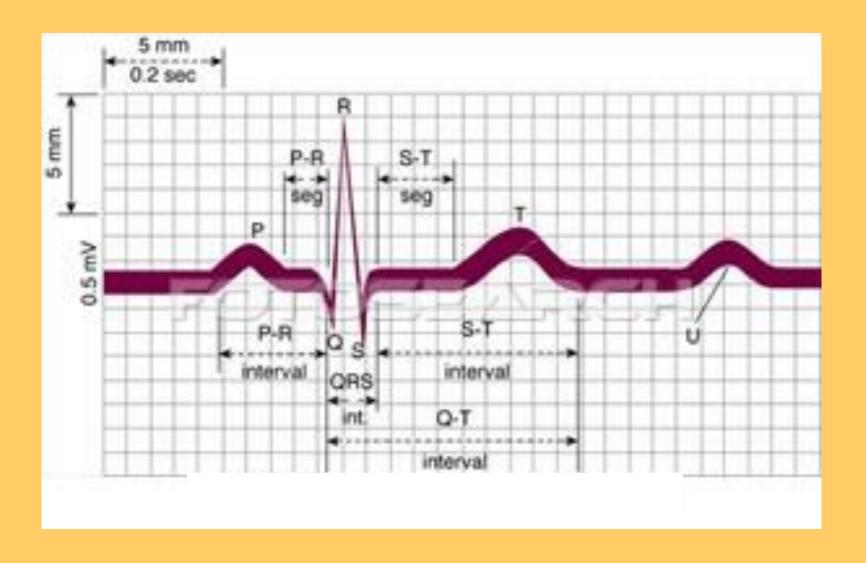
MANAGEMENT OF CARDIAC

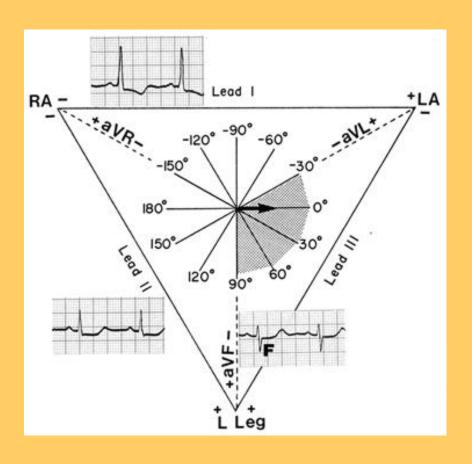
ARRHYTHMIAS

Prof. Arjuna de Silva MBBS,MD,MRCP(UK),MSc(Oxon),FRCP,FCCP, AGAF,FNASSL

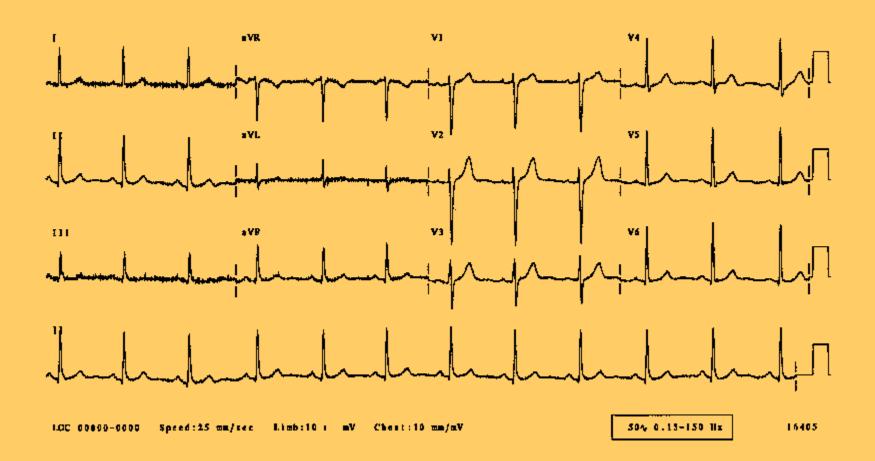
Normal ECG



Normal ECG (continued)



Normal



Normal sinus rhythm

- each P wave is followed by a QRS
- P waves normal for the subject
- P wave rate 60 100 bpm with <10% variation
 - rate <60 = sinus bradycardia
 - rate >100 = sinus tachycardia
 - variation >10% = sinus arrhythmia

