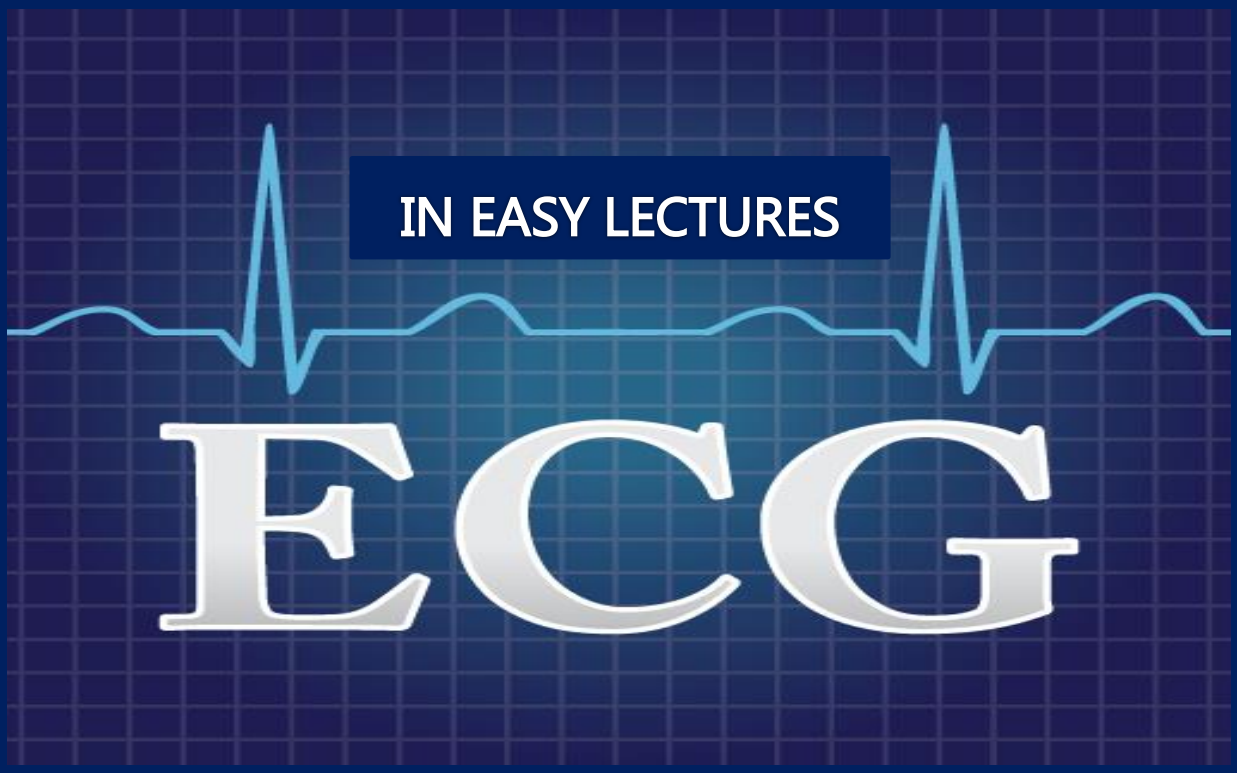

BY:

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Anesthesia Department

2017

الاهداء

إلى من جرع الكأس فارغاً ليسقيني قطرة حب

إلى من كلّت أنامله ليقدم لنا لحظة سعادة

إلى من حصد الأشواك عن دربي ليمهد لي طريق العلم

إلى القلب الكبير (والدي العزيز)

****

إلى من أرضعتني الحب والحنان

إلى رمز الحب وبلسم الشفاء

إلى القلب الناصع بالبياض (والدتي الحبيبة)

****

إلى القلوب الطاهرة الرقيقة والنفوس البريئة إلى رياحين حياتي

(إخوتي)

****

إلى النفوس الطاهرة التي غذتني علما

(أساتذتي)

****

الآن تفتح الأشرعة وترفع المرساة لتنطلق السفينة في عرض بحر واسع مظلم هو بحر الحياة وفي هذه الظلمة لا يضيء إلا قنديل الذكريات ذكريات الأخوة البعيدة إلى الذين أحببتهم وأحبوني

(أصدقائي)

أمجد عدنان

ألمقـــــــــدمة

أضع بين ايديكم عملي هذا الذي ارجو ان يكون على المستوى المطلوب و امل انني لم اقصر في الاحاطة النسبيه بموضوعات الملزمة التي تهدف الى *(تسهيل قراءة تخطيط القلب الكهربائي بشكل عملي ليسهل على المتخصصين معرفة الامراض الشائعة المكتشفة بواسطة تخطيط القلب).*

و ارجو من الاساتذه الافاضل و كذلك اخواني الطلبه ان لا يبخلوا علينا بالملاحظات و المقترحات البناءة لتصويب اخطاءنا لنحاول ان نتفادى زلاتنا و نحاول ان نتلافى العيوب من الممكن اننا ولا شك وقعنا في بعضها

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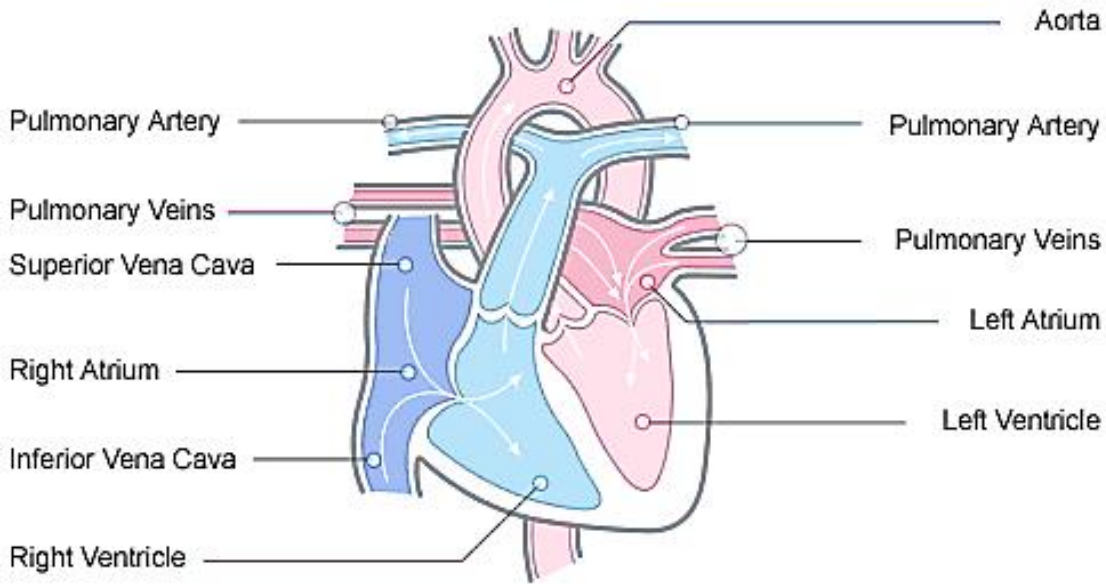
The Heart

The **heart** itself is **made up of** 4 chambers, 2 atria and 2 ventricles. De-oxygenated blood returns to the right side of the heart via the venous circulation. It is pumped into the right ventricle and then to the lungs where carbon dioxide is released and oxygen is absorbed. The oxygenated blood then travels back to the left side of the heart into the left atria, then into the left ventricle from where it is pumped into the aorta and arterial circulation.



The **pressure created** in the arteries by the contraction of the left ventricle is the **systolic blood pressure**. Once the left ventricle has fully contracted it begins to relax and refill with blood from the left atria. The pressure in the arteries falls whilst the ventricle refills. This is the **diastolic blood pressure.**

The **atrio-ventricular septum** completely separates the 2 sides of the heart. Unless there is a septal defect, the 2 sides of the heart never directly communicate. Blood travels from right side to left side via the lungs only. However the chambers themselves work together. The 2 atria contract simultaneously, and the 2 ventricles contract simultaneously.

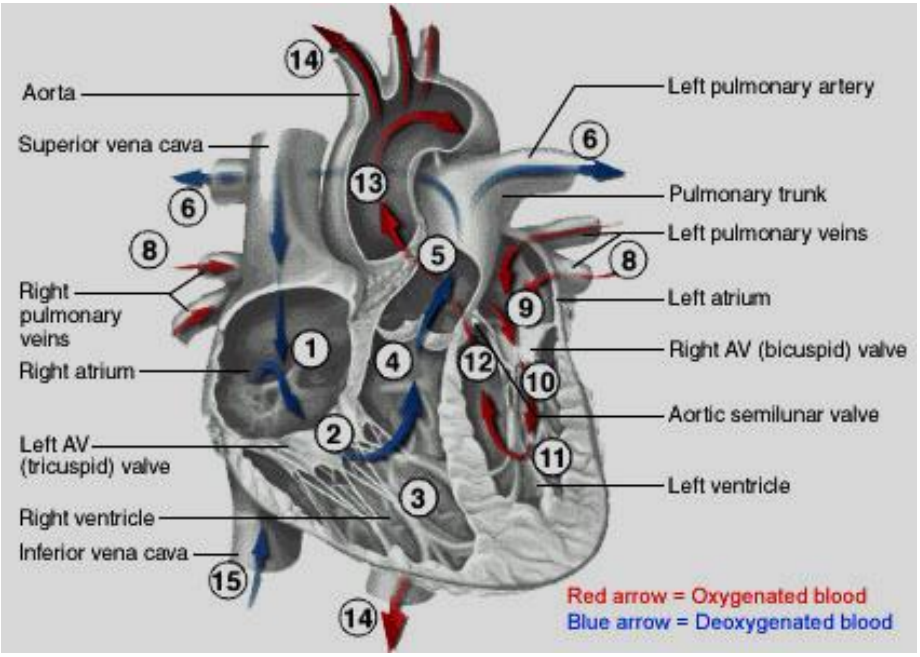
Normal Function of the Heart

The simplest way to describe the heart is as a **"pump".** Quite often doctors and nurses take the analogy further and talk to patients about their "plumbing". This analogy is reasonably accurate. The role the heart plays in the cardiovascular system is similar to the role played by the pump in your central heating system. The heart pumps blood through the arteries

and veins to organs, muscles and tissues, just as the central heating pump forces hot water through the pipes to the radiators.

Blood Flow

- During each heartbeat, the **right side** of the heart receives blood from your body and then sends it to the lungs to pick up oxygen.
- The **left side** of the heart receives the blood from the lungs and then sends the blood to the rest of the body to deliver oxygen.

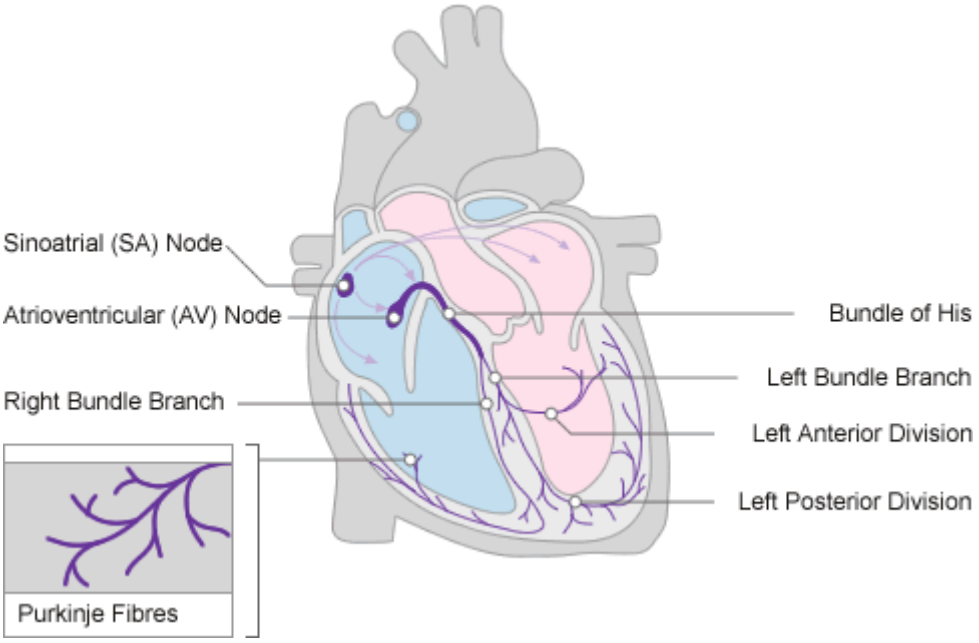


Cardiac Conduction System

Going back to the analogy of the central heating system, the pump, pipes and radiators are of no use unless connected to a power supply. The pump needs electricity to work. The human heart has a similar need for a power source and also uses electricity. Thankfully we don't need to plug ourselves in to the mains, the heart is able to create its own electrical impulses and control the route the impulses take via a specialized conduction pathway.

This pathway is made up of 5 elements:

1. The Sino-atrial (SA) node
2. The atrio-ventricular (AV) node
3. The bundle of His
4. The left and right bundle branches
5. The Purkinje fibres

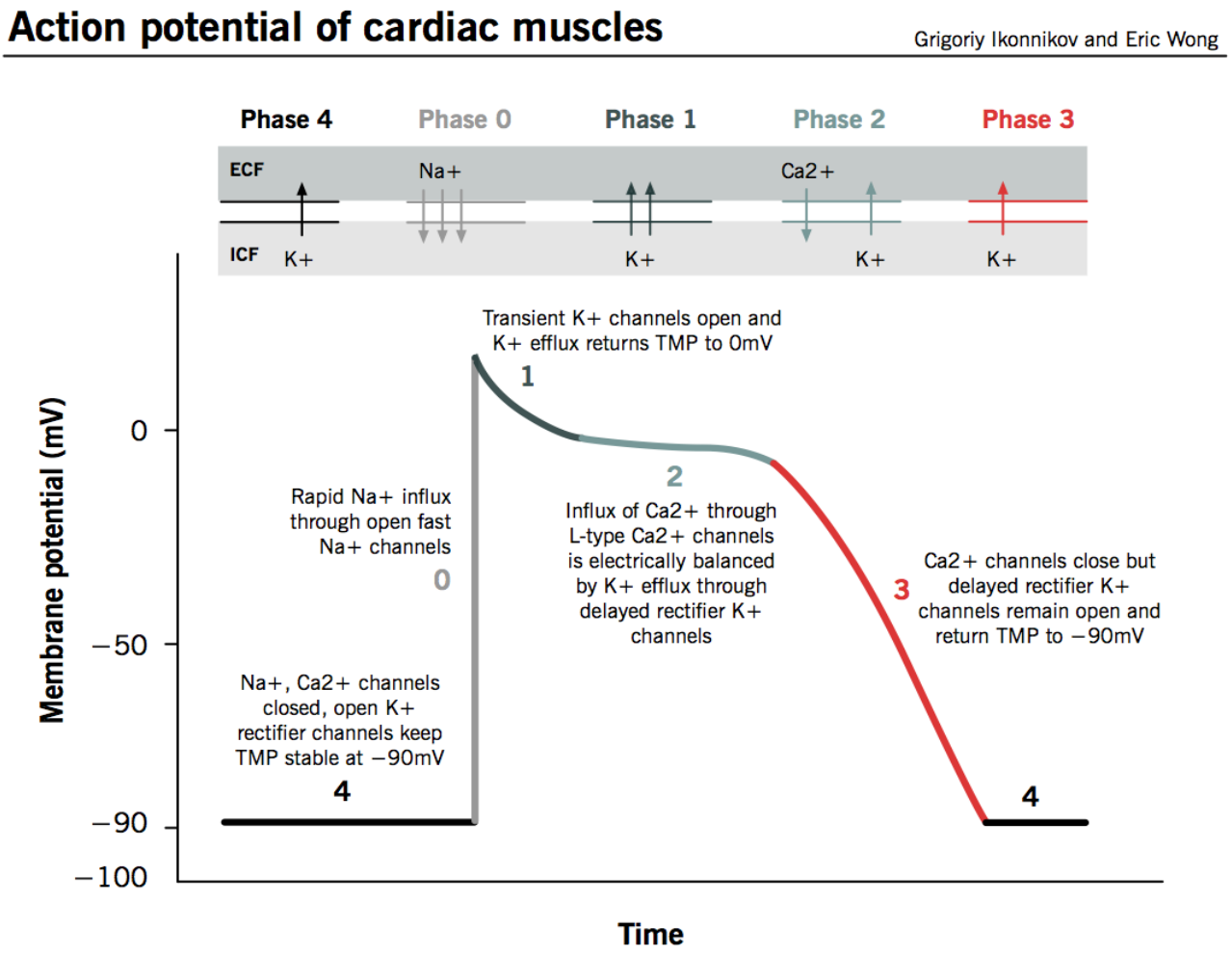


- **Sinoatrial (SA) node** normally generates the action potential, i.e. the electrical impulse that initiates contraction.
  - The SA node excites the right atrium (RA), travels through Bachmann's bundle to excite left atrium (LA).
  - The impulse travels through internodal pathways in RA to the atrioventricular (AV) node.
- **From the AV node**, the impulse then travels through the bundle of His and down the bundle branches, fibers specialized for rapid transmission of electrical impulses, on either side of the interventricular septum.
  - **Right bundle branch (RBB)** depolarizes the right ventricle (RV).
  - **Left bundle branch (LBB)** depolarizes the left ventricle (LV) and interventricular septum.
- Both bundle branches terminate in Purkinje fibers, millions of small fibers projecting throughout the myocardium.

Electrophysiology

- **Two main forces drive ions across cell membranes:**
  - **Chemical potential:** an ion will move down its concentration gradient.
  - **Electrical potential:** an ion will move away from ions/molecules of like charge.
- The **transmembrane potential (TMP)** is the electrical potential difference (voltage) between the inside and the outside of a cell. When there is a net movement of +ve ions into a cell, the TMP becomes more +ve, and when there is a net movement of +ve ions out of a cell, TMP becomes more –ve.

- **Ion channels** help maintain ionic concentration gradients and charge differentials between the inside and outside of the cardiomyocytes.



- **Automaticity:** unlike other cardiomyocytes, pacemaker cells do not require external stimulation to initiate their action potential; they are capable of self-initiated depolarization in a rhythmic fashion. This property is known as automaticity, whereby the cells undergo spontaneous depolarization and an action potential is triggered when threshold voltage is reached.

- **Unstable membrane potential:** Pacemaker cells have an unstable membrane potential and their action potential is not usually divided into defined phases.

- **No rapid depolarization phase:** Pacemaker cells have fewer inward rectifier K^+ channels than do other cardiomyocytes, so their TMP is never lower than −60 mV. As fast Na^+ channels need a TMP of −90 mV to reconfigure into an active state, they are permanently inactivated in pacemaker cells so there is no rapid depolarization phase.

Neural modulation of contractility

- Heart is innervated by both parasympathetic and sympathetic afferent and efferent neurons.
  - **Sympathetic:** postganglionic sympathetic fibers from paravertebral sympathetic ganglia associated with **T1-T5** innervate the atria, ventricles, and conduction system.
  - **Parasympathetic:** parasympathetic innervation is limited to vagal efferent fibers which innervate the SA node and the AV node; parasympathetic innervation to the ventricles is minimal.
- **Sympathetic neurons release** norepinephrine, a catecholamine, which activates β1 receptors on cardiac myocytes, leading to the following effects (note: epinephrine, also a catecholamine, can be made by the adrenal glands and released into the circulation, and has the same effect on β1 receptors):
  - **Chronotropic:** increased heart rate
  - **Dromotropic:** faster conduction through AV node
  - **Inotropic:** increased contractility
  - **Lusitropic:** faster relaxation after contraction
- **Parasympathetic neurons release** acetylcholine, a cholinergic hormone, which activates muscarinic M2-receptors on cardiac myocytes, leading to just one main effect:
  - **Negative chronotropic:** decreased heart rate.

The cardiac muscle has certain special properties which are:

1. **Rhythmicity:** ability of the heart to beat regularly at constant rate.
2. **Contractility:** ability of the heart to contract and push blood into circulation.
3. **Excitability:** ability of the cardiac muscle to respond to an adequate stimulus contraction.
4. **Conductivity:** ability of the cardiac muscle to conduct excitation wave from one part of the heart to another.

In **EKG** study we are concerned with study of **Rhythmicity and conductivity** of the cardiac muscle.

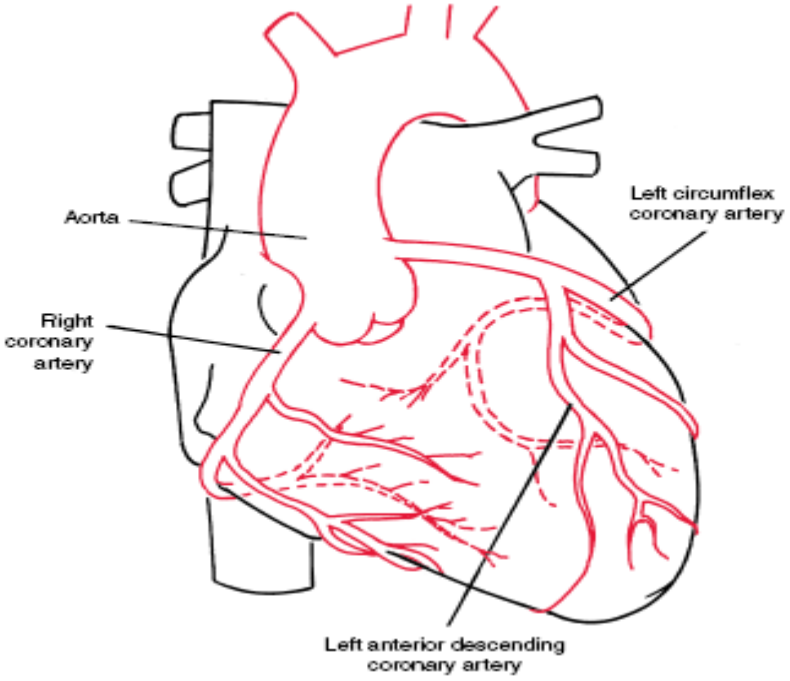
Anatomy of the coronary arteries

The left Coronary artery:

It arises from the left sinus of Valsalva and passes forwards & to the left in the atrioventricular groove for a short distance and then divides into two branches:

1. **The left anterior descending artery:** it passes downwards in the anterior interventricular groove to the apex of the heart & then turns backwards to anastomse with the posterior descending artery.

2. **The circumflex artery:** it continues its course in the left atrioventricular groove to anastomse with the right coronary. It gives several obtuse marginal branches.



The right Coronary artery: It arises from the (right sinus) of Valsalva and runs in the right atrioventricular groove to the posterior surface of the heart to anastomse with circumflex artery.

In the back of the heart it gives the (posterior descending artery which runs downwards, in the posterior interventricular groove, to anastomose with the anterior descending artery.

Pattern of coronary supply

- **Balanced circulation:**

The **left coronary artery** supplies left atrium, left ventricle & anterior part of the interventricular septum. While the **right coronary artery** supplies right atrium, right ventricle & posterior part of the interventricular septum.

- **Right dominance:**

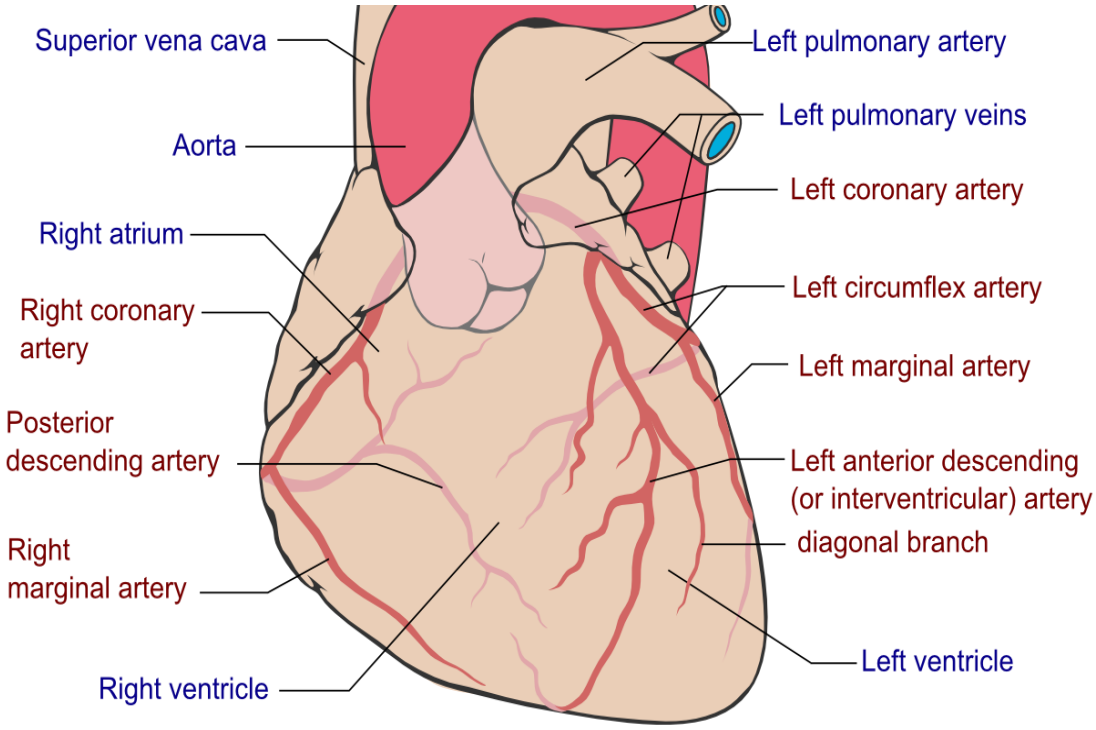
The right coronary supplies also the posterior part of the left ventricle.

