ECG Interpretation.
Hypertrophy and enlargement of the heart.
Automacity disorders.

Outline

1. Review of the conduction system
2. ECG leads and recording
3. Normal ECG and its variants
4. Interpretation and reporting of an ECG
5. Automacity disorders
6. Hypertrophy and enlargement of the heart.

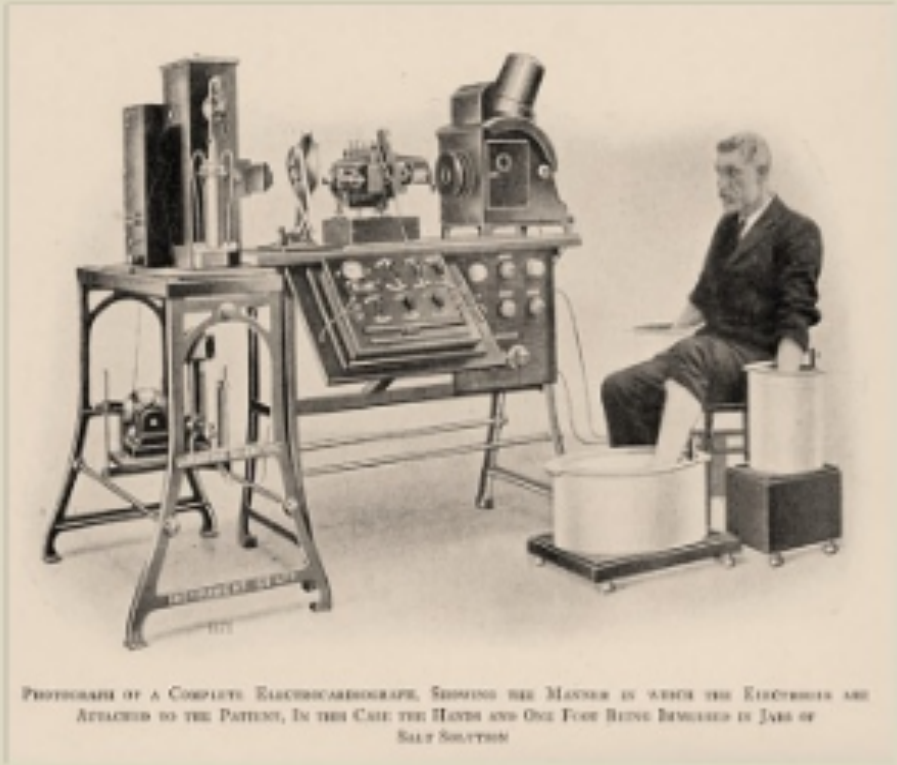
History

- 1842- Italian scientist Carlo Matteucci realizes that electricity is associated with the heart beat
- 1876 – Irish scientist Marey analyzes the electric pattern of frog`s heart
- 1895- Wiliam Einthoven, credited for the invention of EKG
- 1906- using the string electrometer, Wiliam Einthoven, diagnoses some heart problem
- 1924 - the noble prize for physiology or medicine is given to William Einthoven for his work on EKG
- 1938 -AHA and Cardiac society of great Britain defined and position of chest leads
- 1942- Goldberger increased Wilson’s Unipolar lead voltage by 50% and made augmented leads.

1903

Willem Einthoven

A Dutch doctor and physiologist. He invented the first practical electrocardiogram and received the Nobel Prize in Medicine in 1924 for it

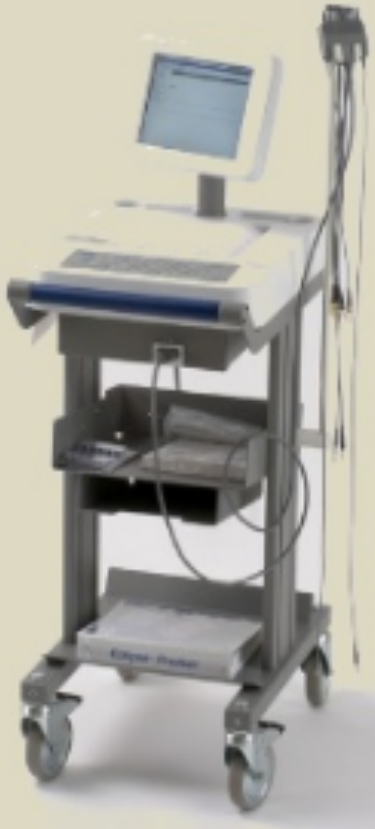


Photograph of a Complete Electrocardiograph, Showing the Manner in which the Electrodes are Attached to the Patient, In this Case the Hands and One Foot Being Immersed in Jars of Salt Solution

NOW

Modern ECG machine

has evolved into compact electronic systems that often include computerized interpretation of the electrocardiogram.



The ECG provides information regarding the electrical activity of the heart and offers:

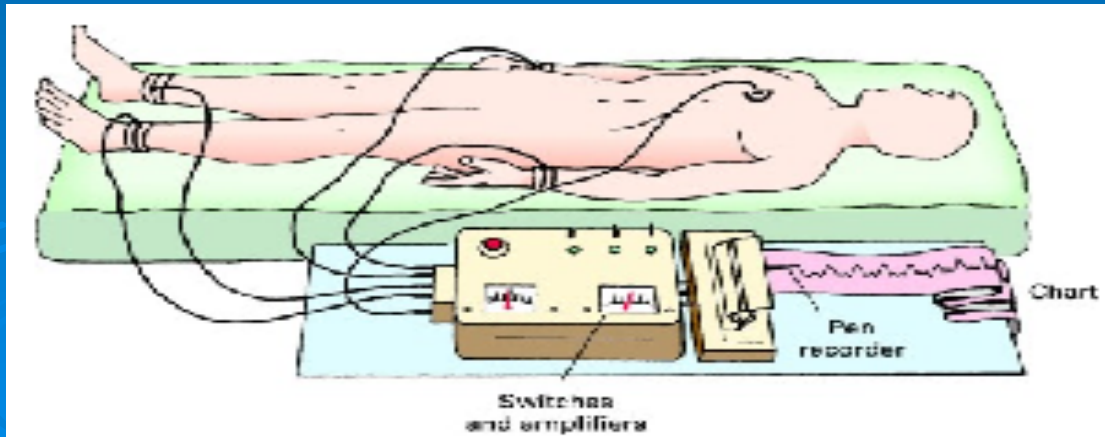
- the possibility to assess the heart‘s ability to generate electrical impulses (***automacity*** or ***chronotropy***);
- to conduct action potentials (***conductivity or drompotropy***);
- the ability of cardiac cells to respond to electrical impulses (***excitability or bathmotropy***);
- but offers no information about contractility (inotropy) or relaxation (lusiotropy).

The genesis of the electrocardiogram

The electrocardiogram (ECG or EKG)- provides a general picture regarding the electrical activity of the heart, recording the electrical changes that take place at the surface of cardiac myocytes at different moments of the cardiac cycles.

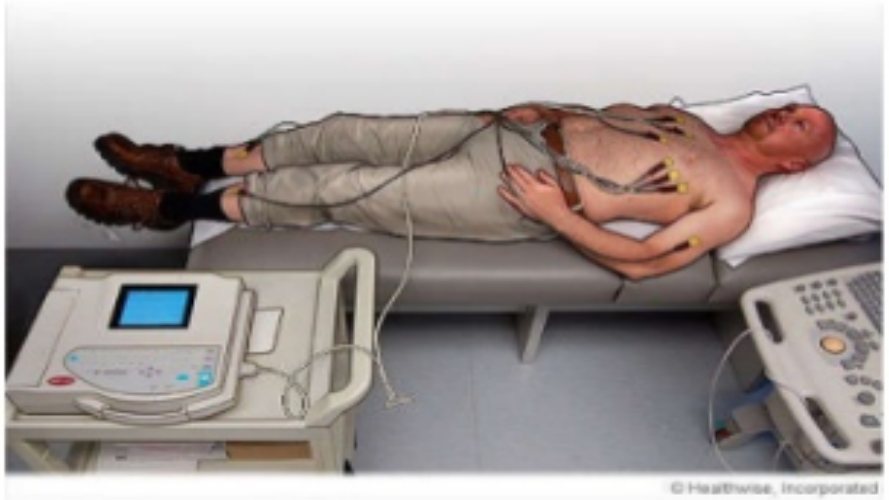
The device used for recording the ECG is called electrocardiograph. The main components of an electrocardiograph are:

- the signal acquisition system- includes the electrodes and the cables;
- the amplification and signal filtering system, used to amplify the relatively small potentials collected by the electrodes (in the order of mV) and to limit the artifacts.
- the signal charting system, displays the ECG trace either on millimeter paper or an a screen.

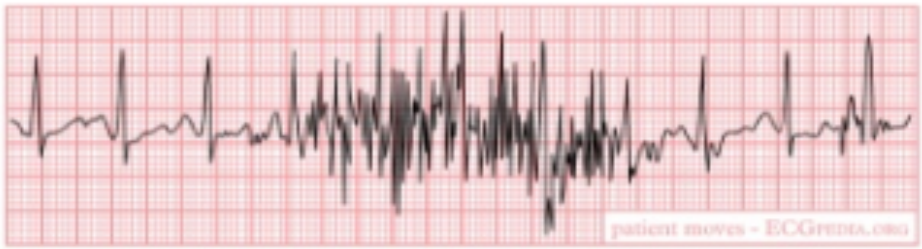


HOW TO DO ELECTROCARDIOGRAPHY

1. Place the patient in a supine or semi-Fowler's position. If the patient cannot tolerate being flat, you can do the ECG in a more upright position.
2. Instruct the patient to place their arms down by their side and to relax their shoulders.
3. Make sure the patient's legs are uncrossed.
4. Remove any electrical devices, such as cell phones, away from the patient as they may interfere with the machine.
5. If you're getting artifact in the limb leads, try having the patient sit on top of their hands.
6. Causes of artifact: patient movement, loose/defective electrodes/apparatus, improper grounding.



Patient, supine position

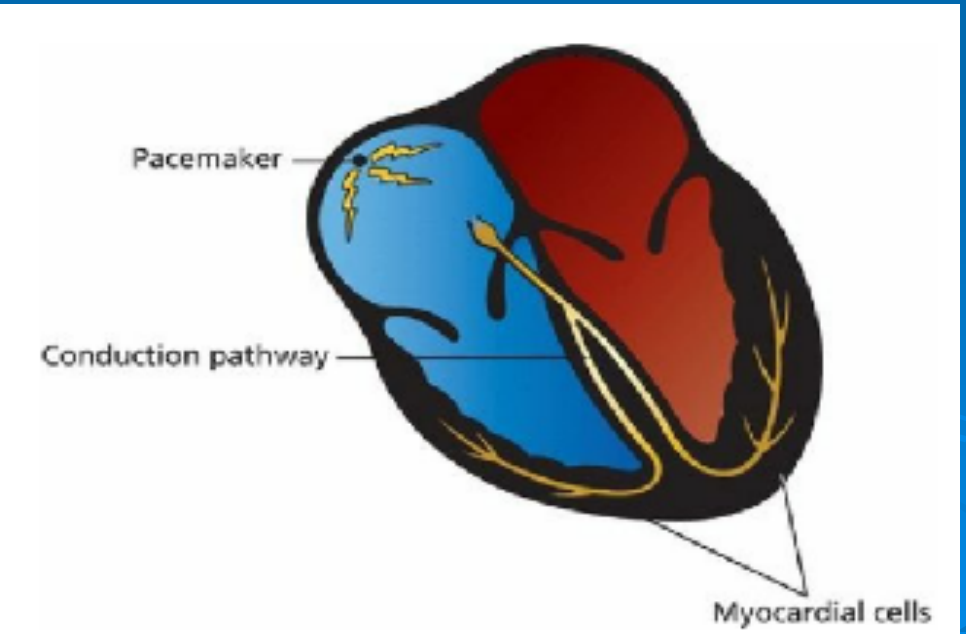


An ECG with artifacts.

The cells of the heart

The heart consists of three types of cells:

- Pacemaker cells- under normal circumstances, the electrical power source of the heart
- Electrical conducting cells- the hard wiring of the heart
- Myocardial cells- the contractile machinery of the heart



Sinoatrial node (SA) node or sinus node – the dominant pacemaker cell of the heart.

- located high up in the right atrium:
  - a branch from SA node is sent to left atria
  - it initiates all heart beat and determine heart rate
  - the wave front travels through the right and left atria in a centrifugal manner.

Atrioventricular node (AV) **:**

- located in the wall of the right atrium just next to the opening of the coronary sinus,
- serve as electrical gateway to the ventricles.

Bundle of His (AV bundle) divided:

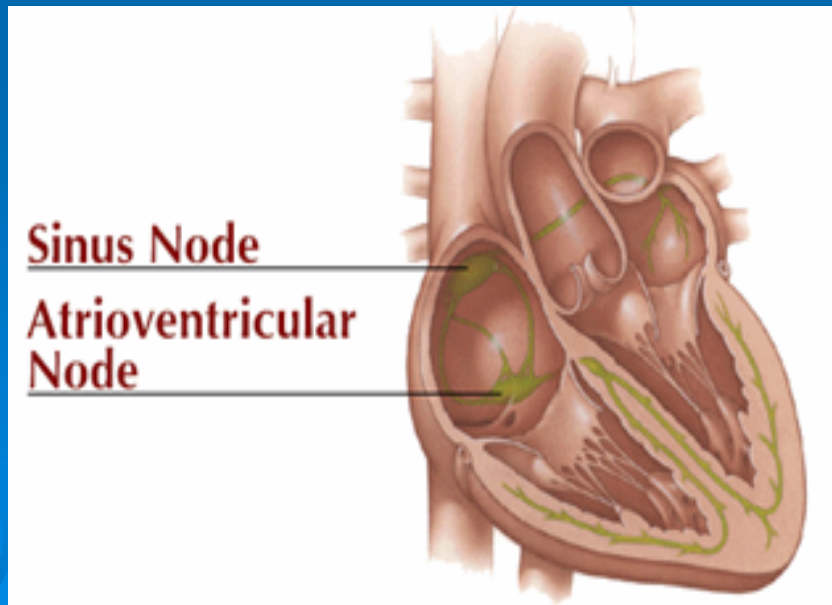
- LBB and RBB

The LBB divides:

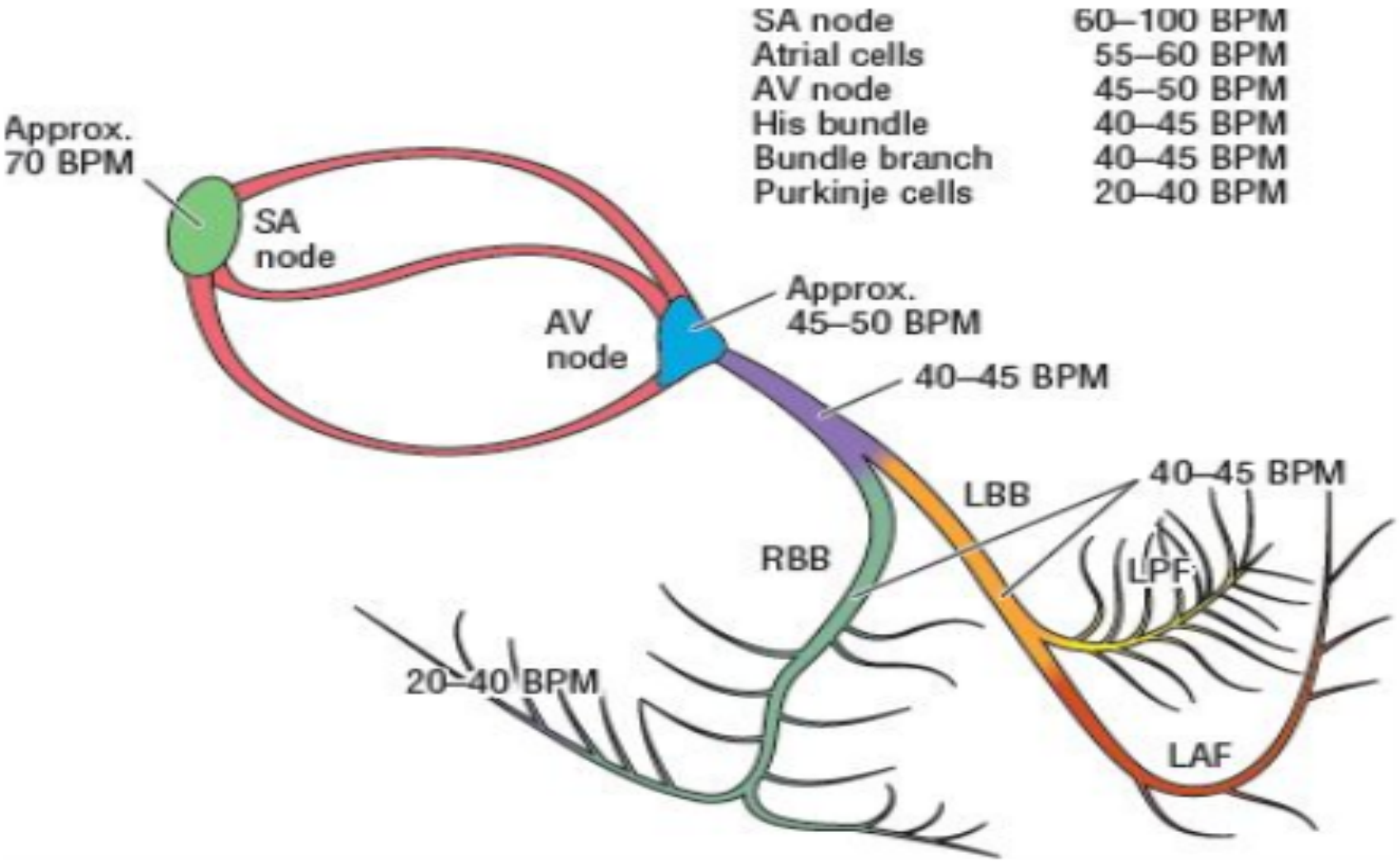
- anterior and posterior branch.

More distally the bundles ramify into Purkinje fibers.

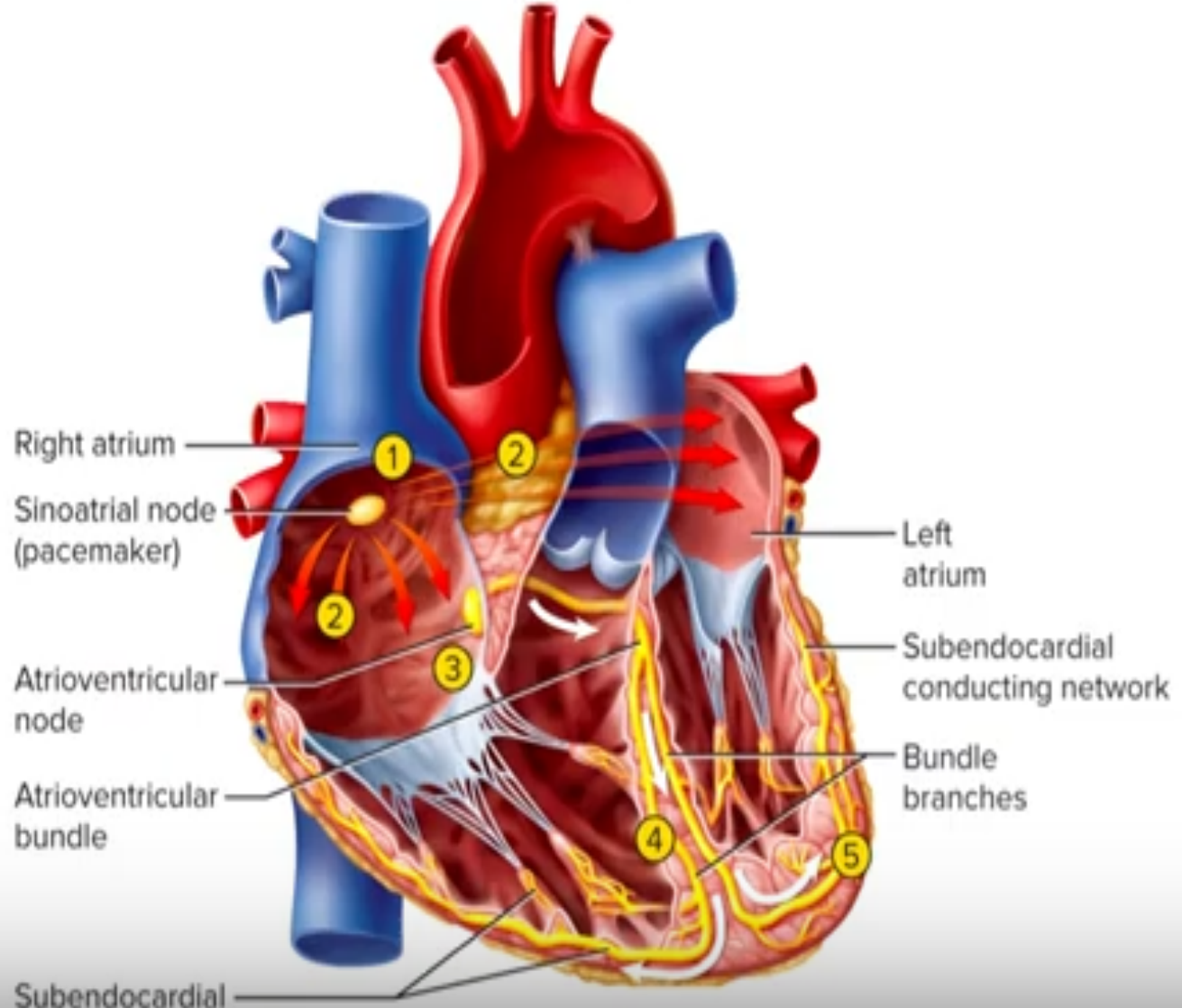
- Bachman‘s bundle - fibers at the top of the intraatrial septum that allow rapid activation of the left atrium from the right.



Intrinsic rates of pacing cells



The electrical conduction system of the heart



1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Subendocardial conducting network distributes excitation through ventricular myocardium.

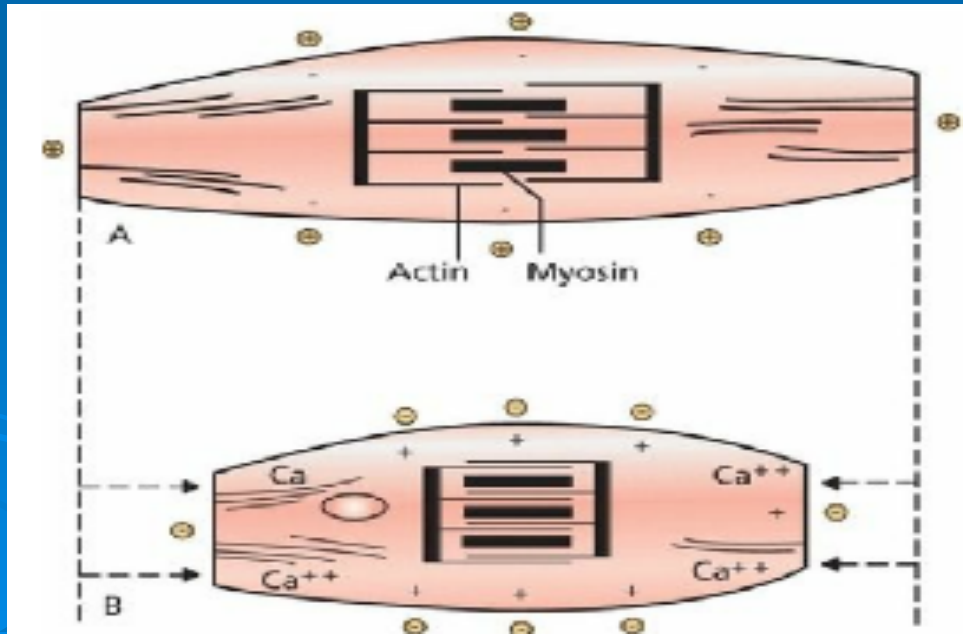
Myocardial cells

- the largest part of the heart tissue;
- are responsible for the heavy labour of repeatedly contracting and relaxing, delivering blood to the rest of the body;
- contain an abundance of the contractile proteins and myosin.

When a wave of depolarization reaches a myocardial cell, calcium is released within the cell, causing the cell to contract (this process- excitation- contraction coupling).

A- resting myocardial cell

B- a depolarized, contracted myocardial cell

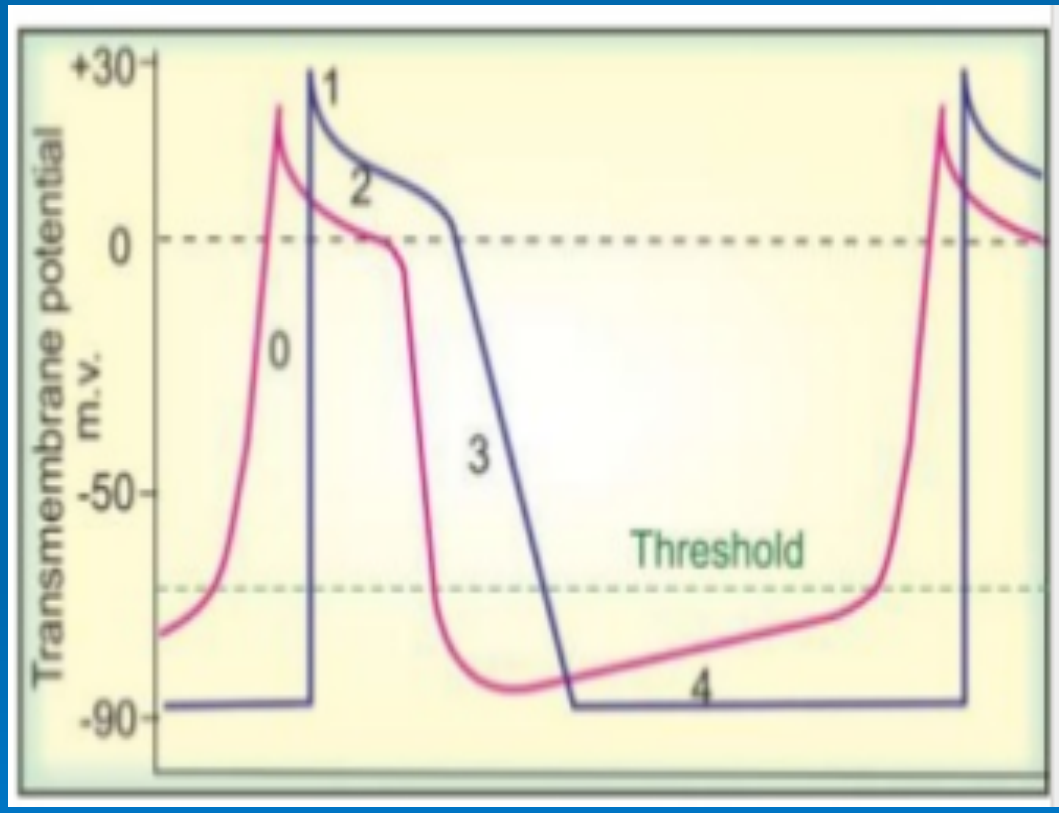


Cardiac Electrophysiology

Impulse generation

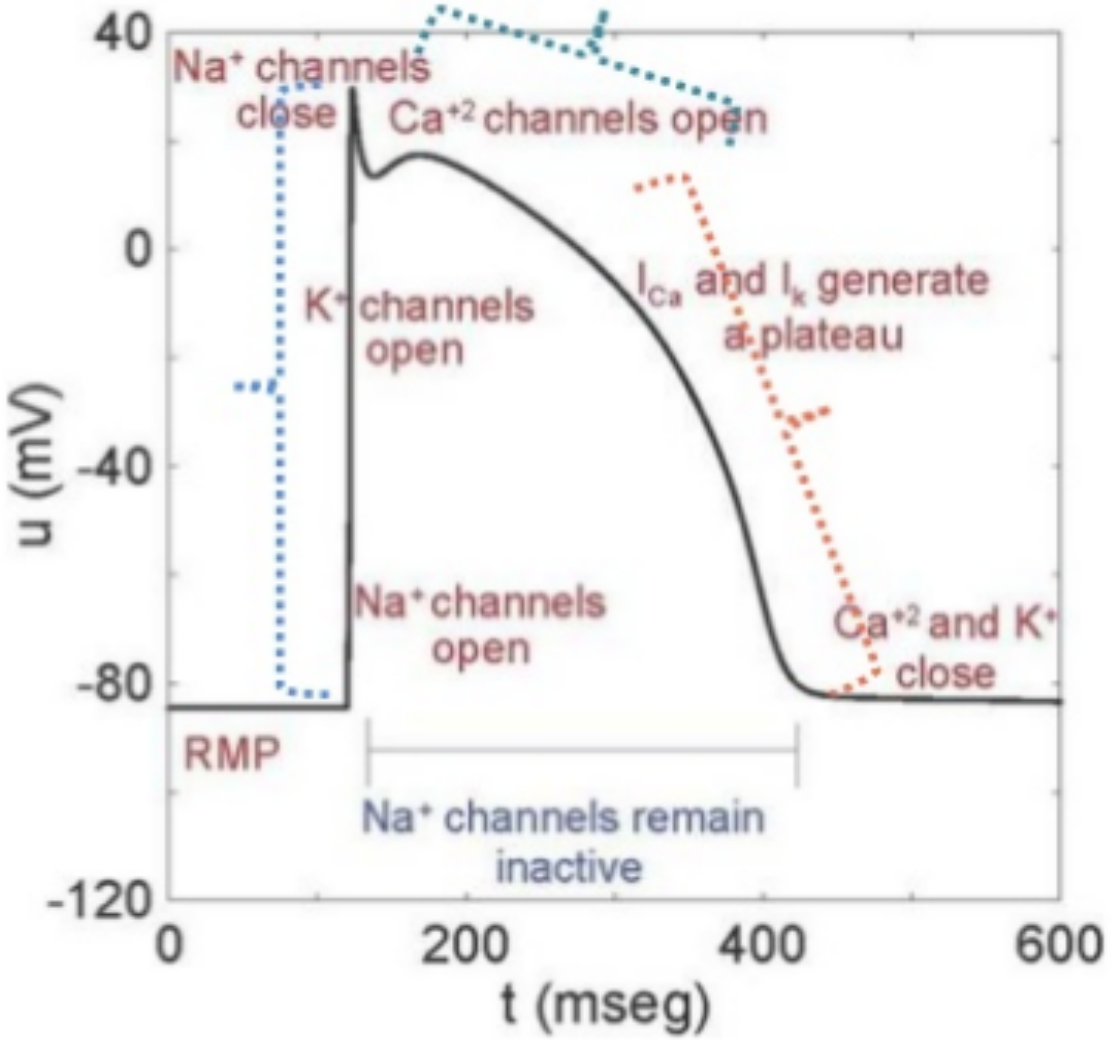
- ***Nonautomatic fibres***: Ordinary working myocardial fibers and cannot generate impulse of their own

- ***Automatic fibres***: SA node, AV node, His- Purkinje system.



Cardiac Electrophysiology

- Impulse generation



Rapid depolarization due to opening of voltage-dated fast Na+ channels

Plateau (maintained depolarization) due to opening of voltage-gated slow Ca+ channels and closing of some K+ channels

Repolarization due to opening of voltage-dated K+ channels and closing of Ca+ channels

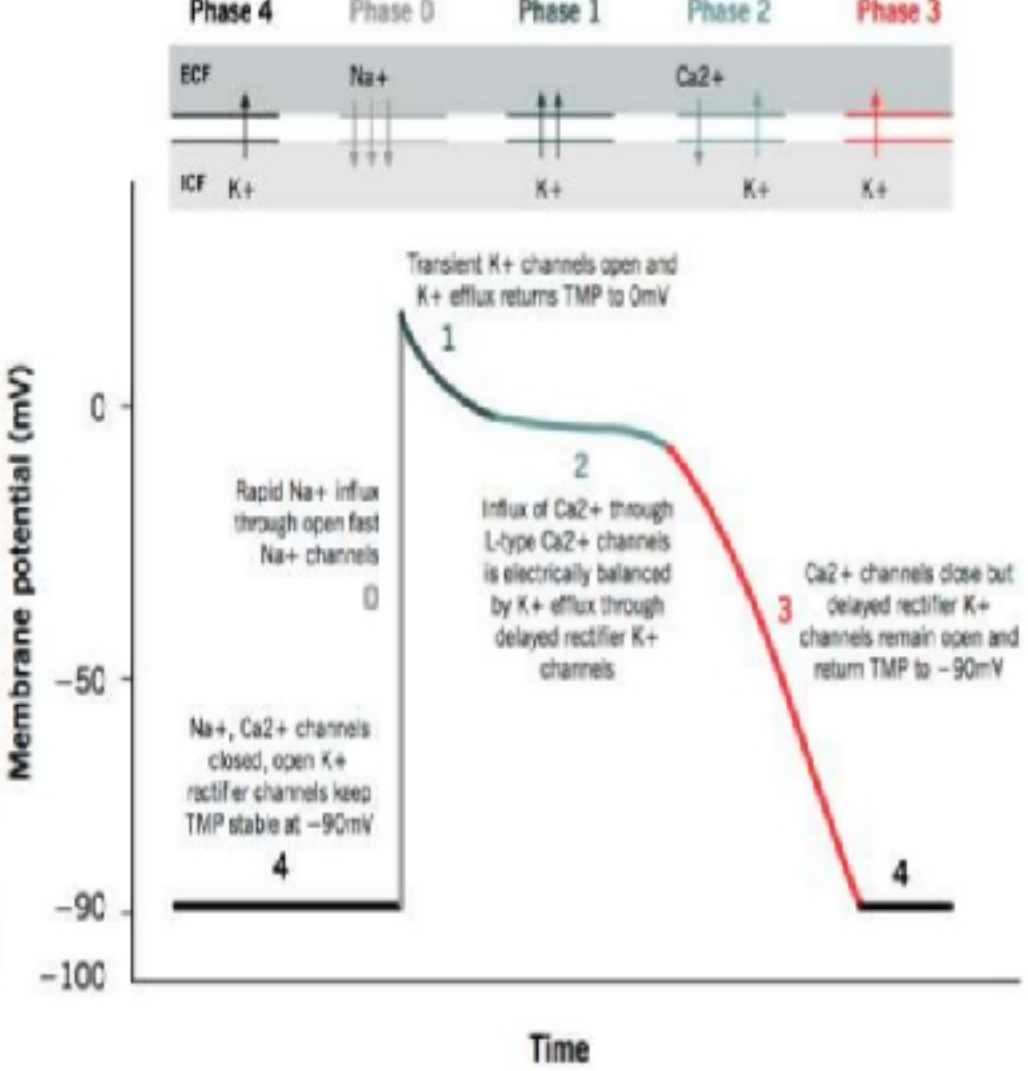
Rapid Na+ entry

Phase 1: Early depolarization
Ca++ slow entry

Phase 2: *Plateau*
continuous repolarization
Slow entry of sodium and calcium

Phase 3: Repolarization
Potassium outflow

Phase 4: Pacemaker potential



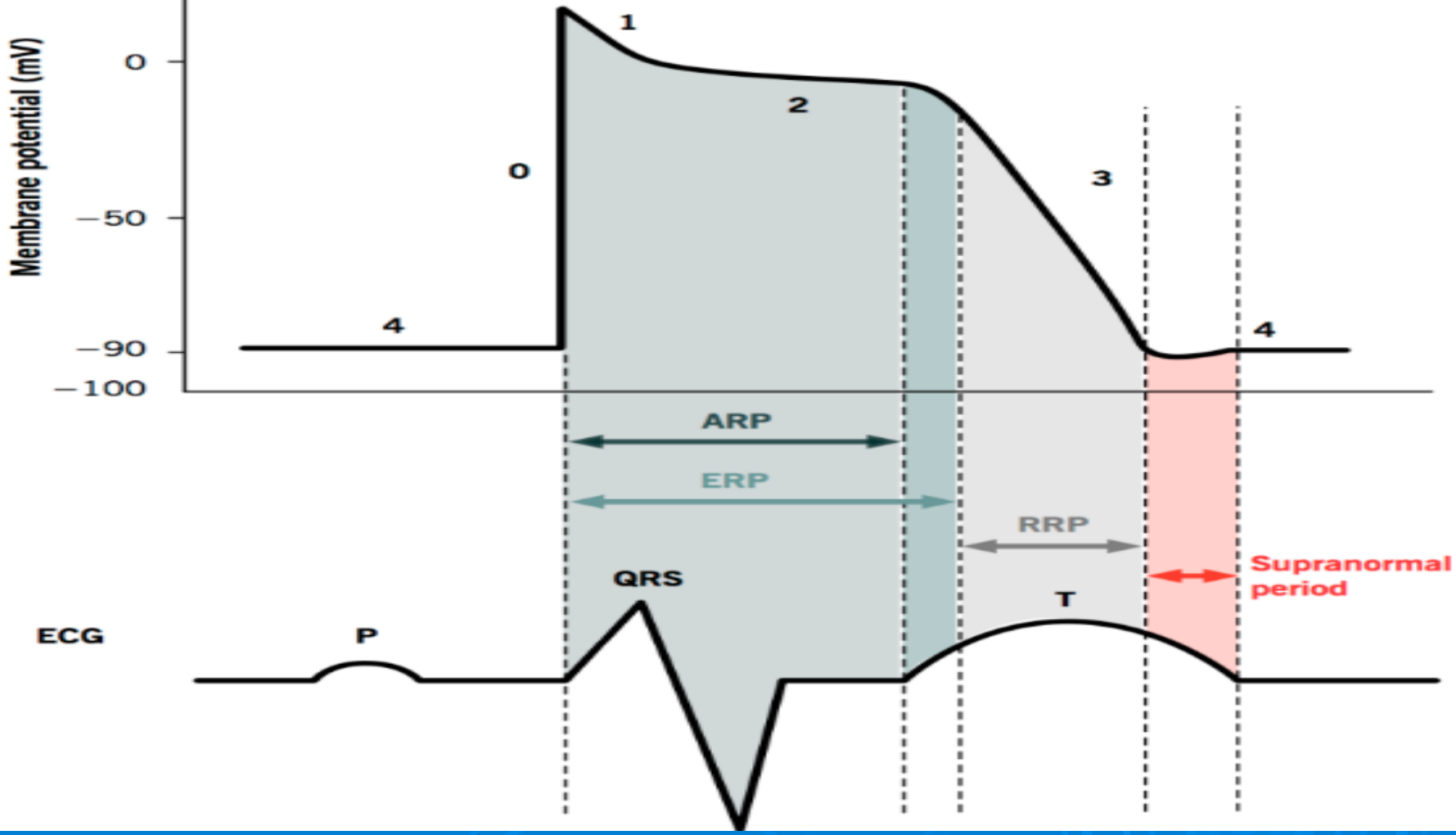
Phase 1 – 3: Refractory period

Ref: Pharmacol Ther. 2005;107(1):59-79.

Refractory periods in cardiac cycle

Grigoriy Ikonnikov and Eric Wong

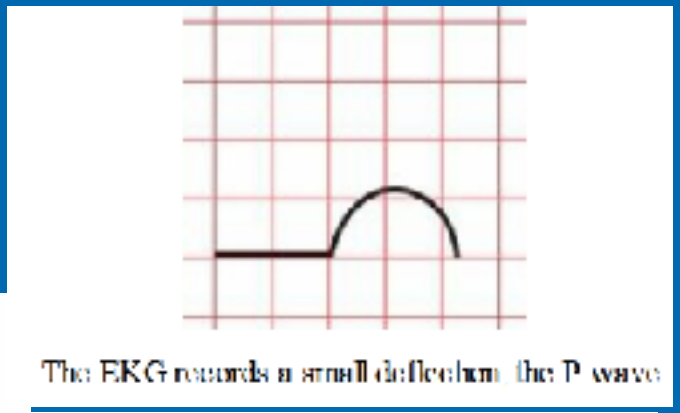
The refractory periods in cardiac muscles allow complete emptying of the ventricles prior to the next contraction. Refractoriness of each phase of the action potential is governed by the number of sodium channels ready to activate. The **absolute refractory period (ARP)** does not allow for any depolarizations. The **effective refractory period (ERP)** may allow for non-propagated depolarization. The **relative refractory period (RRP)** allows a stronger than normal stimulus to cause a full depolarization. The **supranormal period** is a hyperexcitable period where even a weak stimulus can cause an action potential. See text for details.



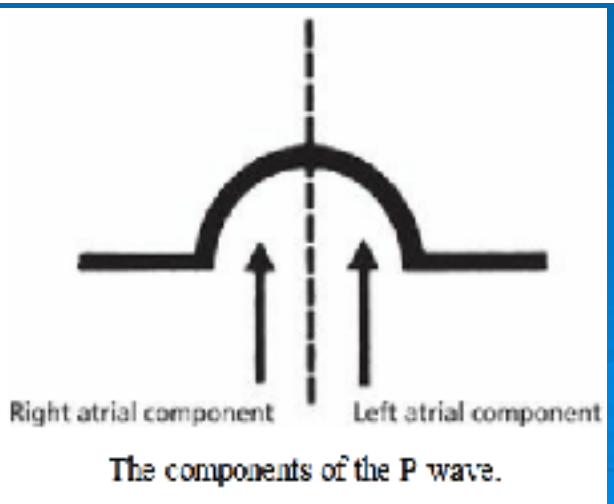
Ikonnikov G, Yelle D. Physiology of cardiac conduction and contractility. Available in http://www.pathophys.org/physiology -of-cardiac-conduction-and- contractility

Each wave or segment of the EKG coresponds to a certain event of the cardiac electrical cycle.

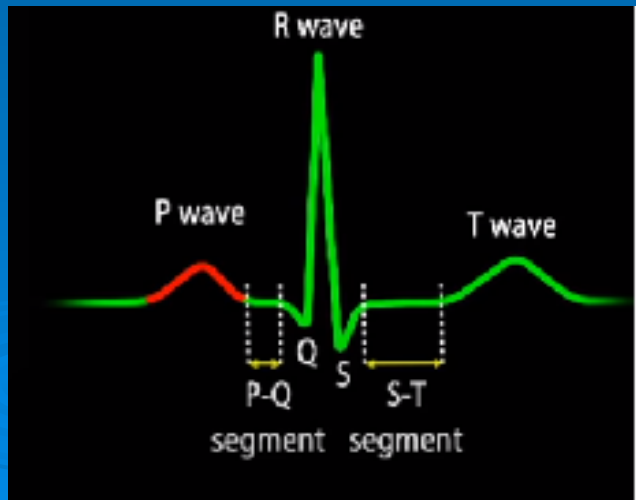
- The sinus nodes fires spontaneously, a wave of depolarization begins to spread outward into the atrial myocardium. During atrial depolarization and contraction, electrodes record a small electrical activity lasting a fraction of second- ***P wave.***



The EKG records a small deflection: the P wave

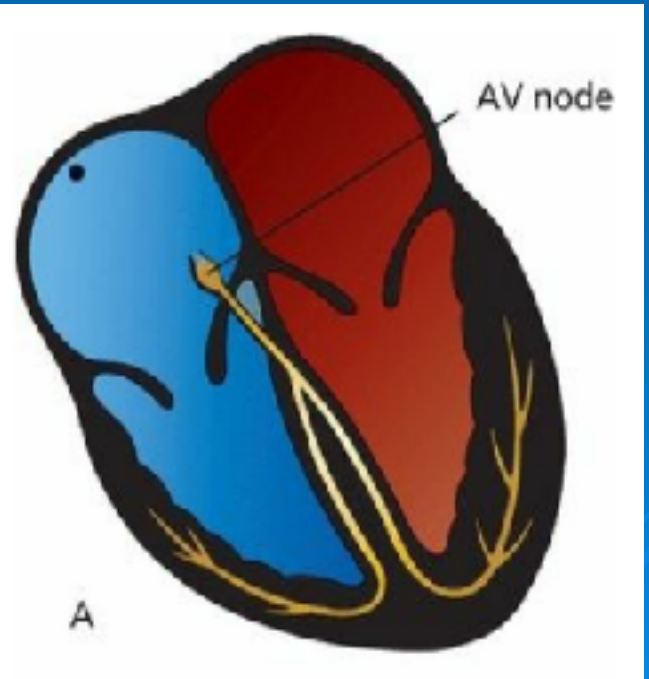


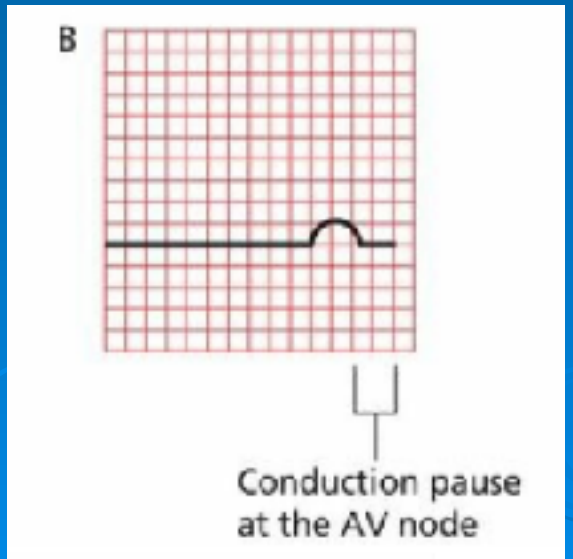
The components of the P wave.



