DISORDERS OF HEART RATE, RHYTHM AND CONDUCTION

Dr. Mohammed Hilal Al-Ali ***L:1***

The heart beat is normally initiated by an electrical discharge from the sinoatrial ***(SA)*** node. The atria and ventricles then depolarise sequentially as electrical depolarisation passes through specialised conducting tissues. The sinus node acts as a pacemaker and is regulated by the autonomic nervous system; vagal activity slows the heart rate, and sympathetic activity accelerates it via cardiac sympathetic nerves and circulating catecholamines. Normal sinus rhythm is characterized by P waves that are upright in leads like in leads I, and II of the ECG, but inverted in the cavity leads AVR and V1.

CARDIAC ARRHYTHMIAS

A cardiac arrhythmia is a disturbance of the electrical rhythm of the heart. Arrhythmias may cause sudden death, syncope, heart failure, chest pain, dizziness, palpitations or no symptoms at all. Arrhythmias are often a manifestation of structural heart disease but may also occur in the context of an otherwise normal heart. There are two main types of arrhythmia:

- *Bradycardia:* the heart rate is slow (< 60 b.p.m.).
- *Tachycardia:* the heart rate is fast (> 100 b.p.m.).

Sinus arrhythmia:

Fluctuations of autonomic tone result in changes of the sinus rate with respiration. During inspiration, parasympathetic tone falls and the heart rate quickens, and on expiration the heart rate falls. This variation is normal, particularly in children and young adults.

Sinus bradycardia:

A sinus rate of less than 60 b.p.m. during the day or less than 50 b.p.m. at night is known as sinus bradycardia. It is usually asymptomatic unless the rate is very slow. It is normal in athletes due to increased vagal tone.

Sinus tachycardia:

Sinus rate acceleration to more than 100 b.p.m. is known as sinus tachycardia. It occur normally during exercise like running and swimming.

Mechanisms of arrhythmia production

There are three main mechanisms of tachycardia:

- ***Re-entry:*** the tachycardia is initiated by an ectopic beat and sustained by a re-entry circuit. Most tachyarrhythmias are due to re-entry (e.g. SVT and WPW).
- ***Increased automaticity:*** the tachycardia is produced by repeated spontaneous depolarisation of an ectopic focus, often in response to catecholamines (e.g. sinus tachycardia, multifocal atrial tachycardia and accelerated idioventricular rhythm).
- ***Triggered activity:*** This is a form of secondary depolarisation arising from an incompletely repolarised cell membrane (e.g. long QT syndrome and digitalis toxicity).

There are two mechanisms of bradycardia:

- ***Reduced automaticity:*** (e.g. sinus bradycardia)
- ***Blocked or abnormally slow conduction:*** (e.g. atrioventricular block).

HEART BLOCK

Heart block or conduction block may occur at any level in the conducting system. Block in either the AV node or the His bundle results in AV block, whereas block lower in the conduction system produces bundle branch block.

Atrioventricular block

There are three forms:

First-degree AV block:

In this condition AV conduction is delayed so the PR interval is prolonged (> 0.20 seconds). It rarely causes symptoms.

Second-degree AV block:

This occurs when some P waves conduct and others do not. There are several forms:

- *Mobitz I block (Wenckebach's phenomenon):* Is progressive PR interval prolongation until a P wave fails to conduct (dropped beat). The cycle then repeats itself. The phenomenon may be physiological and is sometimes observed at rest or during sleep in athletic young adults with high vagal tone.

- *Mobitz II block:* The PR interval of the conducted impulses remains constant but some P waves are not conducted. This is usually caused by disease of the His-Purkinje system and carries a risk of asystole.

- *2 : 1 or 3 : 1 (advanced) block:* Occurs when every second or third P wave conducts to the ventricles. This form of second-degree block is neither Mobitz I nor II.

Third-degree (complete) AV block:

When AV conduction fails completely, the atria and ventricles beat independently (AV dissociation). Ventricular activity is maintained by an escape rhythm arising in the AV node or bundle of His (narrow QRS complexes) or the distal Purkinje tissues (broad QRS complexes).

Aetiology of complete heart block:

(1) Congenital

(2) Acquired:

a) Idiopathic fibrosis

b) Myocardial infarction/ischaemia

c) Inflammation (e.g. aortic root abcess & sarcoidosis)

d) Trauma (e.g. cardiac surgery)

e) Drugs (e.g. digoxin, β-blocker)

Complete heart block produces a slow (25-50/min), regular pulse that, except in the case of congenital complete heart block, does not vary with exercise.

Management

AV block complicating acute myocardial infarction

Acute inferior myocardial infarction is often complicated by transient AV block because the right coronary artery supplies the AV junction. There is usually a reliable escape rhythm, and if the patient remains well, no treatment is required. Symptomatic second-degree or complete heart block may respond to atropine (0.6 mg i.v., repeated as necessary) or if this fails, a temporary pacemaker. In most cases the AV block will resolve within 7-10 days.

