Management of atrial fibrillation

The electrocardiogram and AF

The normal electrocardiogram (ECG) is described as having PQRS waves. The P wave denotes atrial depolarisation, and the QRS wave denotes ventricular depolarisation. In atrial defibrillation there is no coordinated atrial depolarisation, so the P wave is lost.

In patients in AF there are no recognisable P waves on the ECG, nor is any other form of coordinated atrial activity seen in any lead. The baseline is irregular, and chaotic atrial activity is best seen in V1 (the first of the six chest leads, where a waveform irregular in frequency is recorded. The deflections are known as fibrillatory waves. If they are small they are called fine fibrillatory waves, and if large, coarse fibrillatory waves (Huff, 1997).

Transmission of atrial depolarisation to the ventricles is through the atioventricular junction and depends on the refractory period of the tissues involved. The ventricular rate and response will therefore vary accordingly.

ECG characteristics

The characteristics of an electrocardiogram can be summarised as follows:

- Rhythm: grossly irregular; the marked irregularity of the ventricular response is one of the most distinguishing features of AF (Fig 1);
- Atrial rate: 360–600 beats per minute, although this is immeasurable on the surface ECG;
- Ventricular rate: this varies – it may be less than 50 beats per minute to more than 200;
- P wave: there is no P wave; there will be an irregular undulating baseline, the deflections of which are called fibrillatory waves, and are of varying shapes, amplitude and direction;
- P–QRS relationship: QRS complexes occur at irregular intervals in random association with fibrillation waves. The ventricular rate is slower than the atrial rate and will depend on the number of impulses conducted through the AV node to the ventricles.
- P wave: there is no P wave; there will be an irregular undulating baseline, the deflections of which are called fibrillatory waves, and are of varying shapes, amplitude and direction;

**Complications of AF**

In the absence of treatment, the resulting ventricular rate will be rapid, as many of the atrial impulses will arrive at the atioventricular node and be conducted. A ventricular rate in the range of 120–200 beats per minute often results. This can be detected as a radial pulse that is irregular in both timing and volume. There is often a pulse deficit between apical and radial pulses (the radial pulse is less than the apical pulse). Patients often present with palpitations, faintness, hypotension and dyspnoea, and rapid rates may be poorly tolerated. Other common complications include:
- Mural clot formation as a result of stasis of blood in the atria owing to the ineffectively contracting atrium, leading to a stroke;
- Emboli, either pulmonary or systemic;
- A dramatic fall in cardiac output, with rapid ventricular responses for diastolic filling and loss of the atrial contribution to ventricular filling;
- Left ventricular failure;
- Angina;
- Dizziness and syncope.

**Standard treatment of atrial fibrillation**

There are three main components to the treatment of AF:
- Control of ventricular rate;
- Restoration of sinus rhythm;
- Prevention of embolism with anticoagulation therapy.

The priorities of treatment depend on the patient’s tolerance of the rhythm. It is also important to find the underlying cause to try and prevent its recurrence should the rhythm be successfully treated. The Resuscitation Council’s peri-arrest algorithm gives extremely useful guidelines on the treatment of AF (Resuscitation Council UK, 2000). The algorithm is summarised in Fig 2 (p44).

Control of heart rate can be attempted with chemical cardioversion. For example, amiodarone has shown a 75 per cent success rate, while sotalol has been proven useful in preventing recurrence of AF. Cardioversion is the initial treatment of choice if the patient is haemodynamically unstable (Resuscitation Council UK, 2000).

An emergency cardioversion is undertaken if the patient is compromised by the dysrhythmia; that is, if the blood pressure is below 90mmHg, the heart rate is greater than 150 beats a minute or the patient has ongoing chest pain. The patient is sedated/anaesthetised, and asynchronised DC shock starting at 100 Joules is administered. If this is unsuccessful, 200 Joules can be administered, followed by 360 Joules or appropriate...
Normal atrial fibrillation occurs in three clinical circumstances:

- As a primary arrhythmia in the absence of identifiable structural heart disease;
- As a secondary arrhythmia in the absence of structural heart disease but in the presence of a systemic abnormality that predisposes the individual to the arrhythmia;
- As a secondary arrhythmia associated with cardiac disease that affects the atria (Prystowsky et al, 1996).

The most common causes of AF are listed in Box 1. Three types have been identified: acute, chronic, and lone/primary.

- Acute AF: This has an onset within 24–48 hours of the causative event and usually converts spontaneously or in response to an antiarrhythmic agent (cardioversion). It may occur in individuals who are clinically normal but who have a temporary change in their condition; for example, it may occur in people who have consumed excessive alcohol;
- Chronic AF – this may be paroxysmal, and is the most debilitating form of AF because of its abrupt onset. It may be persistent or permanent and requires intervention by cardioversion to sinus rhythm (Marriott and Conover, 1998);
- Lone or primary AF – this occurs in the absence of any other clinical evidence that would suggest a primary cardiac disorder.

**REFERENCES**


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biphasic energy. The patient may also be treated with intravenous amiodarone.

Atrial contraction may be delayed following the return of atrial activity despite the return of apparent P wave activity on the ECG. Because of this atrial paralysis, anticoagulants must be continued for at least two weeks. Patients with chronic AF may not convert to sinus rhythm with any therapy. Treatment of these patients is directed at controlling the ventricular rate and providing anticoagulation for them.

**Nursing interventions**

The main goal of treatment is to maintain adequate cardiac output and tissue perfusion and to ensure that the patient does not develop a thromboembolism. The medical team should be notified immediately if the patient’s heart rate is rapid and if the systolic blood pressure is less than 90mmHg (Resuscitation Council UK, 2000).

If an emergency cardioversion is required for haemodynamic compromise it is important to explain the procedure and reassure the patient. Careful positioning of these patients is important to avoid exacerbating their condition. They should be nursed in a semi-recumbent position and avoid lying flat as this increases pre-load/blood return to the heart, which may increase the danger of heart failure. If the patient is breathless, administration of oxygen may be useful.

To help allay patients’ anxiety, the likely causes of their symptoms should be explained to them, and suggestions made as to what may be done to remedy them. Patients should be asked if they have any chest discomfort, as this is suggestive of myocardial ischaemia, in which case oxygen therapy may be of use. During the acute stages the patient will require continuous cardiac monitoring.

All patients with AF will require anticoagulation therapy, and its effects require continuous monitoring. Also, appropriate teaching about the purpose of intravenous/oral medications is essential.

Once sinus rhythm has been restored further, patient teaching on drug therapy may be required. Patients receiving digoxin should be taught how to take their own pulse, how to recognise the signs and symptoms of toxicity and be aware of the importance of periodic measurement of serum digoxin levels.

**Conclusion**

Patients with AF can present as acutely ill but usually respond rapidly to treatment. During this time, nurses can not only provide physical support to patients but also...