Normal P waves

- height < 2.5 mm in lead II
- width < 0.11 s in lead II
 - for abnormal P waves see <u>right atrial hypertrophy</u>, <u>left atrial hypertrophy</u>, <u>atrial premature beat</u>, <u>hyperkalaemia</u>

Normal PR interval

- -0.12 to 0.20 s (3 5 small squares)
 - for short PR segment consider <u>Wolff-Parkinson-White syndrome</u> or <u>Lown-Ganong-Levine</u>
 <u>syndrome</u> (other causes Duchenne muscular dystrophy, type II glycogen storage disease (Pompe's), HOCM)
- for long PR interval see <u>first degree heart</u> <u>block</u> and <u>'trifasicular' block</u>

Normal QRS complex

- < 0.12 s duration (3 small squares)</p>
 - for abnormally wide QRS consider <u>right</u> or <u>left</u> bundle branch block, ventricular rhythm, <u>hyperkalaemia</u>, etc.
- no pathological Q waves
- no evidence of <u>left</u> or <u>right</u> ventricular hypertrophy

Normal QT interval

- Calculate the corrected QT interval (QTc) by dividing the QT interval by the square root of the preceding R - R interval. Normal = 0.42 s.
- Causes of long QT interval
 - myocardial infarction, myocarditis, diffuse myocardial disease
 - hypocalcaemia, hypothyrodism
 - subarachnoid haemorrhage, intracerebral haemorrhage
 - drugs (e.g. sotalol, amiodarone)
 - hereditary -Romano Ward

Normal ST segment

- no elevation or depression
 - causes of elevation include acute MI (e.g. <u>anterior</u>, <u>inferior</u>), <u>left bundle branch block</u>, normal variants (e.g. athletic heart, Edeiken pattern, high-take off), acute pericarditis
 - causes of depression include myocardial ischaemia, <u>digoxin effect</u>, <u>ventricular hypertrophy</u>, <u>acute posterior MI</u>, <u>pulmonary embolus</u>, <u>left bundle</u> branch block

Normal T wave

- causes of tall T waves include <u>hyperkalaemia</u>, <u>hyperacute myocardial infarction</u> and <u>left bundle</u> branch block
- causes of small, flattened or inverted T waves are numerous and include ischaemia, age, race, hyperventilation, anxiety, drinking iced water, <u>LVH</u>, drugs (e.g. <u>digoxin</u>), pericarditis, <u>PE</u>, intraventricular conduction delay (e.g. <u>RBBB</u>)and electrolyte disturbance.

Classification of arrhythmias

Tachyarrhythmia
 HR>100

Bradyarrhythmia

HR<60

HR<50

Broad complex (QRS) arrhythmias. Narrow complex (QRS) arrhythmias

Classification

- Supraventricular arrhythmias
- Ventricular arrhythmias

Broad complex (QRS) arrhythmias



Broad regular

Broad irregular

VT

VF

SVT

WPW

Symptoms

- Asymptomatic (when no cardiac lesion)
- Palpitation (tachyarrhythmia)
- Ischemic chest pain
- Symptoms of cardiac failure
- Disturbed consciousness

Management

Correct diagnosis with ECG

 Clinical assessment for haemodynamical stability

Signs of haemodyanamical instability

Presence of SBP<90 or

Inadequate peripheral circulation

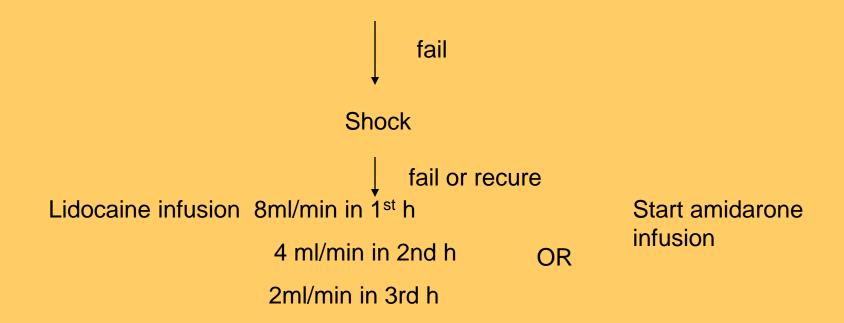
Pulmonary oedema

Management of unstable patient with tachyarrhythmia

Cardioversion with 100 J (Synchronize)

