

ARRHYTHMIAS

1 Introduction

1.1 Definition

Cardiac arrhythmia is an abnormality of cardiac rhythm of any type.

Normal sinus rhythm is arbitrarily defined as a regular rate of between 60 and 100 beats per minute, and any variation from this is, by definition, an arrhythmia. The range of arrhythmias varies from innocent extrasystoles to immediate life-threatening conditions such as asystole or ventricular fibrillation.

Arrhythmias may be present in the absence of cardiac disease, but are more commonly associated with structural heart disease or external provocative factors.

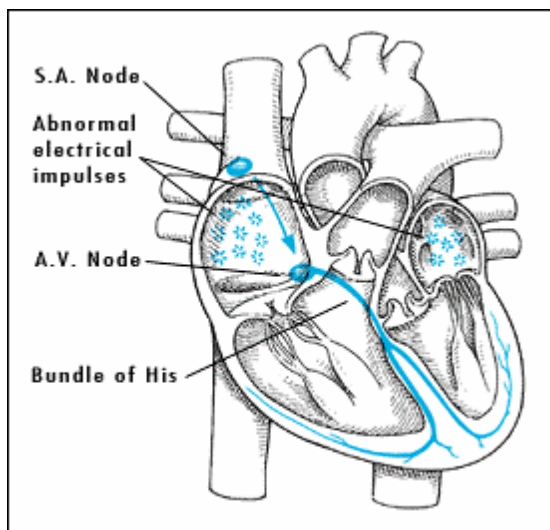
Transient arrhythmias such as extrasystoles are very common and do not usually indicate heart disease. They may be provoked by a variety of substances e.g. alcohol or caffeine.

2 Description

2.1 Aetiology

The normal cardiac impulse originates from the pacemaker cells in the sinus node. Conduction proceeds from the sinus node through the atria to the atrioventricular (AV) node and the His-Purkinje system to activate the ventricles.

Arrhythmias occur due to a disorder of the above system as in the diagram below.



Bradyarrhythmias may result from impairment of the impulse formation or conduction. This may be due to various factors e.g. drugs, myocardial ischaemia, or pathological infiltration.

The mechanism responsible for tachyarrhythmias is not known in all cases. There is usually an interaction between an underlying cause (e.g. ischaemia) and a triggering event such as an extrasystole. (see Appendix A).

2.2 Prevalence

The most common arrhythmia is extrasystole.^[1]

A few individuals will suffer from recurrent arrhythmias. It is difficult to obtain precise figures as to the prevalence of arrhythmias as they can be asymptomatic or unreported.

The commonest is atrial fibrillation (AF) which affects 2 per cent of the population at some time in their lives. AF is found in 15% of all stroke patients and 2-8% of patients with transient ischaemic attacks (TIAs). It tends to be paroxysmal in people of working age. (See Appendix B)

Medical Services

Acute myocardial infarction is one of the commonest causes of arrhythmia, particularly in the first few hours after infarction. Arrhythmias may also occur with chronic ischaemia.

3 Diagnosis

3.1 History and Symptoms

Arrhythmias may cause no symptoms.

The type of symptoms which they cause depends on the degree of irregularity and the amount of underlying cardiac disease.

A careful and accurate history with a detailed description of the symptoms associated with the arrhythmia is essential.

Evidence should be sought for factors which may precipitate the arrhythmia (e.g. drugs, caffeine, alcohol, exercise). A search should be made for other associated diseases (e.g. valvular heart disease, ischaemia or heart failure).

Tachycardias may cause low cardiac output with a reduced cerebral blood flow sufficient to cause syncope. Other symptoms of tachycardia include a feeling of rapid palpitation, angina or dyspnoea. There may be associated polyuria.

Ectopic beats are commonly asymptomatic. They may produce a sensation of the heart missing a beat, often followed by a bump in the chest due to the more powerful post ectopic beat.

The symptoms produced by bradyarrhythmias will depend on the extent of cardiac slowing. They may include syncope (Stokes-Adams attacks) or dizziness. Continuous bradycardia may produce symptoms of fatigue, lethargy, dyspnoea or mental impairment.

Bradyarrhythmias can cause sudden death.

3.2 Examination

Physical examination may be normal if the arrhythmia is intermittent. It may reveal signs of structural heart disease e.g. rheumatic heart disease.

In atrial fibrillation the pulse is “irregularly irregular” and there may be a variable absence of the first heart sound. Extrasystoles are detected as missed beats and occasional irregularities of the pulse.

