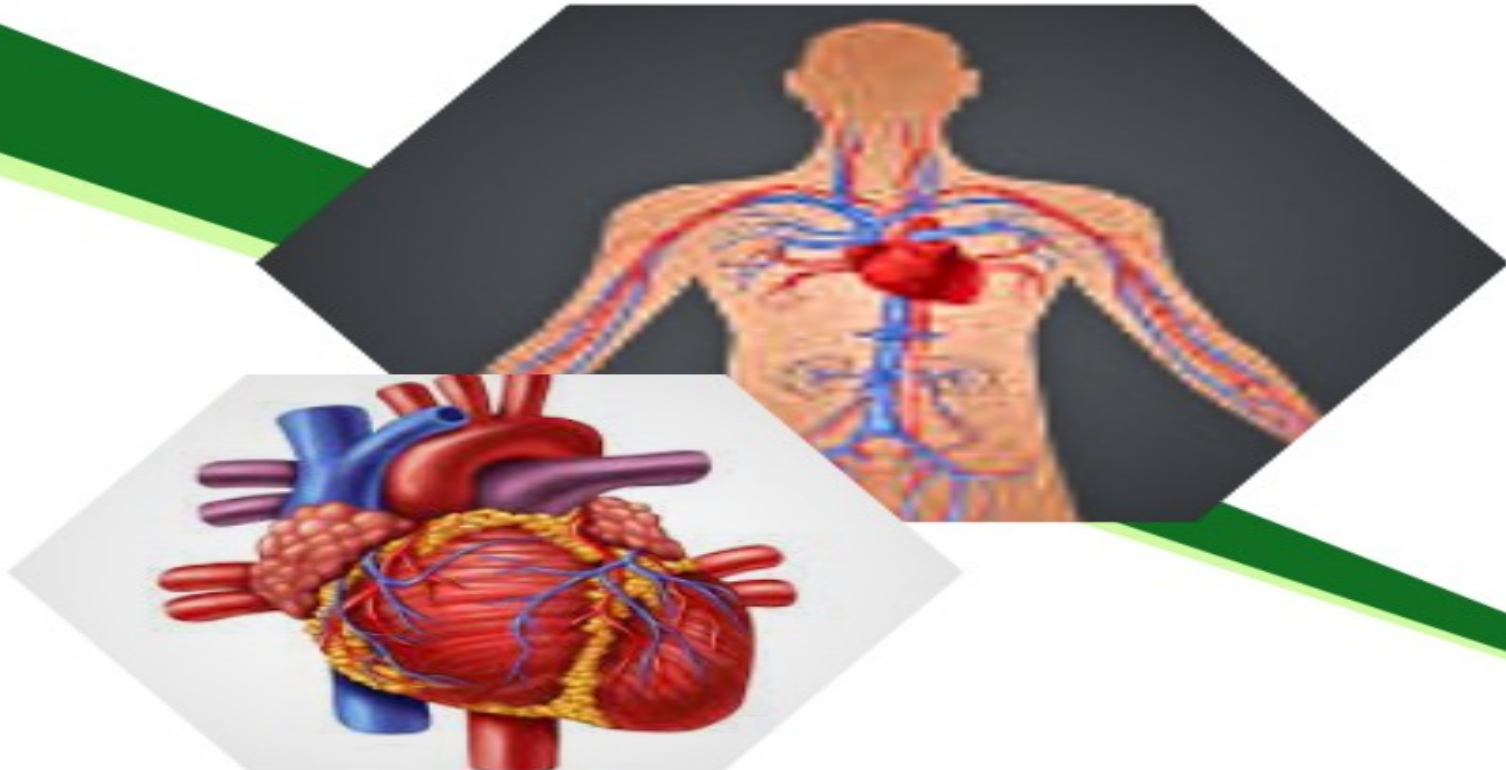


Doctor.021
no. 1

CVS PHYSIOLOGY



Writer: Zaid Alsamarat

Corrector: Mohammad Alsa'ed

Doctor: Yanal Shafagoj



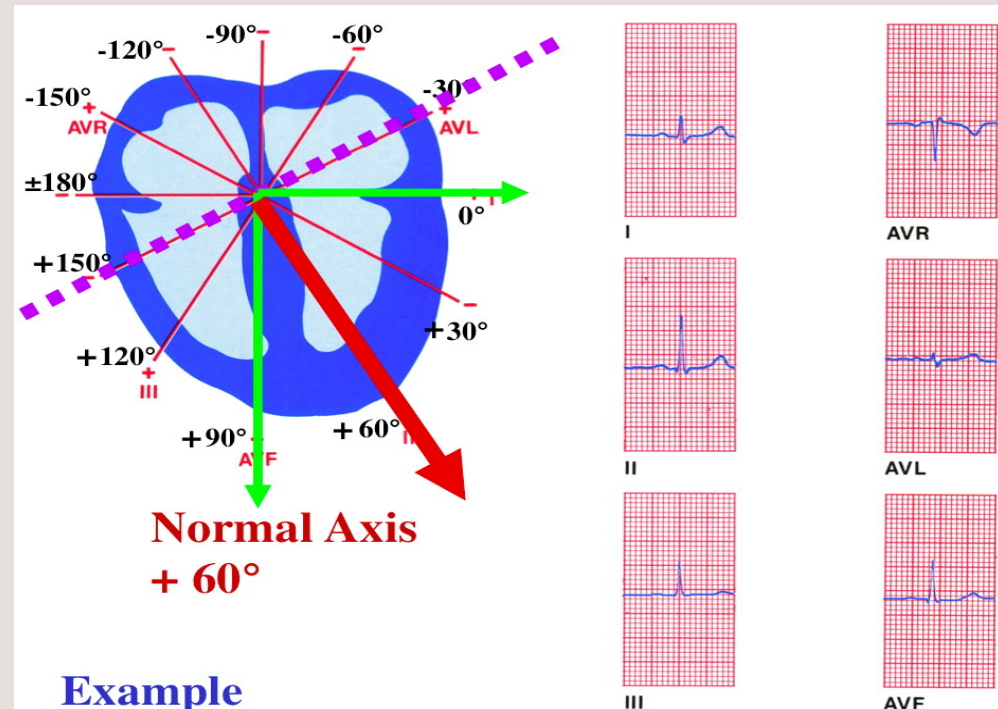
In this lecture the doctor continued discussing certain points about electrocardiograms, how different leads are related to each other, and how such relations can be utilized to solve real life scenarios and find out what abnormalities are present in ECGs.

- First of all, why is it important to understand the polarities of each limb lead?
- Understanding the polarities of each limb lead is essential in order to understand how the mean QRS vector RELATES and RECORDS on the different leads.

For example:

The red arrow resembles the mean QRS vector, the tip of the arrow is where most of the + charge is located, as you can see, this mean vector is moving closer to the + tip of some leads, while moving away from the + tip of other leads, how does this influence what is recorded on each lead?

When the mean QRS vector "moves" closer to the + tip of a lead, the recorded "net deflection" in that lead will be +, and vice versa.



Example

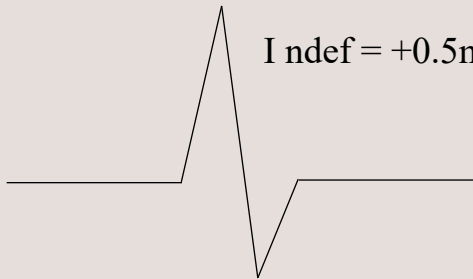
The net deflection in a lead = the amplitude of the positive waves - the amplitude of the negative waves, in most recordings this is done by subtracting the S wave from the R wave.