- **Left dominance:**

The left coronary supplies also the posterior part of the septum & the posterior wall of the right ventricle.

Heart has two types of action

- **Mechanical**: Contraction &relaxation
- **Electrical**: Depolarization & repolarization



Electrode placement

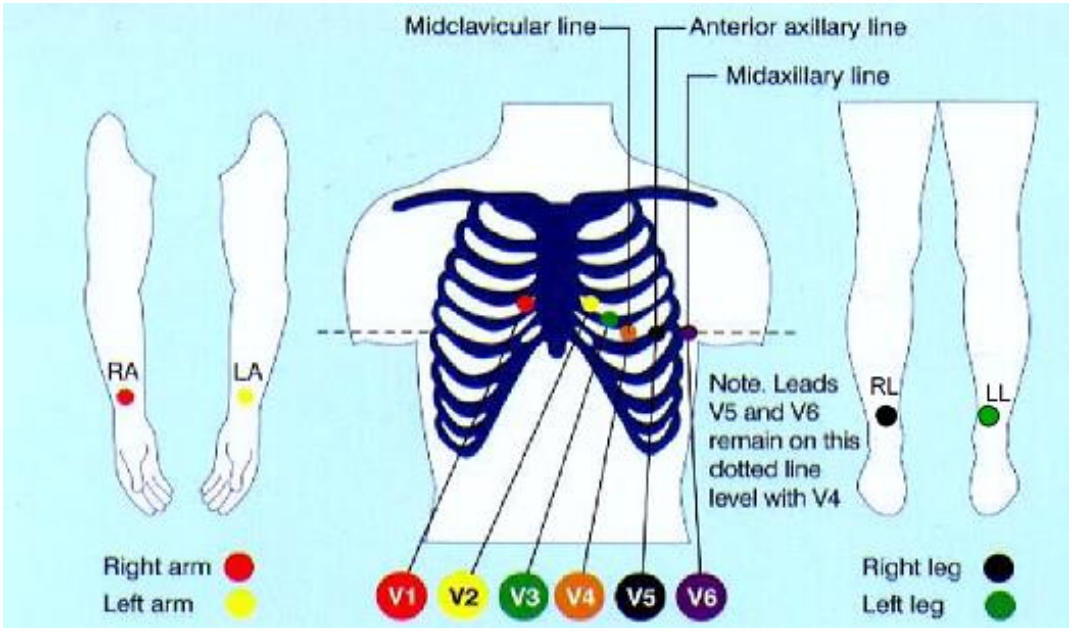
- **10 electrodes in total are placed on the patient.**
- Firstly self-self-adhesive 'dots' are attached to the patient. These have single electrical contacts on them.
- The 10 leads on the ECG machine are then clipped onto the contacts of the 'dots'.

Electrode placement in 12 lead ECG

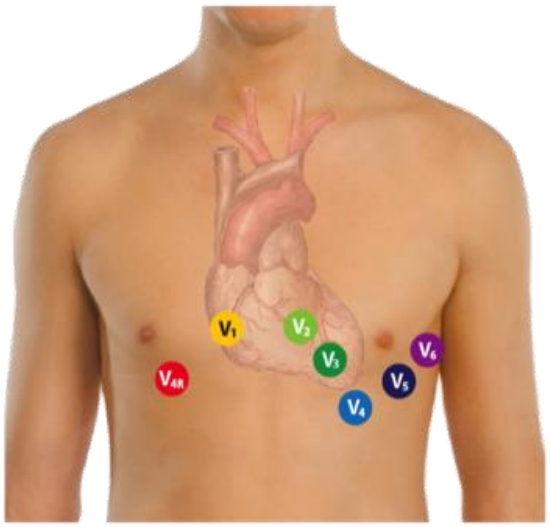
- **6** are chest electrodes: Called **V1-V6** or **C1-C6**
- **4** are limb electrodes
  - **Right arm** **R**ide
  - **Left arm** **Y**our
  - **Left leg** **G**reen
  - **Right leg** **B**ike

Remember

The right leg electrodes a neutral or "dummy"

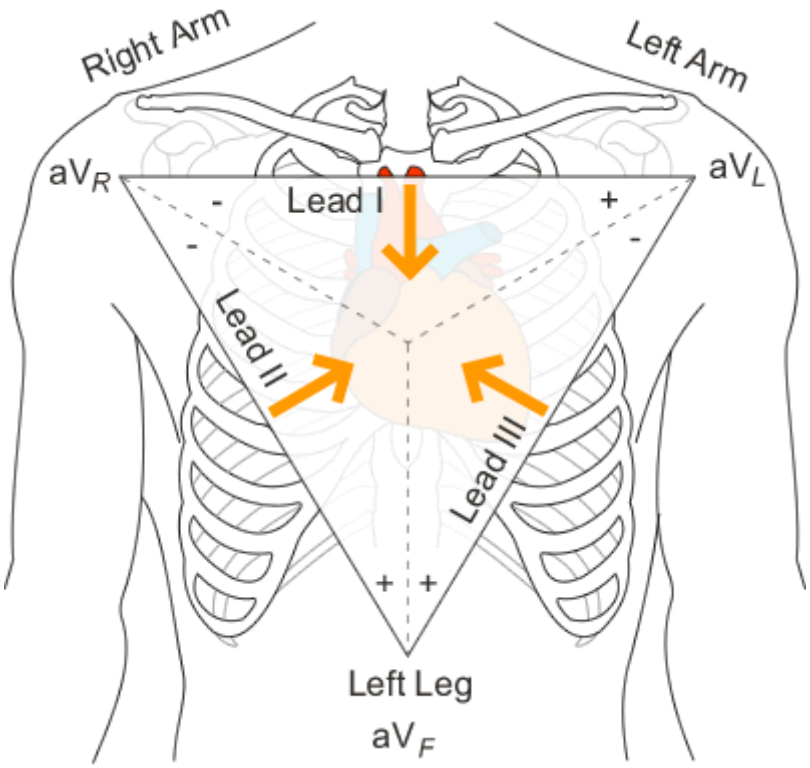


ELECTRODE	PLACEMENT
V1	4th Intercostal space to the right of the sternum
V2	4th Intercostal space to the left of the sternum
V3	Midway between V2 and V4
V4	5th Intercostal space at the **midclavicular line**
V5	Anterior axillary line at the same level as V4
V6	**Midaxillary line** at the same level as V4 and V5
RL	Anywhere above the ankle and below the torso
RA	Anywhere between the shoulder and the elbow
LL	Anywhere above the ankle and below the torso
LA	Anywhere between the shoulder and the elbow



- V_1 — 4th intercostal space to the right of the stemum
- V_2 — 4th intercostal space to the left of the stemum
- V_3 — directly between the leads V_2 & V_4
- V_4 — 5th intercostal space at midclavicular line
- V_5 — Level with V_4 at left anterior axillary line
- V_6 — Level with V_5 at left midaxillary line *(directly under the midpoint of the armpit)*
- V_{4R} — 5th intercostal right midclavicular line

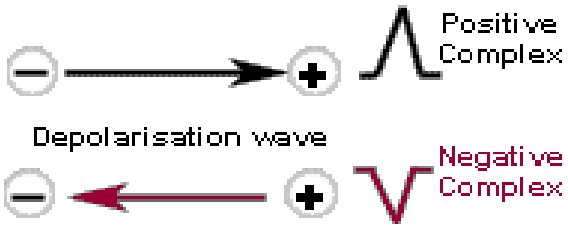
Chest Leads



Einthoven's triangle - Line of site of the bipolar leads

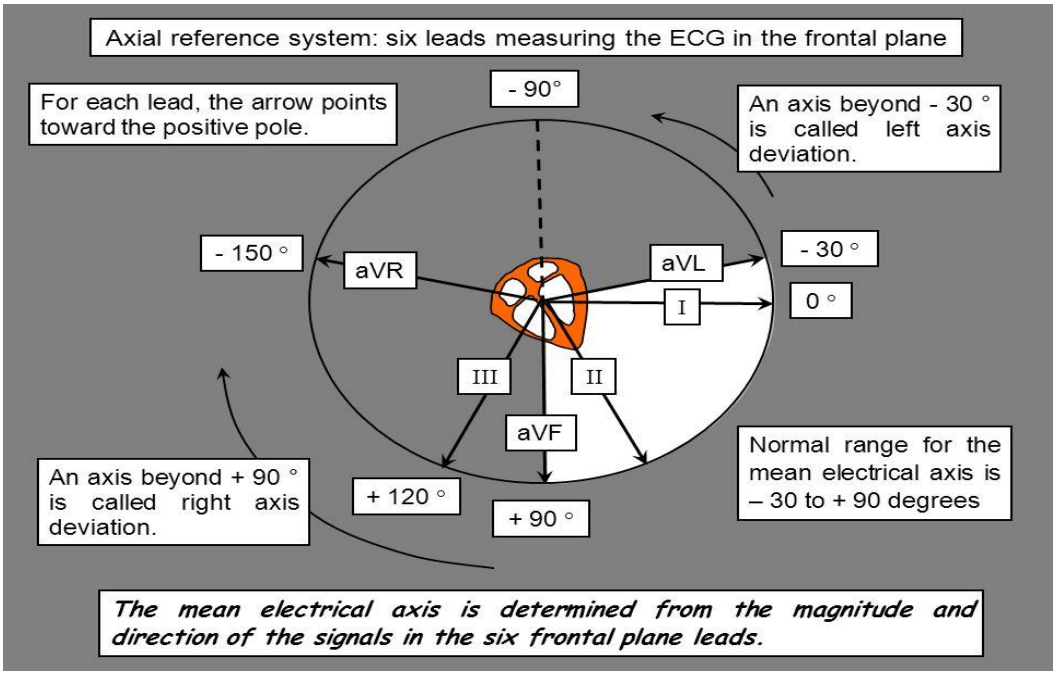
How does the ECG WORK?

- Electrical impulse (wave of depolarization) picked up by placing electrodes on patient.
- The voltage change is sensed by measuring the current change across **2 electrodes – a positive electrode and a negative electrode.**
- If the electrical impulse travels towards the positive electrode this results in a positive deflection.
- If the impulse travels away from the positive electrode this results in a negative deflection.



Types of Leads

- Coronal plane (Limb Leads)
  1. Bipolar leads – I, II, III
  2. Unipolar leads — aVL, aVR, aVF
- Transverse plane V1 — V6 (Chest Leads)

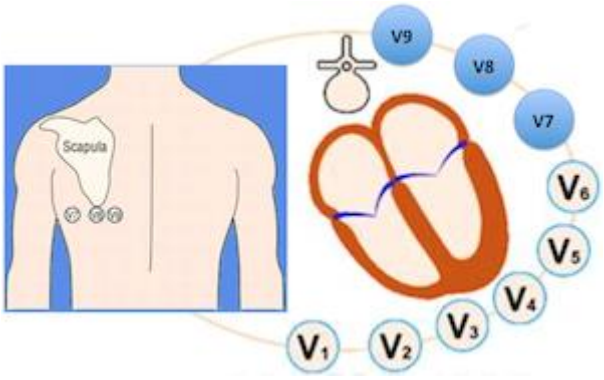


Electrodes around the heart

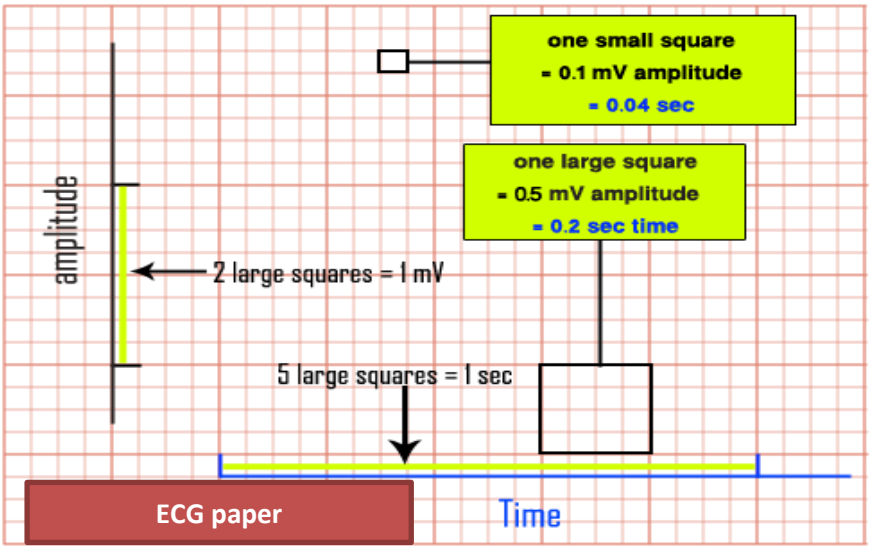
- Each lead can be thought of as 'looking at' an area of myocardium:

يعني ان الاقطاب تمثل كاميرا تصور القلب من كافة الاتجاهات كما في الصورة الاتية

I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral



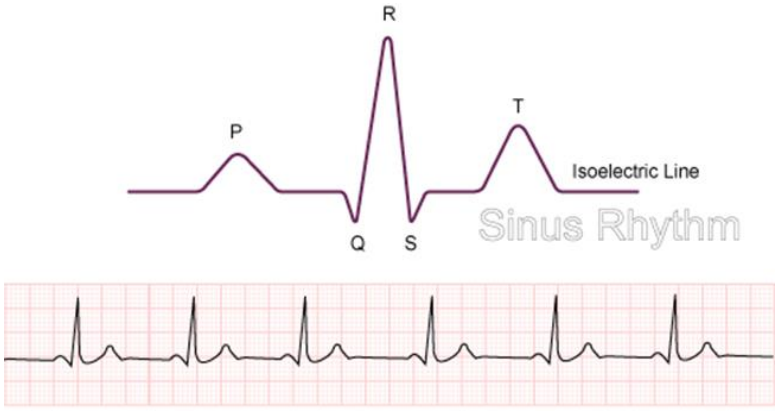
ECG Paper



ECG tracings are recorded on grid paper. **The horizontal axis** of the ECG paper records time, with black marks at the top indicating 3 second intervals. Each second is marked by 5 large grid blocks. Thus each **large block equals 0.2 second**. **The vertical axis** records ECG amplitude (voltage). Two large blocks equal 1 millivolt (mV). **Each small block equals 0.1 mV**. Within the large blocks are 5 small blocks, each representing 0.04 seconds.

Sinus Rhythm

Sinus rhythm is the name given to the normal rhythm of the heart where electrical stimuli are initiated in the SA node, and are then conducted through the AV node and bundle of His, bundle branches and Purkinje fibres.

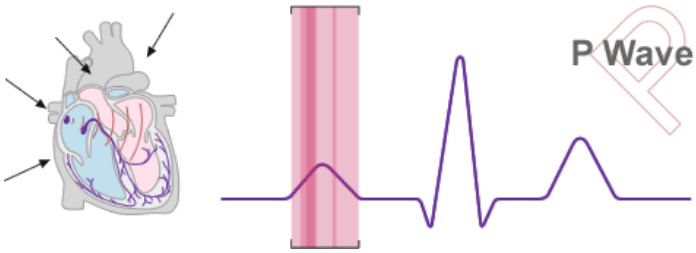


Elements of the trace

The P Wave

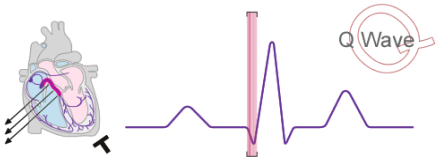
The first wave **(p wave)** represents atrial depolarization. When the valves between the atria and ventricles open, 70% of the blood in the atria falls through with the aid of gravity, but mainly due to suction caused by the ventricles as they expand.

Atrial contraction is required only for the final 30% and therefore a relatively small muscle mass are required and only a relatively small amount of voltage is needed to contract the atria.

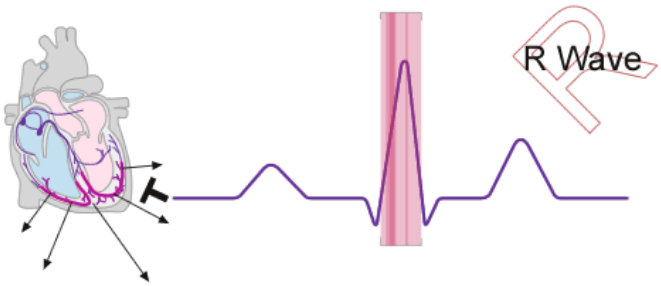


The QRS Complex

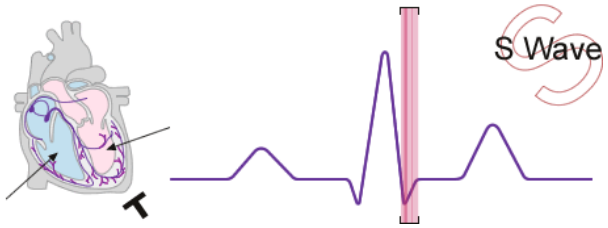
- Represent Ventricles Depolarization
- **Q Wave:** a small negative wave immediately before the large QRS complex. This is known as a Q wave and represents depolarization in the septum.



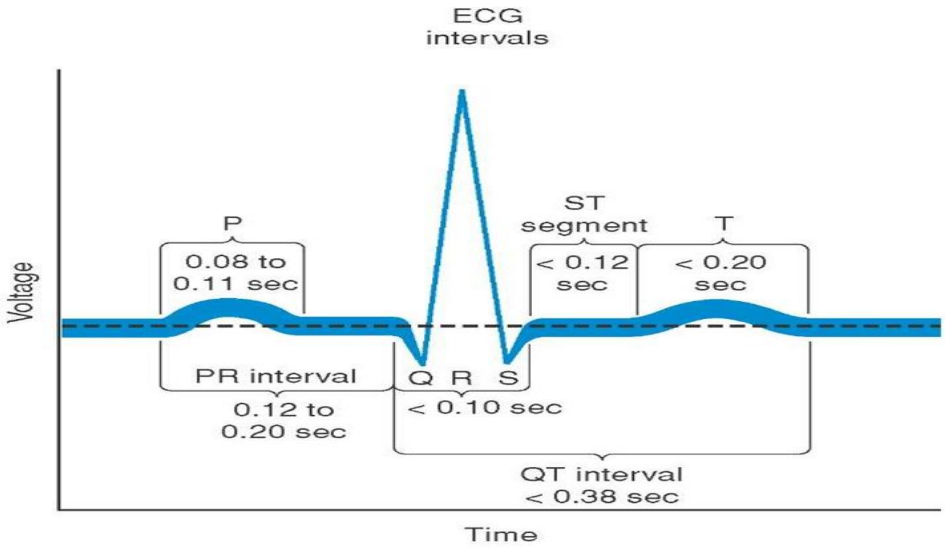
- **R Wave:** The largest wave in the QRS complex is the **R wave**; the R wave represents the electrical stimulus as it passes through the main portion of the ventricular walls.



- **S wave:** You will also have seen a small negative wave following the large R wave. This is known as an S wave and **represents depolarization in the Purkinje fibres**.

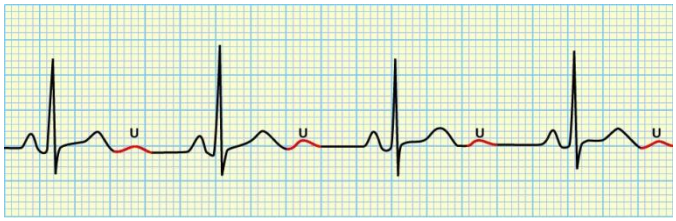


- **T Wave:** Both ventricles repolarize before the cycle repeats itself and therefore a 3rd wave (t wave) is visible **representing ventricular repolarization**.



U Wave

The U wave is a small waveform that follows the T wave and represents repolarization of the Purkinje fibres. It may or may not be present. Normal U waves are small, round, asymmetric and about 10% of the height of the T wave.



PR Interval (PRI)

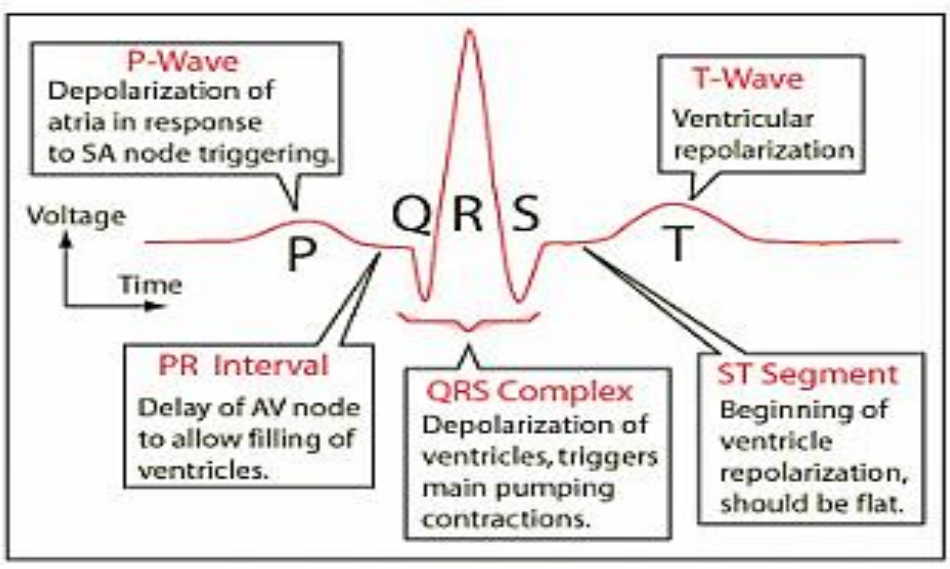
The **PRI** is the time from the beginning of atrial depolarization to the beginning of ventricular depolarization. **A normal PRI is 0.12 to 0.20 seconds** in adults.

ST Segment

The ST segment represents early ventricular repolarization and is normally at the isoelectric line on the ECG. **Elevation or depression of 1 mm (one box) or more is considered abnormal**.

QT Interval

The QT interval is the period from the beginning of the QRS complex to the end of the T wave and represents total ventricular activity (depolarization to repolarization).



عندما ناخذ ورقة التخطيط يجب معرفة التالي :

- **Calibration**
- **Rate**
- **Rhythm**
- **Axis**
- **Elements of the tracing in each lead**

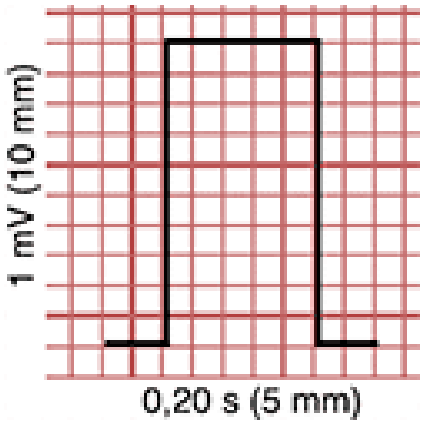
Calibration

Height

- 10mm = 1mV
- Look for a reference pulse which should be the rectangular looking wave somewhere near the left of the paper. It should be 10mm (10 small squares) tall.