A pause separates conduction from the atria to the ventricles.

- AV node slows conduction to a crawl. This pause lasts only a fraction of second.
- This physiological delay in conduction is essential to allow the atria to finish contracting before the ventricles begin to contract. This electrical wiring of the heart permits the atria to empty their volume of blood completely into the ventricles before the ventricles contract.



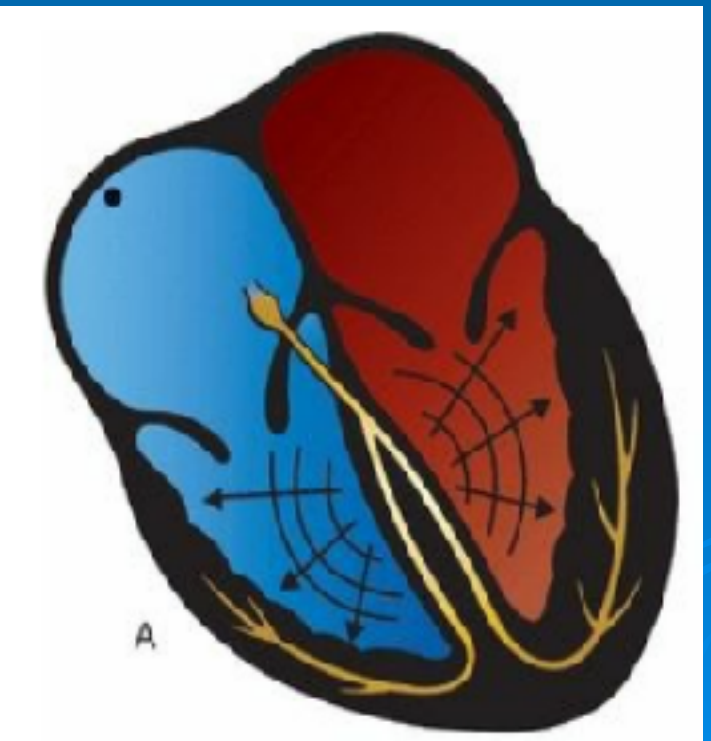


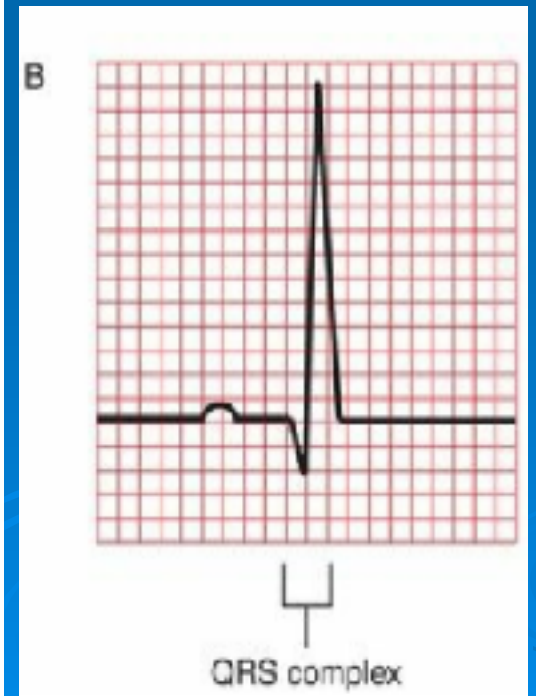
A- The wave of depolarization is briefly held up at the AV node

B-During this pause, the EKG falls silent; there is no detectable electrical activity.

The QRS complex marks the firing of AV node and represent ventricular depolarization.

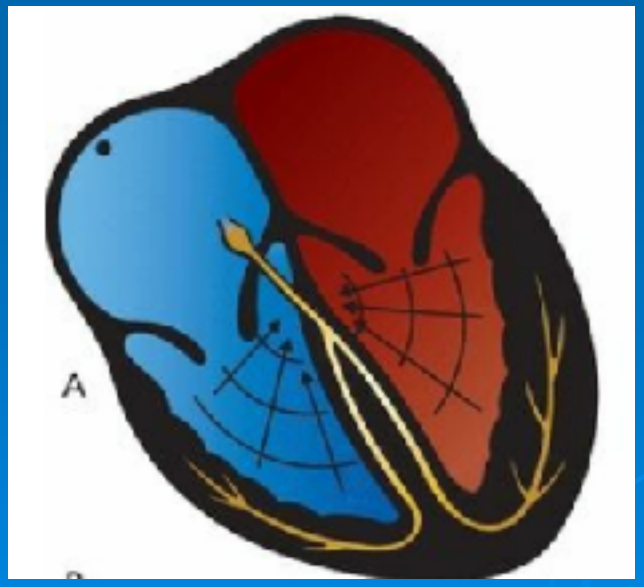
- Impulse travels to the bundle His, causing the depolarization of the interventricular septum, results in a small downward (negativ) deflection- ***Q wave.***
- R wave- the first upward deflection, produced by depolarization of the main mass of ventricles
- S wave- the first downward deflection following an upward deflection, the last phase of ventricular depolarization at the base of the heart.

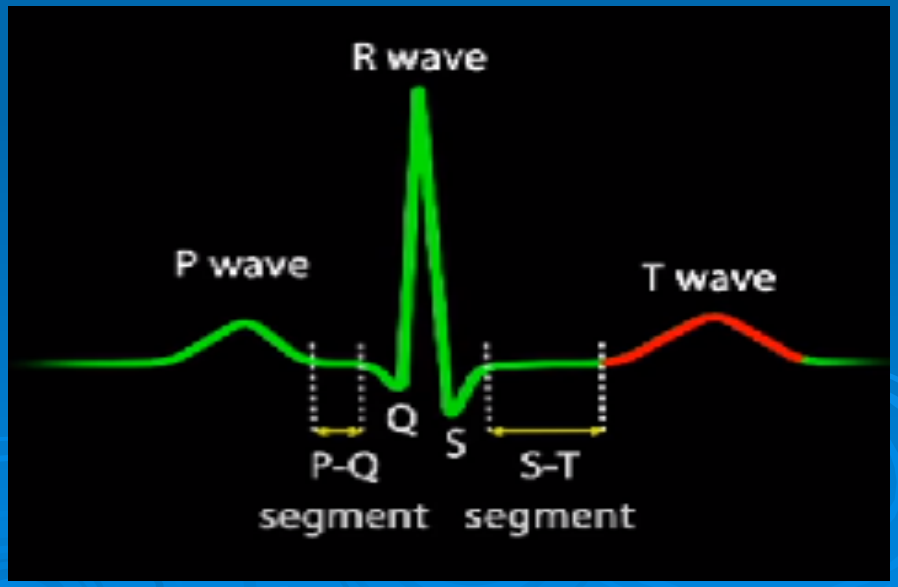


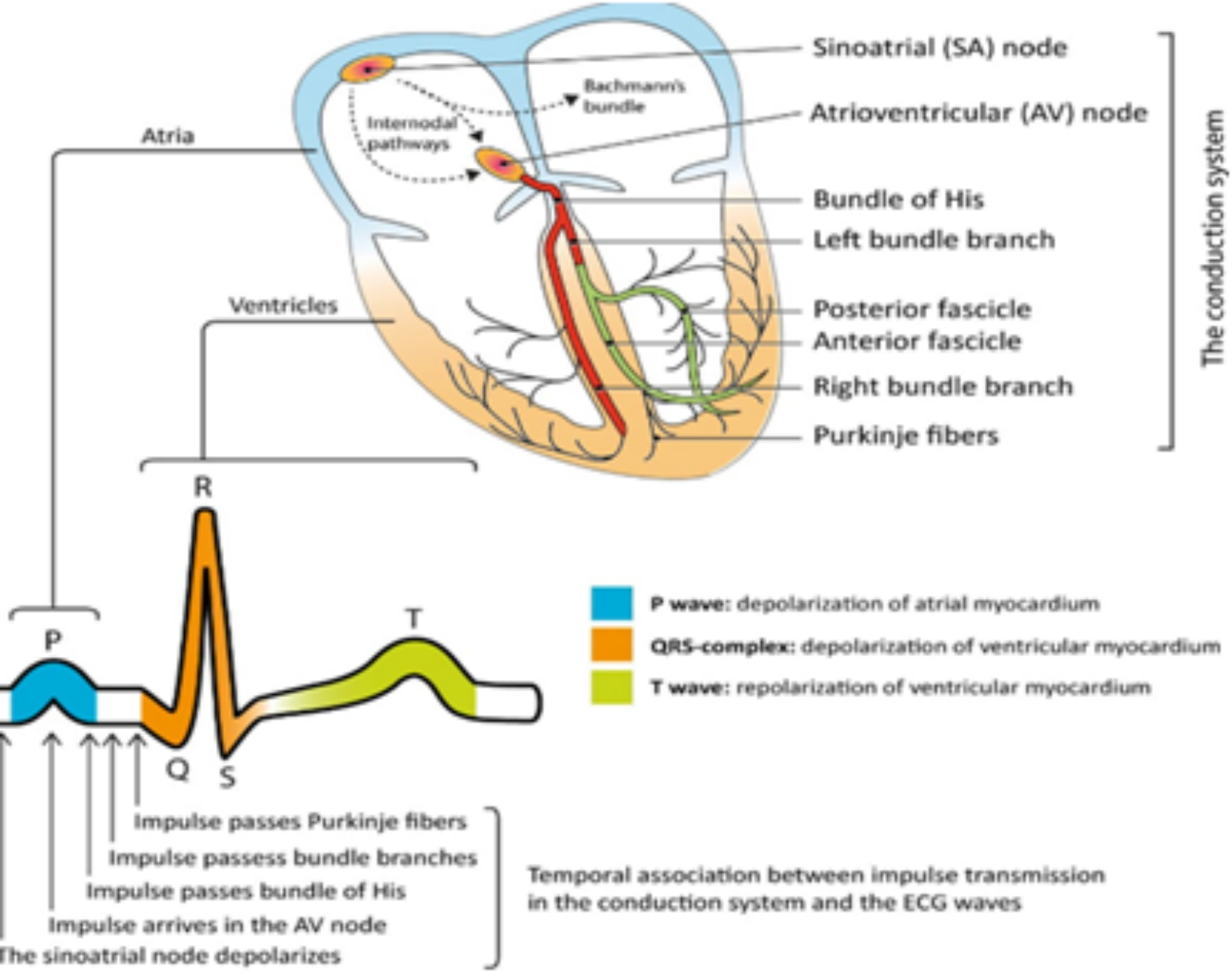


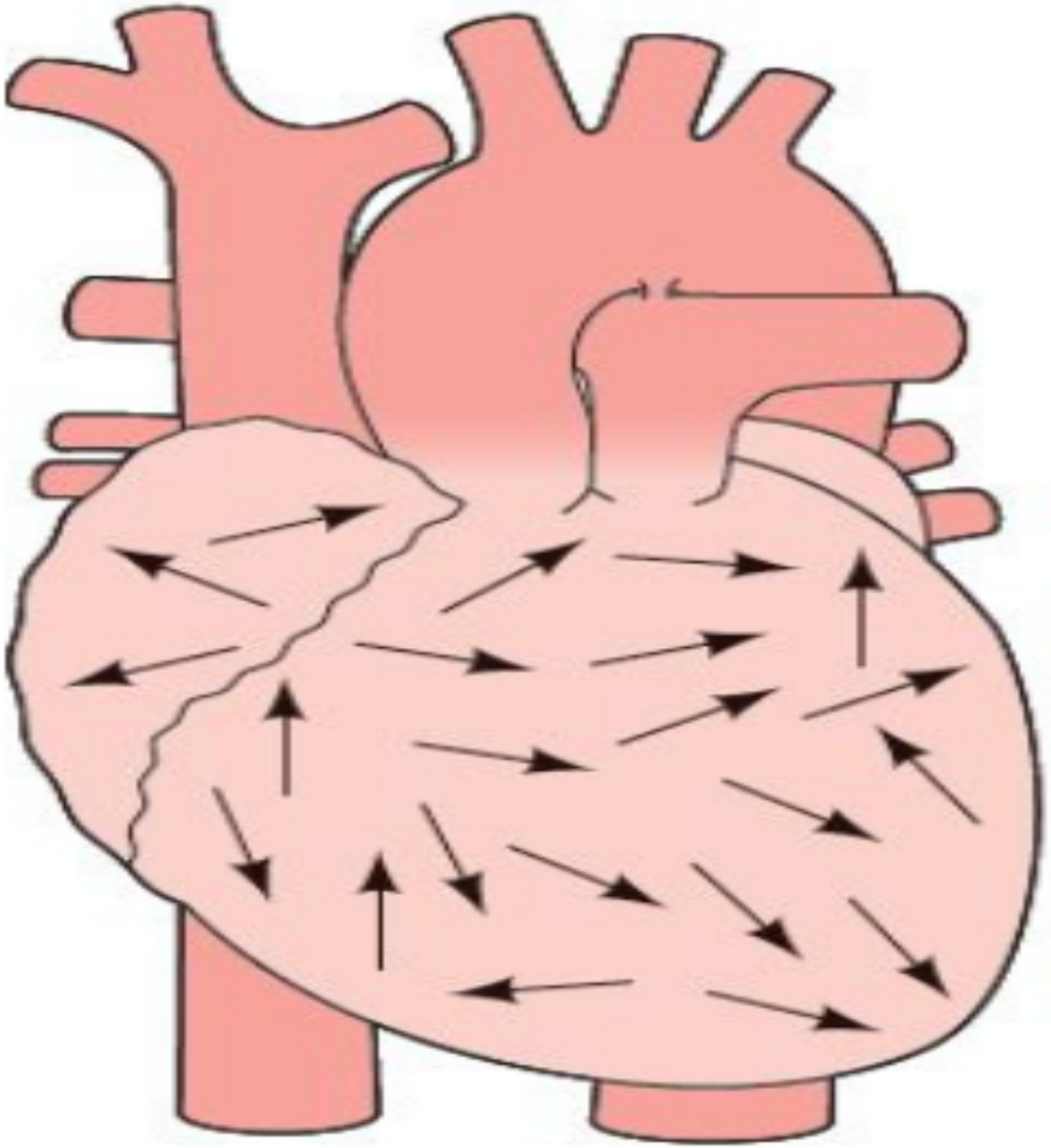
There is a wave of atrial repolarization as well, but it coincides with ventricular depolarization and is hidden by the much more prominent QRS complex.

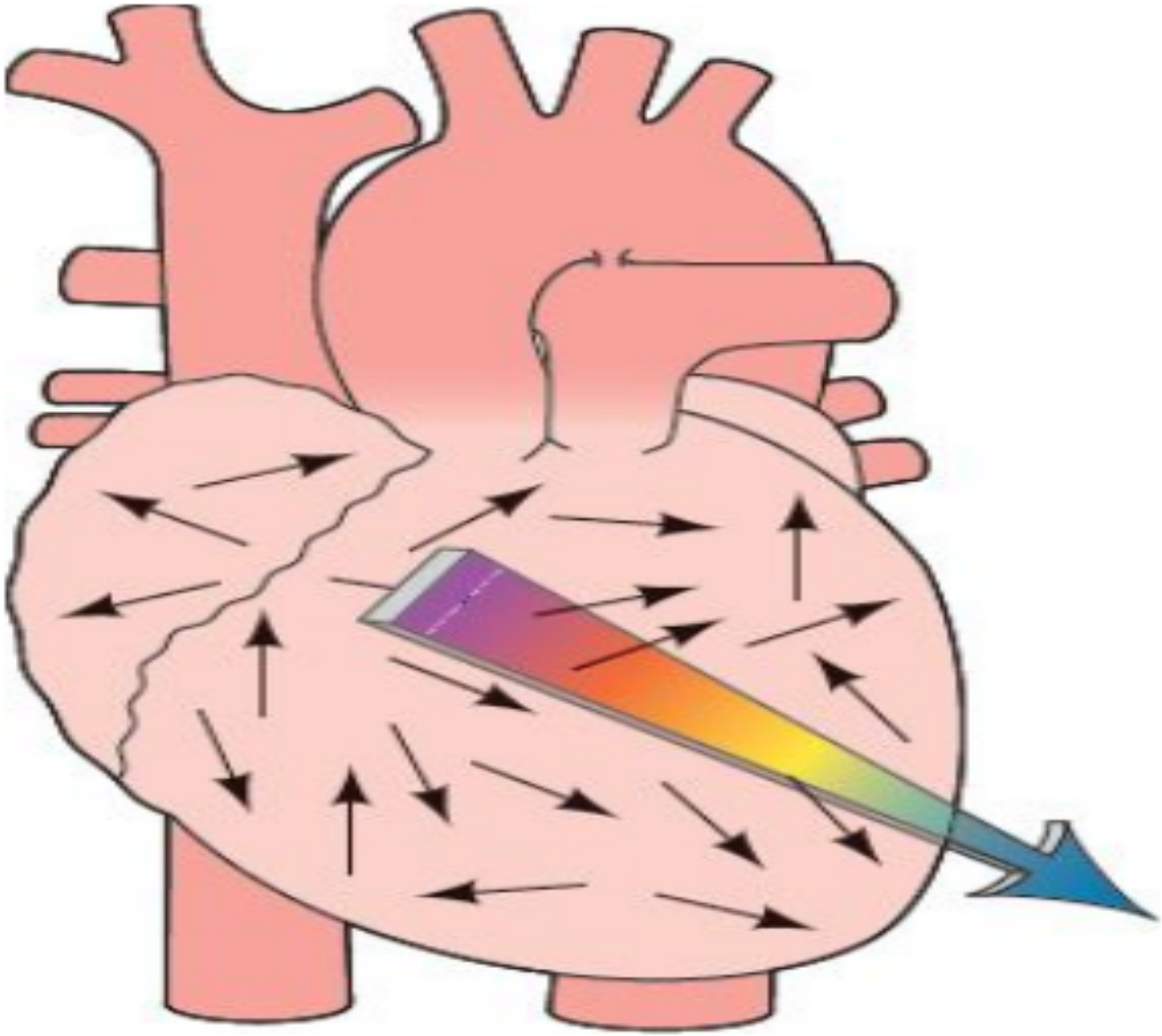
- ST segment reflects the plateau in the myocardial action potential action
- T wave represents ventricular repolarization immediately before the ventricular relaxation or ventricular diastole.
- Ventricular repolarization is a much slower process than ventricular depolarization.






Sinoatrial (SA) node
Bachmann's bundle
Internodal pathways
Atrioventricular (AV) node
Atria
Bundle of His
Left bundle branch
Ventricles
Posterior fascicle
Anterior fascicle
Right bundle branch
Purkinje fibers
The conduction system
R
T
P
Q
S
P wave: depolarization of atrial myocardium
QRS-complex: depolarization of ventricular myocardium
T wave: repolarization of ventricular myocardium
Impulse passes Purkinje fibers
Impulse passess bundle branches
Impulse passes bundle of His
Impulse arrives in the AV node
The sinoatrial node depolarizes
Temporal association between impulse transmission in the conduction system and the ECG waves



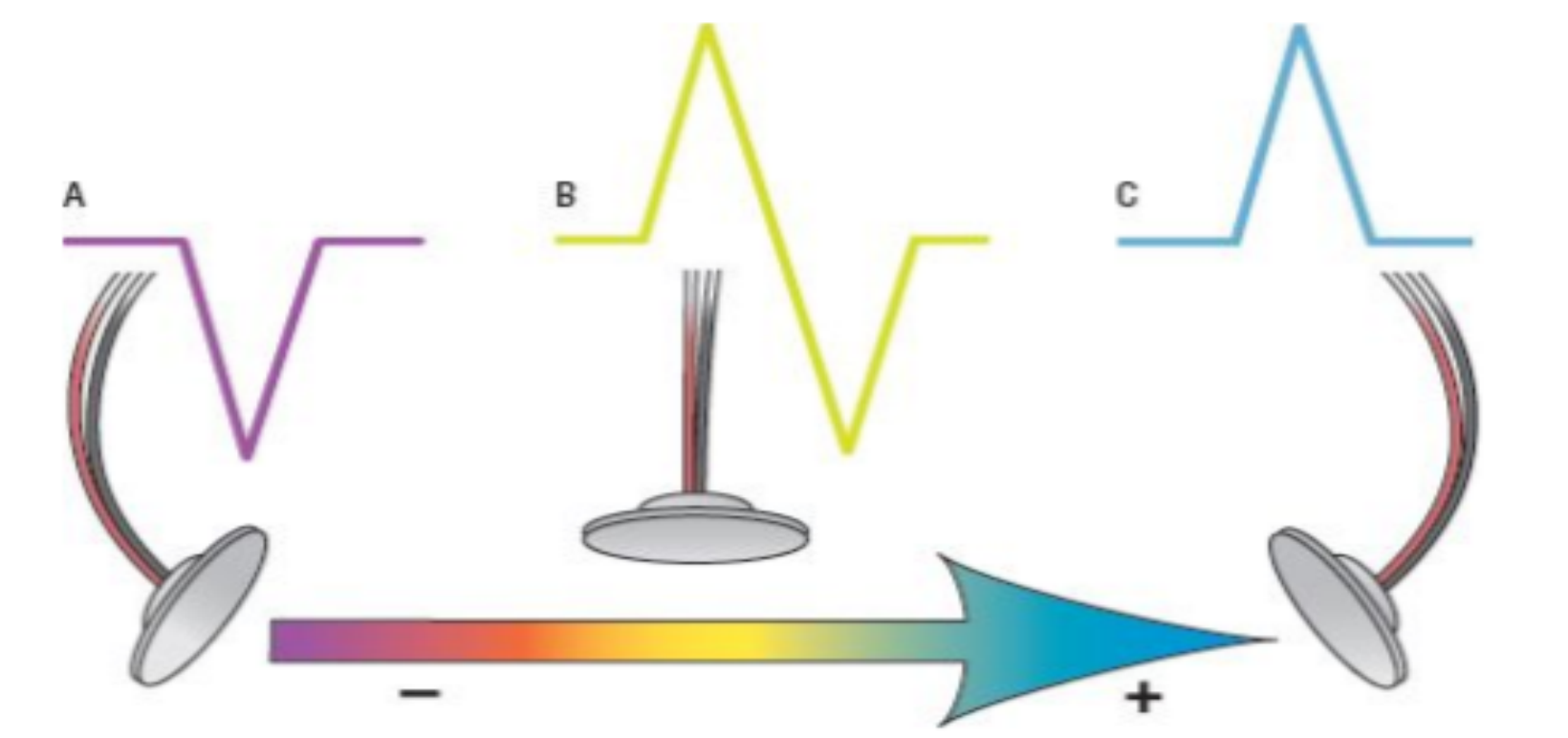


Sum of all ventricular vectors = electrical axis.

Electrodes and wave

- The electrodes are sensing devices that pick up the electrical activity occurring beneath them.

- Three different ECG resulting from the same vector, due to different lead placements.



Electrocardiographic leads

- In order to collect the potentials generated by electrical activity of the heart, electrodes are placed at the surface of the body.
- Graphically, each lead has a coresponding axis, each axis has an orientation.

There are three lead systems that make up the standard ECG:

- Standard Limb Leads (Bipolar): I, III & III
- Augmented Limb Leads (Unipolar): aVR, aVL & aVF
- Precordial Leads: V1- V6

Standard limb leads

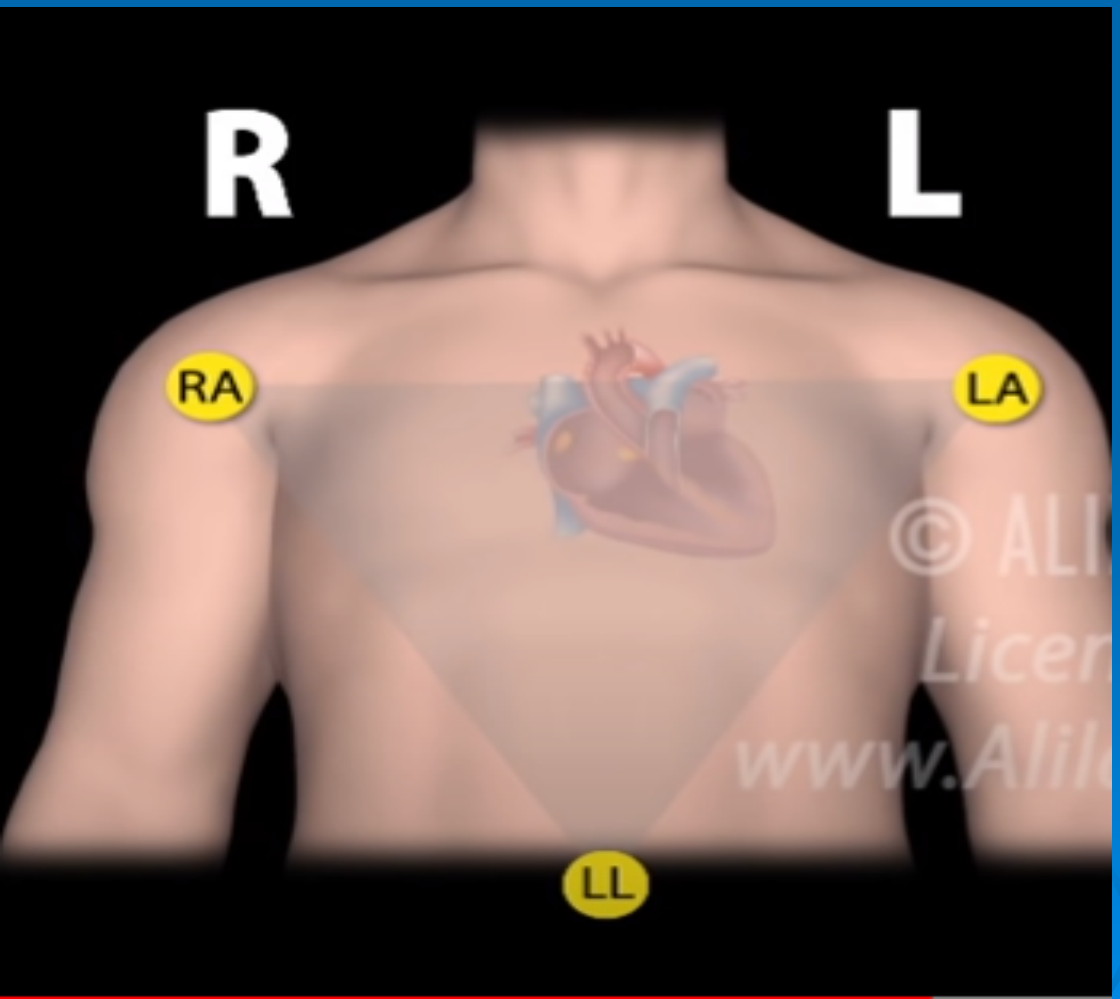
- are bipolar leads, exploring the activity of the heart in frontal plane.
- It is used three active electrodes and a grounding electrode.

The electrodes are named with the initiales of the words indicating their positions and are usually color- coded:

- Right upper limb –R (right)- red
- Left upper limb- L (left)-yellow
- Left lower limb- F(foot)- green

The ground limb- on the right lower limb and is usually black.

The standard (limb) leads



The measurements of a voltage require 2 poles: negative and positive.

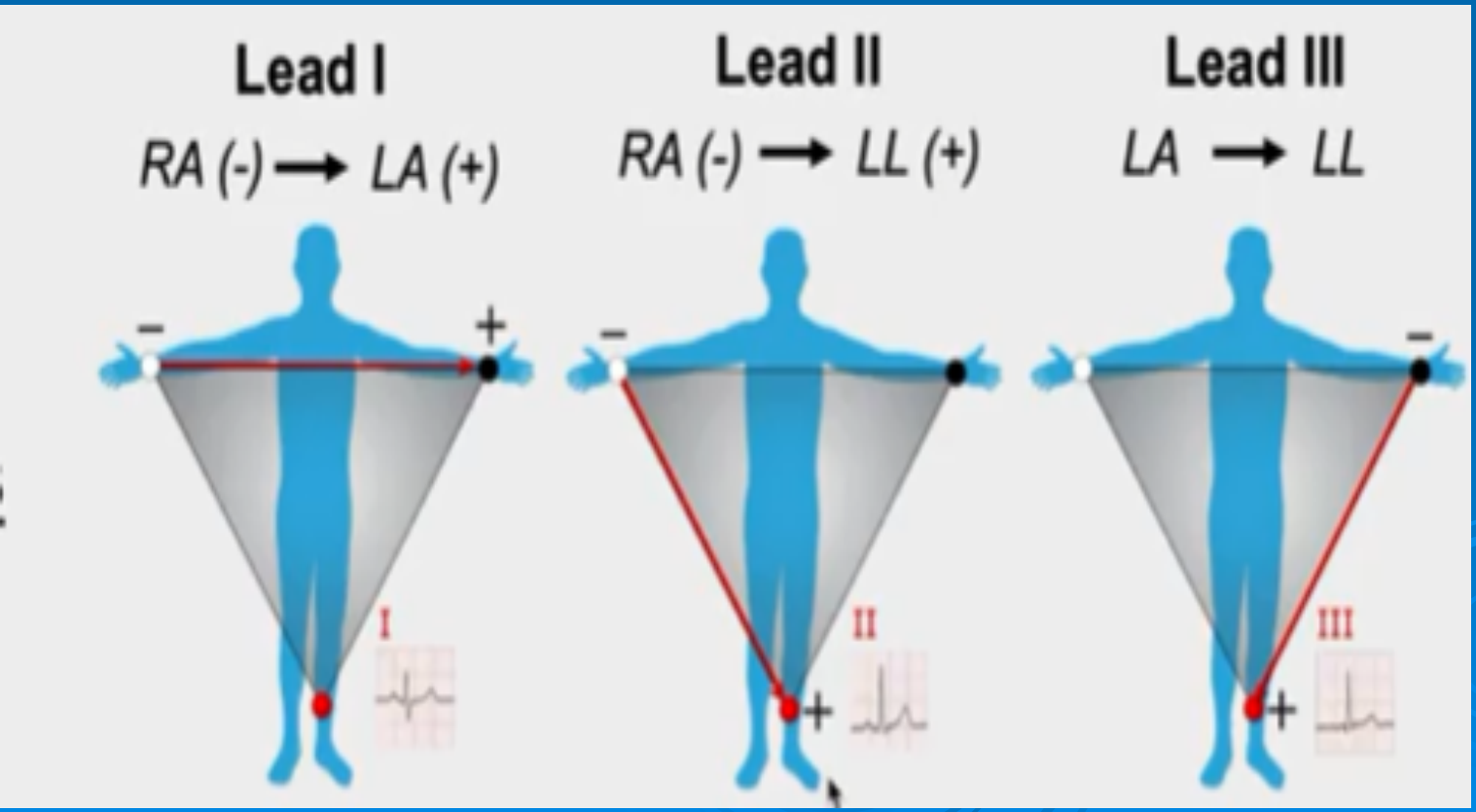
The standard (limb) leads

The electrodes are located on the limbs – one on each arm and one on the left leg.

I = LA - RA

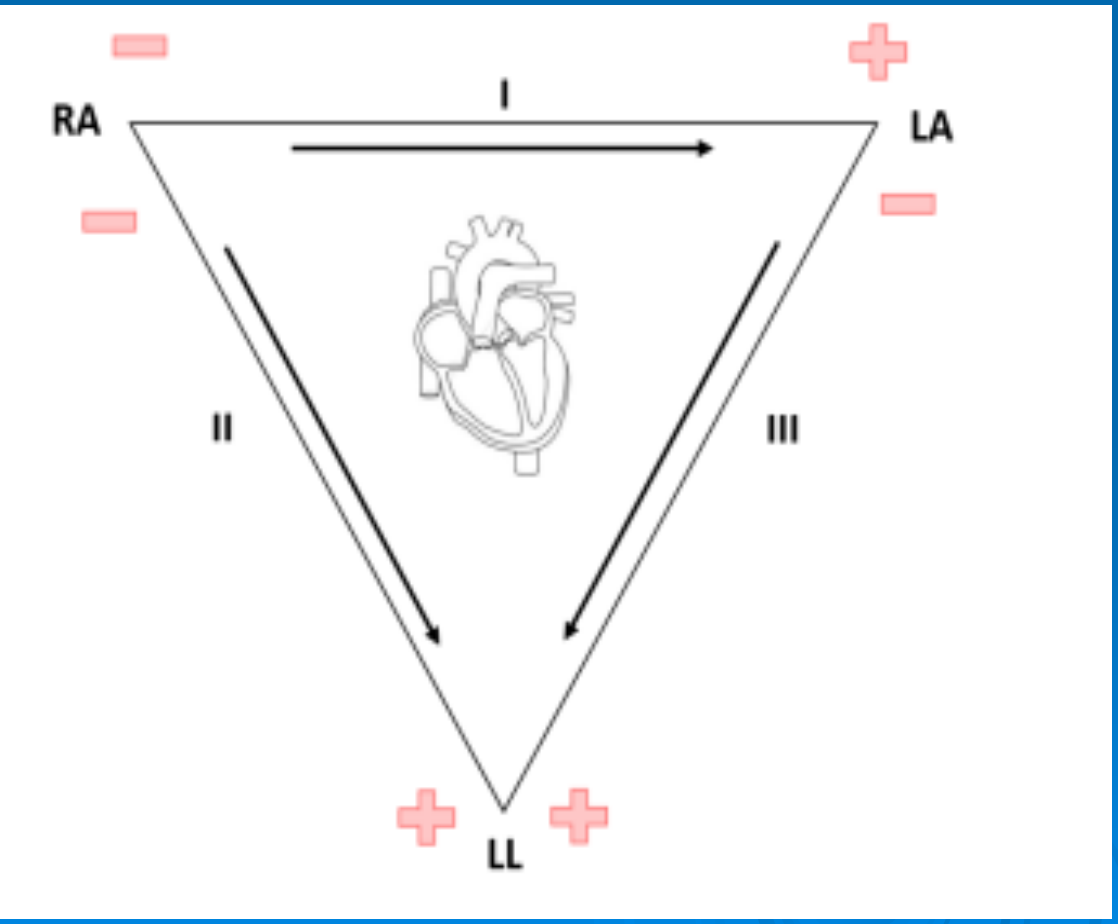
II = LL - RA

III = LL- LA



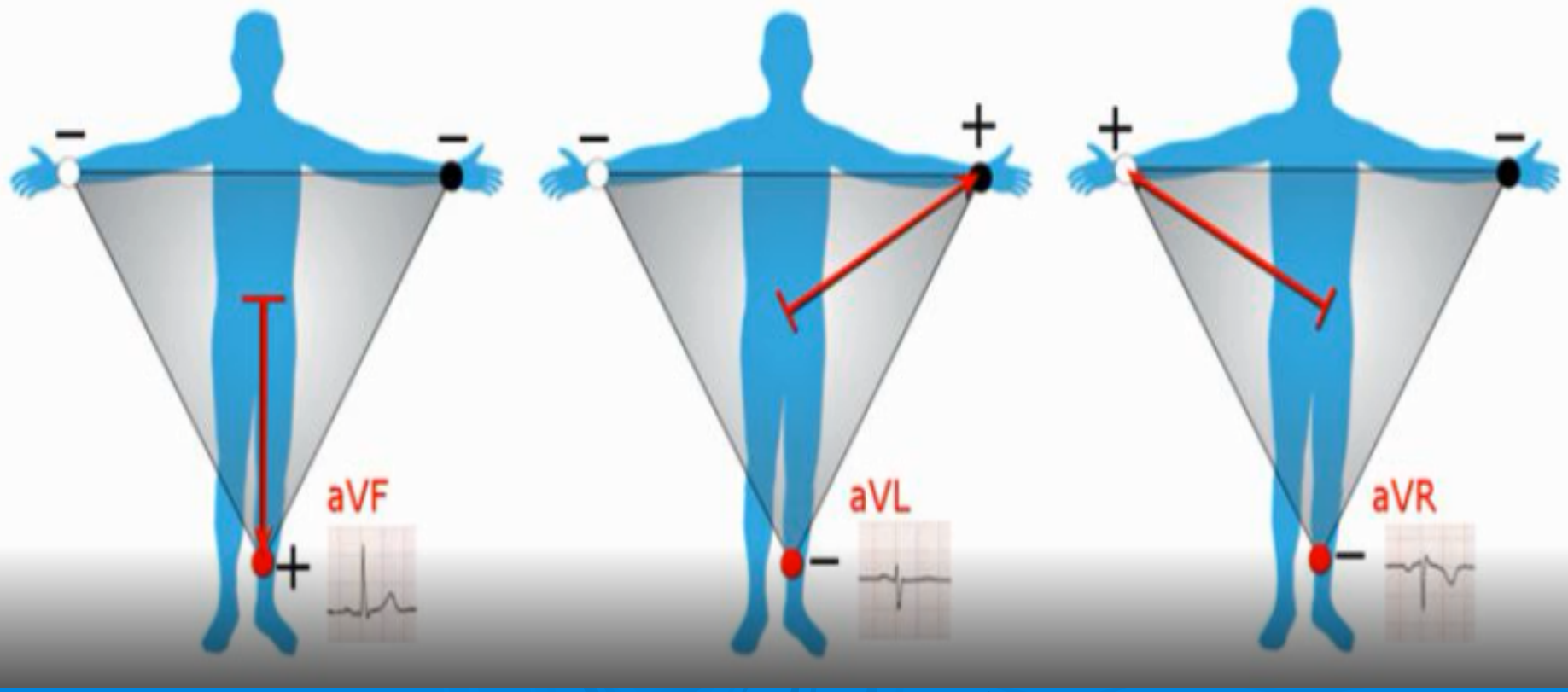
The three limb electrodes I, II and III form a triangle (**Einthoven's Equilateral Triangle**), at the right arm (RA), left arm (LA) and left leg (LL).

Einthoven's Law explains that Lead II's complex is equal to the sum of the corresponding complexes in Leads I and III and is given as **II = I + III.**



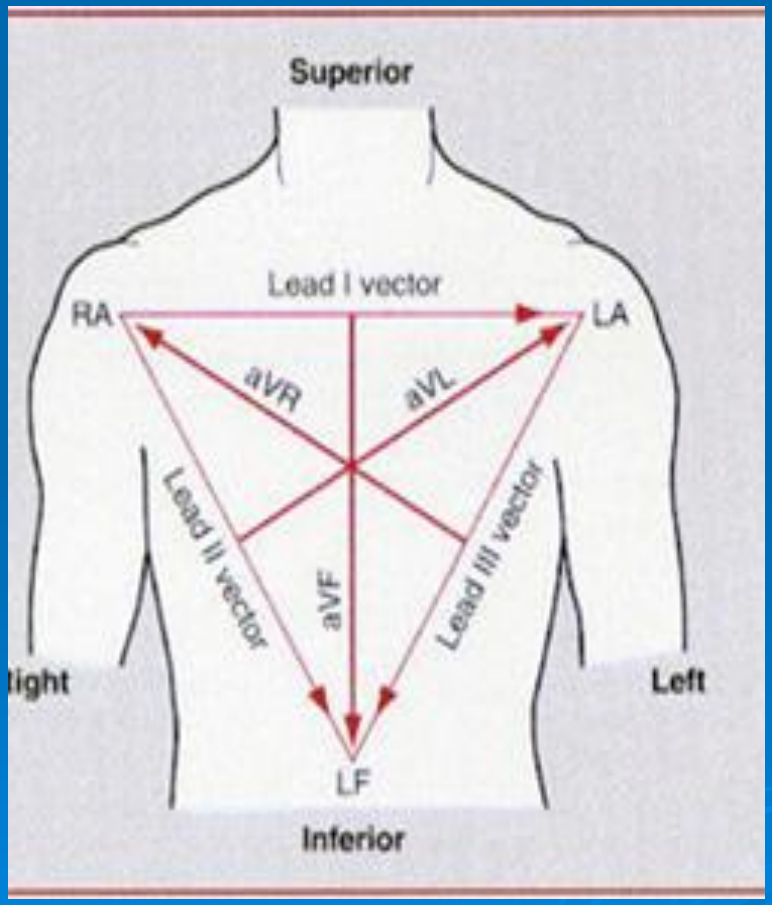
The augmented limb leads

- To obtain the augmented limb leads, the same electrodes are placed in the same as for limb leads position (R, L, F). These are unipolar leads, exploring the activity of the heart in the frontal plane.



The augmented limb leads

The axes of the unipolar limb leads are perpendicular to the axes of the limb leads, poining towards the exploring electrodes.



