Atrial fibrillation is a common finding with major health implications, especially in elderly patients. Dr Ann Robinson discusses new thinking on the management of atrial fibrillation and explains how prompt treatment can prevent strokes.

Atrial fibrillation (AF) is common. The incidence and prevalence are increasing with one in four people over 40 years likely to develop AF in their lifetime. The significance of AF is that it causes haemodynamic instability, cardiomyopathy, heart failure and embolic events such as stroke. At least one in 20 of our patients who are over 65 years, and one in 10 of those over 80 years, will be in AF at any time. For every four patients that we know are in AF, there is at least one we do not know about.

Picking up AF is important because it is responsible for over a third of all strokes in patients over 80 years. It can be tempting to think we are doing patients a favour by not diagnosing and treating AF because of the hassle and risks of anticoagulation. However, it is useful to remind ourselves and patients that AF increases the risk of stroke five times and that strokes associated with AF tend to be worse. Elderly patients may not fear a stroke that causes immediate death, but the prospect of a severe and disabling stroke that results in loss of independence is a terrifying prospect for most.

Current management
Current management involves anticoagulation to reduce the risk of stroke, and symptom control with beta-blockers to slow the pulse rate. Only a minority of patients cannot be anticoagulated because of the risk of bleeding. However, studies have found that just less than half (47 per cent) of patients identified with AF are prescribed anticoagulants. This obviously does not include the large number of patients whose AF remains undiagnosed.

Low-dose aspirin is not effective even though doctors often prescribe it as a compromise solution. In fact, aspirin is the worst of both worlds; ineffective in preventing stroke from AF, and still carrying substantial risks of bleeding in the elderly. A third of people in AF take low-dose aspirin and the false sense of security that this gives them and their doctors may mean they are less likely to get the proper anticoagulation they need. Two-thirds of people admitted to hospital with a stroke related to AF are not anticoagulated.

Figure 1. For suspected paroxysmal atrial fibrillation, ambulatory ECG monitoring will be needed
What is happening in the heart in AF?
The pathophysiology of AF is explained in terms of the concept of “atrial remodelling”. This has developed over the past 15 years to explain the persistent change in atrial structure and function that promotes the occurrence or maintenance of AF by acting on fundamental arrhythmia mechanisms. Rapid ectopic firing and re-entry can maintain AF. For re-entry, there needs to be a trigger and vulnerable substrate. Atrial remodelling increases the likelihood of ectopic or re-entrant activity.

The challenges
There are three main challenges to improving the outcome for people in AF. The first is picking it up. The second is making a decision about anticoagulation and the third is making sure that adequate anticoagulation takes place. It all sounds straightforward but there are significant barriers at each stage.

Diagnosing AF
The old-fashioned way of taking a patient’s pulse opportunistically is a good way of picking up asymptomatic AF, especially in elderly patients. If the pulse feels “irregularly irregular” it could be AF, atrial or ventricular ectopic beats or atrial flutter with variable heart block. The next step is confirming AF on an ECG. For suspected paroxysmal atrial fibrillation (PAF), 24-hour ECG monitoring if it occurs every day, or an event monitor, will be needed.

Encouraging patients to self-diagnose
The Atrial Fibrillation Association has a useful Know Your Pulse video to show people how to check their own pulse and detect irregularity that may be AF. A smart phone app, AliveECG, has video to show people how to check their own pulse and detect the Atrial Fibrillation Association has a useful Know Your Pulse Encouraging patients to self-diagnose

What causes PAF and chronic AF?
The commonest underlying cause of AF is ischaemic heart disease. However, mitral valve disease, hypertension, cardiomyopathy, alcohol excess and thyrotoxicosis can also cause AF so these should be investigated and excluded.

What does the ECG look like in AF?
For those of us not lucky enough to get a computer-generated diagnosis, the ECG will show the following features in AF (see Figure 2): no P waves, variable ventricular rate and QRS complexes usually <120ms unless there is pre-existing bundle branch block, accessory pathways or other conduction problems.

Is echocardiography always indicated?
NICE recommends that a baseline echocardiogram may help guide management, particularly if rhythm control is being considered, as left ventricular function determines choice of treatment. Any suspicion of underlying structural disease or heart failure means echocardiography is needed.

What should we do for acute symptomatic AF?
People with acute, symptomatic AF should be referred within the first 48 hours for consideration of cardioversion. Stable sinus rhythm can be restored if cardioversion takes place within that short time frame. Follow-up is important as recurrences are common. If AF persists for more than 48 hours or the time frame is not certain, the patient will need to start a heart rate-controlling drug (usually bisoprolol), be anticoagulated and wait for at least three weeks before cardioversion can take place. So there is a narrow window of opportunity when a symptomatic person with new-onset AF presents to us.

Which patients in AF should we refer to secondary care?
All those with acute, symptomatic AF should be referred to secondary care in the first 48 hours. Patients with chronic AF who are symptomatic despite our best efforts with medical therapy, also warrant referral. If there is a significant underlying structural, eg valvular, or functional, eg left ventricular systolic dysfunction, problem suspected or confirmed on echocardiogram, the cardiologist will want to see that patient. It is also valid to refer if the patient or GP feels a specialist opinion is needed.

