

# CLINICAL INVESTIGATIONS

NOTES

**FOURTH EDITION**

**PRE-SUMMARIZED  
READY-TO-STUDY  
HIGH-YIELD NOTES**

**FOR THE TIME-POOR  
MEDICAL, PRE-MED,  
USMLE OR PA STUDENT**



PDF



**197 PAGES**

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# CLINICAL INVESTIGATIONS

The following topics will be discussed during the Clinical Investigations tutorials:

**Click to Navigate:**

- Electrocardiogram (ECG)
- Arterial Blood Gases (ABG)
- Pulmonary Function Tests (PFT)
- Liver Function Tests (LFT)
- Full Blood Count (FBC)
- Renal Function Tests

## ECG - Learning objectives

### Essential

#### Pre reading

- ✓ *Correctly identify major anatomical features of the heart :4 chambers, valves, major vessels ( aorta, vena cavae, pulmonary vessels) conducting system, main coronary arteries.*
- ✓ *Understand the orientation of the heart in the thorax*

#### In course

- ✓ Correctly identify which ECG leads correspond to which anatomical part of heart
- ✓ Know how each feature of ECG corresponds to underlying cardiac electrical activity
- ✓ Correctly identify the following features on an ECG:
  - Calibration
  - Rate (n.b. be able to ‘eyeball’ rate as well as accurately calculate)
  - Rhythm : sinus/not sinus
  - Axis
  - Major arrhythmias: sinus tachycardia, sinus bradycardia, AF, VF, ectopic beats
  - LBBB, RBBB, first degree heart block
  - Chamber hypertrophy
  - Ischaemic changes: ST segment and T wave changes (stable angina pectoris and varying degrees of ACS including unstable angina, STEMI and non-STEMI)
  - Effect of abnormalities in electrolyte levels particularly potassium and calcium

### Desirable

Correctly identify the following features in an ECG:

- Pericarditis
- 2<sup>nd</sup> + 3<sup>rd</sup> degree block
- VT + SVT
- Pulmonary embolism
- Low voltage (hypothyroidism, pericardial effusion, obesity, COPD)
- QT changes including congenital prolonged QT
- Wolf-Parkinson-White syndrome
- Pacemaker spikes and complexes
- Atrial flutter

# ECG Tutorials

1. History of the ECG.
2. Information obtained from an ECG
3. Anatomy of the heart
4. Orientation of leads
5. ECG paper
6. ECG interpretation: (15 points)

## 1. History

The ECG has been in use for over a century. A British physiologist, Augustus Waller, performed the first ECG recording in 1887, and he gave demonstrations of his technique, using his dog, Jimmy, as the “patient”. Einthoven, from Holland, was present at such a demonstration. Willem Einthoven constructed his well-known triangle and the hexaxial system to help us gain further insight into the electrical activity of the heart.

## 2. Information obtainable from an ECG

ECGs can give us much information about the heart and also give us clues about other aspects of an individual’s state of health, such as:

- Rhythm – sinus/non-sinus
- Conduction – normal/abnormal
- Size of heart chambers
- Presence of ischaemic heart disease
- Pulmonary embolism
- Inflammation of the pericardium/effusion
- Emphysema
- Drugs the patient may be on e.g. Digoxin, Calcium channel blockers
- Electrolyte status of the patient – potassium, calcium levels
- Temperature – pyrexia or hypothermia
- Endocrine status – AF in thyrotoxicosis; bradycardia in hypothyroidism
- Raised intracranial pressure

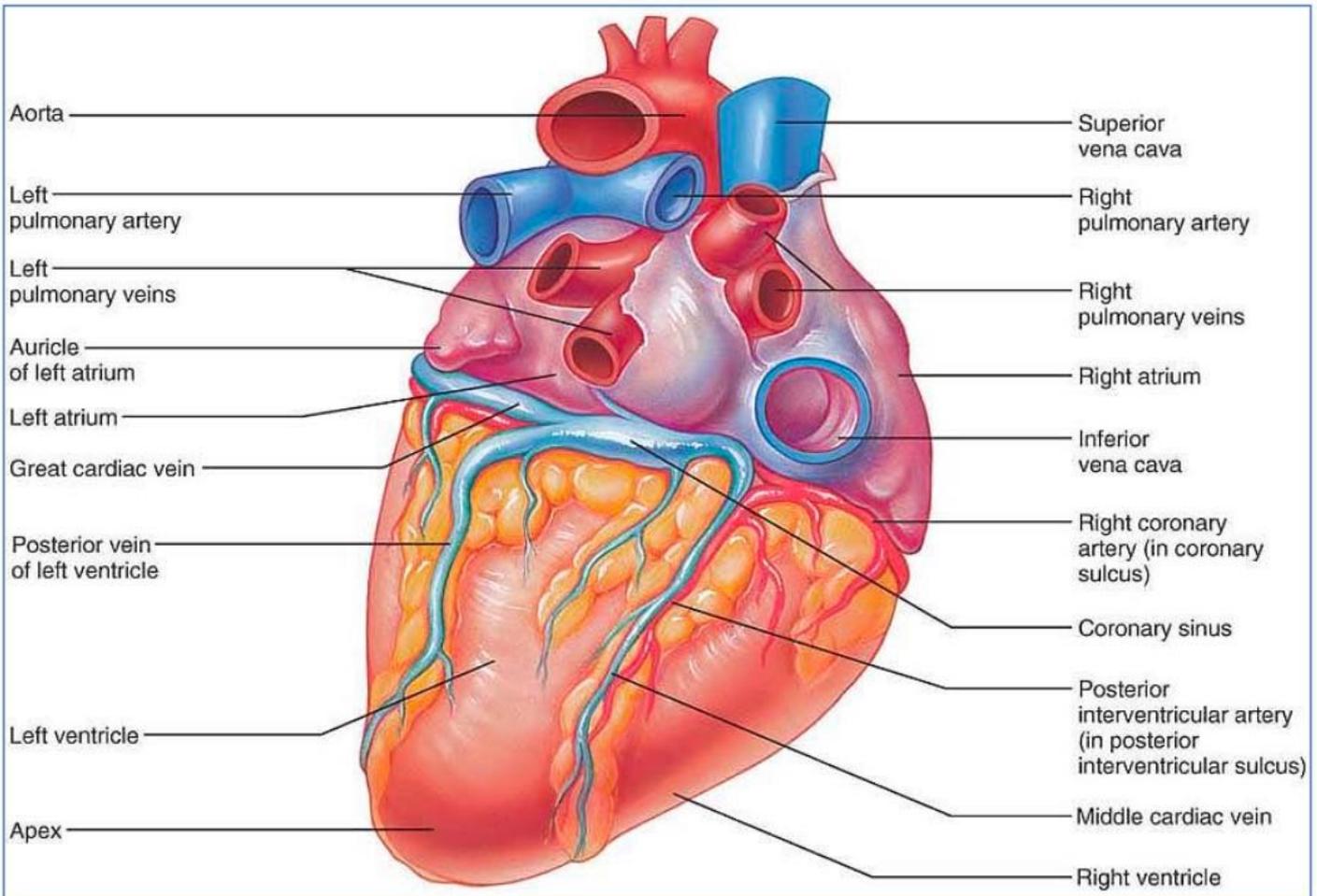
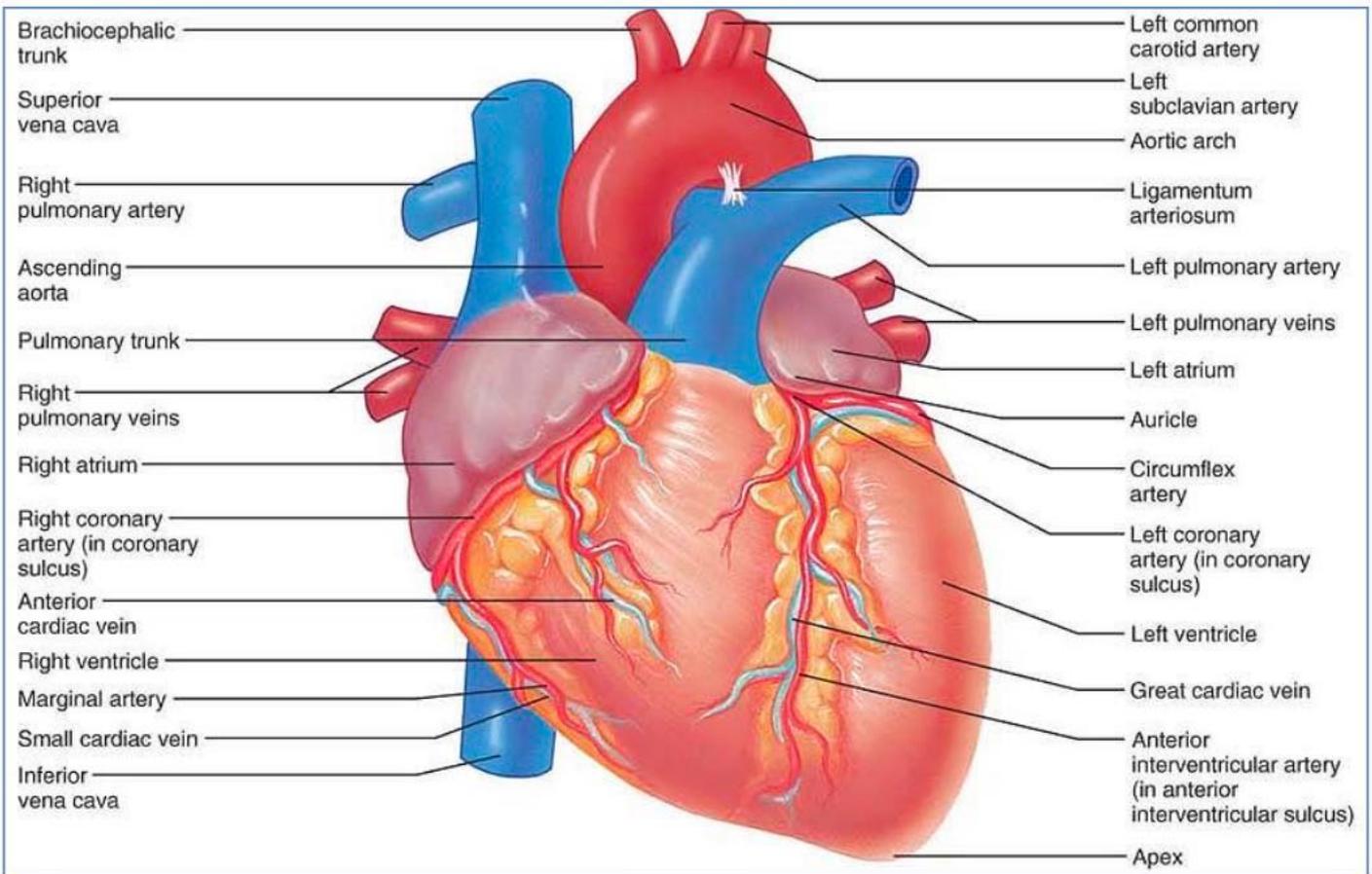
In ischaemic heart disease, the ECG may give us several pieces of information, such as:

