Management of Acute Atrial Fibrillation

W Taggu & VS Rathore

Key points:
- Prevalence of AF doubles with each advancing decades.
- Electrical or pharmacological cardioversion may be effective in restoring sinus rhythm.
- Rate control may be an appropriate treatment option in some situations.
- Electrical cardioversion can be performed safely without anticoagulation if AF is present < 48 hours.

Abstract

Atrial fibrillation (AF) is the most common cardiac arrhythmia; despite many available guidelines the optimal management strategy remains elusive.

The most common presentations of acute AF are palpitations, breathlessness, syncope, dizziness, chest pain, transient ischaemic attack (TIA) and stroke. Patients may need urgent electrical cardioversion (ECV) or pharmacological cardioversion (PCV) and long term anticoagulation therapy.

This review aims to discuss some issues surrounding the early management of acute AF in the Acute Medical Unit.

Keywords

National institute of Health and clinical excellence (NICE), Atrial fibrillation (AF), Paroxysmal atrial fibrillation (PAF), transient ischemic attack (TIA), Electrical cardioversion (ECV), Pharmacological cardioversion (PCV), Transoesophageal echocardiogram (TOE), Warfarin

Introduction

AF is an atrial tachyarrhythmia characterized by predominantly uncoordinated activation with consequent deterioration of atrial mechanical function.[1] It is the commonest sustained arrhythmia. The prevalence appears to double with each advancing decade of age, from 0.5% at aged 52–59 years to almost 9% at aged 80–89 years[2] AF accounts for 3–6% of acute medical admission in UK.[3]

The incidence of post operative AF is approximately 33% after Coronary artery bypass grafting[4] and even higher following valvular heart surgery.[5]

Classification

Atrial fibrillation can be classified as follows:

1. Acute onset (within 48 hours).
2. Paroxysmal (spontaneous termination within less than 7 days and usually less than 48 hours).
3. Persistent (lasting more than 7 days).
4. Permanent (more than 1 year or refractory to cardioversion).
5. 'Lone AF', diagnosed by exclusion of structural heart disease, normal clinical examination, chest x-ray and echocardiogram.

Aetiology

There is an increasing prevalence and incidence of AF with age. In the Framingham study, the development of AF was associated with increasing age, diabetes, hypertension and valve disease.[6] Atrial fibrillation is also associated with and complicated by congestive heart failure and stroke. A full list of Cardiac and non-cardiac causes of AF are shown in Table 1.

Cardiac	Non-Cardiac
Coronary artery disease	Acute infections
Hypertension	Pulmonary embolism
Rheumatic heart disease	Electrolyte imbalance
Sick sinus syndrome	Intrathoracic pathology and Ca Lung
Valvular Heart disease	Thyrotoxicosis
Post cardiothoracic surgery	Alcohol, Caffeine
Cardiomyopathy	Recreational drugs
Pericardial disease	
Structural heart disease	

Table 1. Causes of AF.

Clinical Presentation

AF often presents with vague and non specific symptoms. It may be detected in asymptomatic individuals or after the patient presents with stroke, TIA, heart failure, syncope or dizziness. The most common presenting symptoms in the emergency setting are breathlessness, chest pain and palpitations.[3,7]

Dr Wasing Taggu
MD, MRCP
Registrar Cardiology
Eastbourne DGH, East Sussex NHS Trust
BN21 2UD
Email: wasingtaggu@rediffmail.com
Tel: 01323 435654

Correspondence:
Dr V S Rathore MD MRCP
Consultant Cardiologist
Eastbourne DGH, East Sussex NHS Trust
BN21 2UD
Email: vsrathore@doctors.net.uk
Phone: 01323 435653
Fax: 0118 322 5377

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A detailed history and clinical examination are an essential. Physical findings may include irregularly irregular pulse and changing intensity of the first heart sound, absence of the 'A wave' in the jugular venous pulse and an irregularly irregular ventricular rhythm at the apex.

Investigations

Initial investigations should include:

- Blood tests: Full blood count, electrolytes, thyroid function, troponin
- Chest x-ray
- Electrocardiogram
- Echocardiogram.

An echocardiogram is beneficial to rule out structural heart abnormality but may have limitations acutely among patients with a fast ventricular rate; ideally echocardiogram should be delayed until heart rate has been controlled.

Treatment of acute AF

Rhythm control or Rate Control?

