

ATRIAL FIBRILLATION AND ANAESTHESIA

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Atrial fibrillation (AF) is one of the commonest arrhythmias. It may be paroxysmal (sudden episodes), persistent or permanent. Atrial depolarization is very rapid, irregular and disorganized. This causes irregular and rapid ventricular conduction. AF may be seen in patients presenting for anaesthesia or may occur during anaesthesia.¹

Haemodynamic deterioration occurs due to the loss of atrial mechanical function, irregular ventricular response and a rapid heart rate. The loss of atrial contraction decreases cardiac output by up to 30%, particularly in patients with impaired ventricular diastolic filling. This is of importance in patients with hypertension, left ventricular hypertrophy, mitral stenosis and hypertrophic and restrictive cardiomyopathy.² The ventricular response rate depends on electrophysiological factors in the atrioventricular (AV) node, drugs and sympathetic and vagal tone. A rapid ventricular response results in a reduced cardiac output due to inadequate time for passive filling of the ventricles.³ A persistent rapid ventricular response may result in a dilated ventricular cardiomyopathy.²

Decreased blood flow in the left atrium (LA) and left atrial appendage (LAA) is associated with thrombus formation. Embolism from the LA may result in a stroke or other arterial occlusion. The pathogenesis of thromboembolism is complex and affected by other factors including intrinsic cerebrovascular disease, hypertension, atheroma in the proximal aorta and carotid artery stenosis.²

Pre-operative assessment and atrial fibrillation

Ask about the **first detected episode**. AF is recurrent after 2 or more attacks. Distinguish between paroxysmal, persistent and permanent AF.

Paroxysmal AF is self terminating. The patient may have no symptoms and be unaware of the episode(s) of atrial fibrillation. Symptoms include palpitations, chest pain, dyspnoea, fatigue, light-headedness and syncope. Symptoms may vary depending on the duration, the ventricular rate and the functional status of the patient.²

Persistent AF is sustained and requires electrical or chemical cardioversion to establish sinus rhythm. Inquire about modes of termination and drugs used on a regular or as needed basis: the “Pill-in-the-Pocket” approach.⁴

Permanent AF is long-standing persistent AF where cardioversion was not successful or has not been attempted.²

Although atrial fibrillation may occur without associated disease in younger patients, it may be associated with underlying disease. Treatment of the associated conditions while managing the AF normally resolves the arrhythmia.²

Examination

Expect a completely irregular pulse and jugular venous pulsation. The loudness of the first heart sound may be variable. A rate of between 60 and 80bpm at rest and between 90 and 115bpm during

Associated cardiovascular disease	Non-cardiac associations
<ul style="list-style-type: none"> • Hypertension • Coronary artery disease • Cardiomyopathy • Valvular disease (mitral) • Cardiac surgery • Myocarditis • Pericarditis • Other supraventricular arrhythmia(s) and • Wolff-Parkinson-White syndrome (WPW) 	<ul style="list-style-type: none"> • Alcohol • Hyperthyroidism • Pulmonary disorders • Pulmonary embolism • Obstructive sleep apnoea • Thoracic or oesophageal surgery • Sepsis • Vagal and sympathetic mechanisms.⁵

exercise is considered controlled and minimises the haemodynamic consequences of AF. Associated valvular disease may be diagnosed on auscultation and signs of heart failure should be looked for.²

Investigations

An **ECG** should be done to confirm AF. In AF the p-waves are replaced by fibrillatory waves that rapidly oscillate and are variable in time, size and shape. The ventricular response (QRS complex) is irregular. The ECG should also be studied for LV hypertrophy, pre-excitation (Wolff-Parkinson-White syndrome), bundle branch block, Q waves (previous MI's) and other arrhythmias. Measure the RR, QRS and QT intervals if the patient is on anti-arrhythmic medication.²

A **CXR** evaluates the lungs, and the heart size and shape.

Preoperative **blood analysis** will identify acid base, anaemia and electrolyte abnormalities. AF is more common with hypokalaemia or hypomagnesaemia.