- The *location* of the affected area of myocardium (in order of frequency)
  - Antero-septal area
  - Inferior surface of the heart
  - Antero-lateral aspect of the left ventricle etc
- *Whether the full thickness of the ventricular wall or only part of it is involved*

This is made evident by the presence or absence of pathological Q waves, and the situation of the ST segment. (ST elevation in transmural infarction; ST depression in sub-endocardial MI)

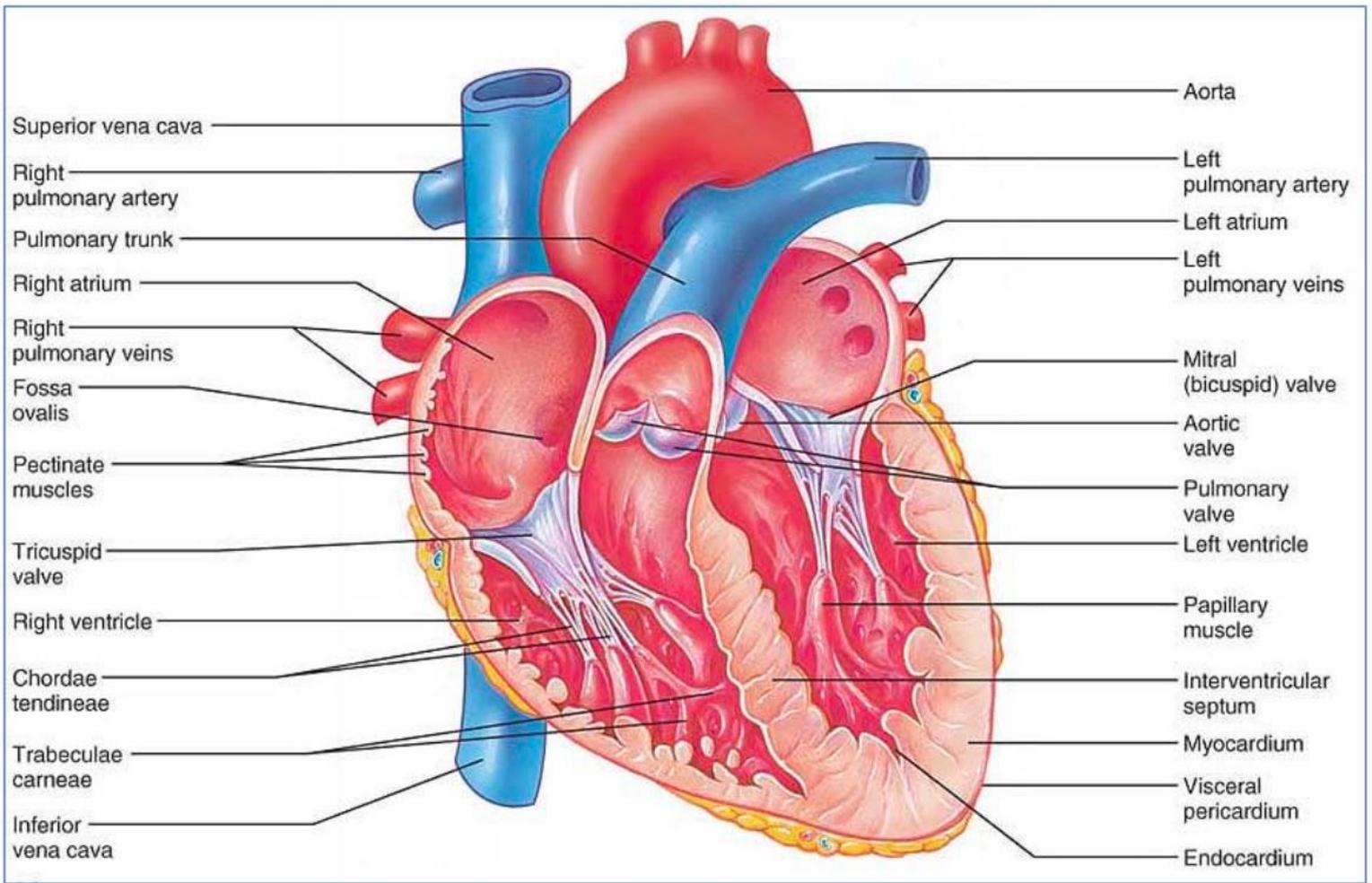
- Whether there are any obvious *sequelae* of the infarction, such as:

- Cardiac failure (sinus tachycardia)
- Rhythm abnormalities
- Left ventricular aneurysm (persistently elevated ST segment)

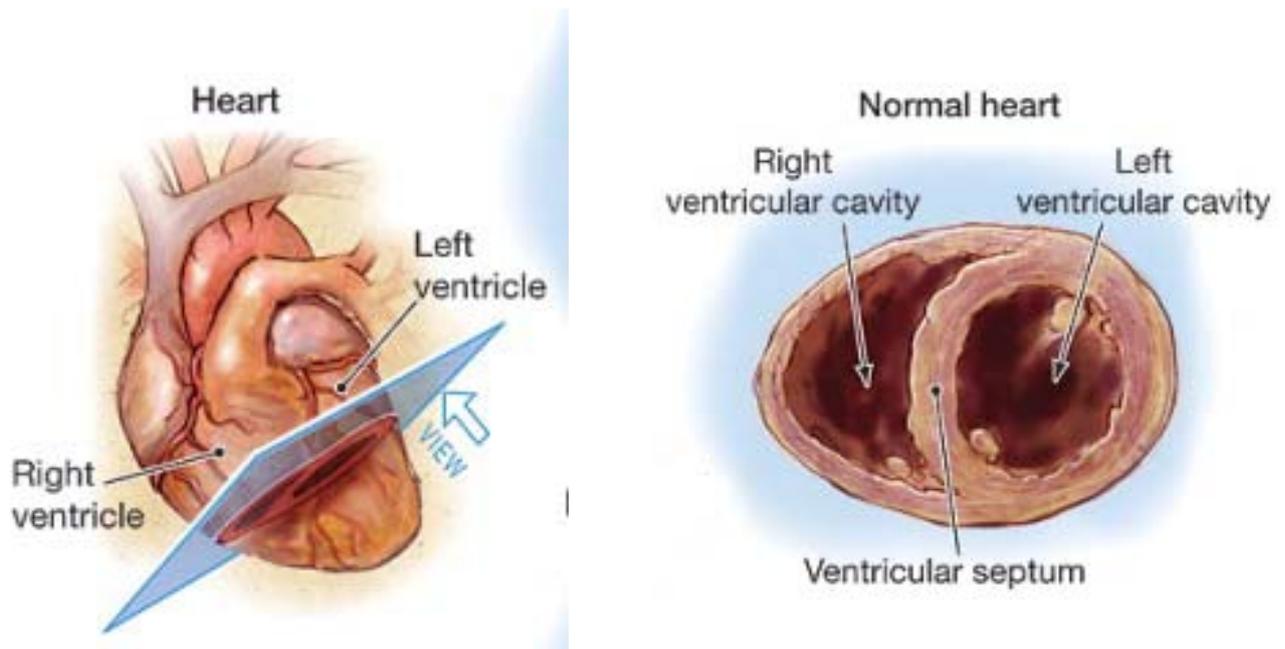


**Heart and Great Vessels**

**Sternocostal Aspect**



**Cutaway of the heart**



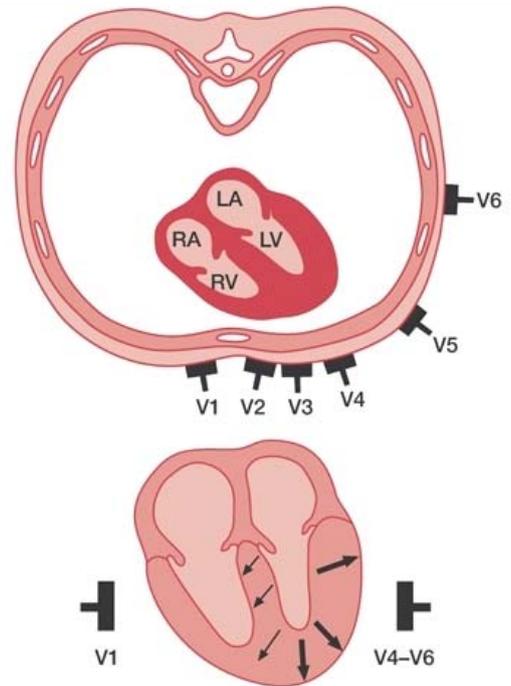
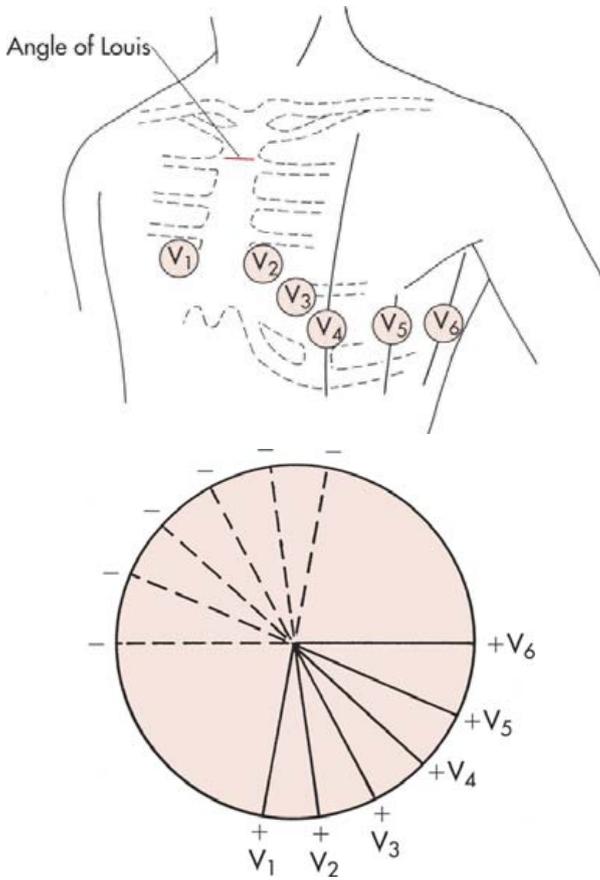
**Oblique section through right and left ventricles**

