

Министерство здравоохранения республики Беларусь
Учреждение образования
«Гомельский государственный медицинский университет»

Кафедра патологической физиологии
Обсуждено на заседании кафедры
Протокол №7 от 30.08.2017

МЕТОДИЧЕСКАЯ РАЗРАБОТКА
Для проведения занятия со студентами
3 курса ФПСЗС, обучающихся на английском языке
по патологической физиологии

Тема: **Нарушения сердечного ритма**

Theme: **Cardiac arrhythmias**

Время 3 ак. часа

1.Actuality of the theme. The disorders of cardiac rhythm concern to complex manifestations of pathology of heart. Its can arise in rather small damage of the conducting system, and in some cases in structural changes. More often arrhythmia arise with infectious illnesses and intoxications as consequence of miocarditis or dystrophy processes in cardiac muscle, and also in heart ischemic disease, cardiosclerosis. The disorders of cardiac rhythm arise also owing to reflex influences from various interreceptors areas (disease of liver, intestinal tract, uterus), and also in hemodynamic disorders (arterial hypertension). Not infrequently arrhythmia is a result of disturbance of functions central and vegetative parts of nervous system. Arrhythmia can be result in development of cardiac insufficiency.

Learning goals of the lesson: to study etiology and pathogenesis of heart rhythm disturbances

Educational goals of the lesson: formation of scientific outlook and theoretical basis of future specialists on the basis of fundamental knowledge and the latest achievements of pathological physiology.

Objectives of the lesson:

1. To know causes, mechanisms of development and main manifestations of cardiac arrhythmias.
2. To know the principles of ECG registration.
3. To be able to determine the main types of arrhythmias from ECG data.
4. To know the disorders of general and coronary circulation in arrhythmias, mechanisms of development of heart failure in heart rhythm disturbances.

To repeat the following questions from related disciplines to ensure absolute mastery of the material:

1. Structure of the conducting system of heart (histology, cytology, embryology disciplines).
2. Mechanisms of occurrence and transfer impulse on conducting system of heart (normal physiology discipline).

Control questions of the lesson:

1. Heart rhythm disturbances (arrhythmias), types.
2. Disorders of cardiac automatism. Causes, mechanisms, electrophysiological mechanisms, ECG signs.
3. Disorders of cardiac conduction. Causes, mechanisms, electrophysiological mechanisms, ECG signs.
4. Disorders of cardiac tissue excitability and pulse excitation. Causes, mechanisms, electrophysiological mechanisms.
5. Disorders of general and coronary circulation in arrhythmias.
6. Fibrillation and defibrillation of heart. Artificial pacemakers.
7. Physico-chemical and metabolic disturbances in myocardium with arrhythmias.
8. Principles of arrhythmias therapy.

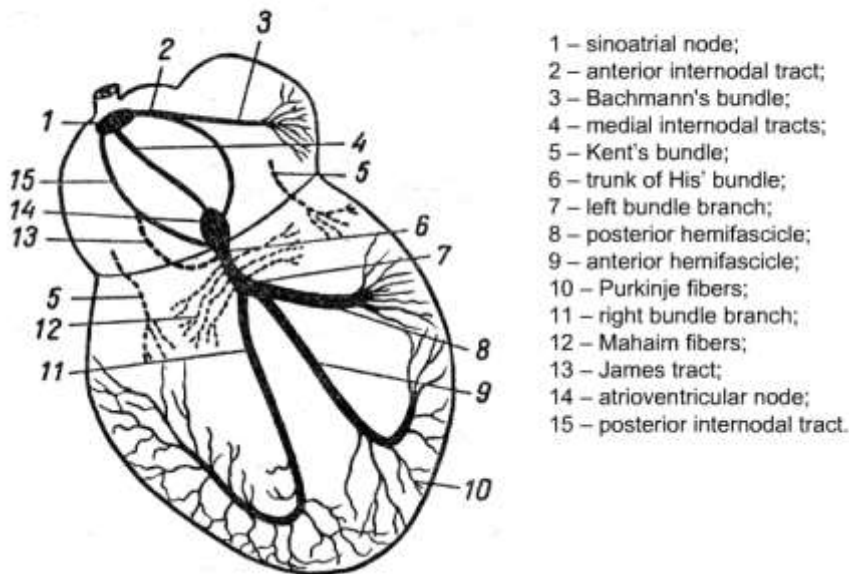
Calculation of study time

Total study time 3 ac.hours

№ п/п	Contents	Calculation of study time
1.	Introduction. Motivational characteristic of the theme	3 minutes
2.	Written control of students on the topic of the lesson	15 minutes
3.	Interviews with students about the topic of the lesson	60 minutes
4.	Self-managed student work	15 minutes
5.	Summing up the results of the lesson	5 minutes
6.	Decision of situational tasks	20 minutes
7.	Task for the next lesson	2 minutes

Additional material:

THE CONDUCTION SYSTEM OF THE HEART



The term arrhythmia refers to alterations in the heart rate or disturbances of regularity of the heart rhythm, implying changes in the cycle duration on ECG records or changes in duration and form of waves within a cycle.

The main classes of arrhythmia include:

- 1) disorders of impulse initiation
- 2) disorders of impulse conduction
- 3) simultaneous abnormalities of impulse generation and conduction

DISORDERS OF IMPULSE INITIATION

Disorders of impulse initiation include:

- a) changes in normal automaticity of the sinoatrial node
- b) abnormal automaticity
- c) triggered activity

Changes in normal automaticity of the sinoatrial node

Changes in the normal automaticity of the sinoatrial node are manifested in **sinus tachycardia, sinus bradycardia, and sinus arrhythmia.**

Sinus tachycardia.

Sinus tachycardia is a heart rate in excess of 100 beats per min.

It represents a physiologic response to a variety of stresses, such as fever, volume depletion, anxiety, exercise, thyrotoxicosis, hypoxemia, hypotension, or congestive heart failure.

Normal heart rates in children

- Newborn: 110 – 150 bpm
- 2 years: 85 – 125 bpm
- 4 years: 75 – 115 bpm
- 6 years+: 60 – 100 bpm

Causes

1. Non-pharmacological

- Exercise
- Pain, anxiety
- Hypoxia, hypercarbia
- Acidaemia
- Sepsis, pyrexia
- Pulmonary embolism

- Hyperthyroidism

2. Pharmacological

- Beta-agonists: adrenaline, isoprenaline, salbutamol, dobutamine
- Sympathomimetics: amphetamines, cocaine, methylphenidate
- Antimuscarinics: antihistamines, TCAs, carbamazepine, atropine
- Others: caffeine, theophylline, marijuana

Sinus bradycardia.

Sinus bradycardia is a heart rate that is less than 60 beats per min.

It results from activation of the vagal influences, or a decrease in sympathetic tone. Some substances can directly inhibit the function of the sinoatrial node, such as drugs (digitalis, quinine, opiates) or metabolites (unconjugated bilirubin, bile acids).

Sinus node dysfunction is commonly manifest as paroxysmal dizziness, presyncope, or syncope. These symptoms usually result from abrupt, prolonged sinus pauses caused by failure of sinus impulse formation (sinus arrest) or block of conduction of sinus impulses to the surrounding atrial tissue (sinus exit block).

Causes

1. Non-pharmacological

- Normal during sleep
- Increased vagal tone (e.g. athletes)
- Vagal stimulation (e.g. pain)
- Inferior myocardial infarction
- Sinus node disease
- Hypothyroidism
- Hypothermia
- Anorexia nervosa
- Electrolyte abnormalities – hyperkalaemia, hypermagnesaemia
- Brainstem herniation (the Cushing reflex)
- Myocarditis

2. Pharmacological

- Beta-blockers
- Calcium-channel blockers (verapamil & diltiazem)
- Digoxin
- Central alpha-2 agonists (clonidine & dexmedetomidine)
- Amiodarone
- Opiates
- GABA-ergic agents (barbiturates, benzodiazepines, baclofen, GHB)
- Organophosphate poisoning

Sinus arrhythmia.

Sinus arrhythmia is characterized by abnormal variations in the R-R interval seen in ECG recordings.