By finding the net deflection of a certain ECG recording, one can tell how the mean vector relates to the lead in use, if the net deflection is + this means that the mean current vector is moving towards the + tip of the lead, if the net deflection is - this means that the mean current vector is moving away from the + tip of the lead, if the net deflection is 0 this means the the mean current vector is PERPENDICULAR on the lead axis, here is an example, let's say you got these readings on leads III, II and I:

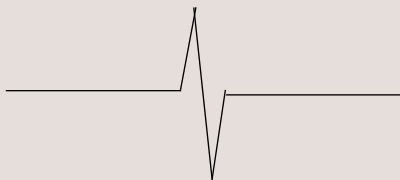
III ndef= -0.5mv



I ndef = +0.5mv



II ndef = 0mv



As you can see, the net deflection in lead I is +, but - in lead III, this means that the mean QRS vector is moving towards the + tip of lead, but away for the + tip of lead III (that is, towards the - tip of lead III).

By doing this, we know that the mean vector is anywhere between angle 0 and angle -60.

The closer the mean vector is to angle 0, the more + lead I net deflection becomes, and the more - that of lead III.

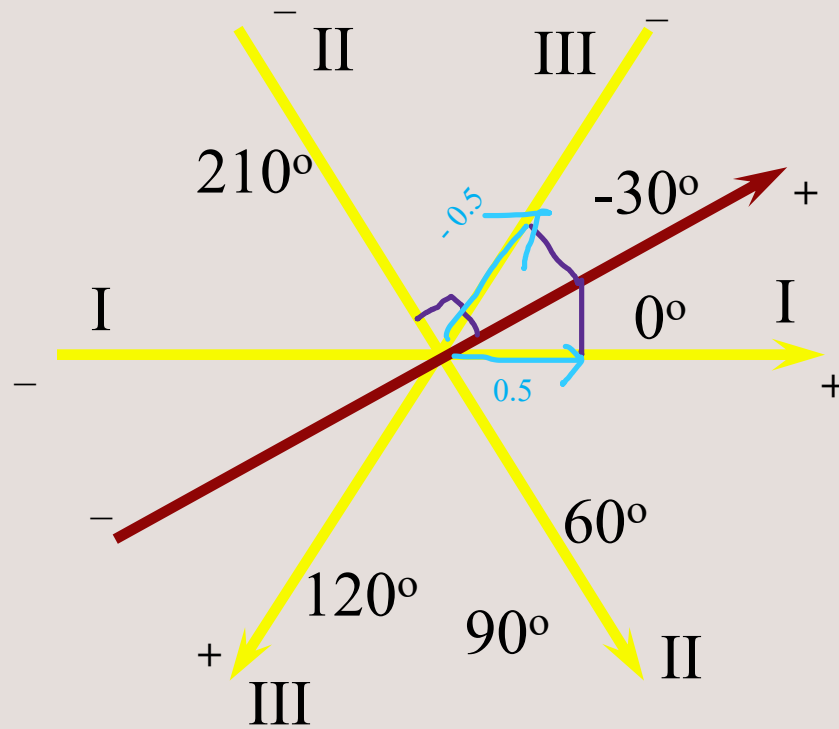
If the mean vector has an angle of -30, we are halfway between leads I and III, and at this point:

Net deflection of lead I= - net deflection of lead III.

At this point too, net deflection of lead II will = 0, because at angle -30 (and 150) the mean vector is PERPENDICULAR TO THE AXIS OF LEAD II.

By combining this info, we know that the mean QRS vector has an angle of -30, and thus is deviated to the left.

Axes of the Three Bipolar and Augmented Leads



Hexaxial diagram is in the frontal plane. Normal *QRS* axis from -30 to $+90$. Lt axis deviation from -30 to -90 . Rt axis deviation from $+90$ to $+180$. Extreme right axis deviation from $+180$ to -90 .

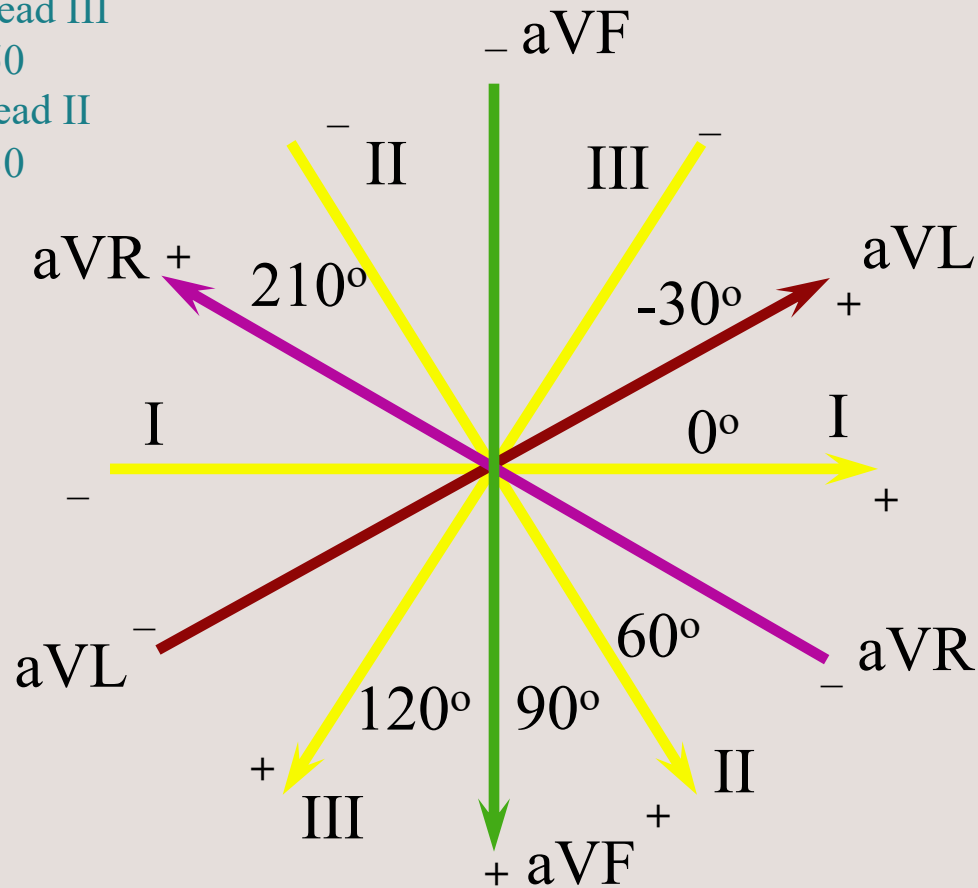
Axes of the Three Bipolar and Augmented Leads