## 4. Orientation of leads

### ◦ Horizontal plane leads

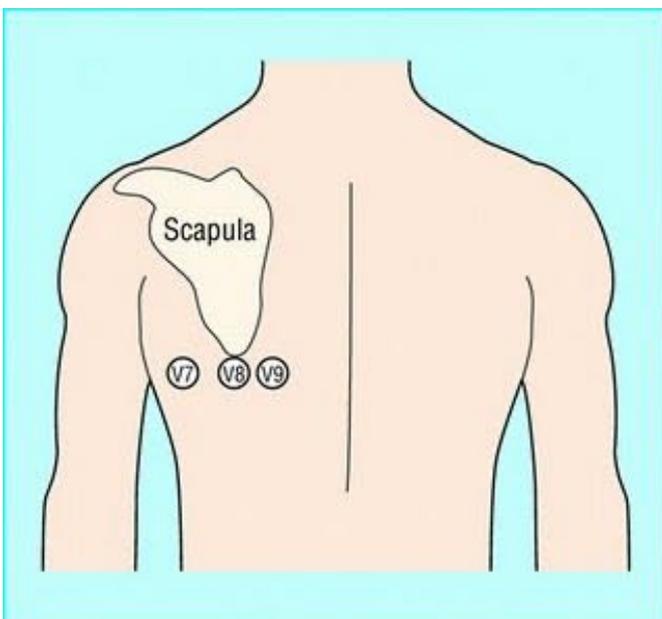
#### Anterior Chest Leads

Location of the electrodes for the chest (precordial) leads

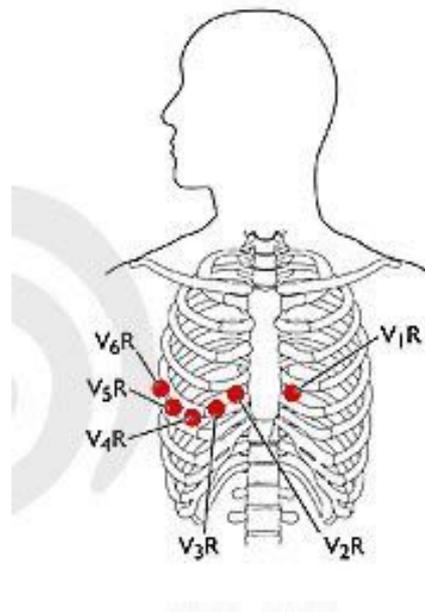


The positive poles of the precordial chest leads point anteriorly and the negative poles point posteriorly.

#### Posterior Chest Leads

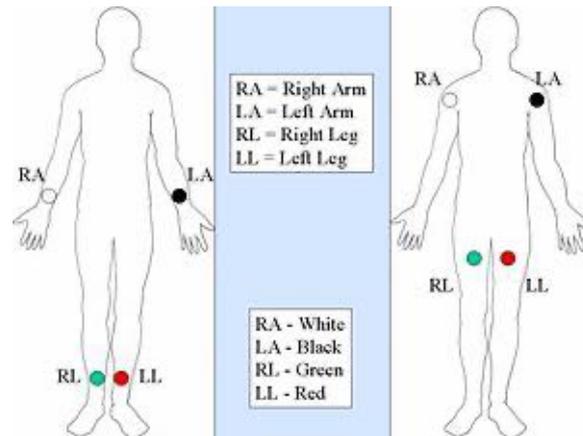


#### Right-sided Chest Leads



◦ Frontal plane leads

**The Limb Leads - Standard and Augmented Leads**

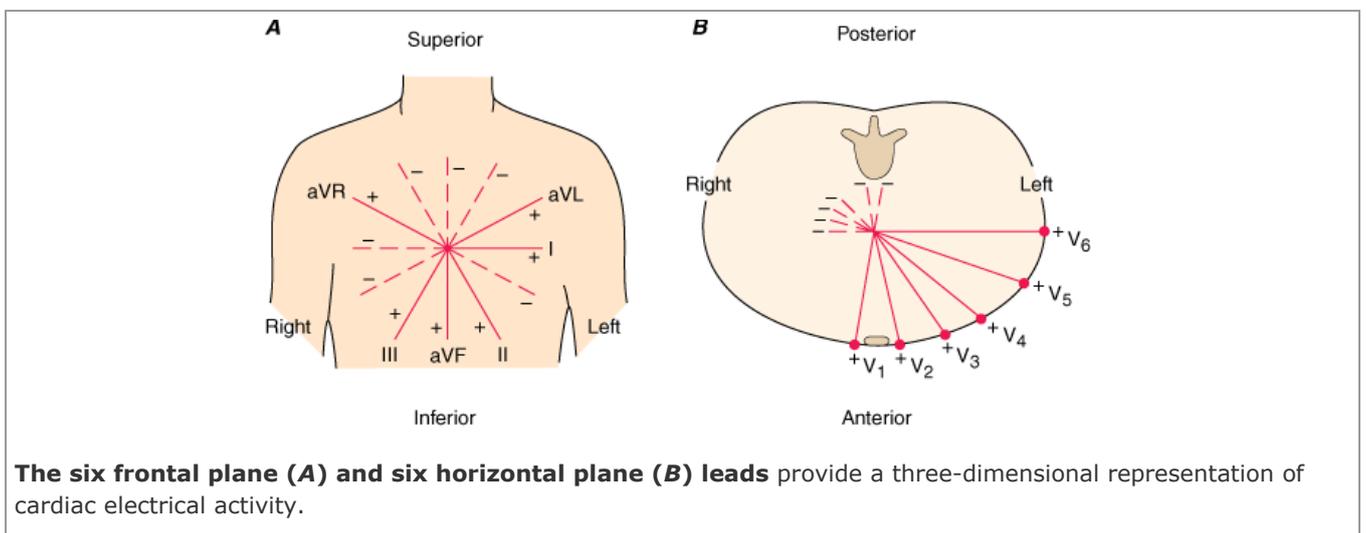
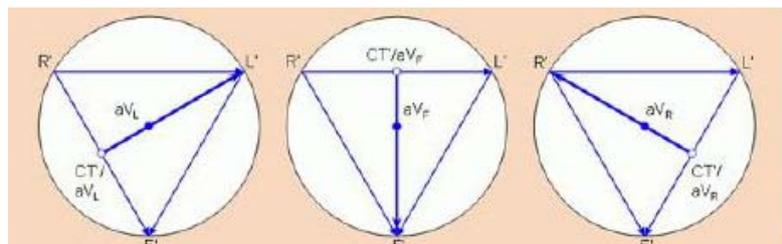
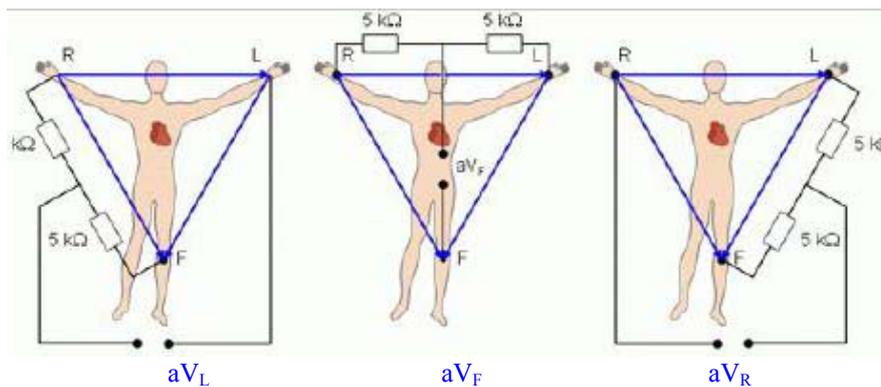


**Standard Leads**

Standard lead I	RA -ve	LA +ve
Standard lead II	RA -ve	LL +ve
Standard lead III	LA -ve	LL +ve

**Augmented Leads**

$AV_L$	LA +ve; RA, LL -ve
$AV_F$	LL +ve; RA, LA -ve
$AV_R$	RA +ve; LA, LL -ve



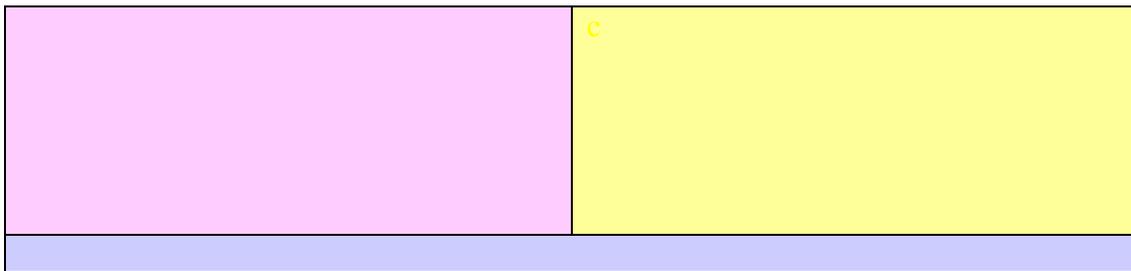
## Areas of the myocardium viewed by specific leads

<b>Std II, Std III, aVF</b>	Inferior (diaphragmatic) surface of LV
<b>Lead V1</b>	Right ventricle Right and left atria Interventricular septum (superior aspect) Endocardial aspect of the posterior left ventricle (Thus V1 has a view of all four cardiac chambers)
<b>Lead V2</b>	Interventricular septum (superior aspect) Endocardial aspect of posterior left ventricle.
<b>Lead V3</b>	Interventricular septum (inferior aspect)
<b>Lead V4</b>	Interventricular septum (inferior aspect) Apex
<b>Leads V5, V6</b>	Lateral aspect of left ventricle (inferior aspect)
<b>Std I, aVL</b>	Lateral aspect of left ventricle (superior aspect)
<b>aVR</b>	Endocardial aspect of left ventricle

## 5. ECG paper

The ECG is recorded such that

- the 6 limb leads (standard & augmented) are recorded on the left (pink)
- the 6 chest (V) leads on the right (yellow)
- the rhythm strip at the bottom (blue), either Std Lead II or Lead V1.