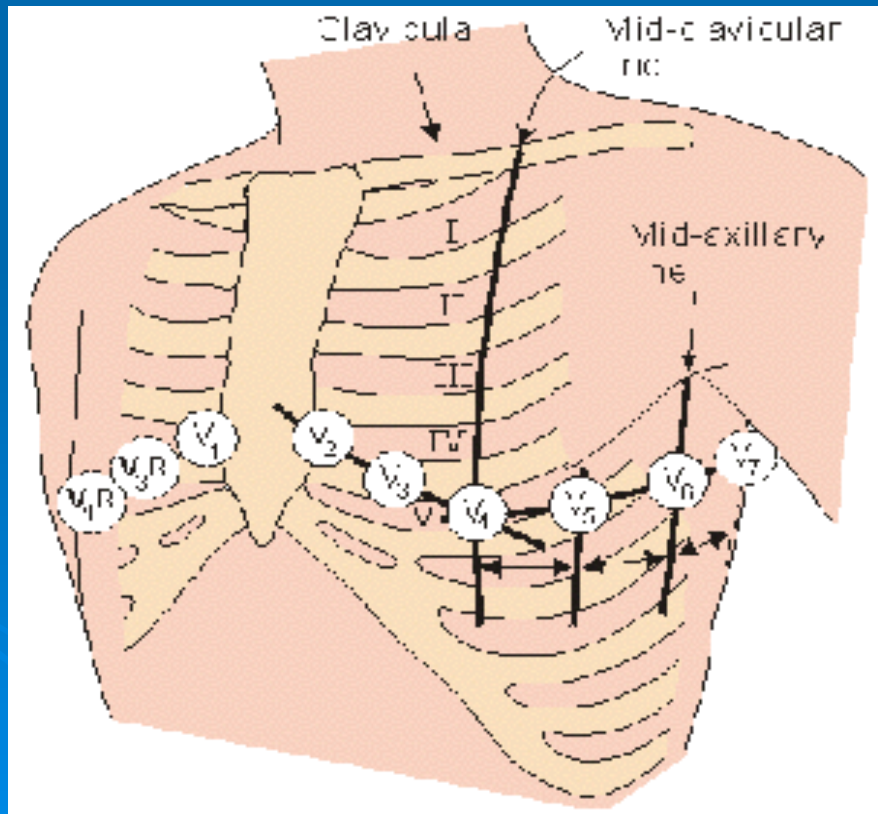
Applying Kirchhoff‘s second law to this electrical circuit the fundamental law of the augmented limb leas can be writtten:

VR+VL+VF=0

The chest (precordial) leads

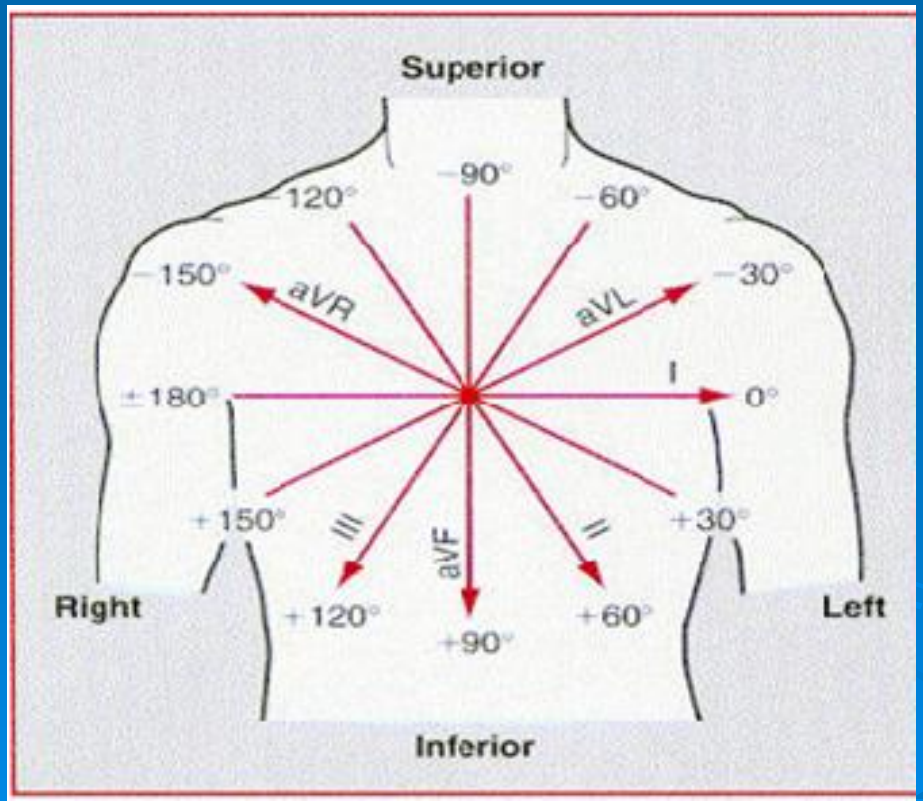
The precordial leads lie in the transverse (horizontal) plane,perpendicular to the other six leads. The exploring electrodes are placed in specific positions at the surface of the thorax, while the indiffernt electrode is obtained by Wilson´s method. The electrodes are placed at the surface of the chest:

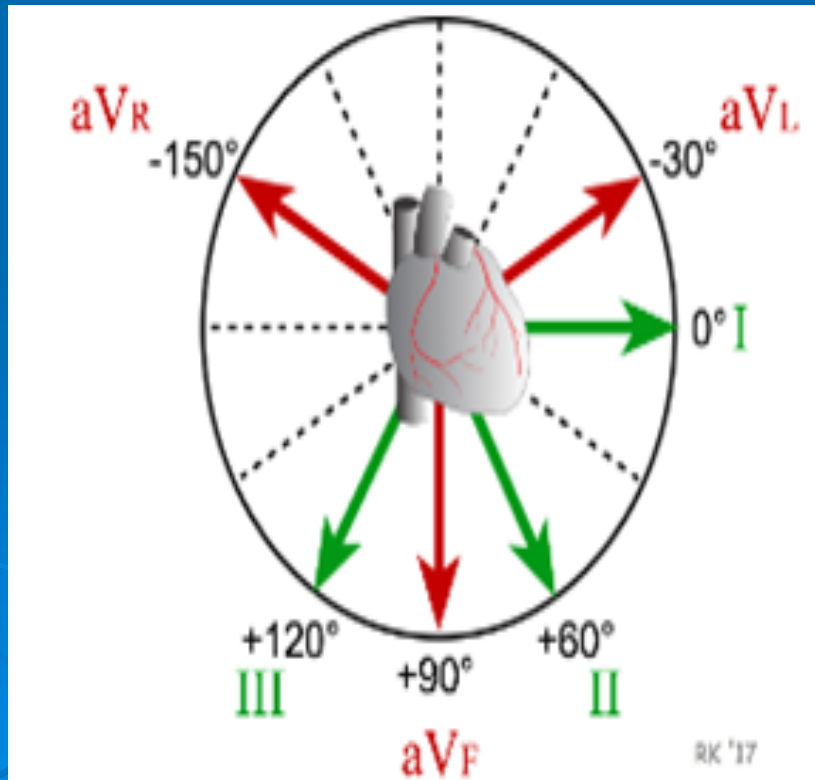
- **V_1- in the 4th intercostal space, right of the sternum;**
- **V_2- in the 4th intercostal space, to the left of the sternum;**
- **V_3- between V_2 and V_4;**
- **V_4- in the 5th intercostal space, on the midclavicular line**
- **V_5- in the 5th intercostal space, on the anterior axillary line,**
- **V_6- in the 5thintercostal space, on the midaxillary line**



The hexaaxial system

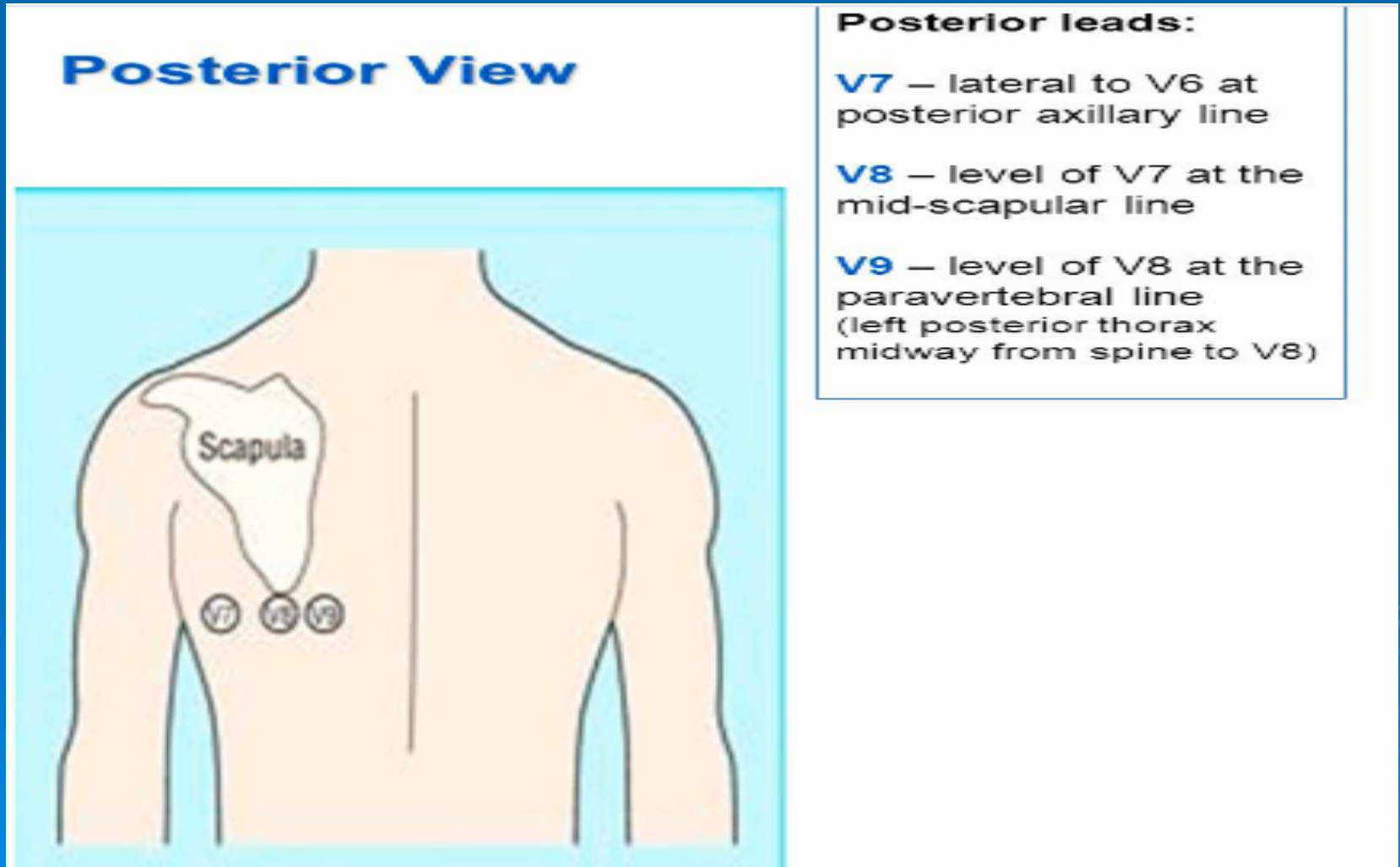
- Since leads I,II, III, aVR, aVL and aVF measure activity in the same plane they are always considered together and represented by a large circle with the negative electrodes for each of the leads aligned in the middle of the chest (hexaaxial system).



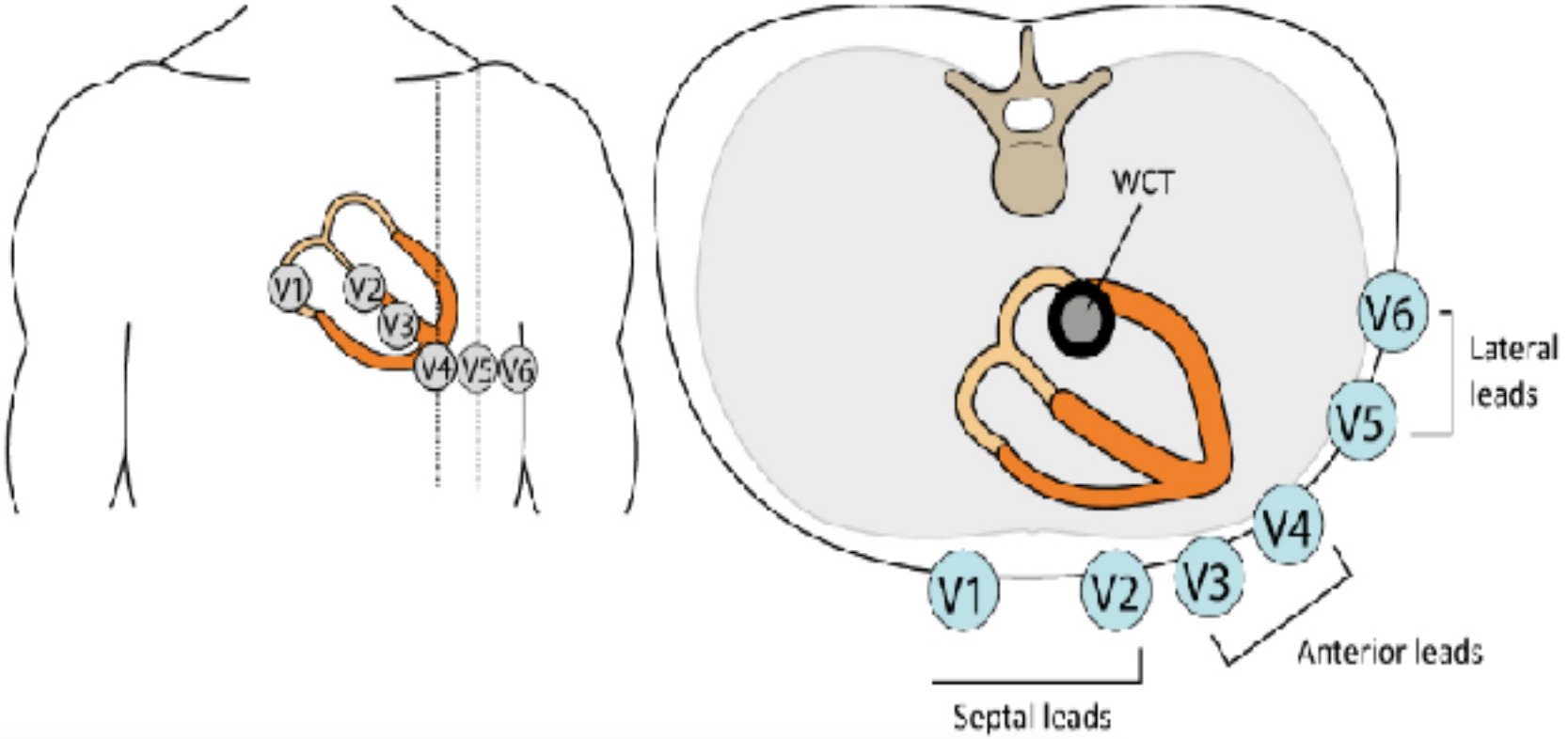


The chest (precordial) leads

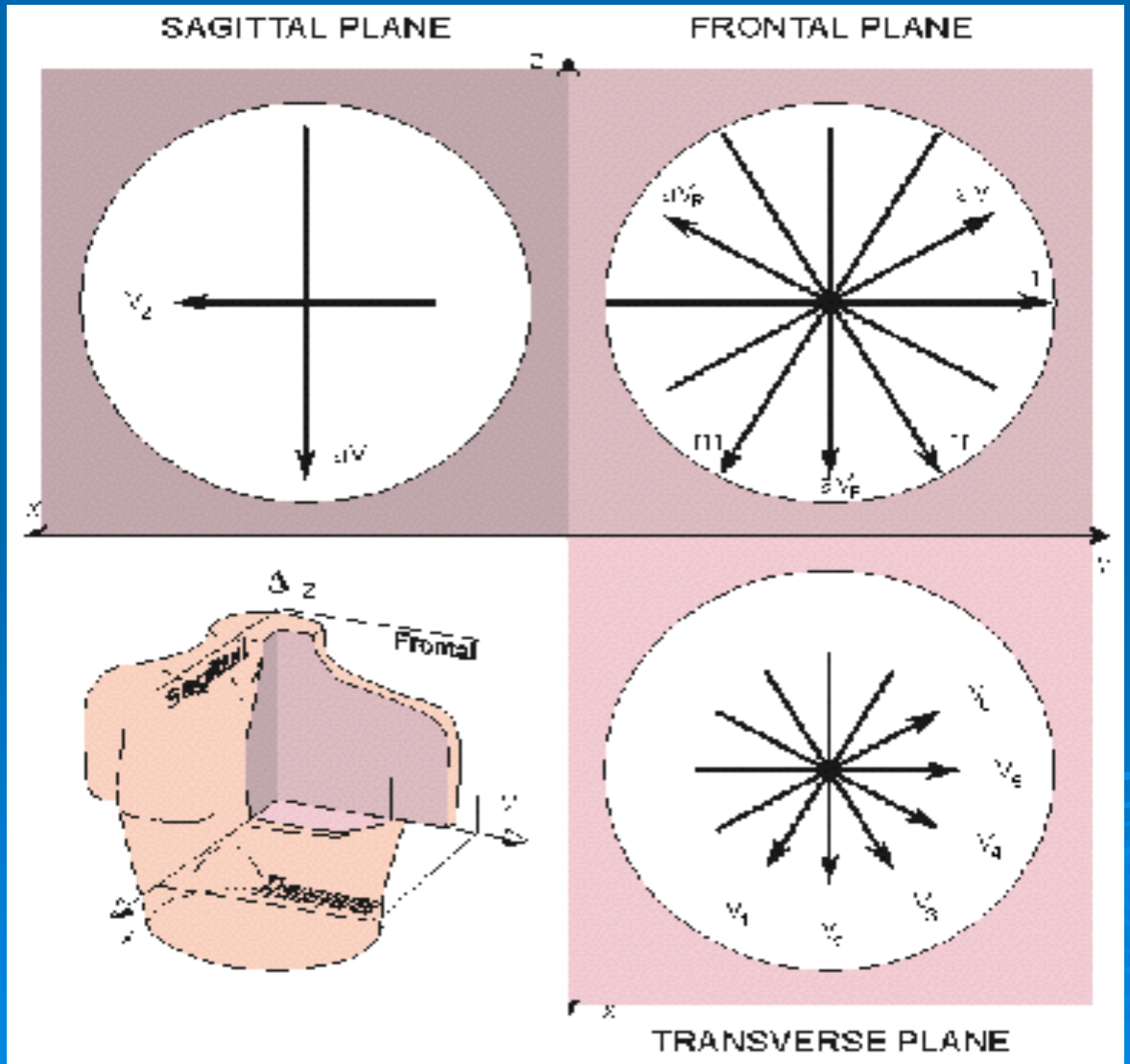
Additional electrodes may rarely be placed to generate other leads for specific diagnostic purposes. *Right-sided* precordial leads may be used to better study pathology of the right ventricle or for dextrocardia (V3R to V6R). *Posterior leads* (V7 to V9) may be used to demonstrate the presence of a posterior myocardial infarction.



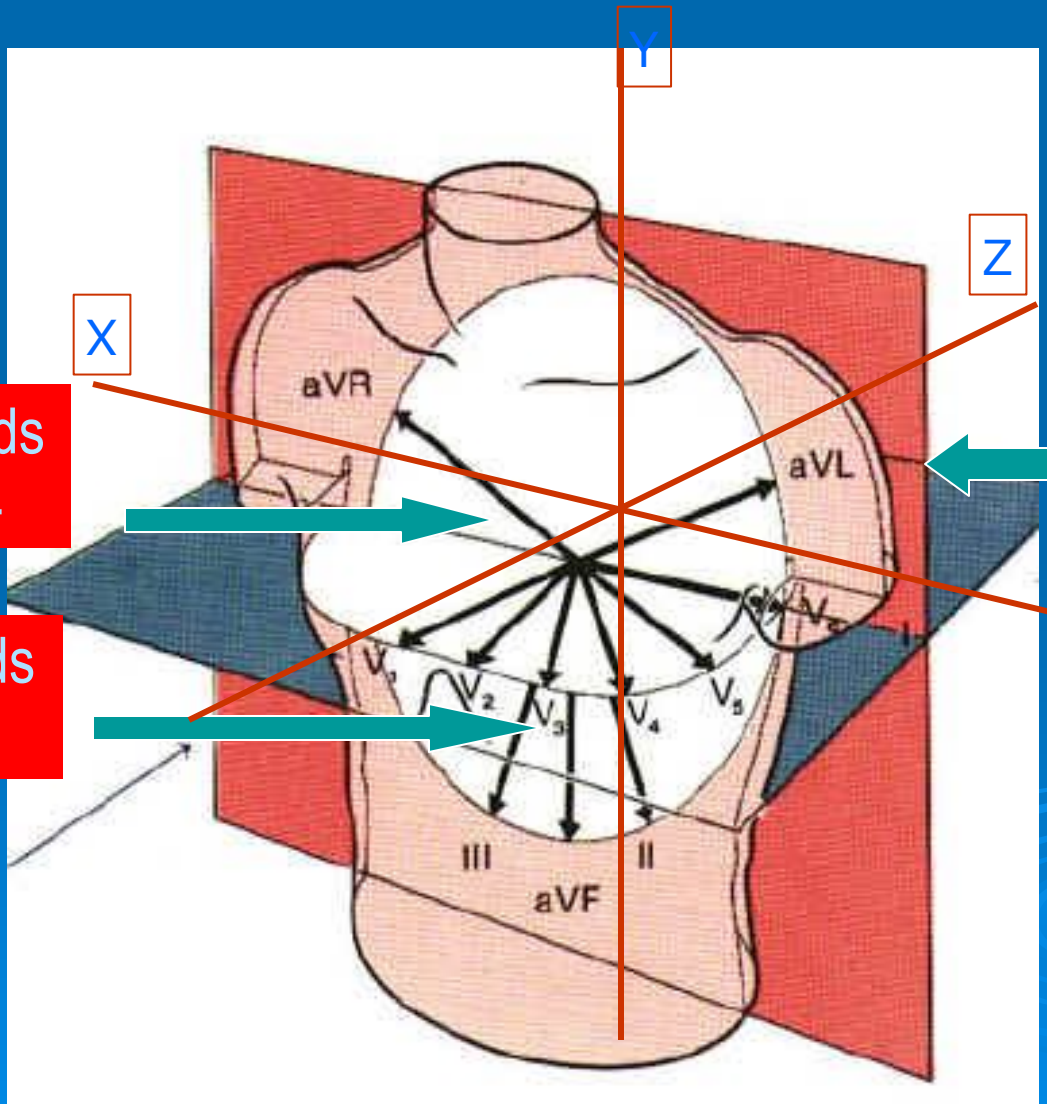
The chest (precordial) leads



Projections of the 12-lead EKG vectors in three orthogonal planes



Review of what each EKG lead looks at



Anterior Leads
V1,V2,V3,V4

Inferior Leads
II,III,AVF

Left lateral
Leads
I, AVL,V5
V6

Incorrect electrode placement

Limb lead reversal:

1. Reversal of right and left arm leads

- Resultant ECG mimics dextrocardia in limb leads with inversion of the P-QRS-T in leads I and aVL
- Leads II and III transposed
- Leads aVR and aVL transposed

2. Reversal of left arm and left leg leads

- Leads I and II transposed
- Leads aVF and aVL transposed
- Lead III inverted

3. Reversal of right arm and left leg leads

- Leads I, II, and III inverted

Arrangement of Leads on the EKG

Each twelve leads records has its own particular line of sight and region of the heart that it views best .

I	aVR	V_1	V_4
II	aVL	V_2	V_5
III	aVF	V_3	V_6

Anatomic Groups (Septum)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups (Lateral Wall)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

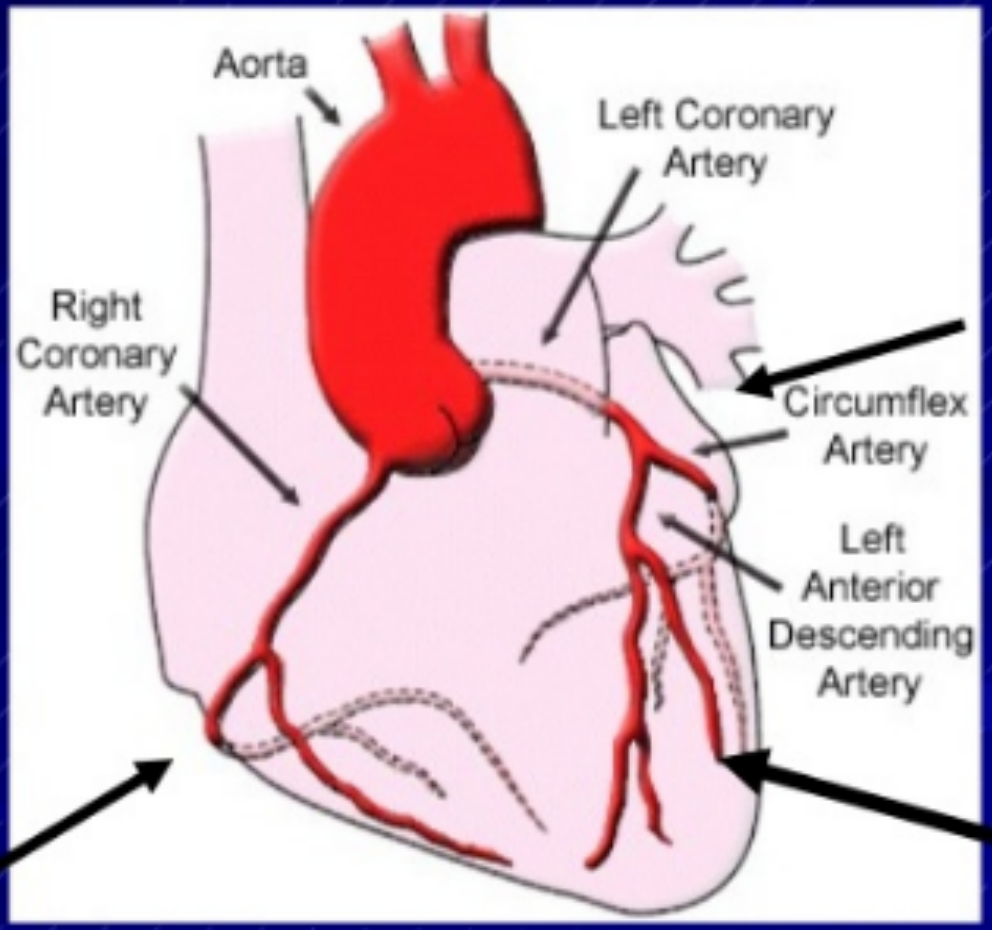
Anatomic Groups (Inferior Wall)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Summary

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Localising the arterial territory



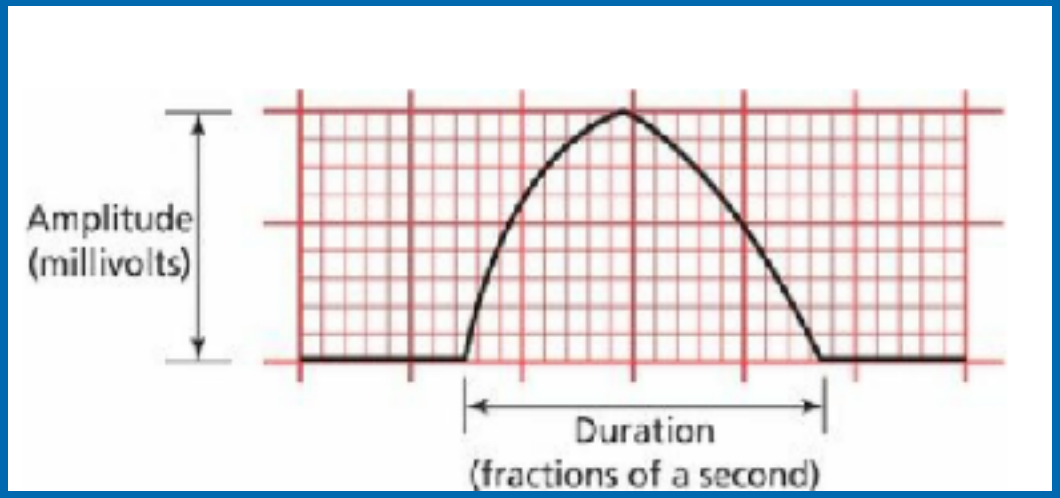
Lateral
I, AVL, V5-V6

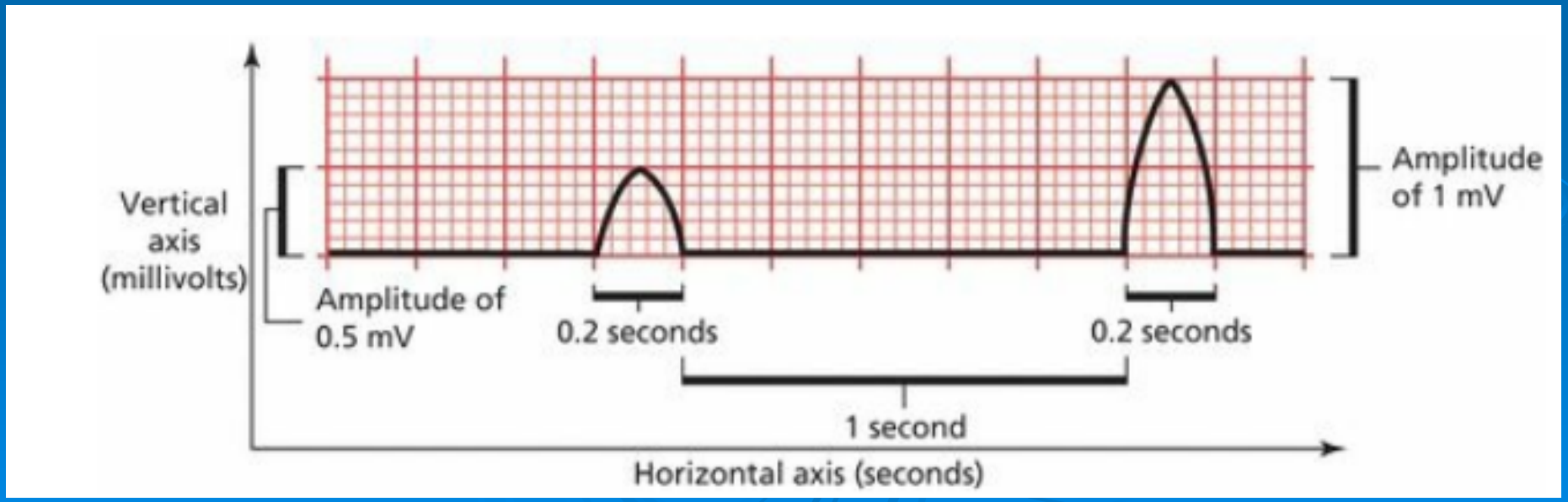
Anterior / Septal
V1-V4

Inferior
II, III, aVF

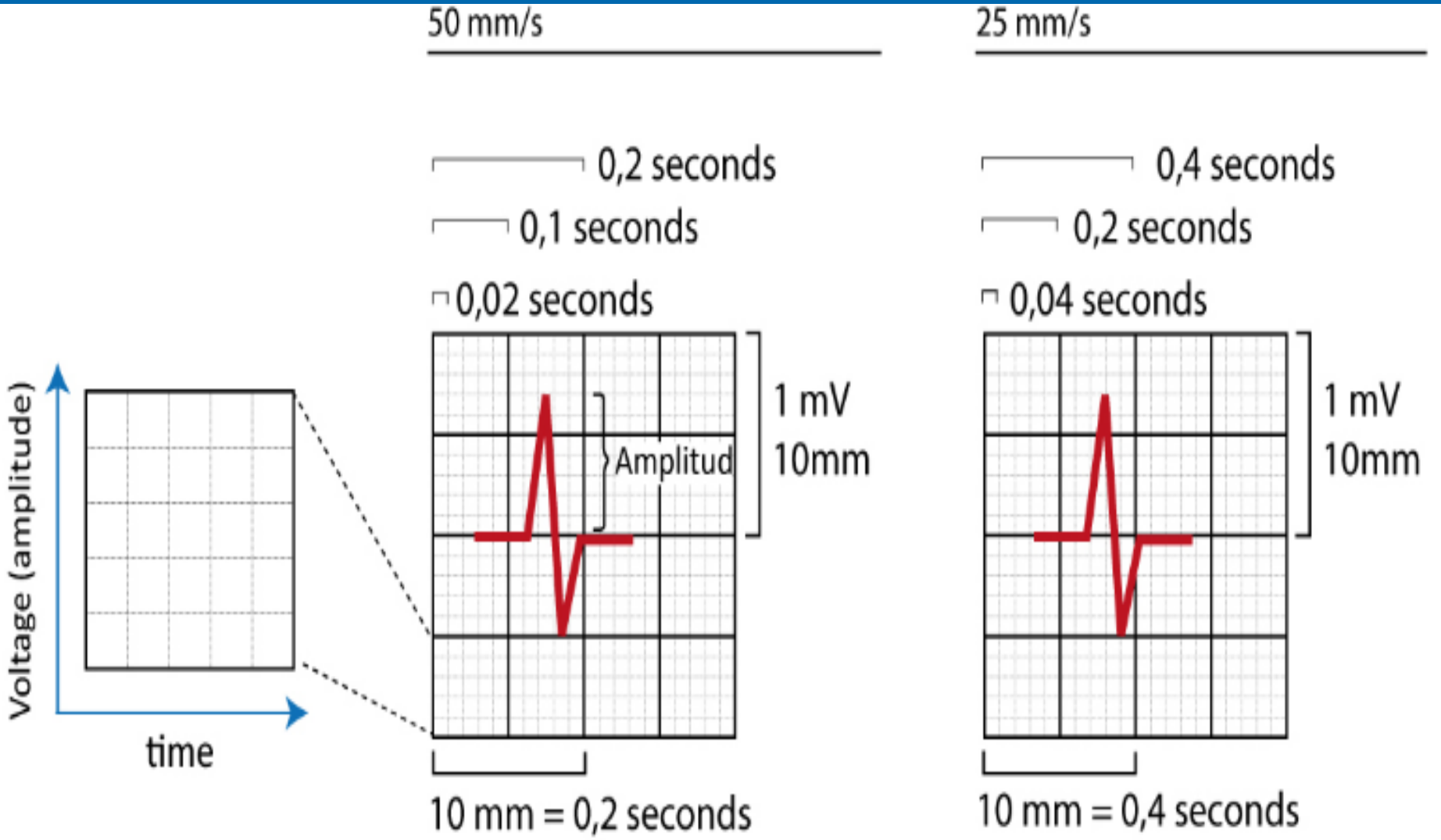
From electrode to paper

- The waves that appear on the ECG reflect the electrical activity of the myocardial cells.





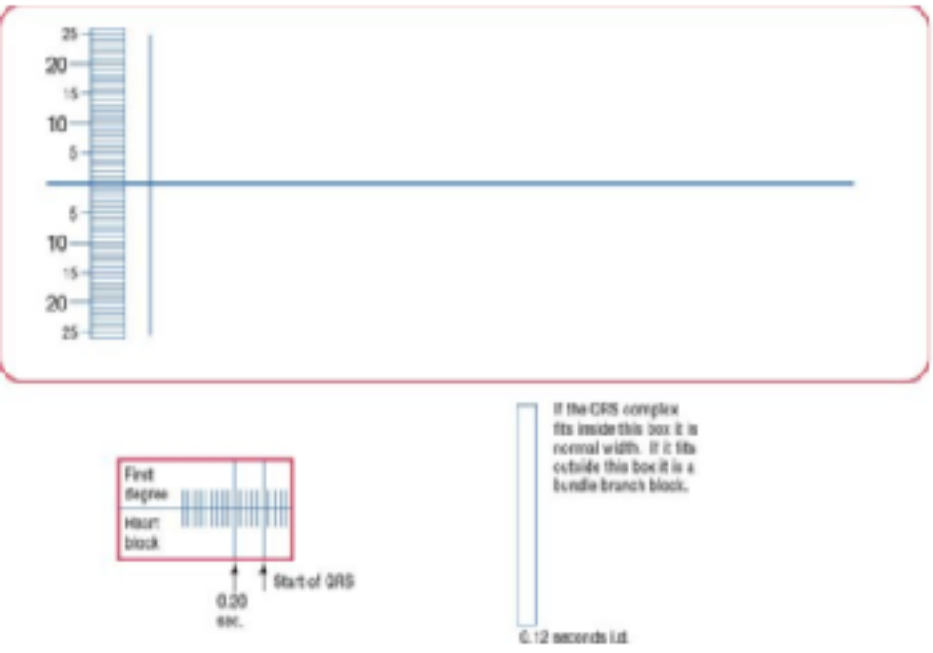
EKG paper speed

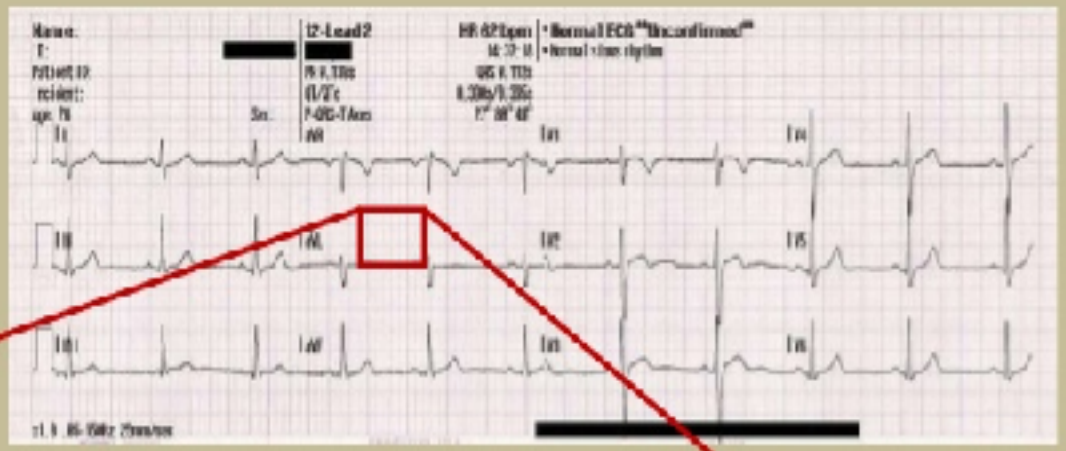


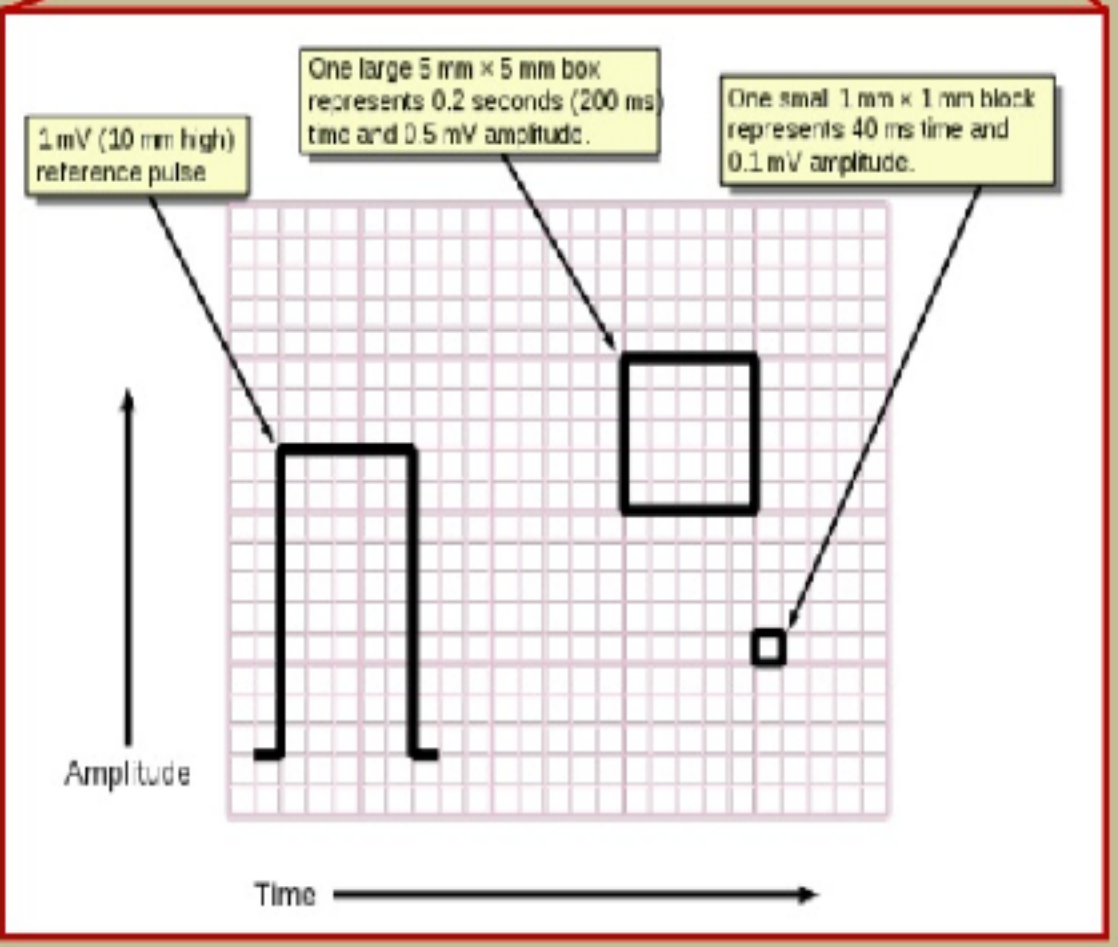
ECG Tools

There are various tools that make reading and interpreting the ECG much easier.