Paper speed

- 25mm/s
- 25 mm (25 small squares / 5 large squares) equals one second



Rate

If the heart rate is regular

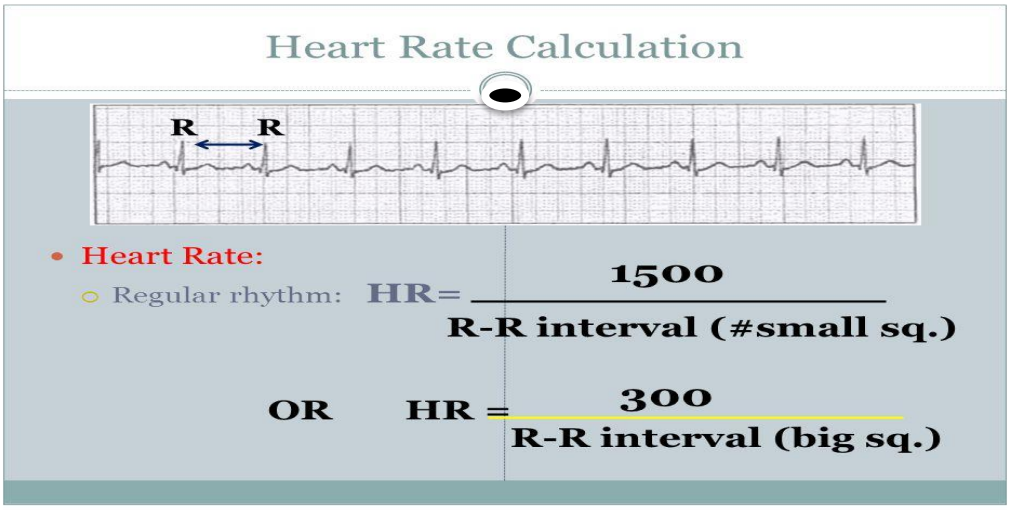
- Count the number of large squares between R waves
- i.e. the RR interval in large squares
- **Rate = 300/ RR**

E.g. RR = 4

- 4 large squares…….. 300/ 4= 75 beats per minute

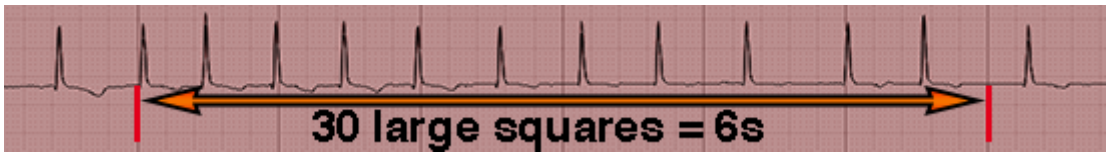
يمكن حساب ضربات القلب اذا كان التخطيط منتظم
من خلال تقسيم الرقم 300 على عدد المربعات الكبيرة بين ضربتين R وال R

الاخرى



For irregular rhythms:

- Usually Electrocardiograms record 10 seconds, so all you have to do is **count all QRS and multiply by 6.**
- If the ECG is not a 10 second one, or you are not aware of its duration, **count 30 large squares (6 seconds), and multiply the number of QRS complexes on them by 10**. The result is an approximate Heart Rate.



For example: ***Count the number of QRS complexes in 30 large squares (6 seconds) and multiply by 10. If there are 11 QRS complexes, the Heart Rate is 110 bpm (11x10= 110).***

حساب ضربات القلب اذا كان التخطيط غير منتظم

من خلال عدد موجات QRS في 30 مربع كبير

انشوف كم بيها QRS ونضربها في 10.) اختار اي Lead لحساب موجات QRS

ويفضل Lead II). في حساب المربعات منه

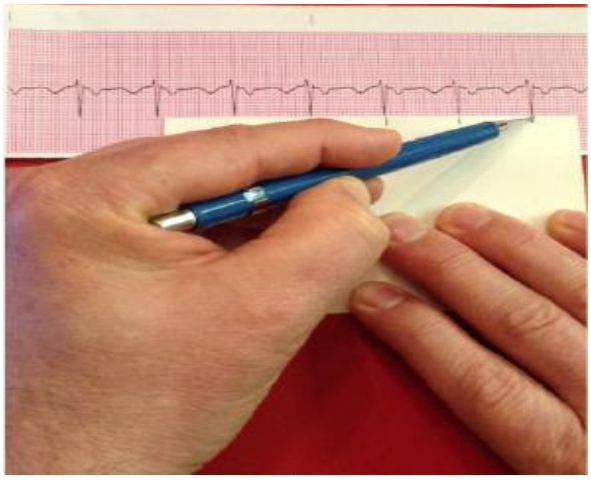
There are multiple methods to estimate the rate (Summary):

- **For regular rhythms**: Rate = 300 / number of *large* squares in between each consecutive R wave.
- **For very fast rhythms**: Rate = 1500 / number of *small* squares in between each consecutive R wave.
- **For slow or irregular rhythms**: Rate = number of complexes on the rhythm strip x 6 (this gives the *average* rate over a ten-second period).

Rhythm

Is the rhythm regular?

- The easiest way to tell is to take a sheet of paper and line up one edge with the tips of the R waves on the rhythm strip.
- Mark off on the paper the positions of **3 or 4 R** wave tips.
- Move the paper along the rhythm strip so that your first mark lines up with another R wave tip.
- See if the subsequent R wave tips line up with the subsequent marks on your paper.
- If they do line up, the rhythm is regular. If not, the rhythm is irregular.

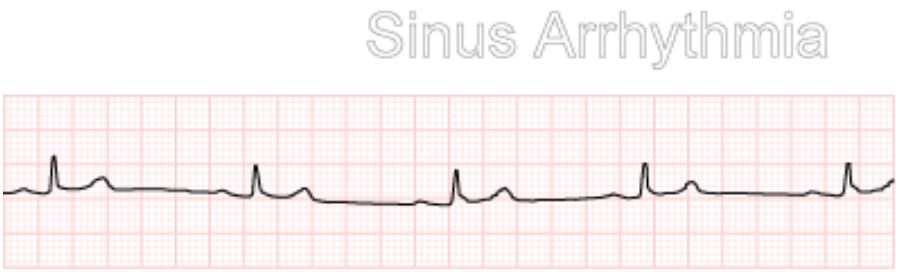


Sinus Rhythm

- **Definition:** Cardiac impulse originates from the sinus node. Every QRS must be preceded by a P wave.
- (This does not mean that every P wave must be followed by a QRS – such as in 2nd degree heart block where some P waves are not followed by a QRS, however every QRS is preceded by a P wave and the rhythm originates in the sinus node, and hence it is a sinus rhythm. It could be said that it is not a normal sinus rhythm).

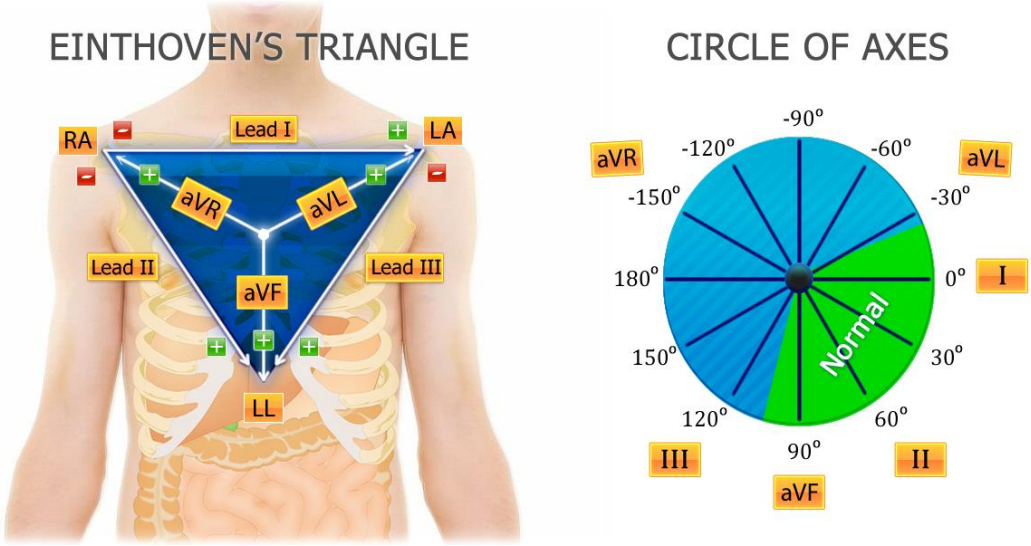
Sinus arrhythmia

- There is a change in heart rate depending on the **phase of respiration**
- **Q.** If a person with sinus arrhythmia inspires, what happens to t their heir heart rate?
- A. The heart rate speeds up. This is because on inspiration there is a decrease in intrathoracic pressure, this leads to an increased venous return to the right atrium. Increased stretching of the right atrium sets off a brainstem reflex (Bainbridge Bainbridge's reflex) that leads to s sympathetic activation of the heart, hence it speeds up)
- This physiological phenomenon is more apparent in children and young adults



Axis

- The axis can be thought of as the overall direction of the cardiac impulse or wave of depolarization of the heart
- An abnormal axis (axis deviation) can give a clue to possible pathology



- **Normal Axis** = QRS axis between -30 and +90 degrees.
- **Left Axis Deviation** = QRS axis less than -30 degrees.
- **Right Axis Deviation** = QRS axis greater than +90 degrees.
- **Extreme Axis Deviation** = QRS axis between -90 and 180 degrees (AKA "Northwest Axis").

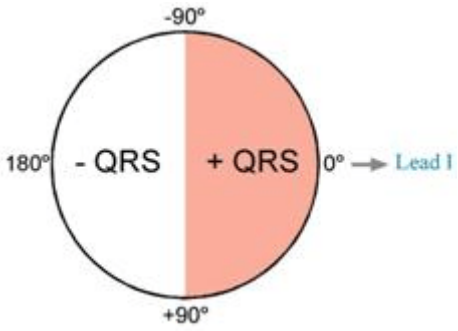
Method 1 – The Quadrant Method

The most efficient way to estimate axis is to look at leads I + aVF.

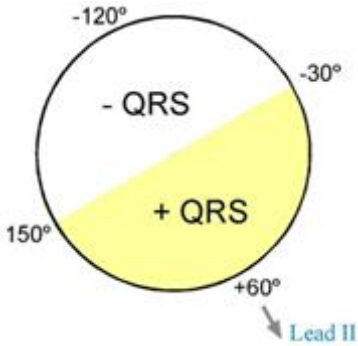
LEAD I	LEAD AVF	QUADRANT	AXIS
Positive	Positive	Left lower quadrant	Normal (0 to +90 degrees)
Positive	Negative	Left upper quadrant	Possible LAD (0 to -90 degrees)
Negative	Positive	Right lower quadrant	RAD (+90 to 180 degrees)
Negative	Negative	Right upper quadrant	Extreme Axis Deviation (-90 to 180 degrees)

Method 2 – Leads I + II

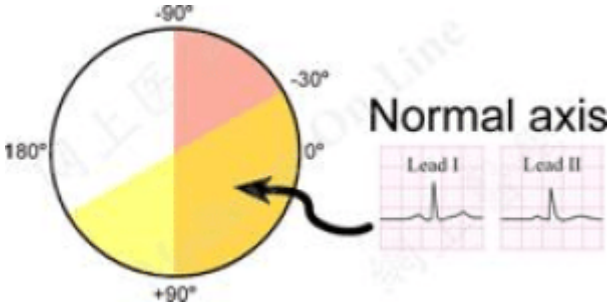
- Another **rapid method** is to look at leads I + II.
- A positive QRS in lead I puts the axis in roughly the same direction as lead I.



- Positive QRS in lead II similarly aligns the axis with lead II.



- Therefore, if leads I and II are both positive, the axis is between -30 and +90 degrees (i.e. normal axis).



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QRS Deflection			Axis
Lead I	**Lead II**	**Lead III**	
Positive	Positive	Positive or Negative	Normal
Positive	Negative	Negative	LAD
Negative	Positive or Negative	Positive	RAD

Summary

Causes of Axis Deviation

Right Axis Deviation

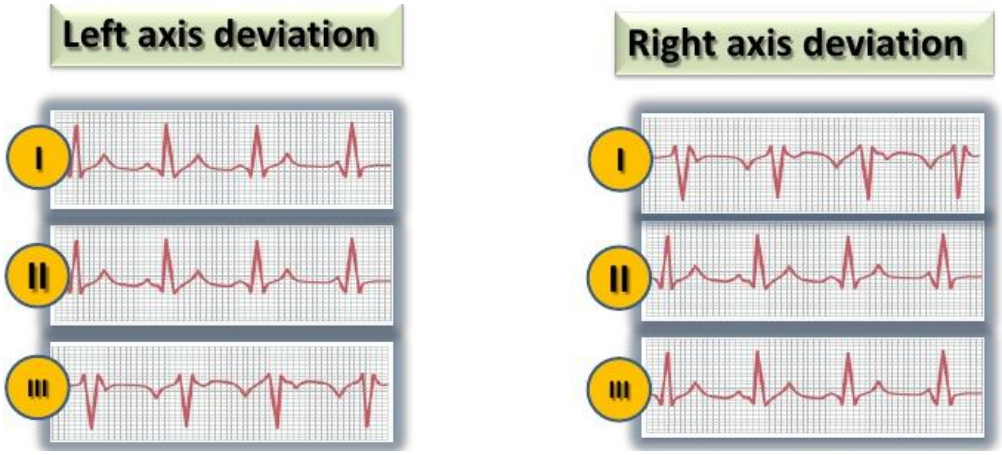
- Right ventricular hypertrophy
- Acute right ventricular strain, e.g. due to pulmonary embolism
- Lateral STEMI
- Chronic lung disease, e.g. COPD
- Hyperkalemia
- Sodium-channel blockade, e.g. TCA poisoning
- Wolff-Parkinson-White syndrome
- Dextrocardia
- Ventricular ectopic
- Normal pediatric ECG
- Vertically orientated heart – tall, thin patient

Left Axis Deviation

- Left ventricular hypertrophy
- Left bundle branch block
- Inferior MI
- Ventricular pacing /ectopic
- Wolff-Parkinson-White Syndrome
- Left anterior fascicular block – diagnosis of exclusion
- Horizontally orientated heart – short, squat patient

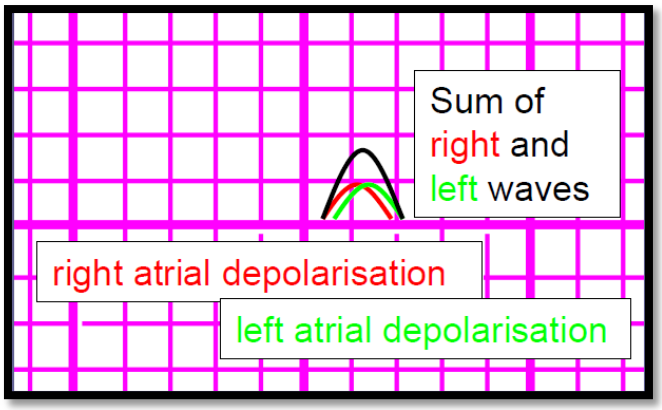
Extreme Axis Deviation

- Ventricular rhythms – e.g. VT, AIVR, ventricular ectopic
- hyperkalemia
- Severe right ventricular hypertrophy



The P wave

The **P wave** represents atrial depolarization, it can be thought of as being made up of two separate waves due to **right atrial depolarization** and **left atrial depolarization**.



Which occurs first?

- Right atrial depolarization

Characteristics of the Normal Sinus P Wave

Morphology

- Smooth contour
- Monophasic in lead II
- Biphasic in V1

Axis

- Normal P wave axis is between 0° and +75°
- P waves should be upright in leads I and II, inverted in aVR

Duration

- < 120 ms

Amplitude

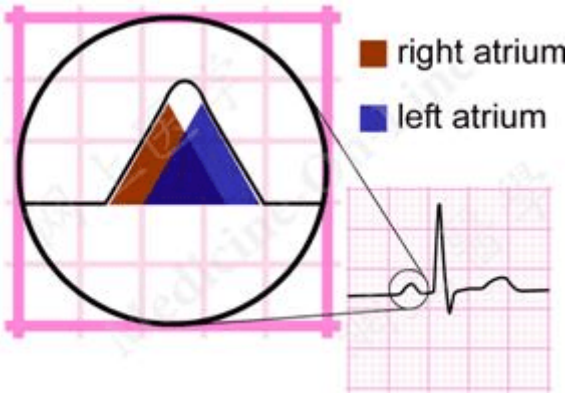
- < 2.5 mm in the limb leads,
- < 1.5 mm in the precordial leads

Note:

Atrial abnormalities are most easily seen in the inferior leads (II, III and aVF) and lead V1, as the P waves are most prominent in these leads.

Normal P-wave Morphology – Lead II

- The right atrial depolarization wave (brown) precedes that of the left atrium (blue).
- The combined depolarization wave, the P wave, is less than 120 ms wide and less than 2.5 mm high.

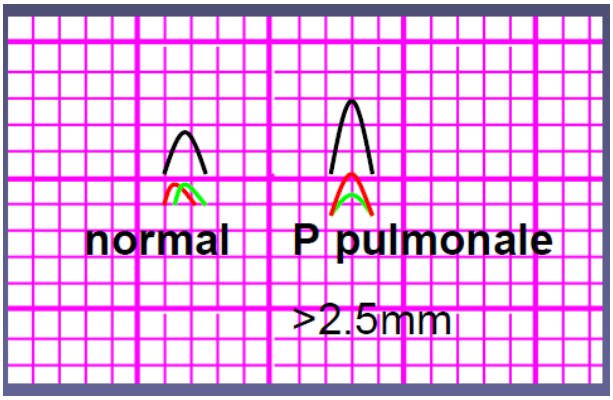


Height

- A tall P wave (over 2.5mm) can be called **P pulmonale**
- Occurs due to **Right atrial hypertrophy**

Causes include:

- Pulmonary hypertension.
- Pulmonary stenosis.
- Tricuspid stenosis.

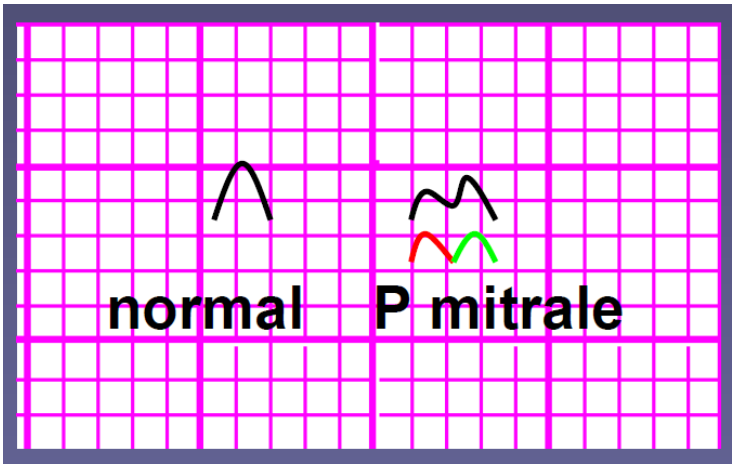


Length

- ✓ P wave with a length >0.08 seconds (2 small squares) and a bifid shape is called **P *mitrale.***
- ✓ It is **caused by** **left atrial hypertrophy** and **delayed left atrial depolarization**

Causes include:

- Mitral valve disease
- LVH



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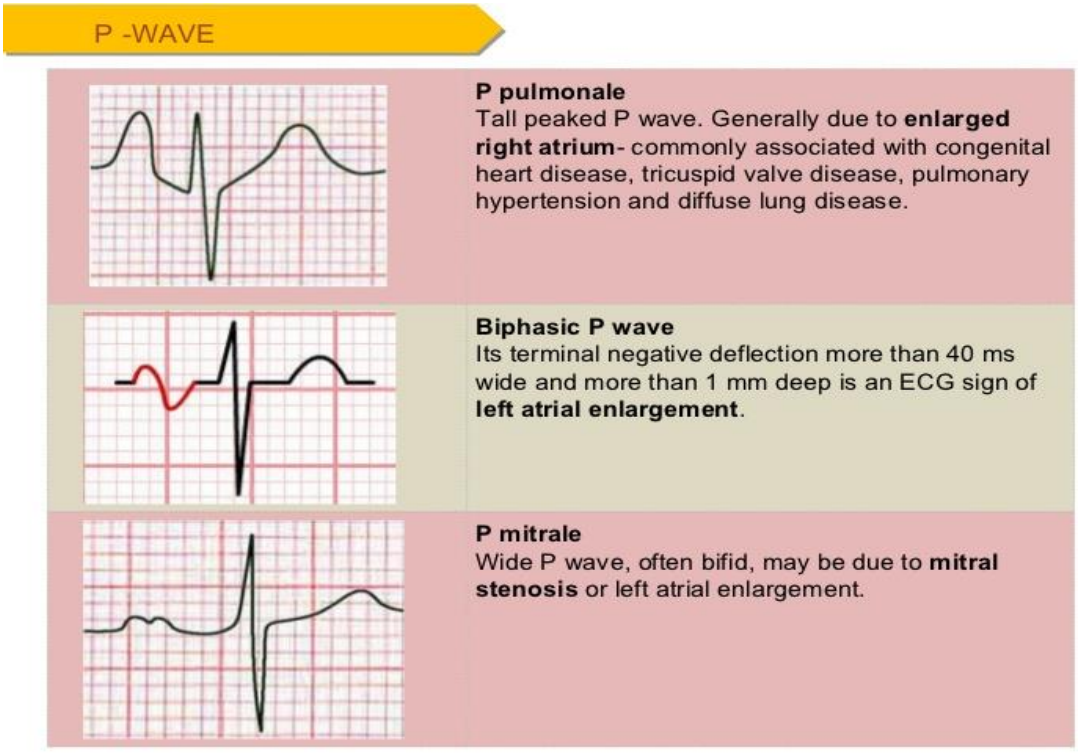
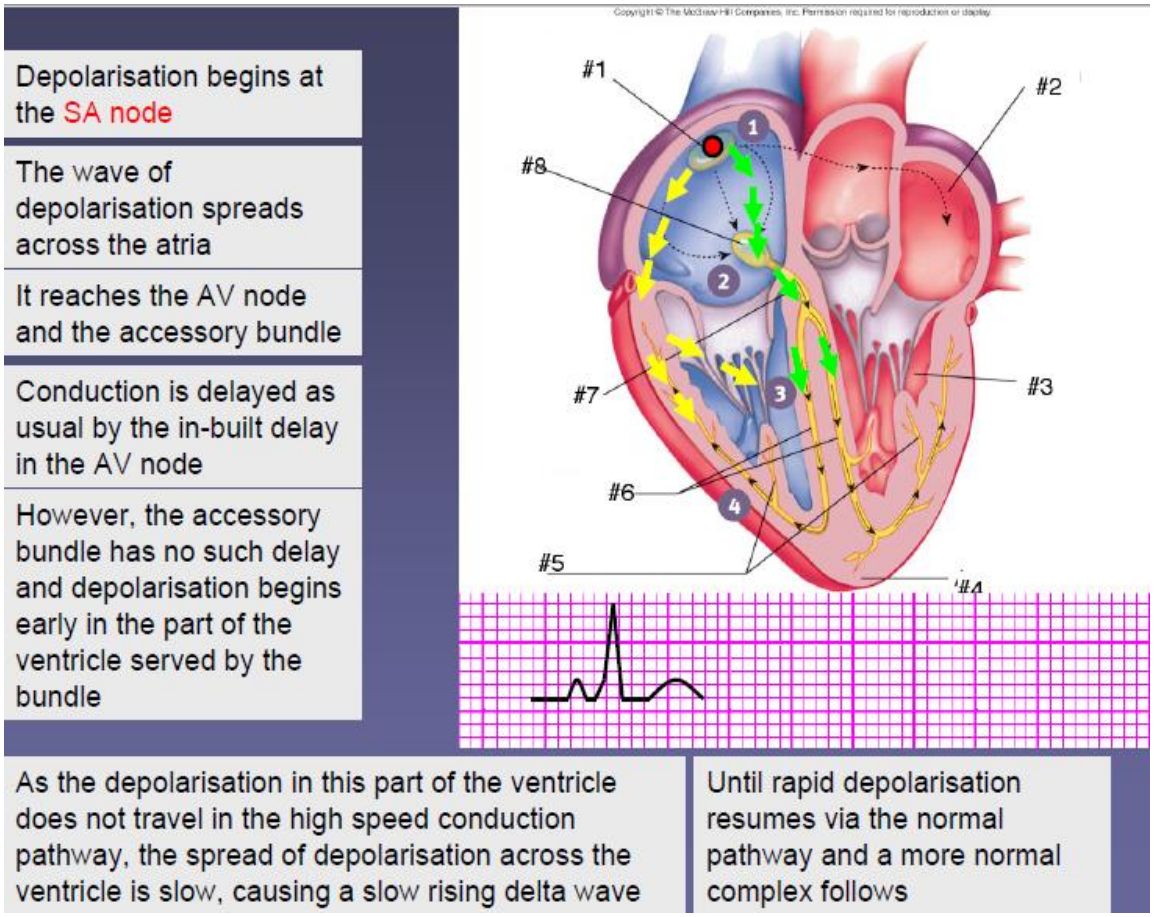


Summary

Normal PR interval

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



➢ **If the PR interval is long (>5 small squares or 0.2s):**
  ✓ If there is a constant long PR interval 1st degree heart block is present
  ✓ First degree heart block is a longer than normal delay in conduction at the AV node

➢ If the PR interval looks as though it is **widening** every beat and then a **QRS complex is missing**, there is **2nd degree heart block, Mobitz type I**. The **lengthening of the PR** interval in subsequent beats is known as the Wenckebach phenomenon.