The ECG paper consists of small squares 1mm x 1mm, and big squares 5mm x 5mm

*In the horizontal plane*

The value of a big square (5mm) is 0.2 seconds; a small square (1mm) is 0.04seconds.

*In the vertical plane*

The value of two big squares (10mm) is 1millivolt (mV), so each small square in the vertical plane is equivalent to 0.1mV

## 6. ECG interpretation: PQRSTU

*When interpreting an ECG recording, we need to assess the following:*

- |                |      |                         |                 |
|----------------|------|-------------------------|-----------------|
| 1. Calibration | QRS: | 8. Pathological Q waves | 12. ST segment  |
| 2. Rate        |      | 9. Duration             | 13. T wave      |
| 3. Rhythm      |      | 10. Voltage             | 14. QT interval |
| 4. Axis        |      | 11. Configuration       | 15. U wave      |
| 5. P wave      |      |                         |                 |
| 6. PR interval |      |                         |                 |
| 7. PR segment  |      |                         |                 |

*To group these observations another way, we need to assess:*

1. Calibration
2. Rate, rhythm and axis
3. The waves – P, T and U waves (and any other waves, such as delta or J waves)
4. The QRS complexes
5. The segments – PR and ST
6. The intervals – PR and QT

- 1. Calibration**      The ECG is calibrated such that the 1mV standardisation mark is 10 mm tall and the horizontal line of the mark is 5mm wide (0.2 seconds) This means that the ECG is being recorded at 25mm/second



Normal 10mm/mV & 25mm/second calibration. Note the box-shaped calibration mark to the left of the complexes, two big squares tall & one big square wide

## 2. Rate

Normal = 60 –100 /min

### Regular rhythm:

Estimate the rate by counting the number of big squares between successive R waves, and dividing this number into 300.

**R – R interval of:**

<b>1 square</b>	<b>corresponds to a heartrate of 300/min</b>
<b>2 squares</b>	<b>150/min</b>
<b>3 squares</b>	<b>100/min</b>
<b>4 squares</b>	<b>75/min</b>
<b>5 squares</b>	<b>60/min</b>
<b>6 squares</b>	<b>50/min</b>

Rate may also be determined by dividing number of small squares between R waves into 1500

### Irregular rhythm:

Count the number of QRS complexes in 50 big squares and multiply your answer by 6 (5 big squares = 1 second, 50 big squares = 10 seconds)

## 3. Rhythm

**Rhythm**      **Sinus**  
                  /  
                  \  
                  **Non-sinus**

### Is the patient in normal sinus rhythm?

In normal sinus rhythm the following pertain:

- The sinus node is the pacemaker of the heart  
Because of the anatomical position of the SA node in relation to the AV node, in sinus rhythm, the P wave is always +ve in Std II, and –ve in aVR. This is because the P wave axis is directed towards the +ve pole of Std II, and away from aVR.
- Each P wave is followed by a QRS complex  
Each atrial depolarisation wave is conducted through the AV node and results in depolarisation of the ventricles.
- The PR interval in a particular lead is constant (the PR interval may vary slightly from lead to lead)  
In sinus rhythm, conduction through the AV node occurs at a normal, constant rate.  
In normal sinus rhythm, the heart rate varies with the phases of respiration, so-called sinus arrhythmia, the rate increasing with inspiration and slowing down on expiration.

### If the patient is *not* in normal sinus rhythm:

Either the SA node is still functioning as the cardiac pacemaker, but there is a partial or complete block to transmission of the depolarisation wave to the ventricles at the level of the AV node (evidence of SA node firing in the form of regular P waves is present)

or

An ectopic focus in atria, AV node or ventricles has taken over the function of cardiac pacemaker and over-ridden the SA node. This may be for a very brief period (e.g. atrial or ventricular ectopic beats) or for longer periods e.g. atrial fibrillation.

If an ectopic rhythm is evident:

- Are the QRS complexes narrow or wide?
- Is the rhythm regular or irregular?

### Width (duration) of QRS complexes

Is the rhythm originating in the atria, the AV nodal tissue or the ventricles?

The QRS complexes are usually narrow when the ventricles are being activated along the normal pathway – along the bundle branches – so will be narrow when an ectopic focus is situated in atrial or nodal tissue.

If the ectopic focus is in the ventricles, the complexes generated will be wider than normal, as the ventricular tissue is activated from myocyte to myocyte, rather than via the specialised conduction tissue, so the ventricular activation takes longer.

### Arrhythmias with narrow QRS complexes:

(rhythms originating in the atria or AV nodal tissue)

<b>1. Sinus arrhythmia</b>	<b>irregular</b>
<b>2. Sinus rhythm with AV block</b>	
<b>First degree</b>	<b>regular</b>
<b>Second degree (Mobitz I)</b>	<b>irregular</b>
<b>Second degree (Mobitz II)</b>	<b>irregular</b>
<b>Third degree (nodal escape)</b>	<b>regular</b>
<b>3. Ectopic atrial or nodal rhythms</b>	
<b>Atrial/nodal ectopic beats</b>	<b>irregular</b>
<b>Supraventricular tachycardias</b>	<b>regular</b>
<b>Atrial flutter</b>	<b>regular</b>
<b>Atrial fibrillation</b>	<b>irregular</b>

### Arrhythmias with wide QRS complexes:

(rhythms originating in the ventricles)

<b>Ventricular ectopic beats</b>	<b>irregular</b>
<b>Ventricular tachycardia</b>	<b>regular</b>
<b>Ventricular flutter</b>	<b>regular</b>
<b>Ventricular fibrillation</b>	<b>irregular</b>
<b>Third degree AV block (ventricular escape)</b>	<b>regular</b>

**In summary, there are three causes of an irregular rhythm (sinus arrhythmia and sick sinus syndrome excluded)**

- 1. Ectopic beats (atrial, nodal or ventricular)**
- 2. Second degree AV block, usually Mobitz I**
- 3. Atrial or ventricular fibrillation**

### Escape rhythms

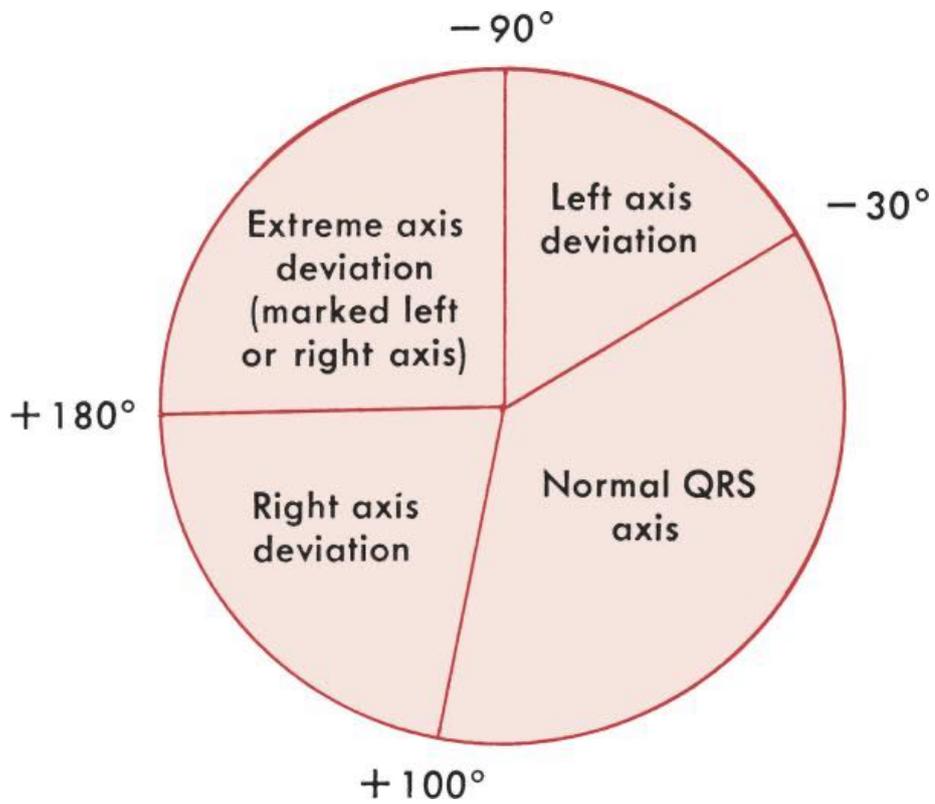
These occur in response to a nodal block, which necessitates that a focus distal to the block takes over the function of pacemaker for the heart.

SA node block → atrial or AV nodal escape rhythm (AV nodal rate = 40 – 60/min)

AV node block → ventricular escape rhythm (Idioventricular rate = 30 - 40/min) if block low down in AV nodal tissue; AV nodal escape rhythm if block high up in AV node.

#### 4. Axis

The normal axis of the heart lies between  $-29$  degrees and  $+100$  degrees.