1. Calipers
2. Axis-wheel ruler
3. ECG ruler
4. Straight edge







STANDARD CALLIBRATION

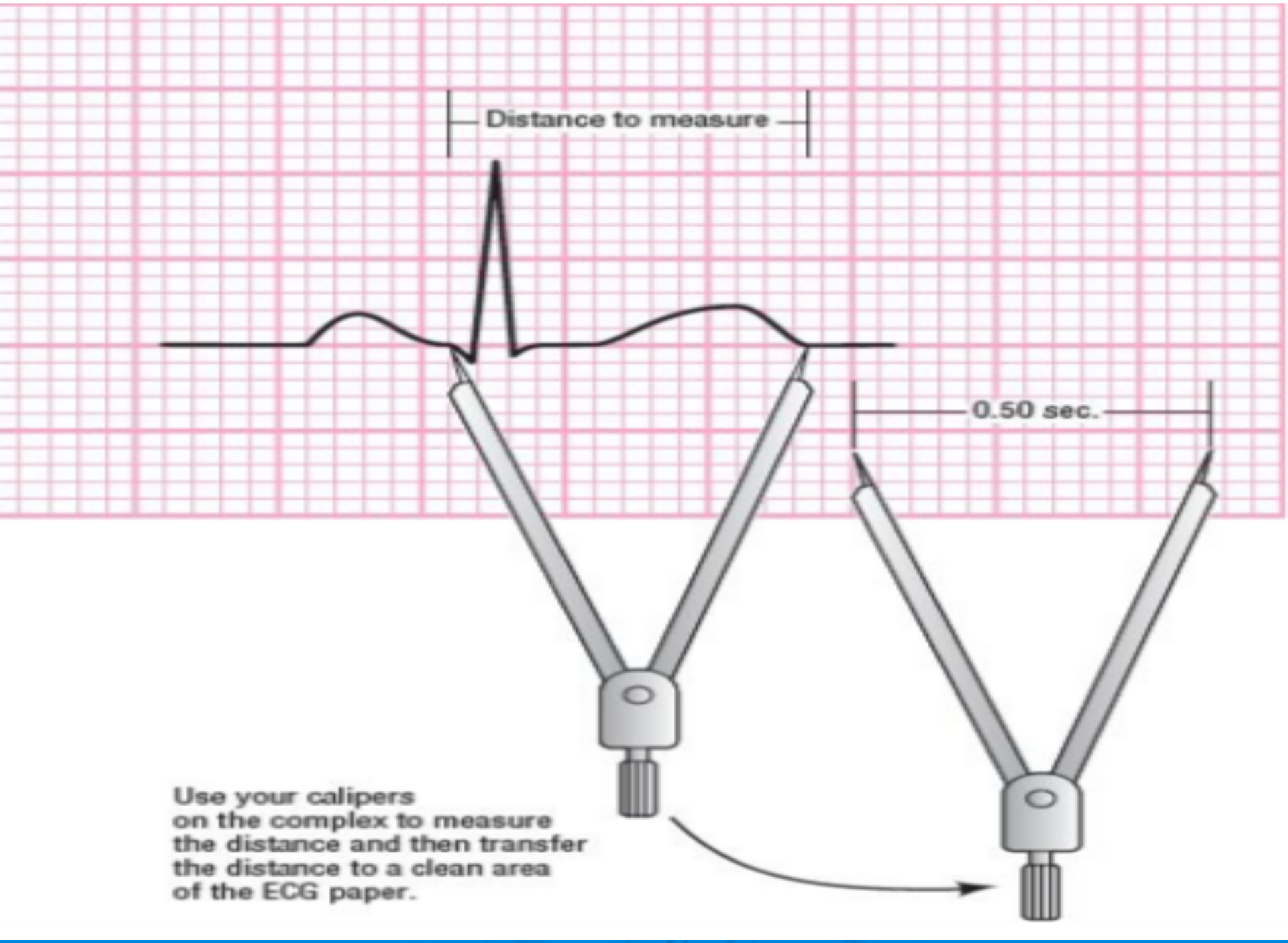
Speed = **25mm/s**

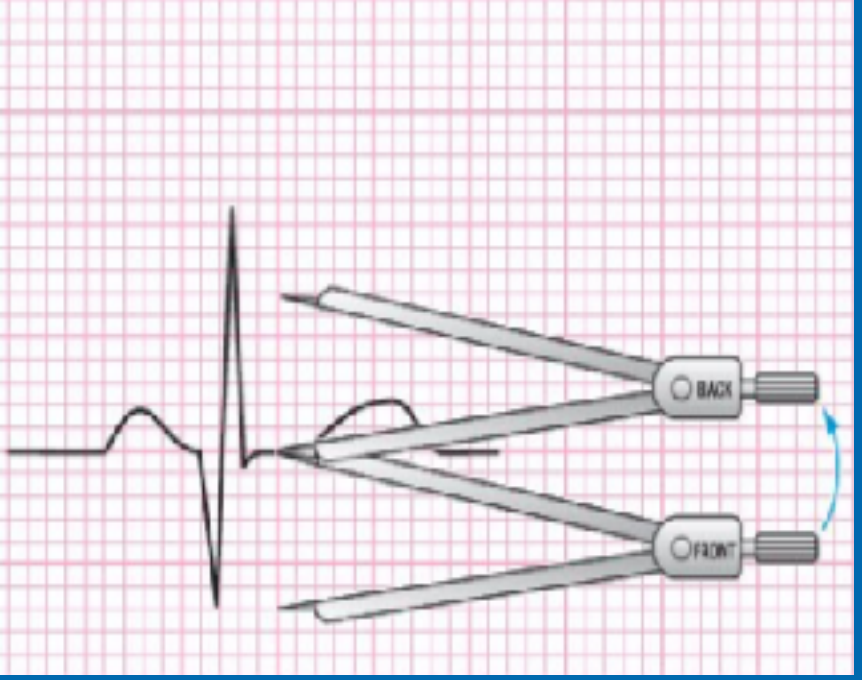
Amplitude = **0.1mV/mm**

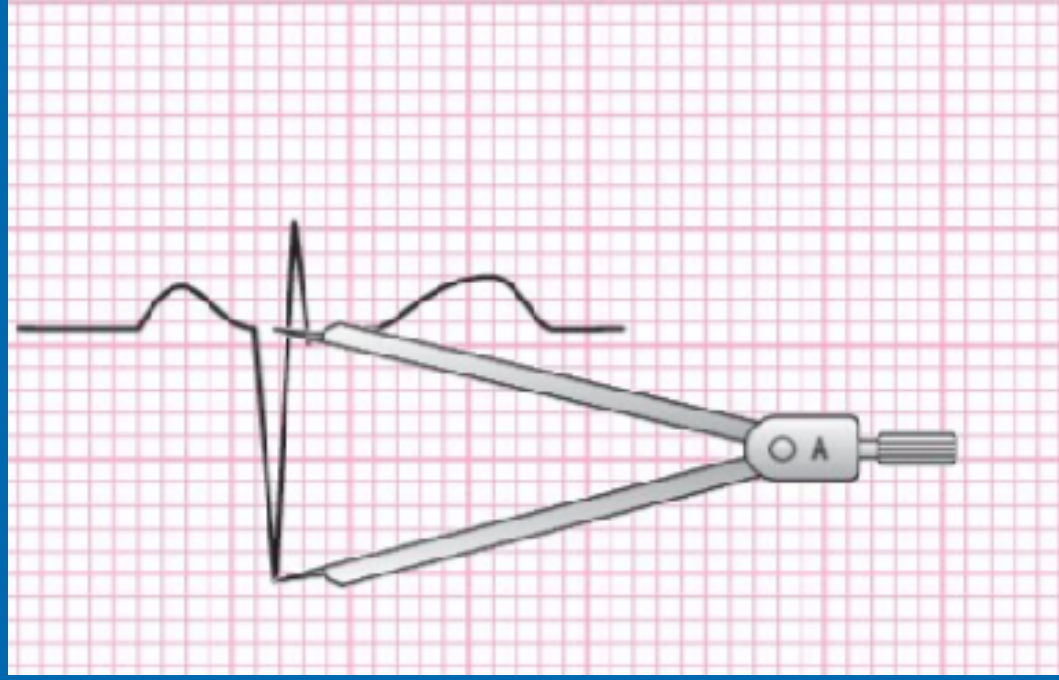
1mV → 10mm high

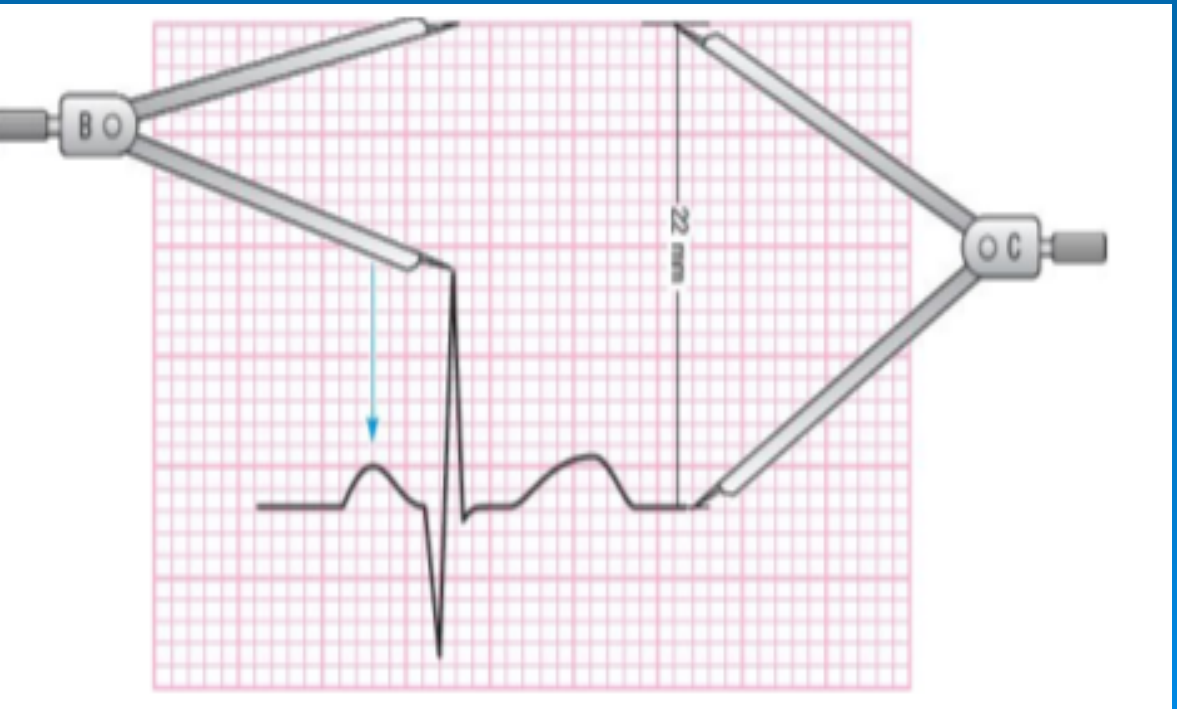
1 large square → 0.2s(200ms)

1 small square → 0.04s (40ms) or 1 mV amplitude


Distance to measure
0.50 sec.
Use your calipers
on the complex to measure
the distance and then transfer
the distance to a clean area
of the ECG paper.


BACK
FRONT


A


B
C
22 mm

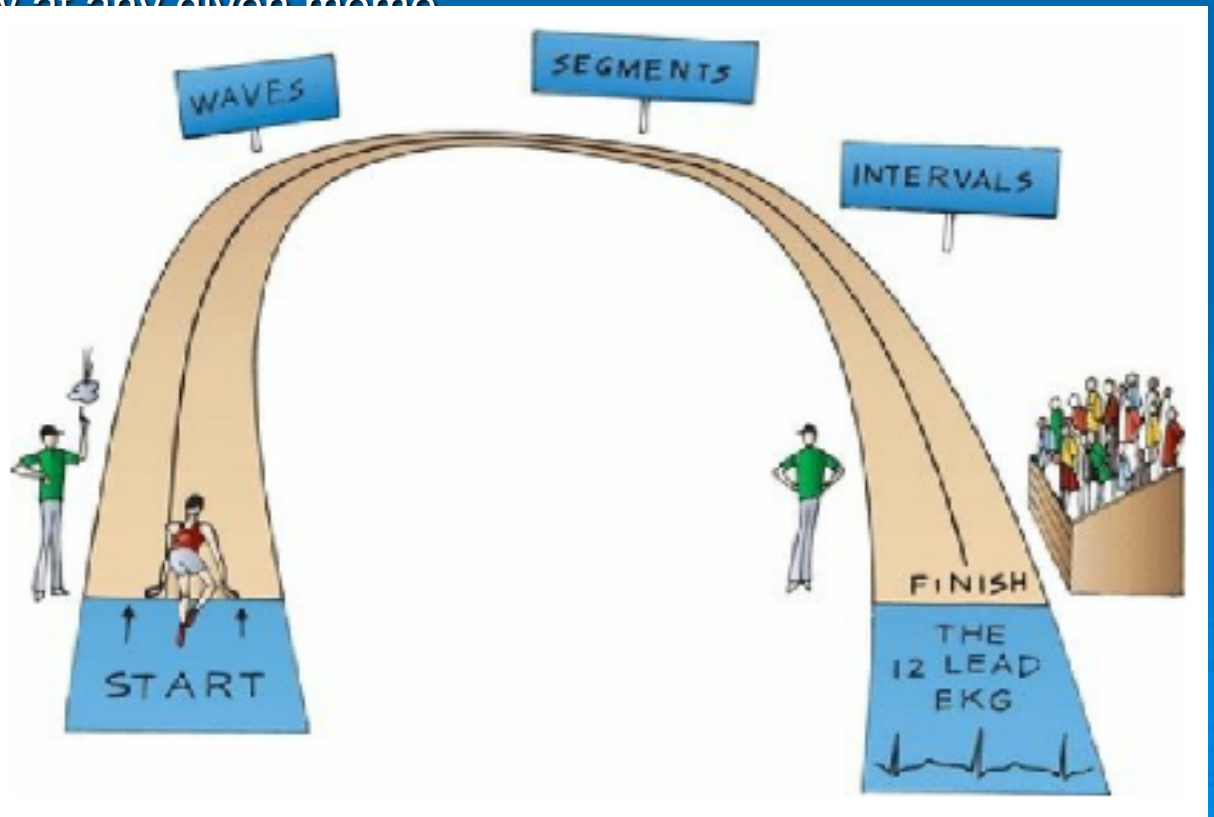
The Normal 12-Lead EKG

The three things necessary to derive the normal 12-lead EKG:

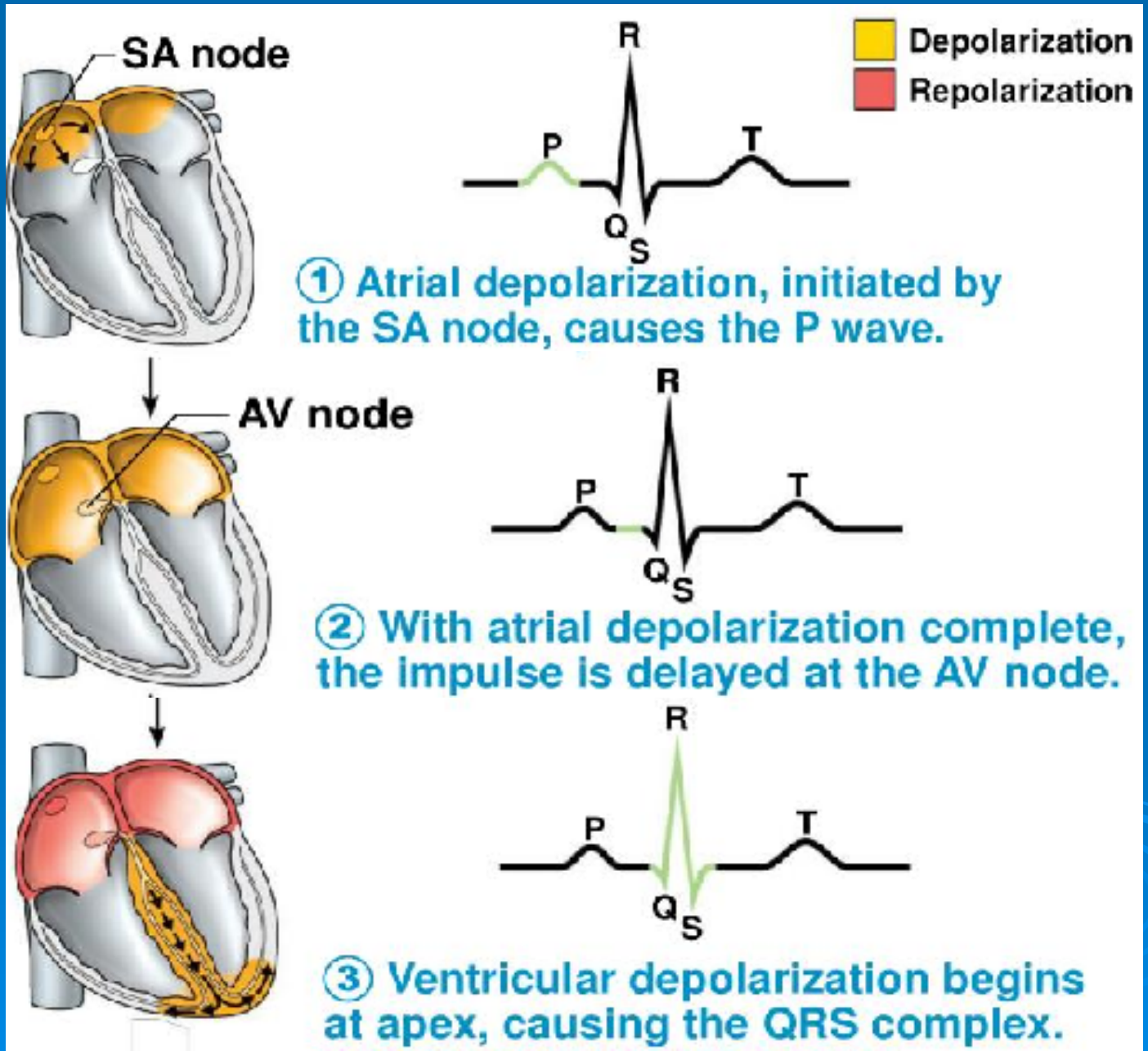
1. The normal pathway of cardiac electrical activation and the names of the segments, waves,and intervals that are generated

2. The orientation of all 12 leads, six in the frontal plane and six in the horizontal plane

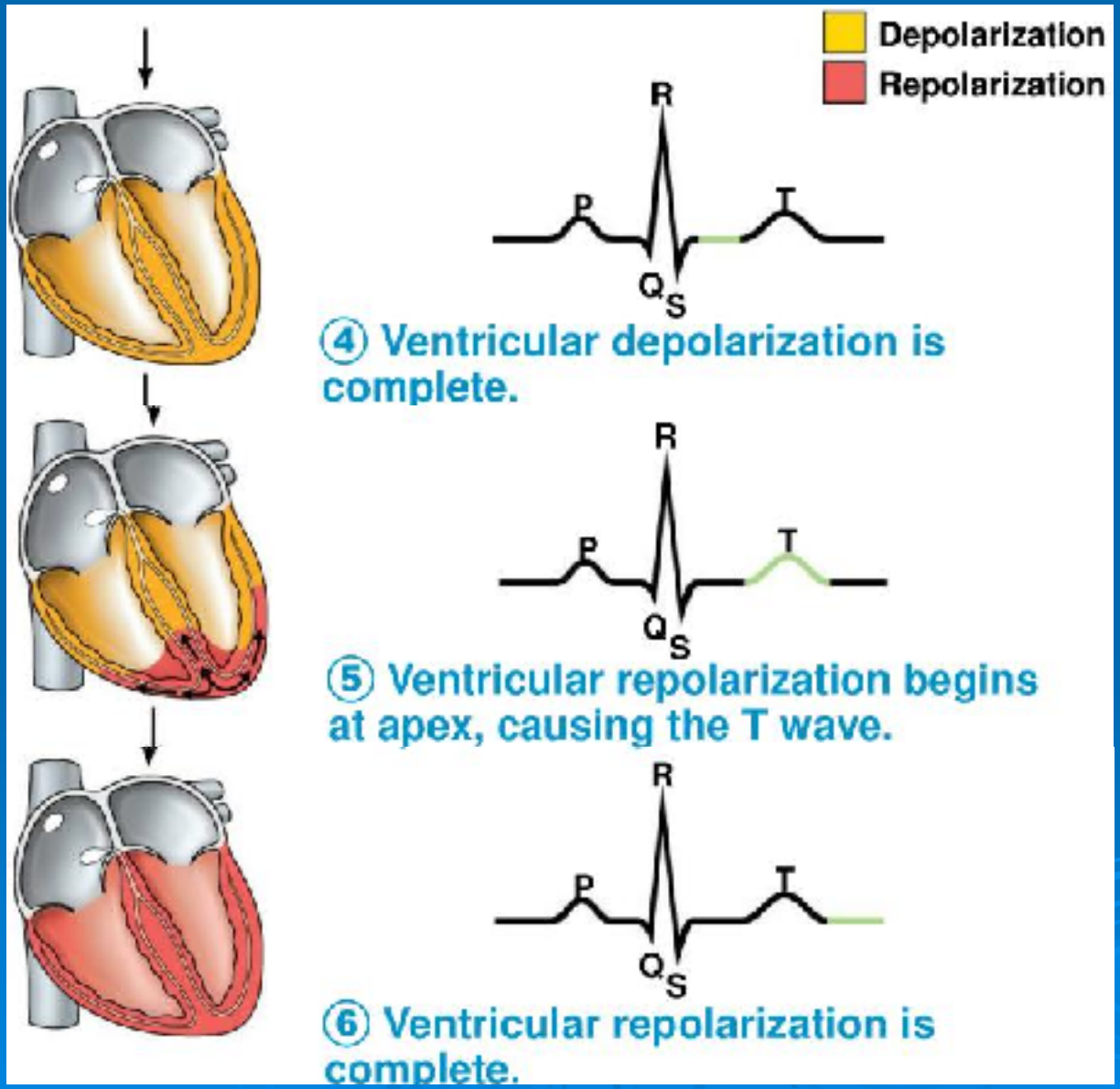
3. The simple concept that each lead records the average current. flow at any given mome



Electrical activity of myocardium

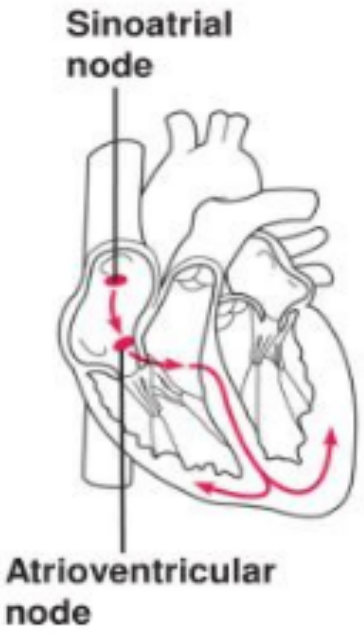


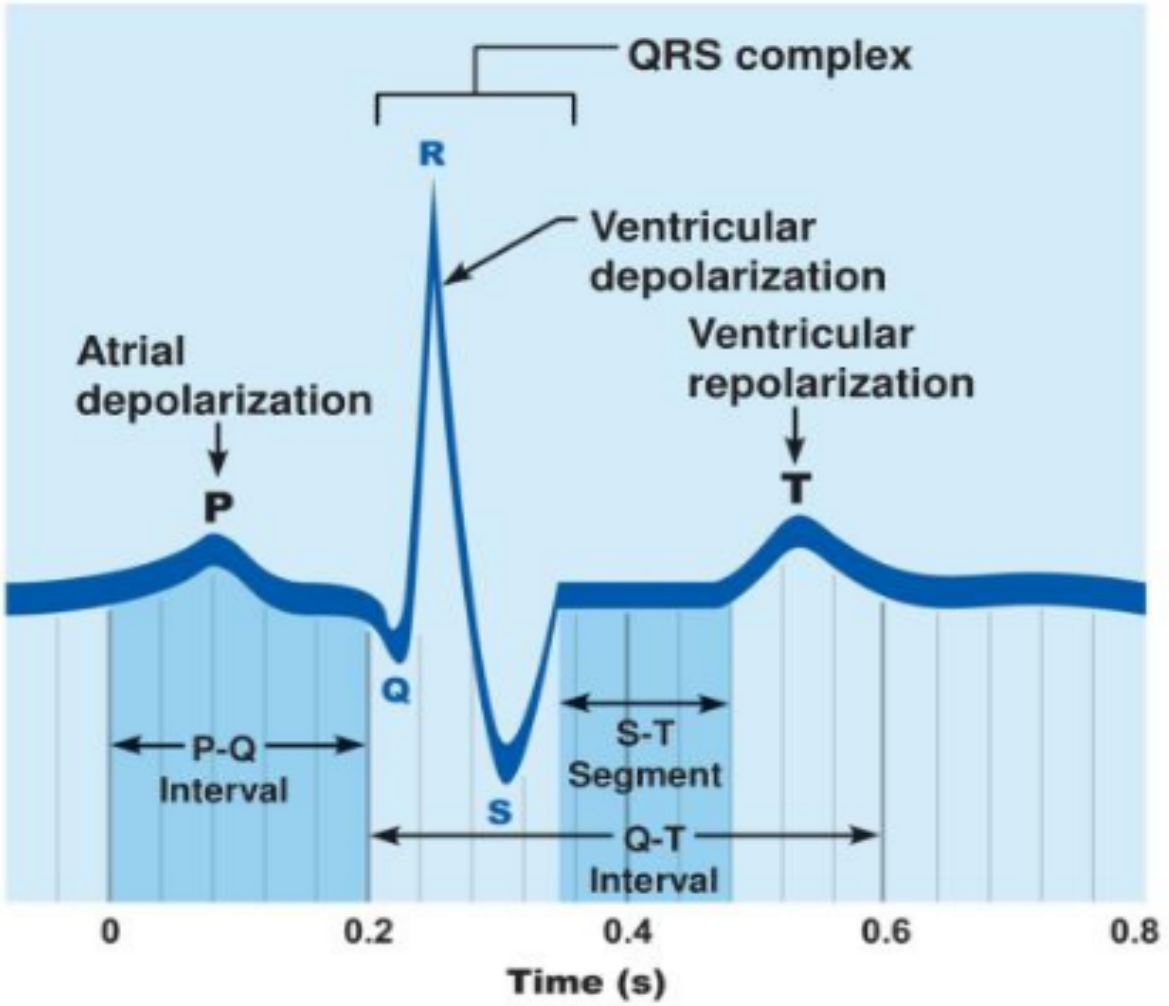
Electrical activity of myocardium



A NORMAL ECG WAVE

REMEMBER



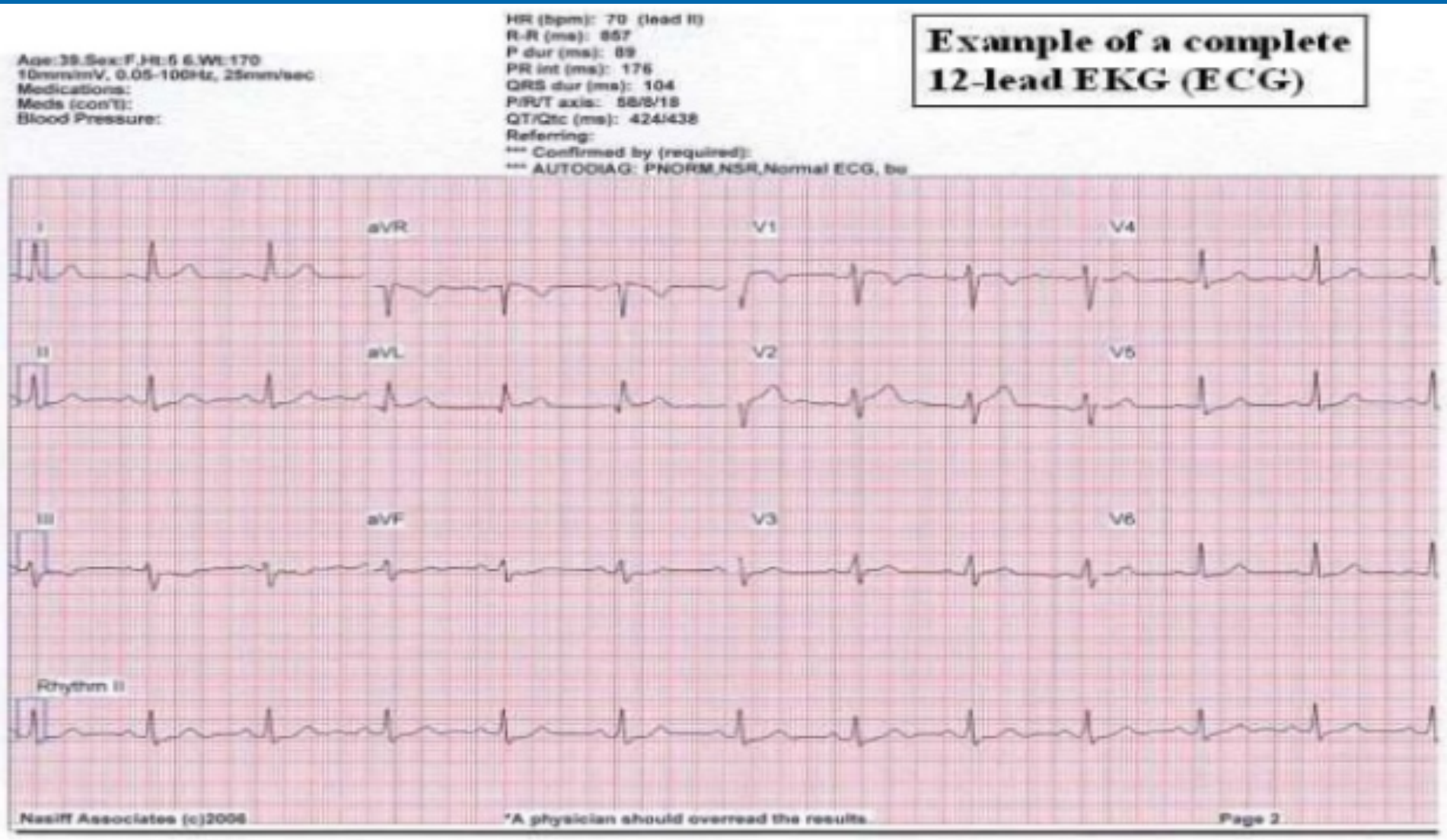


ECG

INTERPRETATION

The More You See, The More You Know

Obtain an ECG, act confident, read the patient details



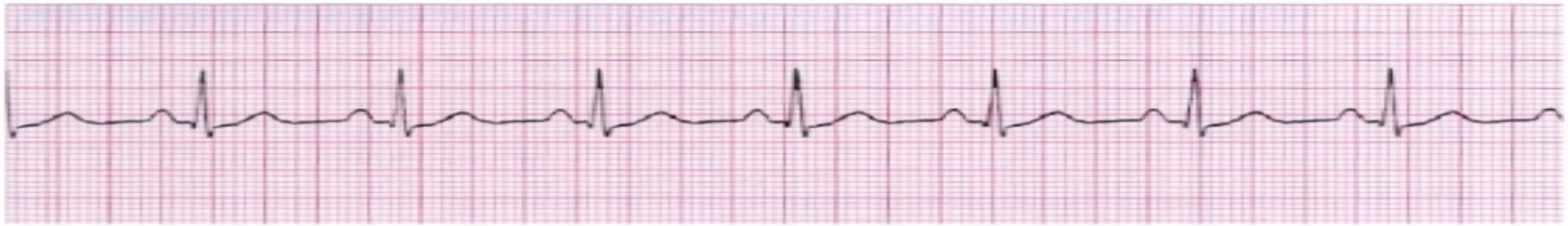
Rhythm

- The P waves – can you find them?
- What is the relationship between the P waves and the QRS complexes?

  P wave before every QRS complex= Sinus rhythm
- Is the rhythm regular or irregular?

Rhythm

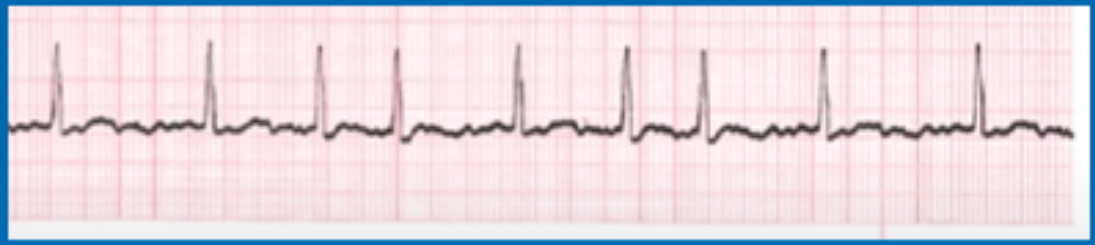
Normal Sinus Rhythm



ECG rhythm characterized by a usual rate of anywhere between 60-99 bpm, every P wave must be followed by a QRS and every QRS is preceded by P wave. Normal duration of PR interval is 3-5 small squares. The P wave is upright in leads I and II

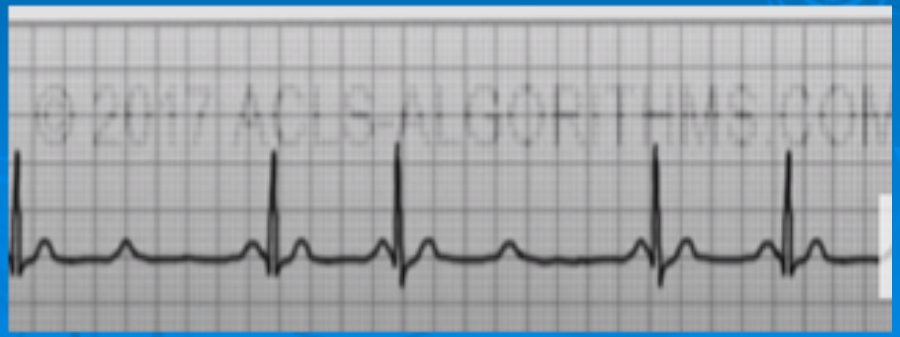
Irregularly Irregular

(atrial fibrilation)



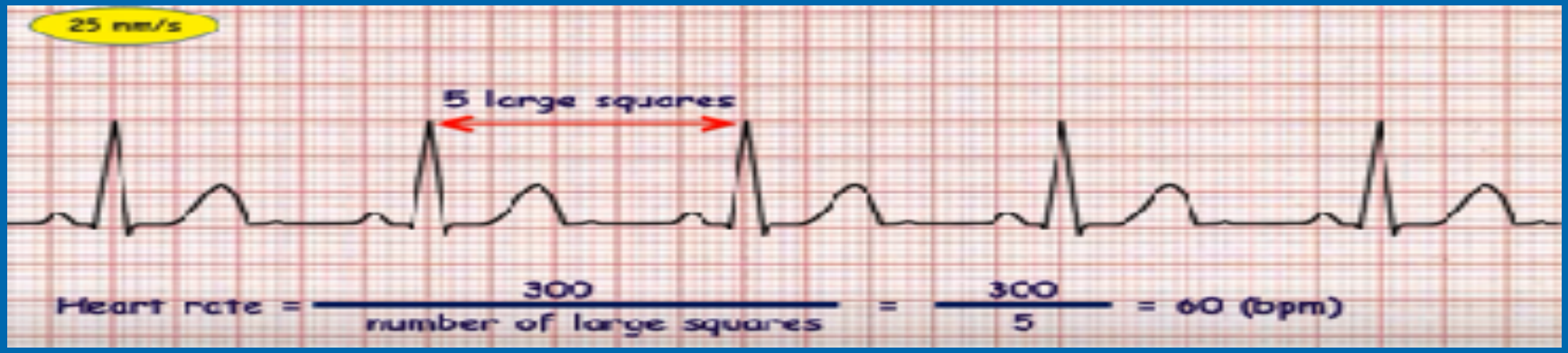
Regularly Irregular

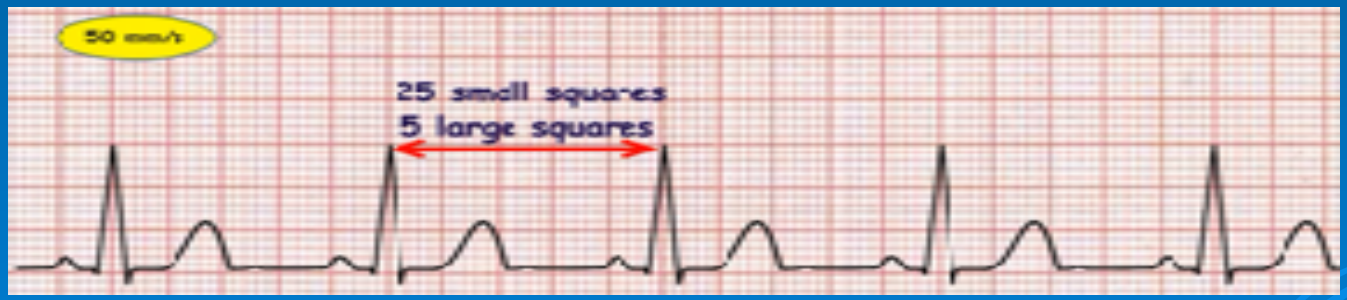
(Second degree heart block type 2)

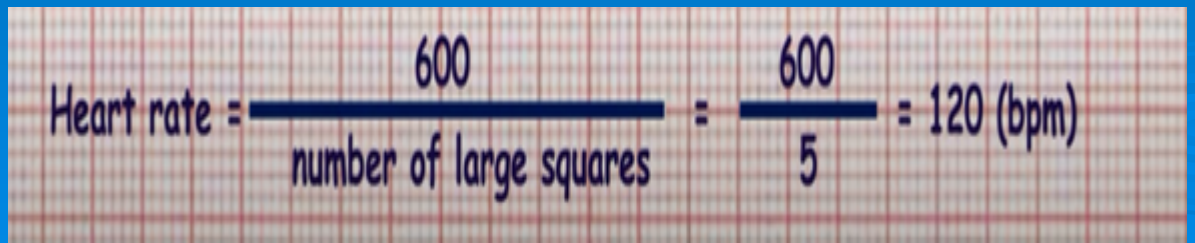


Calculation rate

1. *Large box counting method*

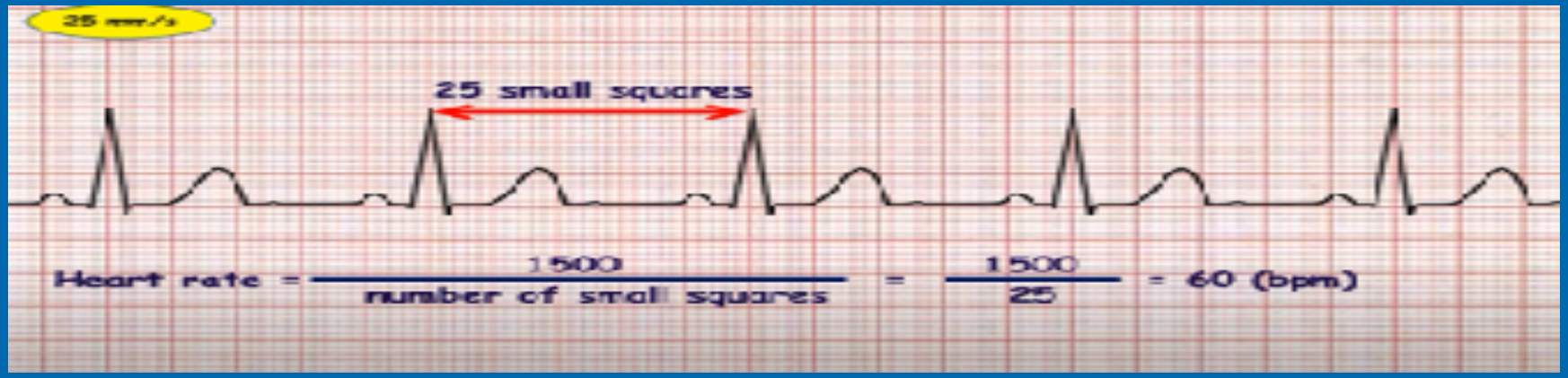


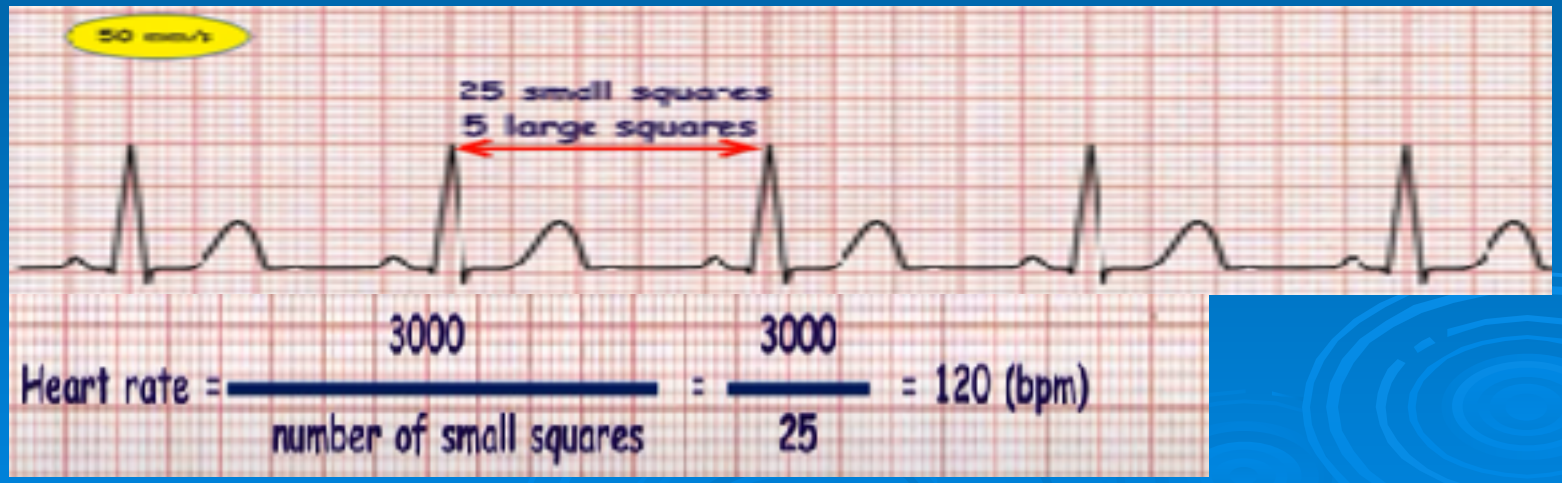




Calculation rate

2. Small box counting

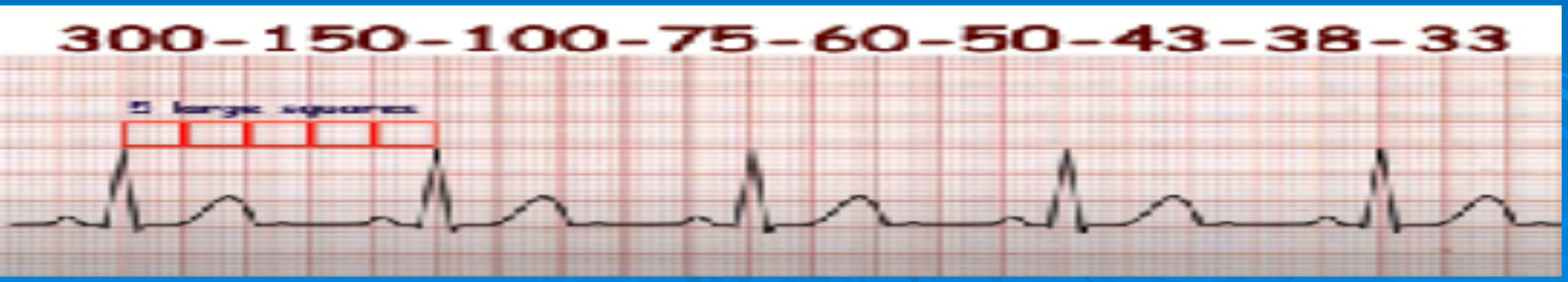




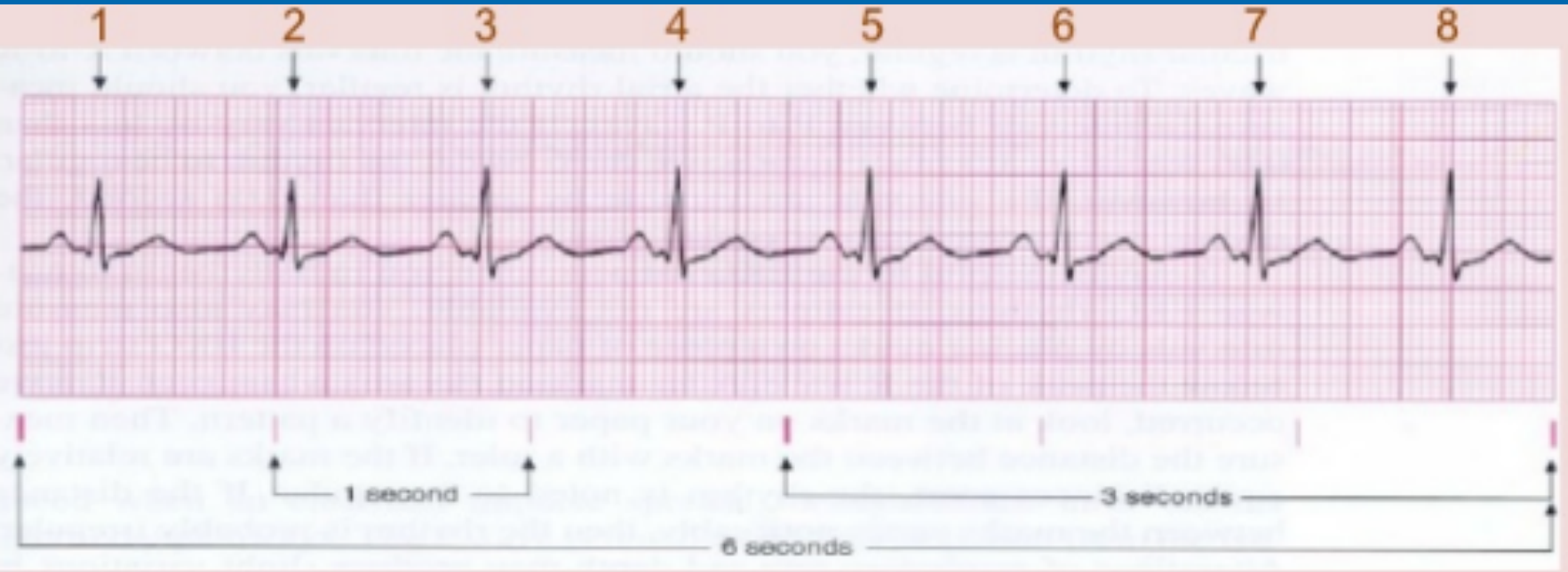
The rule of 300

- It may be easiest to memorize the following table

# of big boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50



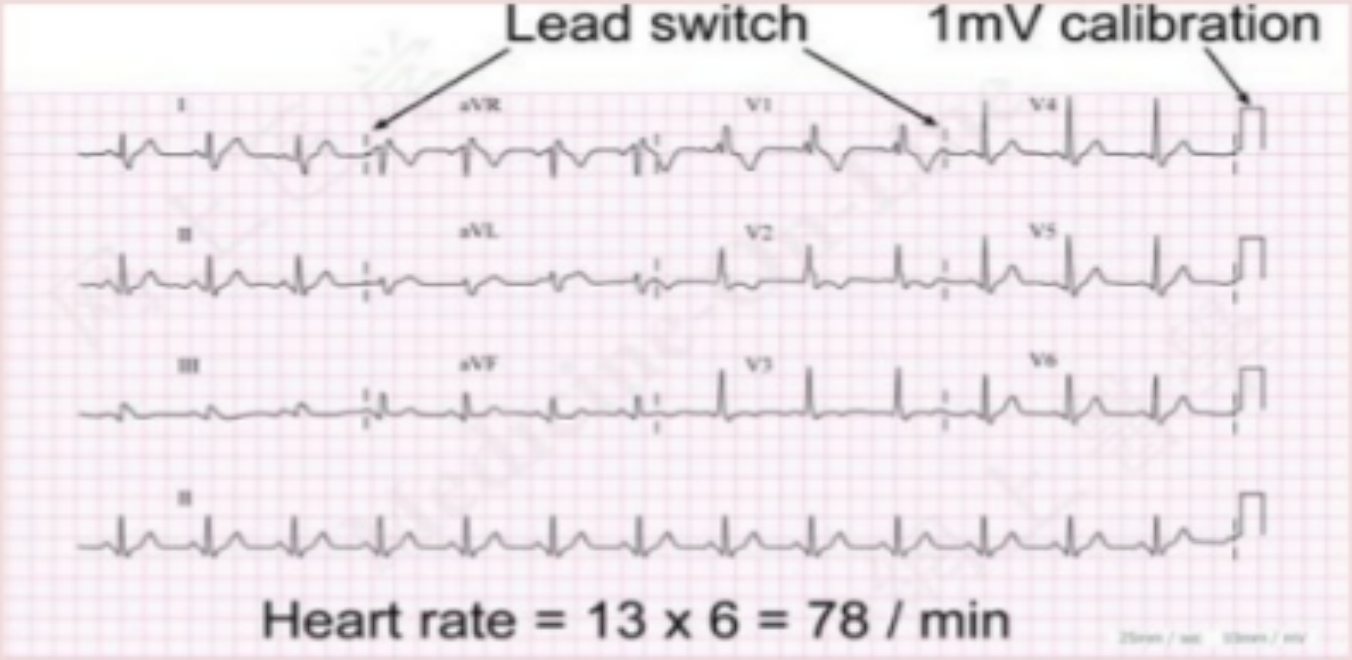
IRREGULAR rhythm



There are 8 waves in this 6-seconds strip.