Making the decision to anticoagulate
A major problem remains a reluctance on the part of GPs to initiate anticoagulation once AF is diagnosed. The decision rests on balancing the risks of stroke, using CHA2DS2-VASc score, against the risk of bleeding, using HAS-BLED score.
A new software tool, (Anticoagulation Programme East London (APEL)), helps practices make these decisions. The software tool, developed by the Centre for Primary Care at Queen Mary, University of London, rapidly identifies high-risk patients in AF who are not anticoagulated.

**Balancing absolute annual stroke risk against absolute annual bleeding risk**

A useful algorithm called QBleed has been developed that calculates the risk of an upper GI or intracranial bleed with and without anticoagulation. It is similar to the HAS-BLED score recommended by NICE but is based on a very much larger cohort. It can only be used for risk assessment prior to starting warfarin, not non-Vitamin K antagonist oral anticoagulants (NOACs), as most of the anticoagulated group received warfarin. NICE says people at risk of falls should not be denied anticoagulation.

**Explaining the benefits of anticoagulation to patients**

To show patients the potential benefit of anticoagulation, we need to be able to explain their chance of having a stroke with and without treatment. A CHA\(_2\)DS\(_2\)-VASc score of 1 gives an annual stroke risk of 0.6 per cent untreated and 0.2 per cent treated. But with a CHA\(_2\)DS\(_2\)-VASc score of 5, the annual untreated stroke risk is 8.4 per cent, falling to 2.7 per cent if treated. Patients with AF may be more likely to accept anticoagulation if they work out their own stroke risk using an online calculator. Results vary depending on whether they use US or UK/EU guidelines but the outcome is broadly similar.

**Stop aspirin when starting anticoagulation**

The twin goals in AF are clearly to cure symptoms so the patient feels better and to prevent a stroke. The key to preventing a stroke is adequate anticoagulation while minimising the risk of serious bleeding. Patients have often been taking low-dose aspirin for years; many buy it over the counter and may not even mention it to the GP. But it is important to stop antiplatelet treatment such as aspirin when starting anticoagulation, otherwise the risk of bleeding increases. Around 1 in 10 anticoagulated patients remain on aspirin or other antiplatelet agents in addition to their anticoagulant. In the vast majority of cases, there is no specific indication for them to remain on the antiplatelet drug (and often no good reason that they started it in the first place). One simple step will help to minimise bleeding risk from anticoagulation; ask whether the patient is taking aspirin and tell them to stop.

**The exceptions: when antiplatelet treatment and anticoagulants should be continued**

The minority of patients who should take an antiplatelet drug and an anticoagulant are those who have acute coronary syndrome or who have had a stent inserted into a coronary artery in the past year. These patients will be under the care of a cardiologist, should be reviewed after a year when the antiplatelet drug is usually stopped and should take a proton pump inhibitor, eg omeprazole, to prevent gastrointestinal bleeding. The preferred anticoagulant will be warfarin, which can be reversed if a bleed occurs, rather than a NOAC, which cannot.

**Warfarin self-monitoring**

For people who are on warfarin rather than a NOAC, it can be very liberating for them to self-monitor rather than having to traipse to a clinic. People on long-term warfarin should be offered the chance to monitor their blood clotting (international normalised ratio; INR) themselves using one of two NICE-approved point-of-care coagulometers (CoaguChek XS and InRatio PT/INR). They can then change the dose of warfarin as needed in agreement with their health professional.

**Making sure that warfarin is doing its job**

Warfarin only works as a way of minimising stroke risk if patients are within the therapeutic range for a majority of the time. Time in the therapeutic range (TTR) is calculated by anticoagulant clinics and the treatment regimen is reviewed if they spend less than 65 per cent TTR or are under- or over-anticoagulated, ie two or more INR readings over 5 or two or more INR readings under 1.5. In these cases of poor control, the first question is whether the patient is actually taking their warfarin regularly and the second is whether other drugs or foods are interacting with the warfarin. If TTR remains suboptimal, a NOAC should be considered.

**NOAC or warfarin?**

At a BMJ Masterclass recently, the assembled 300 GPs were asked, “If you had AF, would you want a NOAC or warfarin?” An overwhelming majority put their hands up for a NOAC. The evidence shows that NOACs work as well as warfarin in reducing stroke and mortality over a wide range of patients. They cause similar overall major bleeding although NOACs are associated with more gastrointestinal bleeding.