3.3 Investigations

Basic investigations include blood tests (FBC, U&E, glucose, TFTs, lipids, LFTS) chest x-ray and ECG. Echocardiography and exercise stress testing are performed when appropriate.^{[2][3]}

Medical Services

The key to successful diagnosis is a systematic analysis of the ECG during the arrhythmia.

When arrhythmias are intermittent, continuous monitoring may be necessary for identification. A 24 hour ECG is therefore the investigation of choice.

This may include inpatient monitoring, particularly in the acute stages of a myocardial infarction, or outpatient ambulatory (Holter) monitoring for periods of up to 48 hours.

Patients who have infrequent sustained palpitations may use a patient activated recorder.

More detailed investigations of arrhythmias may involve electrophysiological testing to record the impulses from the different parts of the conduction system. This may be appropriate if accessory pathways are suspected, or in the evaluation of tachyarrhythmias. These studies may identify an accessory pathway and allow for it to be ablated.

Tachycardia may be artificially stimulated in order to assess the effect of anti-arrhythmic drugs.

4 Treatment

Most cardiac arrhythmias are benign and require no active intervention.

The major indications for treatment are the relief of symptoms and the prevention of complications such as myocardial ischaemia, cardiac failure or embolism.

The presence of structural heart disease is an important factor in the decision to institute active therapy.^[2]

The same arrhythmia may be treated in a patient with underlying heart disease, but left untreated in a patient with a structurally normal heart.

Precipitating factors such as caffeine, thyrotoxicosis, alcohol or electrolyte disturbance should be sought. Avoidance of these may be sufficient to prevent recurrence of the arrhythmia without further intervention.

The type of therapy used will commonly be influenced by the presence of underlying ischaemic heart disease or left ventricular impairment. Any associated cardiovascular disease must be treated.^[1]

Patients with a sustained tachyarrhythmia will normally require therapy to restore sinus rhythm.

Long term prophylaxis may be indicated in the presence of life threatening tachyarrhythmia, or if the patient has a history of recurrent symptomatic attacks of palpitations.

In chronic atrial fibrillation, it may be unrealistic to expect that sinus rhythm will be maintained.

In the bradyarrhythmias, the initial treatment is to increase the ventricular rate by either drugs or pacing. Permanent pacing may be necessary unless the cause of the bradyarrhythmia is transient.

4.1 Drug Therapy

All drugs used in the treatment of arrhythmias have potentially serious side effects and must be used with caution. The ability of an anti-arrhythmic drug to worsen the arrhythmia or produce new arrhythmias is being increasingly recognised.^[4]

No exact classification exists to accurately predict the efficacy of a given drug for a given arrhythmia. Thus, therapy is initiated often on the bases of trial and error (See Appendix C).

Side effects of medication may increase the disabling effects of the condition. They are listed in Appendix D.

4.2 Non-Drug Therapy

Simple non-drug therapy entails manoeuvres which produce vagal stimulation. Carotid sinus massage and particularly the Valsalva manoeuvre are used, and patients with recurrent supraventricular tachycardias should be taught these techniques.^[5]

Pacing is appropriate for certain arrhythmias e.g. atrial flutter, AV nodal re-entry and supraventricular tachycardia.

Facilities for cardioversion must be available due to the danger of ventricular fibrillation. Implantable pacemakers have been used in patients with drug-refractory tachycardias.

Cardioversion can be used to convert persistent atrial fibrillation to sinus rhythm when it is considered likely that the patient will remain in sinus rhythm for the long term.

Catheter ablation is increasingly being used with a consequent decrease in the role of cardiac surgery. It may be curative in the case of supraventricular tachycardia.

An implantable cardioverter defibrillator (ICD) is now the preferred treatment for ventricular tachycardia or fibrillation when the arrhythmia is refractory. Usually, the individual will have experienced at least one cardiac arrest.

5 Prognosis

Prognosis depends on the type of arrhythmia. There may be no excess mortality (e.g. with extra systoles), or a very high mortality (e.g. ventricular fibrillation).

A worsening prognosis occurs if the arrhythmia is associated with other aggravating factors such as ischaemia or macroscopic abnormalities of the myocardium.

Atrial fibrillation carries a particularly poor prognosis if associated with:

- Mitral valve disease
- Enlargement of the left atrium
- Hypertension
- Old age
- Diabetes
- History of embolism.

