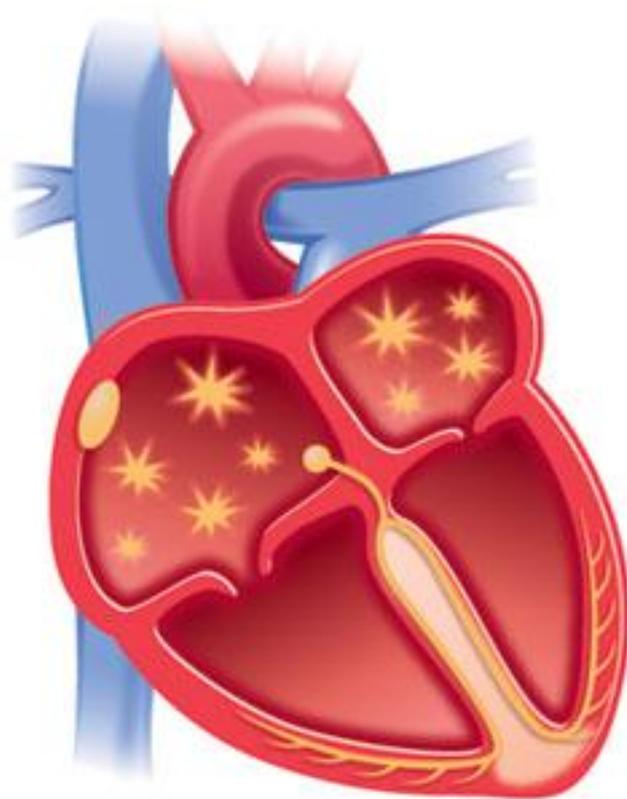


# RUSSELLS HALL HOSPITAL EMERGENCY DEPARTMENT



## CLINICAL GUIDELINE

# ATRIAL FIBRILLATION March 2011

For quick links to AF algorithms: [UNSTABLE PATIENT](#)

[STABLE PATIENT](#)

## Introduction

Atrial fibrillation is the most commonly encountered arrhythmia in clinical practice and is associated with considerable morbidity and mortality, resulting predominantly from an increased risk of stroke and heart failure. The dysrhythmia is characterised by chaotic, irregular and rapid discharge from the atria. This activity reaches the atrioventricular node at varying angles and intervals, producing an irregular ventricular rate. On the ECG, AF is described by the absence of consistent P waves; instead there are rapid oscillations or fibrillatory waves that vary in size, shape and timing.

Research from the National Collaborating Centre for Chronic Conditions estimates that the prevalence of AF is 0.5% in 50-59 year olds and 9% in 80-89 year olds<sup>1</sup>. The annual UK incidence is 46,000 new cases per year<sup>2</sup> and it represents over a third of all dysrhythmia-based hospital admissions in the UK. AF and the associated morbidity represent a significant socioeconomic burden on the healthcare system.

Risk factors for development of AF include increasing age, diabetes, hypertension and valve disease. It is also commonly associated with, and complicated by, congestive heart failure and strokes. Dietary and lifestyle factors associated with AF include excessive alcohol ('holiday heart'), caffeine and emotional or physical stress. AF is also common after surgery, particularly cardiothoracic procedures.

*Table 1: Common Precipitants of Atrial Fibrillation*

<b>CARDIAC</b>	<b>INTRA-THORACIC</b>	<b>METABOLIC</b>
Ischaemic heart disease / MI	COPD	Hypokalaemia
Mitral valve disease	Pneumonia	Hypomagnesaemia
Hypertension	Pulmonary embolus	Acidosis
Cardiomyopathy	Malignancy	Thyrotoxicosis
Pericarditis/Endocarditis	Trauma	Alcohol/drugs
Post-cardiac surgery	Pulmonary hypertension	Sepsis
Pre-excitation (WPW)		

A 2001 study performed amongst Emergency Medicine Consultants found marked variety in individual physicians' management of patients with acute atrial fibrillation<sup>3</sup>. NICE published guidelines in June 2006 to clarify the management of such patients in accordance with the best available evidence<sup>4</sup>. This document aims to provide a solid framework, based on such evidence, for the management of acute atrial fibrillation in the ED at Russells Hall Hospital.