Drugs used for AF

This can be divided into attempted rhythm control,

rate control and anticoagulation for thromboembolic prophylaxis.

Rhythm Control: The aim is to maintain sinus rhythm and prevent the adverse symptoms the patient experiences in AF. Embolism and cardiomyopathy will also be avoided if the patient remains in sinus rhythm.²

Drugs commonly used for rhythm control include amiodarone, sotalol, verapamil and flecainide.

When drugs in class 1c are used the QRS duration should not exceed 150% of the pre-treatment QRS duration. The upper limit for the QT interval for drugs in class Ia and III is 520ms.²

Rate Control: AF is accepted and the aim is to control the ventricular rate during rest and exercise. Drugs used include beta-blockers, calcium channel blockers and digoxin. These agents depress conduction across the AV node and may cause bradycardia or heart block, which requires permanent pacing in order to continue treatment.²

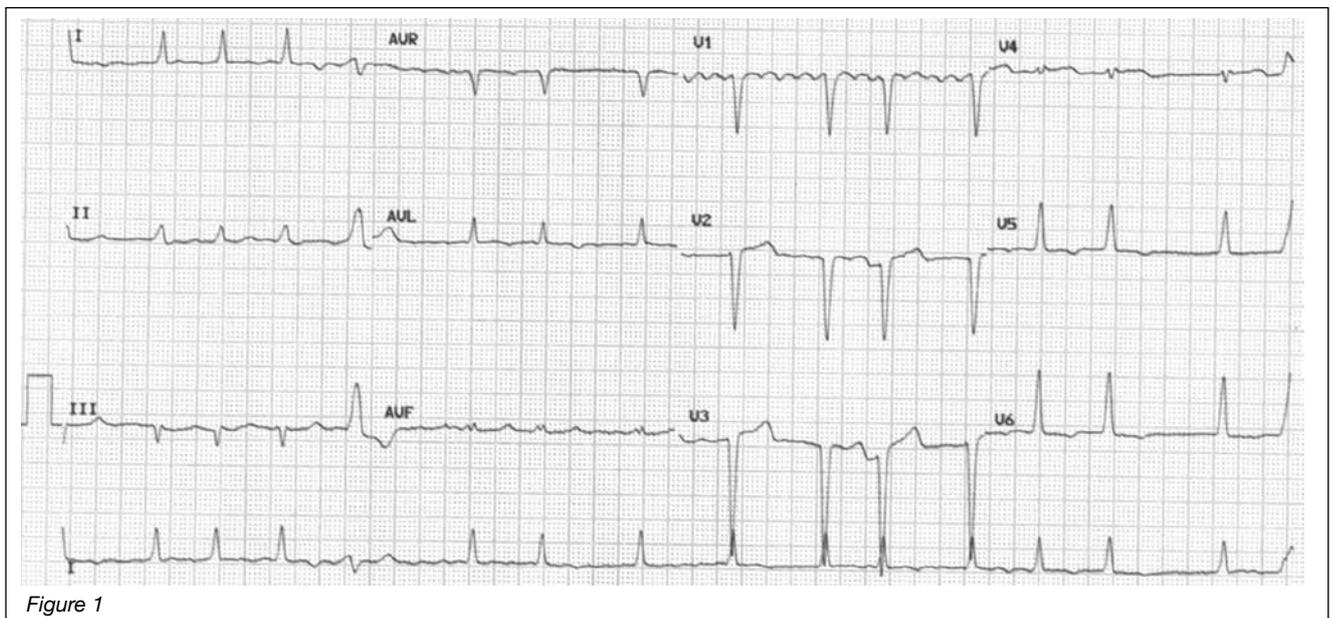


Figure 1

Class	Action	Drugs
Ia	Membrane-stabilising	Procainamide Quinidine Disopyramide
Ic	Membrane-stabilising	Flecainide Propafenone
II	β -adrenoceptor blockers	Esmolol Propranolol
III	Action potential prolongation	Amiodarone Sotalol
IV	Calcium channel antagonist	Verapamil Diltiazem
Others	Cardiac Glycosides	Digoxin ¹