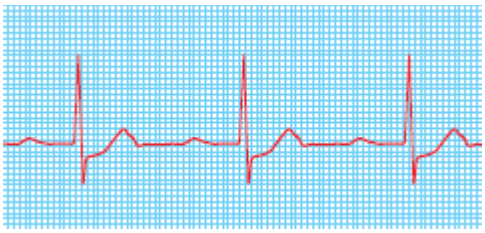
Second-degree or complete heart block complicating acute anterior myocardial infarction is usually a sign of extensive ventricular damage involving both bundle branches and carries a poor prognosis. Asystole may occur and a temporary pacemaker should be inserted as soon as possible until permanent pacemaker is implanted later on. Transcutaneous pacing can provide effective temporary rhythm support.

Chronic AV block

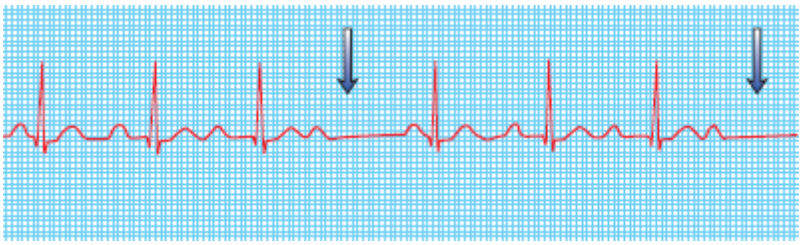
Patients with symptomatic bradyarrhythmias associated with AV block should receive a permanent pacemaker.

Asymptomatic first-degree or Mobitz type I second-degree AV block (Wenckebach's phenomenon) does not require treatment but may be an indication of serious underlying heart disease.

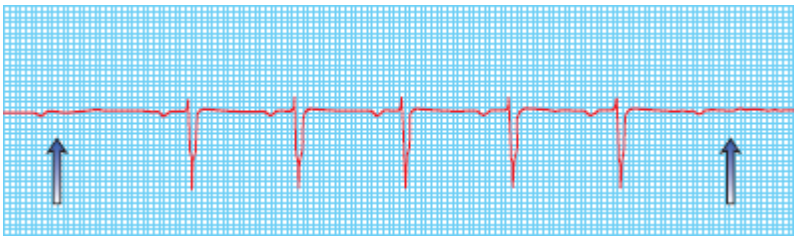
A *permanent pacemaker* is usually indicated in patients with asymptomatic Mobitz type II second-degree or complete heart block because there is evidence that pacing can improve their prognosis. An exception may be made in young asymptomatic patients with congenital complete heart block who have a mean daytime heart rate of more than 50 per minute.



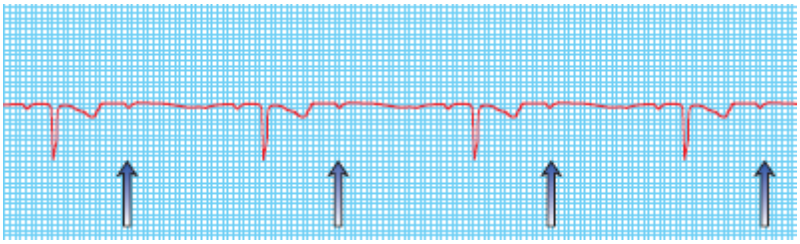
First-degree AV block.



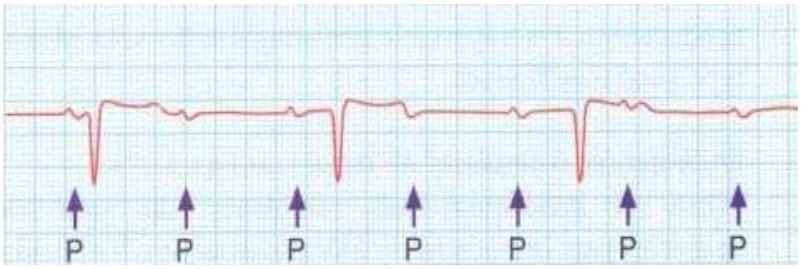
Second-degree AV block: Wenckebach (Mobitz type I) AV block.



Second-degree AV block: Mobitz type II AV block.



Second-degree AV block: 2 : 1 block



Third-degree (complete) AV block

Bundle branch block

The His bundle gives rise to the right and left bundle branches. The left bundle subdivides into the anterior and posterior divisions of the left bundle. Various conduction disturbances can occur.

Bundle branch conduction delay

This produces slight widening of the QRS complex (up to 0.11 s). It is known as ***incomplete bundle branch block***.

Complete block of a bundle branch

This is associated with a wider QRS complex (0.12 s or more). The shape of the QRS depends on whether the right or the left bundle is blocked.