### **Classification of Atrial Fibrillation**

A report published in 2003 established a new classification process for defining the different types of atrial fibrillation<sup>5</sup> and this was adopted in the 2006 NICE Guidelines. It is important for ED doctors to understand which category a patient comes under as this may affect subsequent management.

**INITIAL EVENT (first detected episode):** may or may not be symptomatic and may or may not recur. Time of onset may be unknown.

**PAROXYSMAL AF:** spontaneously terminates in less than 7 days (typically lasts minutes to hours) with a recurrent pattern.

**PERSISTENT AF:** lasts longer than 7 days and is not self-terminating.

**PERMANENT AF:** present for some time, and fails to terminate on cardioversion; or is terminated but relapses within 24 hours.

'Lone AF' is described as AF without overt structural heart disease, co-morbid cardiovascular disease or other recognised precipitants of AF<sup>6</sup>, and is defined by a normal clinical history and examination, chest X-ray and echocardiogram. However it is a diagnosis of exclusion and all patients who present with AF should be investigated for possible precipitants ([table 1](#)); only 11% of patients presenting with acute AF will have true 'lone AF'.

### **Complications of Atrial Fibrillation**

Atrial fibrillation produces a pro-thrombotic state with dyskinetic atria producing intra-atrial blood stasis, abnormal platelets and haemostasis. This contributes to a roughly five-fold increase in stroke and thrombo-embolism in people with AF compared to sinus rhythm<sup>7</sup>. The risk varies for each individual based on their age and concurrent stroke risk factors and all patients should therefore undergo stroke risk stratification ([table 2](#)).

Reduced ventricular filling also leads to an overall reduction in cardiac output and even with a controlled ventricular rate the cardiac output can decrease by 10-20%. This may not be significant in patients with otherwise normal hearts but can result in pump failure in patients with other cardiac abnormalities. With an increase in ventricular rate, the heart spends less time in diastole, and this leads to reduced filling of the coronary blood vessels. Without controlling this rate, there can be progression to critical cardiac ischaemia and subsequent infarction of myocardial tissue.

Atrial fibrillation has been shown to lead to increased mortality, with a 2006 paper estimating a two-fold increase in mortality compared to people in normal sinus rhythm<sup>8</sup>.

## **General Approach Towards Management of Atrial Fibrillation in the Emergency Department**

The complications of increased risk of stroke and heart failure allow us to have an appreciation of the fundamental management strategies in acute atrial fibrillation:



### **Rate control vs. rhythm control**



### **Anti-thrombotic agents**

Prior to considering how best to manage a patient presenting with acute atrial fibrillation, the following should be established:

- ✓ History and examination
- ✓ 12 lead ECG
- ✓ Supplemental oxygen therapy if necessary
- ✓ Intravenous access and relevant blood tests
- ✓ Continuous ECG monitoring
- ✓ Treatment of reversible causes, (e.g. sepsis, electrolyte abnormalities)

### **Diagnosis of Atrial Fibrillation**

A 12 lead ECG should be obtained for all patients who present to the Emergency Department with an irregular pulse. In addition, the following symptoms also warrant ECG assessment for the presence of AF (it should also be borne in mind that it can occur asymptotically in many cases):

- Breathlessness
- Palpitations
- Syncope
- Chest discomfort/pain
- TIA/CVA

On the ECG P waves are absent and are replaced by fibrillatory waves (figure 1). AF is associated with an irregular and frequently rapid ventricular response if AV conduction is intact.



*Figure 1: absent P waves and irregular ventricular response*

## **ECG – Differential Diagnosis**

Careful, expert study of the ECG may be necessary to determine that a tachyarrhythmia is AF with a rapid ventricular response. An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV block. An irregular tachyarrhythmia with broad complexes is most likely to be AF with bundle branch block (figure 2); look for a consistent LBBB or RBBB pattern. Other possible causes are AF with ventricular pre-excitation (in patients with Wolff-Parkinson-White (WPW) syndrome), or polymorphic VT (e.g. torsade de pointes), but polymorphic VT is unlikely to be present without adverse features. Seek expert help with the assessment and treatment of irregular tachyarrhythmias if uncertain of the underlying rhythm or optimal management.