Note that

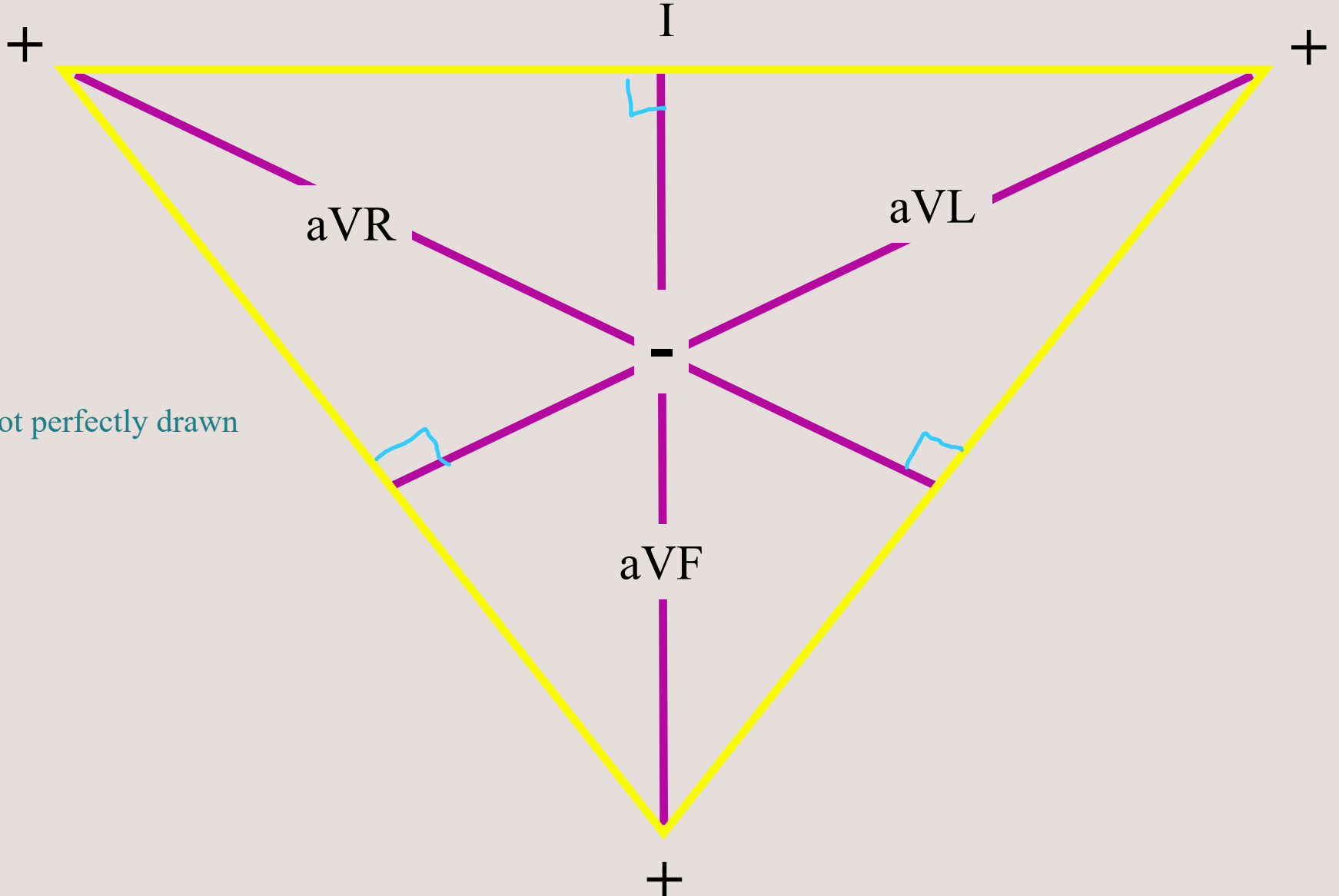
aVF is perpendicular on lead I through angles 90 and -90 .

aVR is perpendicular on lead III through angles 30 and -150

aVL is perpendicular on lead II through angles 150 and -30



Axes of the Unipolar Limb Leads



Let's try to determine the direction of the mean QRS vector from this ECG:

1- look for the isoelectric limb lead (lead with a net def = 0), here it is lead III.

2- what lead is perpendicular to lead III? It is the aVR lead.

Since lead III has a net def = 0, we know that the mean QRS vector has an axis that is very similar to that of lead aVR, however lead aVR is perpendicular on lead III through angles 30 and -150, so which angle is the angle of the mean QRS vector closer too?

3- check lead aVR, it has a net negative deflection! Thus the mean QRS vector is moving away from the + tip of lead aVR, and it's angle is around or equal to +30.



**SEVERE RIGHT
OR
LEFT
AXIS DEVIATION
OF QRS**
From 180 to 360 (-90)

**LEFT AXIS
DEVIATION OF
QRS**

Lead I
□ 180°

Lead I
0° +

**RIGHT AXIS
DEVIATION OF
QRS**
From +90 to + 180

**NORMAL MEAN
ELECTRICAL
AXIS OF QRS**
From 0 to +90

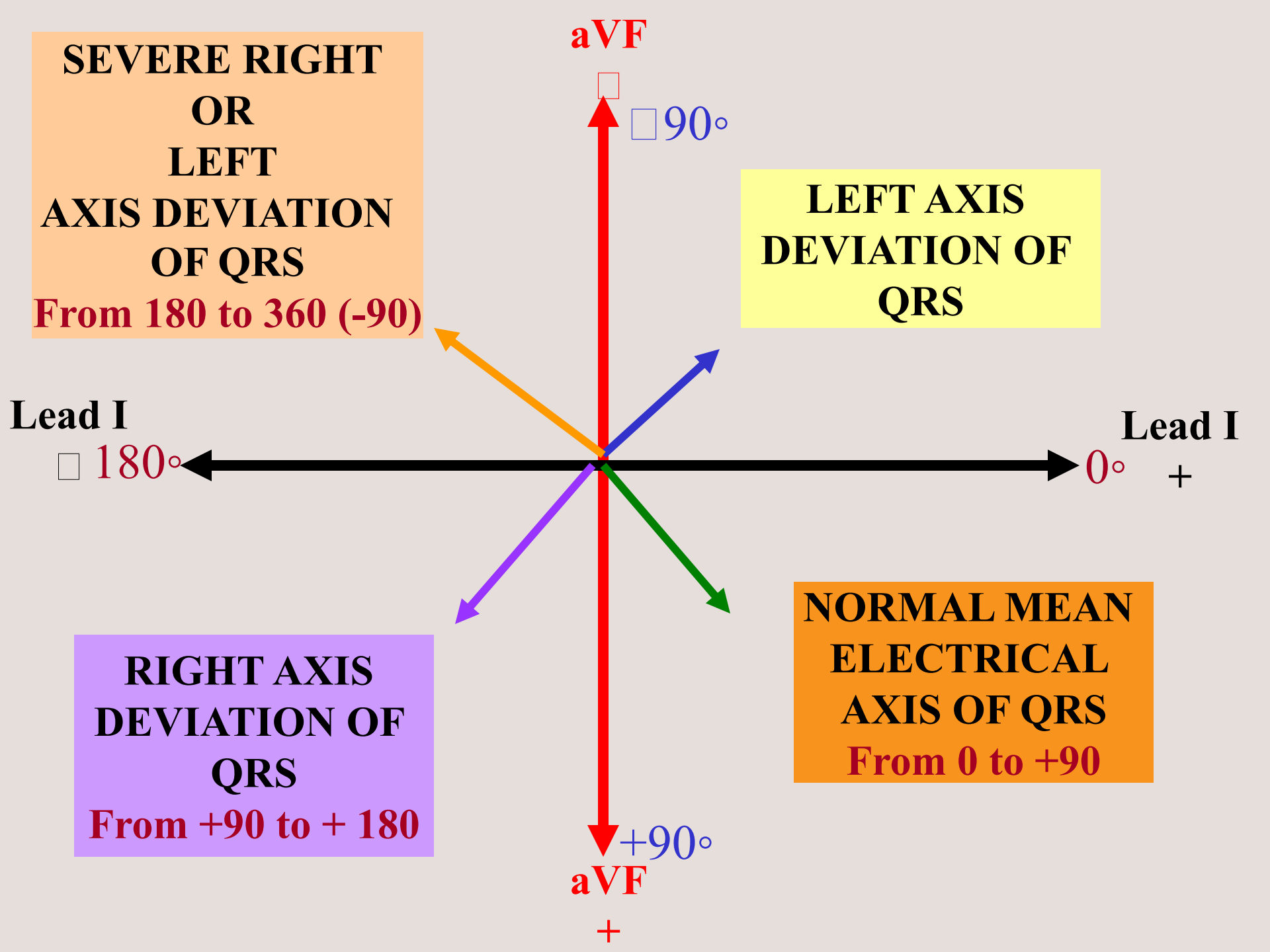
aVF

□ 90°

+90°

aVF

+



- Normal axis is the **left inferior quadrant**
- Look for the equiphasic lead (R wave=S wave)