Right bundle branch block: produces late activation of the right ventricle. This is seen as deep S waves in leads I and V6 and as a tall late R wave in lead V1 (rSR pattern).

Left bundle branch block: produces the opposite – a deep S wave in lead V1 and a tall late R wave in leads I and V6 (M-shaped QRS). Left bundle branch block also produces abnormal Q waves.

Hemiblock: Delay or block in the divisions of the left bundle branch produces a swing in the direction of depolarization (electrical axis) of the heart.

When the anterior division is blocked ***(left anterior hemiblock)***, This produces a superior and leftwards movement of the axis *(left axis deviation)*. Delay or block in the postero-inferior division ***(left posterior hemiblock)*** swings the QRS axis inferiorly to the right *(right axis deviation)*.

Bifascicular block: This is a combination of a block of any two of the following: the *right bundle branch*, the *left antero-superior division* and the *left postero-inferior division*. Block of the remaining fascicle will result in complete AV block.

Causes of right bundle branch block

It is also a normal finding in 1% of young adults and 5% of elderly adults

Congenital heart disease:

Atrial septal defect

Fallot's tetralogy

Pulmonary stenosis

Ventricular septal defect

Pulmonary disease:

Cor pulmonale

Recurrent pulmonary embolism

Acute pulmonary embolism (transient)

Myocardial disease:

Acute myocardial infarction

Cardiomyopathy

Conduction system fibrosis

Chagas' disease

Causes of left bundle branch block

Left ventricular outflow obstruction:

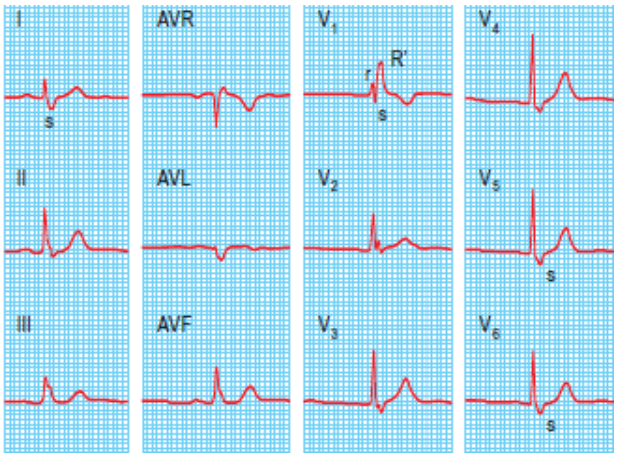
Aortic stenosis

Hypertension

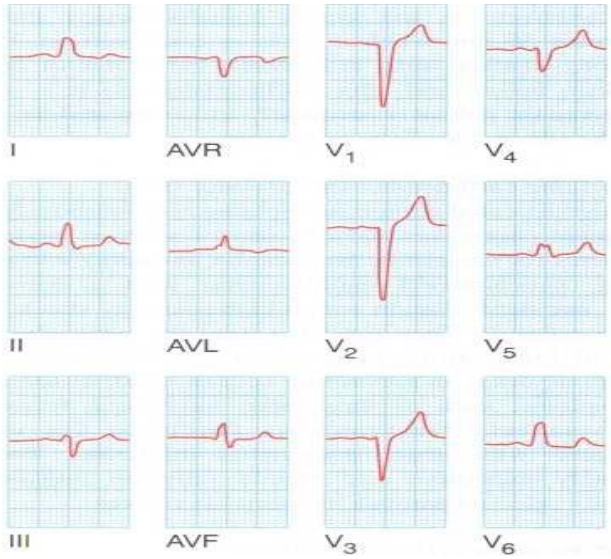
Coronary artery disease:

Acute myocardial infarction

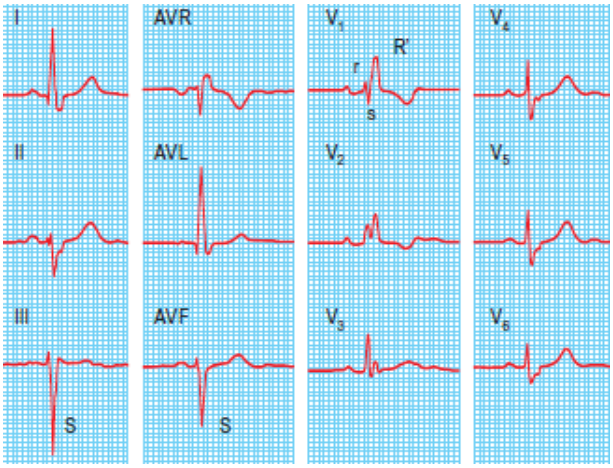
Severe coronary disease (two- to three-vessel disease)



Right Bundle Branch Block (RBBB)



Left Bundle Branch Block (LBBB)



Left anterior hemiblock (Bifascicular Block)