Sinus arrhythmia is usually synchronized with respiration. Usually heart rate increases during inspiration and slows down during expiration as a result of variations in vagal tone that affects the sinoatrial node.

Characteristics

- Variation in the P-P interval of more than 120 ms (3 small boxes).
- The P-P interval gradually lengthens and shortens in a cyclical fashion, usually corresponding to the phases of the respiratory cycle.
- Normal sinus P waves with a constant morphology (i.e. no evidence of premature atrial contractions).
- Constant P-R interval (i.e. no evidence of Mobitz I AV block).

Mechanism

- Sinus arrhythmia is a normal physiological phenomenon, most commonly seen in young, healthy

people.

- The heart rate varies due to reflex changes in vagal tone during the different stages of the respiratory cycle.
- Inspiration increases the heart rate by decreasing vagal tone.
- With the onset of expiration, vagal tone is restored, leading to a subsequent decrease in heart rate.
- The incidence of sinus arrhythmia decreases with age, presumably due to age-related decreases in carotid distensibility and baroreceptor reflex sensitivity.

NB. “Non-respiratory” sinus arrhythmia (not linked to the respiratory cycle) is less common, typically occurs in elderly patients and is more likely to be pathological (e.g. due to heart disease or digoxin toxicity).

Abnormal automaticity.

Abnormal automaticity can initiate an ectopic rhythm in nonpacemaker cells, such as cardiac muscle cells. Abnormal automaticity in the pacemaker cells produces accelerated rhythms. Conditions for abnormal automaticity are formed during partial depolarization and a decrease of the resting potential in the Purkinje fibers from -90-80 to -60-40 mV.

Triggered activity

Triggered activity is induced by early afterdepolarizations, which occur during phases 2 or 3 of the action potential, or delayed afterdepolarization, which occurs following completion of phase 3 of action potential.

Premature ventricular complex (PVC)

A premature beat arising from an ectopic focus within the ventricles.

Origin of Ectopic Beats

- Groups of pacemaker cells throughout the conducting system are capable of spontaneous depolarisation.
- The rate of depolarisation decreases from top to bottom: fastest at the sinoatrial node; slowest within the ventricles.
- Ectopic impulses from subsidiary pacemakers are normally suppressed by more rapid impulses from above.
- However, if an ectopic focus depolarises early enough — prior to the arrival of the next sinus impulse — it may “capture” the ventricles, producing a premature contraction.
- Premature contractions (“ectopics”) are classified by their origin — atrial (PACs), junctional (PJC)s or ventricular (PVCs).

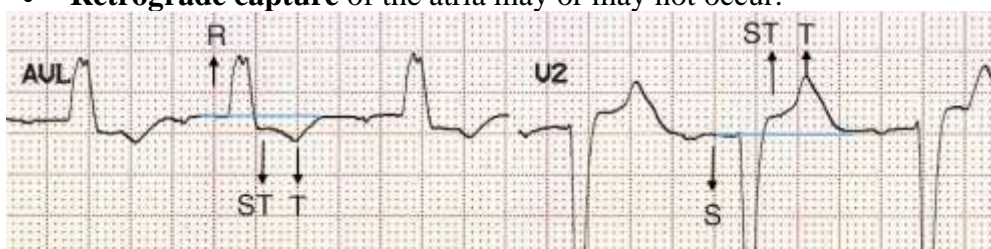
Ventricular Ectopics

- Ectopic firing of a focus within the ventricles bypasses the His-Purkinje system and depolarises the ventricles directly.
- This disrupts the normal sequence of cardiac activation, leading to asynchronous activation of the two ventricles.
- The consequent interventricular conduction delay produces QRS complexes with prolonged duration and abnormal morphology.

Electrocardiographic Features

PVCs have the following features:

- **Broad QRS complex** (≥ 120 ms) with abnormal morphology.
- **Premature** — i.e. occurs earlier than would be expected for the next sinus impulse.
- **Discordant** ST segment and T wave changes.
- Usually followed by a full **compensatory pause**.
- **Retrograde capture** of the atria may or may not occur.

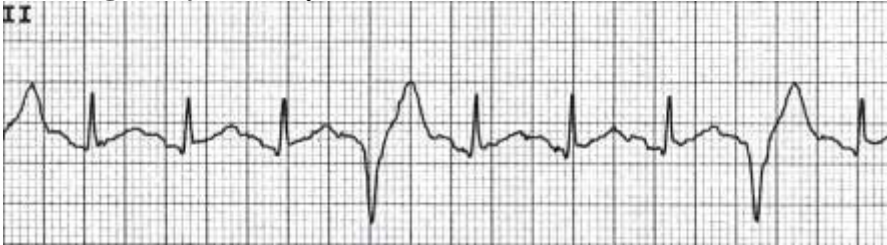


Bigeminy — every other beat is a PVC.



Trigeminy — every third beat is a PVC.

Quadrigeminy — every fourth beat is a PVC.



Couplet — two consecutive PVCs.

Triplet — three consecutive PVCs.

Causes

Frequent or symptomatic PVCs may be due to:

- Anxiety
- Sympathomimetics
- Beta-agonists
- Excess caffeine
- Hypokalaemia
- Hypomagnesaemia
- Digoxin toxicity
- Myocardial ischemia

Premature atrial complex (PAC)

- A premature beat arising from an ectopic focus within the atria.

Origin Of Ectopic Beats

- Groups of pacemaker cells throughout the conducting system are capable of spontaneous depolarisation.
- The rate of depolarisation decreases from top to bottom: fastest at the sinoatrial node; slowest within the ventricles.
- Ectopic impulses from subsidiary pacemakers are normally suppressed by more rapid impulses from above.
- However, if an ectopic focus depolarises early enough — before the arrival of the next sinus impulse — it may “capture” the ventricles, producing a premature contraction.
- Premature contractions (“ectopics”) are classified by their origin — atrial (PACs), junctional (PJC) or ventricular (PVCs).

Atrial Ectopics

- These arise from ectopic pacemaking tissue within the atria.
- There is an abnormal P wave, usually followed by a normal QRS complex.

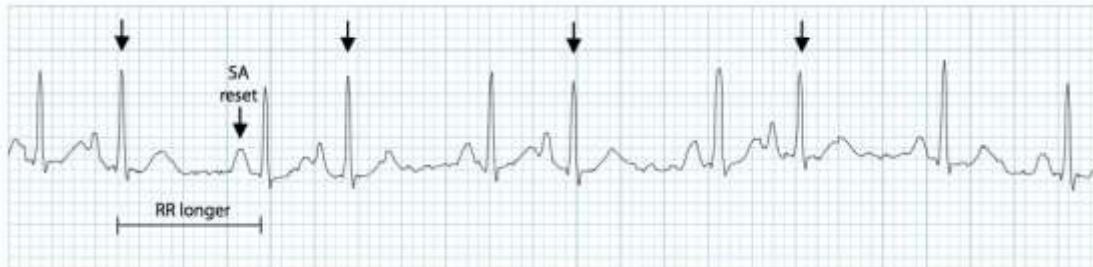
Electrocardiographic Features

PACs have the following features:

- An abnormal (non-sinus) P wave is followed by a QRS complex.
- The P wave typically has a different morphology and axis to the sinus P waves.
- The abnormal P wave may be hidden in the preceding T wave, producing a “peaked” or “camel hump” appearance — if this is not appreciated the PAC may be mistaken for a PJC.
- PACs arising close to the AV node (“low atrial” ectopics) activate the atria retrogradely, producing an inverted P wave with a relatively short PR interval ≥ 120 ms (PR interval < 120 ms is classified as a PJC).
- PACs that reach the SA node may depolarise it, causing the SA node to “reset” — this results in a

longer-than-normal interval before the next sinus beat arrives (“post-extrasystolic pause”). Unlike with PVCs, this pause is not equal to double the preceding RR interval (i.e. not a “full compensatory pause”).

- PACs arriving early in the cycle may be conducted aberrantly, usually with a RBBB morphology (as the right bundle branch has a longer refractory period than the left). They can be differentiated from PVCs by the presence of a preceding P wave.
- Similarly, PACs arriving very early in the cycle may not be conducted to the ventricles at all. In this case, you will see an abnormal P wave that is not followed by a QRS complex (“blocked PAC”). It is usually followed by a compensatory pause as the sinus node resets.