<https://youtu.be/K0eCPwkxHFY>

- The quadrant method: aVF and lead I.
- Physiological left axis deviation from 0 to -30 Superior left quadrant.
- Pathological left axis deviation from -30 to -90 Superior left quadrant
- Pathological Right axis deviation from +90 to +180 Right **inferior quadrant**
- Superior Right quadrant: Extreme Rt axis deviation, or Rt superior axis deviation, or Rt shoulder axis deviation from -90 to -180

Causes of Cardiac Arrhythmias

- Abnormal rhythmicity of the pacemaker
- Shift of pacemaker from sinus node
- Blocks at different points in the transmission of the cardiac impulse
- Abnormal pathways of transmission in the heart
- Spontaneous generation of abnormal impulses from any part of the heart

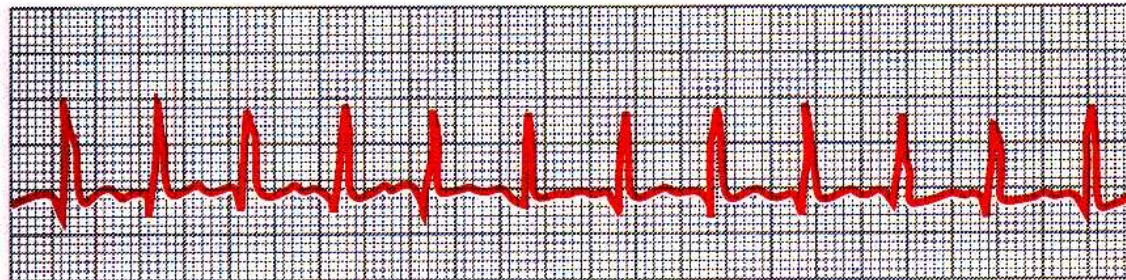
The lecture starts here, all the previous slides were discussed with the doctor for more clarification on ECGs

Abnormal Sinus Rhythms

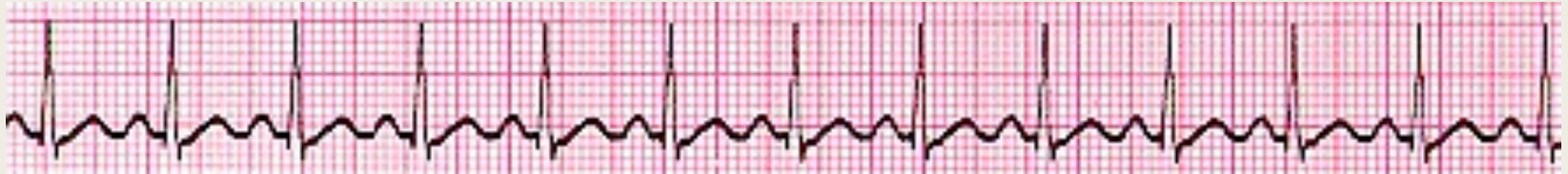
- Tachycardia means a fast heart rate usually greater than 100 beats /min.
- Caused by (1) increased body temperature, (2) sympathetic stimulation (such as from loss of blood and the reflex stimulation of the heart), and (3) toxic conditions of the heart

Other causes: thyrotoxicosis, allergic anaphylaxis (as a result of the sudden elevation of the catecholamines epinephrine and norepinephrine).

There's some correlation between heart rate and body temperature, every 1 Celsius increase in normal body temperature (approx. = 37 Celsius) is faced with an increase of 13 beats in the heart rate. For example if someone has a temperature of 40 Celsius, there heart rate would be increased with $3 * 13$ beats above normal, which is approximately equal to 115 beats/min, however there's a limit for this, as heart contractility may decrease at severely elevated temperatures and bradycardia occurs.



Sinus Tachycardia



- **Etiology:** SA node is depolarizing faster than normal, impulse is conducted normally (i.e. normal PR and QRS interval).
- Remember: sinus tachycardia is a response to physical or psychological stress, **not a primary arrhythmia.**

As you see here , the TP segment has gotten shorter, AKA faster heart rate, but the QRS and PR intervals are normal , that's because the impulse conduction after the SA node firing is still normal and unaffected.

Abnormal Sinus Rhythms (cont'd)

- Bradycardia means a slow heart rate usually less than 60 beats /min
- Present in athletes who have a large stroke volume
- Can be caused by vagal stimulation, one example of which is the carotid sinus syndrome: In these patients, the pressure receptors (baroreceptors) in the carotid sinus region of the carotid artery walls are excessively sensitive. Therefore, even mild external pressure on the neck elicits a strong baroreceptor reflex, causing intense vagal-acetylcholine effects on the heart, including extreme bradycardia. Sometimes this reflex is so powerful that it actually stops the heart for 5 to 10 seconds, leading to loss of consciousness (syncope).
- Physiological sinus arrhythmia during respiratory cycle: During inspiration vagus is inhibited leads to slight increase in H.R, while during expiration vagus is stimulated which leads to slight decrease in H.R.
- In D.M and autonomic neuropathy, this fluctuation does not occur.
- Pulsus paradoxus refers to an exaggerated fall in a patient's blood pressure during inspiration by greater than 10 mm Hg.



Sinus Bradycardia



- **Etiology:** SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval) rate is slower than 60/beats per minute.

In athletes who develop over time a physiological increase in stroke volume output (blood pumped from a single ventricle at the end of one systole), there's a physiological adaptation that lowers heart rate in order for the cardiac output (Q) to stay in the normal range (approx. 5000ml/min).

$$Q = SV \text{ (stroke volume)} * HR.$$

Bradycardia also occurs in sick sinus syndrome (SSS), as a side effect of some drugs (i.e. digoxin, Ca²⁺ channel blockers, beta blockers).

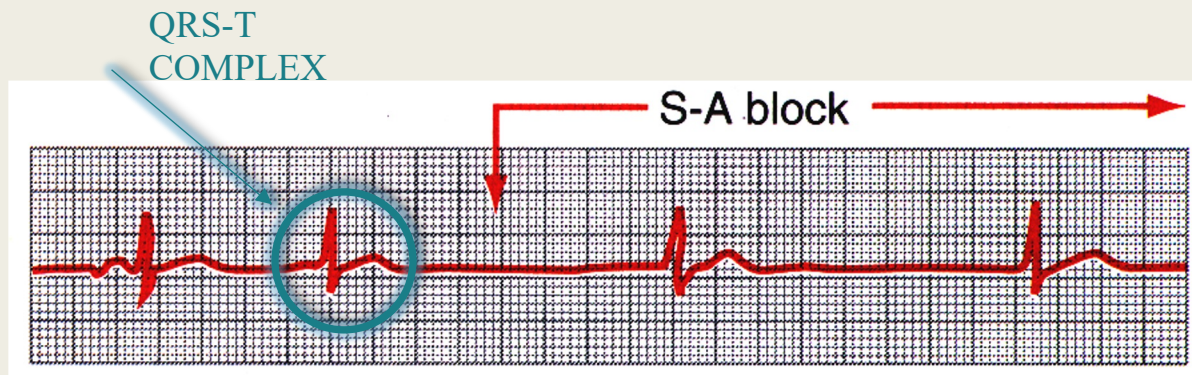
NOT AN ARRHYTHMIA.

Arrhythmias show an abnormal wave patterns (impulse conduction).

Examples include absent P wave, no QRS.