➢ If the PR interval is constant but then there is a missed QRS complex then there is **2nd degree heart block, Mobitz type II**

➢ If there is no discernable relationship between the P waves and the QRS complexes, then **3rd degree heart block** is present

Heart block (AV node block)

Summary

- **1st degree**
- **constant PR, >0.2 seconds**

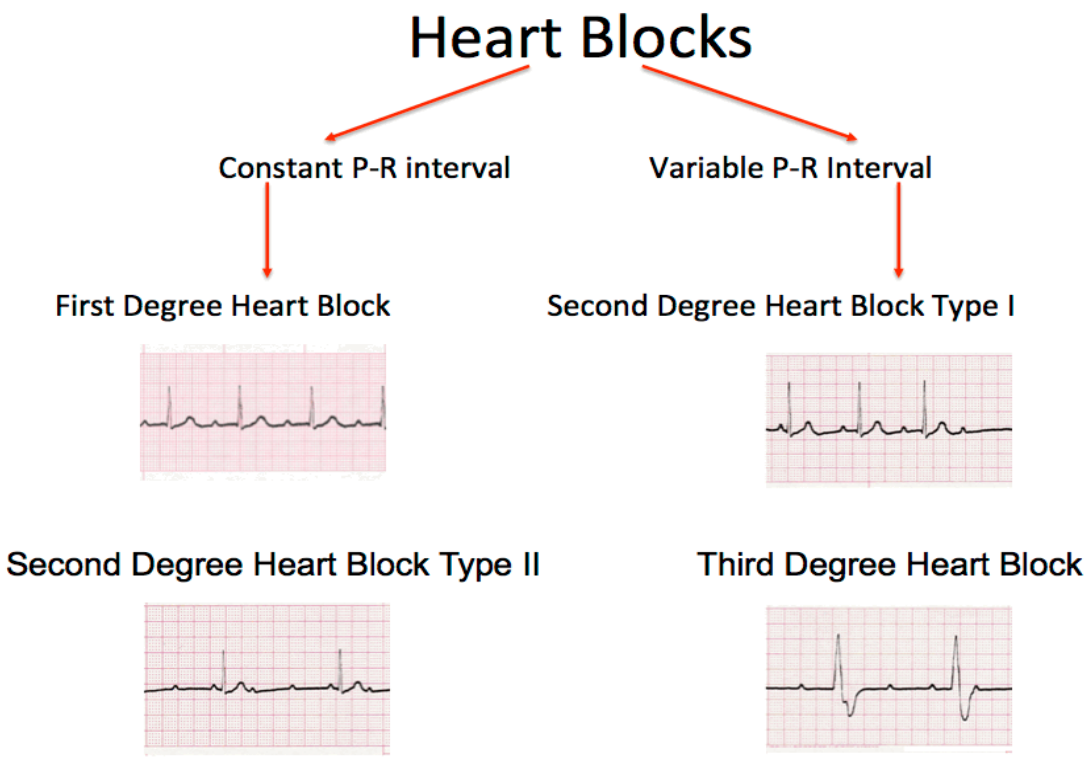
2nd degree type 1 (Wenckebach)

- **PR widens over subsequent beats then a QRS is dropped**

2nd degree type 2

- **PR is constant then a QRS is dropped**

- ✓ **3rd degree**
- ✓ **No discernable relationship between p waves and QRS complexes**



Normal QRS complex

- < 0.12 s duration (3 small squares)
    - For abnormally **wide QRS** consider right or left bundle branch block, ventricular rhythm, hyperkalemia, etc.
- no pathological Q waves
- no evidence of left or right ventricular hypertrophy

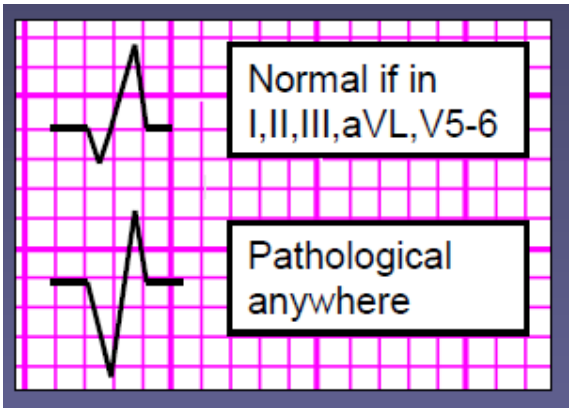
Are there any pathological Q waves?

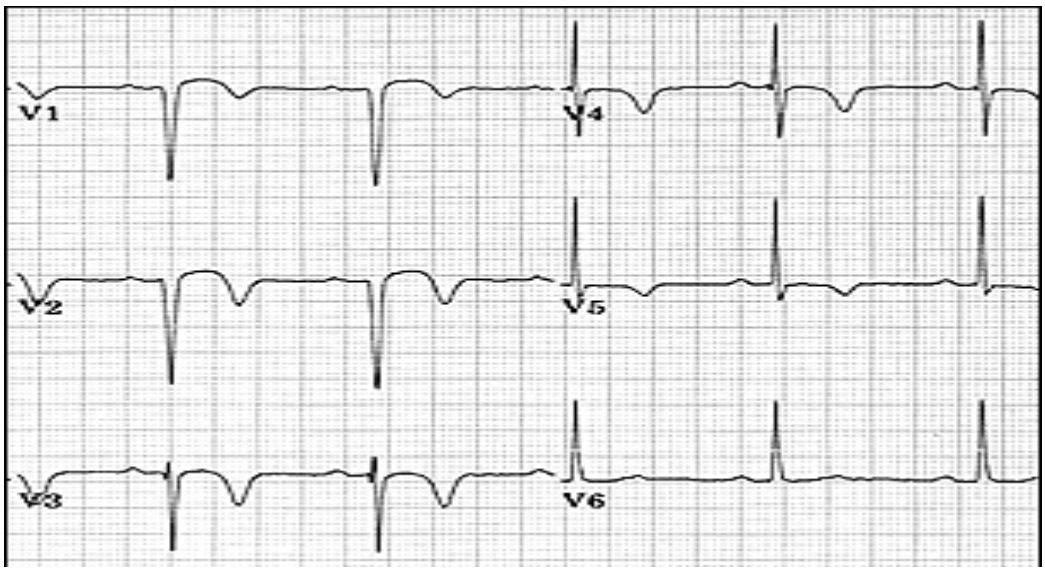
A Q wave can be pathological if it is:

 Deeper than 2 small squares (0.2mV) and/or

 Wider than 1 small square (0.04s) and/or

 In a lead other than III or one of the leads that look at the heart from the left (I, II, aVL, V5 and V6) where small Qs (i.e. not meeting the criteria above) can be normal.





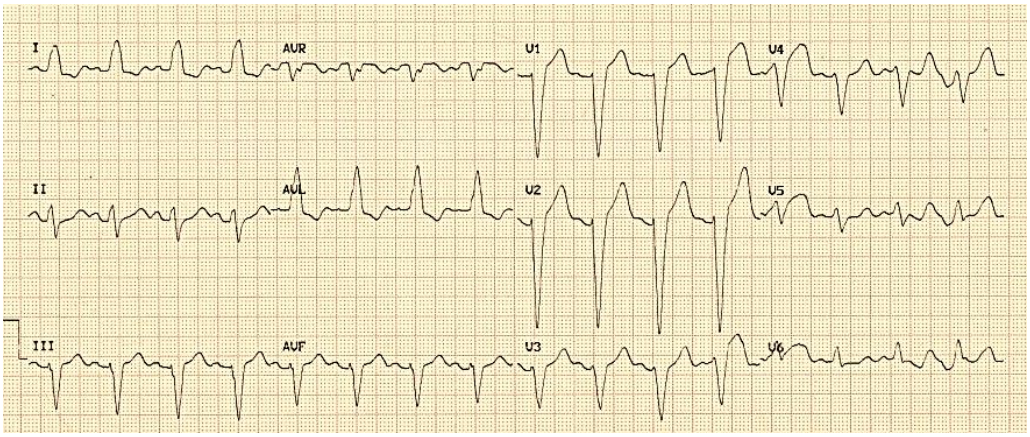
The QRS height

- If the complexes in the chest leads look very tall, consider left ventricular hypertrophy (LVH).
- If the depth of the S wave in V1 added to the height of the R wave in V6 comes to more than 35mm, **LVH is present**.

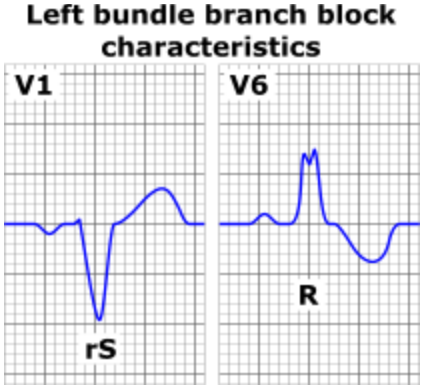
QRS width

- The **width of the QRS complex** should be less than 0.12 seconds (3 small squares).
- Some texts say less than 0.10 seconds (2.5 small squares)
- If the QRS is wider than this, it suggests a ventricular conduction problem: usually **right or left bundle branch block (RBBB or LBBB).**

Left Bundle Branch Block



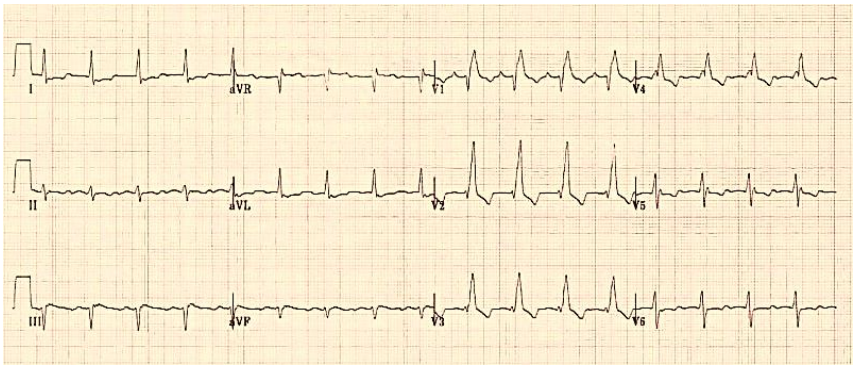
- If **left bundle branch block** is present, the QRS complex may look like a 'W' shape in V1 and/or an "M '' shape in V 6.
- New onset LBBB with chest pain considers **Myocardial infarction**.
- Not possible to interpret the ST segment.



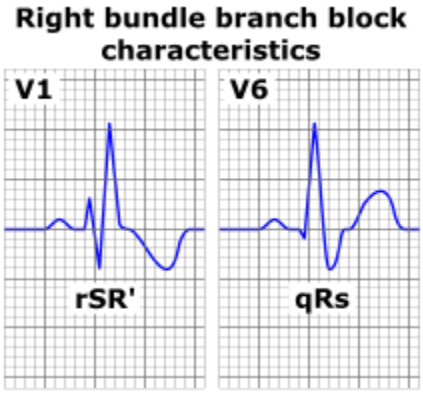
Causes

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

Right Bundle Branch Block



- It is also called RSR pattern
- If right bundle branch block is present, there may be an 'M' in V1 and/or a 'W' in V6.
- Can occur in healthy people with normal QRS width: partial RBBB.



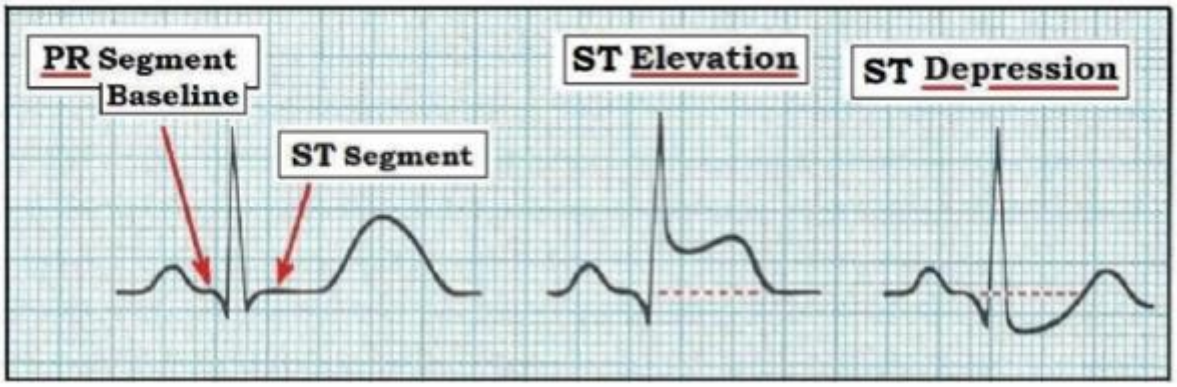
Causes of RBBB

- Right ventricular hypertrophy / corpulmonale
- Pulmonary embolus
- Ischemic heart disease
- Rheumatic heart disease
- Myocarditis or cardiomyopathy
- Degenerative disease of the conduction system
- Congenital heart disease (e.g. atrial septal defect)

Normal ST segment

- The **ST segment** should sit on the isoelectric line

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



Myocardial infarction

Within hours:

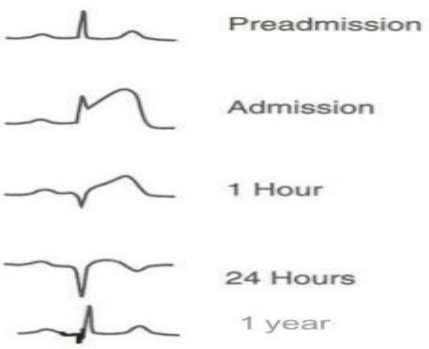
- T wave may become peaked
- ST segment may begin to rise

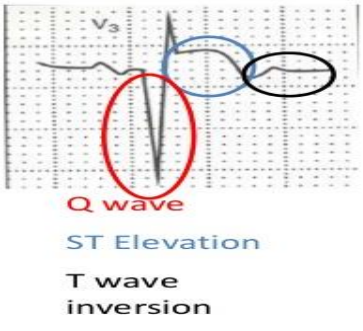
Within 24 hours:

- T wave inverts (may or may not persist)
- ST elevation begins to resolve
- If a left ventricular aneurysm forms, ST elevation may persist

Within a few days:

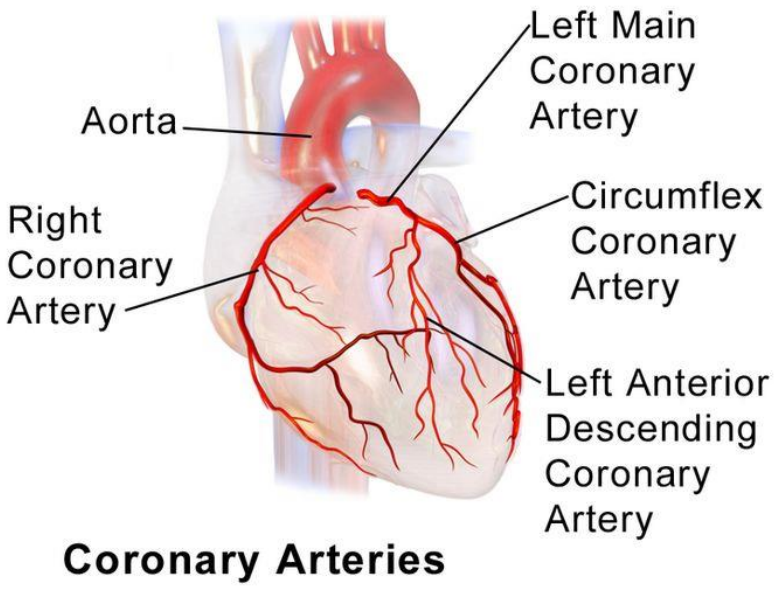
- pathological Q waves can form and usually persist



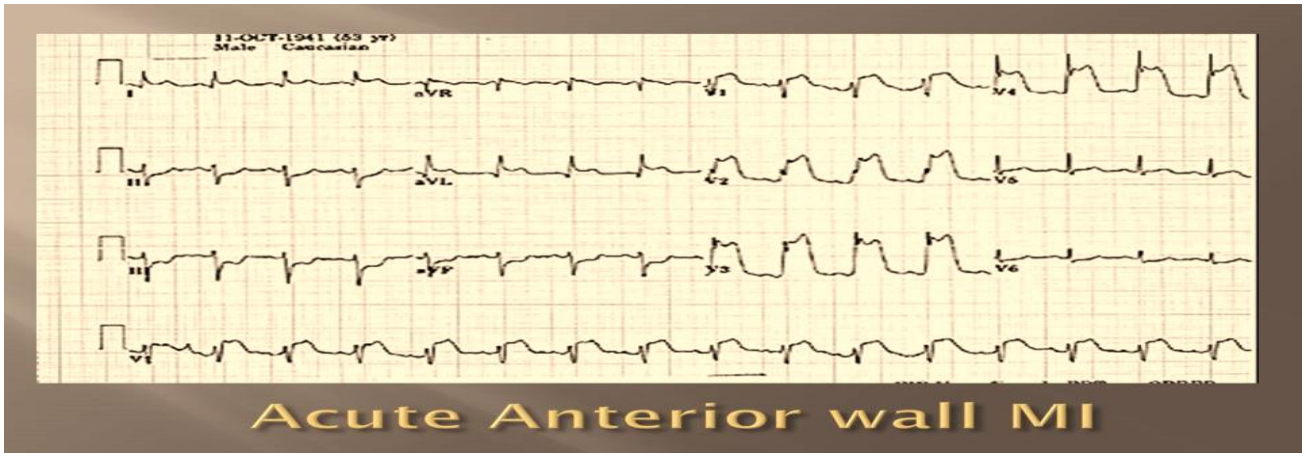


- **The leads affected determine the site of the infarct**

Description	ECG Leads With Changes	Artery Occluded
Inferior	II, III and aVF	RCA
Anteroapical	V3 and V4	Distal LAD
Anteroseptal	V1 and V2	LAD
Anterolateral	I, aVL, V5 and V6	Circumflex Artery
Extensive Anterior	I, aVL and V2-V6	Proximal LCA
True Posterior	Tall R in V1	RCA

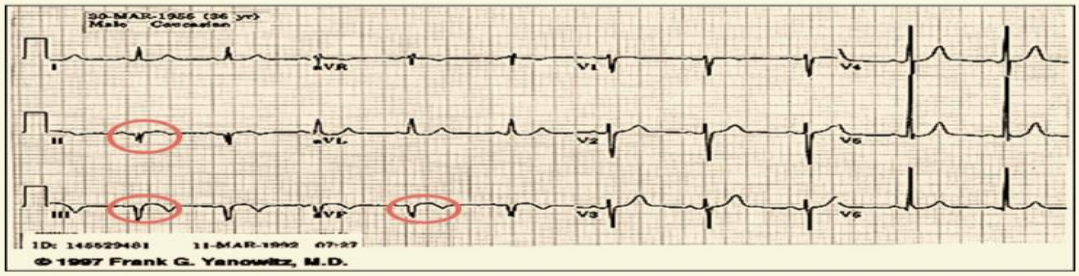


Coronary Arteries



Inferior Wall MI

This is an inferior MI. Note the ST elevation in leads II, III and aVF.



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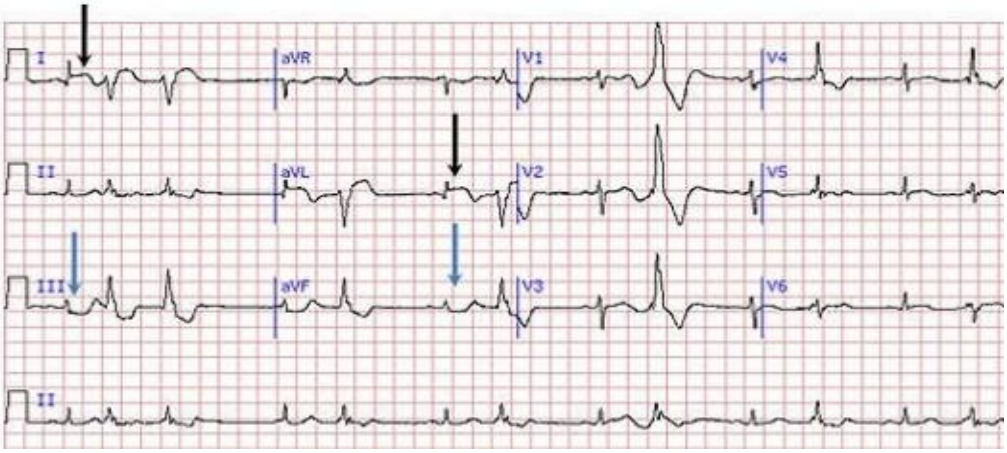
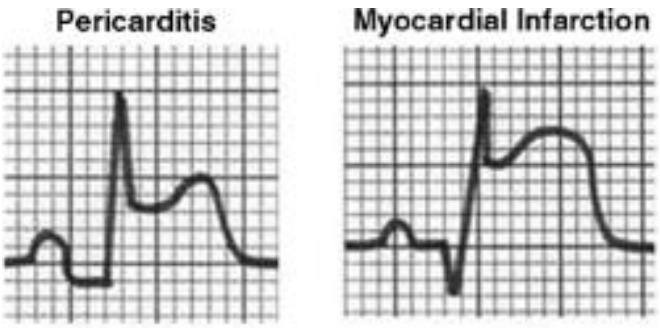
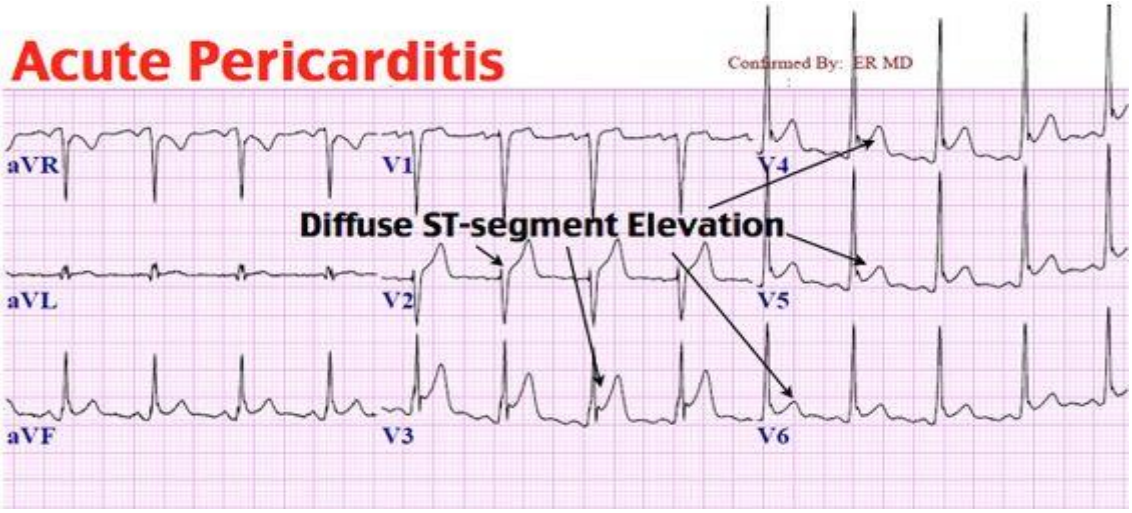
Lateral MI



NOTE:

- ✓ If the ST segment is elevated but slanted, it may not be significant
- ✓ If there are raised ST segments in most of the leads, it may indicate **pericarditis** especially if the **ST segments are saddle shaped**. There can also be PR segment depression.