Causes

Frequent or symptomatic PACs may occur due to:

- Anxiety.
- Sympathomimetics.
- Beta-agonists.
- Excess caffeine.
- Hypokalaemia.
- Hypomagnesaemia.
- Digoxin toxicity.
- Myocardial ischaemia

Atrial flutter

- Atrial flutter is a type of supraventricular tachycardia caused by a macro-re-entry circuit in the right atrium.
- The re-entry circuit results in an atrial rate of 200-400 bpm (typically 300 bpm).
- Ventricular rate is determined by the AV conduction ratio — commonly referred to as the “degree of AV block” (*NB. This term is slightly misleading as the AV block is a physiological response to the rapid atrial rates; these patients usually have no evidence of AV block at normal heart rates*)
- The commonest AV conduction ratio is 2:1 — i.e. the ventricular rate is half the atrial rate (= 150 bpm).
- Higher-degree AV blocks can occur — usually due to medications or underlying heart disease — resulting in lower rates of ventricular conduction, e.g. 3:1 or 4:1 block.
- Atrial flutter with 1:1 conduction can occur due to sympathetic stimulation or in the presence of an accessory pathway (especially if AV-nodal blocking agents are administered to a patient with WPW).
- Atrial flutter with 1:1 conduction is associated with severe haemodynamic instability and progression to ventricular fibrillation.

Typical anticlockwise re-entry circuit within the right atrium

ECG Features

- Regular rhythm in presence of fixed AV block.
- Ventricular rate ~150 bpm in presence of 2:1 AV block.
- Flutter waves / ‘saw-tooth pattern’ best seen in leads II, III, aVF and V₁.
- Flutter wave morphology depending on type of atrial flutter (see above).
- QRS complexes usually < 120 ms unless pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction.
- Variable AV block will result in an irregular rhythm.
- Absence of an isoelectric baseline.



Atrial flutter with a 3:1 block

Clinical Significance

- Atrial flutter is closely related to atrial fibrillation in both causes and symptoms.
- Atrial flutter is usually paroxysmal but may be chronic (in which case it may lead to tachycardia-dependent cardiomyopathy).
- Symptoms of atrial flutter can include dizziness, palpitations, dyspnoea, and chest pain.
- Acute treatment of atrial flutter involves either rate or rhythm control (rhythm control is more likely to be successful).
- Long term cure requires ablation of the tricuspid isthmus to interrupt the re-entry circuit.

Atrial fibrillation

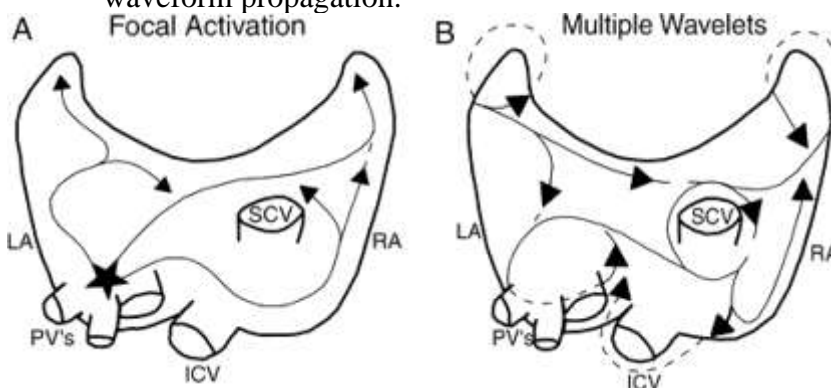
- Atrial Fibrillation (AF) is the most common sustained arrhythmia.
- The incidence and prevalence of AF is increasing.
- Lifetime risk over the age of 40 years is ~25%.
- Complications of AF include haemodynamic instability, cardiomyopathy, cardiac failure, and embolic events such as stroke.
- Characterised by disorganised atrial electrical activity and contraction.

Mechanism of Atrial Fibrillation

The mechanisms underlying AF are not fully understood but it requires an *initiating event* (focal atrial activity / PACs) and *substrate for maintenance* (i.e. dilated left atrium).

Some proposed mechanisms are:

- **Focal activation** – In which AF originates from an area of focal activity. This activity may be triggered, due to increased automaticity, or from micro re-entry. Often located in the pulmonary veins.
- **Multiple wavelet mechanism** – In which multiple small wandering wavelets are formed. The fibrillation is maintained by re-entry circuits formed by some of the wavelets. This process is potentiated in the presence of a dilated LA — the larger surface area facilitates continuous waveform propagation.



Causes of Atrial Fibrillation

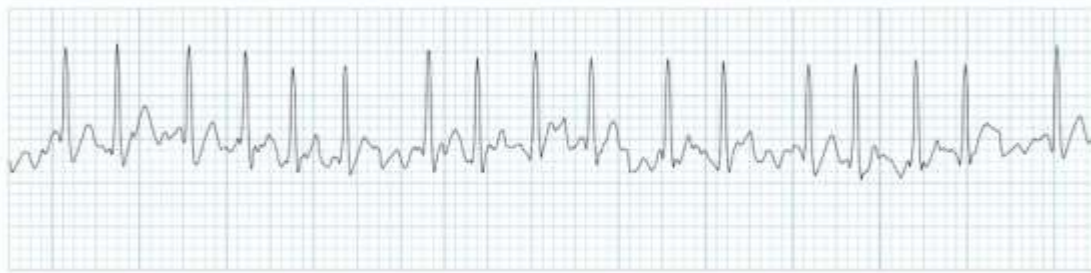
There are multiple causes of AF, including:

- Ischaemic heart disease
- Hypertension
- Valvular heart disease (esp. mitral stenosis / regurgitation)
- Acute infections
- Electrolyte disturbance (hypokalaemia, hypomagnesaemia)
- Thyrotoxicosis
- Drugs (e.g. sympathomimetics)

- Pulmonary embolus
- Pericardial disease
- Acid-base disturbance
- Pre-excitation syndromes
- Cardiomyopathies: dilated, hypertrophic.
- Pheochromocytoma

ECG Features of Atrial Fibrillation

- Irregularly irregular rhythm.
- No P waves.
- Absence of an isoelectric baseline.
- Variable ventricular rate.
- QRS complexes usually < 120 ms unless pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction.
- Fibrillatory waves may be present and can be either fine (amplitude < 0.5mm) or coarse (amplitude >0.5mm).
- Fibrillatory waves may mimic P waves leading to misdiagnosis.



Ventricular tachycardia

- Broad complex tachycardia originating in the ventricles.
- There are several different varieties of VT — the most common is **Monomorphic VT**.



Monomorphic ventricular tachycardia

Clinical Significance

- Ventricular tachycardia may impair cardiac output with consequent hypotension, collapse, and acute cardiac failure. This is due to extreme heart rates and lack of coordinated atrial contraction (loss of “atrial kick”).
- The presence of pre-existing poor ventricular function is strongly associated with cardiovascular compromise.
- Decreased cardiac output may result in decreased myocardial perfusion with degeneration to VF.
- Prompt recognition and initiation of treatment (e.g. electrical cardioversion) is required in all cases of VT.

Sustained = Duration > 30 seconds or requiring intervention due to hemodynamic compromise.

Non-sustained = Three or more consecutive ventricular complexes terminating spontaneously in < 30 seconds.

Mechanisms of Ventricular Tachycardia

Three mechanisms exist for initiation and propagation of ventricular tachycardia:

1. Reentry

- Commonest mechanism.
- Requires two distinct conduction pathways with a conduction block in one pathway, and a region

of slow conduction in the other.

- Develops due to abnormal myocardial scarring usually due to prior ischemia or infarction.

2. Triggered Activity

- Occurs due to early or late after-depolarisations.
- Examples include Torsades and digitalis toxicity.

3. Abnormal Automaticity

- Accelerated abnormal impulse generation by a region of ventricular cells.

Electrocardiographic Features of Ventricular Tachycardia

Ventricular tachycardia can be difficult to differentiate from other causes of broad complex tachycardia. The following characteristics aid in the identification of VT.