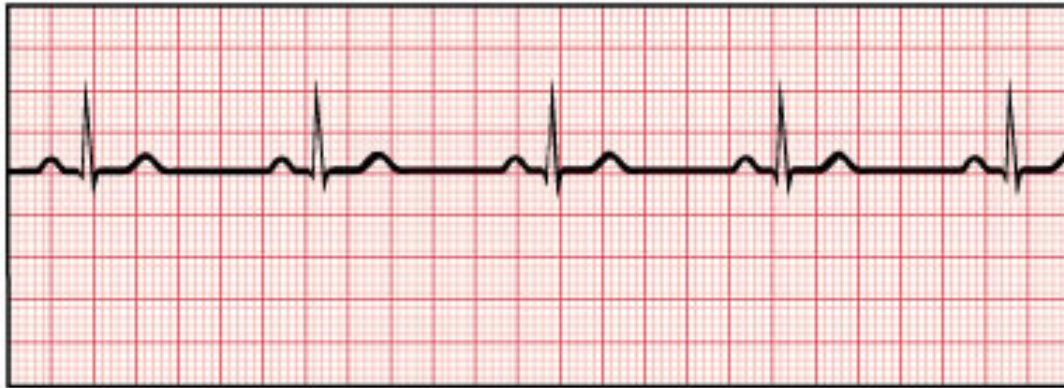
Sinoatrial Block

- ❖ In rare instances impulses from SA node are blocked.
- ❖ This causes cessation of P waves.
- ❖ New pacemaker is region of heart with the fastest discharge rate, usually the A-V node, so the rate of the ventricular QRS-T complex is slowed but not otherwise altered. (slower heart rate)

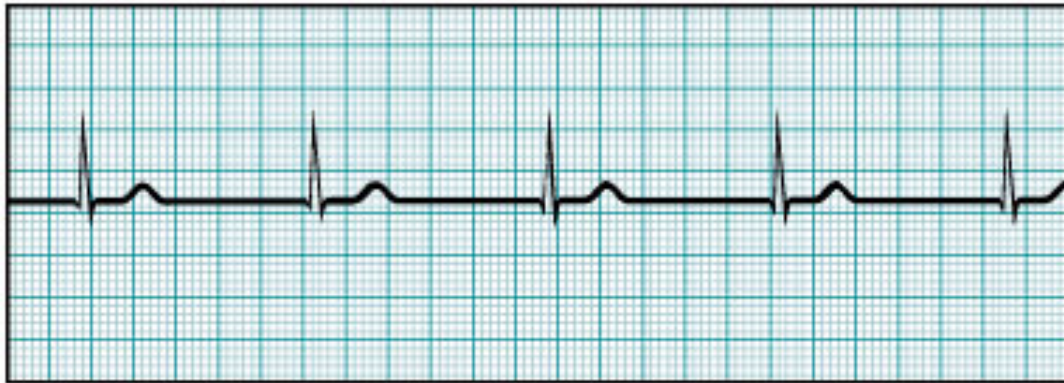


Note: no P waves and slow rate

ECGs, normal sinus rhythm vs SA block



(a) Sinus rhythm (normal)

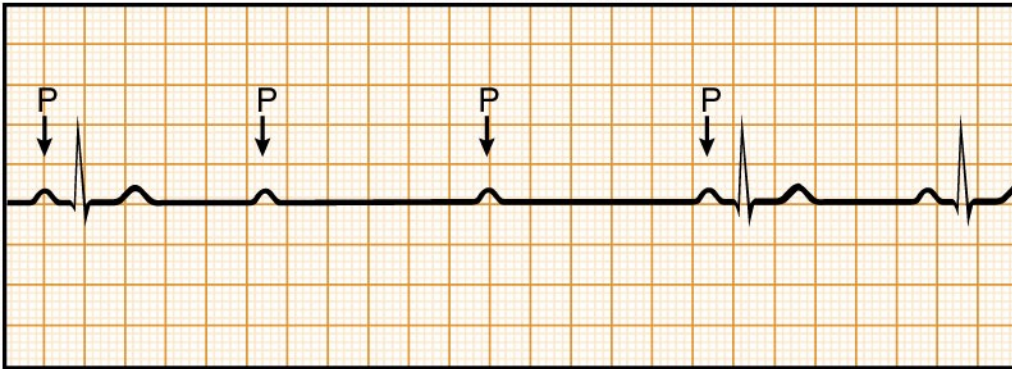


(b) Nodal rhythm – no SA node activity

When the SA node is nonfunctional for whatever reason, the AV node takes the pacemaker role, and thus the P wave stops appearing on the ECG because atrial depolarization is severely slowed and becomes late (and therefore hidden in the QRS complex), or atrial depolarization doesn't happen at all.

ECGs, Abnormal

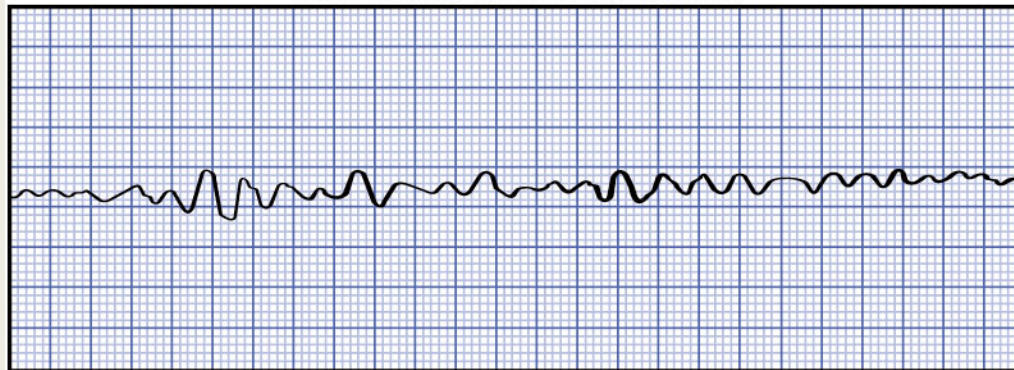
Copyright © The McGraw-Hill Companies, Inc. Permission required for reproduction or display.



(c) Heart block

Arrhythmia: conduction failure at AV node

Copyright © The McGraw-Hill Companies, Inc. Permission required for reproduction or display.