Anticoagulation: Current evidence indicates that patients in paroxysmal, persistent and permanent AF have a similar risk for stroke. The duration of episodes and the time spent in AF has not been shown to determine the risk for stroke. Patients with the greatest risk are the elderly and those with a history of thromboembolism, diabetes mellitus, coronary artery disease, hypertension, heart failure and thyrotoxicosis.⁵

Oral anticoagulation is effective for primary and secondary prevention of stroke in AF with a reduction of more than 60% compared to placebo. Maximum protection occurs with an INR of between 2 and 3. Bleeding is an obvious complication. Balance the risk

of stroke to major bleeding. The protection against stroke with the use of aspirin is modest.²

Oral anticoagulation for a patient in AF, with no mechanical valve, can be discontinued for up to 1 week without the need for additional anticoagulation. If oral anticoagulation needs to be discontinued for a period greater than 1 week, unfractionated heparin or low-molecular-weight heparin can be used.²

Pre-operation medication

In patients undergoing cardiac surgery an oral beta-blocker can prevent postoperative AF. Sotalol or amiodarone may also be given prophylactically in patients with an increased risk of developing AF.²

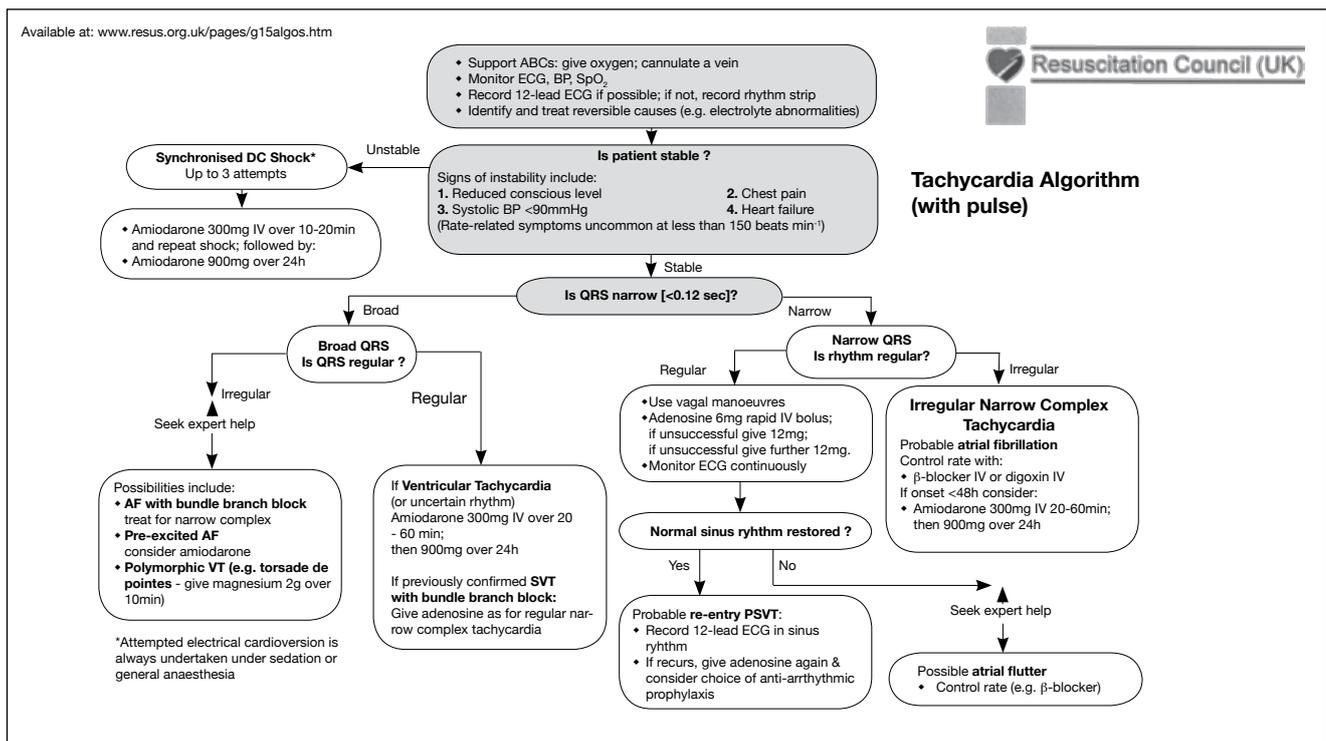


Table 2: Recommended antithrombotic therapy for AF	
Aspirin 325mg od	<ul style="list-style-type: none"> • < 60years, no heart disease. • < 60years, with heart disease, no other risk factors. • 60-75years with no risk factors. • If warfarin is recommended but contraindicated or refused.
Warfarin to INR of 2	<ul style="list-style-type: none"> • > 75years, especially women.
Warfarin to INR between 2 and 3	<ul style="list-style-type: none"> • > 60years with diabetes or coronary artery disease (aspirin is optional). • Heart failure with an ejection fraction less than 35%, thyrotoxicosis or hypertension.
Warfarin to INR between 2.5 and 3.5	Rheumatic heart disease (mitral stenosis), prosthetic valve, previous thromboembolism and current atrial thrombus. ⁵

Peri-operative atrial fibrillation

If possible establish the diagnosis of AF with a 12 lead ECG. Most, if not all anaesthetized patients will have oxygen and IV access in place. Further treatment will depend upon the clinical condition of the patient.⁶ (See figure 2).

Unstable patients