Rate = (Number of waves in 6-second strips) x 10
= 8 x 10
= **80** bpm

IRREGULAR rhythm

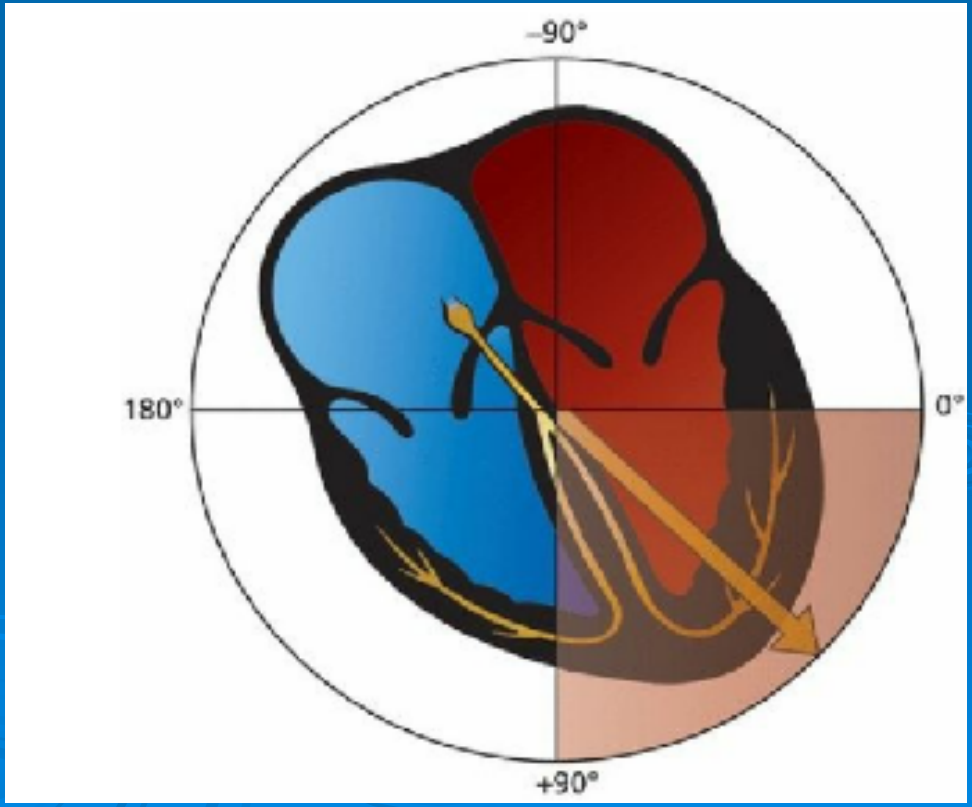


Rate = (Number of waves in 10-second strips) x 6
= 13 x 6
= **78** bpm

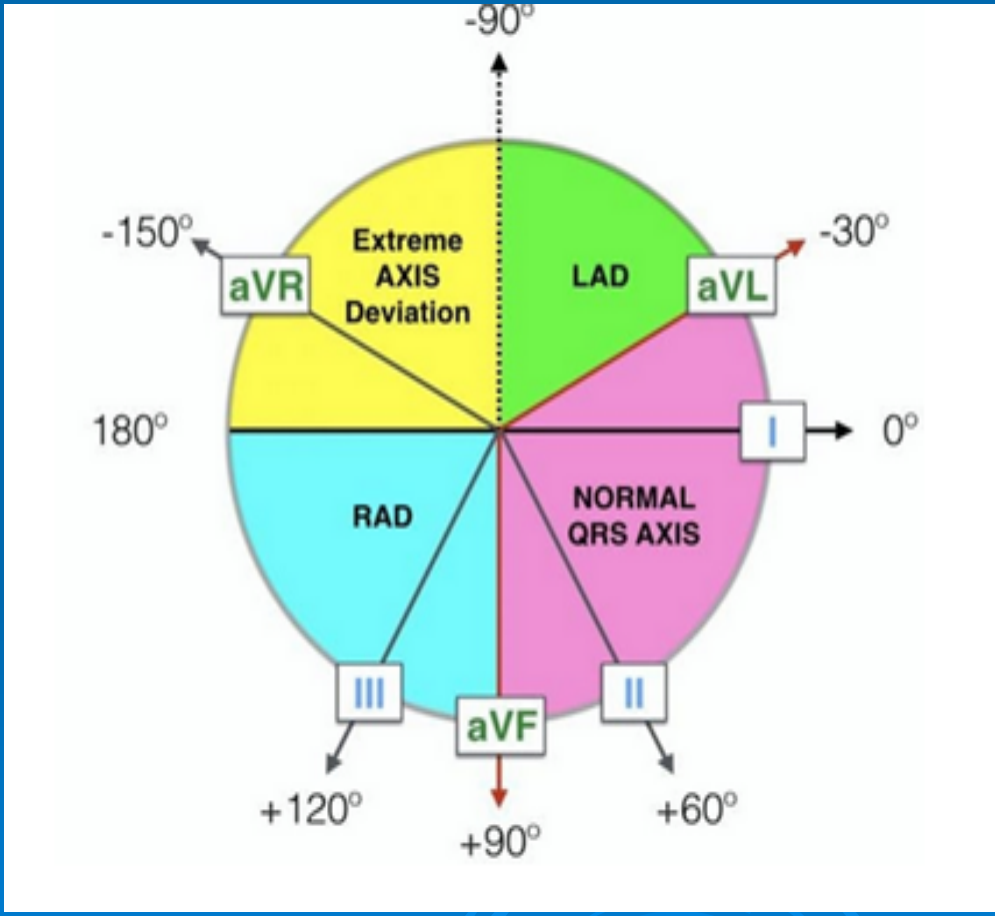
Determining axis

- The term axis refers to the direction of the mean electrical vector, representing the average diretion of current flow. It is defined in the frontal plane only.
- The mean QRS vector points leftward and inferiorly, representing the average direction of current flow during the entirely of ventricular depolarization.

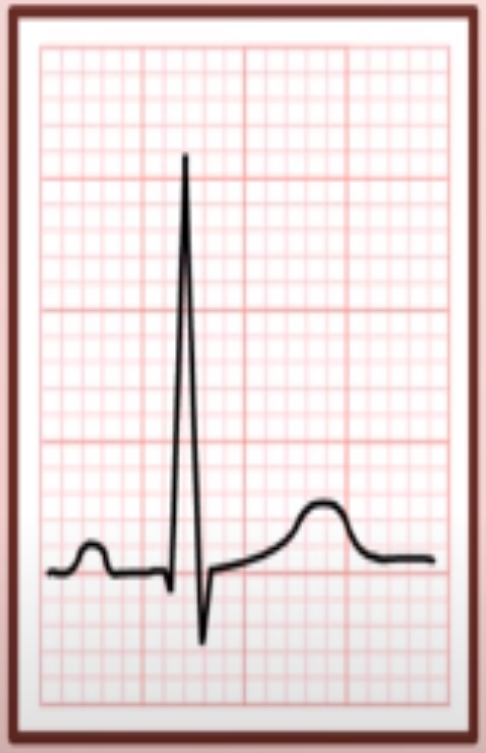
The normal QRS axis- direction of this mean vector- lies between +90° and -30°.



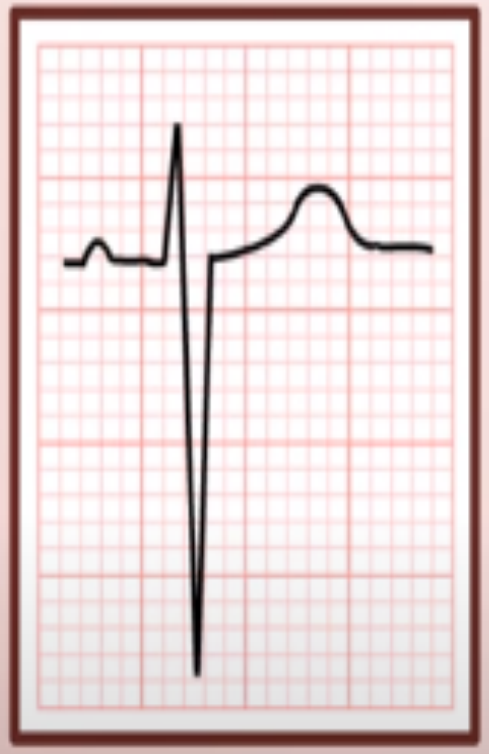
QRS axis


-90°
-150°
-30°
Extreme
AXIS
Deviation
LAD
aVR
aVL
180°
I
0°
RAD
NORMAL
QRS AXIS
III
II
aVF
+120°
+90°
+60°

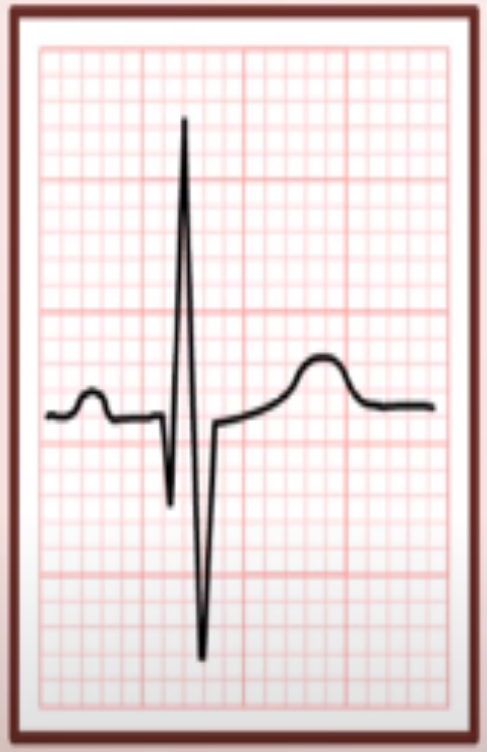
Determining axis- classifying QRS complexes



Predominantly Positive



Predominantly Negative



Equiphasic

Determining Axis- Quadrant Approach

Examine the QRS complex in leads I and aVF.

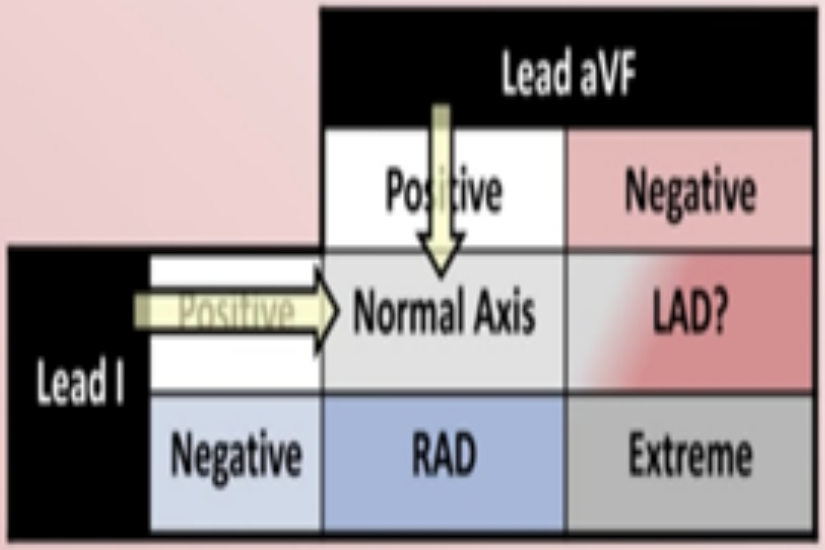
		Lead aVF	
		Positive	Negative
Lead I	Positive	Normal Axis	LAD?
	Negative	RAD	Extreme

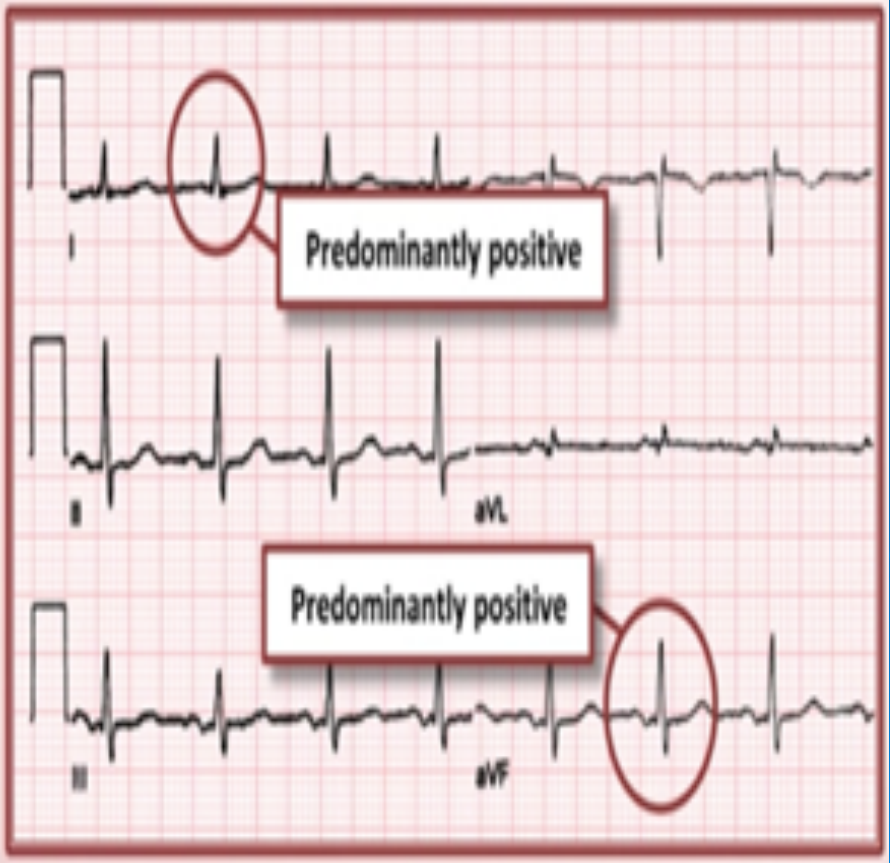
If QRS in I is + and QRS in aVF is -, examine QRS complex in lead II:

Predominantly positive → Normal (-30° to 0°)
Predominantly negative → LAD (-90° to -30°)

Determining Axis- Quadrant Approach

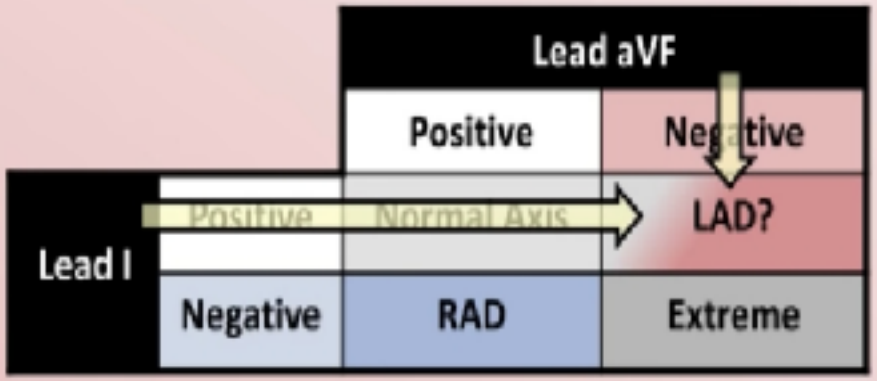
Examine the QRS complex in leads I and aVF.

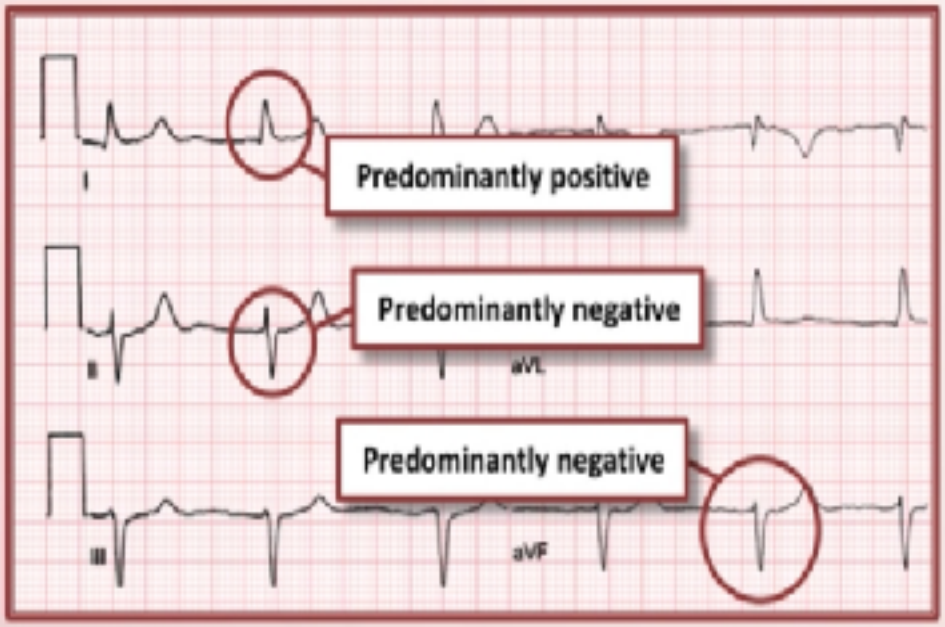




Determining Axis- Quadrant Approach

Examine the QRS complex in leads I and aVF.





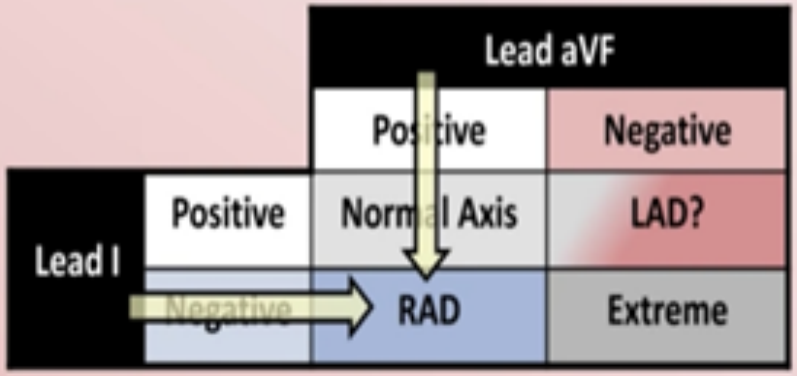
If QRS in I is + and QRS in aVF is -, examine QRS complex in lead II:

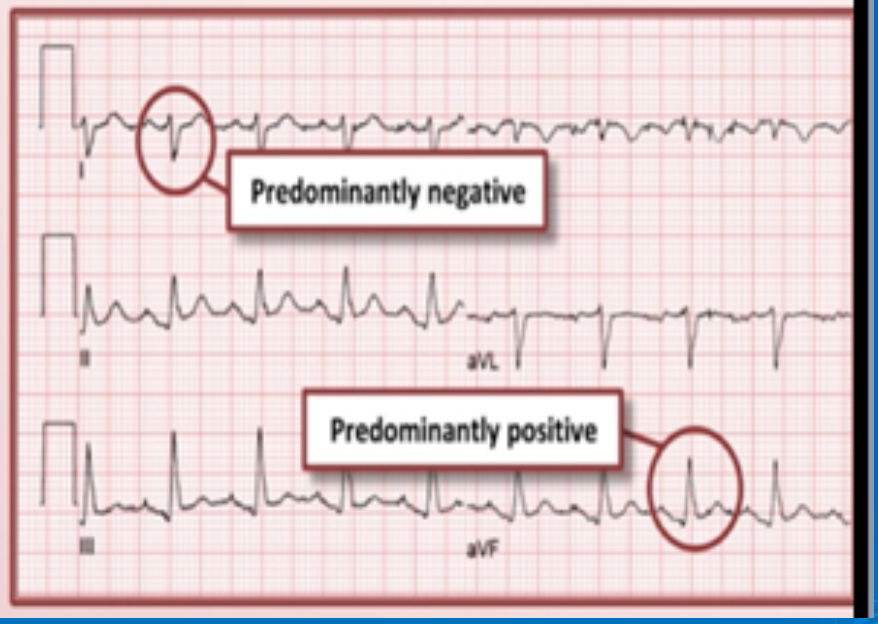
Predominantly positive → Normal (-30° to 0°)
Predominantly negative → LAD (-90° to -30°)

Left Axis Deviation

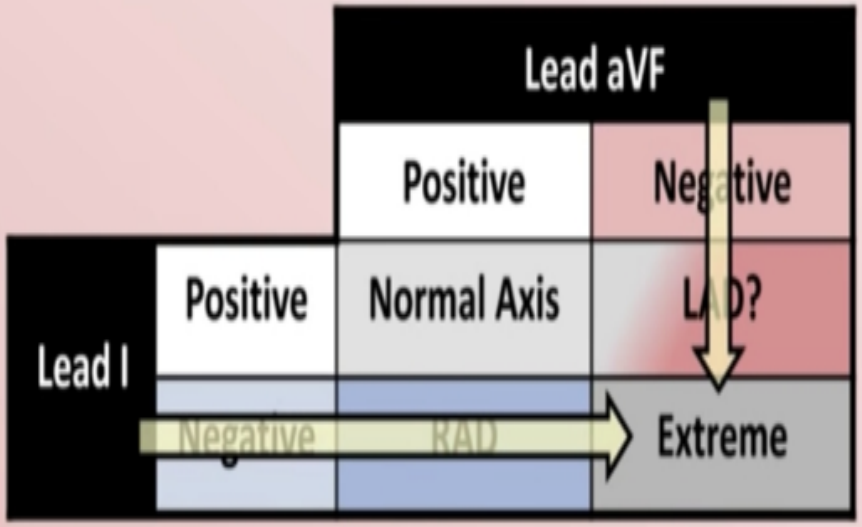
Determining Axis- Quadrant Approach

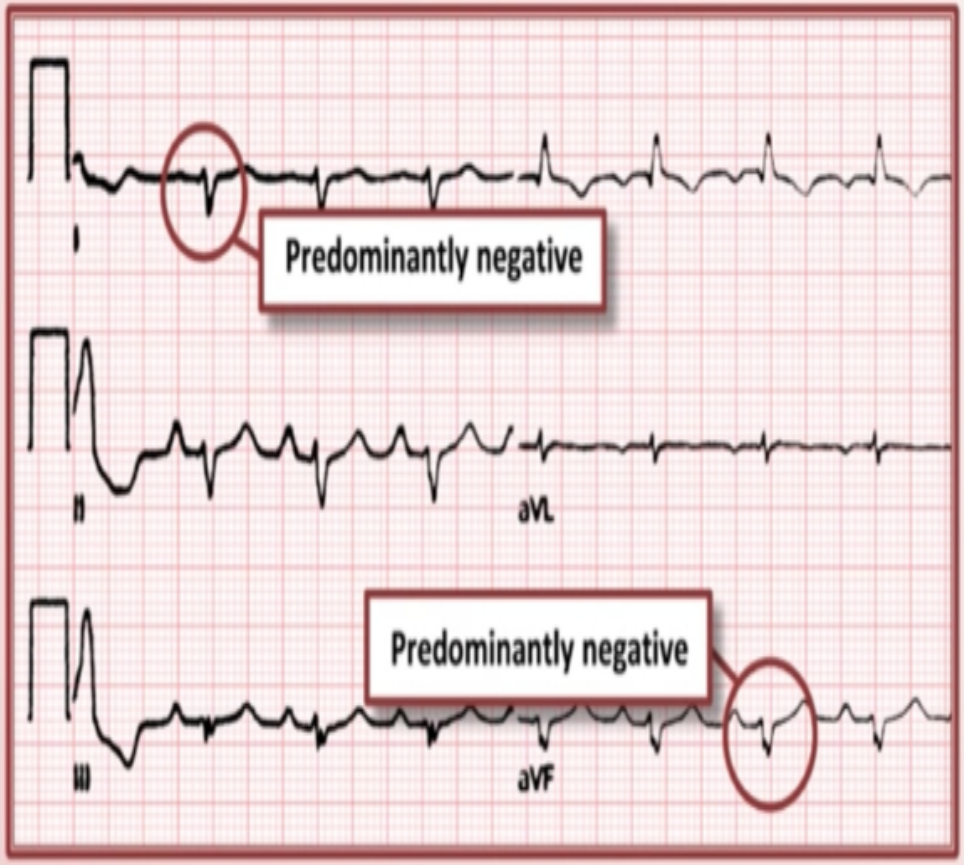
Examine the QRS complex in leads I and aVF.





Examine the QRS complex in leads I and aVF.





If QRS in I is + and QRS in aVF is -, examine QRS complex in lead II:

Predominantly positive → Normal (-30° to 0°)
Predominantly negative → LAD (-90° to -30°)

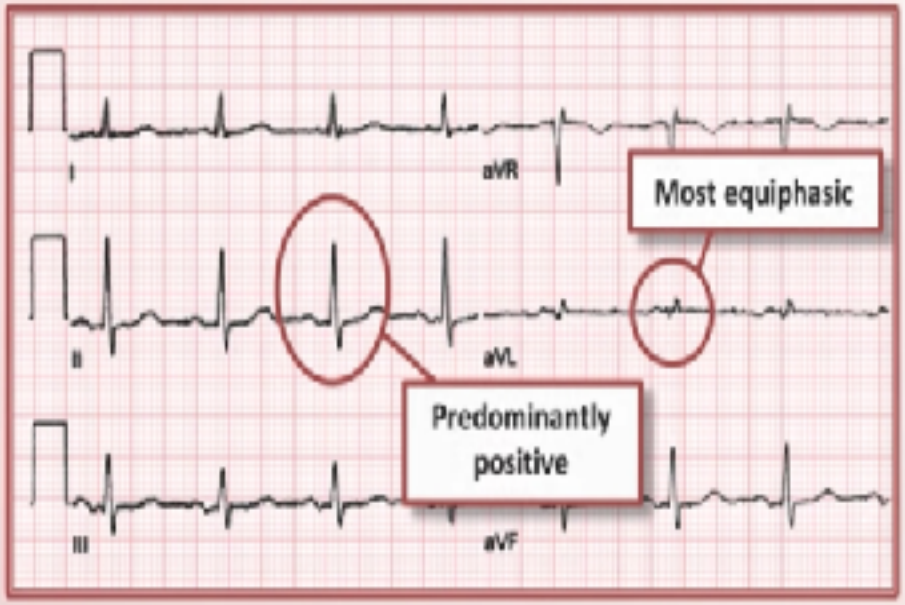
Extreme Axis Deviation

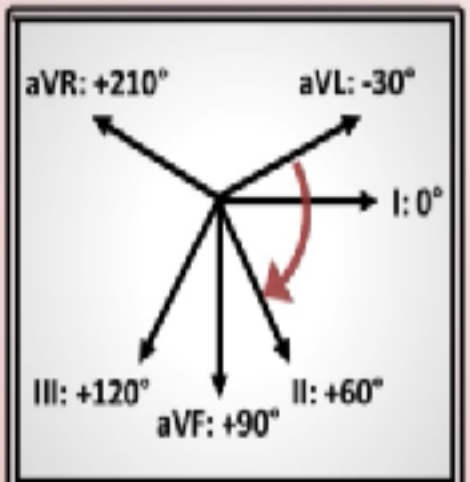
Determining Axis- Equiphasic Approach

1. Determine which lead contains the most equiphasic QRS complex (i.e. the equiphasic lead).

2. Determine which lead lies 90° away from the most equiphasic lead.

3. If the QRS complex in this 2nd lead is predominantly positive, the direction of this lead is approximately the QRS axis. If it is predominantly negative, the QRS axis is 180° away from the direction of this lead.





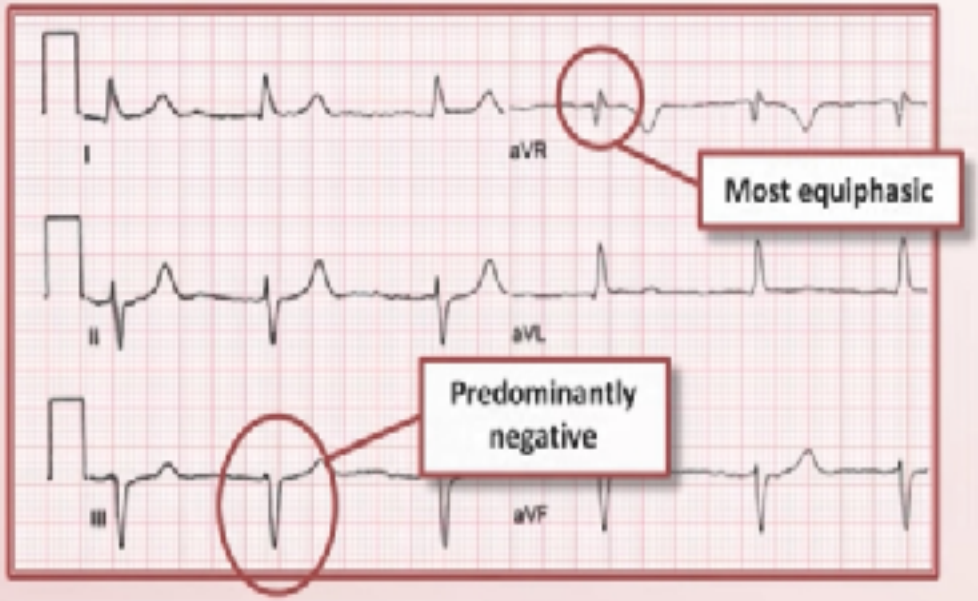
Axis ≈ +60°

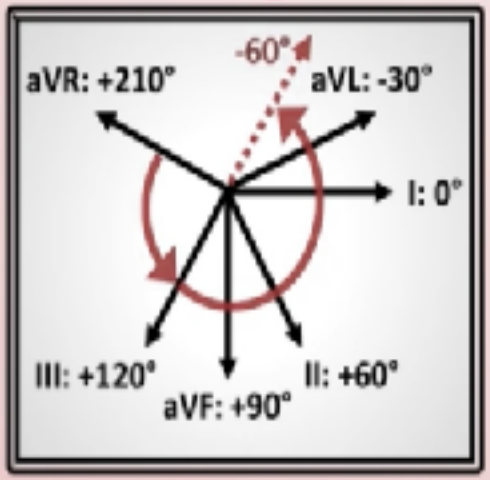
Determining Axis- Equiphasic Approach

1. Determine which lead contains the most equiphasic QRS complex (i.e. the equiphasic lead).

2. Determine which lead lies 90° away from the most equiphasic lead.

3. If the QRS complex in this 2nd lead is predominantly positive, the direction of this lead is approximately the QRS axis. If it is predominantly negative, the QRS axis is 180° away from the direction of this lead.





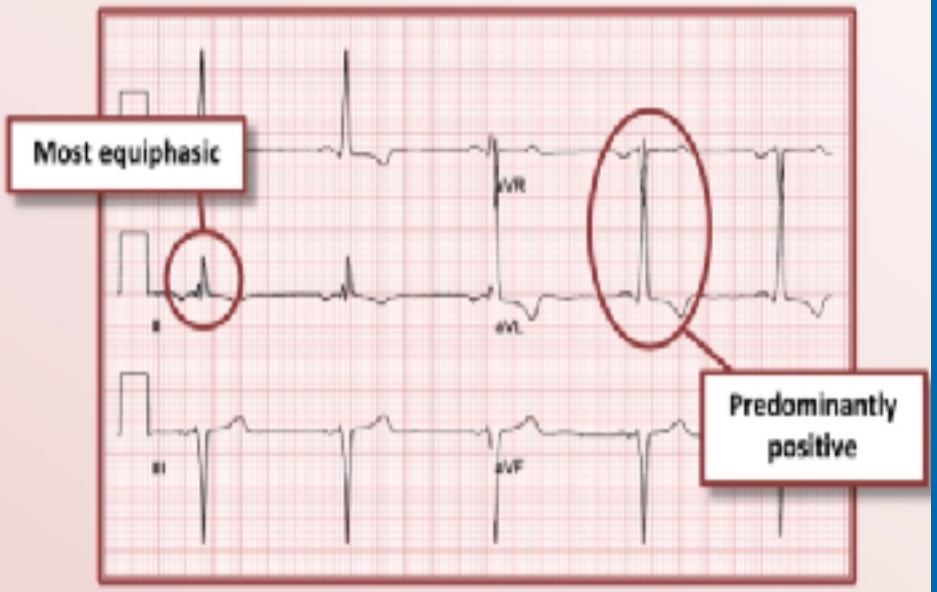
Axis ≈ -60°

Determining Axis- Equiphasic Approach

1. Determine which lead contains the most equiphasic QRS complex (i.e. the equiphasic lead).

2. Determine which lead lies 90° away from the most equiphasic lead.

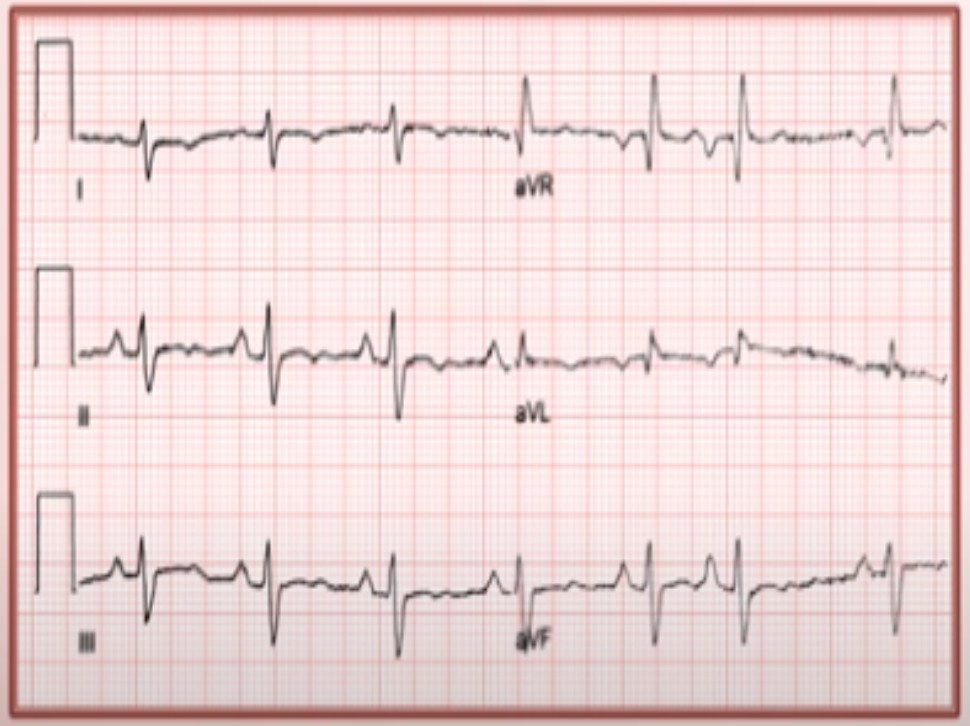
3. If the QRS complex in this 2nd lead is predominantly positive, the direction of this lead is approximately the QRS axis. If it is predominantly negative, the QRS axis is 180° away from the direction of this lead.



Axis ≈ Slightly inferior to -30°

Determining Axis- Equiphasic Approach

- Occurs when all of the limb leads have a QRS complex that is equal parts positive and negative.
- Most commonly seen in COPD as a manifestation of the pulmonary disease pattern.

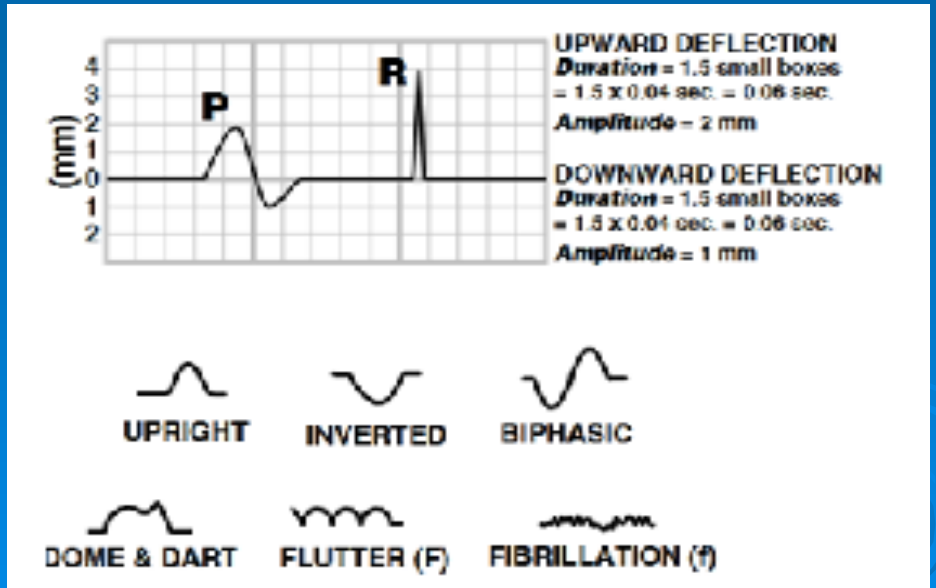


P wave overview

- Monophasic, most positive in lead II
- often biphasic in lead III and V1
- should be upright in leads I and II, most negative in lead aVR
- duration: 0.08-0.10 sec (2.0-2.5 mm), measured at the isoelectric line;

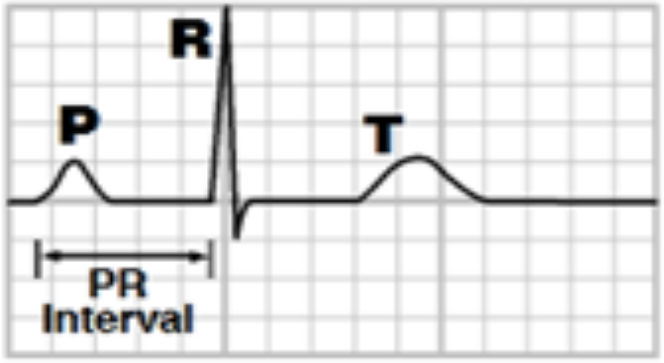
Amplitude

- < 2.5 mm (0.25mV) in the limb leads
- < 1.5 mm (0.15mV) in the precordial leads



PR interval

- reflects conduction through the AV node.
- the normal PR interval is between 120 – 200 ms (0.12-0.20 s) in duration (three to five small squares).
- if the PR interval is > 200 ms, first degree heart block is said to be present.
- PR interval < 120 ms suggests pre-excitation (the presence of an accessory pathway between the atria and ventricles) or AV nodal (junctional) rhythm.

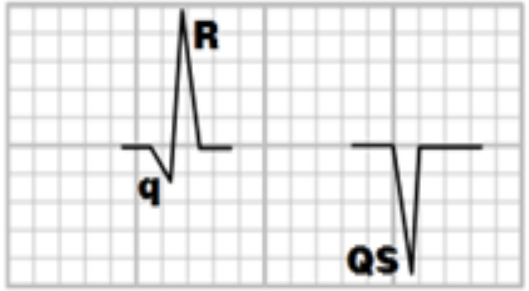


PR INTERVAL = 4 small boxes = 4 x 0.04 = 0.16 sec.

The causes of prolonged P-R interval are:

- Acute rheumatic fever or diphtheria
- Coronary artery disease with fascicular block
- Drugs acting on the A-V node, e.g. digitalis, beta-blockers, calcium-channel blockers.
- P-R interval prolongation is normally observed in vagotonic individuals such as athletes. It is also a normal effect of vagal stimulation (carotid sinus massage) and sympathetic blockade (beta-blocker administration)

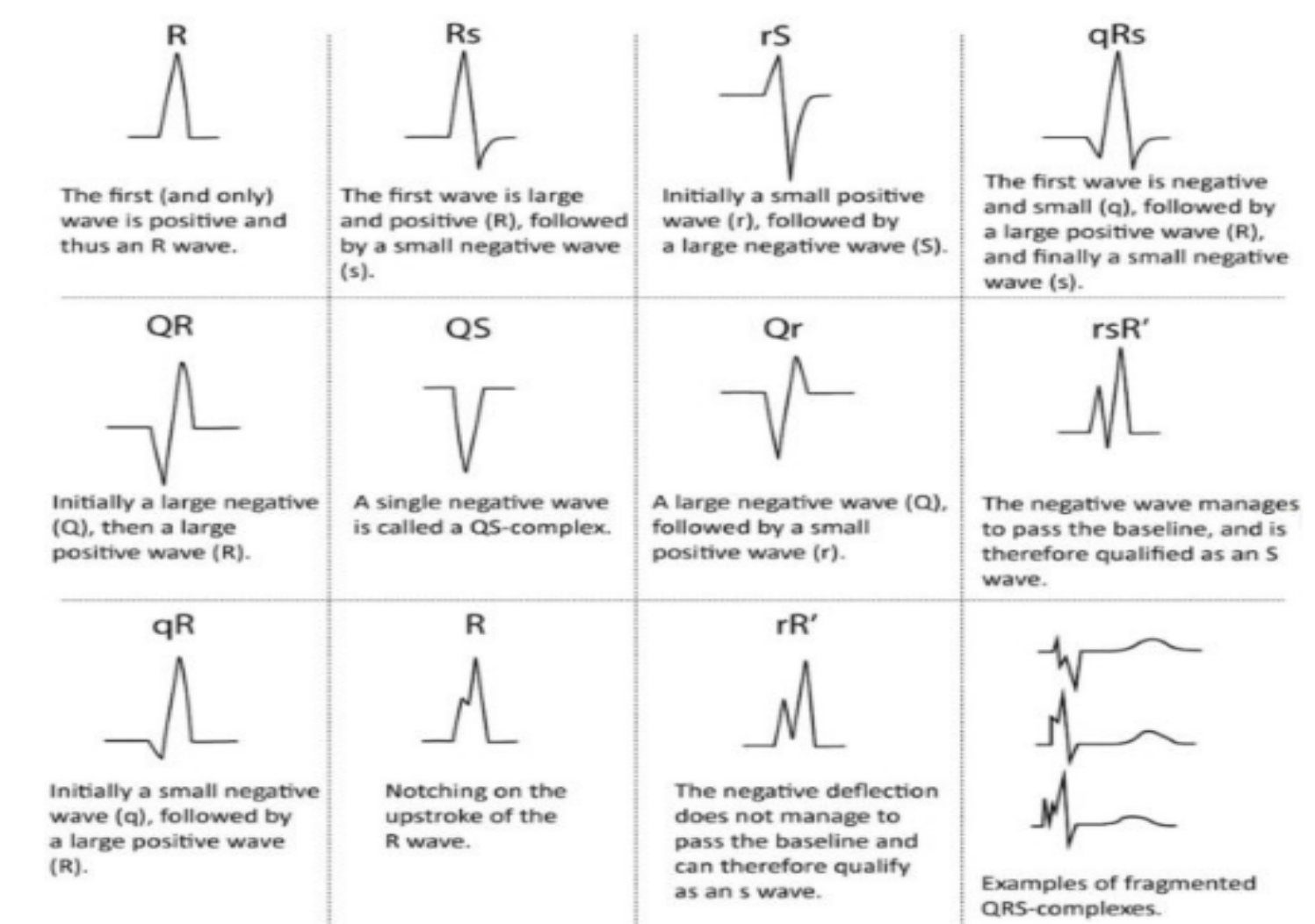
Q wave



Q wave duration = 1 small box
= 0.04 seconds

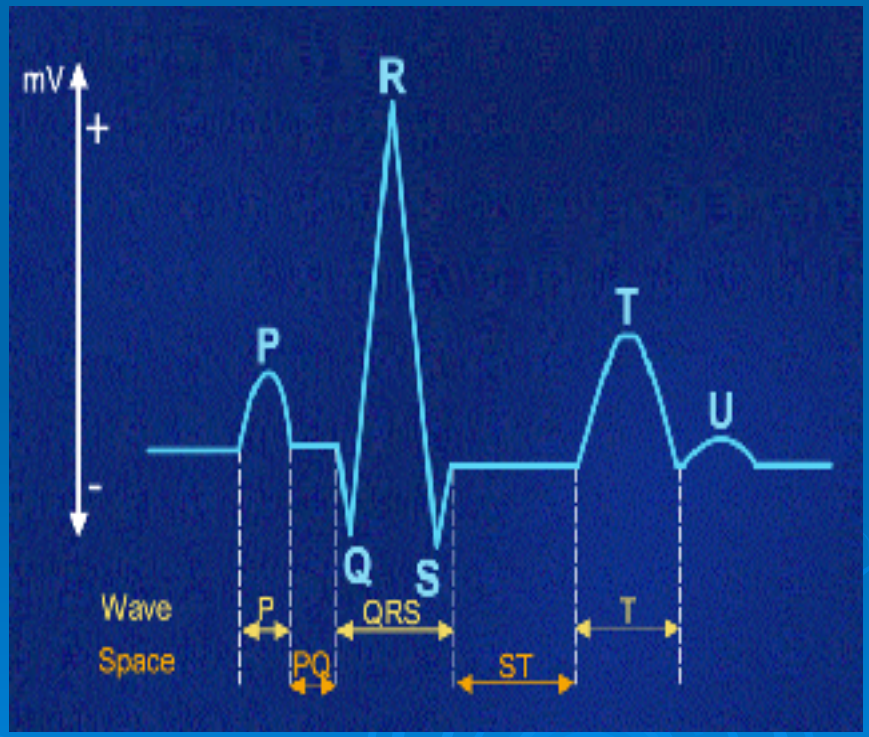
The QRS interval

- The normal QRS duration is about 0.10 sec (or 0.11 sec when measured by computer).
- The QRS duration is slightly longer in males than in females and in large, tall subjects than in small, short subjects.