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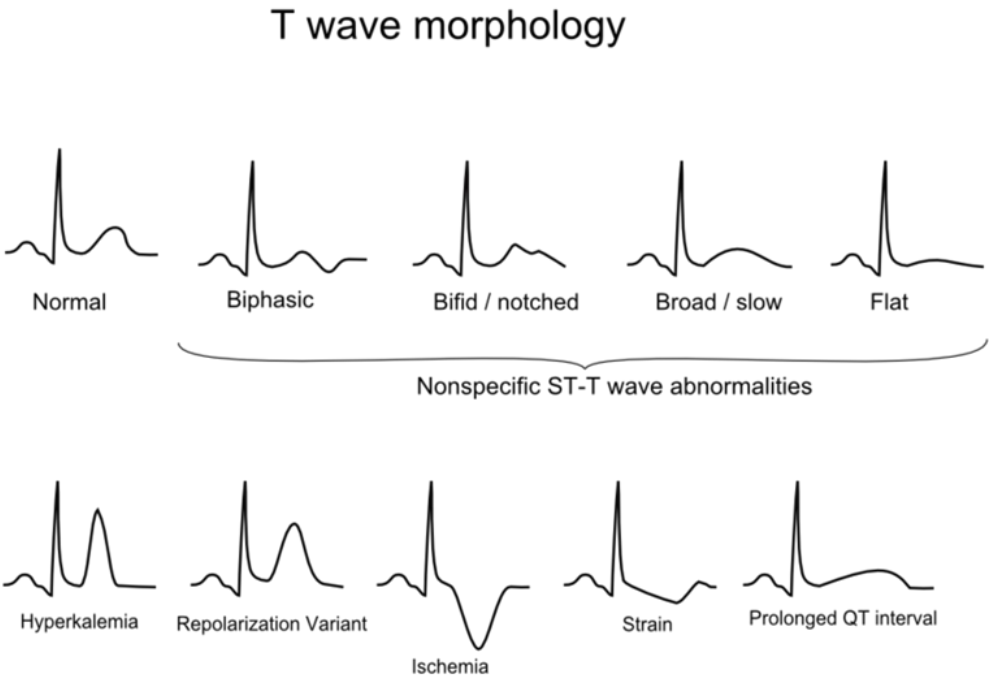
The T wave

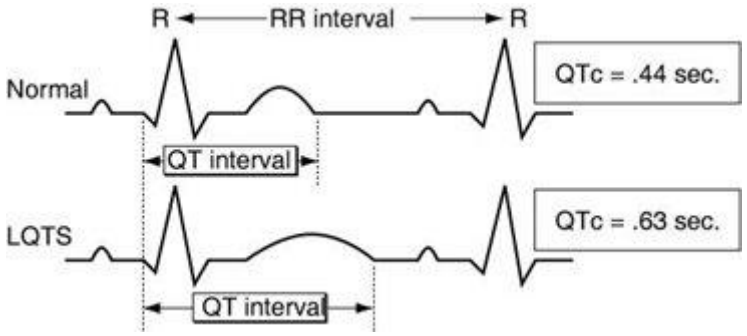
Normal T wave

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

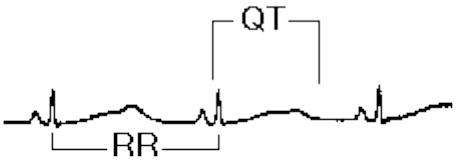


Normal QT interval

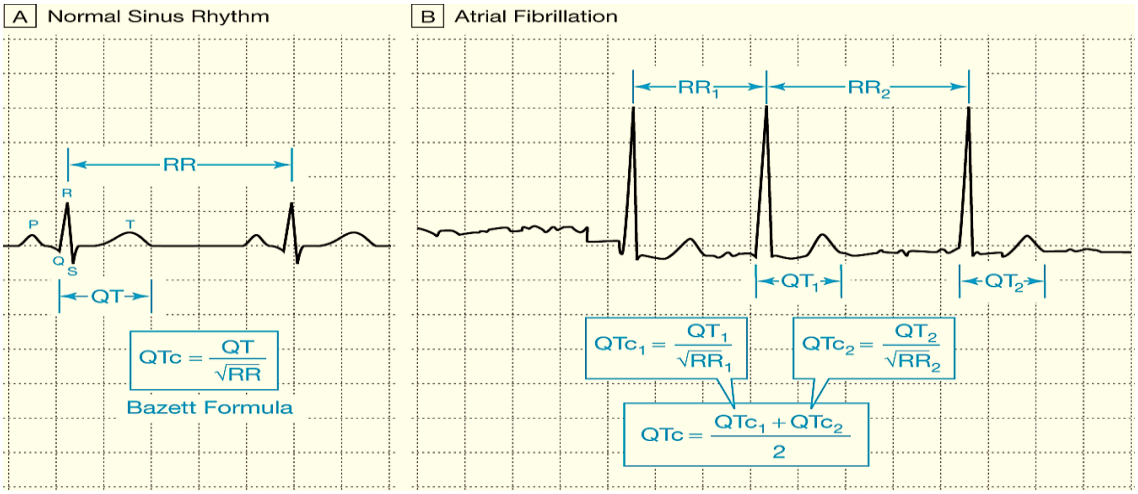
- Calculate the corrected QT interval (QTc) by dividing the QT interval by the square root of the preceeding R - R interval. Normal = 0.42 s.
- Causes of long QT interval
  - myocardial infarction, myocarditis, diffuse myocardial disease
  - hypocalcaemia, hypothyroidism
  - subarachnoid haemorrhage, intracerebral haemorrhage
  - drugs (e.g. sotalol, amiodarone)
  - hereditary
    - Romano Ward syndrome(autosomal dominant)
    - Jervill + Lange Nielson syndrome (autosomal recessive) associated with sensorineural deafness



EX:

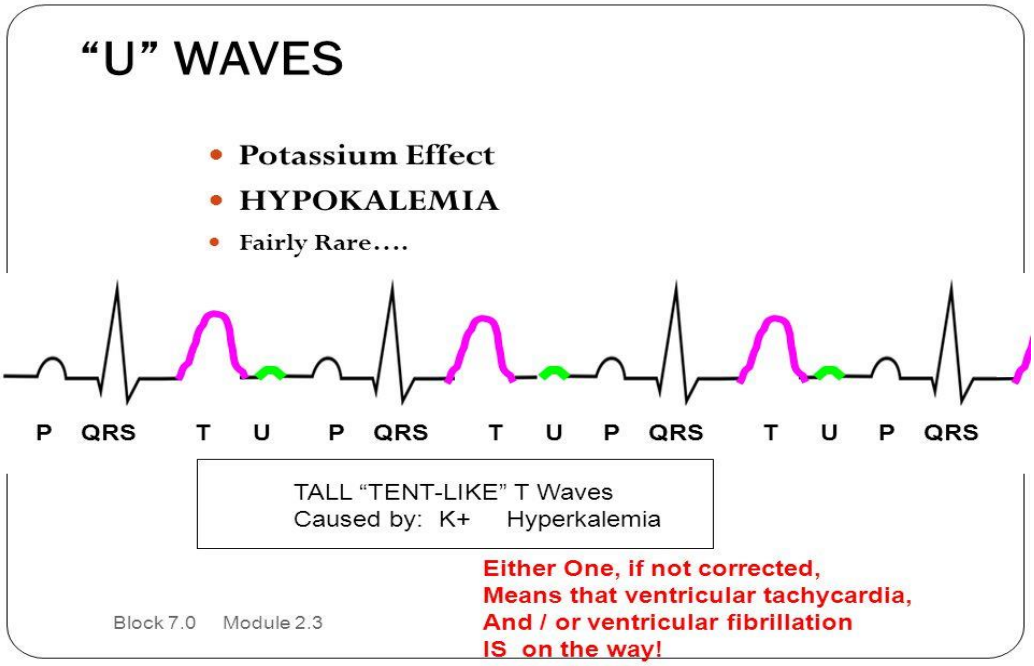


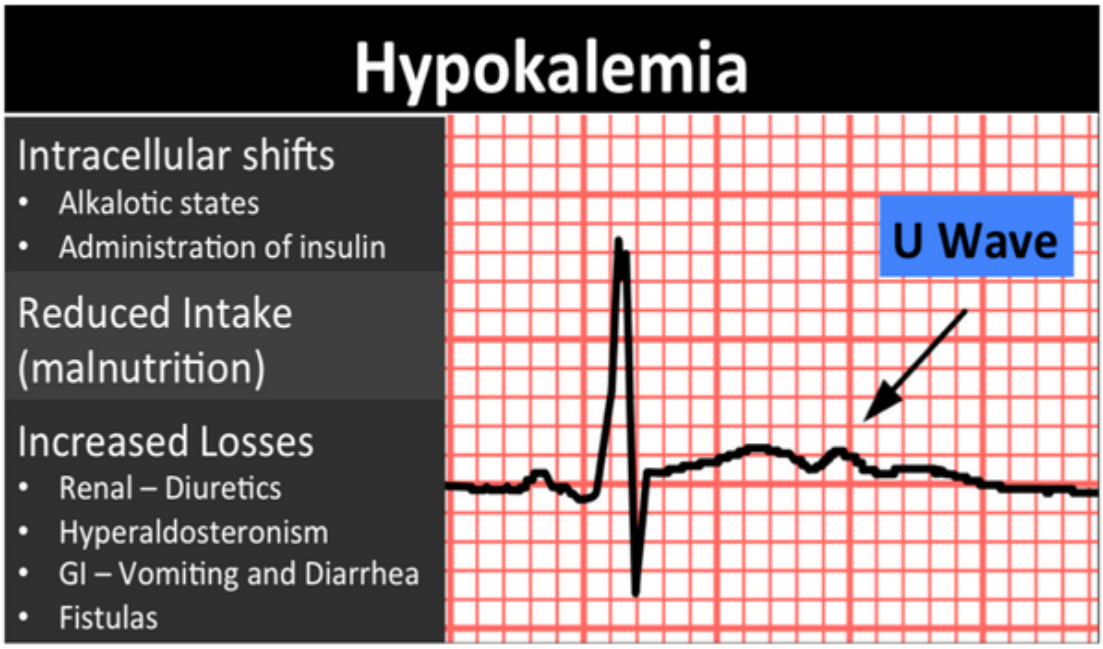
$$QTc = \frac{QT}{\sqrt{RR}} = \frac{0.71}{\sqrt{1.11}} = 0.67 \text{ seconds}$$



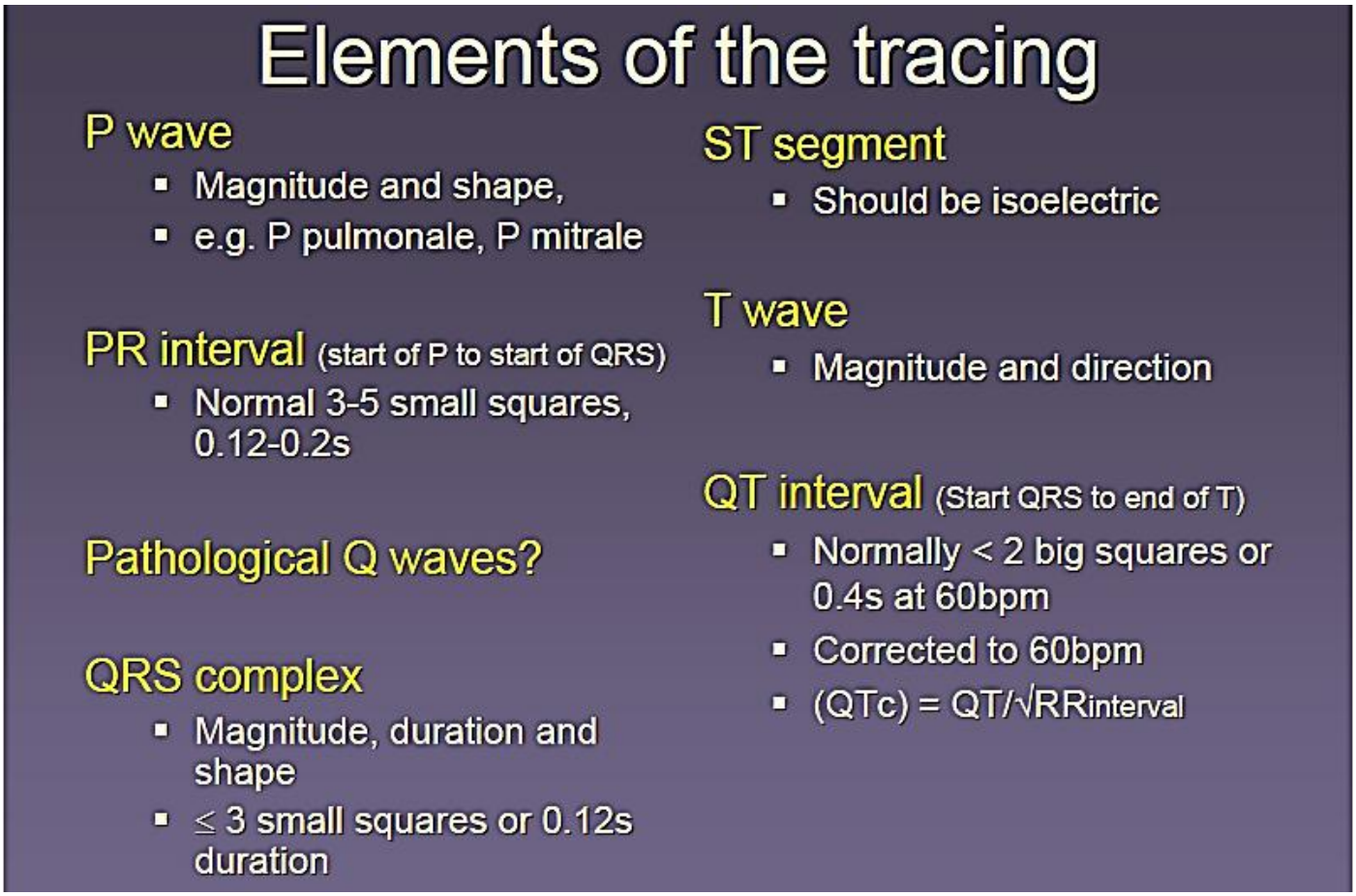
The U wave

- **U waves** occur after the T wave and are often difficult to see
- They are thought to be due to repolarization of the atrial septum
- **Prominent U waves** can be a sign of hypokalemia, hyperthyroidism





Summary



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Conduction Problems

- Always remember the pattern of conduction:

SA node ≫ AV node ≫ His Bundle ≫ bundle branches

- When looking at conduction problems, you are best to look at whichever lead **shows p waves most clearly.** This is usually lead II or V1.

- The **PR interval** the time taken for the depolarisation to spread from the SA node to the ventricular muscle. **This should not be greater than 0.2s –** i.e. 1 big square.

First degree Heart block

If the PR interval is greater than 0.2s, then we call it first degree block. All the waves will still be present; there will just be a gap between the p wave and QRS complex.

First degree heart block is not in itself very important – it can be a sign of coronary artery disease, acute rheumatic carditis, digoxin toxicity or electrolyte disturbance.

Second degree Heart block

This is where there is an **intermittent absence of QRS complexes –** and thus an indication that there is a blockage somewhere between the AV nodes and the ventricles.

There are three types of this:

Mobitz type 2 phenomenons – this is where there is a regular rhythm, and a fairly constant PR interval, but every now and again there is an absent QRS (pictured above). Basically for every QRS, there are 2 or 3 p waves.

Wenckebach phenomenon (aka Mobitz type 1) – progressive lengthening of the PR interval followed by an absence of the QRS, then a shortened PR interval and normal QRS, and the cycle begins again. The cycle is variable in length, and the **R-R interval shortens with the lengthening of the PR interval**

2:1 and 3:1 conduction – there is one normal cycle, then one cycle with an absent QRS (2:1) or there is one normal cycle, then two cycles without a QRS (3:1) – pictured below

Causes

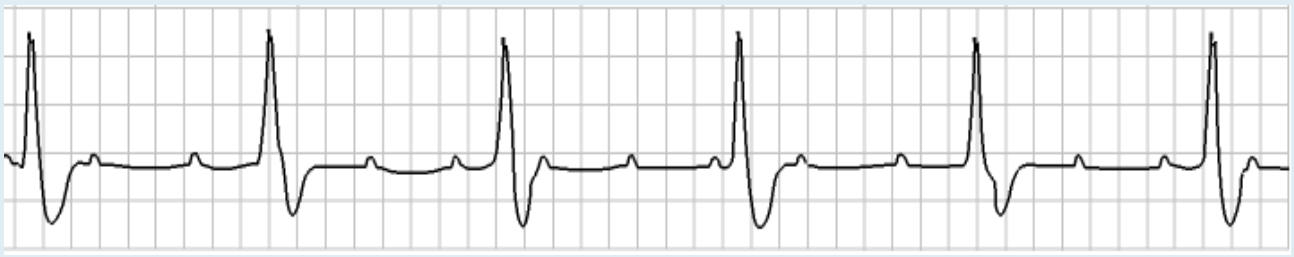
Acute – MI

Chronic – heart disease (CHD)

Third degree Heart block – complete heart block

This occurs when atrial contraction is normal, but no beats are conducted to the ventricles. **The ventricles are still excited by their own internal 'ectopic pacemaker' system!** Thus the definition of complete heart block is:

- P wave ~90/min (more p waves than QRS complexes)
- QRS ~36/min
- Variable PR intervals
- **No relationship between P wave and QRS complexes, but both are present.**
- Abnormally shaped QRS due to abnormal spread of conduction throughout ventricles
- QRS will generally be broad (~160ms – as opposed to a maximum of 120ms in a normal heart – 4 little squares as opposed to 3 little squares)
- Right axis deviation
- Escape rhythms present (more on these later)



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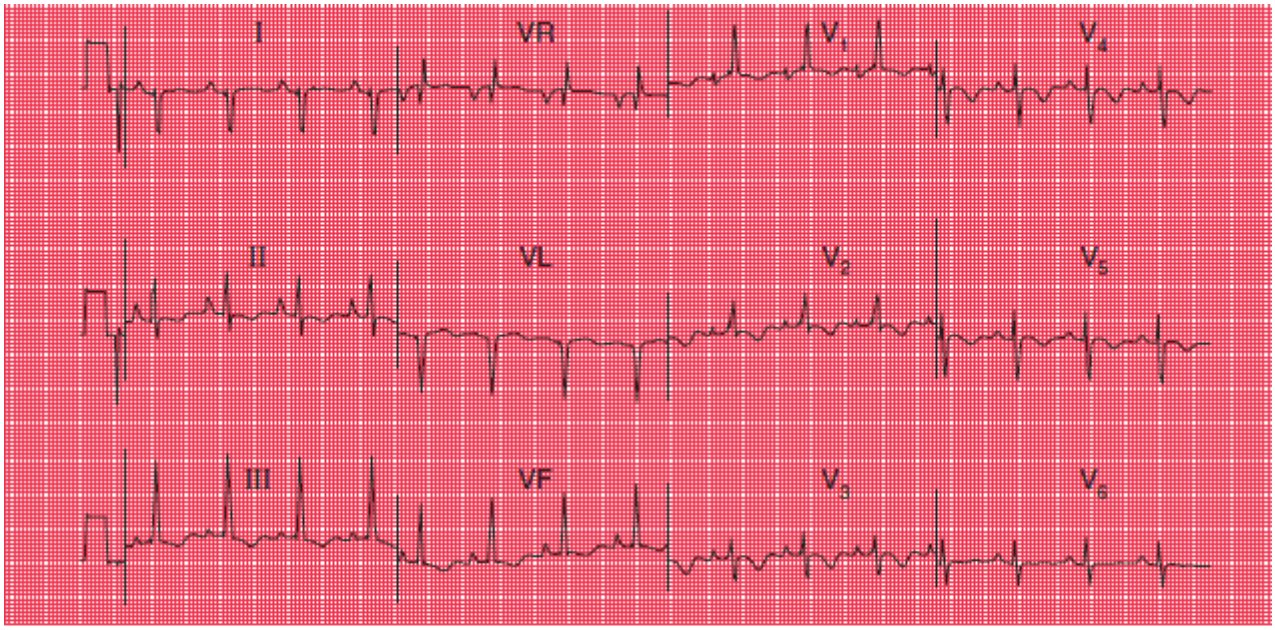
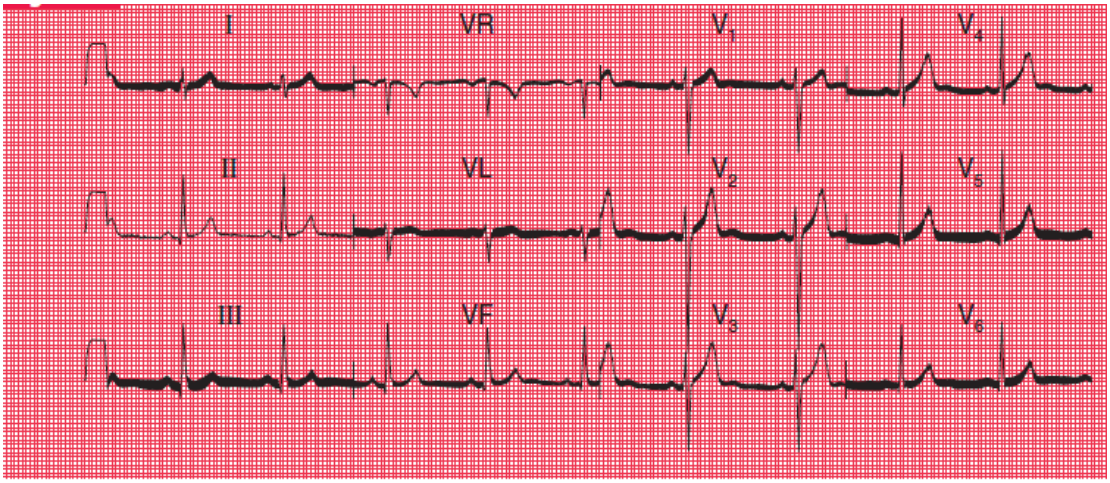
Abnormal ECG



Pulmonary embolism

Note

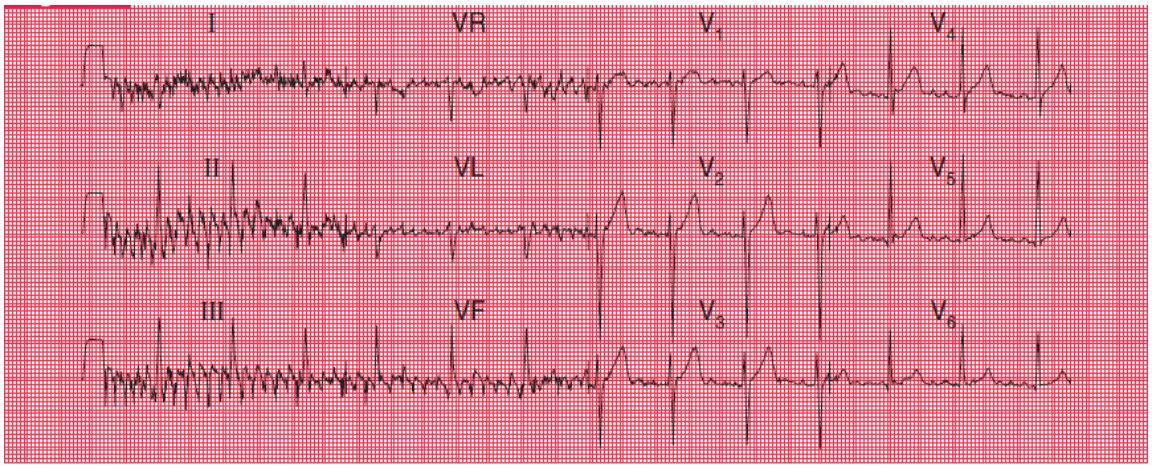
- Sinus rhythm, rate 95/min
- Right axis deviation (QRS complexes predominantly downward in lead I)
- Peaked P waves in lead II, suggesting right atrial hypertrophy
- Persistent S wave in lead V_6
- T waves inverted in leads V_1, V_5, II, III and VF
- Right bundle branch block pattern



The effect of electrical interference

Note

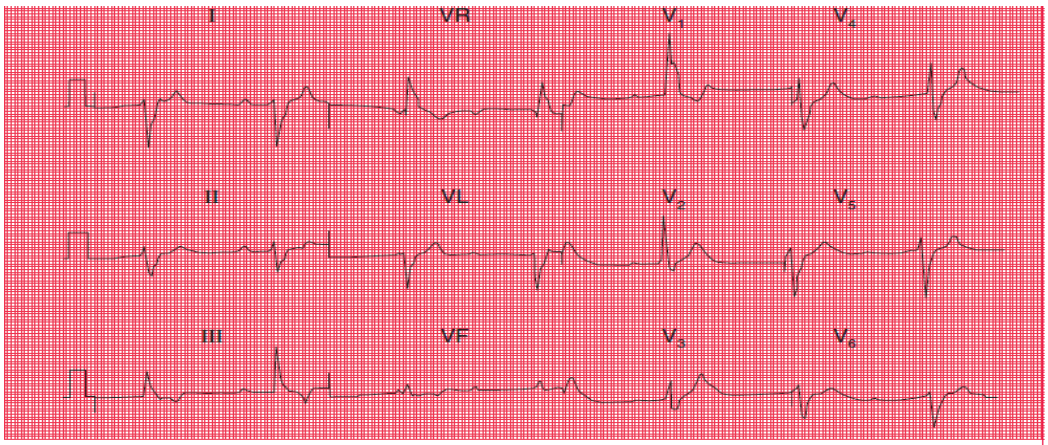
- Regular sharp high-frequency spikes, giving the appearance of a thick baseline