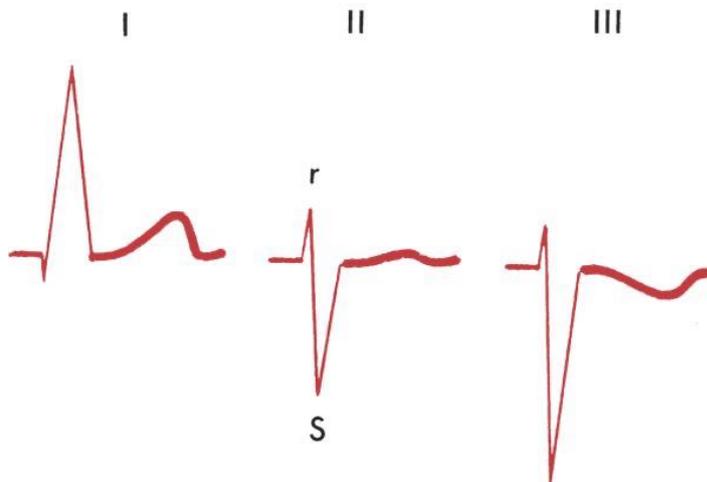
To determine the axis, the standard leads are used as per table below (blue shaded areas)

	Std I	Std II	Std III
<b>Normal</b>	▲	▲	▲ or ▼
<b>LAD</b>	▲	▼	▼
<b>RAD</b>	▼	▲ (occ ▼)	▲
<b>Extreme</b>	▼	▼	▲ or ▼

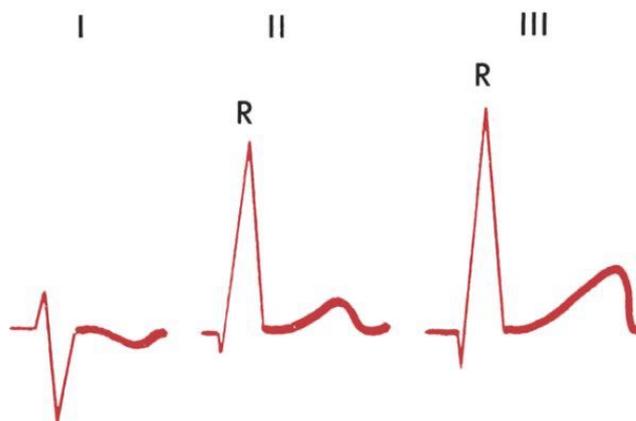
**Causes of left axis deviation**  
( $-30^\circ$  to  $-90^\circ$ )

1. Marked left ventricular hypertrophy
2. Left anterior hemiblock
3. Inferior MI
4. Pregnancy
5. Normal variant

**Left Axis Deviation**



**Right Axis Deviation**



**Causes of right axis deviation**  
( $+100^\circ$  to  $+180^\circ$ )

1. Right ventricular hypertrophy
2. Left posterior hemiblock
3. Lateral MI
4. Acute pulmonary embolism
5. Emphysema
6. Dextrocardia
7. Spurious (Left & right arms interchanged)
8. Normal variant

## 5. P wave

The P wave reflects depolarisation of the atria. This wave is usually a single deflection, and is a composite wave representing depolarisation of both right and left atria. When conduction through the atria is slow, the P wave may be seen as a series of two waves, the first being due to depolarisation of the right atrium, the second caused by left atrial depolarisation occurring shortly thereafter.

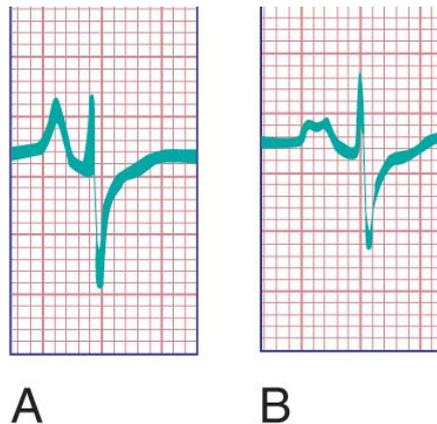
### Height and shape of P wave

A normal P wave is  $< 3\text{mm} \times 3\text{mm}$  in height and width.

**Right atrial hypertrophy** (“P pulmonale”) = P wave  $\geq 3\text{ mm}$  in *height* in limb leads (often best seen in Std lead II)

**Left atrial hypertrophy** (“P mitrale”) = P wave  $\geq 3\text{ mm}$  in *width*. The P mitrale may have a humped or bifid shape.

In left atrial hypertrophy, the P wave in lead V1 may be biphasic, with a wide or deep  $-ve$  component  $\geq 1\text{mm}$



Atrial enlargement. *A*, Peaked narrow P waves characteristic of right atrial enlargement.  
*B*, Wide bifid M-shaped P waves typical of left atrial enlargement

### Vector of P wave

Inverted:	aVR
Upright:	I II aVF V4 – V6
Upright/biphasic/inverted:	V1 – V3 III aVL

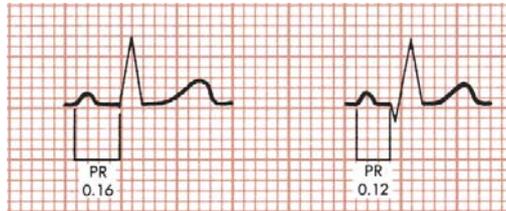
## 6. PR Interval

The PR interval comprises depolarisation of the atria followed by conduction of the wave of depolarisation through the AV nodal tissue to the top of the interventricular septum.

It is measured from beginning of the P wave to the beginning of the QRS complex.

Normal = 0.12 – 0.2 sec (3 – 5 small squares)

>0.2 sec = *first degree heart block*

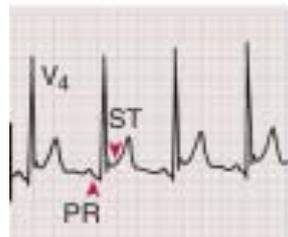


Measurement of the PR interval

## 7. PR Segment

The PR segment reflects the time taken for transmission of the wave of depolarisation from atria to ventricles via the AV node and bundle of His.

The PR segment is normally isoelectric. Pericarditis causes depression of the PR segment



Acute pericarditis is often characterised by two apparent injury currents, one atrial, the other ventricular. The atrial injury current vector produces PR depression. The ventricular injury current is associated with ST elevation.

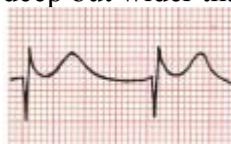
## QRS Complex

The QRS complex reflects depolarisation of the ventricles. Four features of the QRS complex need to be assessed:

- Q waves
- Duration
- Voltage
- Configuration

## 8. Pathological Q waves

*Definition:* A pathological Q wave is >25% the height of the ensuing wave (from tip of Q to peak of R). Sometimes the pathological Q wave is not deep but wider than normal (>0.04sec in duration)



Example of a pathological Q wave in Std III. Note that the depth of the Q wave is > 25% the total height of the complex, from tip of Q to peak of R.

## 9. Duration

Normal duration is  $\leq 0.10\text{sec}$  ( $2\frac{1}{2}$  small squares)

## 10. Voltage

### Criteria for normal voltage of left ventricle

- **Limb leads** (from top of R to bottom of S): 10 – 15 mm
- **V leads:** S in V1/V2 + R in V5/V6:
  - $\leq 60$  mm if  $<30$  years
  - $\leq 40$  mm if 30-40 years
  - $\leq 35$  mm if  $>40$  years
- **Std I** < 15 mm
- **aVL** < 11 mm

### Criteria for normal voltage of right ventricle

- **V1:**  $r < S$ ,
- **Axis** No right axis deviation

## 11. Configuration

- Presence of abnormal waves:
  - Delta waves of Wolff-Parkinson-White syndrome
  - J waves of hypothermia
  - R waves too big in V1 ( $R > s$ ) or
  - R waves too small in V3 ( $r < 3\text{mm}$ )
- Presence of VPBs



Ventricular premature beat (VPB) showing distortion of the QRS and T wave inversion, absence of P wave and compensatory pause before next sinus beat appears.

## 12. ST Segment

The ST segment commences at the J point (the junction of the ST segment and the QRS complex) and extends to the beginning of the T wave. Should be isoelectric as there is no voltage difference between areas of the ventricles in this phase of the ECG.



Lead  $V_5$ .

1 = PQ junction that serves as the baseline reference

2 = J point

3 = ST segment

In this example, slight ST depression is present.(2 should be at same level as 1)

### 13. T wave

The T wave represents ventricular repolarisation.

#### Vector

Its vector is often the same as the preceding QRS complex.

Always inverted in aVR

Upright or inverted in V1, V2 (in young people), Std III

Upright in all other leads.

#### Height

Its height is dependent on the height of the preceding QRS complex, but as a rough guide, should be  $\leq 5$  mm in limb leads and  $\leq 10$  mm in chest leads.

### 14. QT Interval

Encompasses ventricular de- & repolarisation. It is measured from beginning of QRS to end of T wave.

The QT interval is dependent on heart rate, decreasing as rate increases, and becoming longer as rate slows. As a rough guide, the QT interval should normally be  $<$  half the R-R interval.

