

Best Practices in the Diagnosis and Treatment of Atrial Fibrillation

Written by Muriel Cunningham

An overview of best practices for the treatment of patients with atrial fibrillation (AF) was given by 3 experts in the field. Patrick Heck, MD, Papworth Hospital, Cambridge, United Kingdom, started the session by reviewing anticoagulation options for patients with AF. Several novel oral anticoagulant (NOAC) medications have been approved for use in patients with nonvalvular AF, but not in patients with rheumatic mitral valve disease or in patients with mechanical valves. These drugs include the direct thrombin inhibitor dabigatran and the direct factor Xa inhibitors rivaroxaban, apixaban, and edoxaban. Large randomized clinical trials have been conducted in which each of these agents was compared with warfarin [Giugliano RP et al. *N Engl J Med.* 2013; Granger CB et al. *N Engl J Med.* 2011; Patel MR et al. *N Engl J Med.* 2011; Connolly SJ et al. *N Engl J Med.* 2009]. A meta-analysis of the 4 studies found that all NOACs were at least as good as warfarin in preventing stroke or systemic embolism and were at least as safe as warfarin in terms of major bleeds [Ruff CT et al. *Lancet.* 2014].

NOAC antidotes are currently being developed. A monoclonal antibody for dabigatran (idarucizumab) has demonstrated an effective reversal after a 5-minute infusion and should be available soon. The anti-Xa antidote andexanet alfa is currently in phase 3 studies. It is anticipated that this antidote will be effective against apixaban, edoxaban, rivaroxaban, and possibly low-molecular-weight heparin.

Scoring systems are available to help clinicians to determine when anticoagulation is needed. The CHA₂DS₂-VASc score is used to estimate the embolic risk of individual patients, and the HAS-BLED scale is used to estimate bleeding risk. Dr Heck suggested the following resources for those unfamiliar with prescribing NOACs in patients with AF: the European Heart Rhythm Association Practical Guide on the use of NOACs in patients with nonvalvular AF [Heidbuchel H et al. *Europace.* 2013], the European Society of Cardiology Guidelines for the management of AF [Camm AJ et al. *Eur Heart J.* 2012], and an overview by Lip and colleagues [Lip GY et al. *J Am Coll Cardiol.* 2012].

Nicholas Linker, James Cook University Hospital, Middlesbrough, United Kingdom, reviewed initial steps physicians should take in patients with newly diagnosed AF. Patients with AF may present with dyspnea, chest pain, palpitations, dizziness or syncope, stroke, or transient ischemic attacks. Risk factors for AF include higher age, valvular heart disease, increased left atrial size, coronary heart disease, hypertension, diabetes, height (> 6 ft, 2 in), obesity, obstructive sleep apnea, and extreme fitness (eg, endurance athletes such as marathon runners, cyclists, and triathletes). To detect occult AF, pulse palpation is very important and should be performed annually in patients with hypertension, diabetes, coronary heart disease, cerebrovascular disease, and peripheral artery disease, and every 6 months in patients with heart failure. In patients aged ≥ 65 years, manual pulse palpation should be performed whenever a blood pressure reading is taken. Be aware that patients with pacemakers may have a regular pulse but still be in atrial fibrillation and hence at risk of stroke!

Dr Linker noted that a high-quality 12-lead electrocardiogram is required to properly diagnose AF. Assessment of potassium and magnesium levels, cardiac biomarkers, thyroid function, liver function, and echocardiograms may also be necessary. Chest x-rays should also be performed to determine the presence of an enlarged heart, pneumonia, or lung tumors because lung disease is often associated with AF. Comorbidities such as hypertension, diabetes, valvular disease, ischemic heart disease, and heart failure must also be identified and managed.