The effect of shivering

Note

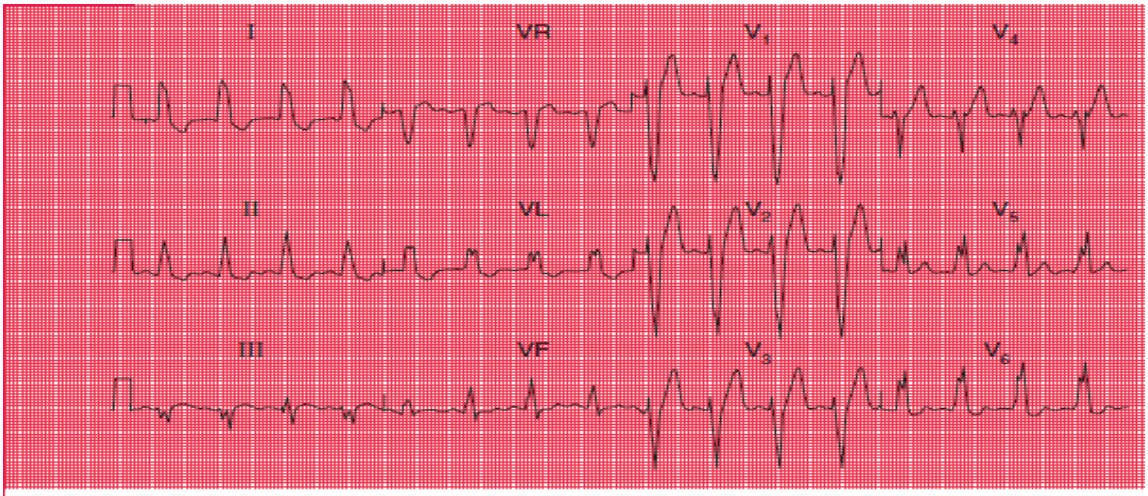
- The spikes are more exaggerated than when a patient is not relaxed
- The sharp spikes are also more synchronized, because the skeletal muscle groups are contracting together
- The effects of skeletal muscle contraction almost obliterate those of cardiac muscle contraction in leads I, II and III



Complete heart block

Note

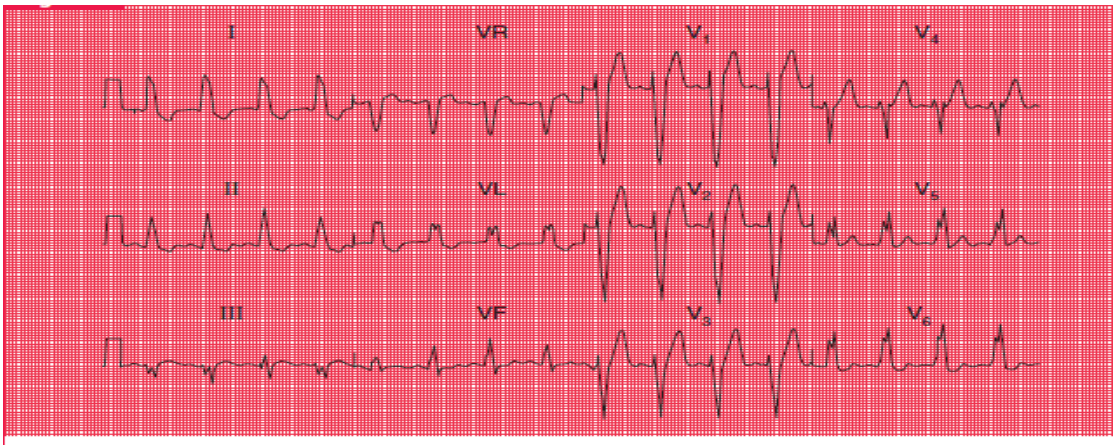
- Sinus rhythm, but no P waves are conducted
- Right axis deviation
- Broad QRS complexes (duration 160 ms)
- Right bundle branch block pattern
- In this case the cause of the block could not be determined, though in most patients it results from fibrosis of the bundle of His



Sinus rhythm with left bundle branch block

Note

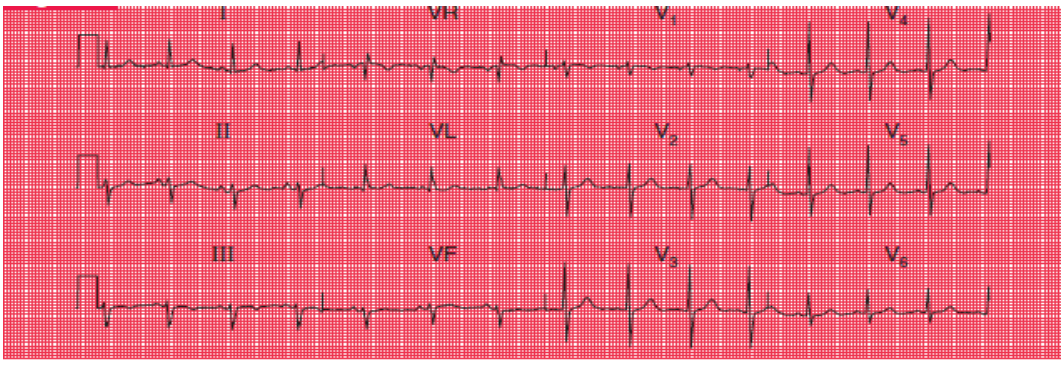
- Sinus rhythm, rate 100/min
- Normal PR interval
- Normal cardiac axis
- Wide QRS complexes (160 ms)
- M pattern in the QRS complexes, best seen in leads I, VL, V_5 and V_6
- Inverted T waves in leads I, II, VL



Sinus rhythm with left bundle branch block

Note

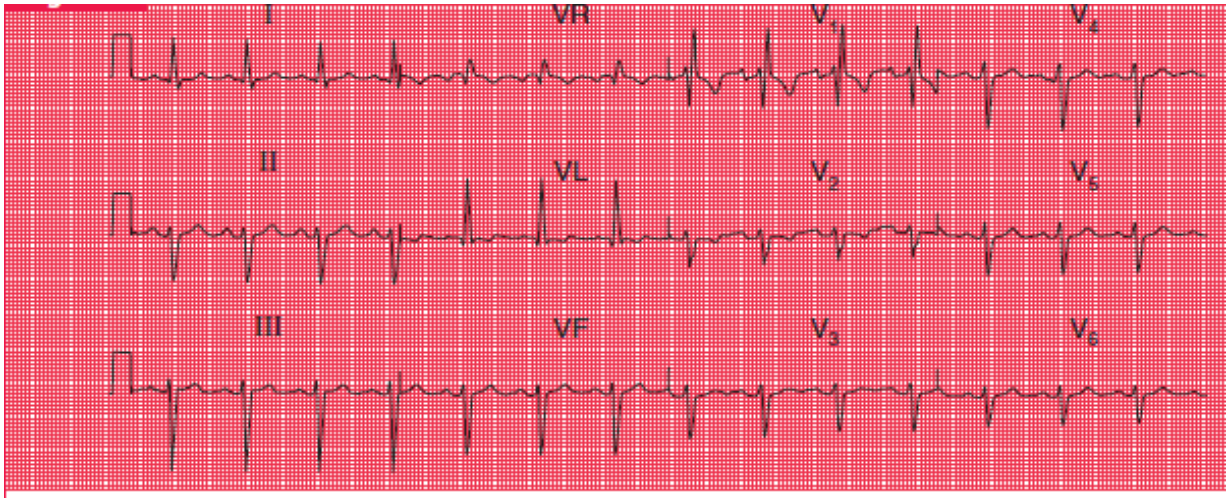
- Sinus rhythm, rate 100/min
- Normal PR interval
- Normal cardiac axis
- Wide QRS complexes (160 ms)
- M pattern in the QRS complexes, best seen in leads I, VL, V_5 and V_6
- Inverted T waves in leads I, II, VL



Sinus rhythm with left axis deviation (otherwise normal)

Note

- Sinus rhythm, rate 80/min
- Left axis deviation: QRS complex upright in lead I, but downward (dominant S wave) in leads II and III
- Normal QRS complexes, ST segments and T waves

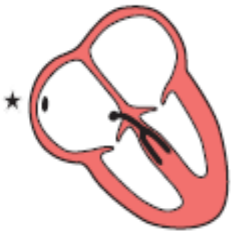


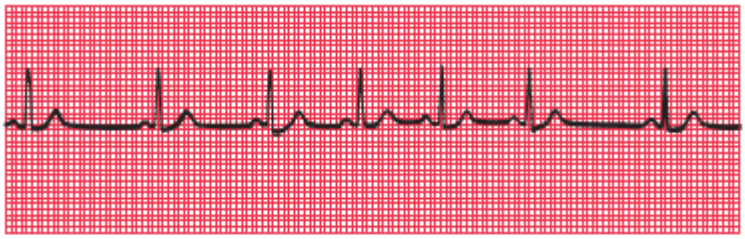
Bifascicular block

Note

- Sinus rhythm, rate 90/min
- Left axis deviation (dominant S wave in leads II and III)
- Right bundle branch block (RSR[1] pattern in lead V_1, and deep, wide S wave in lead V_6)

Sinus arrhythmia

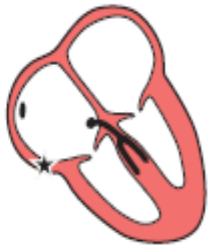


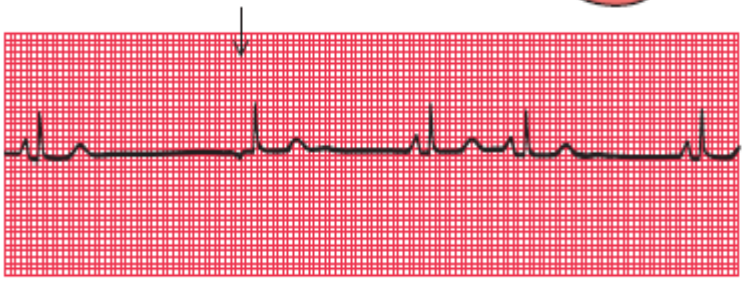


Note

- One P wave per QRS complex
- Constant PR interval
- Progressive beat-to-beat change in the R–R interval

Atrial escape

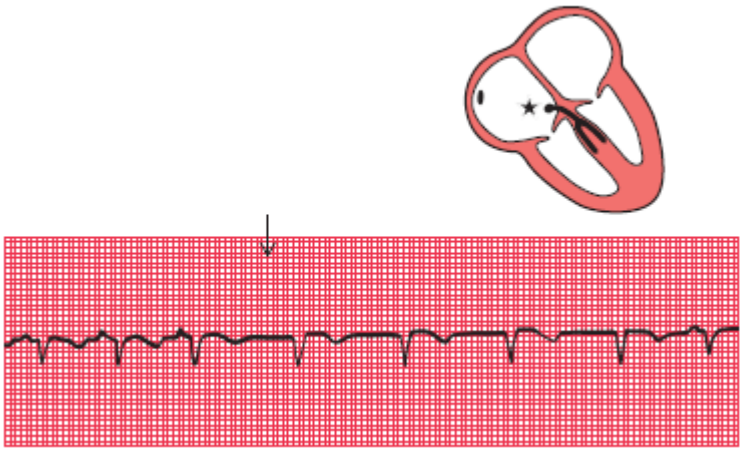




Note

- After one sinus beat the SA node fails to depolarize
- After a delay, an abnormal P wave is seen because excitation of the atrium has begun somewhere other than the SA node
- The abnormal P wave is followed by a normal QRS complex, because excitation has spread normally down the His bundle
- The remaining beats show a return to sinus arrhythmia

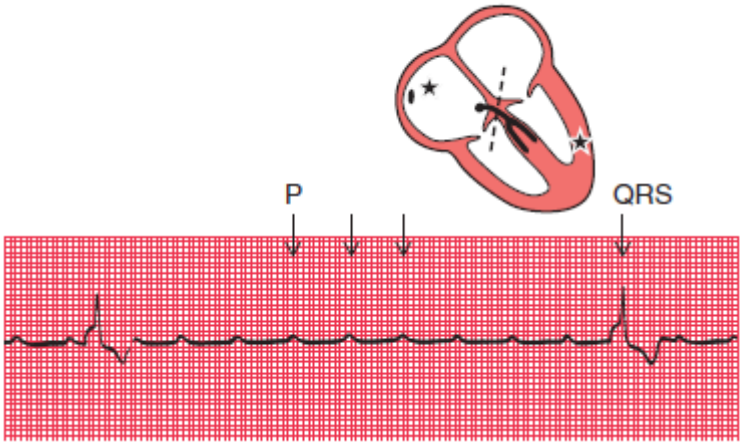
Nodal (junctional) escape



Note

- Sinus rhythm, rate 100/min
- Junctional escape rhythm (following the arrow), rate 75/min
- No P waves in junctional beats (indicates either no atrial contraction or P wave lost in QRS complex)
- Normal QRS complexes

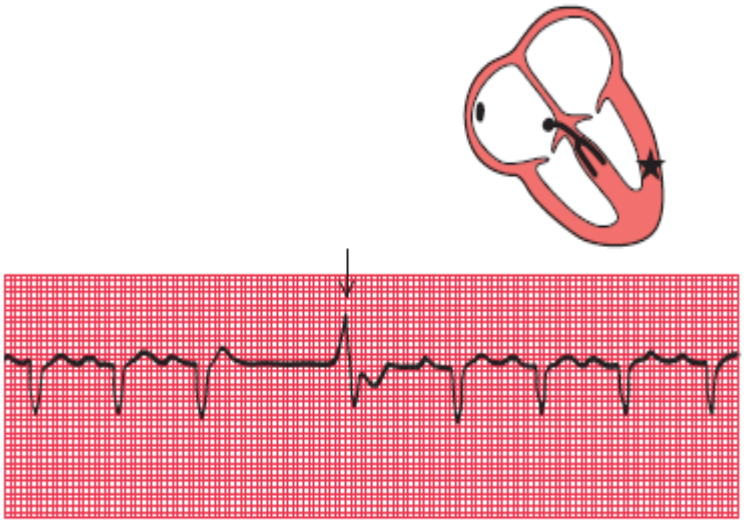
Complete heart block



Note

- Regular P waves (normal atrial depolarization)
- P wave rate 145/min
- QRS complexes highly abnormal because of abnormal conduction through ventricular muscle
- QRS complex (ventricular escape) rate 15/min
- No relationship between P waves and QRS complexes

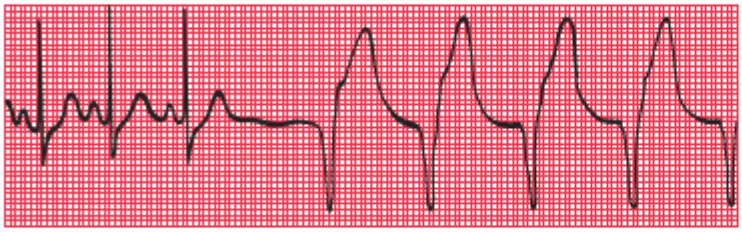
Ventricular escape



Note

- After three sinus beats, the SA node fails to discharge
- No atrial or nodal escape occurs
- After a pause there is a single wide and abnormal QRS complex (arrowed), with an abnormal T wave
- A ventricular focus controls the heart for one beat, and sinus rhythm is then restored

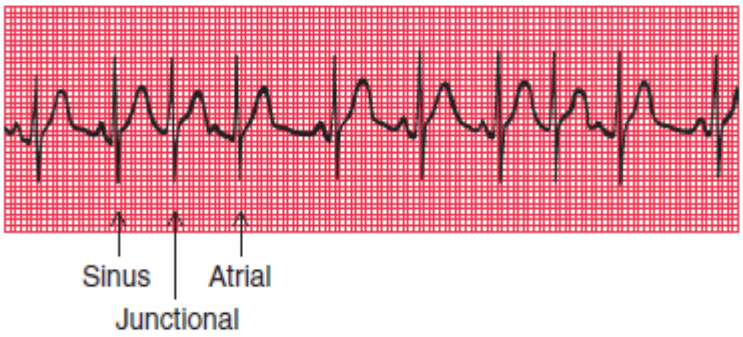
Accelerated idioventricular rhythm



Note

- After three sinus beats, the SA node fails to depolarize
- An escape focus in the ventricle takes over, causing a regular rhythm of 75/min with wide QRS complexes and abnormal T waves

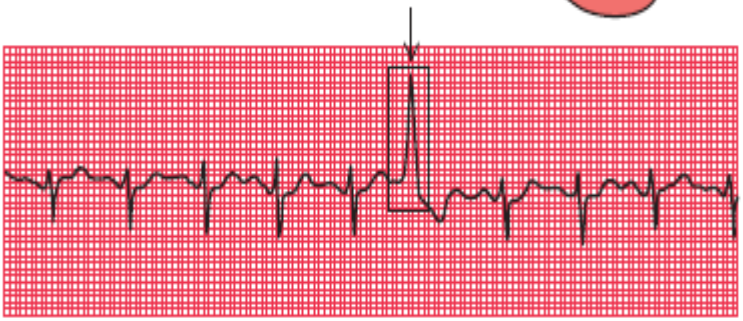
Atrial and junctional (nodal) extrasystoles



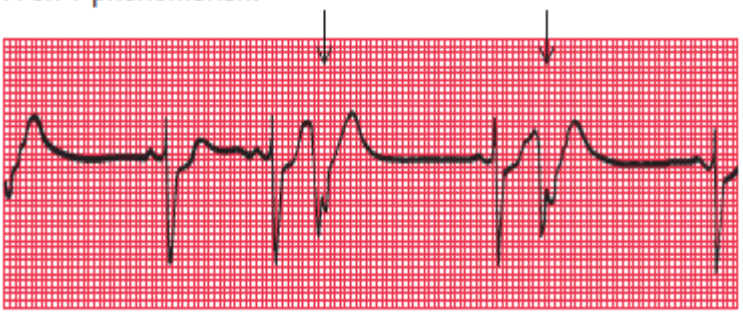
Note

- This record shows sinus rhythm with junctional and atrial extrasystoles
- A junctional extrasystole has no P wave
- An atrial extrasystole has an abnormally shaped P wave
- Sinus, junctional and atrial beats have identical QRS complexes – conduction in and beyond the His bundle is normal

Ventricular extrasystole



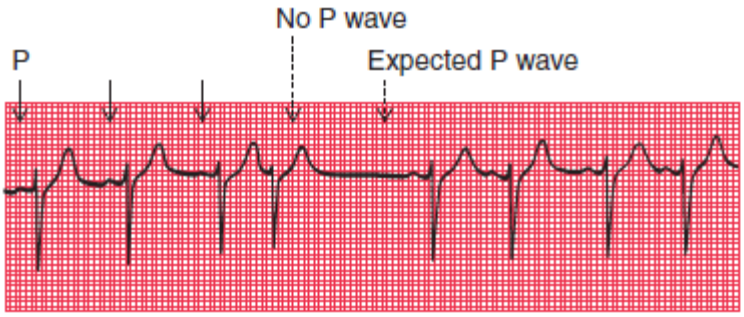
R on T phenomenon:



Note

- The upper trace shows five sinus beats, then an early beat with a wide QRS complex and an abnormal T wave: this is a ventricular extrasystole (arrowed)
- In the lower trace, the ventricular extrasystoles occur (arrowed) at the peak of the T waves of the preceding sinus beats: this is the 'R on T' phenomenon

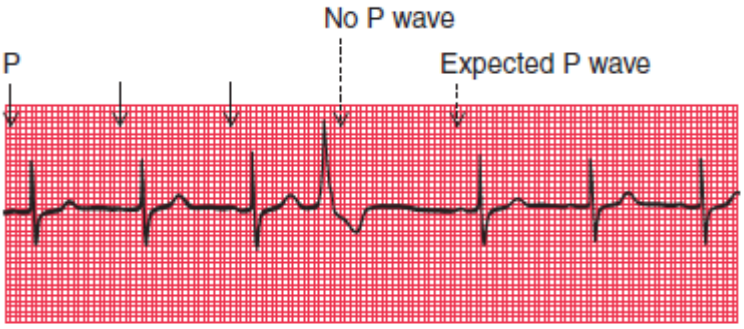
Supraventricular extrasystole



Note

- Three sinus beats are followed by a junctional extrasystole
- No P wave is seen at the expected time, and the next P wave is late

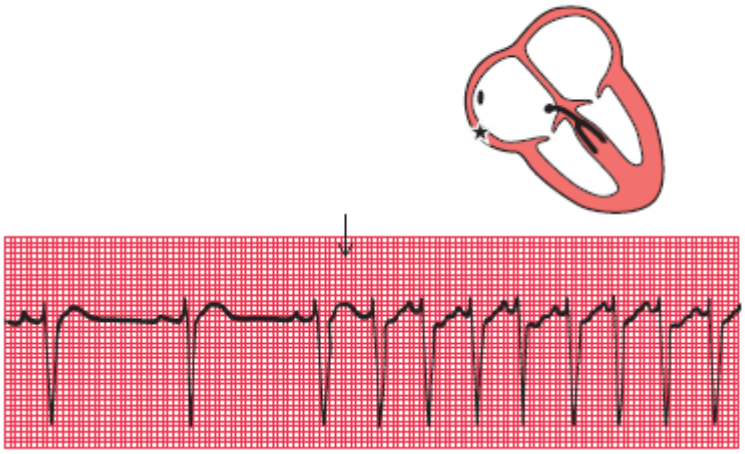
Ventricular extrasystole



Note

- Three sinus beats are followed by a ventricular extrasystole
- No P wave is seen after this beat, but the next P wave arrives on time

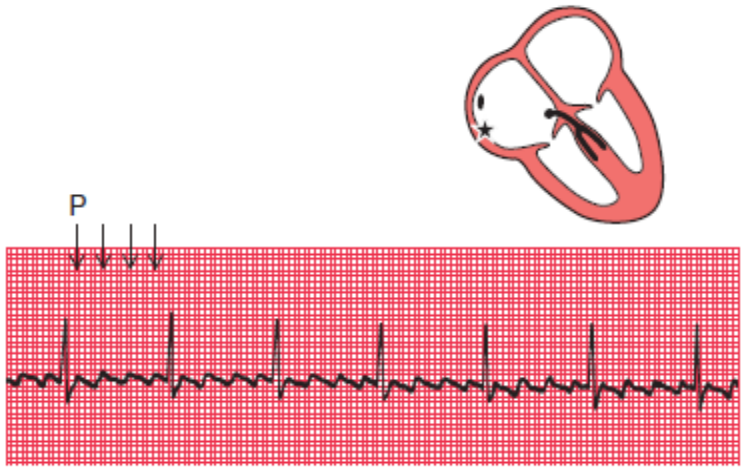
Atrial tachycardia



Note

- After three sinus beats, atrial tachycardia develops at a rate of 150/min
- P waves can be seen superimposed on the T waves of the preceding beats
- The QRS complexes have the same shape as those of the sinus beats

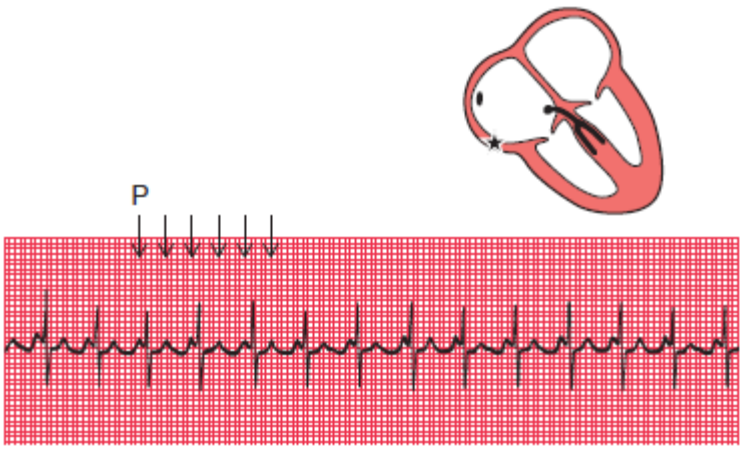
Atrial flutter



Note

- P waves can be seen at a rate of 300/min, giving a 'sawtooth' appearance
- There are four P waves per QRS complex (arrowed)
- Ventricular activation is perfectly regular at 75/min