Initial therapy for AF is often directed toward the restoration of sinus rhythm by means of cardioversion (*'rhythm control'*).[12] The rationale for a rhythm control approach includes the possibility of fewer symptoms, better exercise tolerance, a lower risk of stroke, eventual discontinuation of long term anticoagulation therapy, better quality of life and better survival, if sinus rhythm can be maintained.[13] However, unfortunately at one year following cardioversion approximately 50% of patients are back in AF.[22]

An accepted alternative is a strategy to control the ventricular response rate, while atrial fibrillation is allowed to persist (*'rate control'*). This approach makes use of atrioventricular nodal blocking agents or ablation of the atrioventricular junction and pacemaker implantation in conjunction with continuing anticoagulation.[14] Factors which may predict a high likelihood of recurrence of AF following cardioversion are shown in Table 2. In this context the 'rate control' may be the preferred option.

Drugs commonly used for rate control include: digoxin, calcium antagonists (verapamil, diltiazem) and beta blockers (e.g. bisoprolol, atenolol).

- History of failed DC cardioversion
- Structural heart disease (mitral valve disease, LV dysfunction or dilated Left atrium)
- A prolonged history of AF (> 12 months)
- Previous recurrence of AF

Table 2. Factors indicating high risk of AF recurrence following cardioversion.

Cardioversion

Restoration of sinus rhythm is termed *Cardioversion*. This may be achieved electrically (ECV) or pharmacologically (PCV). Current clinical practice regards PCV as the preferred strategy in haemodynamically stable patients presenting with recent onset AF (within 48 hrs); ECV is regarded as the preferred strategy where the AF is more prolonged or in the emergency setting.

Outside the emergency setting, cardioversion should not be attempted until any underlying precipitant (e.g.: thyrotoxicosis, infection, electrolyte abnormality) has been successfully treated.

Anticoagulation prior to cardioversion

Electrical cardioversion has been shown to precipitate embolisation from the atrium, resulting in ischaemic stroke. Prior to elective cardioversion, anticoagulation for around 6 weeks is advised; this should be continued for at least 1 month following the restoration of sinus rhythm. The onset of AF may be associated with a cluster of thromboembolic events,[9] but the development of intra atrial thrombi is perceived to be minimal within the first 48 hours. Therefore, when considering cardioversion in the *acute setting*, the following points should be noted:

- Cardioversion can be performed safely without oral anticoagulation if AF has been present for less than 48 hours.[8]
- If there is an uncertainty about onset of AF, anticoagulation with heparin, combined with warfarin, is advised until the INR reaches therapeutic range; except in the emergency setting, transoesophageal echo is usually advisable to confirm

- In a patient with acute AF who is receiving no, or sub-therapeutic, anticoagulation therapy :

  In absence of contraindication heparin should be started at initial presentation Heparin should be continued until full assessment has been made and appropriate antithrombotic has been initiated.

- In a patient with a confirmed diagnosis of acute AF of recent onset (< 48 hrs), oral anticoagulation should be used if:

  Stable sinus rhythm is not restored within the same 48 hrs period following onset of acute AF, or

  There are factors indicating a high risk of AF recurrence or

  It is recommended by the stroke risk stratification algorithm (Figure 1)

- In a patient with acute AF where there is uncertainty over the precise time of onset, oral anticoagulation should be used as for persistent AF.

- In cases of acute AF where the patient is hemodynamically unstable, any emergency intervention should be performed as soon as possible and the initiation of anticoagulation should not delay any emergency intervention.

Box 1. NICE Recommendations for antithrombotic therapy in AF.