In complete atrio-ventricular block, the prognosis is poor without pacemaker implantation and improves markedly following permanent pacing. It is still dependent afterwards on the underlying cardiac disease.

Patients with Wolff-Parkinson-White syndrome are at increased risk of the development of atrial fibrillation. The major prognostic concern in this syndrome is the rapid conduction to the ventricle. If the refractory period is particularly short, it may result in ventricular fibrillation with the associated very poor prognosis.

Supraventricular tachycardia (SVT) is a potentially life threatening condition. Unless the acute episode has been clearly precipitated by some transient or reversible factor, there is a high probability of recurrent attacks which may result in sudden death.

The three-year survival in SVT varies from 80 per cent in patients in whom the arrhythmia is suppressed by therapy, to 40 per cent in those in whom effective suppression is not achieved.

The prognosis for cardiac arrest depends on the speed of resuscitation and the attendant heart disease. The long-term survival following discharge from hospital after cardiac arrest is about 50 to 60 per cent at five years.^[1]

6 Main Disabling Effects

6.1 General Considerations

As with prognosis, the disability caused by an arrhythmia depends on the type of arrhythmia and the associated disease(s).

Transient arrhythmias such as extra systoles do not usually cause any disability.

When drug treatment is necessary, temporary absence from work and a short period of rest may be advisable.

First and second degree heart block may be incidental findings in otherwise healthy people.

The presence of an implanted pacemaker is entirely compatible with normal and even strenuous exercise. The underlying heart condition, however, may impose its own restrictions.

The disability in patients with serious arrhythmias is determined by the underlying cardiac disorder such as myocardial scarring or cardiomyopathy.

In common with other cardiovascular disorders, the major difficulty day to day is caused by limitation of exercise tolerance. Arrhythmias also carry the risk of sudden incapacity, e.g. syncope or loss of vision due to an embolus.

Psychological factors such as anxiety or depression may increase the disability in heart disease, as may general anxiety in the individual's family.^{[6][7]}

Complete heart block with a permanent pacemaker needs special consideration in the work place as certain electrical sources can potentially affect pacemakers.

There has been considerable speculation as to whether mobile phones might interfere with pacemakers and ICDs. It has been shown to be a theoretical possibility if the phone is held less than 20 cm from the pacemaker. Individuals are, therefore, advised to use the hand and ear away from the pacemaker, and to dial with the telephone held away from the body.

6.2 Assessing the Claimant

A claimant with a stable arrhythmia on treatment would usually retain the ability to self-care. Similarly, walking on the flat ground would not be contraindicated.

As with all cardiovascular disorders, the assessment in arrhythmias should include full details of:

- Exact medical history

Medical Services

- Medication – with side effects
- Likelihood of further treatment
- Details of current daily activities
- Observed behaviour
- Directed clinical examination.

No generalisations can be made, and each client should be individually assessed. In the IB-PCA the functional areas which may be affected are:

- Rising from sitting
- Bending and kneeling
- Standing
- Walking
- Walking up and down stairs.

Impairment of upper limb function is unlikely to feature as weights of only up to 2.5kg are considered under the PCA regulations.

Remaining conscious is not likely to be affected.

Exemption should be considered if the client is severely restricted by the cardiovascular disorder. A non-functional descriptor may be appropriate if there are uncontrollable episodes of loss of consciousness.

Appendix A - Causes of Arrhythmias

SINUS BRADYCARDIA:

Physiological: youth, athletes, sleep;

Sino-atrial Disease:

Drugs (e.g. beta-blockers, calcium channel blockers, amiodarone);

Vasovagal syndrome;

Inferior myocardial infarction;

Hypothyroidism;

Hypothermia;

Other e.g. jaundice, raised intracranial pressure.

AV BLOCK:

Drugs (e.g. digoxin, verapamil);

Idiopathic Systemic Fibrosis;

Acute myocardial infarction or ischaemia;

Infiltration: aortic stenosis, sarcoid, scleroderma, tumour;

Infection: diphtheria, rheumatic fever, Lyme disease, endocarditis;

Vagal: athletic heart, vasovagal syndrome;

Dystrophia myotonica.

TACHYCARDIAS:

Sinus tachycardias and re- entry;

Atrial tachycardias;

Atrial Flutter;

Atrial Fibrillation.

