

ATRIAL FIBRILLATION - a patient's guide

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What is it?

Atrial fibrillation (AF) is a rhythm irregularity of the heart, where the small chambers of the heart (the atria) contract in an ineffective haphazard fashion, resulting in an irregular heart beat. This may be accompanied by a rapid heart rate, a reduction in the blood pressure, and reduced filling of the main chambers (the ventricles), causing reduced pumping of the blood by the heart. Many patients may be unaware that they have the condition.

AF may be chronic (there all the time) or come and go (paroxysmal).

Important causes of Atrial Fibrillation

Cardiac:

- Ischaemic heart disease (coronary artery disease)
- Hypertension (high blood pressure)
- Rheumatic heart disease
- Cardiac electrical syndromes (e.g. Wolff-Parkinson-White)
- Pericardial diseases (diseases of the lining of the heart)
- Cardiomyopathy (diseases of the heart muscle)

Non-cardiac:

- Overactive thyroid gland
- Acute infections such as pneumonia (usually the AF is transient)
- Alcohol (acute and chronic use)
- Pulmonary embolism (blood clots in lung)
- Following cardiac/chest surgery
- Lung cancer

The first two mentioned causes are the most common, accounting for about 65% of cases. Some patients have no identifiable cause. They are referred to as having "lone" AF.

Diagnosis

The diagnosis can be made by feeling the pulse and listening to the heart. An ECG (electrocardiograph) is needed to confirm the diagnosis and make sure that the irregularity is not due to other conditions.

Consequences of Atrial Fibrillation

As a result of AF, the blood pressure may drop, and the heart rate may be rapid. This reduces blood output from the heart, and can result in congestion in the legs and lungs, especially when pre-existing heart weakness is present. Poor contraction of the atria and

enlargement of the atria can cause clots to form in the atria. These clots can travel in the circulation and cause strokes and blockages of other arteries.

Clinical symptoms

Fatigue, reduced exercise ability and breathlessness may be symptoms due to AF. Palpitations and lightheadedness may also be experienced. Many patients will have minimal symptoms and not be aware that they have the condition. The symptoms depend to a large extent on how rapid the heart rate is and the general fitness of the patient.

There may only be a vague awareness that the heartbeat feels irregular.

Other rarer symptoms include those of a stroke, or pain and swelling in a limb as a result of a limb clot, or even abdominal pain, these symptoms resulting from clots travelling in the circulation.

What can be done about it?

There are four aspects to therapy:

1. Converting AF back to the normal cardiac rhythm (sinus rhythm).
2. Preventing attacks of AF in cases where it comes and goes (paroxysmal AF).
3. Controlling the AF, particularly if the heart rate is rapid.
4. Preventing clot formation and its consequences.

Converting AF back to the normal cardiac rhythm (sinus rhythm)

All patients with AF should be considered for an attempt to convert the heart rhythm back into sinus (normal) rhythm. If reversible causes can be successfully treated first (such as thyroid disease), this increases the success rate of conversion back to the normal rhythm (cardioversion).

Cardioversion can be accomplished by drugs, or by an electrical shock to the heart while under anaesthesia. Drugs that have been used include "class 1" antiarrhythmic drugs, such as flecainide, quinidine, and propafenone, and "class 3" agents such as amiodarone. All these drugs have potentially serious side-effects, and should be supervised by a specialist. Success rates vary from 20% to 80% depending on the underlying cause of the AF. Electrical cardioversion has similar success.

All patients who are being considered for cardioversion should be on anticoagulation therapy (thinning of the blood, usually achieved with a drug called warfarin) prior to the procedure unless the AF is of less than 2 days duration. Anticoagulation should be continued for at least 4 weeks after cardioversion, and in cases where a high risk of relapse exists, longer.

Cardioversion is less likely to be successful in older patients, those who have had AF for more than a year, those with hypertension, and those with structural heart disease.

Preventing attacks of AF in cases where it comes and goes (paroxysmal AF)

The drugs mentioned above may be used to prevent attacks of AF, particularly after successful conversion into sinus rhythm. About 50% of patients remain in sinus rhythm one year after cardioversion, provided they remain on anti-arrhythmic drugs. Flecainide appears to be the most effective drug at maintaining sinus rhythm, but has potential to cause serious side effects, particularly when the patient has coronary artery disease or a weak heart. Amiodarone and sotalol are less effective, but are probably safer.

Controlling the AF, particularly if the heart rate is rapid

In cases where the AF is permanent and cannot be converted to normal rhythm, and where the rate is rapid, the heart rate can be controlled with drugs that slow the electrical impulse from the atria to the ventricles. Useful drugs include beta-blockers, and calcium antagonists (such as diltiazem). Digoxin may also be used for this purpose, particularly when the AF is accompanied by heart failure.

Other drugs directed against the cause of the AF (such as anti-thyroid drugs), as well as drugs such as diuretics (for heart failure) may also be needed.

Preventing clot formation and its consequences

In patients with chronic or paroxysmal AF, it is important to prevent clot formation in most cases. This is usually achieved with warfarin or coumarin drugs that interfere with the formation of the body clotting factors, thereby thinning the blood. Unfortunately, this necessitates regular blood test monitoring, as the dose needs to be individualised.

Research shows that the risk of a stroke for patients with AF on no clot prevention treatment, particularly if they are older or have had a previous stroke, is about 8-10% per year. This risk can be reduced with warfarin to about 4% per year. However, warfarin itself has a risk of causing bleeding of around 0.5% per year. This risk of bleeding increases in patients with poorly controlled dosage, increasing age, history of bleeding (e.g. bleeding ulcers), alcohol excess, certain drugs that interfere with warfarin, and liver disease. In some older patients, the risk of falls may also be an issue that argues against warfarin use. Thus the decision to use warfarin is justified in many cases of AF, but needs to be carefully considered.

An alternative to warfarin is aspirin. Aspirin does reduce the risk of stroke, but is only about half as effective as warfarin. However, it has less risk of causing bleeding, and therefore is useful in situations of increased bleeding risk.

In younger patients, particularly those with "lone" AF (i.e. no identifiable cause and no cardiac abnormality), the risk of clot formation causing stroke is low, and clot prevention therefore may not be required.

Future research and developments

Several drugs are currently under investigation, such as dofetilide. These drugs may prove to be more effective in restoring and maintaining a normal heart rhythm. However, the ideal safe and effective "perfect" drug remains to be discovered.



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The use of transoesophageal ECHO, whereby ultrasound pictures of the chambers of the heart are viewed via a tube down the throat, is gaining use in assessing clearly whether any clot resides in the heart prior to cardioversion. In some cases this reduces the need for anticoagulation therapy.