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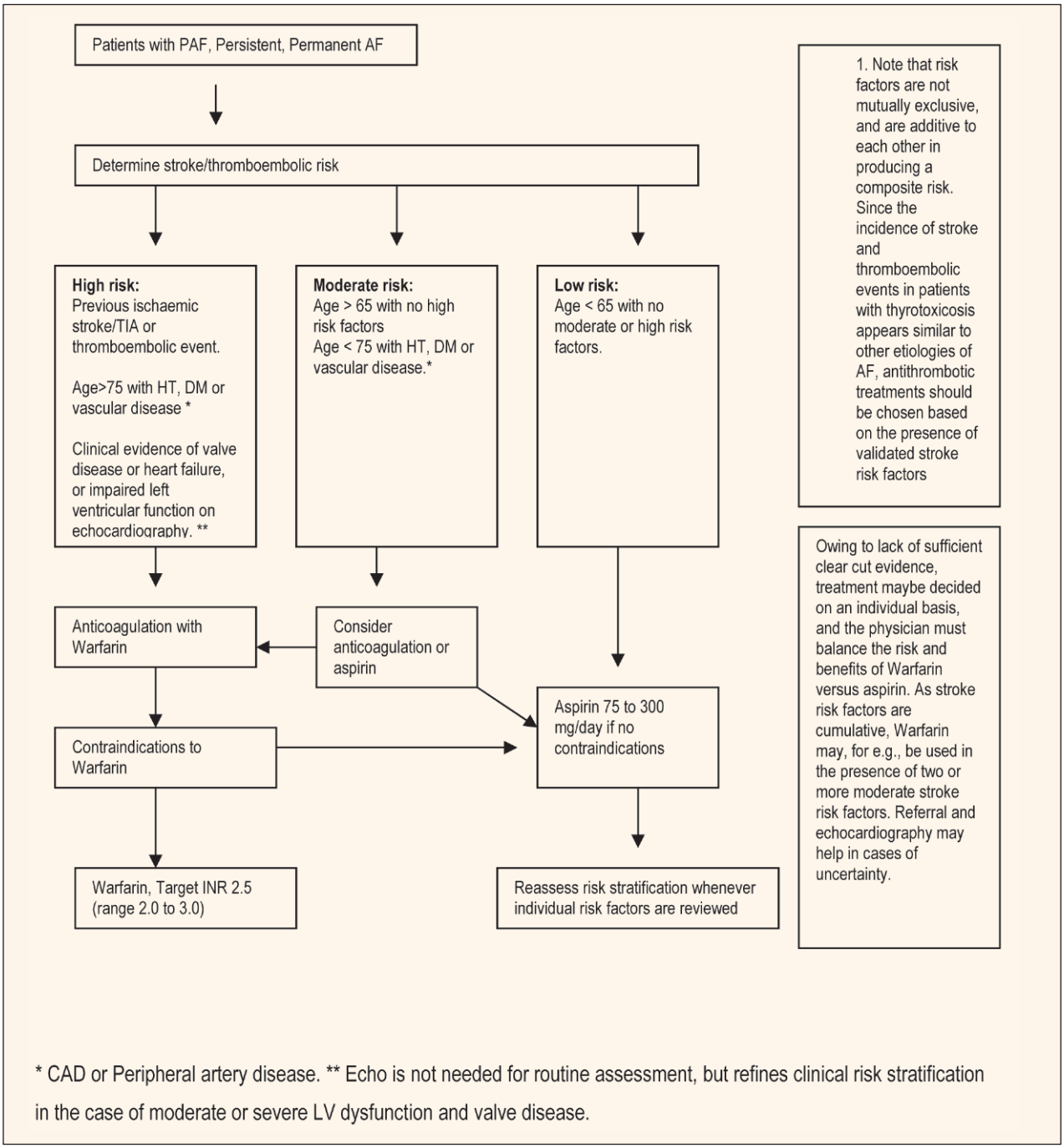


Figure 1. Stroke risk stratification algorithm for patients with atrial fibrillation.
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the absence of clot in the atrium prior to cardioversion in this situation.

- Where there is haemodynamic instability in life threatening situations, cardioversion takes priority over anticoagulation.

Recommendations from the National Institute for Clinical Excellence (NICE) regarding initial antithrombotic therapy are summarized in Box 1.

Haemodynamically unstable AF

Patients with haemodynamically unstable AF (Figure 2) require immediate hospitalization and urgent intervention. Haemodynamic instability caused by AF is usually defined as:[23]

- Ventricular rate > 150.
- Ongoing chest pain.
- Pulmonary oedema.