Appendix B - Aetiology of Atrial Fibrillation

Increased atrial pressure/wall tension: mitral valve disease, heart failure, left ventricular hypertrophy, cardiomyopathy;

Myocardial ischaemia/infarction;

Thyrotoxicosis;

Alcohol;

Sino-atrial disease;

Infiltration: constrictive pericarditis, tumour;

Infection: myo/pericarditis, pneumonia;

Retrograde activation: Wolff-Parkinson-White syndrome, ventricular pacing;

Cardiac or thoracic surgery;

Idiopathic: 'lone' atrial fibrillation.

Appendix C - Commonly Used Antiarrhythmic Drugs

Atrial Fibrillation:

- Digoxin
- Atenolol
- Sotalol
- Amiodarone
- Verapamil.

Ventricular Tachycardia:

- Quinidine
- Disopyramide
- Procaineamide
- Lignocaine
- Mexiletine
- Flecainide
- Sotalol
- Amiodarone

Supraventricular Tachycardia:

- Atenolol
- Verapamil
- Adenosine

Wolff-Parkinson-White Syndrome:

- Flecainide
- Amiodarone
- Sotalol

Appendix D - Common Side effects

Digoxin: anorexia, nausea, vomiting, diarrhoea, abdominal pain, confusion, visual disturbance, heart block.

β blockers: bradycardia, sleep disturbance, nightmares, heart failure, hypotension, bronchospasm, peripheral vasoconstriction.

Amiodarone: light sensitivity, erythema, neuropathy, impaired vision, conduction disturbances, tremor, potentiates digoxin and warfarin, needs regular checks of LFTs, TFTs, and pulmonary function.

Verapamil: constipation, nausea, vomiting, flushing, headaches, fatigue, oedema.

Quinidine/Procainamide: nausea, diarrhoea, rashes, myocardial depression, fever, arrhythmias, thrombocytopenia.

Disopyramide: tachycardia, fibrillation, anticholinergic side effects (dry mouth, blurred vision).

Flucainide: dizziness, visual disturbances, nausea, arrhythmias.

7 References

1. Coronary Heart Disease Statistics, London. British Heart Foundation, 1997.
2. Clark WL, Alpern HL, et al. Suggested Guidelines for Rating Cardiac Disability in Workers Compensation. *West J Med* 1993;**156**: 263-7.
3. Remme WJ, Swedberg K. Task Force for the Diagnosis and Treatment of Chronic Heart Failure. *European Heart Journal* 2001; **22**: 1527-1560.
4. Mitchell LB. Use of the newer anti-arrhythmic drugs in patients with supraventricular tachycardias; let's not throw out the baby with the bath water. *Can J Cardiol* 1996; 12 (No 10) 972-982
5. Paul W Armstrong, Gordon W Moe. Medical advances in the treatment of congestive heart failure. *Circulation* 1993; **88**(No 6): 2941-2952
6. Cay EL, Walker DD. Psychological factors and return to work. *European Heart Journal* 1988; **9** (Supplement L): 74-81.
7. Cay. The influence of psychological problems in returning to work after a myocardial infarction. *Cardiac Rehabilitation* 1983. Proceedings of the Society of Occupational Medicine Research Panel. Symposium London 42-60.

8 Bibliography

1. Lecture Notes on Cardiology 2002 Fourth edition
Iain Sympson; Keith Dawkins; John Morgan; Huon Gray.
2. Oxford Textbook of Medicine 1995 Third edition
3. French's Index of Differential Diagnosis 1996 Thirteenth edition
Editors: Ian Bouchier; Harold Ellis; Peter Fleming
4. Rehabilitation of the Physically Disabled Adult 1997 Second edition
Editors: C John Goodwill; M. Anne Chamberlain; Chris Evans.
5. Price's Textbook of Medicine 1993 Twelfth edition
Edited by Sir Ronald Bodley Scott
6. Disability Evaluation 1996
Stephen L Demeter; Gunnar B J Andersson; George Smith
7. ABC of Heart Failure 2000
Christopher R Gibbs; Michael K Davies; Gregory Y H Lip
8. Fitness for Work 2000 Third Edition
R.A.F. Cox with F.C. Edwards and K. Palmer
9. The Medical Services Incapacity Benefit Handbook for Approved Doctors. April 2000
10. Social Security Legislation 2001 Volume 1: Non Means Tested Benefits
Commentary by David Bonner, Ian Hookes, Robin White.
11. At A Glance guide to the Current Medical Standards of Fitness to Drive.
Swansea; Driver and Vehicle Licensing Agency, March 1998.