- Very broad complexes (>160ms).
- Absence of typical RBBB or LBBB morphology.
- Extreme axis deviation (“northwest axis”) — QRS is positive in aVR and negative in I + aVF.
- AV dissociation (P and QRS complexes at different rates).
- Capture beats — occur when the sinoatrial node transiently ‘captures’ the ventricles, in the midst of AV dissociation, to produce a QRS complex of normal duration.
- Fusion beats — occur when a sinus and ventricular beat coincide to produce a hybrid complex of intermediate morphology.
- Positive or negative concordance throughout the chest leads, i.e. leads V1-6 show entirely positive (R) or entirely negative (QS) complexes, with no RS complexes seen.
- Brugada’s sign – The distance from the onset of the QRS complex to the nadir of the S-wave is > 100ms.
- Josephson’s sign – Notching near the nadir of the S-wave.
- RSR’ complexes with a taller “left rabbit ear”. This is the most specific finding in favour of VT. This is in contrast to RBBB, where the right rabbit ear is taller.

Ventricular flutter

- Extreme form of VT with loss of organised electrical activity
- Associated with rapid and profound hemodynamic compromise
- Usually short lived due to progression to ventricular fibrillation
- As with ventricular fibrillation rapid initiation of advanced life support is required
- Continuous Sine Wave
- No identifiable P waves, QRS complexes, or T waves
- Rate usually > 200 beats / min

Ventricular fibrillation

- Ventricular fibrillation (VF) is the the most important shockable cardiac arrest rhythm.
- The ventricles suddenly attempt to contract at rates of up to 500 bpm.
- This rapid and irregular electrical activity renders the ventricles unable to contract in a synchronised manner, resulting in immediate loss of cardiac output.
- The heart is no longer an effective pump and is reduced to a quivering mess.
- Unless advanced life support is rapidly instituted, this rhythm is invariably fatal.
- Prolonged ventricular fibrillation results in decreasing waveform amplitude, from initial coarse VF to fine VF and ultimately degenerating into asystole due to progressive depletion of myocardial energy stores.

ECG Findings

- Chaotic irregular deflections of varying amplitude
- No identifiable P waves, QRS complexes, or T waves
- Rate 150 to 500 per minute
- Amplitude decreases with duration (coarse VF -> fine VF)

Mechanism

In the presence of ischemic heart disease VF may be preceded by:

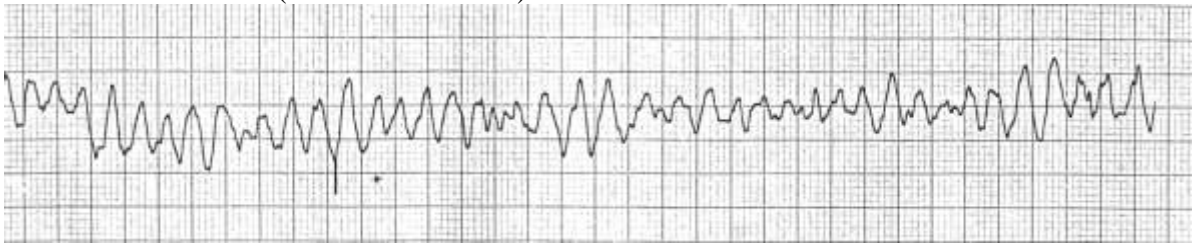
- Premature ventricular contractions (PVCs)
- ST changes

- R on T phenomenon
- Pauses
- QT prolongation
- Ventricular tachycardia
- Supraventricular arrhythmias
- Sinus tachycardia

The underlying mechanism of Ventricular Fibrillation is not fully understood, several mechanisms have been hypothesised:

Causes

- Myocardial ischemia / infarction
- Electrolyte abnormalities
- Cardiomyopathy (dilated, hypertrophic, restrictive)
- Long QT (acquired / congenital) causing TdP → VF
- Brugada syndrome
- Drugs (e.g. verapamil in patients with AF+WPW)
- Environmental – electrical shocks, drowning, hypothermia
- Pulmonary embolism
- Cardiac tamponade
- Blunt trauma (Comotio Cordis)



- Typical rhythm strip of ventricular fibrillation

CONDUCTION DISTURBANCES

Types of conduction disorders include:

1. slow conduction or block of conduction leading to ectopic "escape" rhythms.
2. unidirectional block and reentry.
3. accelerated conduction.

Slow conduction or block of conduction.

Manifestations and consequences of the conduction disturbances depend on the site and extent of the impulse blockade.

1. Sinoatrial nodal block (sick sinus syndrome).

First degree sinoatrial exit block denotes a prolonged conduction time from the sinus node to the surrounding atrial tissue. Second-degree sinoatrial exit block denotes the intermittent failure of conduction of sinus impulses to the surrounding atrial tissue. Third-degree, or complete, sinoatrial block is characterized by a lack of atrial activity or by the presence of an ectopic subsidiary atrial pacemaker.

Causes

1. *Intrinsic*

- Idiopathic Degenerative Fibrosis (commonest).
- Ischaemia.
- Cardiomyopathies.
- Infiltrative Diseases e.g. sarcoidosis, haemochromatosis.
- Congenital abnormalities.

2. *Extrinsic Causes*

- Drugs e.g. digoxin, beta-blockers, calcium channel blockers.
- Autonomic dysfunction.
- Hypothyroidism.

- Electrolyte abnormalities — e.g. hyperkalaemia.

ECG in Sinus Node Dysfunction

ECG abnormalities can be variable and intermittent. Multiple ECG abnormalities can be seen in sinus node dysfunction including:

- Sinus Bradycardia.
- Sinus Arrhythmia – associated with sinus node dysfunction in the elderly in the absence of respiratory pattern association.
- Sinoatrial Exit Block.
- Sinus Arrest — pause > 3 seconds.
- Atrial fibrillation with slow ventricular response.
- Bradycardia – tachycardia syndrome.

Bradycardia – tachycardia syndrome

- Alternating bradycardia with paroxysmal tachycardia, often supraventricular in origin.
- On cessation of tachyarrhythmia may be a period of delayed sinus recovery e.g. sinus pause or exit block.
- If significant this period of delayed recovery may result in syncope.

Clinical Manifestations

- Commonly seen in the elderly but sinus node dysfunction can affect all age groups.
- Symptoms are due to decreased cardiac output and end-organ hypoperfusion associated with cardiac rhythm abnormality.
- Wide range of clinical symptoms including syncope, near-syncope, dizziness, fatigue and palpitations.

2. AV conduction disturbances.

First-degree AV block is characterized by a PR interval > 0,2s. (Marked' first degree block if PR interval > 0,3s)

Causes

- Increased vagal tone
- Athletic training
- Inferior MI
- Mitral valve surgery
- Myocarditis (e.g. Lyme disease)
- Hypokalaemia
- AV nodal blocking drugs (beta-blockers, calcium channel blockers, digoxin, amiodarone)
- May be a normal variant

Clinical significance

- Does not cause haemodynamic disturbance
- No specific treatment is required

Second-degree AV block (intermittent AV block) is present when some atrial impulses fail to conduct to the ventricles.

Mobitz type I second-degree AV block (AV Wenckebach block) is characterized by progressive PR interval prolongation prior to block of an atrial impulse.

- Progressive prolongation of the PR interval culminating in a non-conducted P wave
- The PR interval is longest immediately before the dropped beat
- The PR interval is shortest immediately after the dropped beat

Other Features

- The greatest increase in PR interval duration is typically between the first and second beats of the cycle.
- The RR interval progressively shortens with each beat of the cycle.
- The Wenckebach pattern tends to repeat in P:QRS groups with ratios of 3:2, 4:3 or 5:4.

Mechanism

- Mobitz I is usually due to reversible conduction block at the level of the AV node.
- Malfunctioning AV node cells tend to progressively fatigue until they fail to conduct an impulse. This is different to cells of the His-Purkinje system which tend to fail suddenly and unexpectedly (i.e. producing a Mobitz II block).