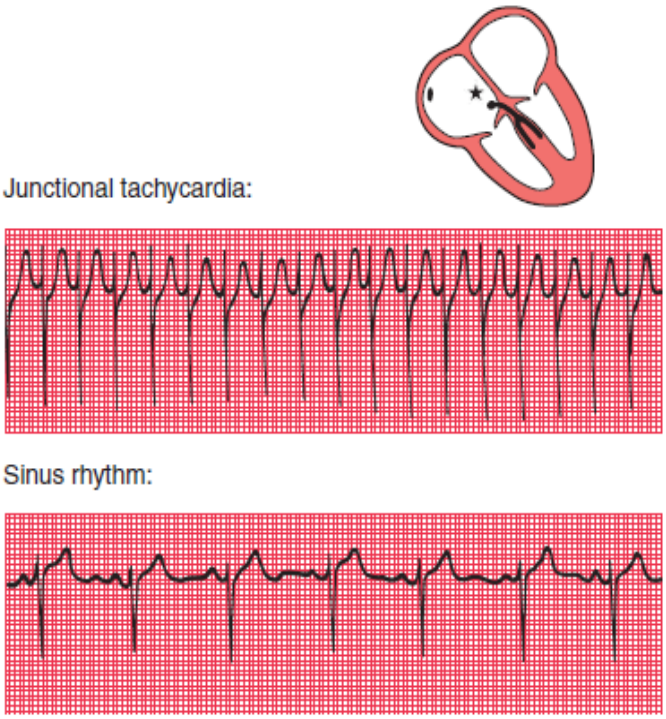
Atrial flutter with 2:1 block



Note

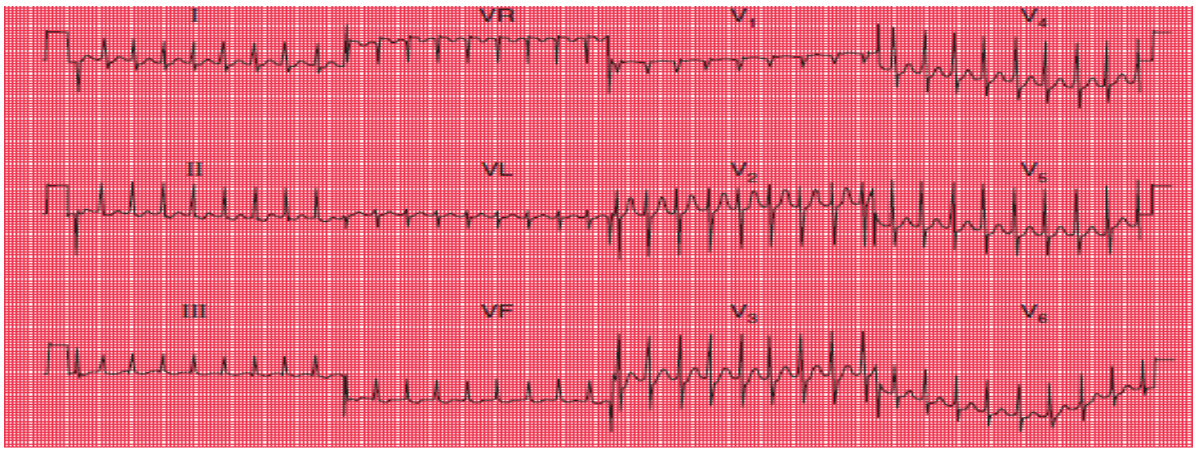
- Atrial flutter with an atrial rate of 250/min is present, and there is 2:1 block, giving a ventricular rate of 125/min
- The first of the two P waves associated with each QRS complex can be mistaken for the T wave of the preceding beat, but P waves can be identified by their regularity
- In this trace, T waves cannot be clearly identified

Junctional (nodal) tachycardia



Note

- In the upper trace there are no P waves, and the QRS complexes are completely regular
- The lower trace is from the same patient, in sinus rhythm. The QRS complexes have essentially the same shape as those of the junctional tachycardia

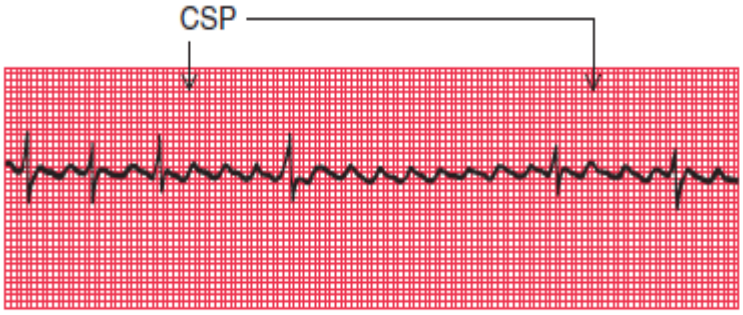


Junctional tachycardia

Note

- No P waves
- Regular QRS complexes, rate 200/min
- Narrow QRS complexes of normal shape
- Normal T waves

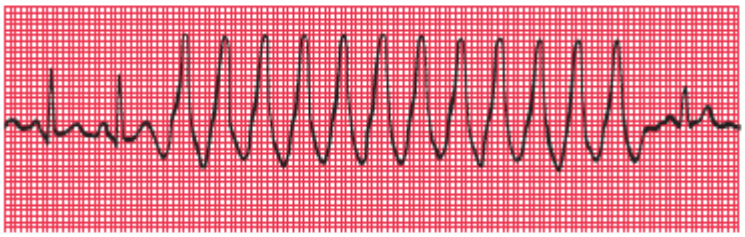
Atrial flutter with carotid sinus pressure (CSP)



Note

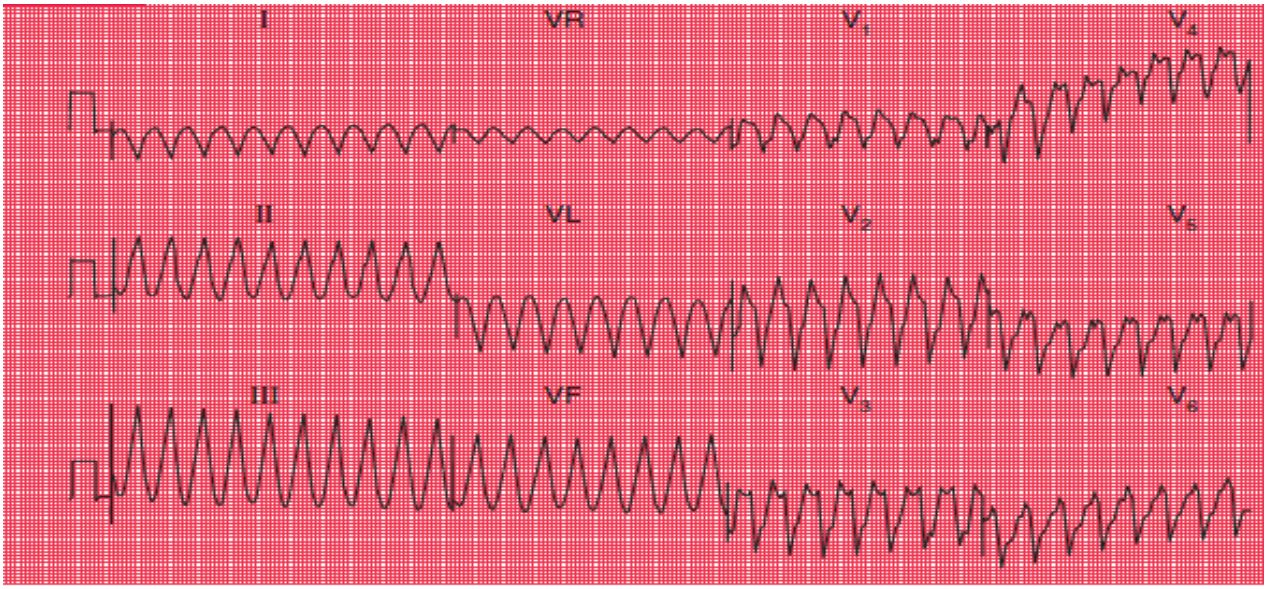
- In this case, carotid sinus pressure (applied during the period indicated by the arrows) has increased the block between the atria and the ventricles, and has made it obvious that the underlying rhythm is atrial flutter

Ventricular tachycardia



Note

- After two sinus beats, the rate increases to 200/min
- The QRS complexes become broad, and the T waves are difficult to identify
- The final beat shows a return to sinus rhythm



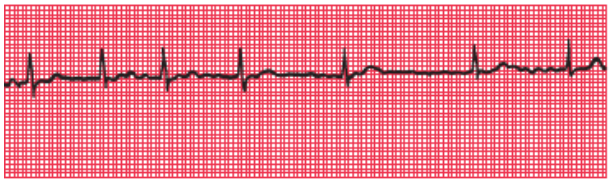
Ventricular tachycardia

Note

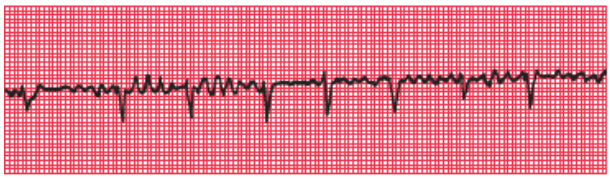
- No P waves
- Regular QRS complexes, rate 200/min
- Broad QRS complexes, duration 280 ms, with a very abnormal shape
- No identifiable T waves

Atrial fibrillation

Lead II:



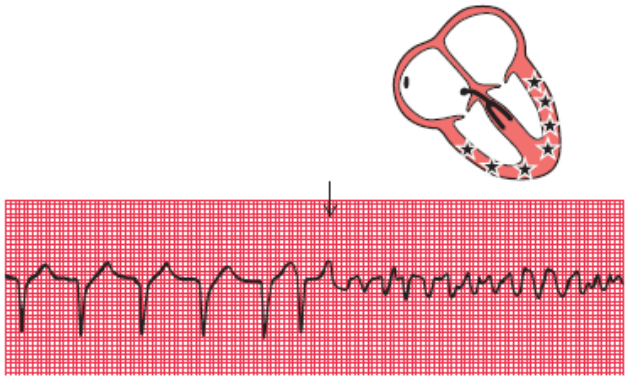
Lead V_1:

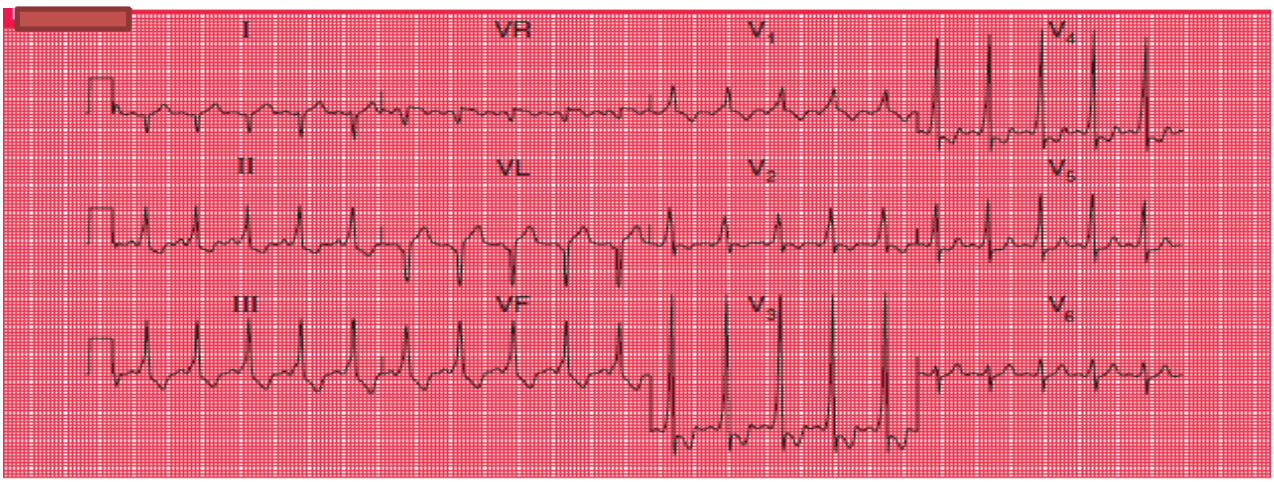


Note

- No P waves, and an irregular baseline
- Irregular QRS complexes
- Normally shaped QRS complexes
- In lead V_1, waves can be seen with some resemblance to those seen in atrial flutter – this is common in atrial fibrillation

Ventricular fibrillation



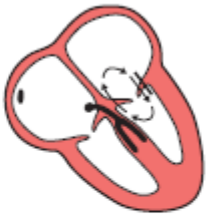


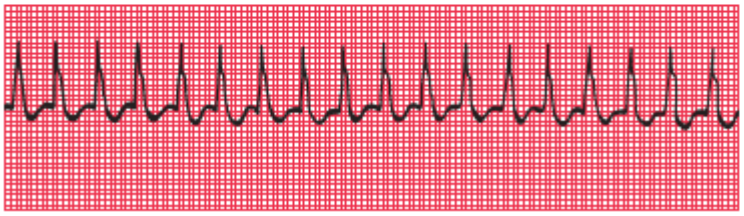
The Wolff–Parkinson–White syndrome

Note

- Sinus rhythm, rate 125/min
- Right axis deviation
- Short PR interval
- Slurred upstroke of the QRS complex, best seen in leads V_3 and V_4. Wide QRS complex due to this 'delta' wave
- Dominant R wave in lead V_1

Sustained tachycardia in the Wolff–Parkinson–White syndrome

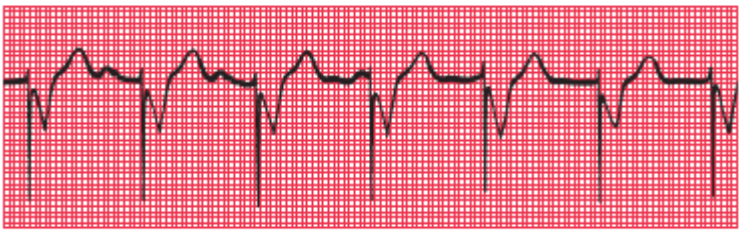




Note

- During re-entry tachycardia, no P waves can be seen

Pacemaker

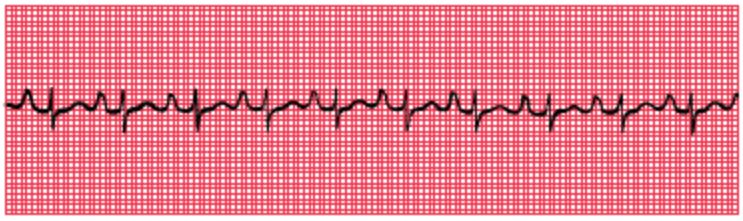


Note

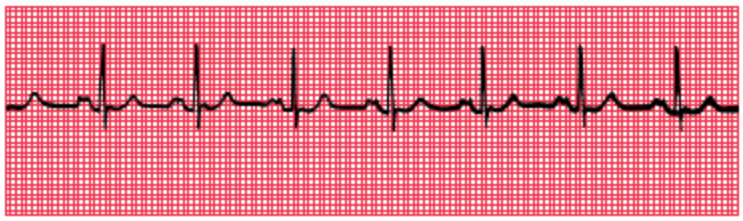
- Occasional P waves are visible, but are not related to the QRS complexes
- The QRS complexes are preceded by a brief spike, representing the pacemaker stimulus
- The QRS complexes are broad, because pacemakers stimulate the right ventricle and cause 'ventricular' beats

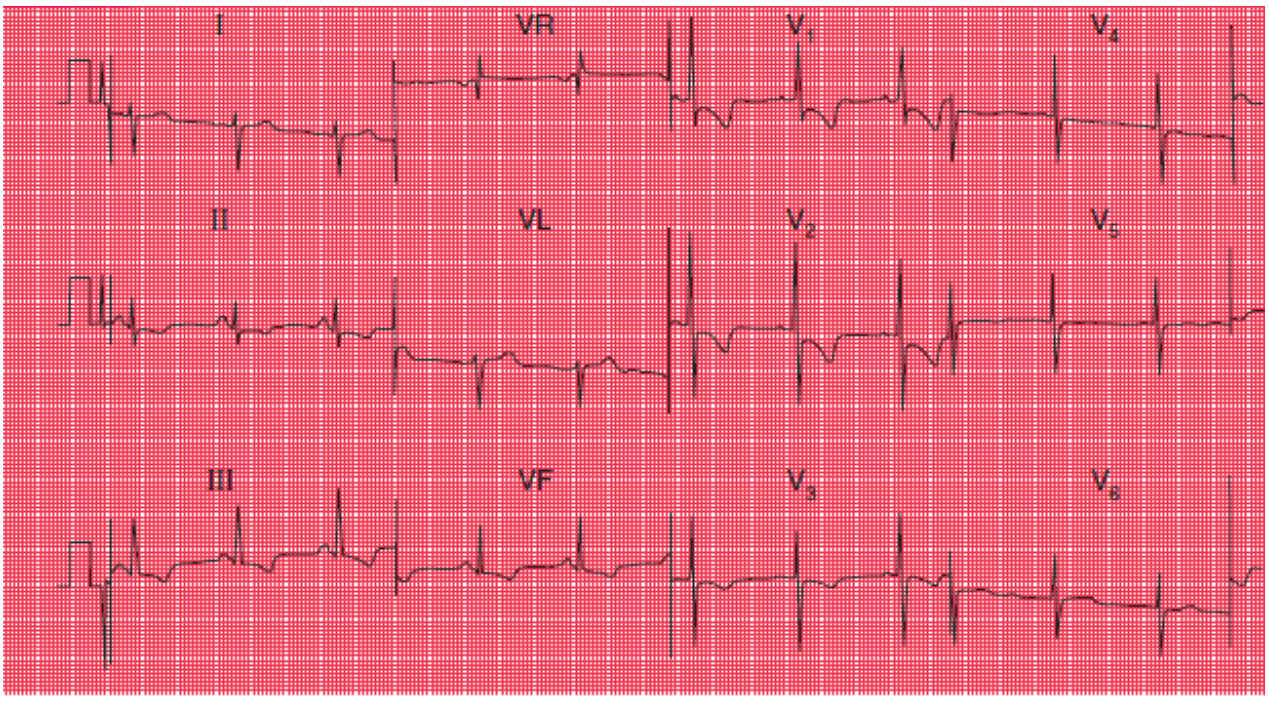
Abnormality	P wave	P:QRS ratio	QRS regularity	QRS shape	QRS rate	Rhythm
Occasional (i.e. extrasystoles)				Normal		Supraventricular
				Abnormal		Ventricular
Sustained	Present	P:QRS = 1:1	Regular	Normal	Normal	Sinus rhythm
					≥150/min	Atrial tachycardia
			Slightly irregular	Normal	Normal	Sinus arrhythmia
					Slow	Atrial escape
		More P waves than QRS complexes	Regular	Normal	Fast	Atrial tachycardia with block
					Slow	Second degree heart block
				Abnormal	Slow	Complete heart block
	Absent	n/a	Regular	Normal	Fast	Junctional tachycardia
					Slow	Junctional escape
				Abnormal	Fast	Junctional tachycardia with bundle branch block or ventricular tachycardia
			Irregular	Normal	Any speed	Atrial fibrillation
				Abnormal	Any speed	Atrial fibrillation and bundle branch block
		QRS complexes absent				Ventricular fibrillation or standstill

Right atrial hypertrophy



Left atrial hypertrophy

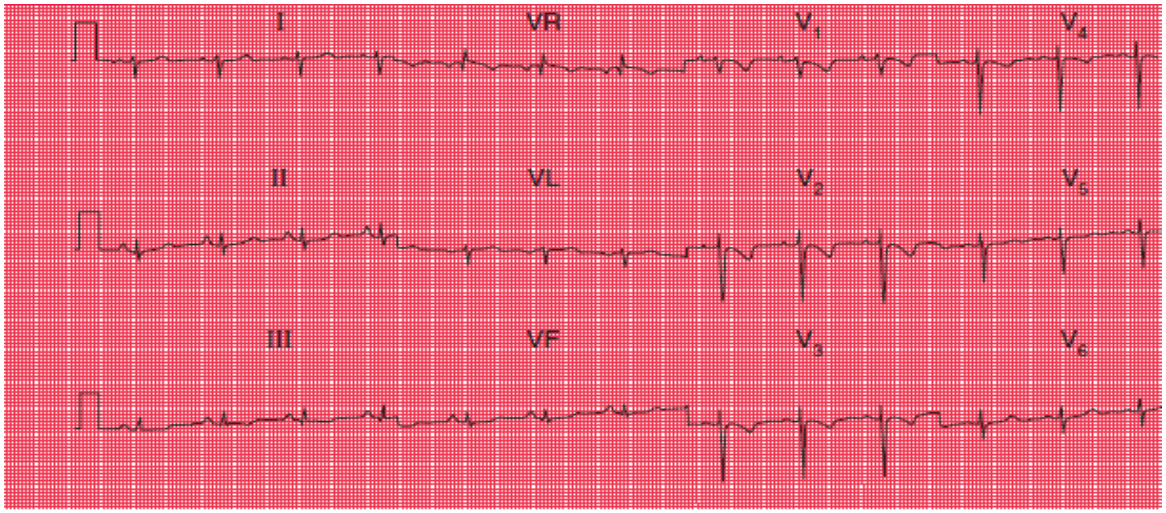




Severe right ventricular hypertrophy

Note

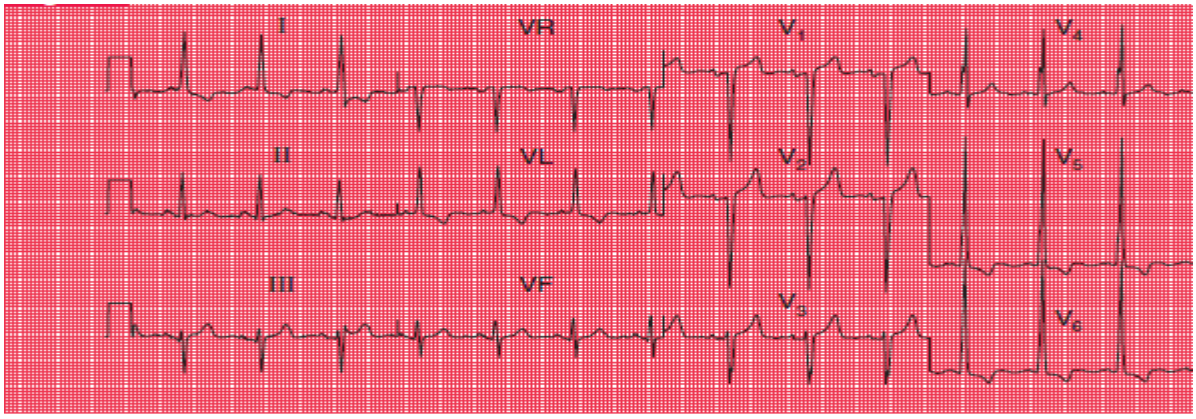
- Sinus rhythm, rate 63/min
- Right axis deviation (deep S waves in lead I)
- Dominant R waves in lead V_1
- Deep S waves in lead V (clockwise rotation)
- Inverted T waves in leads II, III, VF and V_1–V_3
- Flat T waves in leads V_4–V_5



Pulmonary embolism

Note

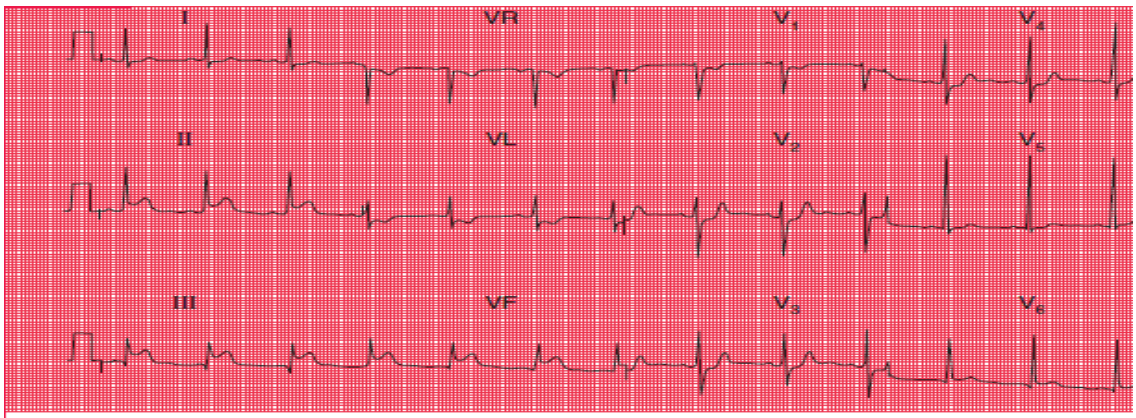
- Sinus rhythm, rate 75/min
- Right axis deviation
- Peaked P waves, especially in lead II
- Persistent S wave in lead V_6
- T wave inversion in leads V_1–V_4



Left ventricular hypertrophy

Note

- Sinus rhythm, rate 83/min
- Normal axis
- Tall R waves in leads V_5–V_6 (R wave in lead V_5, 40 mm) and deep S waves in leads V_1–V_2
- Inverted T waves in leads I, VL and V_5–V_6