Management of stable patient with tachyarrhythmia

lidocaine 50-100mg iv within 2mins



Amidarone

- Iv 150mg over 10 minutes
- Iv 300mg over 6 hours
- Iv 900mg over 18 hours
- Then switch to oral

Supraventrycular arrhythmias

- Atrioventricular nodal re-entrant tachy cardia (AVNRT)
- Atrioventricular reciprocating tachycardia(AVRT)
- Atrial fibrillation
- Atrial flutter
- Atrial tachycardia

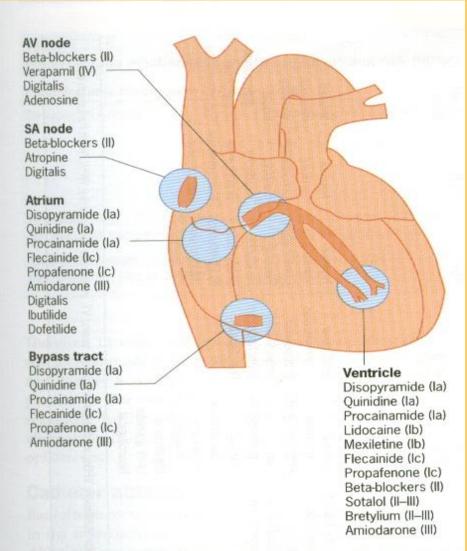


Fig. 13.46 Drugs that affect various parts of the heart. The Vaughan Williams' class is given in parentheses.

Atrial fibrillation

- Identify and treat the cause
 - IHD
 - Hypertension
 - Valvular heart disease
 - Heart failure
 - Pulmonary infection or embolism
 - metabolic abnormalities
 - toxins

Treatment of recent onset (within 48 hr) poorly tolerated(unstable) AF

- Direct current cardioversion
- Heparinization followed by warfarin (for at least 1 month)

Treatment of recent onset (within 48 h) well tolerated(stable) AF

 IV bolus of amiodorone 300mg followed by an infusion of 900mg over 24 hours.

if fails

cardioversion

Heparinization followed by warfarin (at least for 1 month)

Treatment of well tolerated(stable) AF of long or unknown duration

- Significant thromboembolic risk associated with cardioversion
- Heparinization followed by warfarin
- Atenolol 5mg by slow iv infusion followed by oral atenolol(5-10mg)
- If coexistent ventrycular dysfunction, digoxin(0.125-0.5mg) DOC.
- Cardioversion should be considered after I month's anticoagulation.

Atrial flutter

- If unstable cardiovert
- If stable Adenosine iv

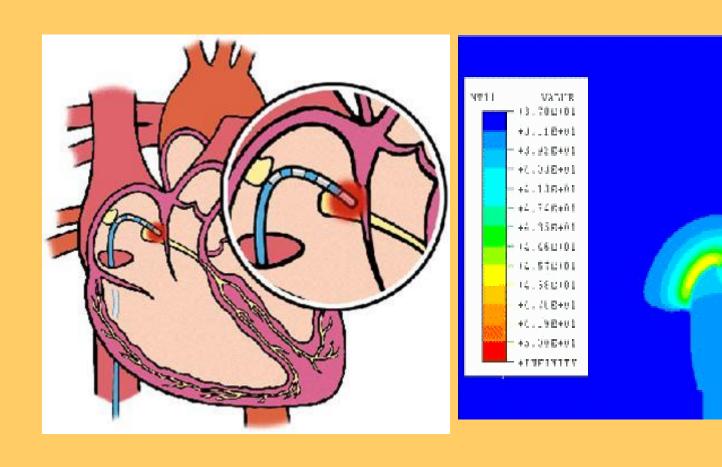
Treatment of chronic arrhythmias

- Drugs
- RF ablation
- Implanted devices

Atrial tachycardia

- If haemodynamically unstable cardiovert
- If haemodynamically stable
 Adenosine iv

Radiofrequency ablation (RF)