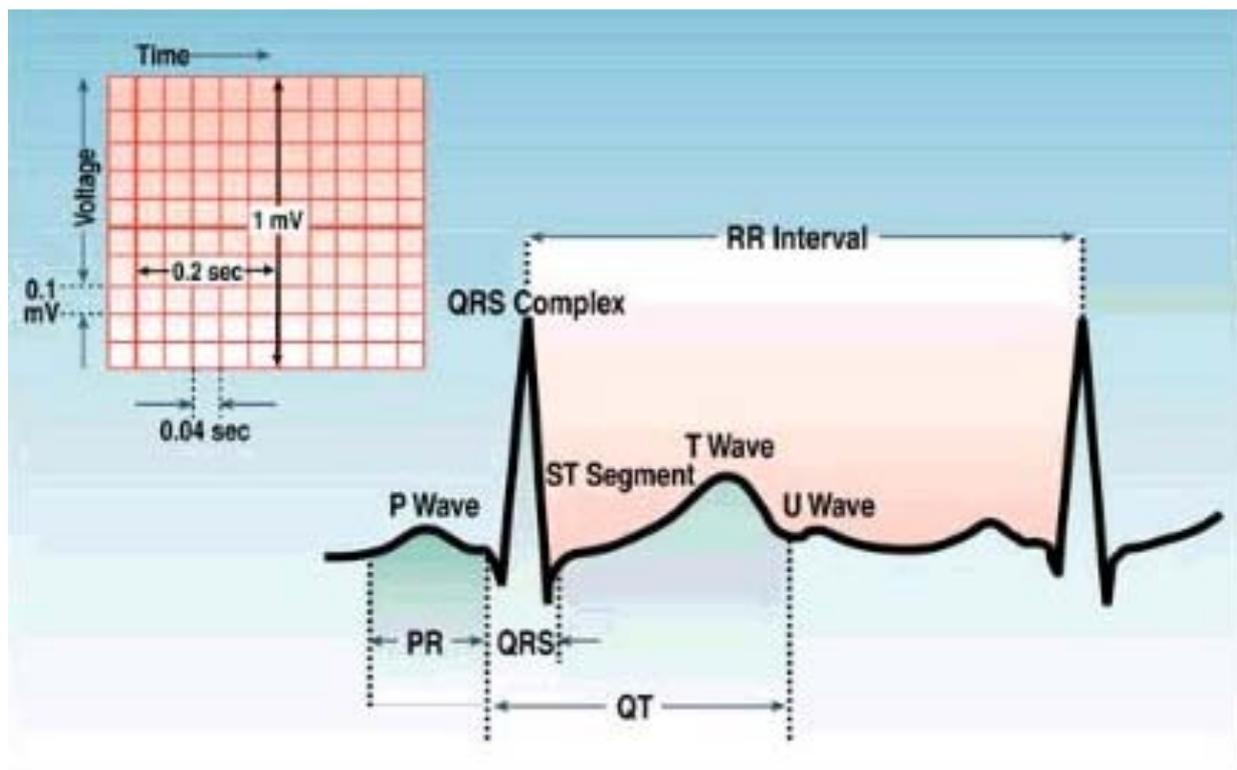
Corrected QT is the QT calculated for a heart rate of 60 bpm.

$$QTc = QT/\sqrt{R-R} \text{ (seconds)}$$

Corrected QT in males is 0.43 sec, in females 0.45 sec.

### 15. U wave

The U wave is a small wave ( $\leq 25\%$  the height of the T wave) which follows the T wave and which is thought to represent repolarisation of the Purkinje fibers. It is often only visible at slow heart rates, and is most prominent in the right sided chest leads.



## DIRECTION of P and T WAVES

### *The P wave*

< 0.12 sec ( < 3 small squares)

≤ 3mm height ( ≤3 small squares)

Inverted in aVR (all deflections are always negative in this lead)

Upright/biphasic/inverted V1 – V3, III, aVL

Upright I, II, aVF, V4 – V6

### *The T wave*

<5 mm height in limb leads

<10 mm in praecordial leads

Inverted in aVR

Upright or inverted in V1, V2 (young people), III

Upright in all other leads.

**Easy way to remember:** in Leads V1 and V2

- P and T waves may be upright or inverted (especially in young people)
- ↑ST segment may be a normal variant (<3mm) - “early repolarisation” pattern
- ↓ST segment may be a normal variant (<1mm)

\*Important always to take note of the history, and to compare current ECG with previous ECGs. If ST deviation from isoelectric line is not present in previous ECGs, the elevation or depression is likely to be significant.

## Pathological T waves

**Tall T waves** (>10 mm in V leads / >5mm in limb leads)

- Ischaemic causes:
  - Acute transmural MI - “hyperacute T waves”
  - Coronary artery spasm - Prinzmetal’s Angina / Cocaine
- Non- Ischaemic causes:
  - Hyperkalaemia
  - Acute pericarditis

**Inverted T waves**

Myocardial ischaemia/infarction  
Ventricular hypertrophy  
BBB  
Pulmonary embolism

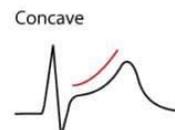
Cardiomyopathy (CMO)  
Digoxin  
CVA

### A Characteristics of ST-segment elevations caused by ischemia



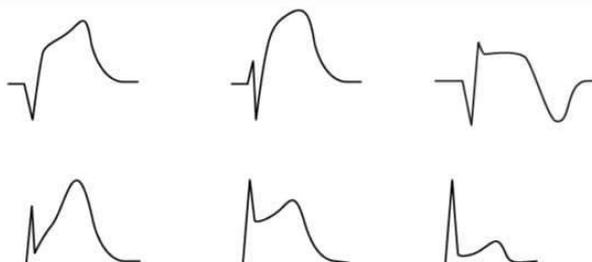
ST-segment elevations caused by ischemia typically displays a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

### B Typical non-ischemic ST-segment elevation



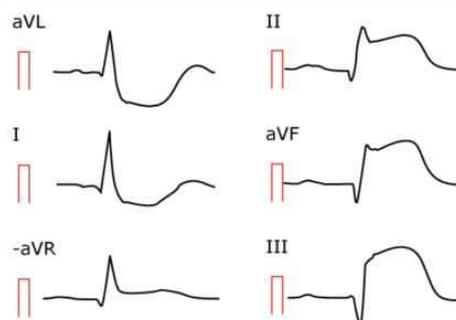
Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

### C Examples of ST-segment elevations caused by ischemia



ST-segment elevation can vary markedly in appearance. These six examples were retrieved from six different patients with STEMI.

### D Real life example (limb leads shown)



ECG from a male patient (age 61) who experienced chest pain while driving to work. Note ST-segment elevations as well as reciprocal ST-segment depressions. There are also pathological Q-waves (leads III, aVF and perhaps II).

### A Normal T-waves



**Normal T wave**  
Smooth transition from ST-segment to T wave. T wave is slightly asymmetric with a steeper downslope.

**Normal variant**  
Large, asymmetric T wave with broad base. Often in conjunction with slight J point elevation in leads V2-V4.

### B Large T-waves



**Hyperkalemia**  
Large, symmetric, pointed with short base.

**Hyperacute T wave**  
can be seen in transmural ischemia. High, broad based, symmetric, not pointed. Almost always seen in conjunction with ST-segment elevation.

### C Biphasic (diphasic) T-waves

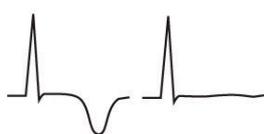


Both these T waves are negative (inverted) since the terminal portions are negative.

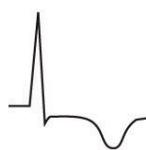
This T wave is positive by definition since the terminal portion is positive.

Whenever spotting a biphasic T wave, try to determine whether it is actually a positive or negative (inverted) T-wave by viewing the terminal portion of the T wave.

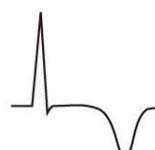
### D Negative (inverted) T-waves



**Post-ischemic**  
Symmetric T wave, with varying depth. Ranges from flat T wave to very deep T wave inversion. Inverted T waves do not equate acute (ongoing) ischemia, but rather appear after an episode of ischemia!



**Acute (ongoing) ischemia**  
T wave inversion with simultaneous ST-segment deviation (most commonly ST-depression). Note that it is the ST-segment deviation that represents the acute ischemia!



**Cerebrovascular insult pattern**  
Very deep (gigantic) T wave inversions in the chest leads. Some studies report this finding in up to 30% of patients with intracerebral hemorrhage.



**Hypertrophic cardiomyopathy**  
Symmetric T wave inversions, most commonly in V1-V3. Often very deep and accompanied by large R waves. Occasionally accompanied by ST-segment depression.



**PERIMYOKARDIT**  
T wave inversions occur after normalization of ST-segment elevations in perimyocarditis. T wave inversions often seen in most leads.

## ECG Differential Diagnoses

### P waves

#### ▫ Tall P waves

P wave  $\geq 3$  mm in height in limb leads (“P pulmonale”)

- Right atrial hypertrophy

#### ▫ Wide P waves

P wave  $\geq 3$  mm in width in limb leads (“P mitrale”)

- Left atrial hypertrophy
- Slow atrial conduction due to IHD

#### ▫ P wave with altered configuration

P wave in sinus rhythm is always  $-ve$  in aVR,  $+ve$  in Std II.

If pacemaker of the heart is a focus low down in the atrium or in AV nodal tissue, P wave will be  $+ve$  in aVR and  $-ve$  in Std II

The P wave of an atrial ectopic beat close to the SA node has a slightly different configuration from the sinus P wave.

### PR Duration

#### Short PR

Accessory pathways such as W-P-W

Nodal tachycardia (atria depolarised just before ventricles)

Ectopic atrial rhythm (focus close to AV node)

#### Long PR

Elderly

Drugs “ABCD” e.g. Adenosine, Beta blockers, Calcium channel blockers, Digoxin

Myocarditis

IHD especially involvement of RCA which supplies AV node in 90% of individuals. AV block is common in inferior myocardial infarction, when caused by RCA occlusion

### QRS Complex

#### Pathological Q waves

Normal variant in aVR, aVL, aVF, Std III, V1

Myocardial infarction (transmural)

Left ventricular hypertrophy

Absent r waves V1 – V3 → QS waves in these leads

Right ventricular hypertrophy

Poor/ reversed r wave progression may → QS waves in right-sided chest leads

Left bundle branch block.