Examine for adverse signs and clinical evidence of low cardiac output. Pallor, sweating, cold and clammy extremities are secondary to sympathetic activity. Hypotension may develop. If awake, observe for decreasing level of consciousness. A very high ventricular response rate (>150) can reduce coronary blood flow and cause myocardial ischemia with chest pain. Look for signs of heart failure: pulmonary oedema (left sided failure) raised JVP and hepatic engorgement (right sided failure).⁶

Electrical cardioversion is the preferred method of treatment. Synchronized direct-current cardioversion (DCCV) should be attempted as soon as possible. When using a biphasic defibrillator, shocks should start between 120-150J and at 200J with a monophasic defibrillator. Increase in increments during the initial 3 attempts.⁶

If the patient remains in AF and unstable, 300mg amiodarone should be given intravenously over 10-20 minutes (ideally through a central line) before electrical cardioversion is tried again. A further 900mg amiodarone may be given intravenously over 24 hrs.⁶

Look for precipitating and reversible factors:

- Central venous catheters
- Electrolyte and acid base abnormalities (especially hypokalaemia or hypomagnesaemia)
- Hypertension
- Hypovolaemia
- Hypoxia
- Myocardial ischemia
- Pre-excitation syndromes (WPW)
- Pulmonary embolism

- Sepsis, especially pneumonia

Surgery may also play part in the development of AF:

- Atrial and pericardial manipulation
- Cardiac surgery
- Electroconvulsive therapy
- Oesophagectomy
- Pneumonectomy¹

The stable patient

If there are no adverse cardiovascular signs, treatment options include:

- Rate control
- Chemical cardioversion
- Electrical cardioversion
- Prevention of complications with anticoagulation

Control rate with an IV beta-blocker, digoxin, magnesium or combinations of these 3 agents. If the onset of AF is less than 48hrs, amiodarone (may result in chemical cardioversion) may be given as 300mg over 20-60 min followed by 900mg over 23hours.⁶ Digoxin as the sole agent for rate control in persistent AF is commonly used, but is thought to be less effective. Digoxin has no benefit in preventing episodes of paroxysmal AF.²

Other drugs which may be used for rate control in the emergency setting:

- Diltiazem 0.25mg/kg IV over 2 min with a maintenance of 5-15mg/hr
- Esmolol 0.5mg/kg IV over 1 min, maintenance 0.05-0.2mg/kg/min
- Metoprolol 2.5-5mg IV over 2min up to 3 times
- Propranolol 0.15mg/kg IV
- Verapamil 0.075-0.15mg/kg IV over 2 min
- Digoxin 0.25mg IV every 2 hrs up to 1.5mg²

It is vital never to use beta blockers and calcium channel blockers together as the combination may prove fatal.