R wave overview

- The positive wave of the QRS complex is called the R wave, whether or not it is preceded by a Q wave.
- When a second positive deflection occurs, it is termed R'.
- Dominant R wave in V1
- Dominant R wave in aVR
- Poor R wave progression

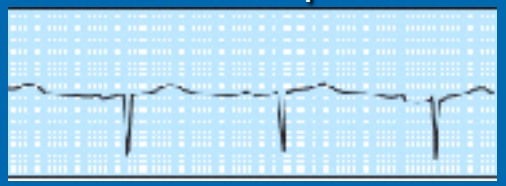


R wave overview

Chest leads - the R wave increases its amplitude and duration from V1 to V4 or V5.

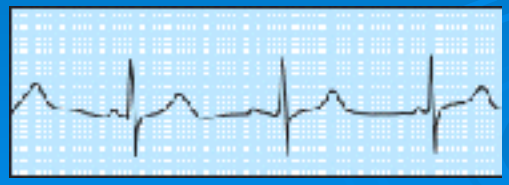
The amplitude of the R wave in leads V5 and V6 varies directly with left ventricular dimension.

- Lead V_1



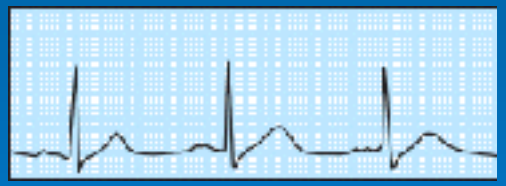
- Lead V_2

- Lead V_3

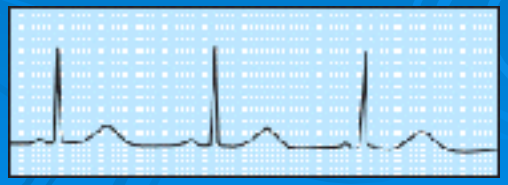


Lead V_4

Lead V_5

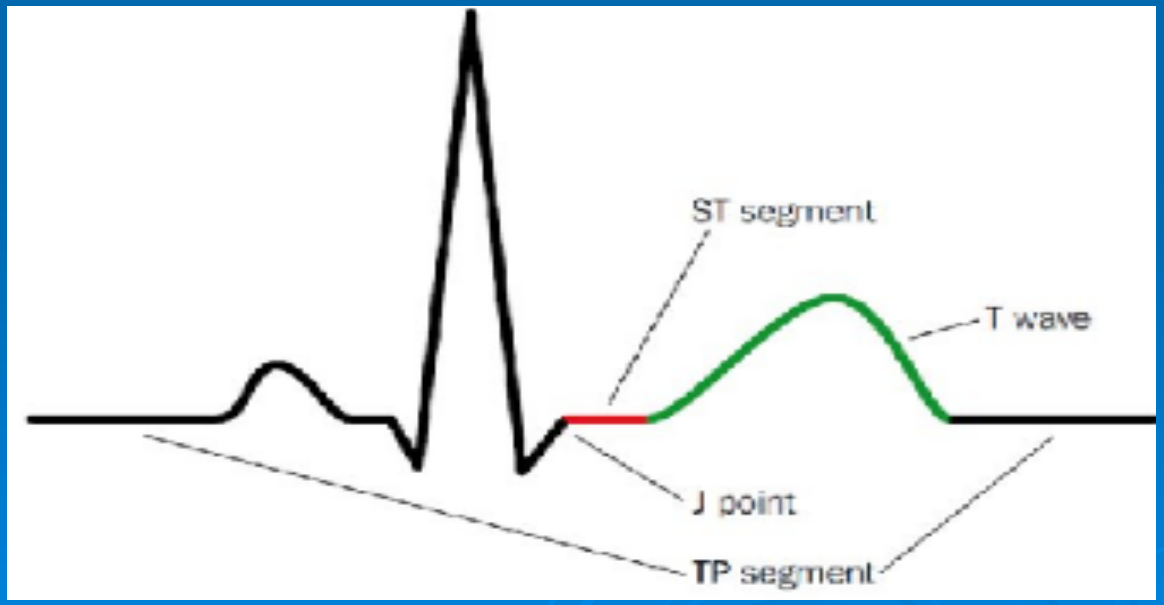


Lead V_6



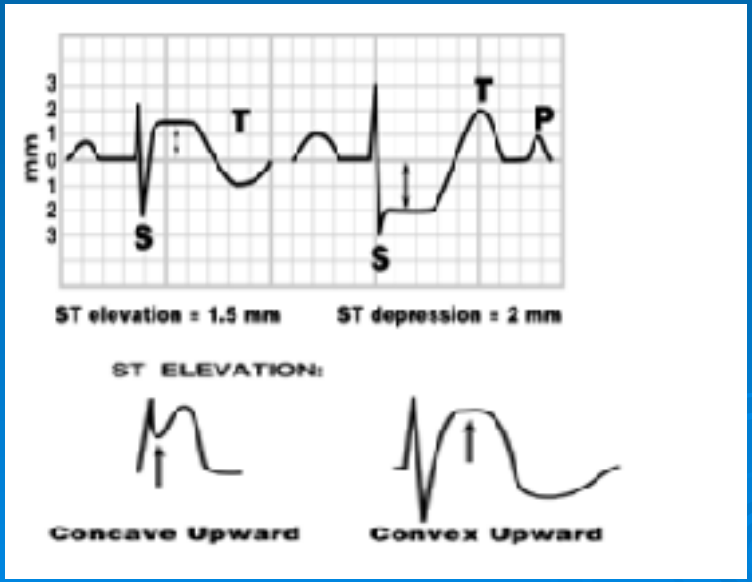
ST segment

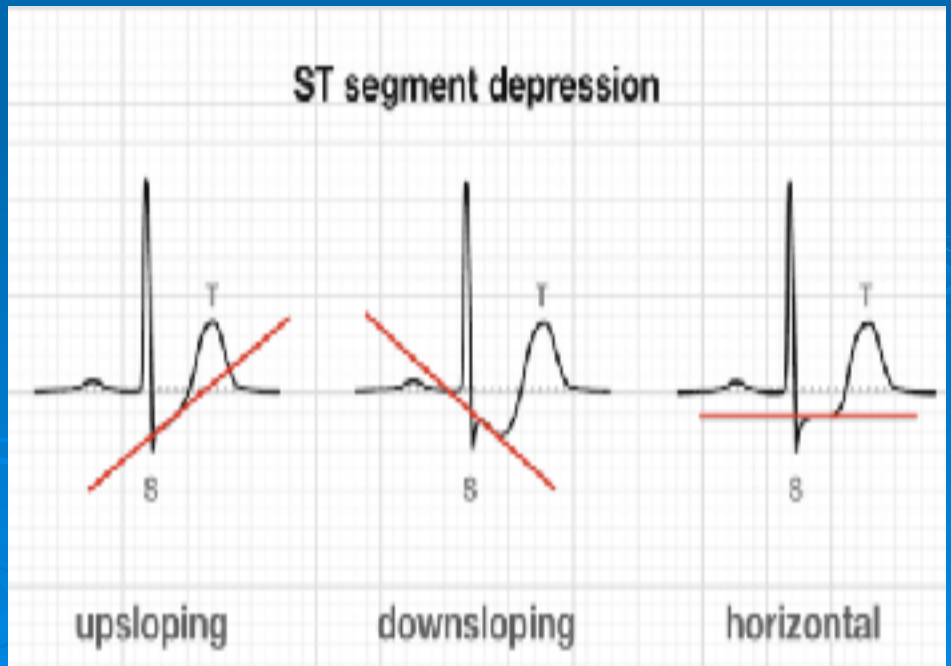
- **The ST segment represents the time from the end of ventricular depolarization to the start of ventricular** repolarization
- is the flat, isoelectric section of the EKG between the end of the S wave (the J point) and the beginning of the T wave**.**
- **The ST segment is normally on the isoelectric line, on the same level with the PR and TP segments**.



ST segment

- The most important cause of ST segment abnormality (elevation or depression) is myocardial ischaemia or infarction.
- ST depression can be either upsloping, downsloping, or horizontal.
- Reciprocal change has a morphology that resembles “upside down” ST elevation and is seen in leads electrically opposite to the site of infarction.





The causes of ST segment depression

- Myocardial ischemia: horizontal or downsloping
- Repolarization changes secondary to ventricular hypertrophy or bundle branch block
- Digitalis effect
- Central nervous system disorder
- Hypokalemia
- Antiarrhythmic drug effect
- Mitral valve prolapse

Non specific causes of ST segment depression are:

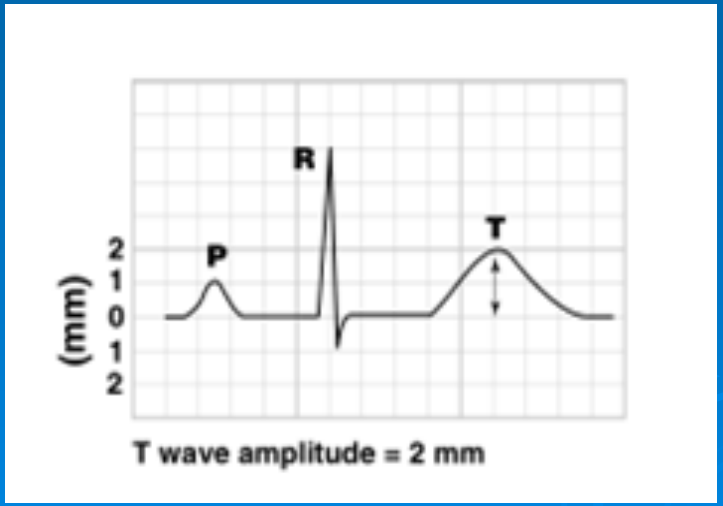
- Physiological states: anxiety, tachycardia, hyperventilation
- Extra-cardiac disorders: hemorrhage, shock, cerebrovascular accident, pancreatitis, cholecystitis, pulmonary embolism.

The causes of ST segment elevation

- Myocardial injury
- Acute pericarditis
- Early repolarization: concave upward ST elevation that ends with an upward T wave, with notching on the downstroke of the R wave. T waves are usually large and symmetrical. ST-T wave changes are stable over a long time period.
- LVH
- Central nervous system disease
- Apical hypertrophic cardiomyopathy
- Hyperkalemia
- Acute cor pulmonale
- Myocarditis
- Myocardial tumor

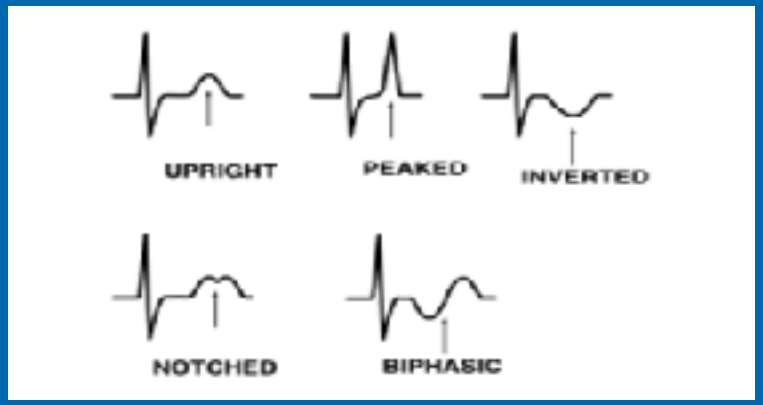
T wave overview

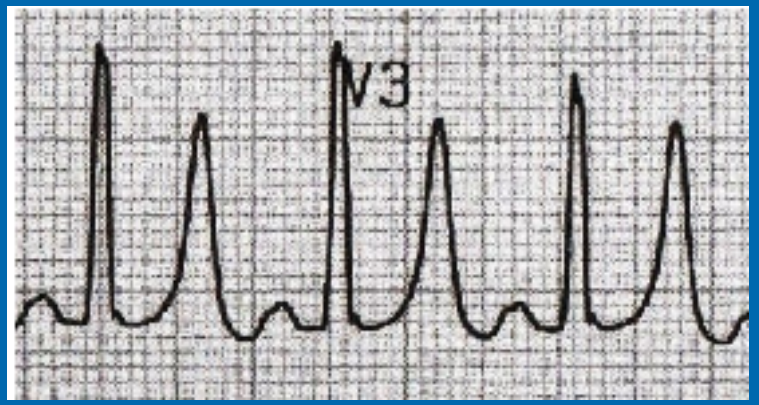
- represents ventricular repolarisation;
- upright in all leads except aVR and V1
- amplitude < 5mm in limb leads, < 10mm in precordial leads (10mm in men, 8mm in women)
- typical and normal to find positive T waves in the same leads that have tall R waves
- Slight “peaking” of the T wave may occur as a normal variant.
- the amplitude or height of normal T wave is one-third to two thirds that of the corresponding R wave



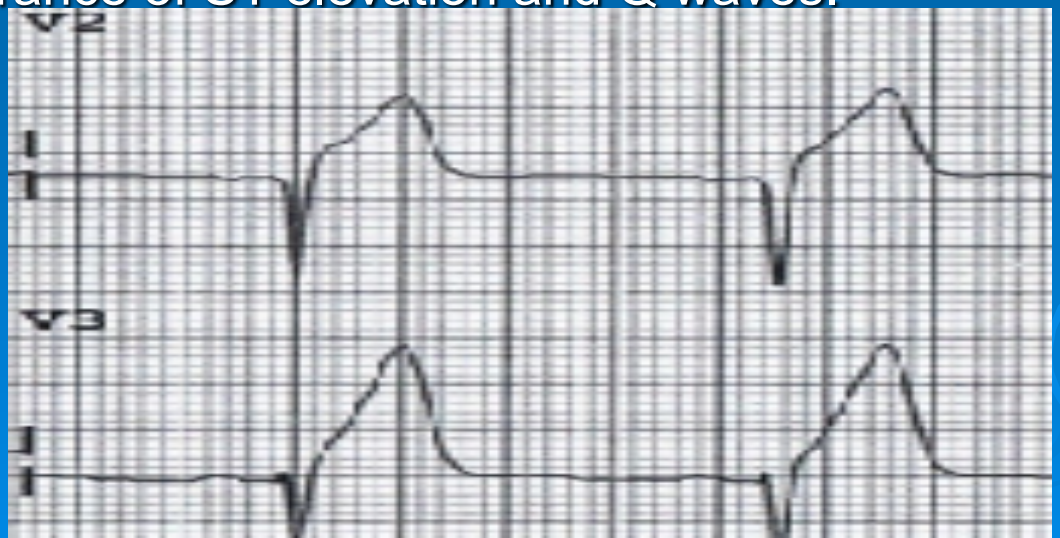
T wave overview

T wave abnormalities



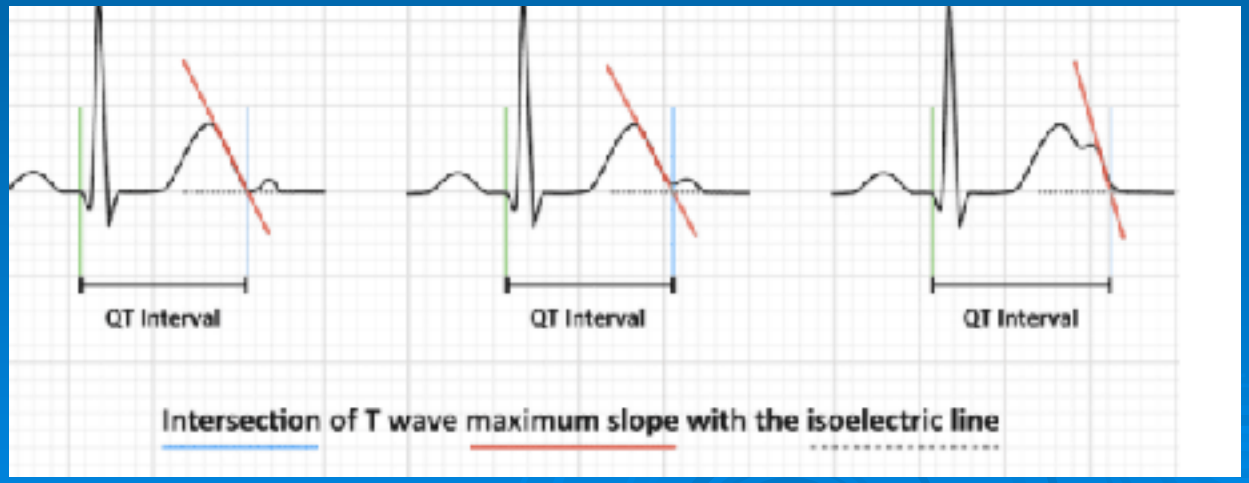


- Broad, asymmetrically peaked or '*hyperacute*' T-waves are seen in the early stages of ST-elevation MI (STEMI) and often precede the appearance of ST elevation and Q waves.



QT interval

- The **QT interval** is the time from the start of the Q wave to the end of the T wave.
- The QT interval *shortens* at faster heart rates
- The QT interval *lengthens* at slower heart rates
- The QT interval should be measured in either lead II or V5-6
- QTc is prolonged if > 440ms in men or > 460ms in women
- QTc > 500 is associated with increased risk of torsades de pointes



Causes of a prolonged Q-T interval

1. Acquired conditions

- Drugs (quinidine, procainamide, disopyramide, amiodarone, sotalol)
- Hypomagnesemia, hypocalcemia
- Marked bradyarrhythmias
- Intracranial hemorrhage
- Myocarditis
- Mitral valve prolapse
- Myxedema
- Hypothermia

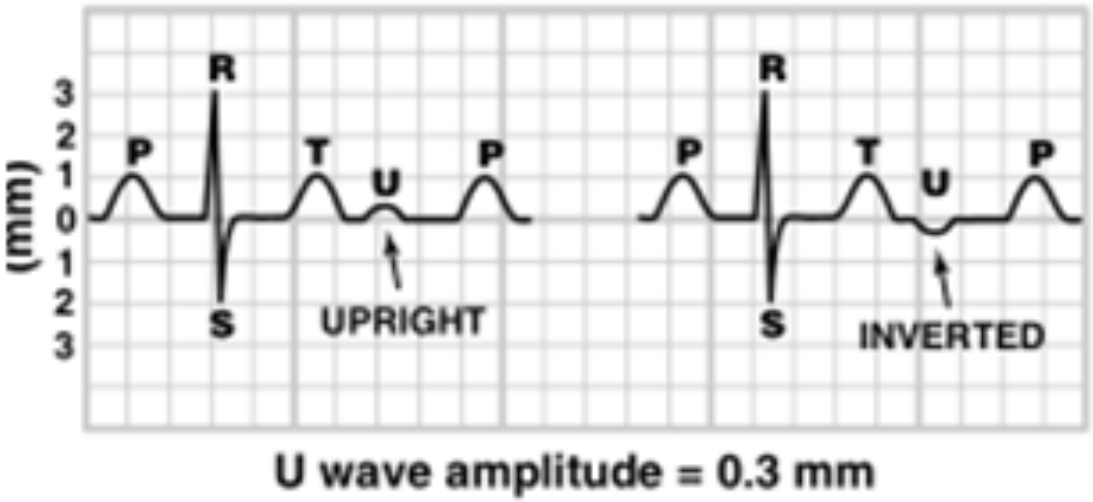
- 2. Congenital disorders

U wave overview

- The U wave is a small (0.5 mm) deflection immediately following the T wave
- U wave is usually in the same direction as the T wave.
- U wave is best seen in leads V2 and V3.

The source of the U wave is unknown. Three common theories regarding its origin are:

- Delayed repolarisation of Purkinje fibres
- Prolonged repolarisation of mid-myocardial “M-cells”
- After-potentials resulting from mechanical forces in the ventricular wall

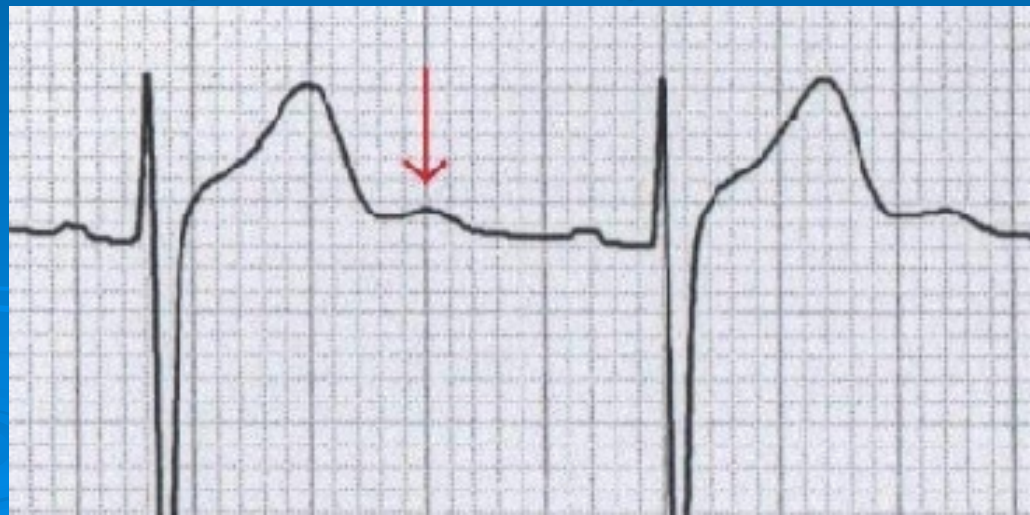


U wave amplitude = 0.3 mm

U wave overview

1. Prominent U wave
   - Hypokalemia
   - Bradyarrhythmias
   - Hypothermia
   - LVH
   - Coronary artery disease
   - Drugs (digitalis, quinidine, amiodarone)

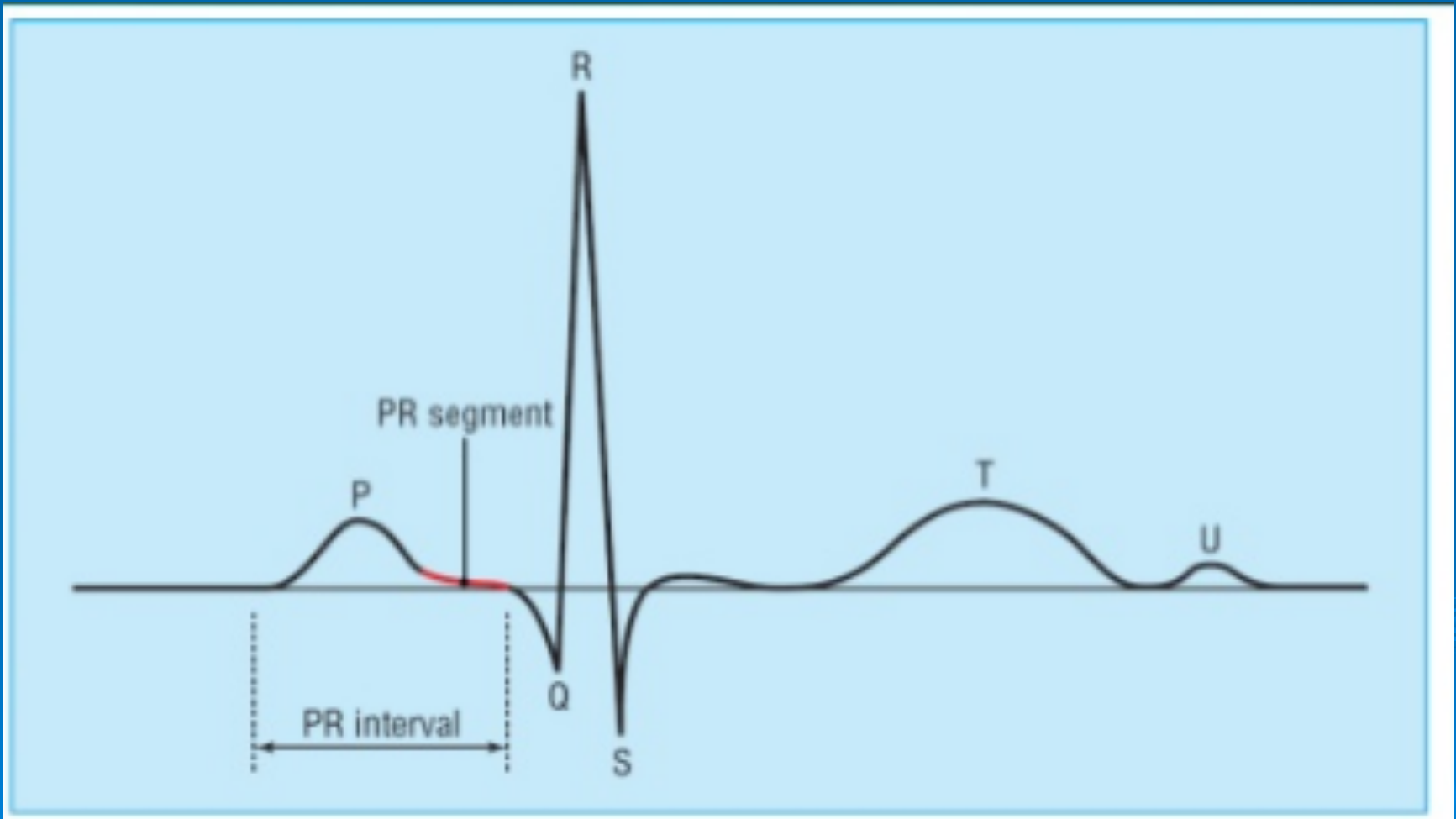
2. Inverted U wave (LVH, severe RVH, Myocardial ischemia)



What do we look for ?
How to report an EKG?

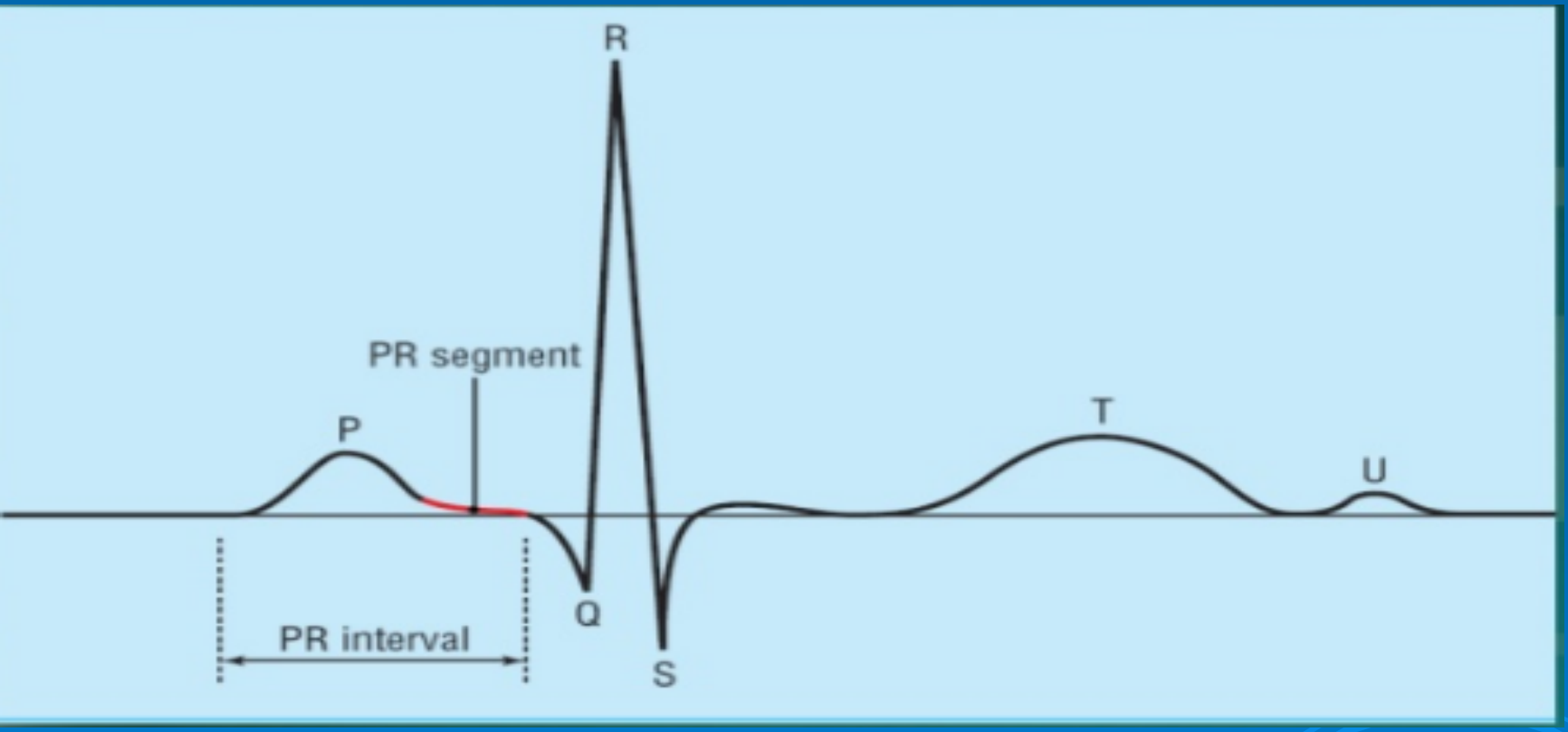
- Patient details, situation details (the time)
- Standardization (voltage calibration), speed paper
- Rhythm (sinus or not)
- Rhytmicity (rhytmical or not)
- Heart rate
- Mean QRS axis
- PR, QRS and QT intervals (duration)
- P wave (polarity, duration and height)
- Precordial R-wave progression
- Abnormal Q wave (wide and deep)
- ST segment (position according isoelectric line)
- T waves (polarity, symmetricity and height)
- U waves (if present)

ECG Rules
Rule 1



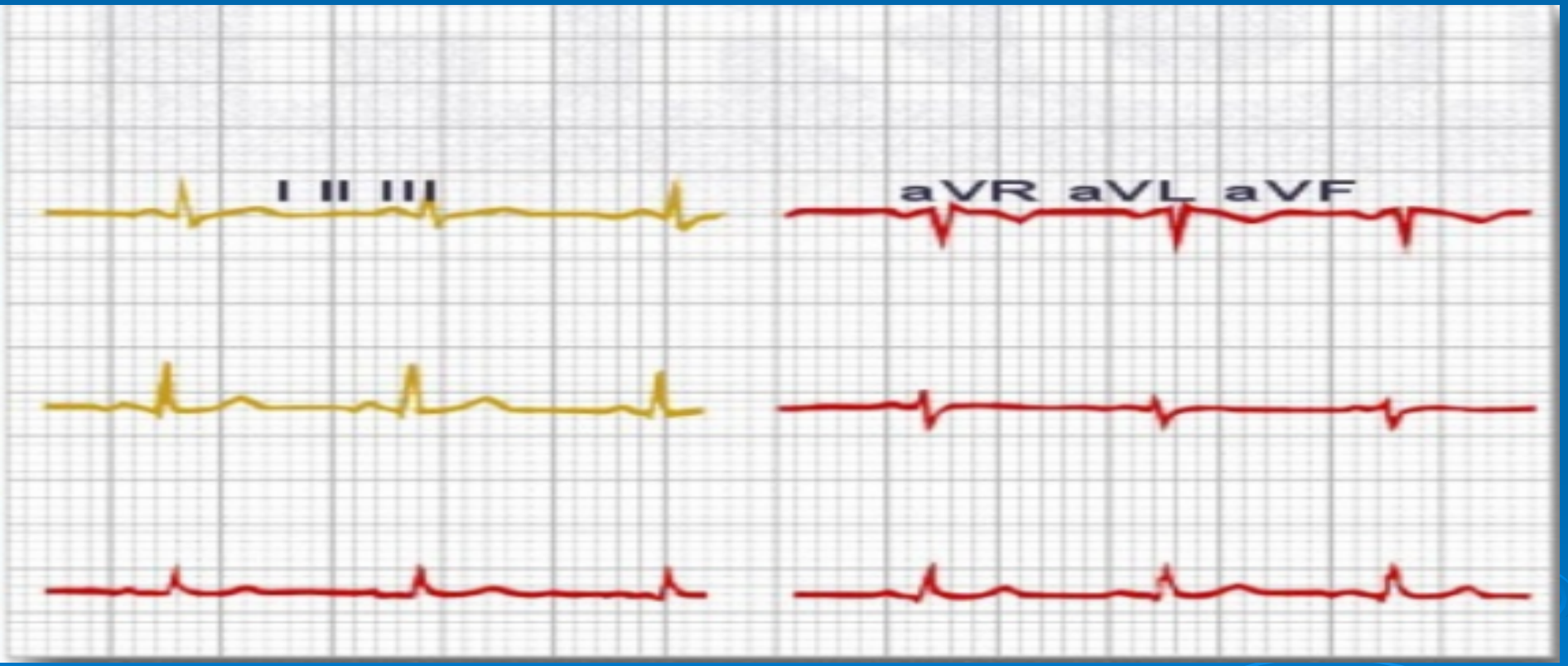
Normal duration of PR interval is 0.12-0.20 s (three to five small squares)

Rule 2



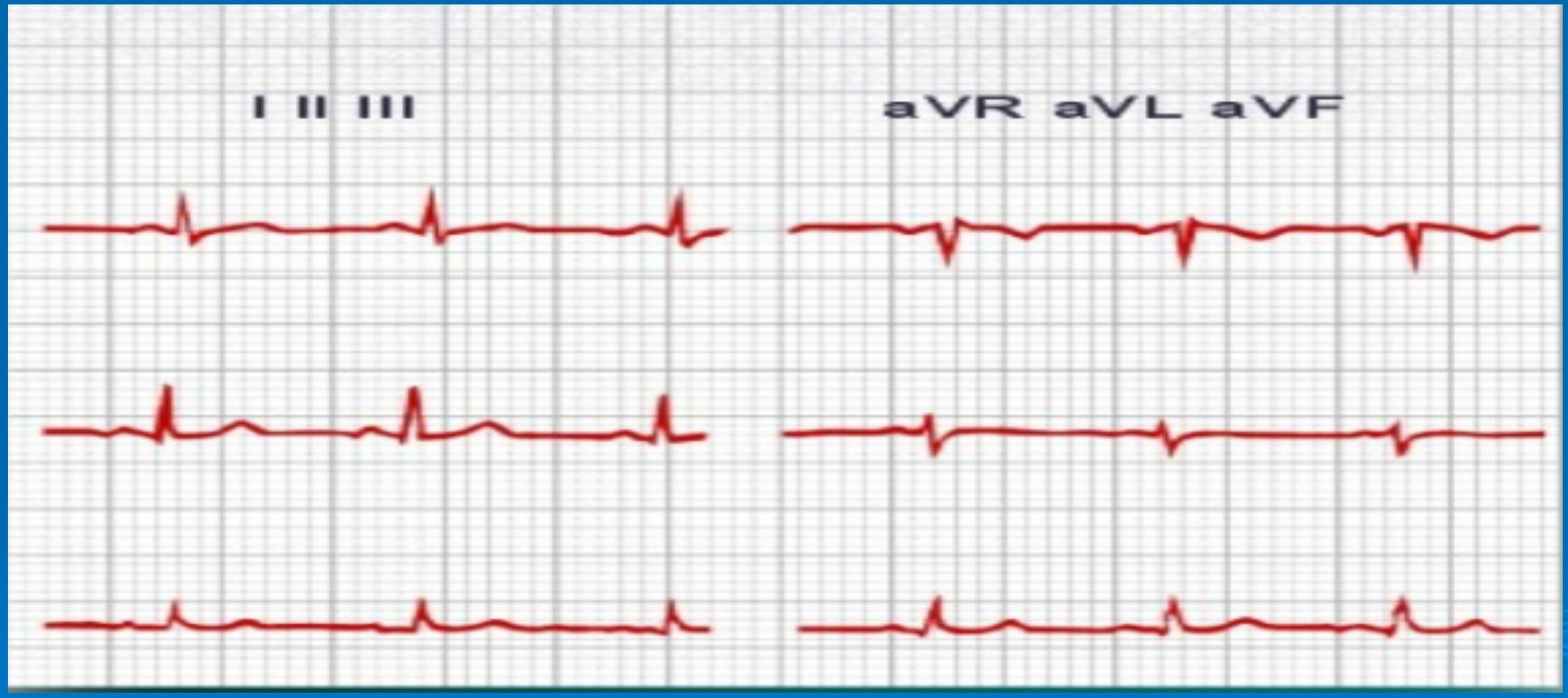
The width of the QRS complex should not exceed 110 ms, less than 3 little squares

Rule 3



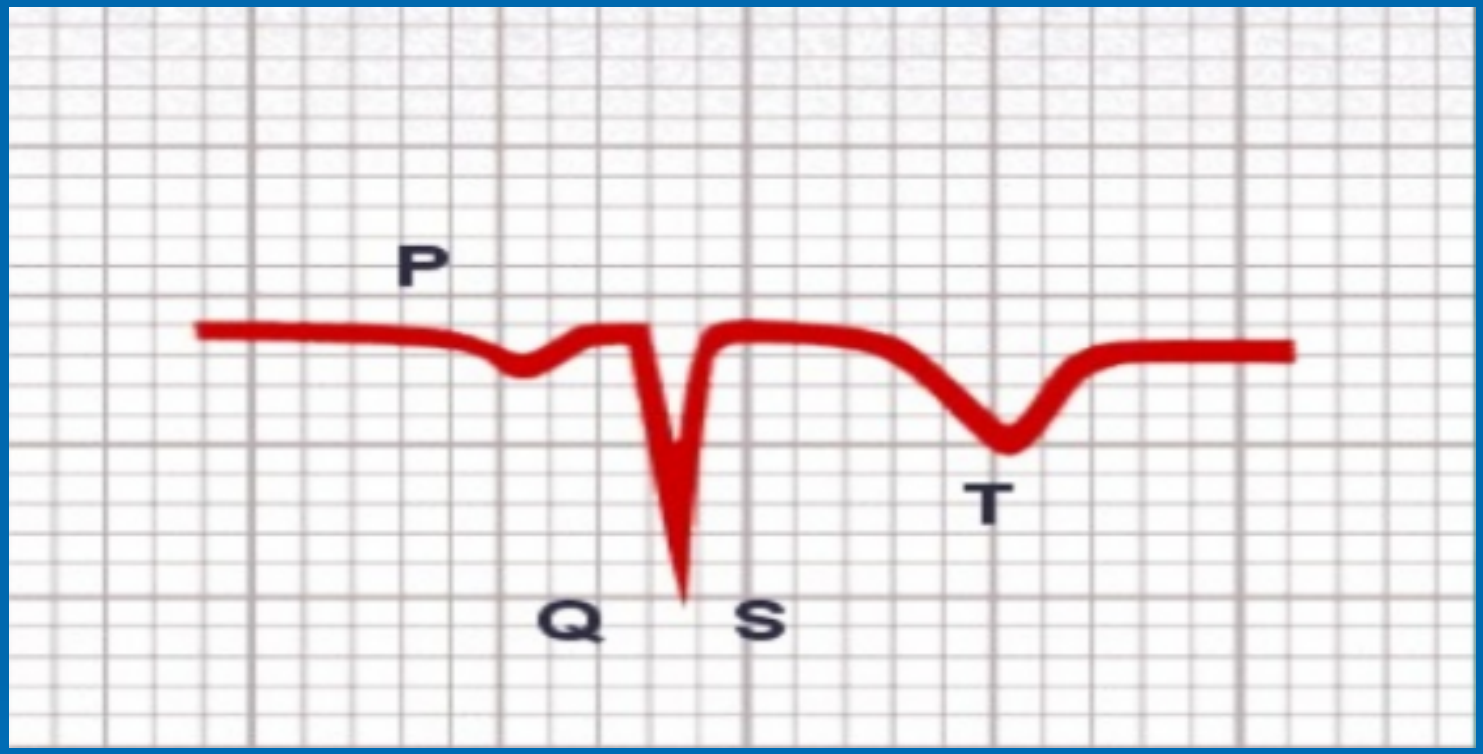
The QRS complex should be dominantly upright in leads I and II .

Rule 4



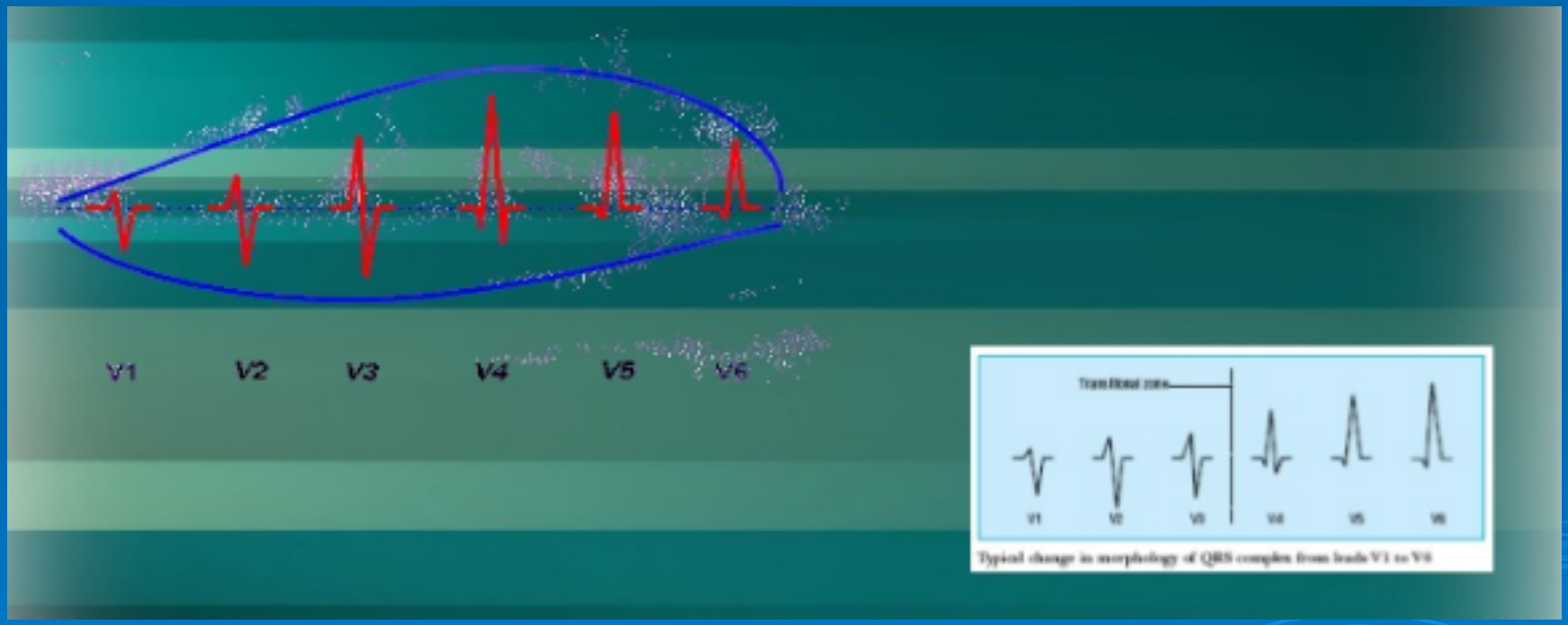
QRS and T waves tend to have the same general direction in the limb leads .