(e) Ventricular fibrillation

No pumping action occurs

Atrioventricular Block

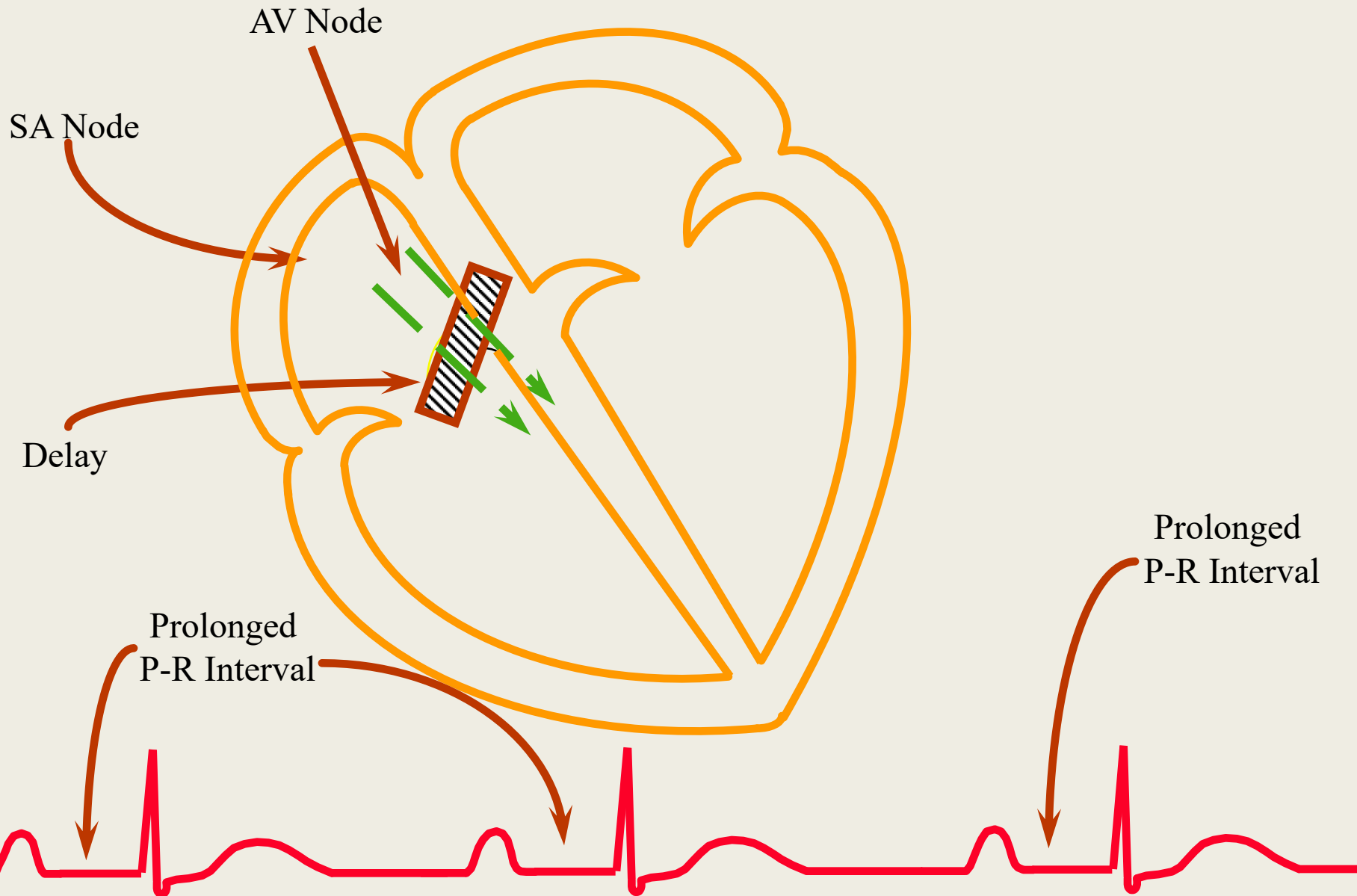
- ✓ **Impulses through A-V node and A-V bundle (bundle of His) are slowed down or blocked due to :**
 - ✓ *Ischemia of A-V nodal or A-V bundle fibers (can be caused by coronary ischemia)*
 - ✓ *Compression of A-V bundle (by scar tissue or calcified tissue)*
 - ✓ *A-V nodal or A-V bundle inflammation*
 - ✓ *Excessive vagal stimulation*
 - ✓ *Degeneration of the A-V conduction system, which is sometimes seen in older patients.*

Medications such as digitalis or beta-adrenergic antagonists can, in some cases, impair A-V conduction.

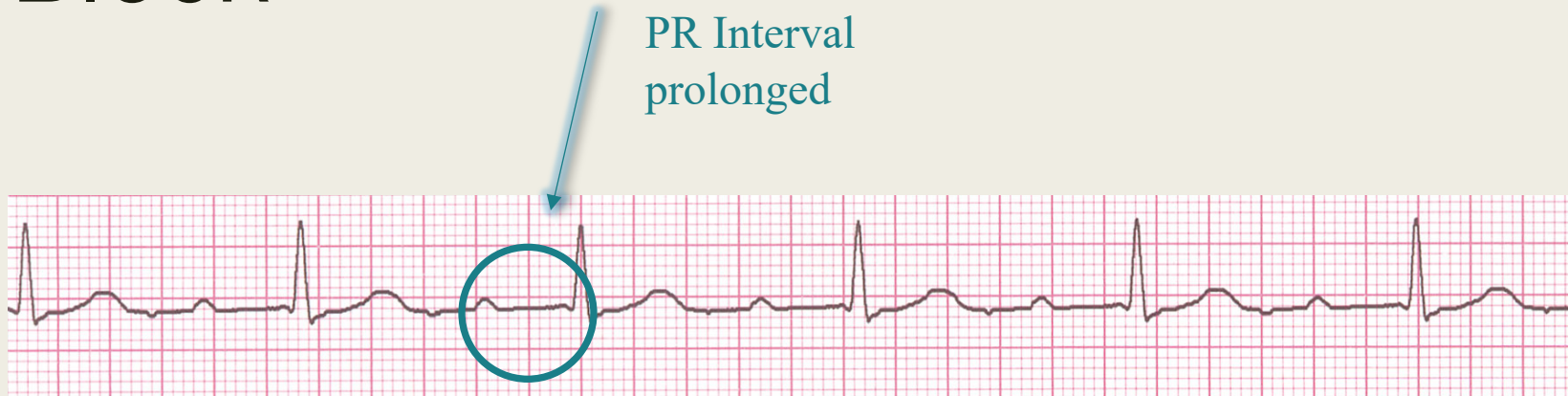
Incomplete Heart Block: First Degree Block

- ❖ Normal P-R interval is 0.16 sec
- ❖ If P-R interval is > 0.20 sec, first degree block is present (but P-R interval rarely increases above 0.35 to 0.45 sec, because, by that time, conduction through the A-V bundle is depressed so much that conduction stops entirely).

First Degree **incomplete** Heart Block



1st Degree **incomplete** AV Block



- **Etiology:** Prolonged conduction delay in the AV node or Bundle of His.
- Note how the PR prolongation is consistent and stable, that is, the PR interval stays constant even though it is prolonged.

Second Degree Incomplete Block

- P-R interval increases to 0.25 - 0.45 sec
- the action potential is sometimes strong enough to pass through the bundle into the ventricles, and sometimes not strong enough to do so. In this case, there will be an atrial P wave but no QRS-T wave, and it is said that there are “dropped beats” of the ventricles. This condition is called second-degree heart block.
- Atria beat faster than ventricles.
- There are two types of second-degree A-V block— Mobitz type I (also known as Wenckebach periodicity) and Mobitz type II.

Type 1 2nd degree AV block vs Type 2:

Type I block is characterized by progressive prolongation of the P-R interval until a ventricular beat is dropped and is then followed by resetting of the P-R interval (back to the original PR interval) and repeating of the abnormal cycle. A type I block is almost always caused by abnormality of the A-V node. In most cases, this type of block is benign, and no specific treatment is needed.

In type II block, there is usually a fixed number of non-conducted P waves (do not reach the ventricles) for every QRS complex, For example, a 2:1 block implies that there are two P waves for every QRS complex. At other times, rhythms of 3:2 or 3:1 may develop. In contrast to type I block, with type II block the P-R interval does not progressively prolong before the dropped beat; it remains fixed.

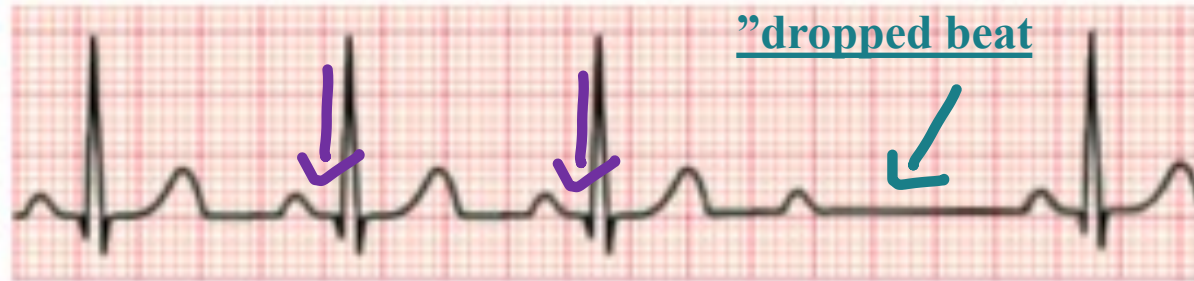
Type I 2nd degree AV block vs Type II 2nd degree AV block:

Mobitz I or Wenckebach Note how the PR interval is progressively becoming longer



PR interval is now reset back to the original duration

Mobitz II Note how the PR interval duration is constant until a QRS complex is dropped "dropped beat"