Causes

- Drugs: beta-blockers, calcium channel blockers, digoxin, amiodarone
- Increased vagal tone (e.g. athletes)
- Inferior MI
- Myocarditis
- Following cardiac surgery (mitral valve repair, Tetralogy of Fallot repair)

Clinical significance

- Mobitz I is usually a benign rhythm, causing minimal haemodynamic disturbance and with low risk of progression to third degree heart block.
- Asymptomatic patients do not require treatment.
- Symptomatic patients usually respond to atropine.
- Permanent pacing is rarely required.

In **Mobitz type II second-degree AV block**, conduction fails suddenly and unexpectedly without a preceding change in PR intervals.

- Intermittent non-conducted P waves *without* progressive prolongation of the PR interval (compare this to Mobitz I).
- The PR interval in the conducted beats remains constant.
- The P waves ‘march through’ at a constant rate.
- The RR interval surrounding the dropped beat(s) is an exact multiple of the preceding RR interval (e.g. double the preceding RR interval for a single dropped beat, treble for two dropped beats, etc).

Mechanism

- Mobitz II is usually due to failure of conduction at the level of the His-Purkinje system (i.e. below the AV node).
- While Mobitz I is usually due to a functional suppression of AV conduction (e.g. due to drugs, reversible ischaemia), Mobitz II is more likely to be due to *structural* damage to the conducting system (e.g. infarction, fibrosis, necrosis).
- Patients typically have a pre-existing LBBB or bifascicular block, and the 2nd degree AV block is produced by intermittent failure of the remaining fascicle (“bilateral bundle-branch block”).
- In around 75% of cases, the conduction block is located *distal to the Bundle of His*, producing *broad QRS complexes*.
- In the remaining 25% of cases, the conduction block is located within the His Bundle itself, producing narrow QRS complexes.
- Unlike Mobitz I, which is produced by progressive fatigue of the AV nodal cells, Mobitz II is an “all or nothing” phenomenon whereby the His-Purkinje cells suddenly and unexpectedly fail to conduct a supraventricular impulse.
- There may be no pattern to the conduction blockade, or alternatively there may be a fixed relationship between the P waves and QRS complexes, e.g. 2:1 block, 3:1 block.

Causes of Mobitz II

- Anterior MI (due to septal infarction with necrosis of the bundle branches).
- Idiopathic fibrosis of the conducting system (Lenegre’s or Lev’s disease).
- Cardiac surgery (especially surgery occurring close to the septum, e.g. mitral valve repair)
- Inflammatory conditions (rheumatic fever, myocarditis, Lyme disease).
- Autoimmune (SLE, systemic sclerosis).
- Infiltrative myocardial disease (amyloidosis, haemochromatosis, sarcoidosis).
- Hyperkalaemia.
- Drugs: beta-blockers, calcium channel blockers, digoxin, amiodarone.

Clinical Significance

- Mobitz II is much more likely than Mobitz I to be associated with haemodynamic compromise, severe bradycardia and progression to 3rd degree heart block.

- Onset of haemodynamic instability may be sudden and unexpected, causing syncope (Stokes-Adams attacks) or sudden cardiac death.
- The risk of asystole is around 35% per year.
- Mobitz II mandates immediate admission for cardiac monitoring, backup temporary pacing and ultimately insertion of a permanent pacemaker.

AV block: 2nd degree, “fixed ratio” blocks

- Second degree heart block with a fixed ratio of P waves: QRS complexes (e.g. 2:1, 3:1, 4:1).
- Fixed ratio blocks can be the result of either Mobitz I or Mobitz II conduction.

Third-degree AV block is present when no atrial impulse propagates to the ventricles; atria and ventricles follow their own rhythms.

Third-degree AV block may be associated with prolonged ventricular standstill until a ventricular focus begins firing. The cardiac arrest, if prolonged, can result in cerebral ischemia, syncope, or death - Stokes-Adams syndrome.

- In complete heart block, there is complete absence of AV conduction – *none* of the supraventricular impulses are conducted to the ventricles.
- Perfusing rhythm is maintained by a junctional or ventricular escape rhythm. Alternatively, the patient may suffer ventricular standstill leading to syncope (if self-terminating) or sudden cardiac death (if prolonged).
- Typically the patient will have severe bradycardia with independent atrial and ventricular rates, i.e. AV dissociation.

Mechanism

- Complete heart block is essentially the end point of either Mobitz I or Mobitz II AV block.
- It may be due to progressive fatigue of AV nodal cells as per Mobitz I (e.g. secondary to increased vagal tone in the acute phase of an inferior MI).
- Alternatively, it may be due to sudden onset of complete conduction failure throughout the His-Purkinje system, as per Mobitz II (e.g. secondary to septal infarction in acute anterior MI).
- The former is more likely to respond to atropine and has a better overall prognosis.

Causes of complete heart block

The causes are the same as for Mobitz I and Mobitz II second degree heart block. The most important aetiologies are:

- Inferior myocardial infarction
- AV-nodal blocking drugs (e.g. calcium-channel blockers, beta-blockers, digoxin)
- Idiopathic degeneration of the conducting system (Lenegre’s or Lev’s disease)

Clinical significance

- Patients with third degree heart block are at high risk of ventricular standstill and sudden cardiac death.
- They require urgent admission for cardiac monitoring, backup temporary pacing and usually insertion of a permanent pacemaker.

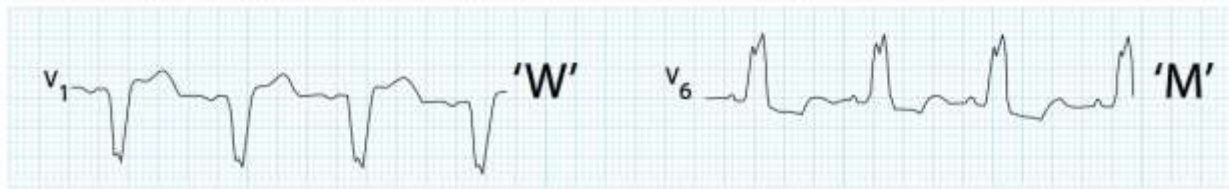
3. Bundle branch block.

Blockade of the right or left common bundles or the left anterior or posterior fascicles results in an abnormal sequence of ventricular excitation.

Left bundle branch block

- Normally the septum is activated from left to right, producing small Q waves in the lateral leads.
- In LBBB, the normal direction of septal depolarisation is reversed (becomes right to left), as the impulse spreads first to the RV via the right bundle branch and then to the LV via the septum.
- This sequence of activation extends the QRS duration to > 120 ms and eliminates the normal septal Q waves in the lateral leads.
- The overall direction of depolarisation (from right to left) produces tall R waves in the lateral leads (I, V5-6) and deep S waves in the right precordial leads (V1-3), and usually leads to left axis deviation.
- As the ventricles are activated sequentially (right, then left) rather than simultaneously, this

produces a broad or notched ('M'-shaped) R wave in the lateral leads.



Dominant S wave in V1 with broad, notched ('M'-shaped) R wave in V6

Diagnostic Criteria

- QRS duration of 120 ms
- Dominant S wave in V1
- Broad monophasic R wave in lateral leads (I, aVL, V5-V6)
- Absence of Q waves in lateral leads (I, V5-V6; small Q waves are still allowed in aVL)
- Prolonged R wave peak time > 60ms in left precordial leads (V5-6)

Associated Features

- *Appropriate discordance*: the ST segments and T waves always go in the opposite direction to the main vector of the QRS complex
- Poor R wave progression in the chest leads
- Left axis deviation

Causes

- Aortic stenosis
- Ischaemic heart disease
- Hypertension
- Dilated cardiomyopathy
- Anterior MI
- Primary degenerative disease (fibrosis) of the conducting system (Lenegre disease)
- Hyperkalaemia
- Digoxin toxicity

NB. It is unusual for left bundle branch block to exist in the absence of organic disease.

Incomplete LBBB is diagnosed when typical LBBB morphology is associated with a QRS duration < 120ms.