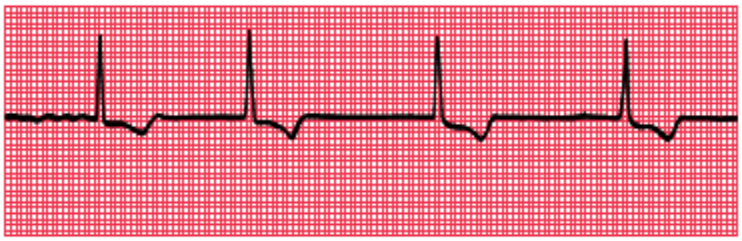


Acute inferior infarction; lateral ischaemia

Note

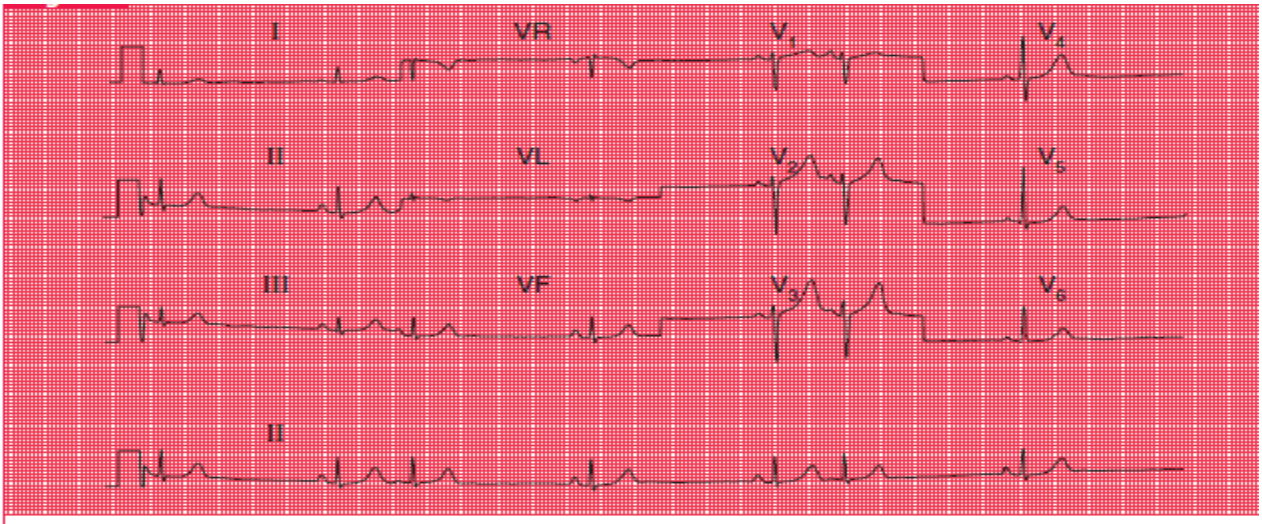
- Sinus rhythm, rate 70/min
- Normal axis
- Q waves in leads III and VF
- Normal QRS complexes
- Raised ST segments in leads II, III and VF
- Inverted T waves in lead VL (abnormal) and in lead V_1 (normal)

Digoxin effect



Note

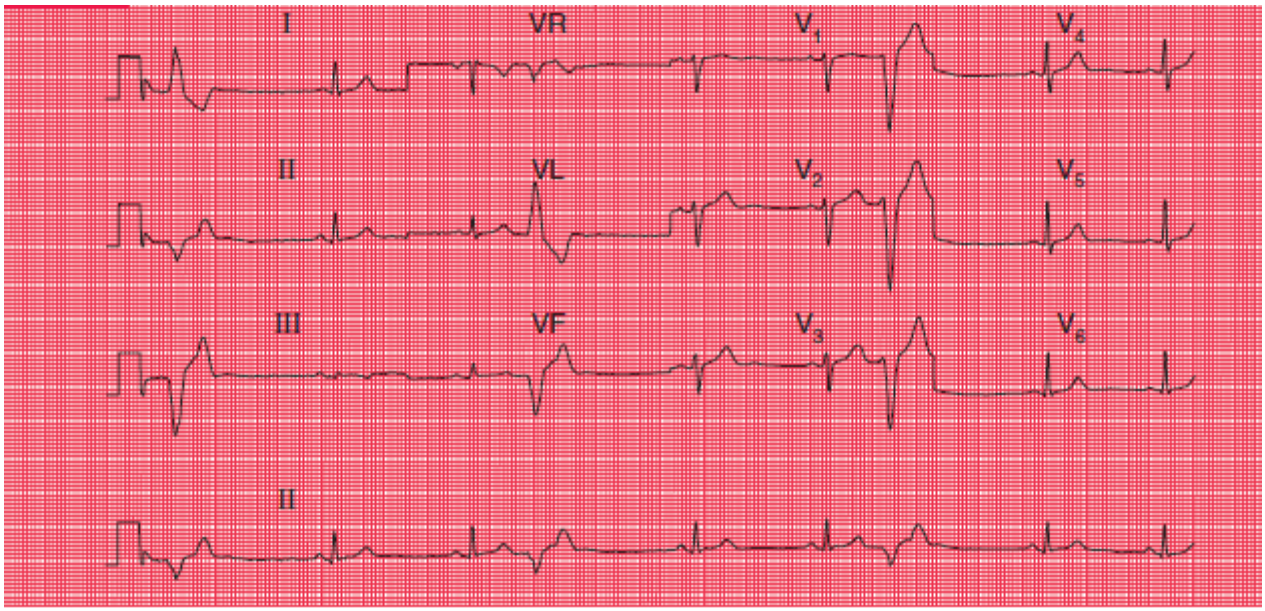
- Atrial fibrillation
- Narrow QRS complexes
- Downward-sloping ST segments ('reversed tick')
- Inverted T waves



Atrial extrasystoles

Note

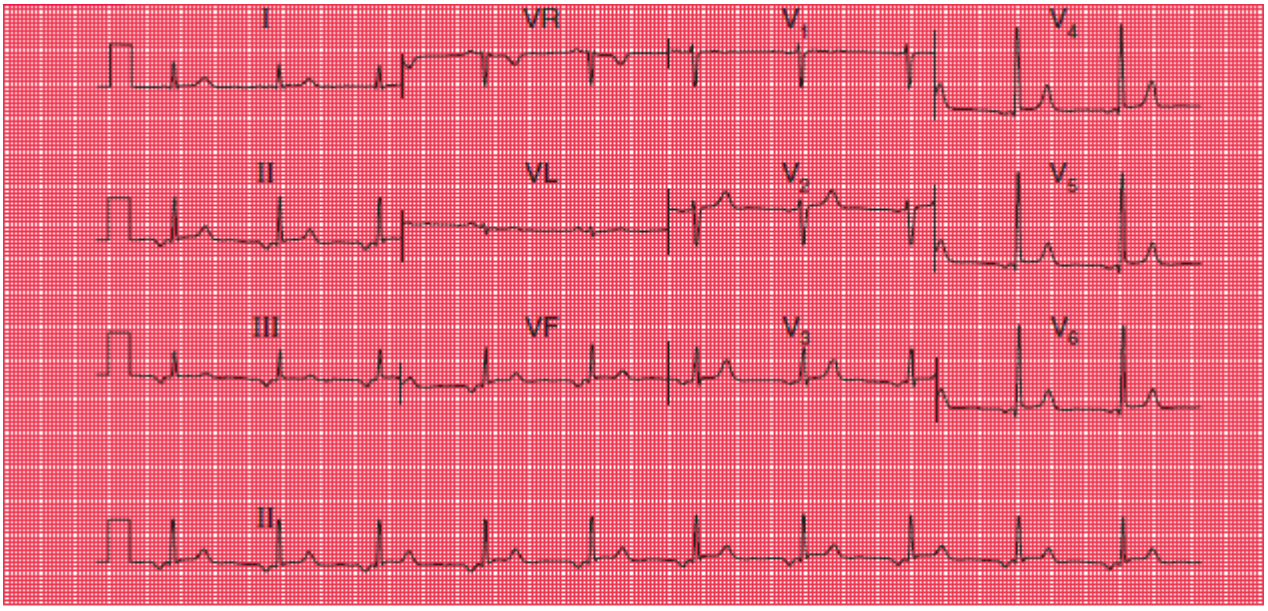
- Sinus rhythm; rate as judged from adjacent sinus beats is about 35/min
- The overall heart rate, calculated including the extrasystoles, is about 45/min
- Extrasystoles are identified by early P waves that are differently shaped compared with those associated with the sinus beats
- The QRS complexes and T waves are the same in the sinus and atrial beats



Ventricular extrasystoles

Note

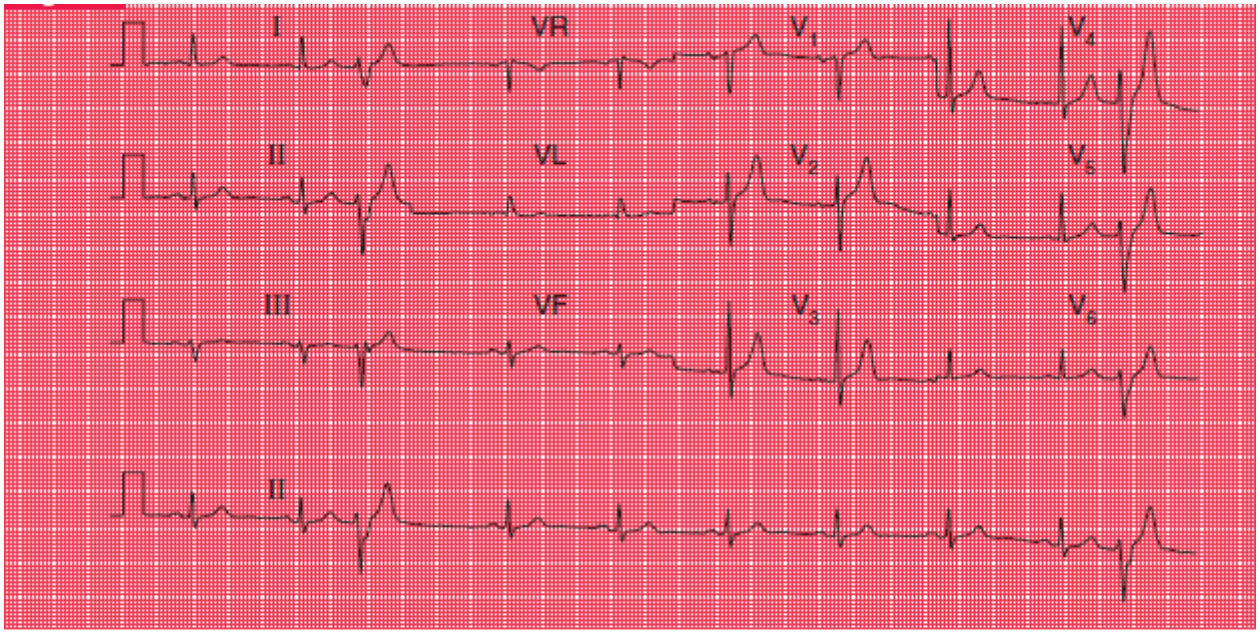
- Sinus rhythm, rate 50/min
- Frequent ventricular extrasystoles, identified by their early occurrence without preceding P waves, and by their wide and abnormal QRS complex and differently shaped T wave compared with the sinus beats
- In the sinus beats the QRS complexes and T waves are normal



Ectopic atrial rhythm

Note

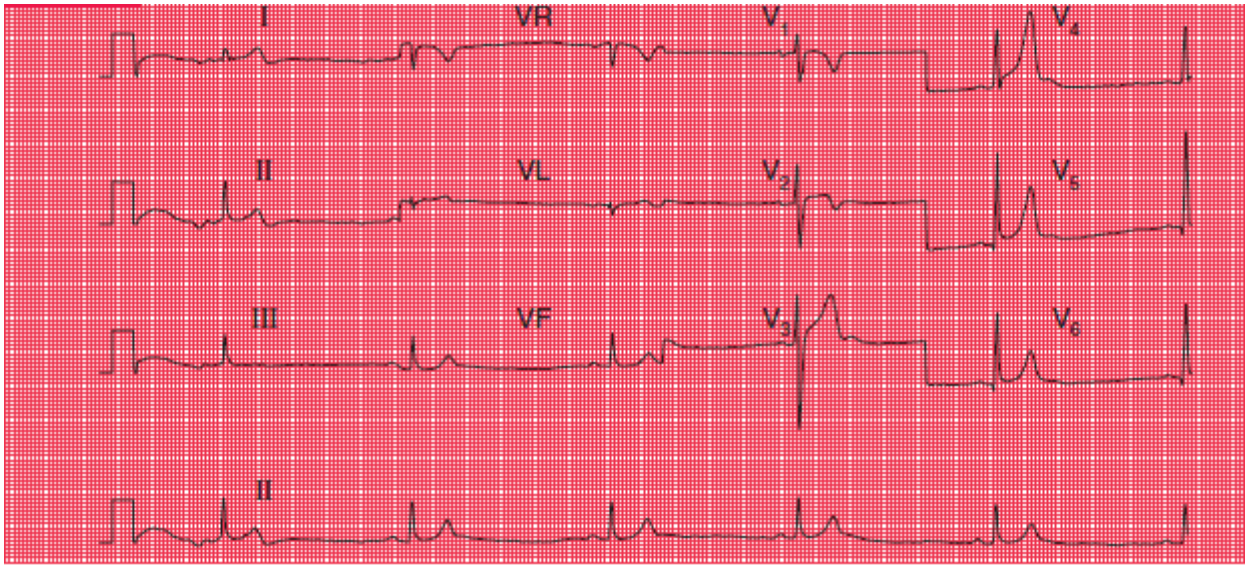
- Regular rhythm with inverted P waves in most leads, indicating that an atrial focus controls the heart rate
- The PR interval is at the lower end of normal, at 140 ms
- Heart rate 60/min
- The QRS complexes, ST segments and T waves are all normal



Bifid P waves

Note

- Sinus rhythm, rate 60/min
- There are two ventricular extrasystoles
- In leads V_2, V_3 and V_4 the P wave is 'bifid'. This can be a sign of left atrial hypertrophy, but is often seen in normal ECGs
- In the sinus beats the QRS complexes, ST segments and T waves are normal



Normal ECG with prominent U waves

Note

- The ECG appearance at the beginning is due to movement of the patient
- Sinus rhythm, rate 35/min (sinus bradycardia)
- Normal axis
- Normal QRS complexes
- Peaked T waves in leads V_4–V_6
- Prominent U waves in leads V_3–V_5

LOOK

REMINDERS

THE NORMAL ECG

Limits of normal durations

- PR interval: 220 ms.
- QRS complex duration: 120 ms.
- QT_c interval: 450 ms.

Rhythm

- Sinus arrhythmia.
- Supraventricular extrasystoles are always normal.

The cardiac axis

- Normal axis: QRS complexes predominantly upward in leads I, II and III; still normal if the QRS complex is downward in lead III.
- Minor degrees of right and left axis deviation are within the normal range.

QRS complex

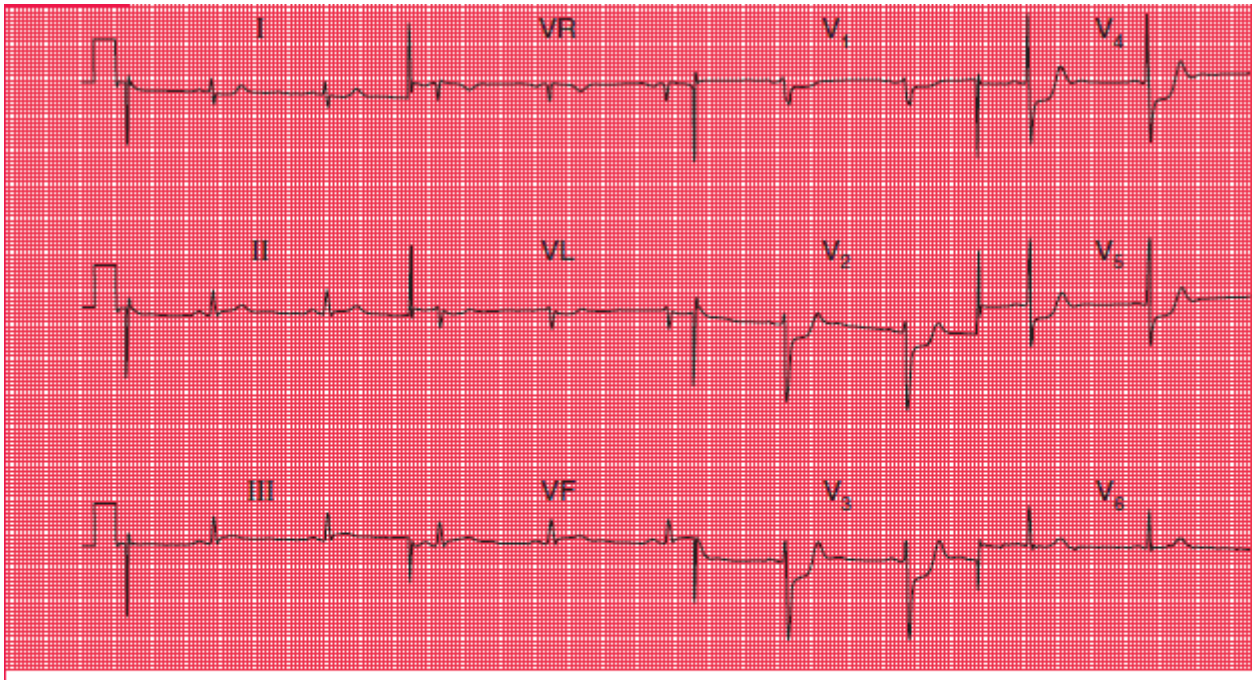
- Small Q waves are normal in leads I, VL and V_6 (septal Q waves).
- RSR[1] pattern in lead V_1 is normal if the duration is less than 120 ms (partial right bundle branch block).
- R wave is smaller than S wave in lead V_1.
- R wave in lead V_6 is less than 25 mm.
- R wave in lead V_6 plus S wave in lead V_1 is less than 35 mm.

ST segment

- Should be isoelectric.

T wave

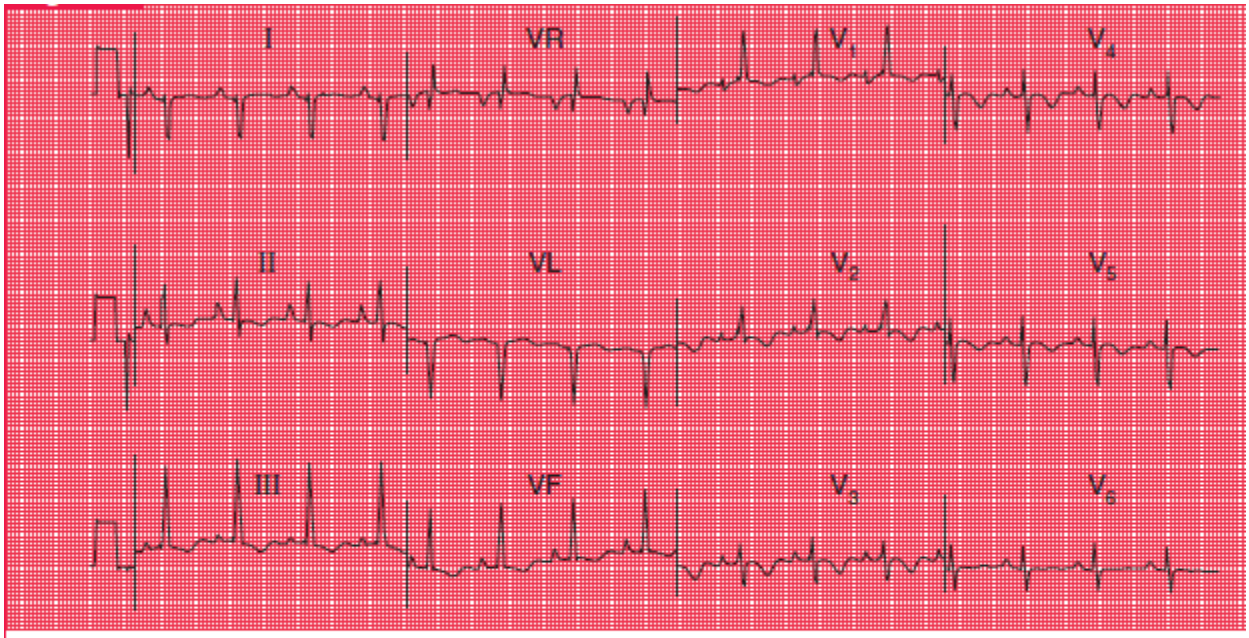
- May be inverted in:
  - lead III
  - lead VR
  - lead V_1
  - leads V_2 and V_3, in black people.



ST segment depression in unstable angina

Note

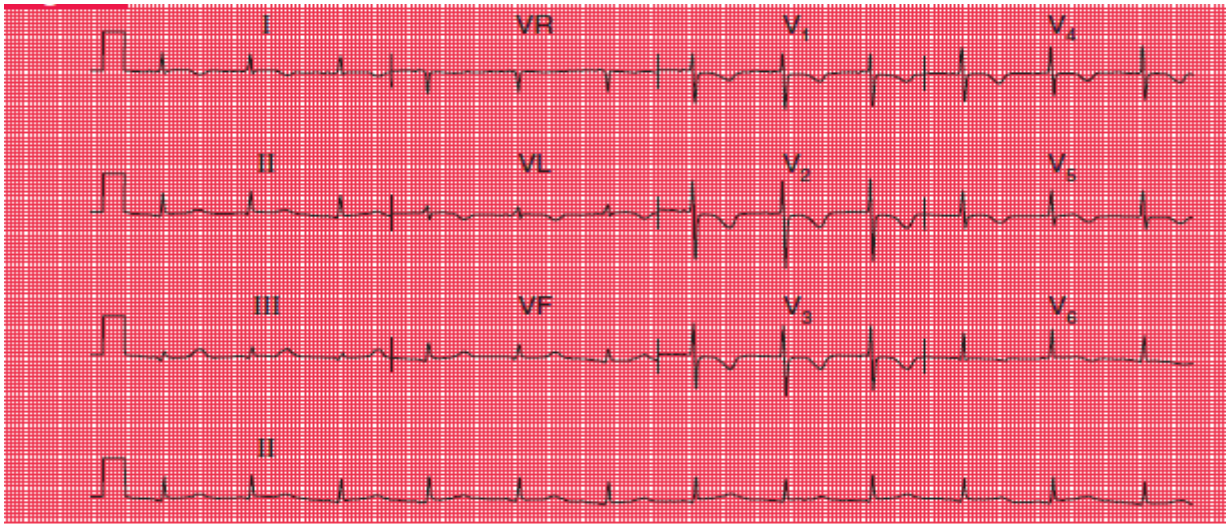
- Sinus rhythm, rate 60/min
- Normal axis
- Normal QRS complexes
- ST segments depressed horizontally in leads V_3–V_5
- Normal T waves



Pulmonary embolism

Note

- Sinus rhythm, rate 95/min
- Right axis deviation (QRS complexes predominantly downward in lead I)
- Peaked P waves in lead II, suggesting right atrial hypertrophy
- Persistent S wave in lead V_6
- T waves inverted in leads V_1, V_5, II, III and VF
- Right bundle branch block pattern



Prolonged QT interval

Note

- Sinus rhythm, rate 75/min
- Normal axis
- P wave difficult to see in some leads, but most obvious in leads I and VL
- Normal QRS complexes
- T wave inversion in leads I, VL and V_1–V_6
- QT interval 480 ms, QT_c interval 520 ms
- In this patient the prolonged QT interval was due to amiodarone

REMINDERS

CONDUCTION PROBLEMS

First degree block

- One P wave per QRS complex.
- PR interval greater than 200 ms.

Second degree block

- Wenckebach (Mobitz type 1): progressive PR lengthening then a nonconducted P wave, and then repetition of the cycle.
- Mobitz type 2: occasional nonconducted beats.
- 2:1 (or 3:1) block: two (or three) P waves per QRS complex, with a normal P wave rate.

Third degree (complete) block

- No relationship between P waves and QRS complexes.
- Usually, wide QRS complexes.
- Usual QRS complex rate less than 50/min.
- Sometimes, narrow QRS complexes, rate 50–60/min.

Right bundle branch block

- QRS complex duration greater than 120 ms.
- RSR^1 pattern.
- Usually, dominant R^1 wave in lead V_1.
- Inverted T waves in lead V_1, and sometimes in leads V_2–V_3.
- Deep and wide S waves in lead V_6.

Left anterior hemiblock

- Marked left axis deviation – deep S waves in leads II and III, usually with a slightly wide QRS complex.

Left bundle branch block

- QRS complex duration greater than 120 ms.
- 'M' pattern in lead V_6, and sometimes in leads V_4–V_5.
- No septal Q waves.
- Inverted T waves in leads I, VL, V_5–V_6 and, sometimes, V_4.

Bifascicular block

- Left anterior hemiblock and right bundle branch block (see above).

Summary Table

References

1- ECGs by Example, 3rd Edition, **Churchill Livingstone.**
2- The ECG Made Easy, 8th EDITION, **John R. Hampton.**
3- 150 ECG problems, 2nd Edition, **John R. Hampton.**
4- **WEB SITES:**
   - nottingham.ac.uk
   - almostadoctor.co.uk ,(ECG Abnormalities)
   - advancedpctuneup.com

With Best Wishes;

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2017