Broad complex (QRS > 0.12 sec) irregular tachycardia in a stable patient may be AF with aberrant conduction, pre-excited AF or polymorphic VT. Compare the pre-operative ECG with a new 12 lead ECG. Look on the initial ECG for bundle branch block or a delta wave as in Wolff-Parkinson-White syndrome (WPW). Seek expert advice if available. AF with bundle branch block may be treated as above. Amiodarone could be considered in WPW with AF, but avoid adenosine, diltiazem, verapamil and digoxin.⁶

Wolff-Parkinson-White syndrome (WPW): this group of patients have an accessory pathway connecting the atria and the ventricles. In AF the accessory pathway can conduct rapidly leading to a very rapid ventricular response, hypotension and arrest. Digoxin, calcium channel blockers and beta-blockers do not block the accessory pathway and can enhance conduction through the accessory pathway. They should therefore be avoided. If the patient is haemodynamically unstable, electrical cardioversion is indicated. If not under a Cardiologist, this group of patients should be transferred for possible ablation therapy.²

Chemical cardioversion takes longer to work than DCCV and is not as reliable. It tends to be reserved for stable patients and it is therefore advisable to seek the help of an expert.⁶

Reversible precipitants should be identified and dealt with before the administration of anti-arrhythmic drugs.²

In a patient with uncomplicated AF a beta-blocker could be tried, but flecainide and sotalol are very effective.²

In **adrenergically associated** AF the first choice is a beta blocker followed by sotalol or amiodarone.²

Congestive cardiac failure: Patients are prone to the pro-arrhythmic effect of anti-arrhythmic drugs. Most agents are negatively inotropic. Amiodarone is recommended.²

Coronary artery disease: Beta-blockers may be considered first. Sotalol has good beta-blocking activity and can be the initial agent. Amiodarone is associated with long-term toxicity. Flecainide is not recommended.²

Hypertensive heart disease: Left ventricular hypertrophy causes early post-depolarization with increased risk of 'torsade de pointes' VT. Drugs which do not increase the QT interval are the best option. If the patient is free from coronary artery disease, flecainide is a reasonable choice. Although amiodarone lengthens the QT interval the risk of it being pro-arrhythmic is low.²

WPW syndrome: Radiofrequency ablation is the preferred management option.²

Combinations of a beta-blocker, sotalol or amiodarone with a class Ic agent may be tried if a single agent fails.²

Some of these drugs may become pro-arrhythmic if used with other drugs that prolong the QT interval. Check <http://www.torsades.org.com> for further details about drugs which prolong the QT interval. Deterioration in renal function could lead to accumulation of anti arrhythmic drugs and should be monitored along with K⁺ and Mg⁺⁺ levels. Patients should be warned about symptoms such as dyspnoea, angina and syncope.²

A large proportion of patients with new AF will spontaneously cardiovert within 1-2 days.²

DCCV is still an option in the stable patient with AF for less than 48hrs and this is more effective than drugs in restoring sinus rhythm.⁶

The recovery phase and atrial fibrillation

If a patient develops AF postoperatively in recovery:

- Control the patients rate with AV nodal blocking agents.
- DCCV may be attempted.
- If the AF is recurrent or refractory give anti arrhythmic drugs as for a patient with coronary artery disease.
- Anticoagulation is recommended as for non surgical patients.²

In a patient with newly diagnosed AF there is a suggested "minimal evaluation":

- 12 lead ECG.
- CXR.
- Transthoracic echo to look at valve function, chamber size, peak right ventricular pressure, hypertrophy and pericardial disease.
- Thyroid function tests.

Additional investigations may be indicated:

- Exercise tolerance test.
- 24 hour cardiac monitoring.
- Transoesophageal echocardiogram and
- Electrophysiological studies.⁵

Consider referring the patient to a specialist. It may not be possible to start anticoagulation immediately, but if indicated it should not be omitted.