The NICE Implementation Collaborative report of 2014 notes that there are three currently licensed NOACs – dabigatran, rivaroxaban and apixaban – that have been approved by NICE as options for the prevention of stroke and systemic embolism in patients with nonvalvular AF. The report states: “The drugs must therefore be made available for prescribing within their licensed indications, and should be automatically included in local formularies.” It urges review of local arrangements for the use of antithrombotic therapies in AF and policies.
Who can have a NOAC?
Anyone with nonvalvular AF who has one or more associated risk factors, can be considered for a NOAC to reduce the risk of stroke. The risk factors are having a history of stroke/transient ischaemic attack, being over 75 years old, having high blood pressure, symptomatic heart failure or diabetes.22

Who cannot have a NOAC?
NOACs are not licensed in pregnancy or breastfeeding, should not be used with some drugs, eg clarithromycin with dabigatran, carbamazepine with rivaroxaban or apixaban, should be used with extreme care with antiplatelet therapy and, clearly, should not be started if there is active or significant risk of a major bleed.22

Which NOAC?
There is no head-to-head data comparing the NOACs. Cost, bleeding risk and renal function are the three parameters that guide decision making. Local formularies may make the decision for us or clinical algorithms can guide the choice.22

Heart rate control is first-line treatment
Treatment for heart rate control is with a beta-blocker, eg bisoprolol or, less commonly, a rate-limiting calcium-channel blocker, eg diltiazem. Digoxin23 is less often used now but is sometimes advised for elderly and sedentary people in whom sympathetic tone is low, so beta-blockers are less likely to work. Elderly patients are more likely to suffer from digoxin toxicity as their elimination may be reduced. Starting with a low dose and titrating up, and checking urea and electrolytes as well as digoxin levels, help to reduce toxicity. If rate control is not achieved with one drug, a second of the three drugs (diltiazem, beta-blocker or digoxin) can be added. After that, referral to secondary care is advised.

When is rhythm control important?
Heart rate control is the main priority. Rhythm control is only important if someone has symptoms despite the rate being well controlled, or is in heart failure. Amiodarone or cardioversion are options in secondary care. After cardioversion, the drug dronedarone24 helps to maintain the patient in sinus rhythm but is contraindicated in heart failure.

Treating sporadic attacks of AF
A single dose of an antiarrhythmic drug, eg flecainide,25 may be prescribed by a cardiologist for healthy patients with no underlying cardiovascular disease who get very occasional paroxysmal AF.

When is ablation therapy a good idea?
Left atrial ablation is carried out for PAF when drug treatment has failed. It is useful when patients are symptomatic despite medical treatment, have left ventricular systolic dysfunction and have not been in continuous AF for a long time. Ablate and pace26 is a technique in which a pacemaker is inserted and then the AV node is ablated. It is used when a patient is in heart failure and has a fast ventricular rate.

What is left atrial appendage occlusion?
In AF, a thrombus can develop in the left atrial appendage (LAAO) using a catheter technique and have not been in continuous AF for a long time. Ablate and pace26 is a technique in which a pacemaker is inserted and then the AV node is ablated. It is used when a patient is in heart failure and has a fast ventricular rate.

Key groups in whom NOACs should especially be considered include patients who cannot take vitamin K antagonists (such as warfarin), those who cannot be stabilised on vitamin K antagonists with poor time in therapeutic range, eg <65 per cent, despite adequate adherence, and those taking aspirin for stroke prevention. Because there is no active monitoring with NOACs at an anticoagulant clinic, patients need to understand the need for and agree to continue with the treatment. Doctors and pharmacists have an important role in educating and reinforcing the reasons for treatment and answering patients’ concerns. There are, as yet, no specific antidotes for the NOACs but there are steps that can be taken to reverse the drugs’ effects in the event of a major bleed, says the report.20

What about safety concerns with NOACs?
NOACs have been around for about six years now. They act by inhibiting thrombin (dabigatran) or factor Xa (rivaroxaban, apixaban). On the plus side, unlike warfarin, they have a rapid onset of action, short duration, few interactions with other drugs or foods and do not need close monitoring. Because of their short half-life, if a major bleed occurs, it is unlikely to be more dangerous than if the patient were on warfarin. The main downside of NOACs is that there is no specific antidote. Another limitation to their use is poor renal function because the NOACs, especially dabigatran, are excreted by the kidneys.21

How integrate NOACs into the care pathway. It quotes the 2014 NICE guidelines saying “Continued use of aspirin is a barrier to appropriate stroke prevention with oral anticoagulants.”

Figure 3. NICE recommends that a baseline echocardiogram may help guide management of atrial fibrillation, particularly if rhythm control is being considered, as left ventricular function determines choice of treatment.

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What does the future hold?
There are many aspects of management of AF that remain controversial. These include the roles of rate and rhythm control, the need for early cardioversion to prevent chronicity, whether electrical or pharmacological cardioversion is better, and sorting out who will benefit from long-term anticoagulation, NOAC therapy, ablation therapy and left atrial appendage occlusion. Ablation therapy may be developed to treat persistent, as well as paroxysmal, AF.28

Current therapy aims to manage the condition, reducing symptoms and stroke risk. But better strategies are needed to predict AF risk, diagnose and treat underlying disease and tailor treatment to individuals. The hope is that AF will become curable or, better still, preventable.

References
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Declaration of interests
None to declare.

Dr Robinson is a GP and health writer