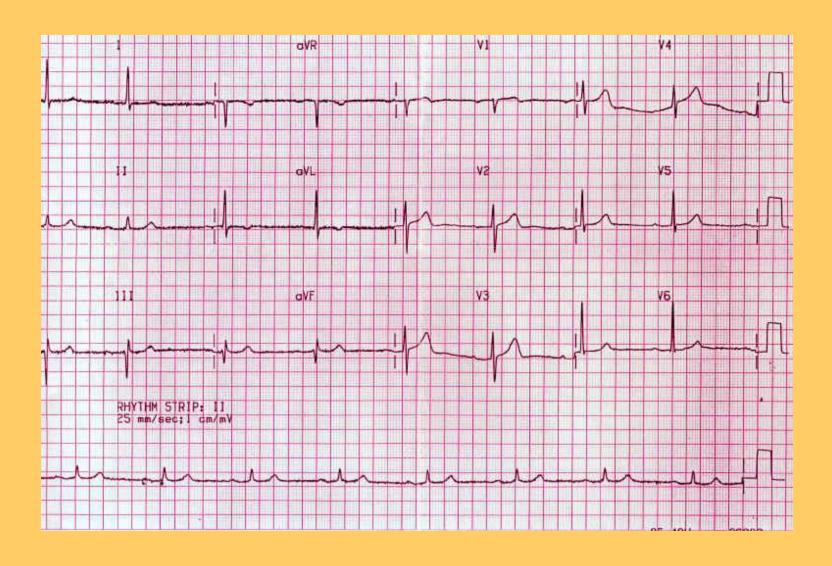
Bradyarrhythmias

1st degree heart block

2nd degree heart block

Complete heart block

1st degree heart block



2nd degree

Wenckebach or Mobitz I Mobitz II Second Degree Heart Block Type II

Complete HB



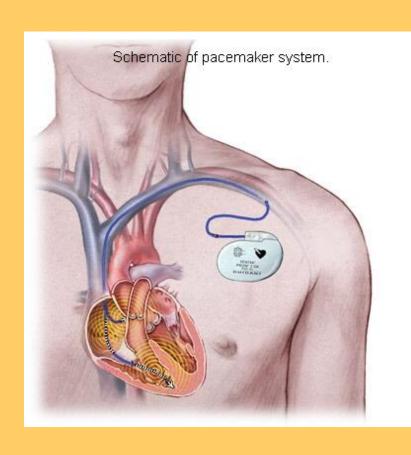
Complete heart block treatment

- Pacing Temporary
 - Permanent

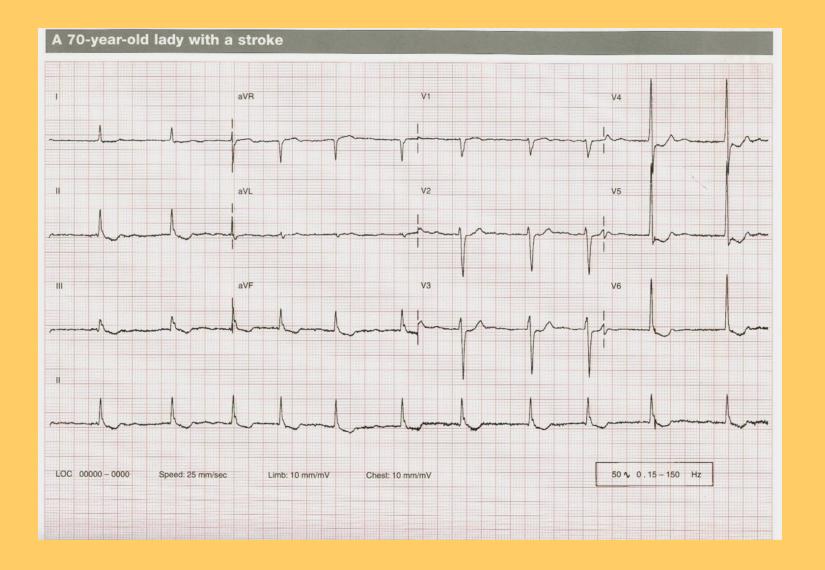
External pacemaker

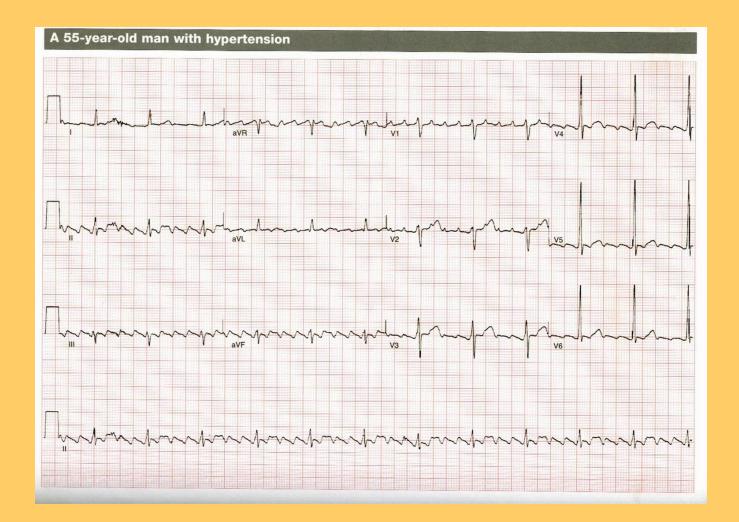


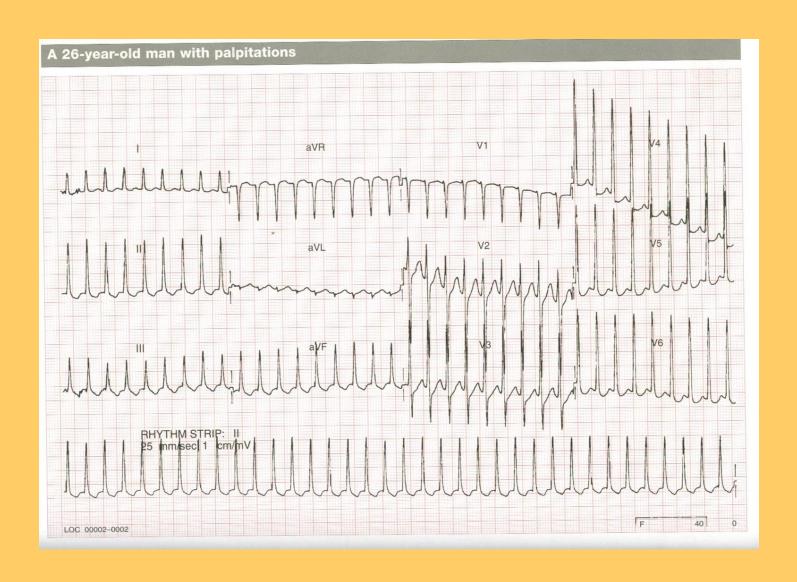