Causes of AF and treatment approaches are presented in Tables 1 and 2. In addition to pharmacotherapy, recent research has shown the importance of addressing lifestyle factors to manage AF. The LEGACY [Pathak RK et al. *J Am Coll Cardiol.* 2015] and CARDIO-FIT studies [Pathak RK

Official Peer-Reviewed
Highlights From





Table 1. Causes of Atrial Fibrillation

Cardiac Causes	Noncardiac Causes
Common causes	Acute infections, especially pneumonia
Ischemic heart disease	Electrolyte depletion
Rheumatic heart disease	Lung carcinoma
Hypertension	Other intrathoracic pathology (eg, pleural effusion)
Sinus node disease	Pulmonary embolism
Less common causes	Thyrotoxicosis
Cardiomyopathy	
Pericardial disease (including effusion and constrictive pericarditis)	
Atrial septal defect	
Atrial myxoma	
Pre-excitation syndromes (eg, Wolff Parkinson White syndrome)	

Reproduced with permission from NJ Linker, MD.

Table 2. Treatment of Atrial Fibrillation

Description	Recommendation
Hemodynamically unstable AF: AF with a rapid ventricular rate causing ongoing chest pain, hypotension, shortness of breath, dizziness, or syncope	DCCV
Hemodynamically stable symptomatic AF	
Rate control	If rate control is inadequate with monotherapy, consider adding a calcium channel blocker (in absence of heart failure) or digoxin
No heart failure	β-Blockers (metoprolol, bisoprolol) Calcium channel blockers (verapamil, diltiazem)
Heart failure	β-Blockers (use caution) Digoxin (oral) Amiodarone
Cardioversion	If duration of AF known to be < 48 h and no left atrial thrombus on transesophageal echocardiogram, consider DCCV or pharmacological cardioversion (flecainide or amiodarone) If duration of AF > 48 h or unknown, cardioversion should be deferred until anticoagulated
Lifestyle measures	Weight reduction Exercise

AF, atrial fibrillation; DCCV, direct current cardioversion.

Reproduced with permission from NJ Linker, MD.

et al. *J Am Coll Cardiol.* 2015] demonstrated that weight loss and exercise reduce symptoms and the burden of AF in obese patients.

Nick Linton, Imperial College, London, United Kingdom, concluded the session with a presentation on rhythm control in patients with AF. The primary goal of rhythm control is to improve symptoms, so it is important to establish a link between rhythm and symptoms. Treatment must be individually tailored, and decision making should not be delayed because restoring sinus rhythm becomes more difficult the longer a person has AF. Dr Linton emphasized that a key decision point is when patients go from paroxysmal to persistent AF because delays would then make the restoration of sinus rhythm more difficult.

Drugs have somewhat limited efficacy in sinus rhythm maintenance, with a success rate of 30% to 50% 1 year after direct current cardioversion [Freemantle N et al. *Europace.* 2011]. Amiodarone has the best efficacy, and, although it has a low risk of pro-arrhythmia, it may cause a long QT interval. Amiodarone has a high risk of side effects, particularly with high cumulative doses. For this reason, it is important to monitor thyroid, liver, skin, lungs, and eyes in patients taking this medication. The sodium channel blocker flecainide has increased mortality in patients with a previous myocardial infarction and should not be prescribed to patients with structural heart disease, conduction system disease, or systolic

heart failure. Flecainide can potentiate flutter with 1:1 atrioventricular conduction, so it is typically co-prescribed with a rate-slowing agent such as a β -blocker. QRS duration should be monitored in patients taking flecainide. Sotalol is not effective for sinus rhythm conversion but helps prevent recurrence. Sotalol is renally cleared and can cause QT prolongation, so patients should be monitored. Fewer long-term side effects are seen with dronedarone compared with amiodarone, but it is not as effective. Dronedarone has been shown to increase mortality in patients with recent decompensation of heart failure and is contraindicated in patients with persistent AF.

Many patients with persistent AF who have undergone cardioversion and are on antiarrhythmic drugs will relapse. "It is important that drugs that have a risk are discontinued if they are not working," Dr Linton said. Patients with a permanent AF strategy who are not going to be converted back to sinus rhythm should stop antiarrhythmic drugs and return to a rate control strategy. In his concluding remarks, Dr Linton noted the importance of conducting a thorough assessment of a patient's cardiovascular system and lifestyle when they present with AF. There is new evidence that patients presenting with AF have less arrhythmia during follow-up after multidisciplinary interventions targeting weight, fitness, sleep apnea, diabetes management, and hypertension [Pathak et al. *JACC.* 2015].