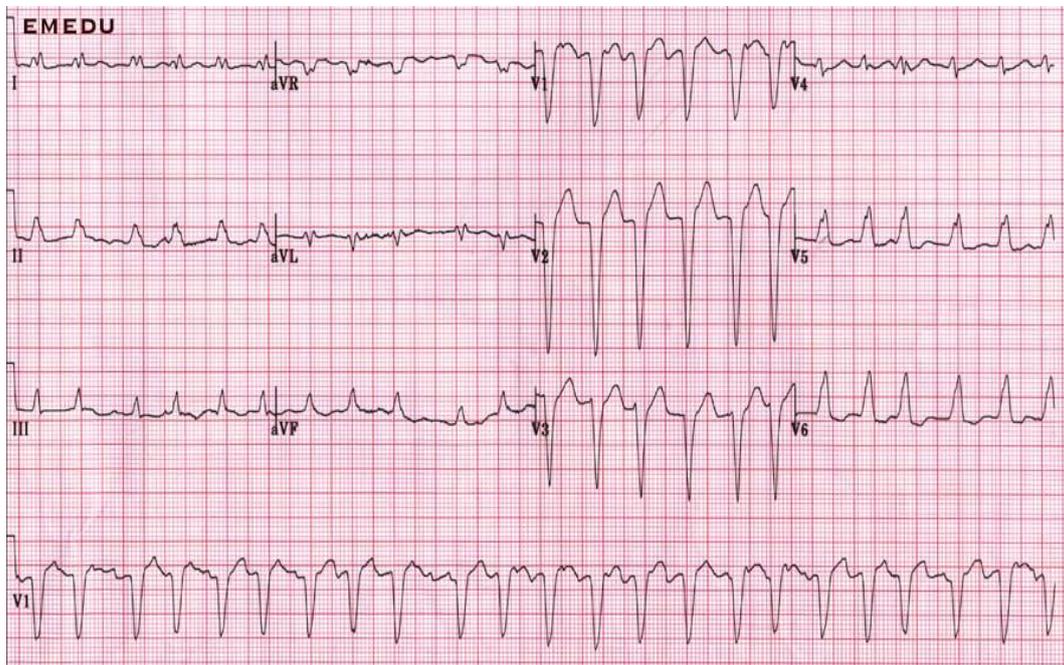


Figure 2: Atrial fibrillation with pre-existing LBBB (from emedu.org)

If any of the conditions in [table 1](#) are identified, treatment should incorporate the underlying condition. The subsequent management of the patient will depend on their haemodynamic status (including the presence of cardiac chest pain and acute pulmonary oedema) and the classification of their AF.

**\*\*Pitfall – Beware!\*\***

As previously stated, WPW can present as AF. Clues may exist in the history or the ECG. Look for evidence of pre-excitation with wide and bizarre QRS complexes; delta waves are not usually seen. Ventricular response is typically very fast (>200 bpm). **Never give digoxin or verapamil** but seek urgent Cardiology advice with a view to restoring sinus rhythm with chemical or DC cardioversion.

## **'Fast' AF Management in the Compromised Patient**

As per UK [Resuscitation Council Guidelines](#), if the patient is unstable and deteriorating, consider urgent DC cardioversion (**synchronised** biphasic shock 150J > 200J > 360J) with sedation if the patient has any of the following signs and symptoms caused by tachycardia:

- signs of acute pulmonary oedema
- consciousness level is reduced
- chest pain
- hypotension (systolic BP <90)

*However* it should be remembered that AF (particularly at rates <150bpm) is usually well tolerated unless the patient has impaired cardiac function secondary to critical CAD, severe LV systolic impairment or severe obstructive valvular disease. A fast ventricular response may be secondary to sepsis / PE / anaemia etc. and the haemodynamic compromise not due to the AF per se. If this is the case then the primary cause should of course be treated urgently.

The success of DC cardioversion appears to be greater with anteroposterior paddle positioning (sternum and left subscapular) than with standard paddle positions. In a conscious patient, short-acting sedation or general anaesthesia are required. Studies have shown that the success rate for electrical cardioversion performed in the ED setting is 80-89%, compared to 50% for pharmacological cardioversion<sup>9</sup>.