Anaesthesia for DC Cardioversion

It is not in the best interest of all patients to attempt electrical cardioversion. In a patient with recurrent persistent AF who has had at least one attempt of electrical cardioversion, it may be reasonable to accept the progression of persistent AF to permanent AF and to focus on anticoagulation and rate control.²

Anaesthesia technique

Elective cardioversion is a day case procedure and should be performed on a fasted patient. Adequate anaesthesia must be obtained by using short acting anaesthetic agents enabling rapid recovery (e.g. propofol).²

In the emergency setting the patient may not be starved and airway management must then be as for the non-starved patient with rapid sequence induction.

The anterior-posterior paddle position with one paddle on the sternum and the other on the left scapula seems to be superior to the anterior-lateral position where one paddle is on the ventricular apex and the other inferior to the right clavicle.²

Implanted permanent pacemakers (PPM) or defibrillators (AICD) should be interrogated just before and after electrical cardioversion to establish good working order. Anterior-posterior paddle position is the preferred position since the paddles should be as far away from the device as possible.²

The anaesthetic position of the patient could be improved by using hands free paddles.

The risks of electrical cardioversion mainly relate to arrhythmias and thromboembolism. Transient ST-segment elevation may appear and cardiac enzymes may be elevated without obvious myocardial damage. A variety of arrhythmias are possible:

- Ventricular and supraventricular premature beats
- Bradycardia (may indicate a conduction defect in the absence of AV nodal blocking agents)
- Sinus arrest
- VT and VF (with hypokalemia and digoxin toxicity).²

Anticoagulation

If the duration of AF is more than 48hrs (or unknown) the patient should be anticoagulated for 3 to 4 weeks before cardioversion (electrical or chemically). The patient should also be anticoagulated for 3-4 weeks after successful cardioversion. The left atrium and left atrial appendage (LA/LAA) may take several weeks after cardioversion to recover from mechanical "stunning" and during this recovery the patient remains at risk of thrombus formation.²

Alternatively, the patient could be screened for a LA/LAA thrombus with transoesophageal echo. If thrombus has been identified the patient should be anticoagulated for 3-4 weeks prior to attempted cardioversion. If there is no thrombus heparin should be given before cardioversion, as a bolus followed by an infusion, aiming for an activated partial thromboplastin time (APTT) of 1.5-2 times the control time. Successful cardioversion should be followed by oral anticoagulation for 3 – 4 weeks.²

In the acute setting with haemodynamic instability where DCCV is urgent, unfractionated or low-molecular-weight fractionated heparin should be used. Protection against late embolism may require further oral anticoagulation, but this will depend on the risk of recurrence and the patient's intrinsic risk for thromboembolism.²

Recurrent and refractory AF

Some patients have resistant AF and DCCV is entirely unsuccessful. Others convert back to AF 1-2 minutes after cardioversion. In a further group AF will recur from 1 day up to 2 weeks after successful cardioversion. The first 2 groups account for 25% of patients and the last group for a further 25%.²

Drugs which increase the DC cardioversion success rate and decrease early recurrence are most appropriate in patients who have previously failed to convert to sinus rhythm and those with immediate or subacute recurrence of AF.²

Aim to obtain therapeutic plasma levels before and for a few weeks after cardioversion with one of the following agents:

- Amiodarone
- Flecainide
- Quinidine
- Sotalol²

Patients receiving drugs which increase the QT interval should be observed in hospital for 24 to 48 hours after cardioversion.²

Possible risks involved include:

- Paradoxical increase in defibrillation threshold (flecainide)
- Accelerated ventricular rate in the absence of AV nodal blocking (Ia and Ic) and
- Ventricular pro-arrhythmic effect.²

Summary

Management of AF is divided into rhythm control, rate control and anticoagulation. In the unstable patient DCCV is indicated. If the patient is stable, cardioversion may be attempted electrically or chemically. An asymptomatic patient may only require rate control. Anticoagulation reduces the risk of thromboembolic complications. Several factors need to be kept in mind when prescribing or administering drugs for patients in AF.

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