Left anterior fascicular block

- In left anterior fascicular block (aka *left anterior hemiblock*), impulses are conducted to the left ventricle via the left posterior fascicle, which inserts into the infero-septal wall of the left ventricle along its endocardial surface.
- On reaching the left ventricle, the initial electrical vector is therefore directed downwards and rightwards (as excitation spreads outwards from endocardium to epicardium), producing small R waves in the inferior leads (II, III, aVF) and small Q waves in the left-sided leads (I, aVL).
- The major wave of depolarisation then spreads in an upwards and leftwards direction, producing large positive voltages (tall R waves) in the left-sided leads and large negative voltages (deep S waves) in the inferior leads.
- This process takes about 20 milliseconds longer than simultaneous conduction via both fascicles, resulting in a slight widening of the QRS.
- The impulse reaches the left-sided leads later than normal, resulting in a increased R wave peak time (the time from onset of the QRS to the peak of the R wave) in aVL.

Diagnostic Criteria for LAFB

- Left axis deviation (usually between -45 and -90 degrees)
- Small Q waves with tall R waves (= 'qR complexes') in leads I and aVL
- Small R waves with deep S waves (= 'rS complexes') in leads II, III, aVF
- QRS duration normal or slightly prolonged (80-110 ms)
- Prolonged R wave peak time in aVL > 45 ms
- Increased QRS voltage in the limb leads

Left posterior fascicular block

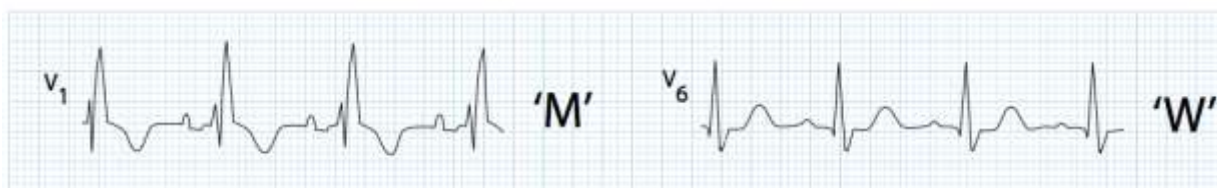
- In left posterior fascicular block (aka *left posterior hemiblock*), impulses are conducted to the left ventricle via the left anterior fascicle, which inserts into the upper, lateral wall of the left ventricle along its endocardial surface.
- On reaching the ventricle, the initial electrical vector is therefore directed upwards and leftwards (as excitation spreads outwards from endocardium to epicardium), causing small R waves in the lateral leads (I and aVL) and small Q waves in the inferior leads (II, III and aVF).
- The major wave of depolarisation then spreads along the free LV wall in a downward and rightward direction, producing large positive voltages (tall R waves) in the inferior leads and large negative voltages (deep S waves) in the lateral leads.
- This process takes up to 20 milliseconds longer than simultaneous conduction via both fascicles, resulting in a slight widening of the QRS.
- The impulse reaches the inferior leads later than normal, resulting in an increased R wave peak time (= the time from onset of the QRS to the peak of the R wave) in aVF.

Diagnostic Criteria for LPFB

- Right axis deviation ($> +90$ degrees)
- Small R waves with deep S waves (= 'rS complexes') in leads I and aVL
- Small Q waves with tall R waves (= 'qR complexes') in leads II, III and aVF
- QRS duration normal or slightly prolonged (80-110ms)
- Prolonged R wave peak time in aVF
- Increased QRS voltage in the limb leads
- No evidence of right ventricular hypertrophy
- No evidence of any other cause for right axis deviation

Right bundle branch block

- In RBBB, activation of the right ventricle is delayed as depolarisation has to spread across the septum from the left ventricle.
- The left ventricle is activated normally, meaning that the early part of the QRS complex is unchanged.
- The delayed right ventricular activation produces a secondary R wave (R') in the right precordial leads (V1-3) and a wide, slurred S wave in the lateral leads.
- Delayed activation of the right ventricle also gives rise to secondary repolarization abnormalities, with ST depression and T wave inversion in the right precordial leads.
- In isolated RBBB the cardiac axis is unchanged, as left ventricular activation proceeds normally via the left bundle branch.



Tall R' wave in V1 ("M" pattern) with wide, slurred S wave in V6 ("W" pattern)

ECG changes in RBBB

Diagnostic Criteria

- Broad QRS > 120 ms
- RSR' pattern in V1-3 ('M-shaped' QRS complex)
- Wide, slurred S wave in the lateral leads (I, aVL, V5-6)

Causes of RBBB

- Right ventricular hypertrophy / cor pulmonale
- Pulmonary embolus
- Ischaemic heart disease
- Rheumatic heart disease
- Myocarditis or cardiomyopathy
- Degenerative disease of the conduction system

- Congenital heart disease (e.g. atrial septal defect)

Incomplete RBBB

- Incomplete RBBB is defined as an RSR' pattern in V1-3 with QRS duration < 120ms.
- It is a normal variant, commonly seen in children (of no clinical significance).

Unidirectional block and reentry.

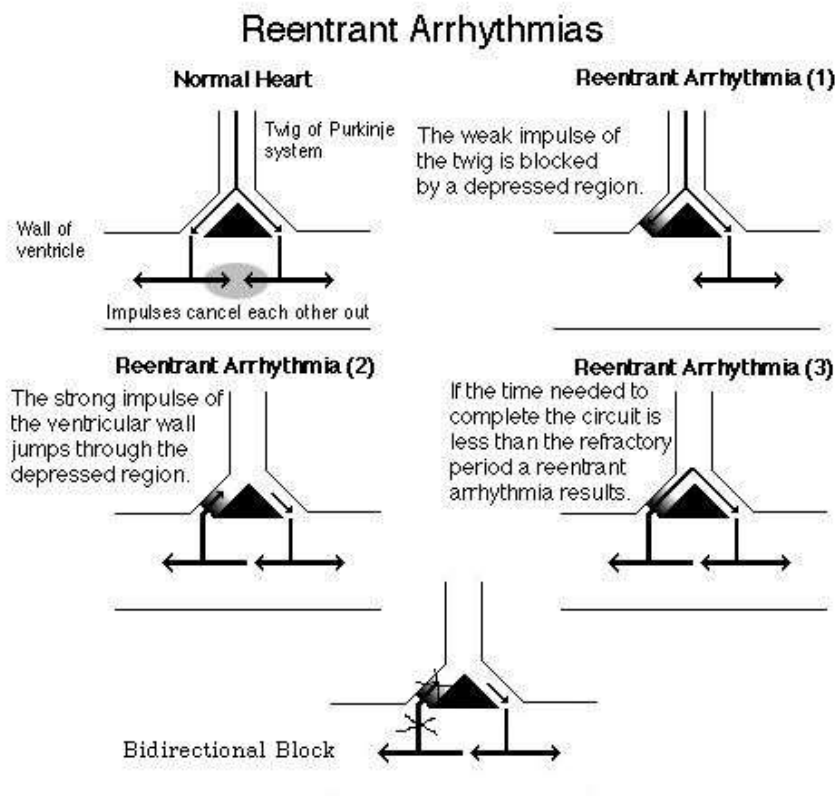
Reentry is a phenomenon of repetitive circulation of the impulse over a closed loop. Repetitive circulation of the impulse over this loop can produce a sustained tachyarrhythmia.

The requirements for initiating reentry include:

1. electrophysiologic inhomogeneity;
2. unidirectional block in one pathway;
3. slow conduction over an alternative pathway, allowing time for the initially blocked pathway to recover excitability; and
4. reexcitation of the initially blocked pathway to complete a loop of activation.

Two types of reentry arrhythmias are described:

1. "Ordered" reentry, or macroreentry (circulation of impulse over a closed loop of large diameter; sometimes this loop is formed by natural conduction pathways, or anatomic obstacle);
2. Microreentry (circulation of impulse over random, short pathways). This mechanism underlies atrial or ventricular fibrillation.



Accelerated conduction.

Wolf-Parkinson-White syndrome.

It results from an aberrant conduction pathway between atria and the ventricles. The aberrant pathway eliminates or reduces the normal delay between atrial and ventricular activation. The ECG shows a shortened P-R interval, usually less than 0.1 sec, and a widened QRS complex with a slurred initial upstroke (delta wave).