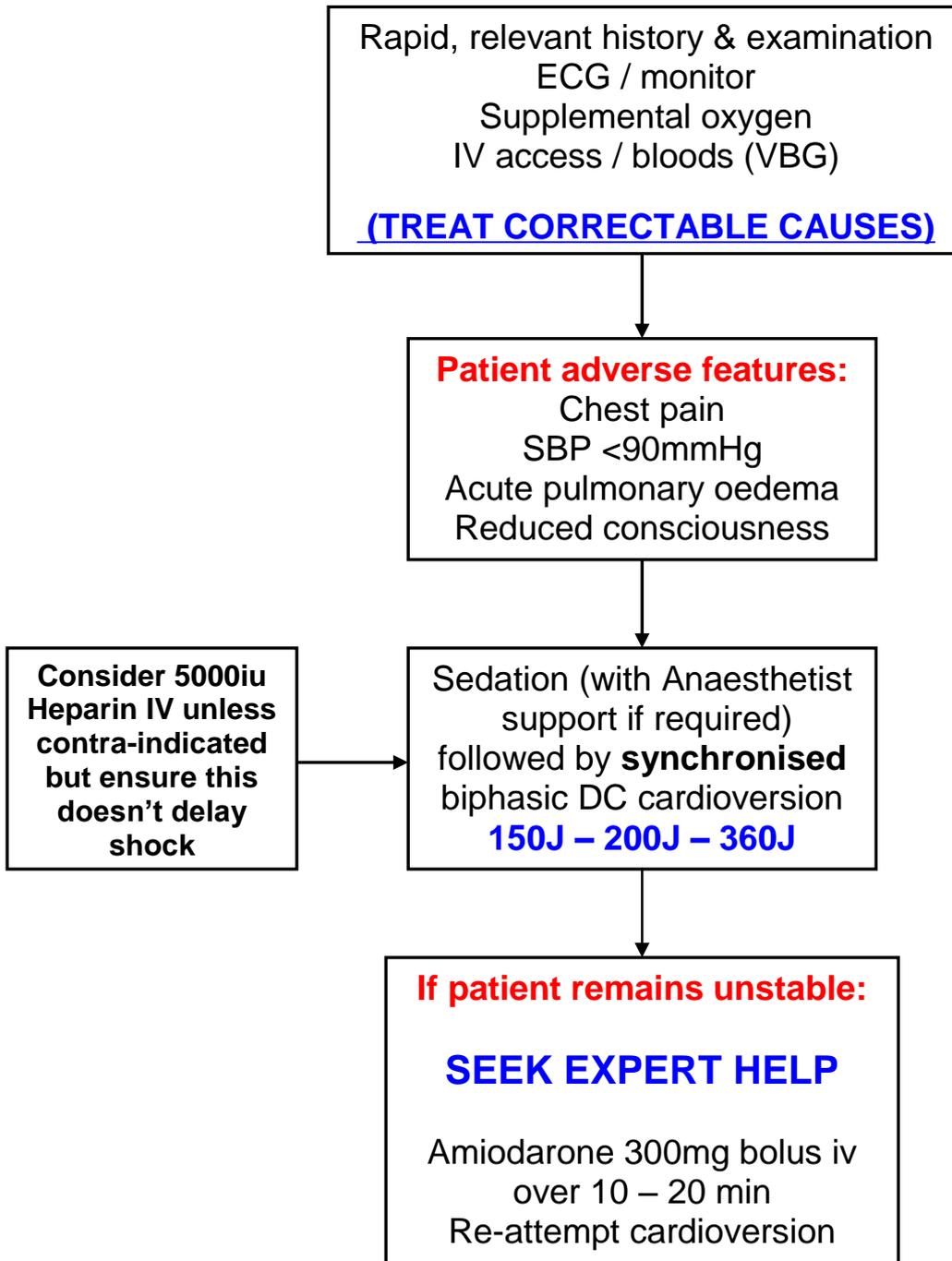
If DC cardioversion fails to restore sinus rhythm and the patient remains unstable give amiodarone 300mg IV over 10-20 min and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900mg over 24h.

It is safe to cardiovert patients with implanted devices such as a permanent pacemaker or internal defibrillator provided the implanted device is interrogated after cardioversion for any malfunction. The paddles used for cardioversion should be placed as far as possible from the implanted device; preferably in the AP position. Brief arrhythmias may arise immediately following cardioversion. In addition, patients with underlying conduction defects are at risk of developing profound bradycardia, CHB or asystolic periods. Pacing facilities must be at hand before attempting cardioversion.

