excessive alcohol intake (Lip et al, 2011; Camm et al, 2010; Pisters et al, 2010). Patients with a HAS-BLED score of three or above are deemed to have a high risk of bleeding; however, risk factor management is recommended before deciding against using an OAC (Camm et al, 2010).

---

**TABLE 1. TYPES OF ATRIAL FIBRILLATION**

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>First diagnosis AF</td>
<td>Applies to all patients presenting with AF for the first time</td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>Self-terminating, usually within seven days</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>Has episodes lasting longer than seven days, is continuous and responds to pharmaceutical or electrical cardioversion</td>
</tr>
<tr>
<td>Long-standing persistent AF</td>
<td>Lasting one year or more before a rhythm control strategy is adopted</td>
</tr>
<tr>
<td>Permanent AF</td>
<td>The arrhythmia is accepted by the patient and physician</td>
</tr>
</tbody>
</table>

**TABLE 2. ASSESSING BLEED RISK: HAS-BLED SCORE**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension*</td>
<td>1</td>
</tr>
<tr>
<td>Abnormal liver/renal function</td>
<td>1 or 2</td>
</tr>
<tr>
<td>Stroke risk</td>
<td>1</td>
</tr>
<tr>
<td>Bleeding predisposition</td>
<td>1</td>
</tr>
<tr>
<td>Labile INRs</td>
<td>1</td>
</tr>
<tr>
<td>Older (age &gt;65)</td>
<td>1</td>
</tr>
<tr>
<td>Drugs/alcohol usage **</td>
<td>1 or 2</td>
</tr>
<tr>
<td>Maximum score</td>
<td>9</td>
</tr>
</tbody>
</table>

Score of 3 or above = high risk

*Hypertension is defined as systolic >160mmHg. **Drugs/alcohol use refers to concomitant use of drugs, for example, antiplatelet drugs, non-steroidal anti-inflammatory drugs or alcohol use

Adapted from Pisters et al (2010)
During AF, blood clots develop in the left atrial appendage (LAA) in the majority of cases (Lip et al, 2011). The LAA can be surgically removed, but this is only performed as a concomitant procedure during open heart surgery. Recently, minimally invasive techniques in which an expandable frame is introduced to occlude the appendage during cardiac catheterisation have been developed.

Management of rate or rhythm

Initial assessment will determine the underlying cause and type of AF and how the patient is affected by the arrhythmia (Camm et al, 2010; NCC-CC, 2006). Symptoms can be controlled by a rate control strategy using drugs or a rhythm control strategy. Some patients may benefit from both (Camm et al, 2010; NCC-CC, 2006).

Tables 3 and 4 show these options.

---

**TABLE 3. TREATMENT SUMMARY**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Aim</th>
<th>When recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral anticoagulants such as warfarin</td>
<td>67% stroke risk reduction</td>
<td>First line in high-risk patients</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Can be unpopular</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased risk of bleeding</td>
</tr>
<tr>
<td>Novel oral anticoagulants – dabigatran,</td>
<td>Non-inferior to warfarin</td>
<td>No regular blood tests needed</td>
</tr>
<tr>
<td>rivaroxaban, apixaban</td>
<td></td>
<td>Fewer drug/food interactions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fixed daily or twice daily dose</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No antidote</td>
</tr>
<tr>
<td>Aspirin</td>
<td>22% stroke risk reduction</td>
<td>Limited benefits in those under 65 and no risk factors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Can be used in high risks, if patient declined OAC</td>
</tr>
<tr>
<td>Aspirin and clopidogrel combination</td>
<td>Slightly more effective than aspirin alone</td>
<td>Considered in those who cannot take or decline OAC but bleed risk similar to that of OAC</td>
</tr>
<tr>
<td>Left atrial appendage occlusion</td>
<td>A potential alternative to taking OAC</td>
<td>For selected individuals only</td>
</tr>
<tr>
<td></td>
<td></td>
<td>High-risk procedure</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>Possible “upstream effect in preventing recurrences” Optimising</td>
<td>Still limited evidence</td>
</tr>
<tr>
<td></td>
<td>intermittent heart failure</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statins</td>
<td>Possible “upstream therapy”</td>
<td>Still limited evidence</td>
</tr>
</tbody>
</table>

**TABLE 4. TREATMENT RECOMMENDATIONS**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Aim</th>
<th>When recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta–blockers, for example bisoprolol</td>
<td>● Control heart rate in permanent and persistent AF. Reduces symptoms and prevents heart failure</td>
<td>First line, less efficacious than class I and class III anti-arrhythmics, but less toxic</td>
</tr>
<tr>
<td></td>
<td>● Prevent recurrences in paroxysmal AF and cardioverted persistent AF</td>
<td>Avoid in uncontrolled heart failure and asthma</td>
</tr>
<tr>
<td>Rate limiting Ca2+ channel antagonists,</td>
<td>● Control heart rate in permanent and persistent AF</td>
<td>First line where beta-blockers unsuitable</td>
</tr>
<tr>
<td>for example diltiazem, verapamil</td>
<td>● Reduces symptoms and prevents heart failure</td>
<td>Avoid in moderate-severe left ventricular systolic impairment</td>
</tr>
<tr>
<td>Digoxin</td>
<td>● Control heart rate in permanent and persistent AF</td>
<td>Unsuitable for monotherapy unless patient sedentary</td>
</tr>
</tbody>
</table>

**Left atrial appendage occlusion**

During AF, blood clots develop in the left atrial appendage (LAA) in the majority of cases (Lip et al, 2011). The LAA can be surgically removed, but this is only performed as a concomitant procedure during open heart surgery. Recently, minimally invasive techniques in which an expandable frame is introduced to occlude the appendage during cardiac catheterisation have been developed.