ECG features of WPW in sinus rhythm are:

- PR interval < 120ms
- Delta wave – slurring/slow rise of initial portion of the QRS
- QRS prolongation > 110ms
- ST Segment and T wave discordant changes – i.e. in the opposite direction to the major component of the QRS complex

- Pseudo-infarction pattern can be seen in up to 70% of patients – due to negatively deflected delta waves in the inferior / anterior leads (“pseudo-Q waves”), or as a prominent R wave in V1-3 (mimicking posterior infarction).

WPW may be described as type A or B. Type A has a positive delta wave in all precordial leads with $R/S > 1$ in V₁. Type B has a negative delta wave in leads V₁ and V₂

SIMULTANEOUS ABNORMALITIES OF IMPULSE GENERATION AND CONDUCTION

This mechanism is exemplified by parasystole - an ectopically originating rhythm.

ELECTROCARDIOGRAPHY

Skin Preparation:

Clean with an alcohol wipe if necessary. If the patients are very hairy – shave the electrode areas.

ECG standard leads

There are three of these leads, I, II and III.

Lead I: is between the right arm and left arm electrodes, the left arm being positive.

Lead II: is between the right arm and left leg electrodes, the left leg being positive.

Lead III: is between the left arm and left leg electrodes, the left leg again being positive.

Chest Electrode Placement

V1: Fourth intercostal space to the right of the sternum.

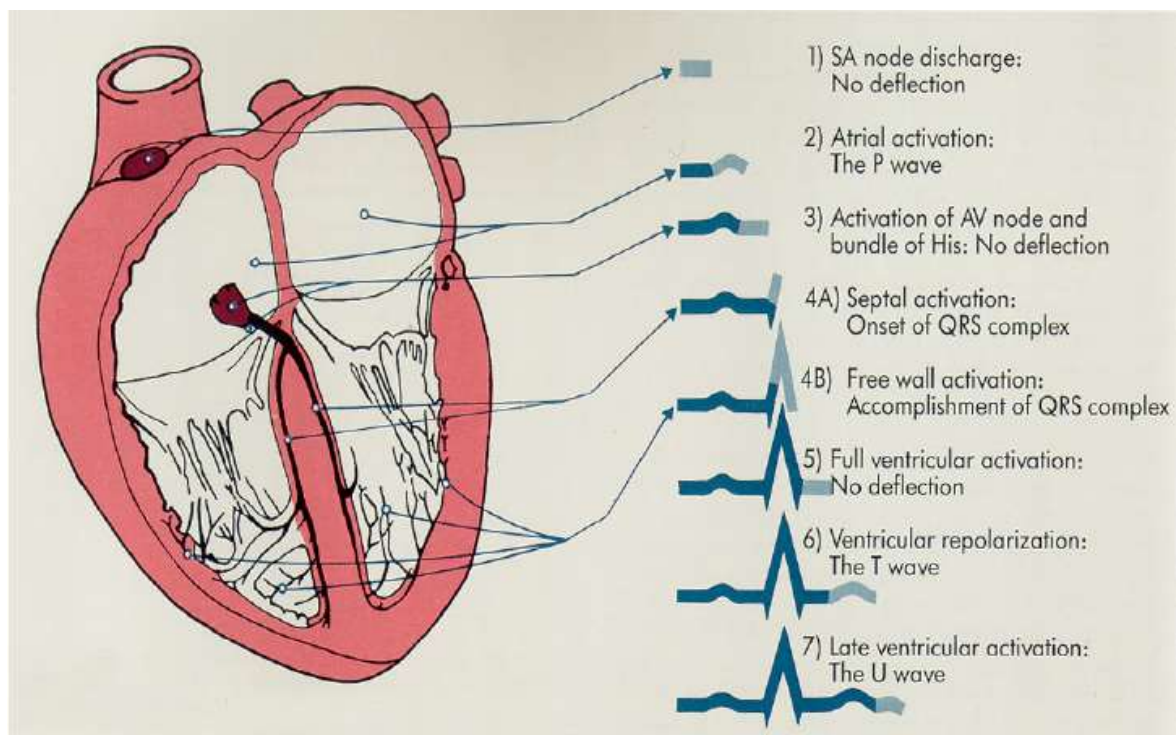
V2: Fourth intercostal space to the Left of the sternum.

V3: Directly between leads V2 and V4.

V4: Fifth intercostal space at midclavicular line.

V5: Level with V4 at left anterior axillary line.

V6: Level with V5 at left midaxillary line. (Directly under the midpoint of the armpit)



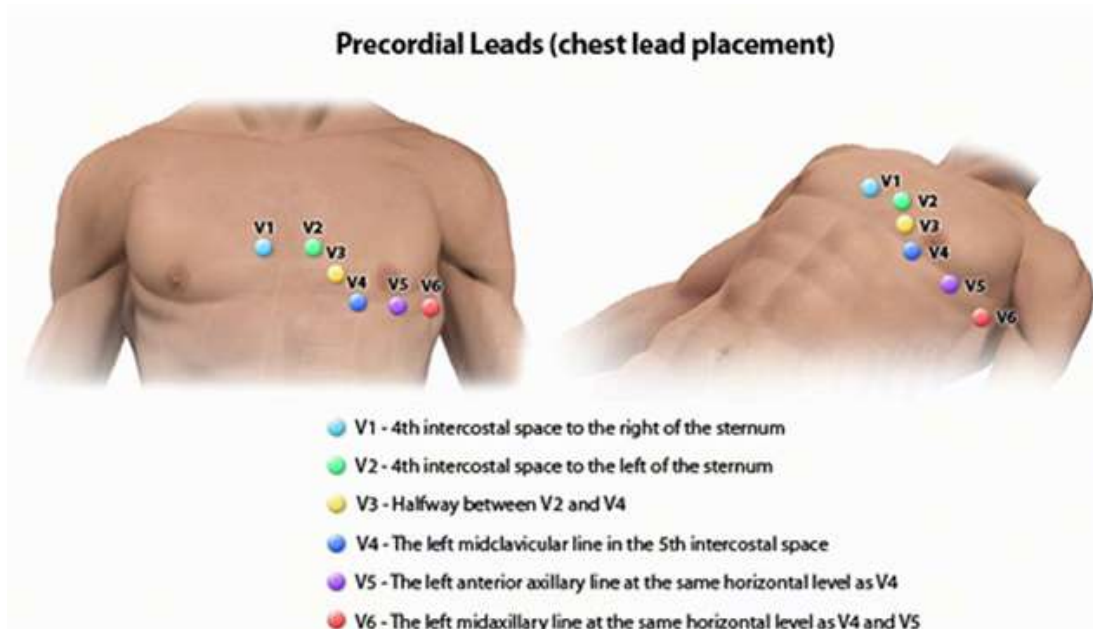


Fig 1. ECG Leads - Views of the Heart

Chest Leads

V1 & V2

V3 & V4

V5 & V6

View

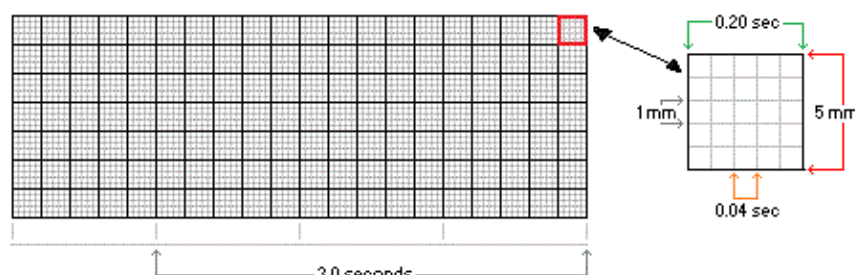
Right Ventricle

Septum/Lateral Left Ventricle

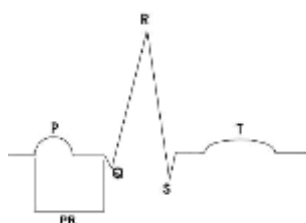
Anterior/Lateral Left Ventricle

ECG Interpretation

The graph paper that the ECG records on is standardised to run at 25mm/second, and is marked at 1 second intervals on the top and bottom. The horizontal axis correlates the length of each electrical event with its duration in time. Each small block (defined by lighter lines) on the horizontal axis represents 0.04 seconds. Five small blocks (shown by heavy lines) is a large block, and represents 0.20 seconds.