There is evidence to suggest that these patients benefit from 5000iu heparin IV prior to cardioversion, to prevent thrombo-embolic events on restoration of sinus rhythm.

**Remember that improvement may not be achieved or maintained without optimization of correctable factors and careful attention should be paid to any pathology underlying the patient's condition.**

**Flow Chart for ED Management of Unstable Patient  
with AF and Rapid Ventricular Response**





## **Cardioversion of the Stable Patient**

If AF has been present for less than 48 hours, seek urgent Cardiology advice. Unless there is an underlying cause (e.g. thyrotoxicosis, pneumonia) it may be appropriate to restore sinus rhythm immediately with chemical or electrical cardioversion. No high-quality studies directly comparing these two techniques have demonstrated any superiority.

## **Role of Digoxin in the Emergency Department**

Digoxin is best used as an adjunct in the rate control of patients with AF. It may be used after beta-blockers or calcium antagonists for rate control if the rate does not fall sufficiently. It may also be used as first-line therapy in patients with significant heart failure.

Digoxin enhances vagal tone, and slows the ventricular rate by reducing the sympathetic drive and vagotonic action. It has no effect on the SA node or conduction in the AV node. Given orally, digoxin has a delay of approximately 60 minutes in reducing ventricular rate, and peak plasma levels don't occur until 6 hours after ingestion. There is **NO** advantage of IV digoxin over oral digoxin. It is ineffective for rate control in patients with a high sympathetic drive, (i.e. sepsis, severe heart failure). See nomogram for loading dose:

<http://thehub/departments/acutemedicine/Prescribing%20Regimens/digoxin.html>

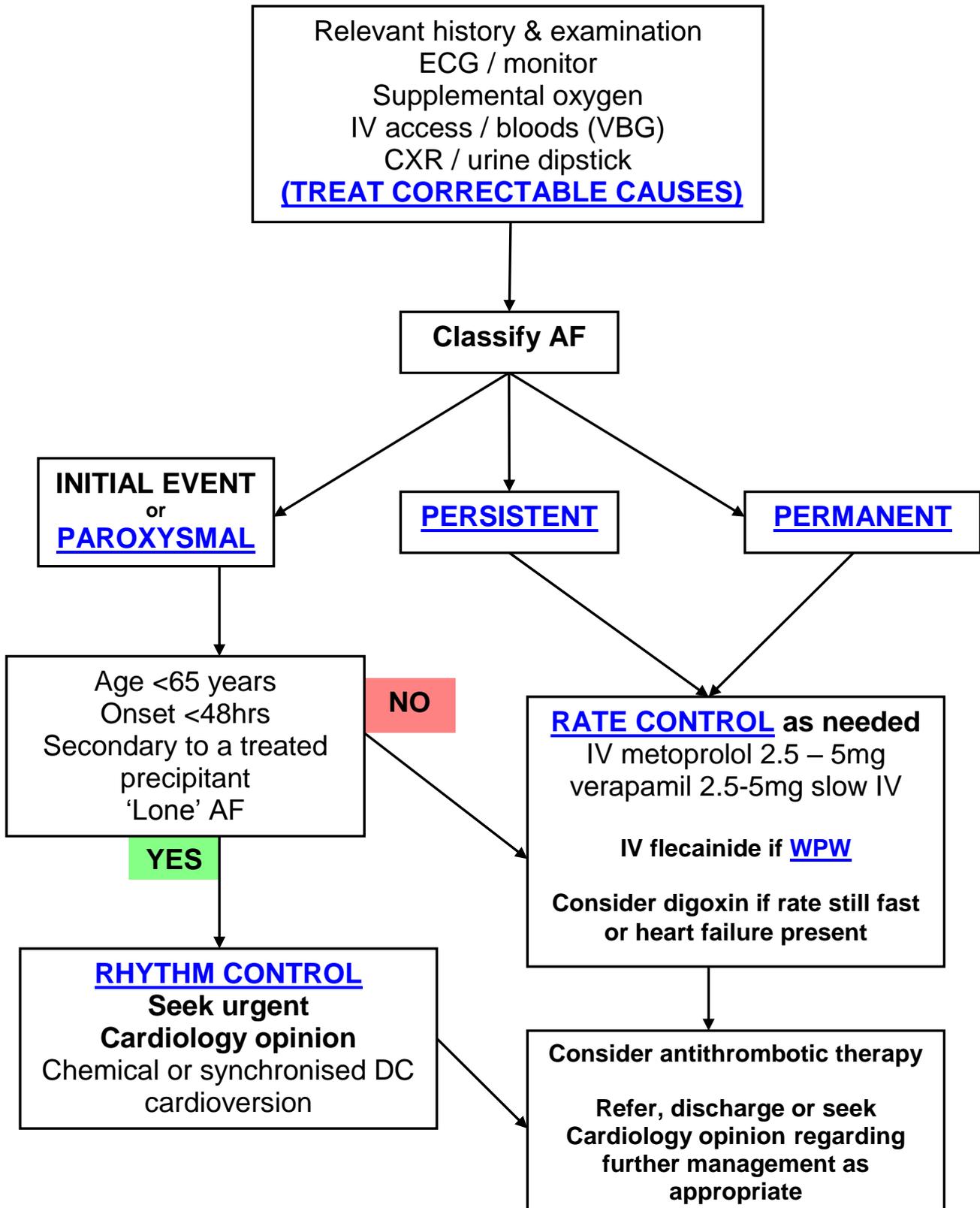
## **Antithrombotic Therapy in Atrial Fibrillation**