Rule 5



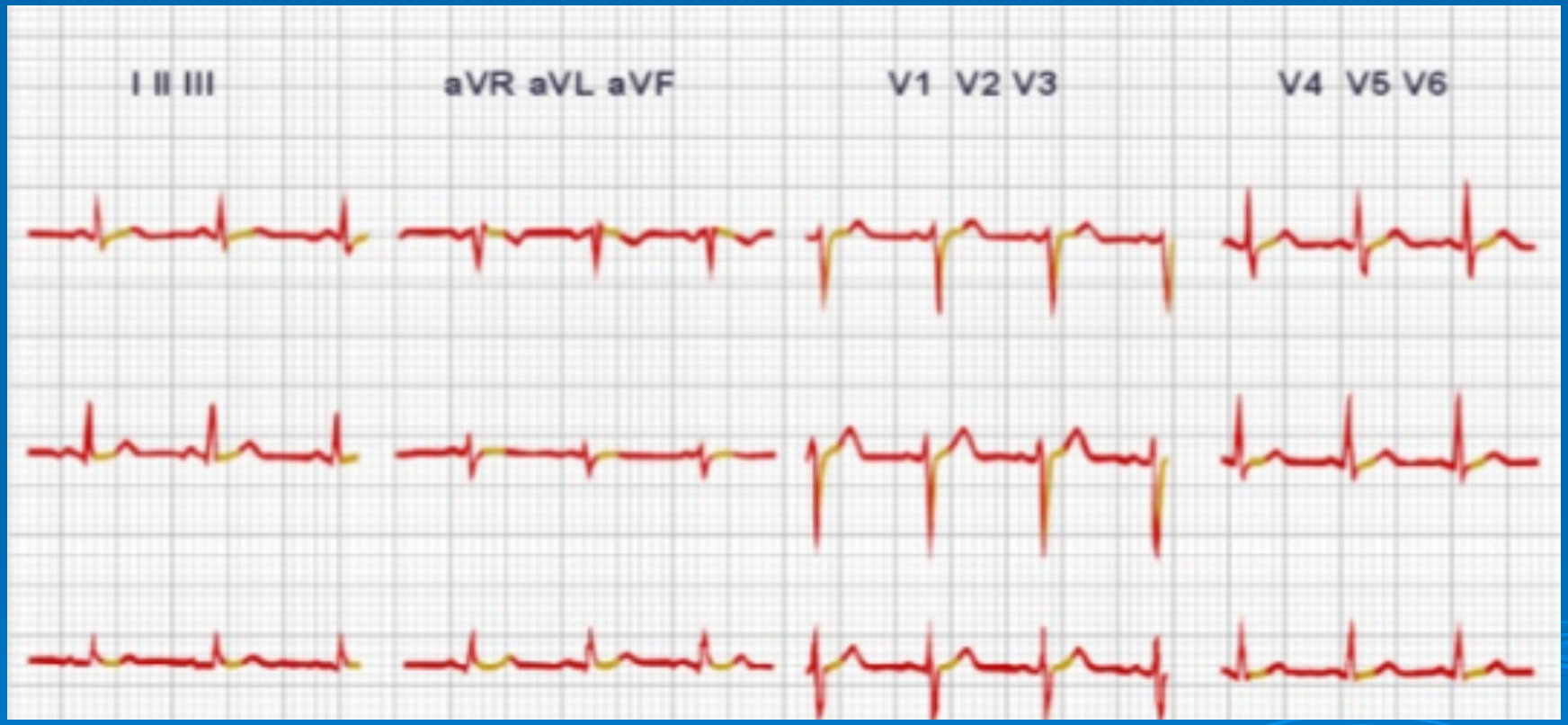
All waves are negative in lead aVR.

Rule 6



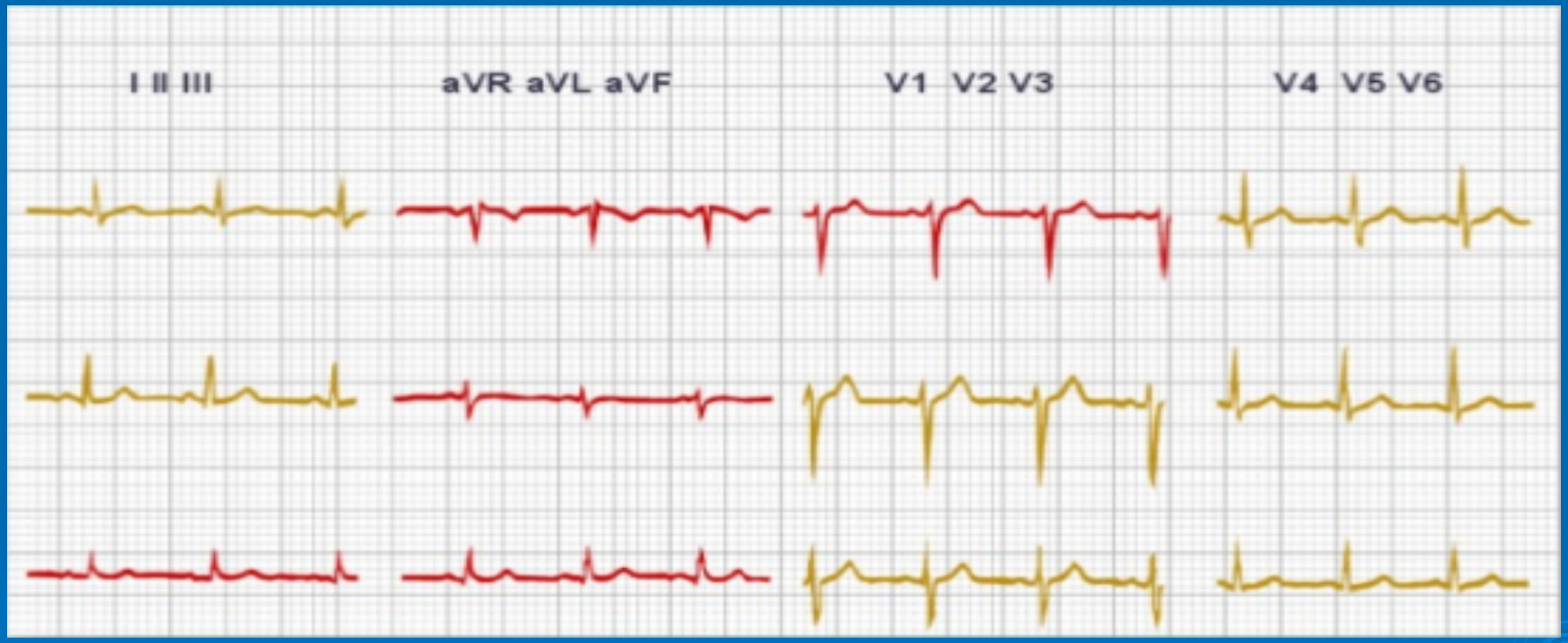
The R wave must grow from V1 to at least V4.
The S wave must grow from V1 to at least V3 and disappear in V6 .

Rule 7



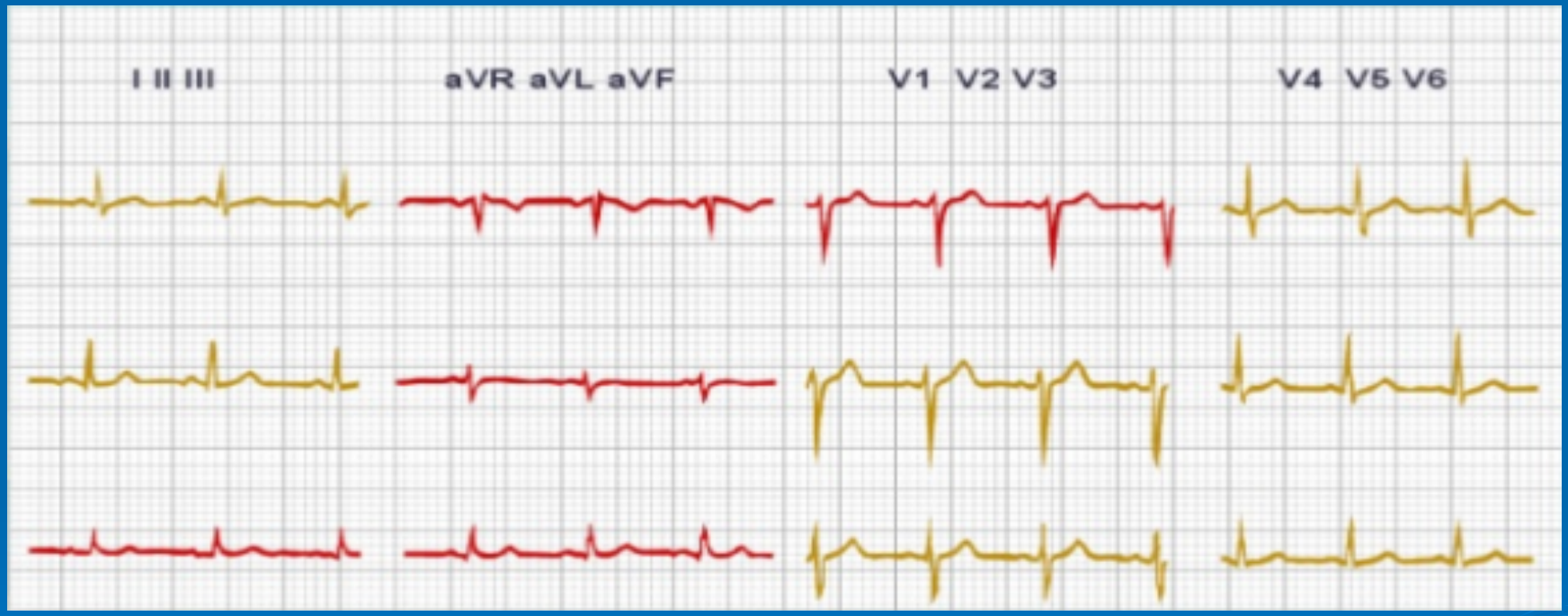
The ST segment should start isoelectric except in V1 and V2 where it may be elevated.

Rule 8



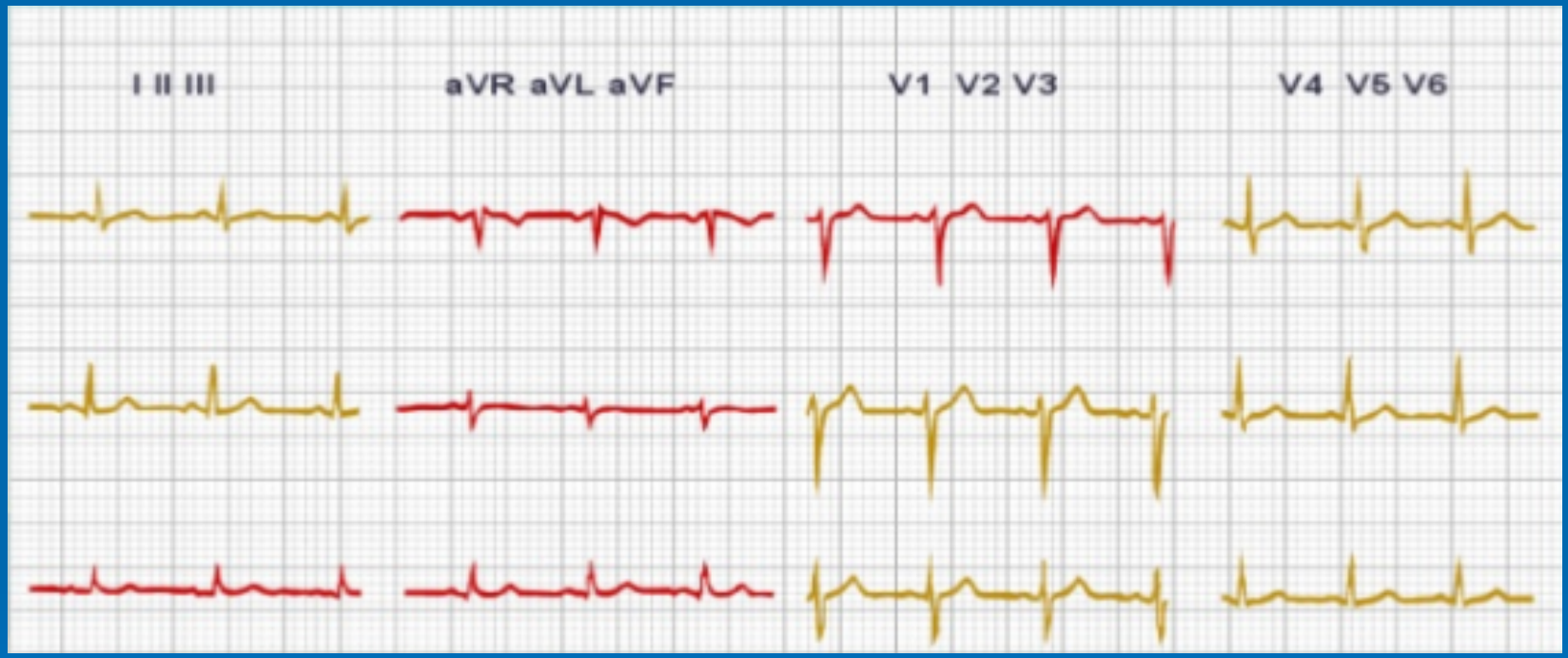
The P waves should be upright in I, II, and V2 to V6 .

Rule 9

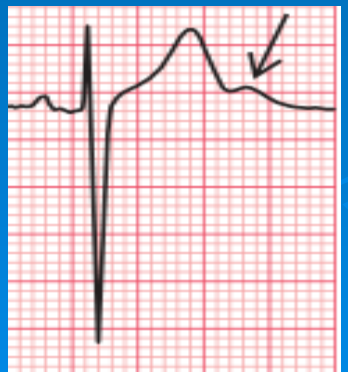


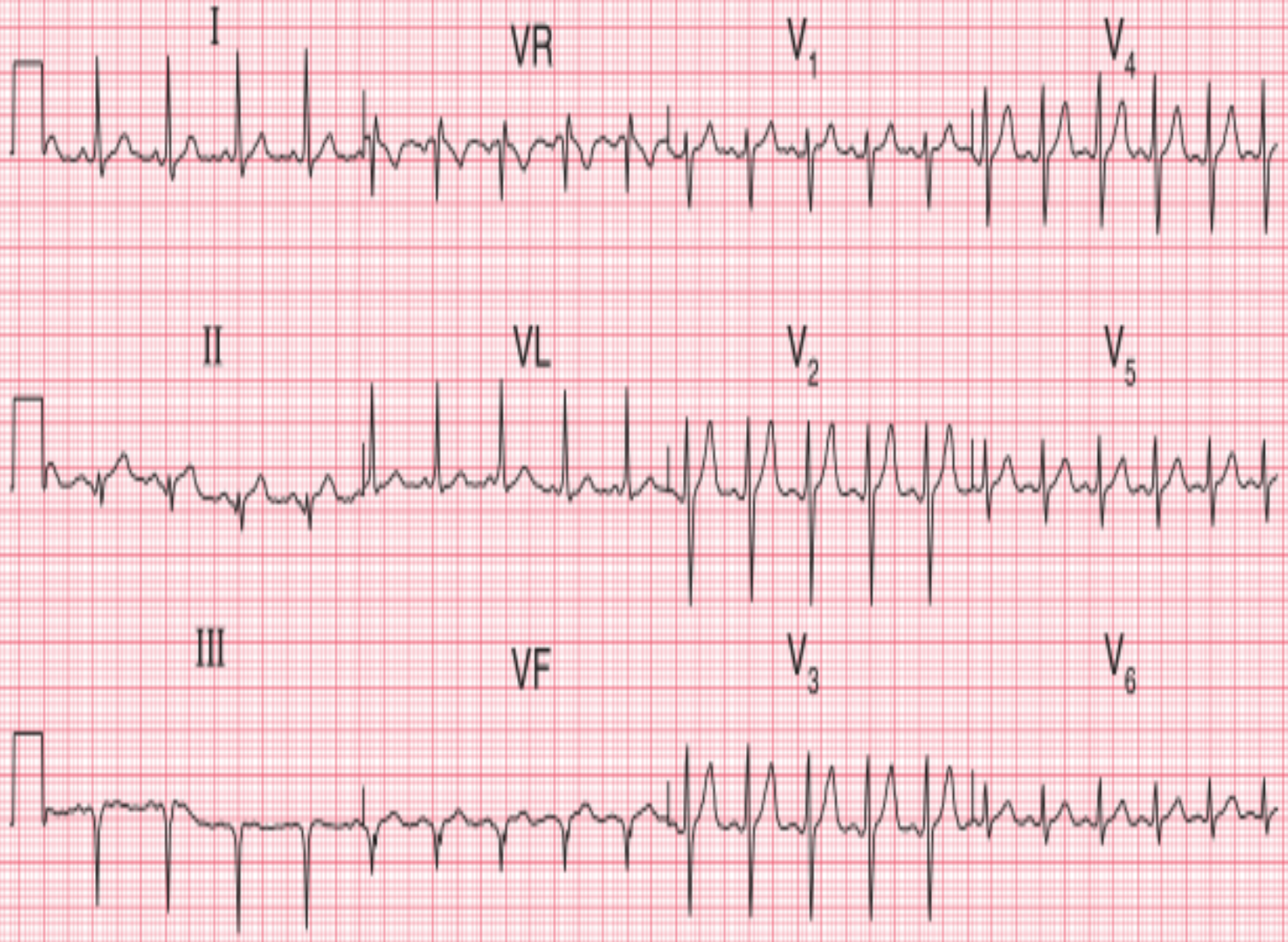
There should be no Q wave or only a small q less than 0.04 seconds in width in I, II, V2 to V6.

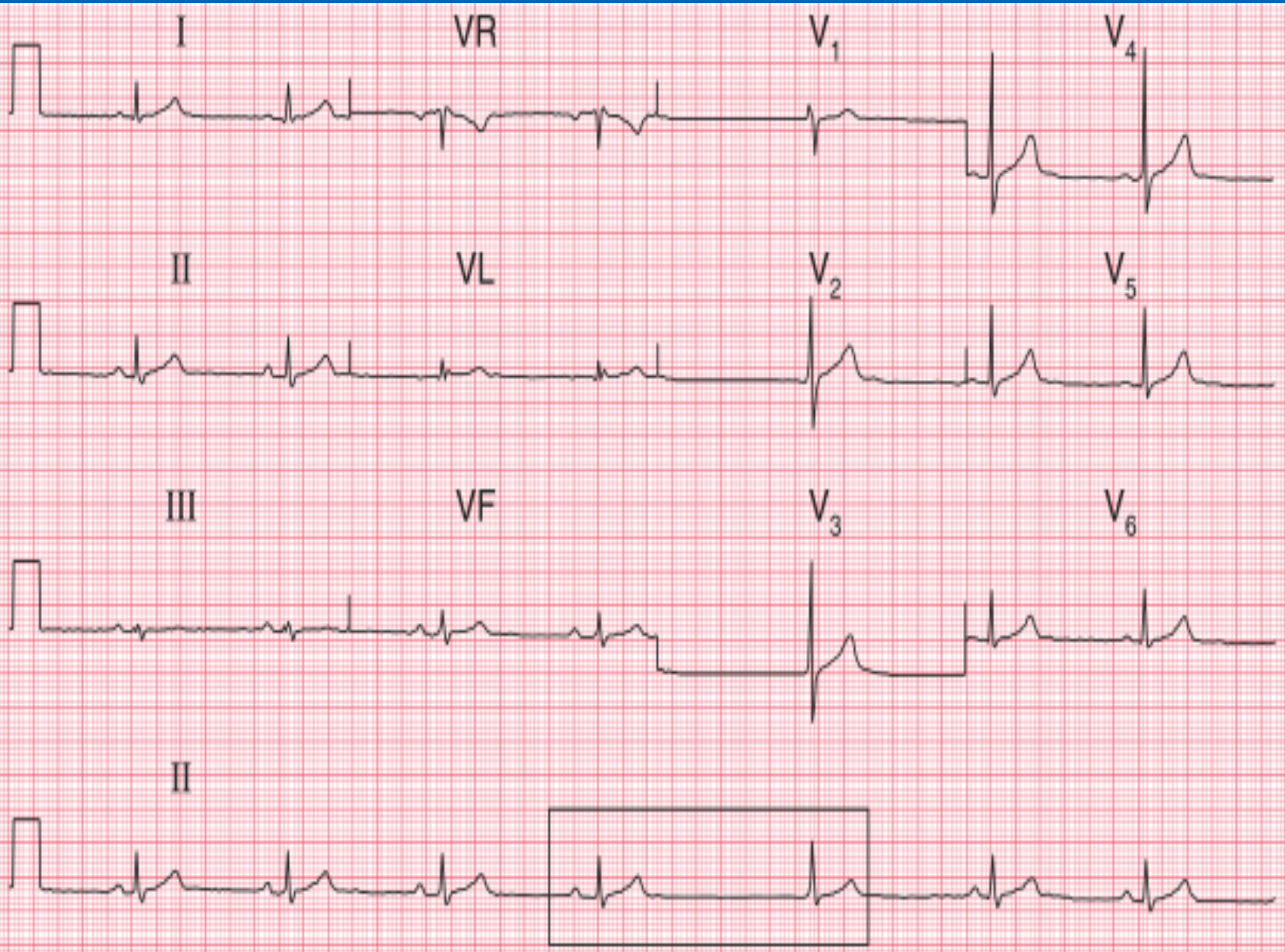
Rule 10



The T wave must be upright in I, II, V2 to V6 .



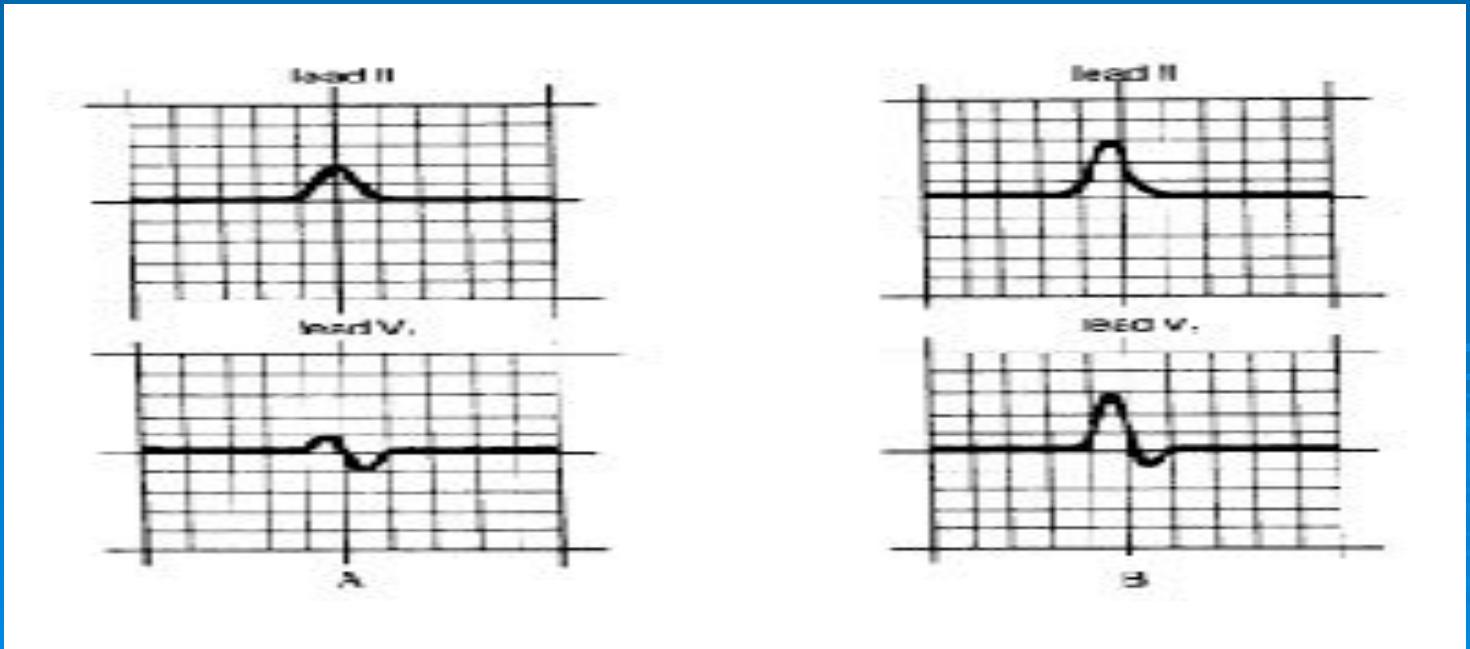

I
VR
V1
V4
II
VL
V2
V5
III
VF
V3
V6


I
VR
V1
V4
II
VL
V2
V5
III
VF
V3
V6
II

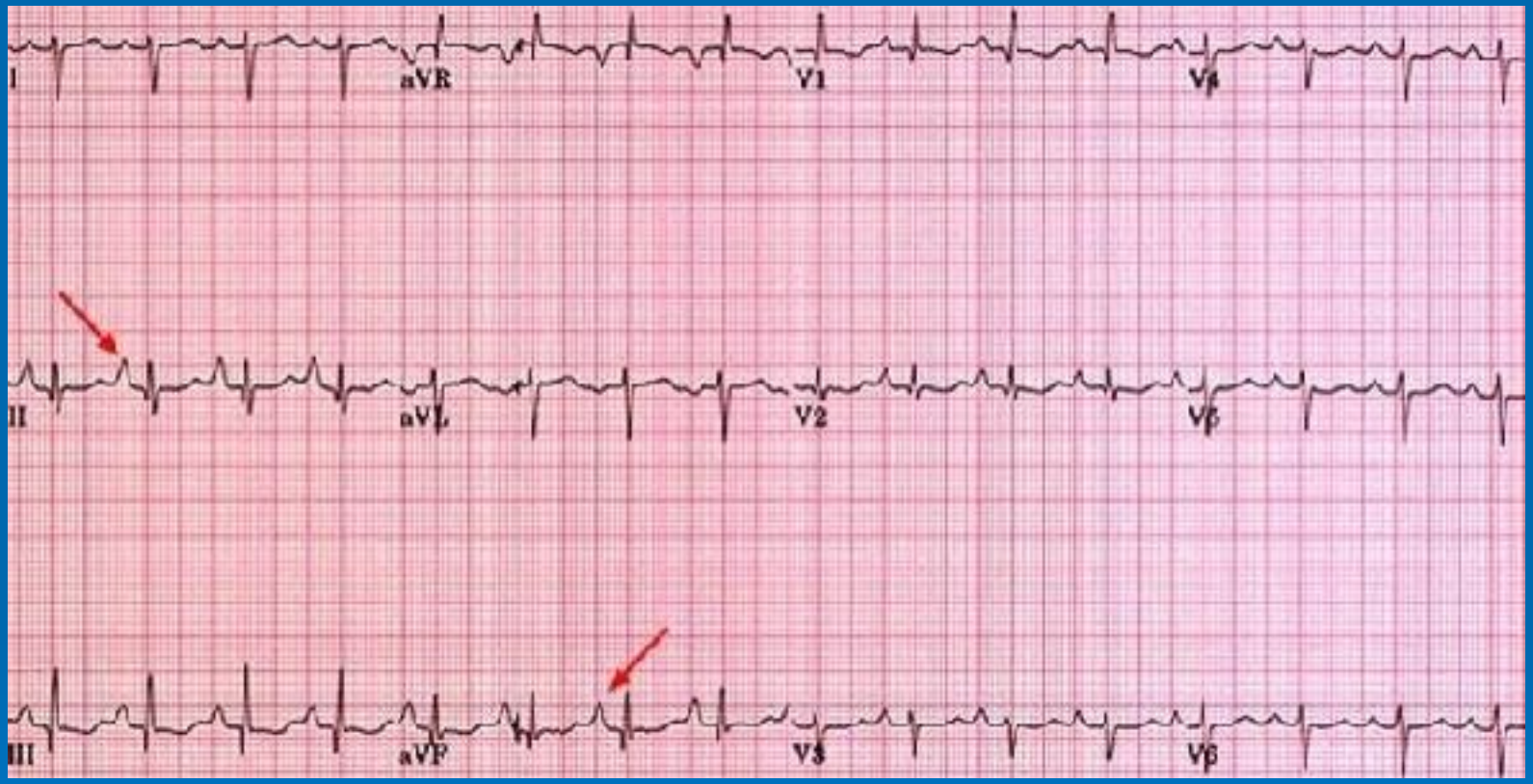
Right atrial enlargement

Tall upright P wave:

- 2.5 mm in leads II, III, and aVF *(P-pulmonale*), or
- 1.5 mm in leads V1 or V2
- It is called “P pulmonale”, because it is often met in cor pulmonare.
- Possible right axis deviation of the P wave



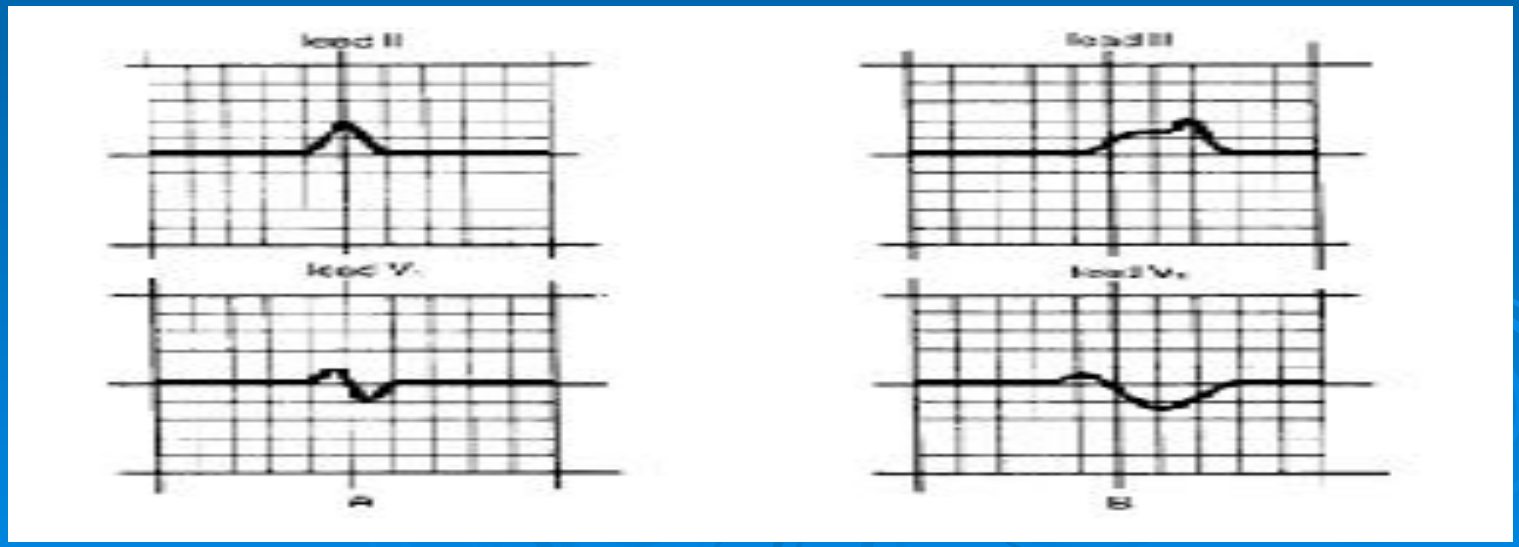
Right atrial enlargement



Right atrial enlargement is commonly associated with congenital heart disease, tricuspid valve disease, pulmonary hypertension and diffuse lung disease.

Left atrial enlargement

- The P wave sometimes has a distinctive humped or notched appearance;
- Terminal negative portion of the P wave in lead V1 ≥1mm deep and ≥ 0.04 seconds in duration (one small box deep and one small box wide), or
- Notched P wave with a duration ≥0.12 seconds in leads II, III or aVF (*P-mitrale*).



LEFT ATRIAL ENLARGEMENT

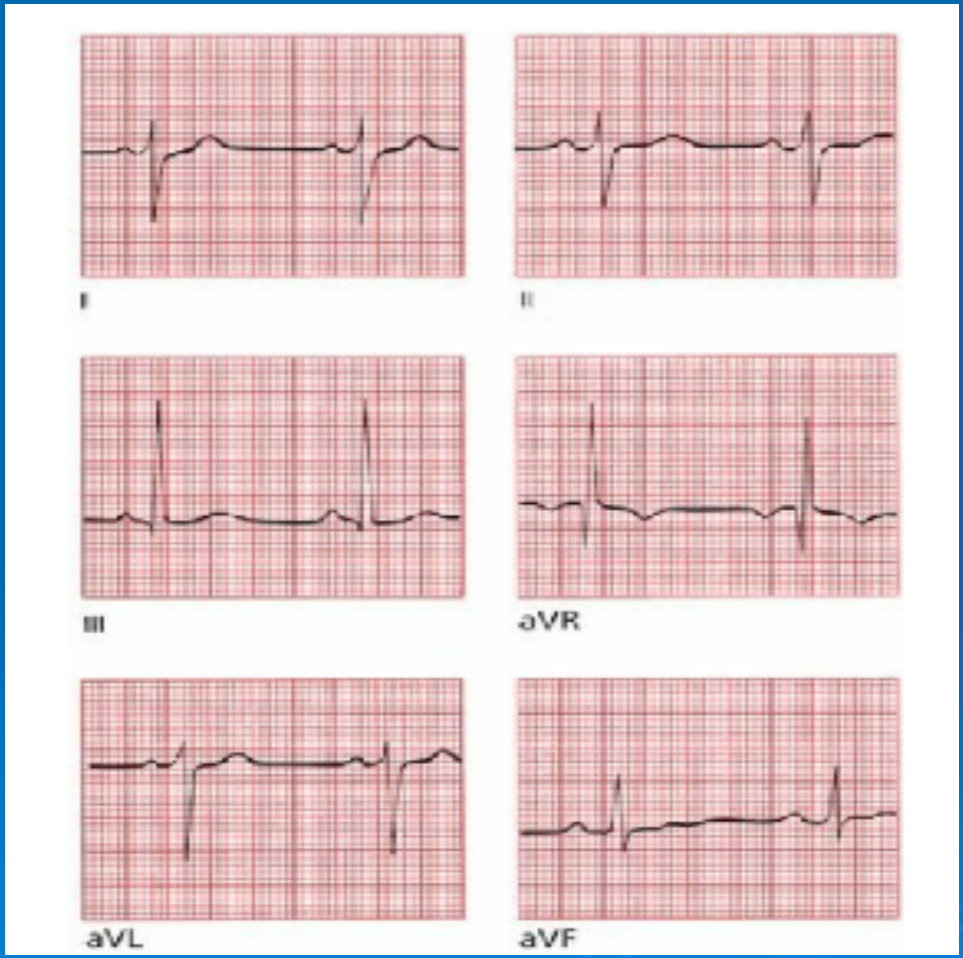
CAUSE : STENOTIC MITRAL VALVE

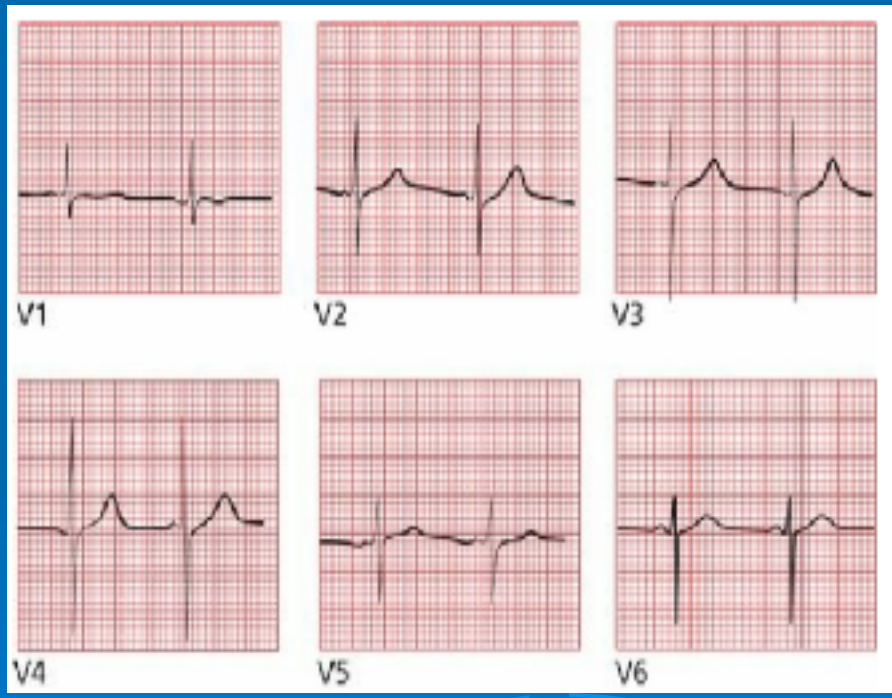
Right Ventricular Hypertrophy

- The R wave is larger than S wave in V1, whereas the S wave is larger than the R wave in V6
- R in V1 > 7 mm;
- S in V5 or V6 >7 mm;
- Right axis deviation is present, with the QRS axis exceeding + 100°;
- sometimes a small q wave precedes the tall R wave in lead V1 (qR pattern);
- Negative T wave in V1 in the presence of R >5 mm
- the presence of a complete or incomplete right bundle branch block (RBBB) pattern

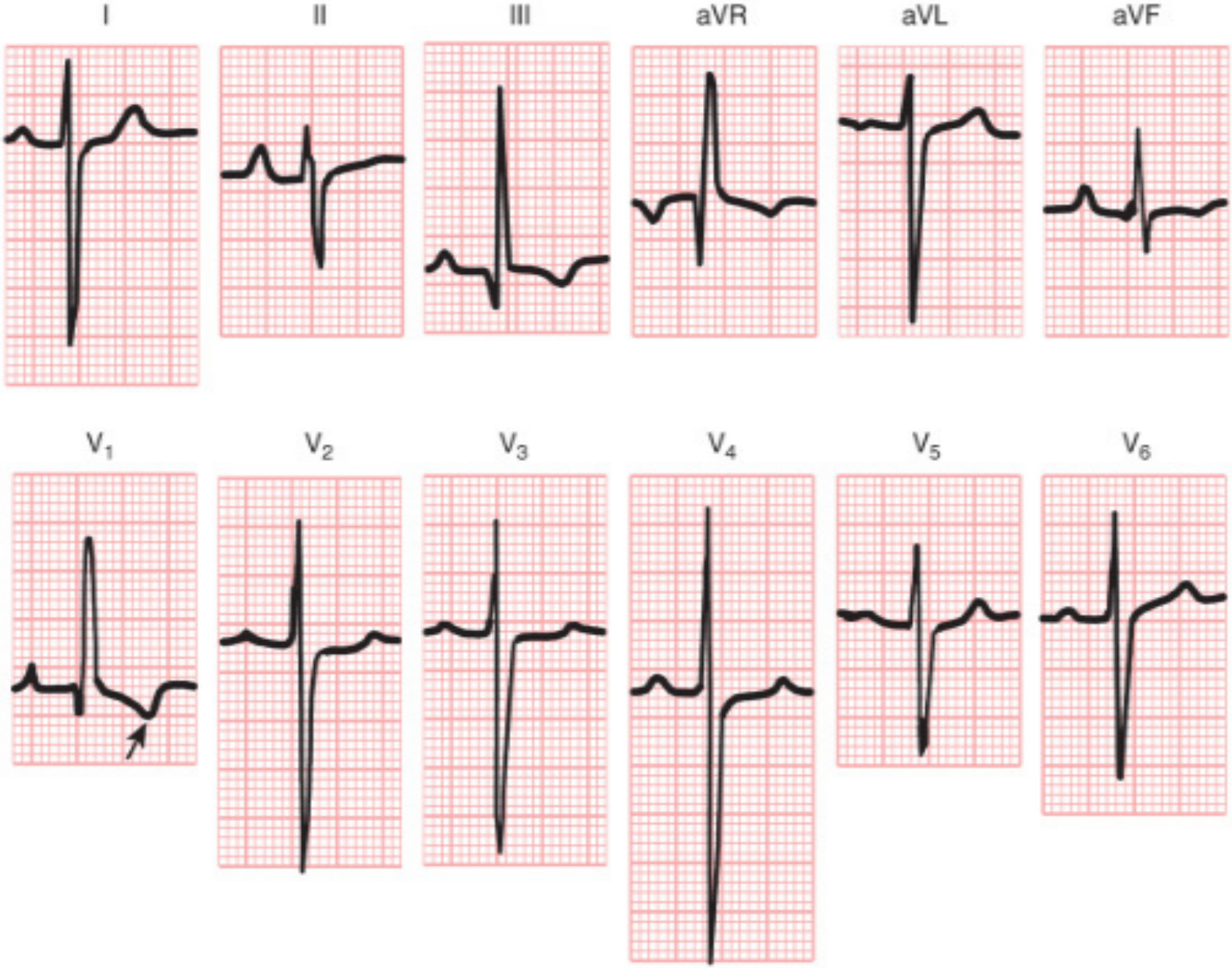
Causes: chronic obstructive pulmonary disease, primary or secondary pulmonary hypertension, mitral stenosis, mitral regurgitation, chronic LV failure, congenital heart disease, atrial septal defects

Right Ventricular Hypertrophy





Right Ventricular Hypertrophy



Left Ventricular Hypertrophy

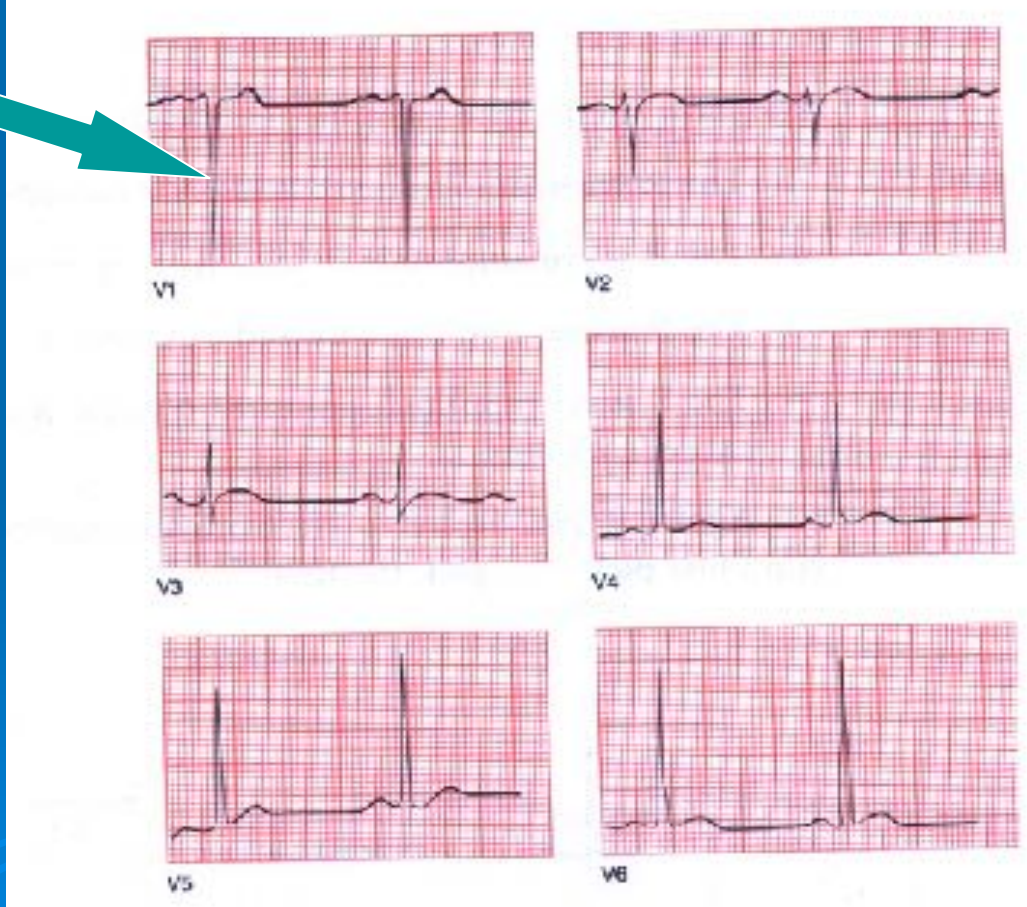
- SV1 (or SV2) + RV5 (or RV6) ≥35 mm (Sokolow/Lyon index)
- the Cornell criteria: R in aVL + S in V3 ≥28 mm for males and R in aVL + S in V3 ≥20 mm for females
- S wave in aVR >14 mm
- R wave in aVF >20 mm
- R wave in V5 or V6 >2.6 mV
- Left axis deviation exceeding -15° is also often present
- Onset of the intrinsicoid deflection in V5 or V6 ≥0.05 second
- Largest R wave + largest S wave in the precordial leads> 45mm

Causes of LVH:

- hypertension,
- heart valve disorders such as aortic valve stenosis,
- congenital heart disease (coarctation of aorta, patent ductus arteriosus),
- hypertrophic cardiomyopathy,
- endocrine disorders.