**Rhythm control**

**Electrical cardioversion (DCCV)**

Patients presenting with new onset persistent AF who are symptomatic, have no structural heart disease and where no reversible cause is found have a good chance of restoring and maintaining normal sinus rhythm with electrical (DC) cardioversion (Camm et al, 2010; NCC-CC, 2006).

However, if AF has been present in the long term and there is associated heart disease, it is unlikely that sinus rhythm will be restored. In this scenario, controlling the ventricular rate is often preferable to reduce the symptoms of AF.

During DCCV, a brief electric shock of high energy is delivered to the patient’s chest to terminate the rapid heart rate. The shock depolarises the myocardium, which interrupts the tachycardia, allowing the sinus node to reset and resume control of a normal rhythm (Bennett, 2006). Patients are at risk of thromboembolism during the procedure so are required to have anticoagulation therapy for three weeks before and at least 4-6 weeks afterwards (Camm et al, 2010; NCC-CC, 2006).

**Pharmaceutical cardioversion**

Patients presenting with paroxysmal AF may be considered for beta-blockers or calcium antagonists to improve symptoms. If symptoms persist, antiarrhythmic medications should be considered.

Treatment options are oral or intravenous administration of class I antiarrhythmic drugs such as flecainide or propafenone or class III drugs, such as amiodarone. Flecainide should be avoided in people with structural heart disease as it can be proarrhythmic; an ECG should be recorded before it is started and one week
Pharmacological cardioversion becomes less effective after seven days’ duration and electrical cardioversion is often needed (Lip et al, 2011).

**Left atrial catheter ablation**

If symptoms continue in persistent or paroxysmal AF despite the use of one or two drugs, left atrial catheter ablation or pulmonary vein isolation can be considered.

This involves advancing flexible catheters through the blood vessels, often the femoral vein, into the right side of the heart. These are passed into the left side of the heart through an atrial trans-septal puncture. Radiofrequency energy is then used to ablate or destroy the areas of endocardium that are responsible for triggering the abnormal rhythm.

In patients with paroxysmal AF, the triggers often originate around the pulmonary veins so this area is frequently targeted with good outcomes (Lip et al, 2011). However, treating patients with persistent AF is more complex and involves using additional ablation approaches such as multiple linear ablation techniques in the left atria, which have lower success rates (Lip et al, 2011).

Owing to its complexity and high-risk profile, AF ablation is used only in people who are highly symptomatic and refractory to anti-arrhythmic drugs (Camm et al, 2010). Patients must be made aware that repeat procedures are often required and that anticoagulation therapy is needed for life if they are at high risk of stroke due to the risk of AF recurring (Camm et al, 2010; 2012).

**Rate control**

**Drugs**

Beta-blockers, calcium antagonist agents or digoxin are commonly used for ventricular rate control (Camm et al, 2010; NCC-CC, 2006). These drugs all work by blocking conduction through the AV node.

They should be avoided in patients whose AF is complicated by presence of a pre-excitation syndrome such as Wolf-Parkinson-White. These patients have an accessory pathway and, if the AV node is blocked, impulses will be conducted via the accessory pathway, resulting in extremely rapid ventricular rates, which may trigger ventricular fibrillation. They should be treated with a class I anti-arrhythmic drug, such as flecainide, or DC cardioversion (Lip et al, 2011).

Beta-blockers are contraindicated in people with asthma as they can cause bronchospasm. Calcium channel blockers should be avoided in patients with moderate to severe left ventricular impairment due to a risk of exacerbating heart failure. Digoxin has little effect on rate control in active people but can be an effective addition to beta blockers or calcium antagonists or used in sedentary individuals (Camm et al, 2010; NCC-CC, 2006).

**Atrioventricular node ablation**

This method of radiofrequency ablation is used to control ventricular rate rather than cure the AF.

The AV node is ablated, which isolates the ventricles from the atria and leads to complete heart block, so a permanent pacemaker is implanted. It has a high success rate and can improve symptoms and quality of life (Wood et al, 2000). The main disadvantage of this procedure is that patients will be pacemaker dependent. It is therefore reserved for those experiencing rapid ventricular rates despite drug therapy and where left atrial catheter ablation has failed (Bennett, 2006).

**The Cox maze procedure**

This is the most aggressive approach to treating troublesome AF. It is performed during open heart surgery, often as an adjunctive measure to heart valve repair or replacement.

Lines of conduction block in the atria are created by incision, radiofrequency energy or cryothermy. This forms barriers to atrial conduction so that the multiple wavelets of electrical activity circulating in the atria that result in AF cannot be sustained (Bennett, 2006). A summary of invasive treatment options is shown in Table 5.

**Conclusion**

AF is highly prevalent in our ageing population, so it is encountered in most settings. Early detection, diagnosis and treatment are paramount to reduce the risks associated with it, in particular stroke. A sound understanding of the mechanisms and management of AF will enable nurses to help diagnosis and manage it.

---

**TABLE 5. INVASIVE TREATMENT RECOMMENDATIONS**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Aim</th>
<th>When recommended</th>
</tr>
</thead>
</table>
| Pulmonary vein isolation with or without atrial ablation | Potential "cure" | - Limited use  
- 60–70% success rate  
- Invasive  
- Repeated procedures often necessary  
- 3–5% complication rate |
| Pacemaker and AV node ablation | Reduce symptoms | - Destructive, creates pacemaker dependence  
- Risk of pacemaker-induced heart failure |
| Cox maze procedure | Potential "cure" | - Only available during concomitant valve surgery |

---

**References**