The 2006 NICE Guidelines suggest that all patients with atrial fibrillation have an assessment of stroke risk to determine which anti-thrombotic therapy would be appropriate. This should be started as soon as possible, and if not commenced in the ED, individual patients should be referred to their GP to commence appropriate therapy. Assessment of stroke risk is based on the following criteria:

*Table 2: Stroke Risk Stratification*

	<b>HIGH RISK</b>	<b>MODERATE RISK</b>	<b>LOW RISK</b>
<b>FEATURES</b>	TIA Thromboembolism Valve disease Impaired LV function Age > 75 years with the following: -hypertension -diabetes -peripheral vascular disease -IHD	Age > 65 years with no high-risk factors  Age > 75 years with the following: -hypertension -diabetes -peripheral vascular disease -IHD	Age < 65 years with no moderate or high-risk factors
<b>THERAPY</b>	Warfarin (INR 2-3) with target 2.5	Aspirin or warfarin	Aspirin 75mg – 300mg / day

**Flow Chart for ED Management of Stable Patient with AF**



### **Patients Suitable for Discharge from the ED**

For asymptomatic patients under 65 years with no evidence of structural heart disease and a controlled ventricular rate (60-80 bpm), they may be discharged provided that they have appropriate follow-up organised (i.e. via cardiology specialist nurse or GP) and none of the features below:

- Patient requires further tests to exclude ACS
- Further rate control required
- Possible cardioversion indicated
- Embolic event (e.g. TIA) or high risk for stroke
- Mitral valve or structural heart disease
- Heart failure
- Non-cardiac cause of AF requiring further investigation or therapy

Patients suitable for discharge may be admitted to CDU overnight if necessary. Outpatient ECHO should be arranged prior to discharge. These discharge criteria are only a guide and do not take into account all possible scenarios; consult with Cardiology if unsure.

### **Summary**

Atrial fibrillation is a common presentation to Emergency Departments in the UK and NICE Guidelines published in 2006 offer an evidence-based approach to the management of a condition that has previously been treated in many different ways. It is important to consider the causative features of AF, and treat where appropriate. In the unstable patient correction of any underlying cause and synchronised DC cardioversion remain the first choice therapies. However, it is important to consider the use of IV heparin in these patients (if not already anti-coagulated), and amiodarone if there is likely to be a delay in sedating the patient.

For the stable patient with new onset or paroxysmal AF (short duration of less than 48 hours) cardioversion remains the first choice therapy, but for persistent or permanent AF, it would be more appropriate to offer rate-control, with the use of beta-blockers or verapamil. Amiodarone is probably the safest agent for pharmacological cardioversion and flecainide is the safest drug to use in Wolff-Parkinson White.

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**With thanks to Dr Joe Martins (Consultant Cardiologist, RHH) and Dr David Raven (Consultant in Emergency Medicine, Heartlands Hospital)**

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