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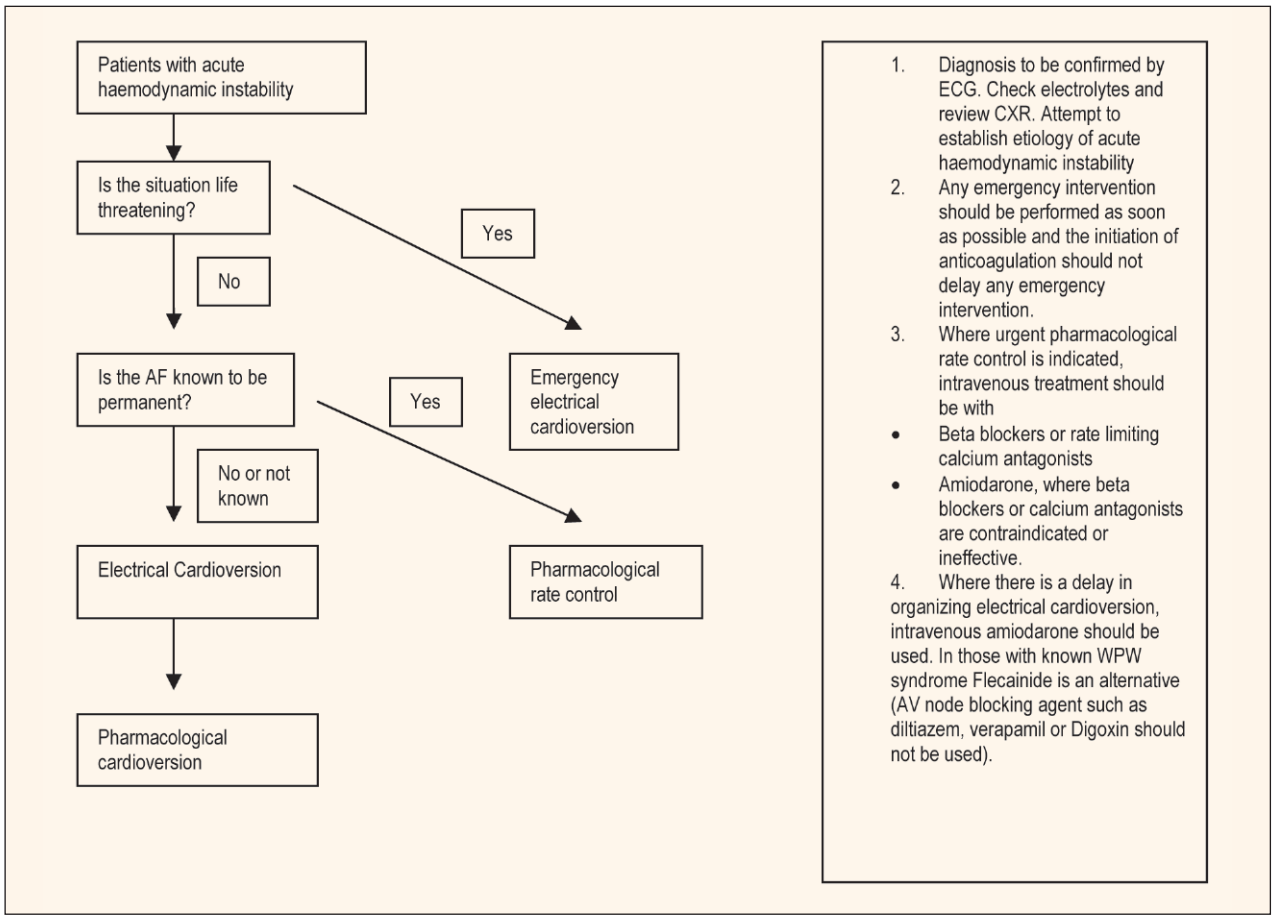


Figure 2. Hemodynamically unstable AF treatment algorithm.
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- Critical perfusion (hypotension, evidence of tissue ischaemia, etc).

A number of specific issues should be recognized in the management of acute AF in the presence of haemodynamic instability[22]

- The slow onset of digoxin make it a poor choice for use in this situation.
- A patient whose AF is associated with thyrotoxicosis will not respond to any strategy that does not first treat the underlying thyroid disease.
- Patients with an accessory pathway such as Wolf Parkinson White (WPW) syndrome are particularly at risk following the onset of AF. They may present with high ventricular rate (> 200/min) which is often exacerbated by AV node blocking drugs[24].

- In patients with life threatening deterioration in haemodynamic stability following the onset of AF, emergency ECV should be performed irrespective of the duration of the AF.
- In a patient with non life threatening haemodynamic instability following the onset of AF:
  1. Electrical cardioversion should be performed.
  2. Where there is delay in organizing electrical cardioversion intravenous Amiodarone should be used.
  3. In those with WPW syndrome Flecainide maybe used as an alternative for attempting pharmacological cardioversion. AV node blocking agents: Diltiazem Verapamil or Digoxin should not be used.
- In patient with non permanent AF where the haemodynamic instability is caused mainly by a poorly controlled ventricular rate, a pharmacological rate control strategy should be used.
- Where urgent pharmacological rate control is indicated this can be achieved by intravenous administration of one of the following:
  - Beta blockers or rate limiting calcium antagonist (e.g. verapamil).
  - Amiodarone, where Beta blocker and calcium antagonist are contraindicated or ineffective.

Box 2. NICE recommendations for treatment of AF.