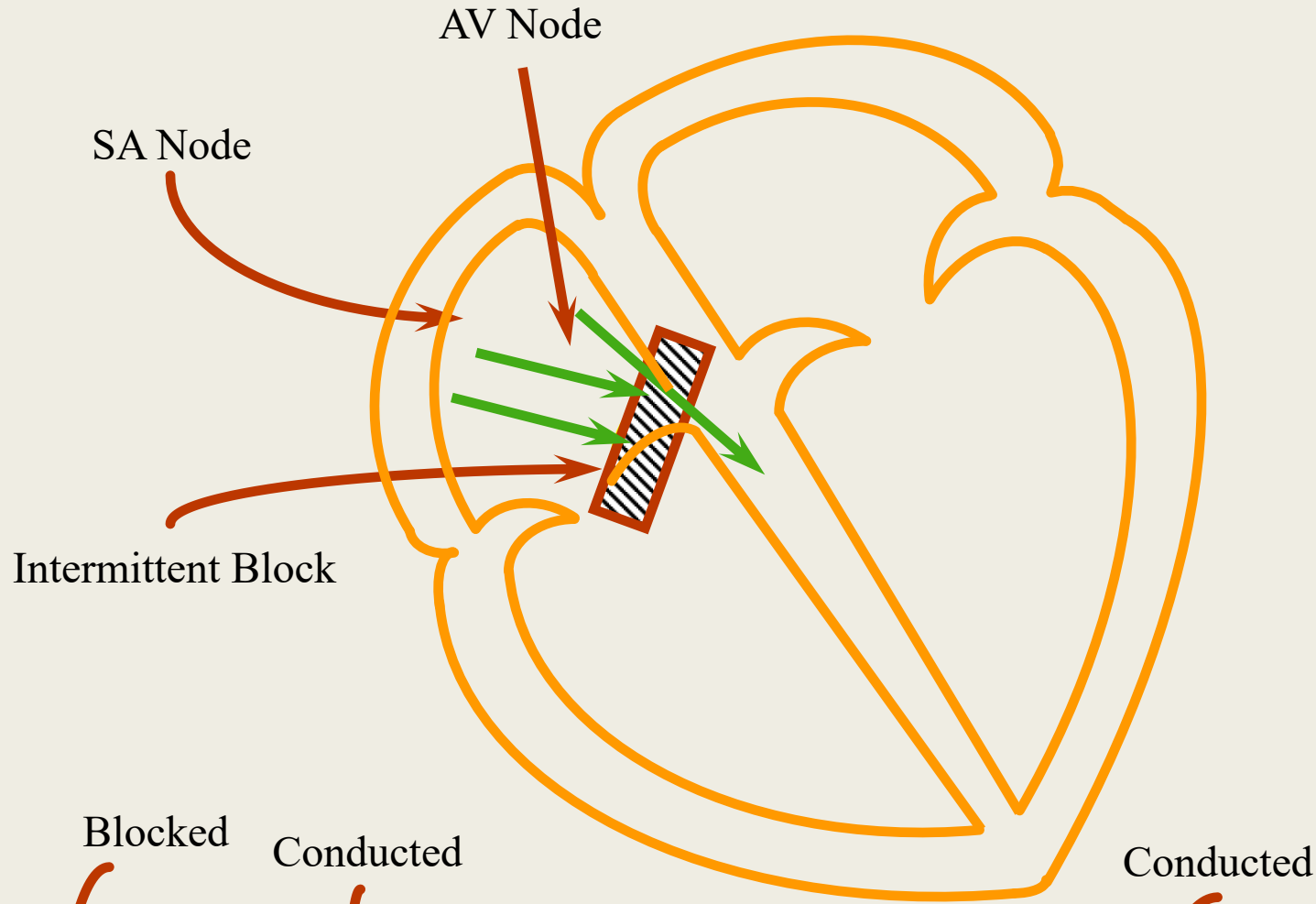


2:1 block



Note that in the 2nd ECG, the type II 2nd degree block starts at the green arrow, however the all the previous reading is shown to emphasize on how the PR interval is constant, the 3rd ecg (2:1 block) is just a continuation of the 2nd ecg after the type II 2nd degree block has started.

Second Degree Heart Block



Shown in the following ECG is a type II 2nd degree block, with 2:1 ratio (2 p waves for each QRS complex)

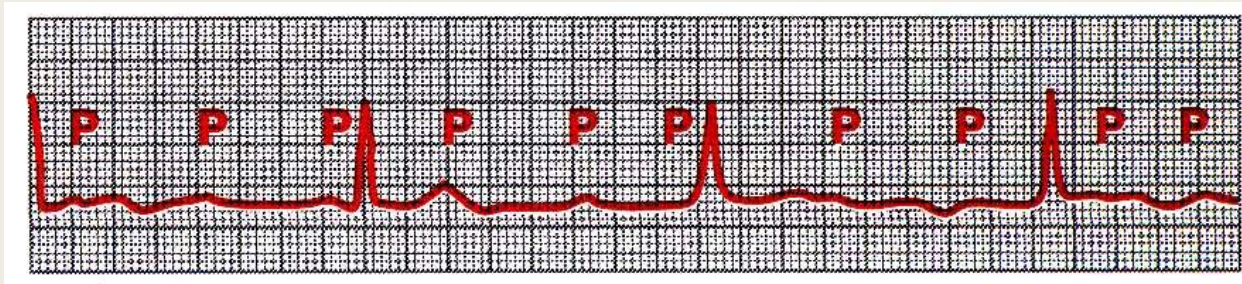
Type I 2nd Degree AV Block,



- **Etiology:** Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.

Third Degree Complete Block

- ☞ Total block through the A-V node or A-V bundle
- ☞ P waves are completely dissociated from QRST complexes
- ☞ Ventricles escape and A-V nodal rhythm ensues



HR = 37

3rd Degree AV Block



- **Etiology:** There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker beats at around 15 - 40 beats/minute.
- in this case, the ventricles spontaneously establish their own signal, usually originating in the A-V node or A-V bundle distal to the block.

Note that the rate of rhythm of the atria in this ECG is about 100 beats/min, whereas the rate of ventricular beat is less than 40 beats/min. Furthermore, there is no relationship between the rhythm of the P waves and that of the QRS-T complexes because the ventricles have “escaped” from control by the atria and are beating at their own natural rate, controlled most often by rhythmical signals generated distal to the A-V node or A-V bundle where the block occurs.



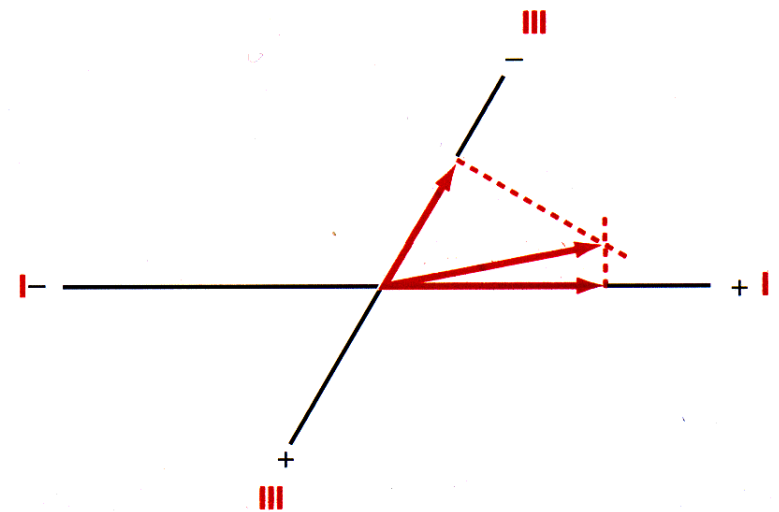
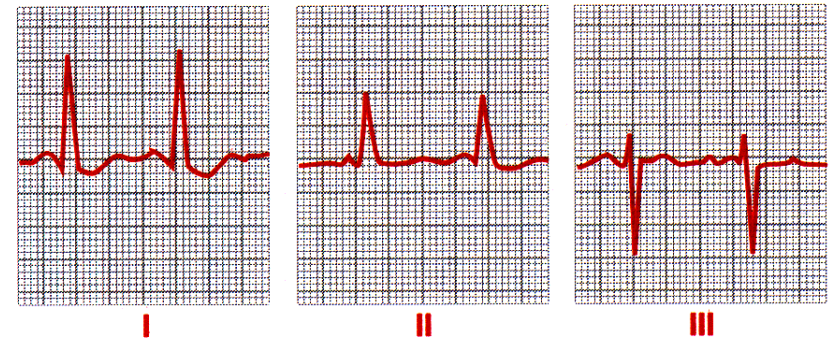
Figure 13-7. Complete atrioventricular block (lead II).