Absent r waves V1 – V3 → QS waves in these leads

HOCM

Septal hypertrophy → deep Q waves in lateral chest leads (Leads V5 & V6)

#### Widened QRS complex ( $> 0.10$ sec)

Bundle branch block (left or right)

Non-specific intraventricular conduction delay

Ventricular beats

Hyperkalaemia

Drugs e.g. tricyclics

W-P-W

**Low voltage QRS complexes**  
( $<5\text{mm}$  limb leads,  $<10\text{mm}$  chest leads)

Spurious (ECG calibration altered to  $5\text{mm/mV}$ )  
COPD  
Obesity  
Pericardial effusion  
Infiltration of the myocardium e.g. hypothyroidism/amyloid  
Extensive myocardial infarction

**R wave  $>$  s wave in Lead V1**

Posterior MI (transmural)  
Right ventricular hypertrophy  
Right bundle branch block  
W-P-W

**R wave in Lead V3  $<$  3 mm (“poor R wave progression”)**

Anteroseptal MI  
LVH/RVH  
LBBB  
COPD

**ST segment elevation**

Transmural myocardial infarction (STEMI)  
Coronary artery spasm:                      Prinzmetal’s angina  
   Cocaine  
Ventricular aneurysm  
Normal variant (V1 – V2,  $\leq 3\text{ mm}$ )  
LVH                      (V1 – V3)  
LBBB                    (V1 – V3)  
Acute pericarditis  
Acute myocarditis  
Hyperkalaemia

**ST segment depression**

Myocardial ischaemia:                      · angina pectoris  
   · sub-endocardial MI  
Reciprocal change in acute transmural myocardial infarction  
Normal variant (only in V1 – V2,  $< 1\text{ mm}$ )  
Left ventricular hypertrophy (in left chest leads, V5-V6)  
Right ventricular hypertrophy (in right chest leads, V1-V2)  
LBBB (V5 –V6)  
RBBB (V1-V2)  
Digoxin  
Hypokalaemia

## T wave abnormalities

**Tall T waves** (> 10 mm in V leads/> 5 mm in limb leads)

Acute transmural MI  
Coronary artery spasm  
Acute pericarditis  
Hyperkalaemia

### Inverted T waves

Myocardial ischaemia/infarction  
Ventricular hypertrophy  
BBB  
CMO  
Digoxin  
CVA  
Acute pulmonary embolism (Leads V1 – V4)  
(Hypokalaemia → T wave flattening)

## QT Interval

### Short QT

Congenital  
Drugs e.g. digoxin

Hyperkalaemia  
Hypercalcaemia  
Hyperthermia  
Acidosis

### Long QT

Congenital  
Drugs e.g. Amiodarone  
Erythromycin

Hypokalaemia  
Hypocalcaemia  
Hypothermia

### IHD

Myocarditis  
Head injury/Sub-arachnoid Hx/Vasovagal

## U Waves

### Prominent U waves

Hypokalaemia  
Antiarrhythmic drugs e.g. Amiodarone  
LVH  
Sub-arachnoid Hx

### Inverted U waves

Myocardial ischaemia

## Axis Deviation

### LAD

LVH  
Left anterior fascicular block  
Inferior MI  
Pregnancy  
Normal variant

### RAD

RVH  
Left posterior fascicular block  
Lateral MI  
Acute pulmonary embolism  
COPD  
Dextrocardia  
Normal variant  
Spurious (arm electrodes interchanged)

# Classification of arrhythmias

Arrhythmias arise due either to a disorder of impulse

- **formation**

or

- **conduction**

## Disorders of impulse formation

In addition to the specialised tissue known as the sinu-atrial node (SA node), which normally fills the role of pacemaker for the heart, all elements of the conducting tissue, such as the atrio-ventricular node (AV node), bundle branches and Purkinje fibers are capable of performing this pacemaker function, as well as the cardiac myocytes, which all have inherent rhythmicity – the ability to generate an impulse de novo.

An arrhythmia is any rhythm which does not fulfil the criteria for normal sinus rhythm (the impulses originate in the SA node at a rate of 60 -100 times per minute, each impulse is conducted through the AV node to the ventricles and the time taken for this to occur is the same for each impulse generated)

Arrhythmias may therefore arise in the SA node, the atria, the AV node, or the ventricles.

- **Sinus node**

- Sinus arrhythmia

- Sinus bradycardia

- Sinus tachycardia

- **Atria**

- Atrial extrasystoles

- Paroxysmal atrial tachycardia\*

- Atrial flutter

- Atrial fibrillation

- **AV node**

- AV nodal extrasystoles

- AV nodal re-entrant tachycardia\*

- **Ventricles**

- Ventricular extrasystoles

- Ventricular tachycardia

- Ventricular flutter

- Ventricular fibrillation

## Disorders of impulse conduction

- **SA node block**
- **AV node block** First degree  
Second degree (Mobitz I or Mobitz II)  
Third degree (complete heart block)
- **Accessory pathways e.g. Wolff-Parkinson-White syndrome** → re-entrant tachycardia \*

\* Supra-ventricular tachycardias (SVTs)

### Escape Rhythms

A focus in the atria, AV node or ventricle will start to generate impulses if the impulse generating mechanism of the heart fails, and the focus then becomes the pacemaker of the heart for as long as it is the primary impulse generator. The location of the escape rhythm is dependent on the level of the defect. These rhythms are termed escape rhythms and provide a safety net for the heart in times of a block in transmission of the cardiac impulse.

SA node block (caused by, for example, inhibition of the SA node by beta blockers) will result in an **atrial escape rhythm** or one originating lower down in the AV node (**nodal escape rhythm**)

AV node block, if third degree, will result in a **nodal escape rhythm** if the block is proximal in the AV nodal tissue (sufficient normal AV nodal tissue distal to the block to generate impulses and take over pacemaker function)

or

in a **ventricular escape rhythm** if the block is distal in the AV nodal tissue (no normally functioning AV nodal tissue to take over the function of pacemaker)

Nodal escape rhythms may be differentiated (for the most part) from ventricular escape rhythms by:

- **Duration of the QRS complexes** (narrow, normal appearing QRS complexes in AV nodal escape rhythms, wide complexes in ventricular escape rhythms, because ventricles being activated from cell to cell, not along normal efficient conducting pathways, so activation takes longer)
- **Rate of the escape rhythm**

AV nodal escape rhythm rate 40 – 60 per minute

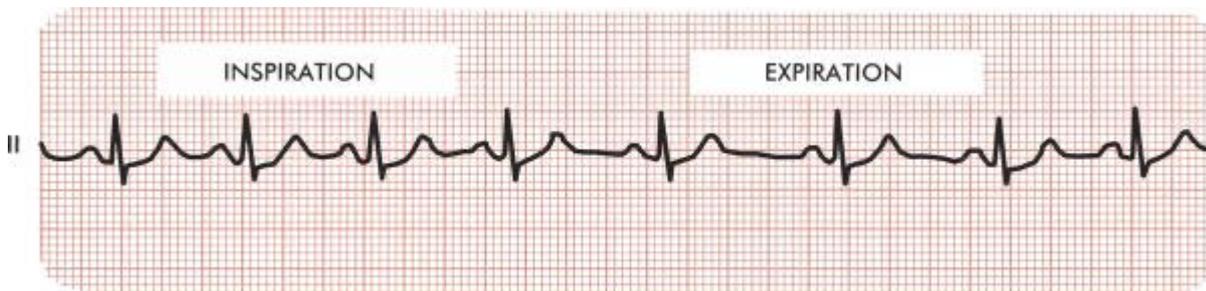
Ventricular escape rhythm rate 15 - 40 per minute (however, sometimes accelerated to >40/minute)

## Arrhythmias affecting the sinus node

### Sinus Arrhythmia

In healthy people, especially younger subjects, the SA node does not pace the heart at a perfectly regular rate. Instead, a slight beat-to-beat variation is present. When this variability is more accentuated, the term *sinus arrhythmia* is used.

The most common cause of sinus arrhythmia is respiration. Respiratory sinus arrhythmia is a normal finding and may be quite marked particularly in children and young adults. The heart rate normally increases with inspiration and decreases with expiration because of changes in vagal tone that occur during the different phases of respiration.



**Respiratory sinus arrhythmia.**

### Sinus bradycardia

With sinus bradycardia, sinus rhythm is present; the heart **rate is less than 60 beats/min.**

This arrhythmia commonly occurs in the following conditions:

- Normal variant (Many people have a resting pulse rate of less than 60 beats/min, and trained athletes may have a resting or sleeping pulse rate as low as 35 beats/min.)
- Drugs that
  - increase vagal tone (e.g., digitalis)
  - decrease sympathetic tone (e.g., beta blockers)
  - calcium channel blockers
- Hypothyroidism
- Hyperkalaemia
- Sick sinus syndrome (Some patients, particularly elderly ones, have marked sinus bradycardia without obvious cause, probably from degenerative disease of the SA node or surrounding tissue.)
- Sleep apnoea syndromes
- Carotid sinus hypersensitivity
- Vasovagal reactions

## Sinus Tachycardia

In general, sinus tachycardia occurs with any condition that produces an *increase* in sympathetic tone or a *decrease* in vagal tone.

**Rate 100 – 200 bpm** (young)  
**100 – 150 bpm** (elderly >70 years)

The following conditions are commonly associated with sinus tachycardia:

- Anxiety, excitement, exertion, and pain
- Drugs that increase sympathetic tone (e.g., epinephrine, dopamine, tricyclic antidepressants and cocaine)
- Drugs that block vagal tone (e.g., atropine)
- Fever, many infections, and septic shock
- Congestive heart failure (CHF)
- Pulmonary embolism  
Sinus tachycardia is one of the most common arrhythmias which occurs in acute pulmonary embolism.
- Acute myocardial infarction; sinus tachycardia generally a bad prognostic sign and implies extensive heart damage.
- Hyperthyroidism (sinus tachycardia at rest may be an important diagnostic clue)
- Pheochromocytoma
- Intravascular volume loss because of bleeding, vomiting, diarrhoea, acute pancreatitis, dehydration.
- Alcohol intoxication or withdrawal

## Ectopic arrhythmias originating above the ventricles

- Atrial premature beats
- Supraventricular tachycardias
- Atrial Flutter
- Atrial Fibrillation

### Atrial premature beats (APBs)

An ectopic focus in left or right atrium discharges and depolarises the atria before the sinus node was due to fire again.

#### Aetiology

Normal hearts (APBs are the most common arrhythmia)

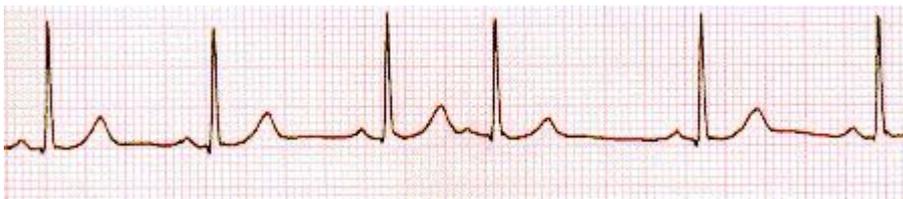
Emotional stress

Caffeine excess

Drugs e.g. those used for asthma, such as epinephrine, aminophylline

Hyperthyroidism

Structural heart disease e.g. valvular lesions



The fourth complex in this recording is an atrial premature complex.