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- Amiodarone is the most commonly used agent in management of patient with hemodynamically unstable AF. The beta blocker Esmolol is an alternative choice. Treatment with AV node blockers (e.g. Adenosine, Digoxin, Verapamil, and Diltiazem) will unmask or exacerbate rapid AF due to WPW syndrome and are therefore contraindicated for patients in whom WPW cannot be excluded.[24]

Electrical Cardioversion

Electrical Cardioversion (ECV) is carried out in the fasting post absorptive state with sedation or general anaesthesia, following informed consent. The strategy chosen varies amongst clinicians, although sedation with benzodiazepines has become increasingly popular.[25] More recently a nurse led service has been successfully described.[26]

Paddles are positioned in the anteroposterior or base-apex location. A DC shock synchronized to the peak of the QRS is delivered. Previous practice has moved from low energy (50J) to high energy (200–360J). Newer biphasic defibrillators delivering 200J are more frequently used. We advocate up to 3–4 shocks varying the position with a maximum energy of 360J. Procedural risks are low but VF/VT, asystole and transiently depressed myocardial function have been reported.

Post procedural anticoagulation with warfarin should be maintained for a minimum of 4 weeks, aiming for INR between 2 and 3.

Chemical Cardioversion

For patients not requiring urgent cardioversion, chemical cardioversion with antiarrhythmic drugs has been found to be effective in 35 to 75 percent depending on the population studied.[17] A variety of drugs are effective for chemical cardioversion. (see Table 3)

With the possible exception of amiodarone, no single drug appears clearly superior and selection is often based on the risk of proarrhythmia and side effect profile.[21]

Maintaining sinus rhythm

Recurrent AF is common following cardioversion; although antiarrhythmic drugs may reduce the incidence of recurrence, their benefits must be balanced against the long-term side effects of drugs such as amiodarone. Our practice is not to use antiarrhythmic prophylaxis following cardioversion for a single episode of AF, but to reserve these agents for patients who require recurrent cardioversion.

Post Operative AF

Post operative AF is associated with greater risk of mortality and morbidity. Evidence is also emerging[25] that post operative AF predisposes to significantly increased risk of stroke and thrombo-embolism, suggesting that patients should be anticoagulated where AF persists more than 48 hours.[28]

Treatment is indicated for those patients who remain symptomatic, become hemodynamically unstable, and develop cardiac ischemia or heart failure. The treatment strategies include ECV, atrial overdrive pacing if atrial flutter is dominant, pharmacological rate control and anti-thrombotic therapy. Electrolye abnormalities (e.g. hypokalaemia, hypomagnesaemia) are common following surgery and correction may be effective[29–30] in the prevention of post operative AF.

Long-term anticoagulation

The pro-thrombotic state predisposes to stroke and thromboembolism in AF, with an approximately 5 fold greater risk than that of people without AF.[31] The incidence of stroke attributed to AF increases from 1.5 % at age 50–59 years to 23.5% at age 80–89 years [32].

To identify high risk patients with AF who should be targeted for longer term anticoagulation, the ***CHAD2 risk stratification*** scheme can be used.[10–11]

One point is allocated to each of the following:

- congestive cardiac failure
- hypertension
- Age > 75 years
- Diabetes Mellitus

Two points are allocated for:

- Stroke or TIA.

Score > 2 = high risk; Score 1–2 = intermediate risk; score 0 = low risk.

Prior to initiating anticoagulation therapy, attention should be paid to factors which increase the risk of bleeding complications. Risk of bleeding is increased in the elderly, patients with uncontrolled hypertension or

Drug	Class	Dose	Route	Comments
Flecainide	Ic	2mg/kg 10- 30 minutes MAXIMUM 150 mg. followed by infusion if required 1.5 mg/kg for 1 hour	IV	To be used in absence of structural heart disease
Propafenone	Ic	150 mg 3 times daily	PO	To be used in absence of structural heart disease
Esmolol	II	5- -200 microgram /kg/minute	IV	Avoid in Asthma or uncontrolled heart failure
Sotalol	III	80- 240 mg bd	PO	Avoid in Asthma or uncontrolled heart failure
Amiodarone	III	300 mg in 1 hour followed by 900 mg in 24 hours	IV	Administer in Large vein or central access

Table 3. Drugs commonly used for pharmacological cardioversion.

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history of bleeding (e.g. peptic ulcer, cerebral hemorrhage). Concomitant use of antiplatelet agents and non-steriodal anti-inflammatory agents also increases the risk of haemorrhage, and where possible these should be stopped prior to introduction of anticoagulants.

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