Duration of a waveform, segment, or interval is determined by counting the blocks from the beginning to the end of the wave, segment, or interval.



P-Wave: represents atrial depolarization - the time necessary for an electrical impulse from the sinoatrial (SA) node to spread throughout the atrial musculature.

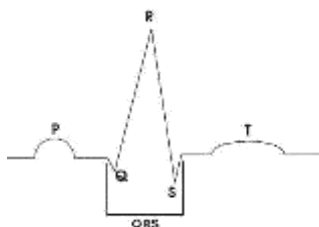
Location: Precedes QRS complex

Amplitude: Should not exceed 2 to 2.5 mm in height Duration: 0.06 to 0.11 seconds

P-R Interval: represents the time it takes an impulse to travel from the atria through the AV node, bundle of His, and bundle branches to the Purkinje fibres.

Location: Extends from the beginning of the P wave to the beginning of the QRS complex

Duration: 0.12 to 0.20 seconds.



QRS Complex: represents ventricular depolarisation. The QRS complex consists of 3 waves: the Q wave, the R wave, and the S wave.

- **The Q wave** is always located at the beginning of the QRS complex. It may or may not always be present.

- **The R wave** is always the first positive deflection.

- **The S wave**, the negative deflection, follows the R wave

Location: Follows the P-R interval

Amplitude: Normal values vary with age and sex

Duration: No longer than 0.10 seconds

Q-T Interval: represents the time necessary for ventricular depolarization and repolarization.

Location: Extends from the beginning of the QRS complex to the end of the T wave (includes the QRS complex, S-T segment, and the T wave)

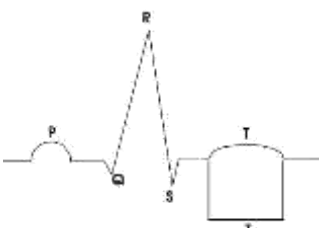
Duration: Varies according to age, sex, and heart rate

T Wave: represents the repolarization of the ventricles. On rare occasions, a U wave can be seen following the T wave. The U wave reflects the repolarization of the His-Purkinje fibres.

Location: Follows the S wave and the S-T segment

Amplitude: 5mm or less in standard leads I, II, and III; 10mm or less in precordial leads V1-V6.

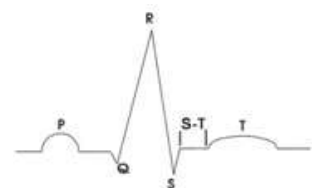
Duration: Not usually measured



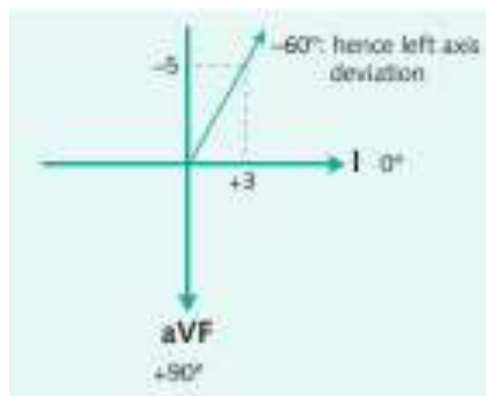
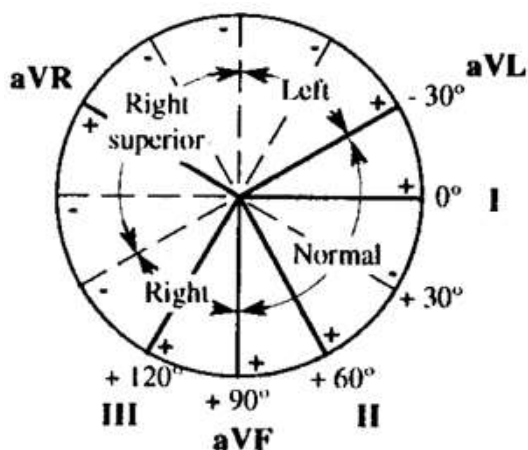
S-T Segment: represents the end of the ventricular depolarization and the beginning of ventricular repolarization.

Location: Extends from the end of the S wave to the beginning of the T wave

Duration: Not usually measured



CARDIAC AXIS



The rules for working out the cardiac axis are as follows:

- Calculate the net deflection of each lead – e.g. in lead I, if there is a Q wave measuring three small squares and an R wave height of six small squares, the net deflection is +3. Do this for leads I and aVF.
- A net positive deflection goes in the direction of the vector; negative deflections go in the opposite direction of the vector – e.g. net deflection of +3 in I goes 3 points in the direction of I; a net deflection of -5 in aVF goes in the opposite deflection of the vector (i.e. upwards) by 5 points.
- The cardiac vector is therefore the sum of the individual vectors from I and aVF – e.g. +3 in I, -5 in aVF gives a vector of about -60° . A normal axis is between 0° and $+90^\circ$; anything to the

left of 0° is termed left axis deviation; anything to the right of 90° is right axis deviation.

Causes of a Right superior axis deviation

- emphysema
- hyperkalaemia
- lead transposition
- artificial cardiac pacing
- ventricular tachycardia

Causes of right axis deviation

- normal finding in children and tall thin adults
- right ventricular hypertrophy
- chronic lung disease even without pulmonary hypertension
- anterolateral myocardial infarction
- left posterior hemiblock
- pulmonary embolus
- Wolff-Parkinson-White syndrome - left sided accessory pathway
- atrial septal defect
- ventricular septal defect

Causes of left axis deviation

- left anterior hemiblock
- Q waves of inferior myocardial infarction
- artificial cardiac pacing
- emphysema
- hyperkalaemia
- Wolff-Parkinson-White syndrome - right sided accessory pathway
- tricuspid atresia
- ostium primum ASD
- injection of contrast into left coronary artery

note: left ventricular hypertrophy is not a cause left axis deviation

NORMAL SINUS RHYTHM

Definition

- The default heart rhythm.
- Pacemaking impulses arise from the sino-atrial node and are transmitted to the ventricles via the AV-node and His-Purkinje system.
- This results in a regular, narrow-complex heart rhythm at 60-100 bpm.

Characteristics of normal sinus rhythm

- Regular rhythm at a rate of 60-100 bpm (or age-appropriate rate in children).
- Each QRS complex is preceded by a normal P wave.
- Normal P wave axis: P waves should be upright in leads I and II, inverted in aVR.
- The PR interval remains constant.
- QRS complexes are < 100 ms wide (unless a co-existent interventricular conduction delay is present).

Normal heart rates in children

- Newborn: 110 – 150 bpm
- 2 years: 85 – 125 bpm
- 4 years: 75 – 115 bpm
- 6 years+: 60 – 100 bpm



Sinus rhythm

Questions for self-control of knowledge:

1. What is the classification of cardiac arrhythmias.
2. Give the definition of "sinus tachycardia."
3. Describe sinus bradycardia, what are its causes?
4. Give the definition of "extrasystoles."
5. Explain the concept - heterotopic (ectopic) foci of excitation?
6. Give the definition of "paroxysmal tachycardia".
7. What is the commonality between paroxysmal tachycardia and arrhythmia?
8. What is dangerous paroxysmal tachycardia?
9. Give the definition of "ventricular fibrillation" heart. Which diseases of the heart appears this pathology?
10. What does the PQ interval prolongation on the ECG?
11. What is the basis of the periods of Wenckebach-Samoilov? As they appear on the ECG?
12. How does a complete atrioventricular heart block on ECG?
13. Specify the causes, mechanisms of development, and manifestations of the longitudinal heart block.
14. When and why does syndrome Morgagni-Stokes-Edemsa develop? How does it manifest? What it can cause disorders of activity?

Tasks for self-managed student work:

1. Stokes-Adams attack: etiology, pathogenesis, outcomes, methods of therapy.
2. Wolff-Parkinson-White syndrome.
3. Mechanism of development of reperfusion arrhythmias.

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