Permanent pacemaker

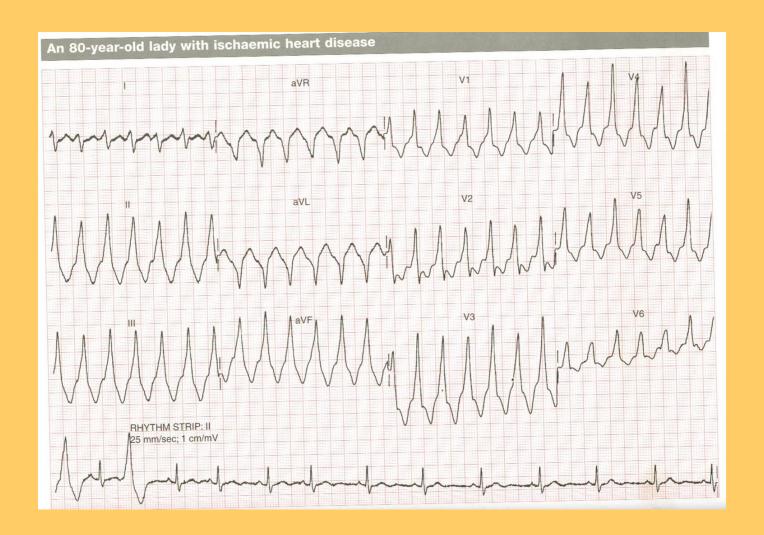












Summary

- Patients with good cardiac function often tolerate the arrhythmias.
- Patients with coexistent cardiac impairment may be severely compromised by an arrhythmia.
- Tachyarrythmias associated with major haemodynamic compromise usually require urgent cardioversion.
- Bradyarrhythmias associated with major haemodynamic compromise often require pacing.
- Patients with better tolerated arrhythmias can be treated with drug therapy.