Note the following:

↳ configuration of the ectopic P wave is slightly different from that of the sinus P waves

↳ narrow, normal appearing QRS complex follows the ectopic P wave

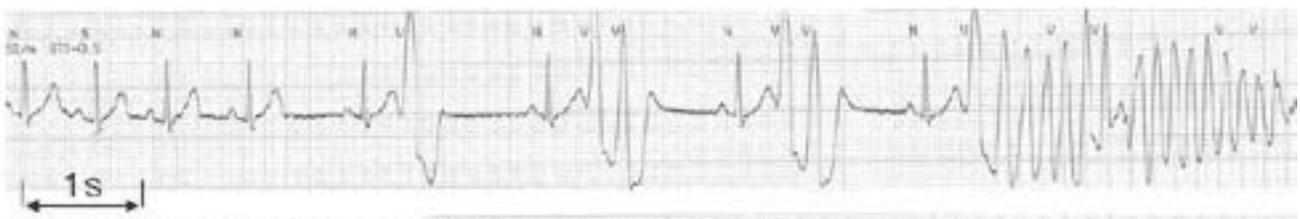
↳ short R-R interval preceding the complex

↳ long R-R interval following the complex, the so-called “compensatory pause”

The APB is distinguishable from a VPB (ventricular premature complex) by:

*f* no discernible P wave precedes the VPB

*f* the VPB differs obviously in configuration from the normal QRS complexes, being prolonged in duration (wide), often bizarre looking and followed by T wave inversion.

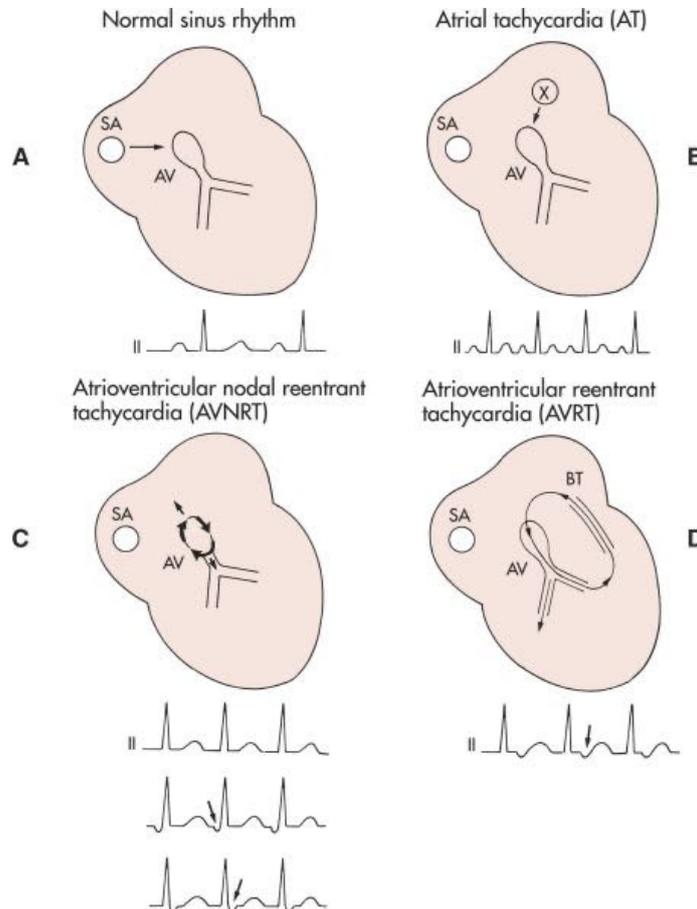


The 6<sup>th</sup>, 8<sup>th</sup>, 9<sup>th</sup>, 11<sup>th</sup>, 12<sup>th</sup> complexes are VPBs; after the 13<sup>th</sup> complex (normal QRS) an episode of VT has commenced.

## Supraventricular tachycardias

Rate  $\leq 250$  bpm

- **Atrial tachycardia** ( $\geq 3$  consecutive APBs) – P wave *before* QRS (usually +ve in Std II) or buried in preceding T wave
- **Nodal re-entrant tachycardia** – P wave often *hidden in QRS*, may be just before/after QRS, when it will be seen to be –ve in Std II (atria and ventricles being depolarised at approximately the same time)
- **Bypass tract re-entrant tachycardia** – ventricles activated before atria, therefore P wave occurs *after* QRS and –ve in Std II

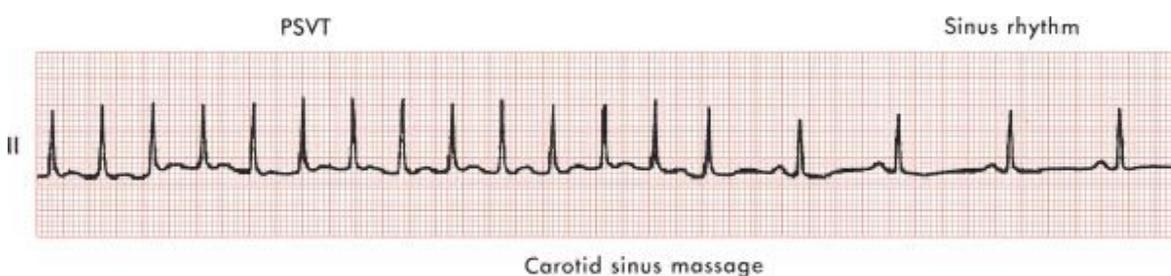


**A**, Normal sinus rhythm.

**B**, With atrial tachycardia (AT), a focus (X) outside the sinoatrial node fires off automatically at a rapid rate.

**C**, With atrioventricular (AV) nodal reentrant tachycardia (AVNRT), the cardiac stimulus originates as a wave of excitation that spins around the AV nodal (junctional) area. As a result, retrograde P waves may be buried in the QRS or appear immediately before or just after the QRS complex (*arrows*) because of nearly simultaneous activation of the atria and ventricles.

**D**, A similar type of reentrant (circus-movement) mechanism may occur with a bypass tract (BT) of the type found in Wolff-Parkinson-White syndrome (see ). This mechanism is referred to as *atrioventricular re-entrant tachycardia* (AVRT). Note the negative P wave (*arrow*) in lead II, somewhat after the QRS complex.



Paroxysmal supraventricular tachycardia (PSVT) treated with carotid sinus massage. The first 14 beats in this rhythm strip show the regular tachyarrhythmia with a rate of about 140 beats/min and no visible P waves.

## Atrial Flutter *Rate: 250 – 350 per minute (average 300)*

Re-entrant tachycardia travelling anti-clockwise in right atrium (80%), clockwise (20%). Route is around the tricuspid valve, inbetween the vena caval orifices.

Seen as a sawtooth appearance in inferior leads.

A regular atrial tachycardia and therefore usually a regular ventricular rhythm as well; sometimes if there is impairment of conduction through the AV node by disease or drugs, the ventricular response will be irregular depending on how variable the atrio-ventricular block is e.g. varying between 2:1, 3:1, 4:1 block etc.

### Causes:

Usually occurs only in a diseased heart:

Mitral valve disease  
Ischaemic heart disease  
Cardiomyopathy  
Hypertension

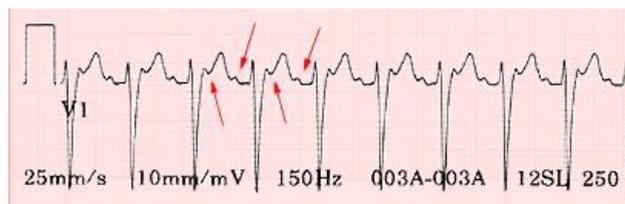
Or, secondary to:

Chronic obstructive pulmonary disease (COPD)  
Pulmonary embolism  
As a complication of cardiac surgery

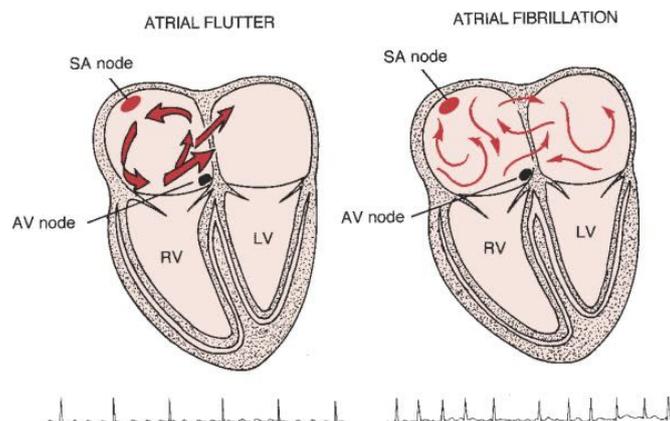
Atrial rate often 300 pm, with a physiological AV block → 2:1 conduction, so ventricular rate = 150 bpm. Conduction block may increase to 3:1 → ventricular rate = 100 bpm.; 4:1 → ventricular rate = 75 bpm. (AV node cannot normally conduct impulses faster than 200 per minute)

### Treatment:

1. Anticoagulation
2. Slow conduction through AV node with  $\beta$  blocker, Calcium channel blocker or Digoxin. ( $\beta$  blockers inhibit sympathetic tone of AV node, Digoxin enhances parasympathetic tone of AV node, Ca channel blockers directly inhibit impulse conduction by impairing cellular entry of Calcium ions)
3. Antiarrhythmic agents, such as Amiodarone
4. DC cardioversion
5. Radiofrequency ablation of tract
6. Atrial pacemaker



The arrows are directed at the flutter waves; this patient has a physiological 2:1 AV block (every 2<sup>nd</sup> flutter wave is blocked at the AV node, as it is still in its refractory period, having just conducted the previous flutter wave to the ventricles)



Comparison of mechanisms of atrial flutter and atrial fibrillation (AF). Atrial flutter is typically due to a large reentrant wave originating in the right atrium. With typical atrial flutter, the wave spreads in counterclockwise direction. AF is attributed to either multiple reentrant wavelets and/or to multiple sites of atrial automaticity.

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