Left Ventricular Hypertrophy

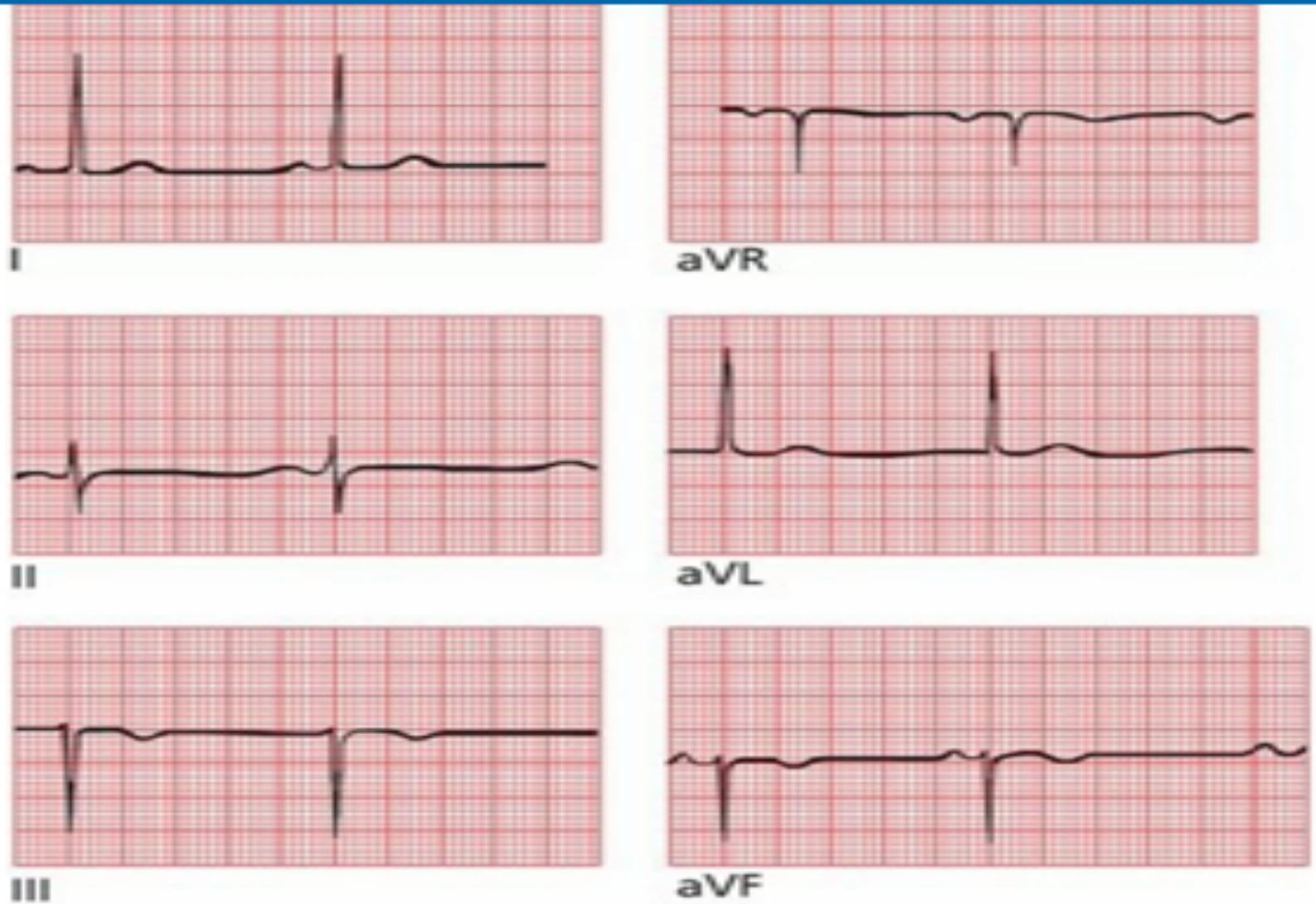
S wave



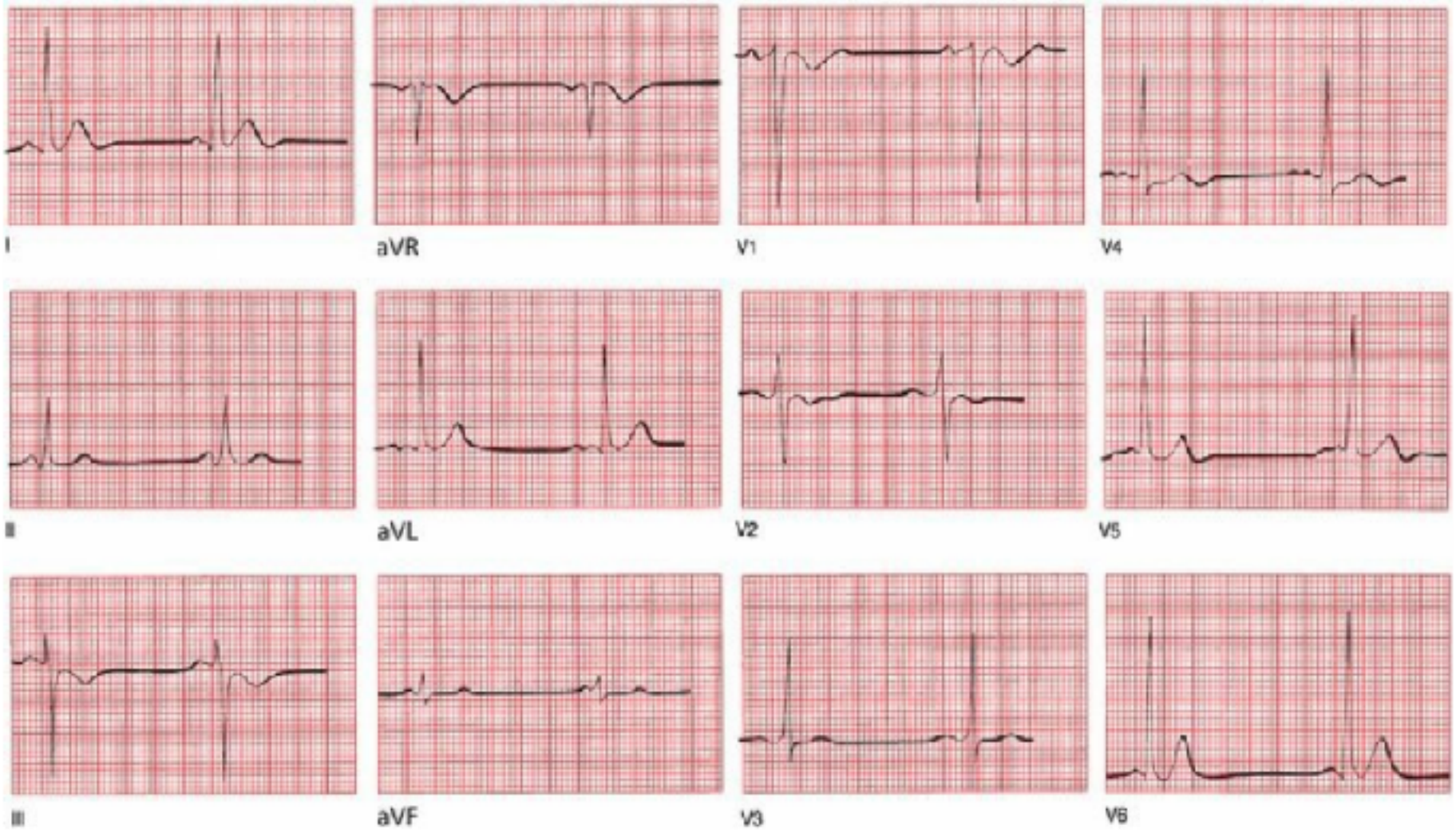
R wave amplitude in V5 or V6 + S wave in V1 or V2 > 35 mm (RV5-6 +SV1-2 > 35 mm)

R wave amplitude in V6 exceeds the R-wave amplitude in lead V5.

Left Ventricular Hypertrophy



Is there Ventricular Hypertrophy in the tracing below?
The patient is a 50-year-old female.

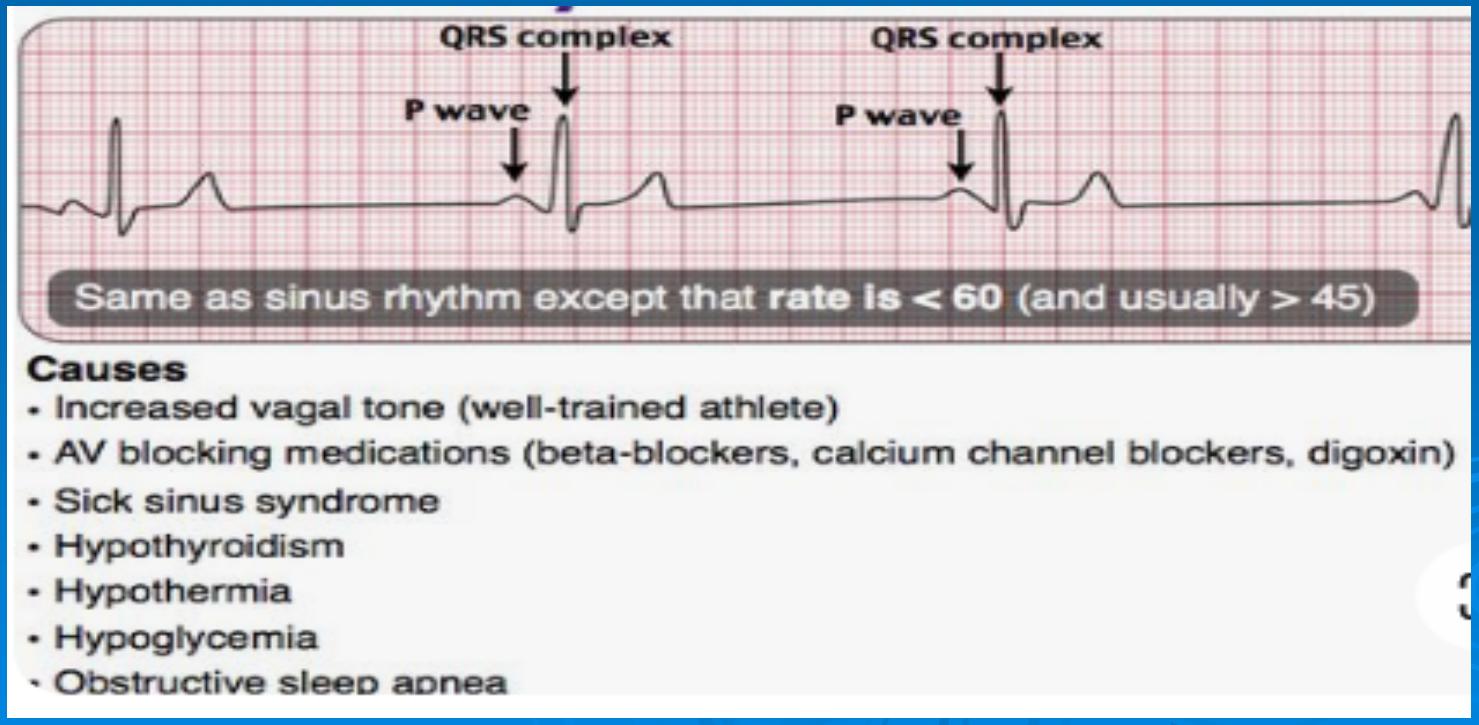


Automacity disorders

1. Sinus bradycardia
2. Sinus tachycardia
3. Sinus arrhythmia
4. Sinus node dysfunction (its historical name sick sinus syndrome)

Sinus bradycardia

- rhythm in which the rate of impulses arising from the sinoatrial (SA) node is lower than expected.
- a normal upright P wave in lead II — sinus P wave — preceding every QRS complex with a ventricular rate of less than 60 beats per minute.



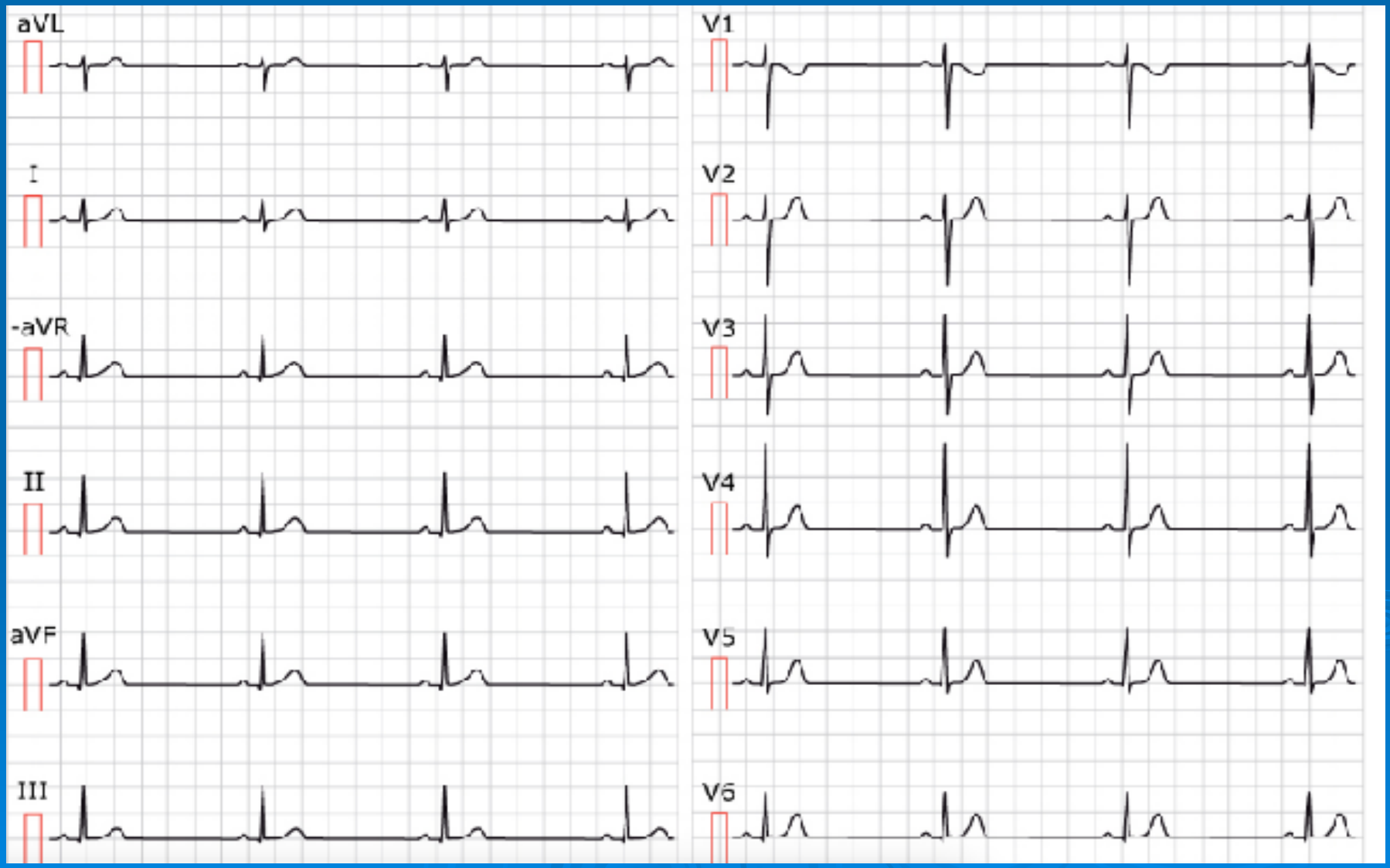
Normal (physiological) causes of sinus bradycardya

- During sleep
- Well- trained individuals display SB due to high vagal tone
- During vagal syncope (intense emotional stress)
- During vagal maneuvres
- It' not uncommon to discover in healthy young individuals who are not well-trained, this is a normal finding.

Symptoms of Bradycardia

- Lightheadedness or dizziness (especially with exertion)
- Easy fatiguability
- Syncope (fainting) or near-syncope
- Dyspnea (shortness of breath)
- Chest pain or discomfort
- Confusion

Sinus bradycardia



Sinus tachycardia

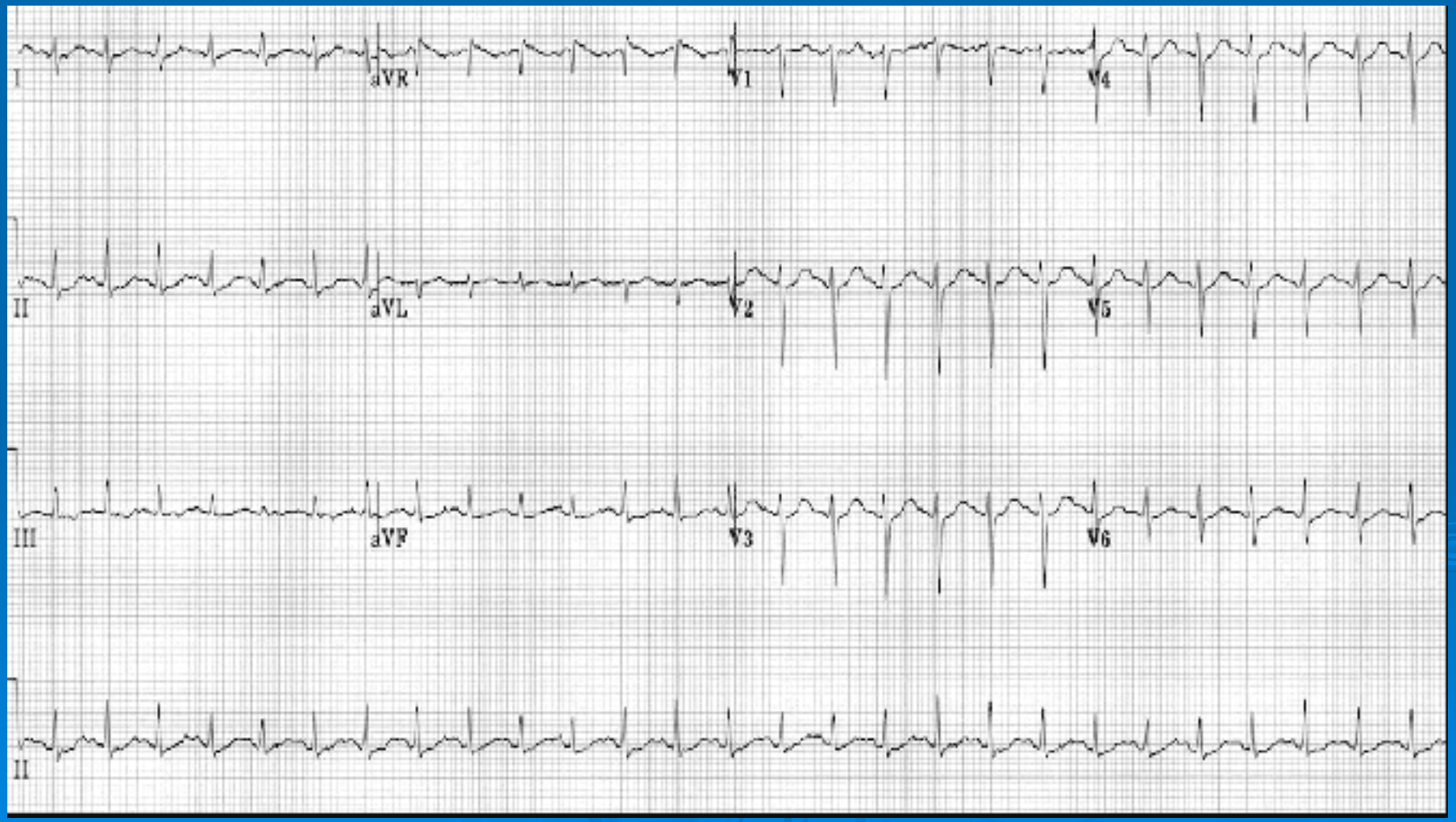
- The occurrence of sinus node discharge at a rate exceeding 100 beats/min constitutes sinus tachycardia.
- Each sinus P wave is followed by a QRS complex, indicating sinus rhythm with 1:1 AV conduction.
- P wave amplitude often increases and PR interval often shortens with increasing heart rate (during exercise).

Sinus tachycardia

Sinus tachycardia is usually a response to normal physiological situations, such as exercise and an increased sympathetic tone with increased catecholamine release—stress, fright, flight, anger:

- Pain
- Fever
- Anxiety
- Dehydration
- Anemia
- Heart failure
- Hyperthyroidism
- Mercury poisoning
- Pheochromocytoma
- Sepsis
- Pulmonary embolism
- Acute coronary ischemia and myocardial infarction
- Chronic pulmonary disease
- Hypoxia
- Intake of stimulants such as caffeine, nicotine, cocaine

Sinus tachycardia



Sinus arrhythmias

- is an irregularity of the sinus rhythm defined as a variation in the P-P interval by 0.16 sec (160 msec) or more in the presence of normal P waves.

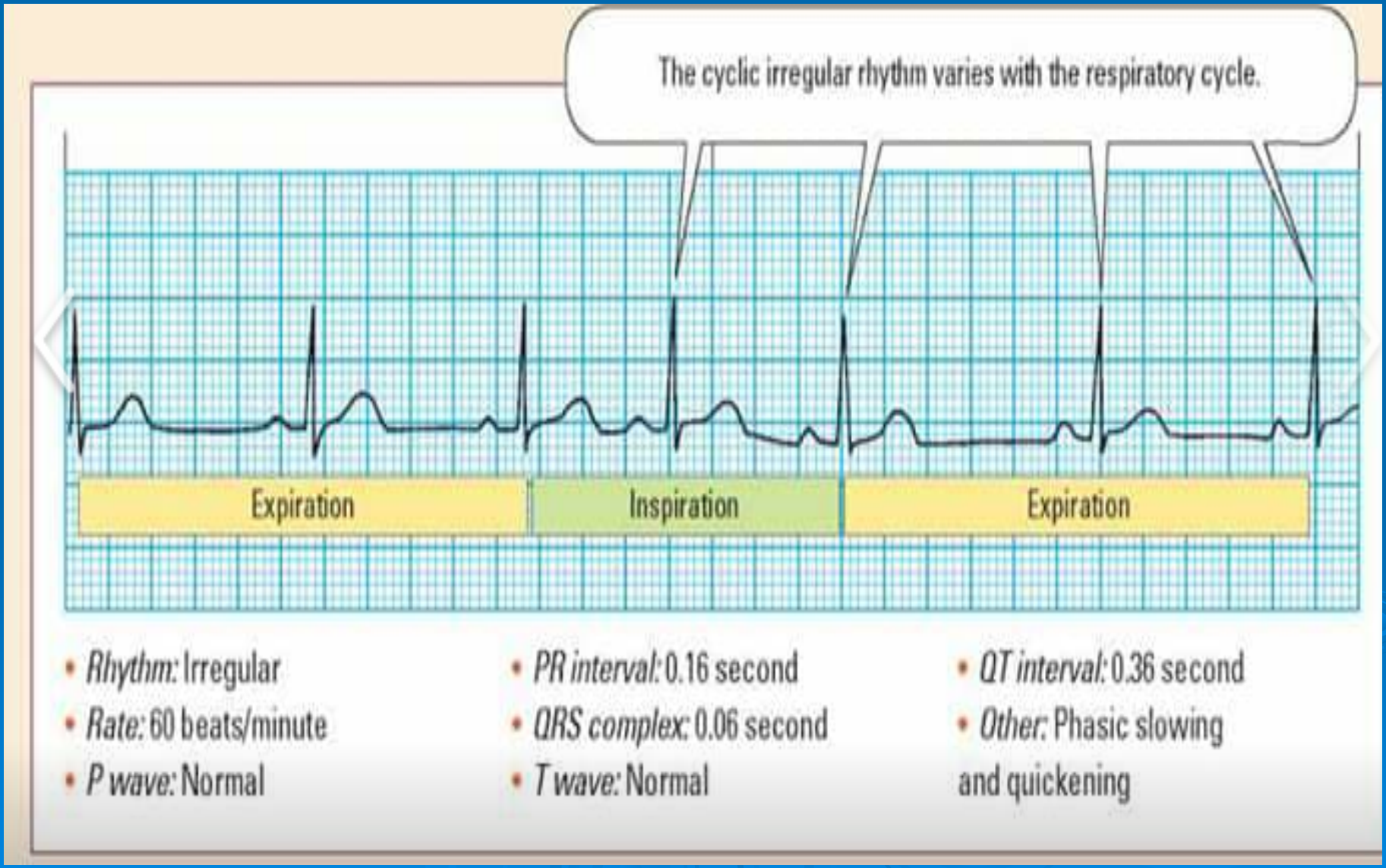
Classification

- Respiratory sinus arrhythmia
- **Non-respiratory Sinus Arrhythmia**
- **Ventriculophasic Sinus Arrhythmia**

Respiratory sinus arrhythmia

- the variation in heart rate is related to the respiratory cycle.
- the sinus rate increases gradually during inspiration and decreases with expiration.
- the variation is attributed to changes in vagal tone as a result of reflex mechanisms arising from the pulmonary and systemic vascular systems during respiration
- It is a normal variant that is most present in young people

Respiratory sinus arrhythmia



- *Rhythm:* Irregular
- *Rate:* 60 beats/minute
- *P wave:* Normal
- *PR interval:* 0.16 second
- *QRS complex:* 0.06 second
- *T wave:* Normal
- *QT interval:* 0.36 second
- *Other:* Phasic slowing and quickening

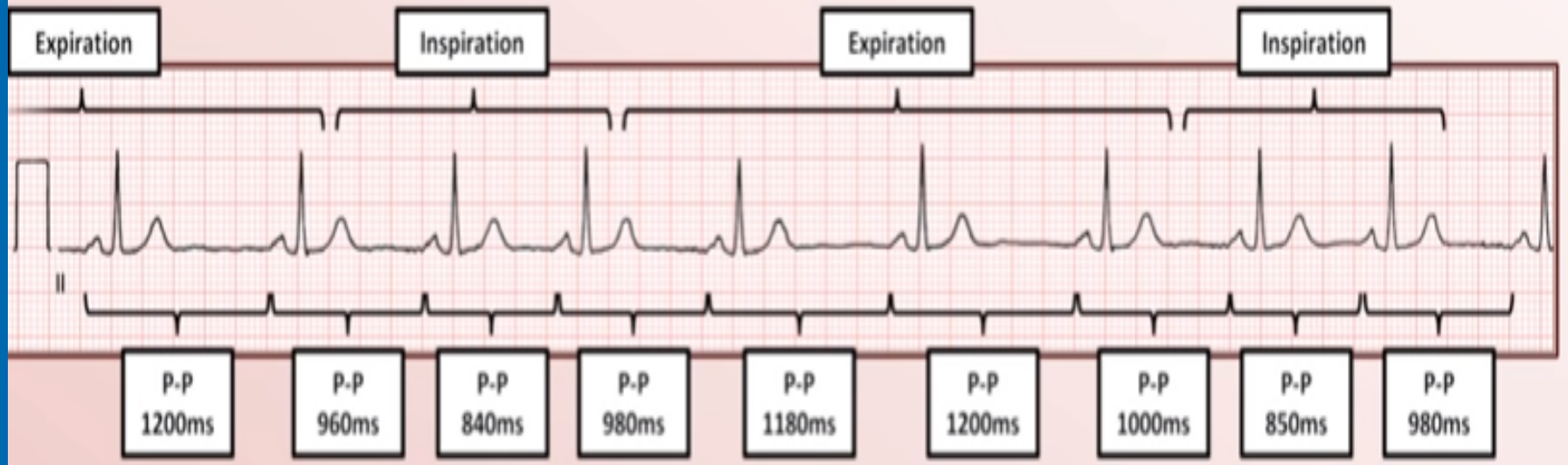
Non-respiratory Sinus Arrhythmia

- In non-respiratory sinus arrhythmia, the variation in the P-P interval is unrelated to the respiratory cycle.
- It can occur in the normal heart; however, it is more common among elderly with heart disease.

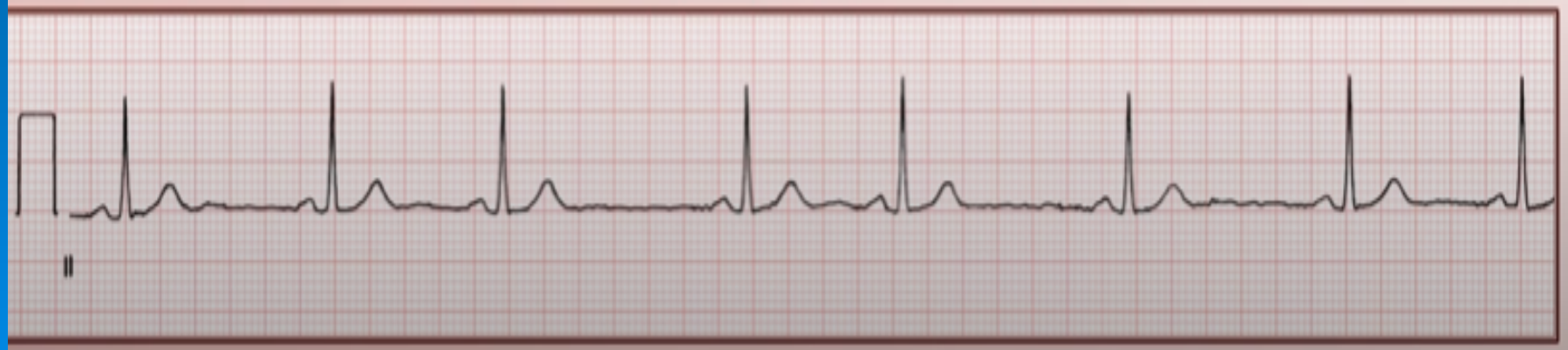
The cause of non-respiratory sinus arrhythmia is usually unknown, known causes include:

- Side effect of medications (digitalis, morphine)
- High intracranial pressure
- Inferior myocardial infarction
- Recovery from illnesses

Respiratory Sinus Arrhythmia



Non-Respiratory Sinus Arrhythmia



Sick sinus syndrome (SSS)

- also known as sinus node dysfunction (SND), is a disorder of the sinoatrial (SA) node caused by impaired pacemaker function and impulse transmission producing a constellation of abnormal rhythms.

This disease has different electrocardiographic presentations, such as:

- Periods of inappropriate and often severe sinus bradycardia.
- Sinus pauses, sinus arrests and sinus exits blocks that can happen with and without appropriate escape rhythm.
- Alternating tachycardia and bradycardia, referred to as a tachy-brady syndrome, which could also be associated with other supraventricular tachycardias.
- Prolonged sinus node recovery time after atrial premature complex or atrial tachyarrhythmias
- Additional conduction system disease is often present, including AV block , and/or bundle branch block

The causes of SSS

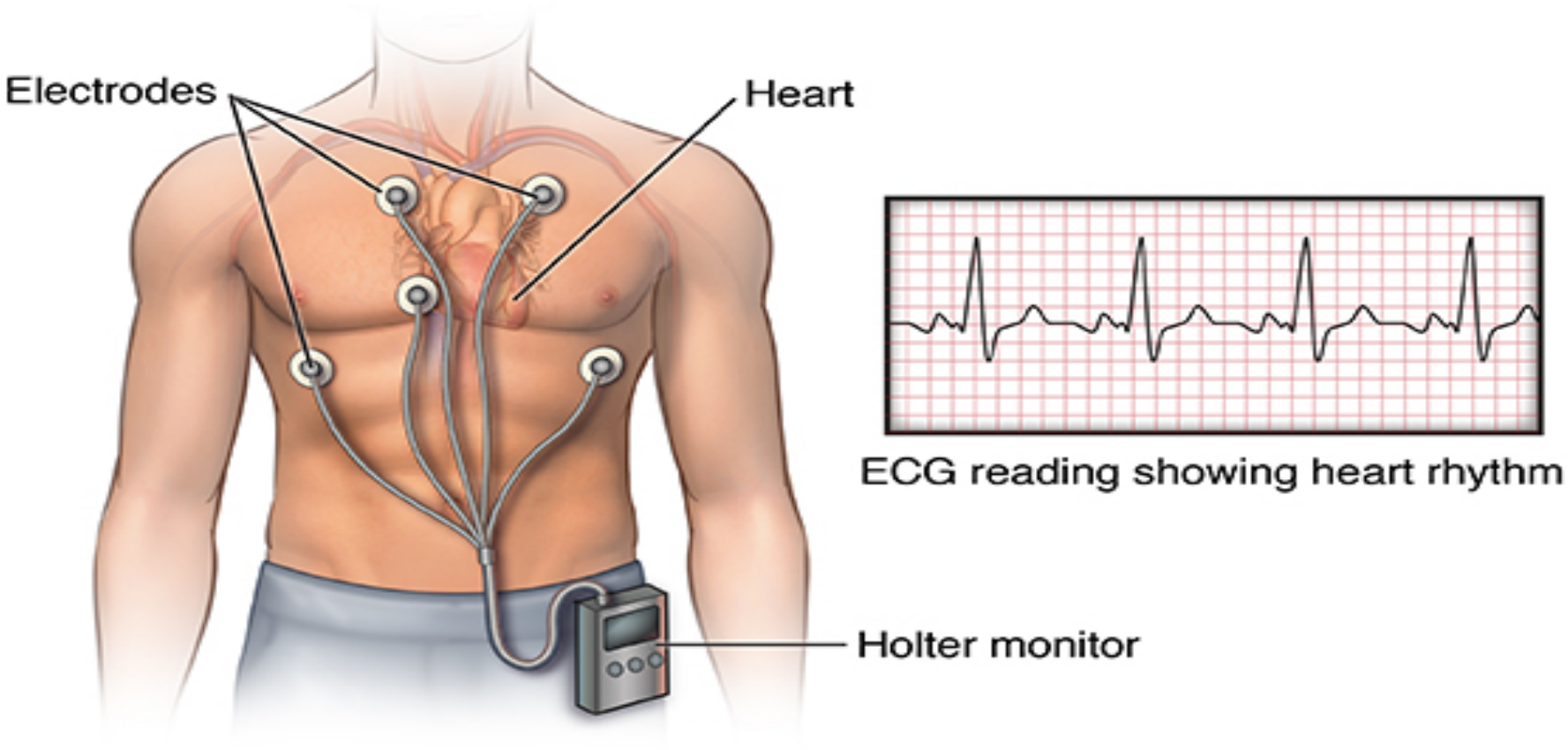
- idiopathic;
- ischemic heart disease;
- primary nonischemic cardiomyopathy;
- hypertensive heart disease;
- secondary cardiomyopathy including those resulting fromconnective tissue disease, syphilis, metastatic tumor, amyloidosis, myxedema;
- mitral valve prolapse;
- rheumatic heart disease;
- acute myocarditis;
- congenital heart disease

Symptoms of SSS

- fatigue
- dizziness
- presyncope
- syncope
- palpitations
- very slow pulse (bradycardia)
- difficulty breathing
- chest pain
- mental confusion
- memory problems
- disrupted sleep

The key to diagnosing sinus node dysfunction is to establish a correlation between the patient symptoms and the ECG findings at the time of symptoms.

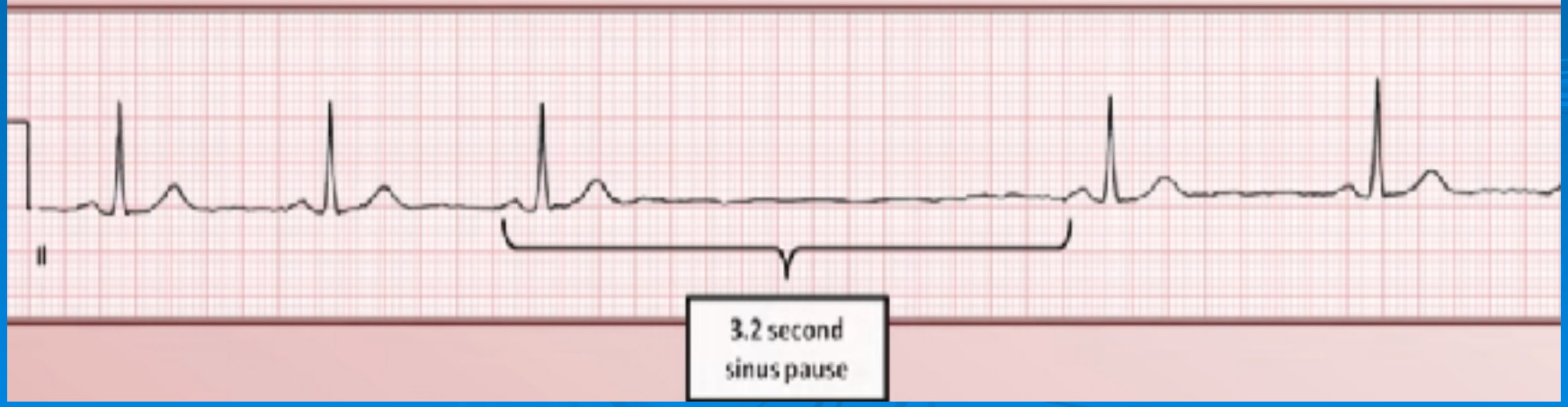
Holter monitor with EKG reading



ECG reading showing heart rhythm

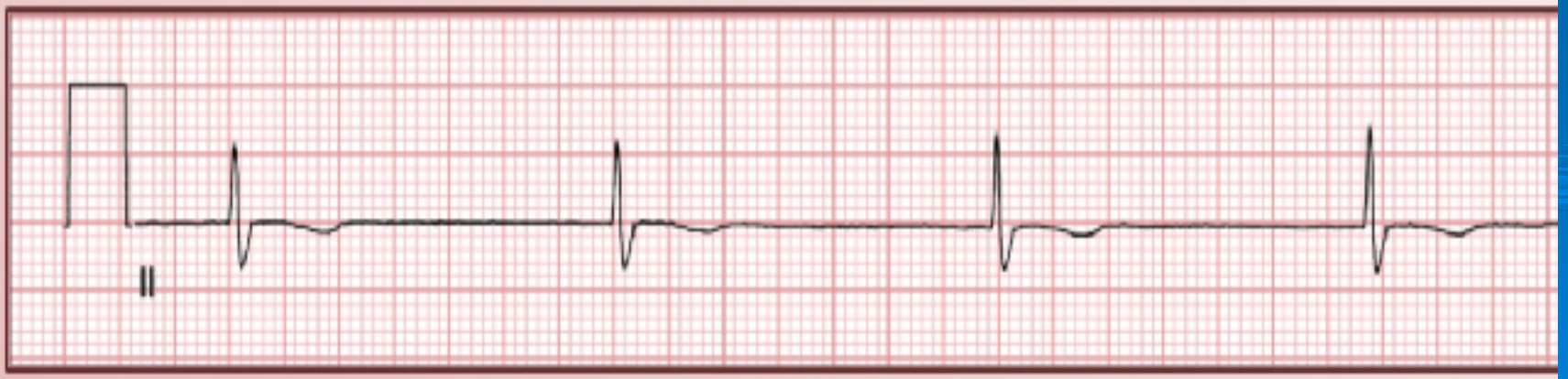
Sinus pause

- Sinus pause – A temporary interruption in sinus rhythm caused by failure of impulse generation within the SA node.
- Typically defined as being > 2-3 seconds.
- Must be distinguished from SA nodal exit block, if possible.



Sinus arrest

- Sinus arrest – A prolonged failure of impulse generation within the SA node.
- Will result in asystole if there is not an escape rhythm present.



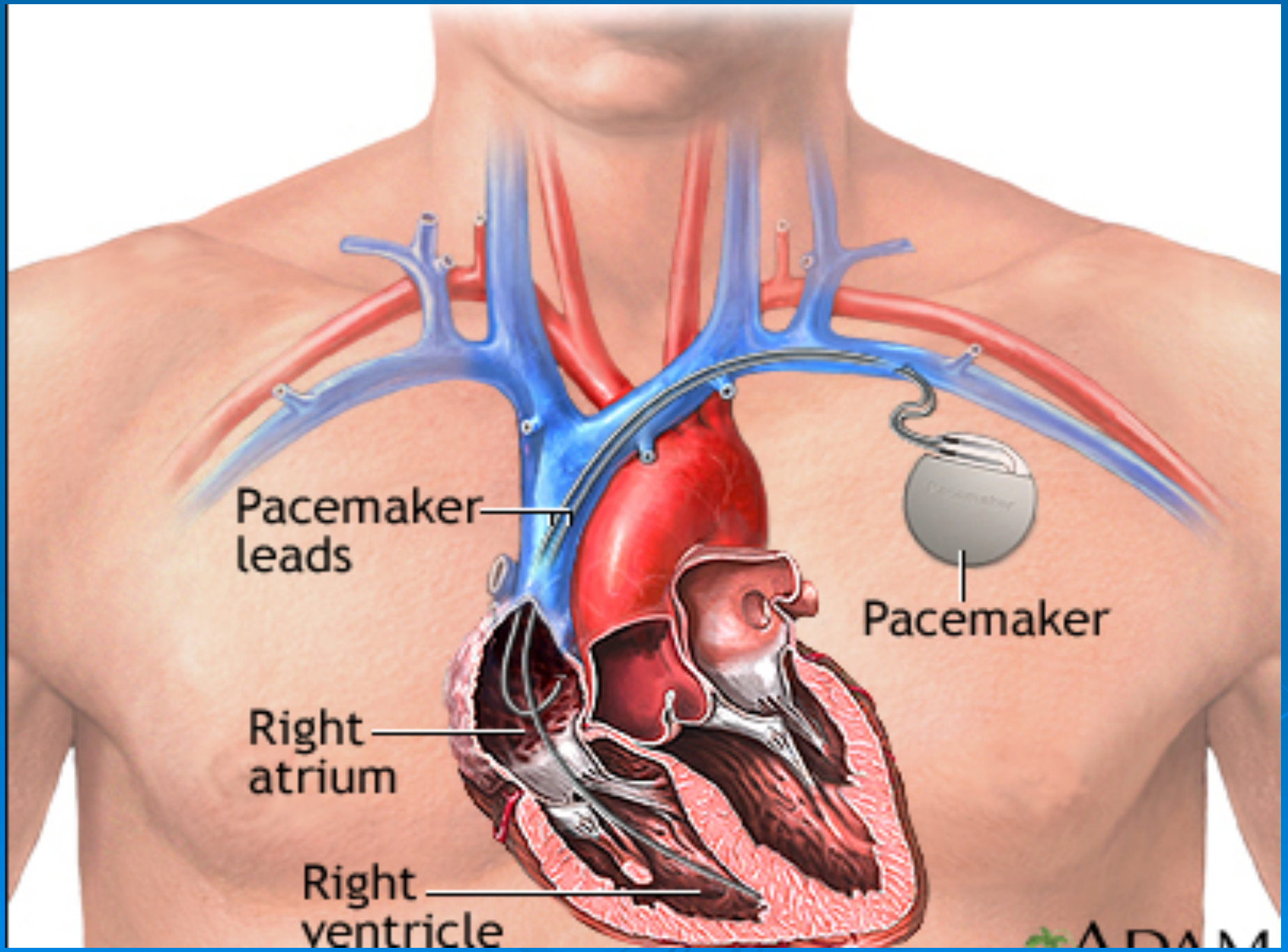
Sinus arrest with junctional escape rhythm at 43 bpm.

Tachy-brady syndrome

- is identified by bradycardia alternating with paroxysmal supraventricular arrhythmias, most frequently atrial fibrillation.

This results from abnormal automaticity and conduction within the atrial tissue.

A permanent pacemaker is indicated in symptomatic patients who have documented bradycardia responsible for their symptoms.


Pacemaker leads
Pacemaker
Right atrium
Right ventricle

THANK YOU