Stokes-Adams Syndrome

- Complete A-V block comes and goes. The duration of block may be a few seconds, a few minutes, a few hours, or even weeks or longer before conduction returns. This condition occurs in hearts with ischemia of the conductive system.
- Ventricles stop contracting for 5-30 sec because of overdrive suppression meaning they are used to atrial drive.
- Patient faints because of poor cerebral blood flow
- Then, ventricular escape occurs with A-V nodal or A-V bundle rhythm (15-40 beats /min).
- Artificial pacemakers connected to right ventricle are provided for these patients.
- overdrive suppression means that ventricular excitability is at first suppressed because the ventricles have been driven by the atria at a rate greater than their natural rate of rhythm. However, after a few seconds, some part of the Purkinje system beyond the block, usually in the distal part of the A-V node beyond the blocked point in the node, or in the A-V bundle, begins discharging rhythmically at a rate of 15 to 40 times/min, acting as the pacemaker of the ventricles. This phenomenon is called ventricular escape.

Factors Causing Electrical Axis deviation

- Changes in heart position: left shift caused by expiration, lying down and excess abdominal fat, short and obese.
- Right shift caused by thin and tall person



Factors Causing Electrical Axis Deviation

...cont'd

- Hypertrophy of left ventricle (left axis shift) caused by hypertension, aortic stenosis or aortic regurgitation causes slightly prolonged QRS and high voltage.

A high voltage ECG is an ECG that gives a voltage greater than 4mv after summing the voltage difference in each bipolar limb lead.

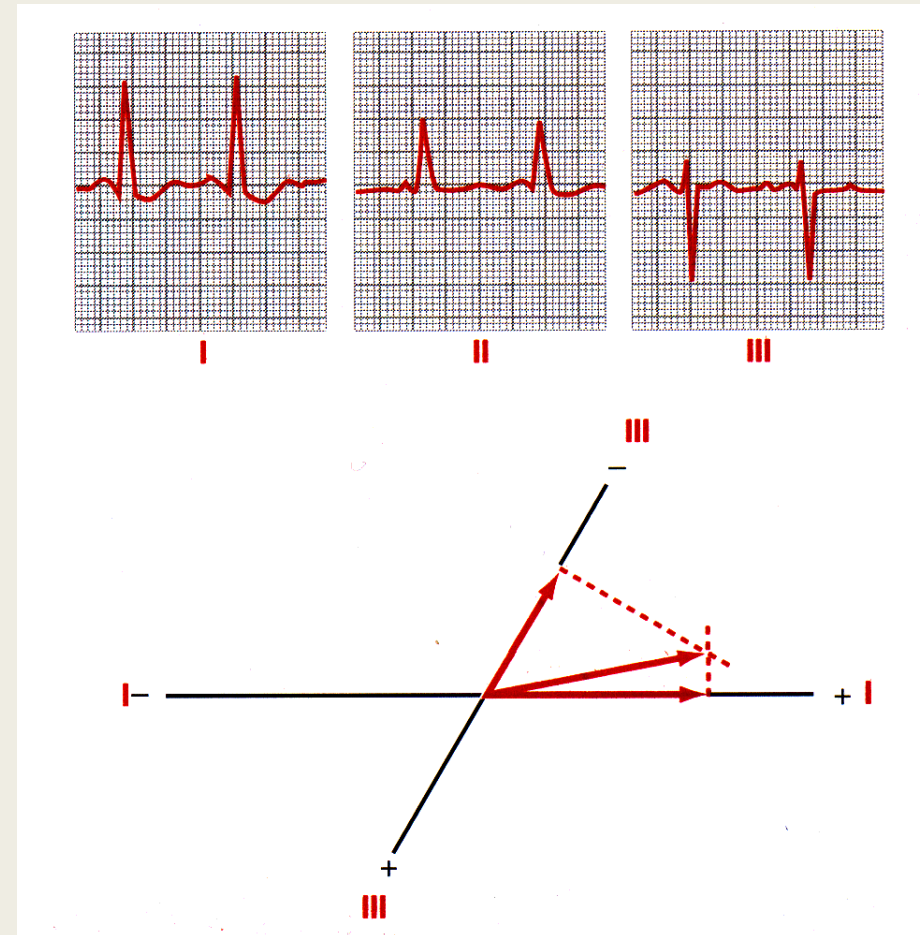
For example in the picture the voltage difference in lead I = $1.6 \text{ mv} - (-0.2 \text{ mv}) = 1.8 \text{ mv}$

Lead II = $1 \text{ mv} - (-0.1 \text{ mv}) = 1.1 \text{ mv}$

Lead III = $0.3 \text{ mv} - (-1.4 \text{ mv}) = 1.7 \text{ mv}$

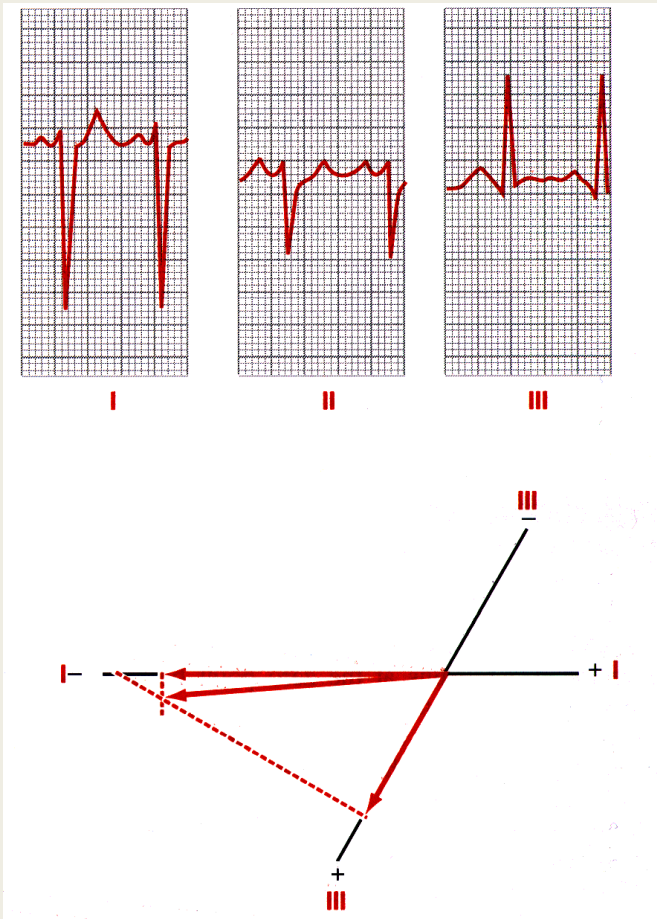
I+II+III = 4.6mv

This is a high voltage ECG, those are only seen in axis deviation due to hypertrophy, and not seen in deviations due to bundle branch block (left or right)



It occurs because the hypertrophied side now carries a higher than normal charge, augmenting the length of the mean QRS vector.

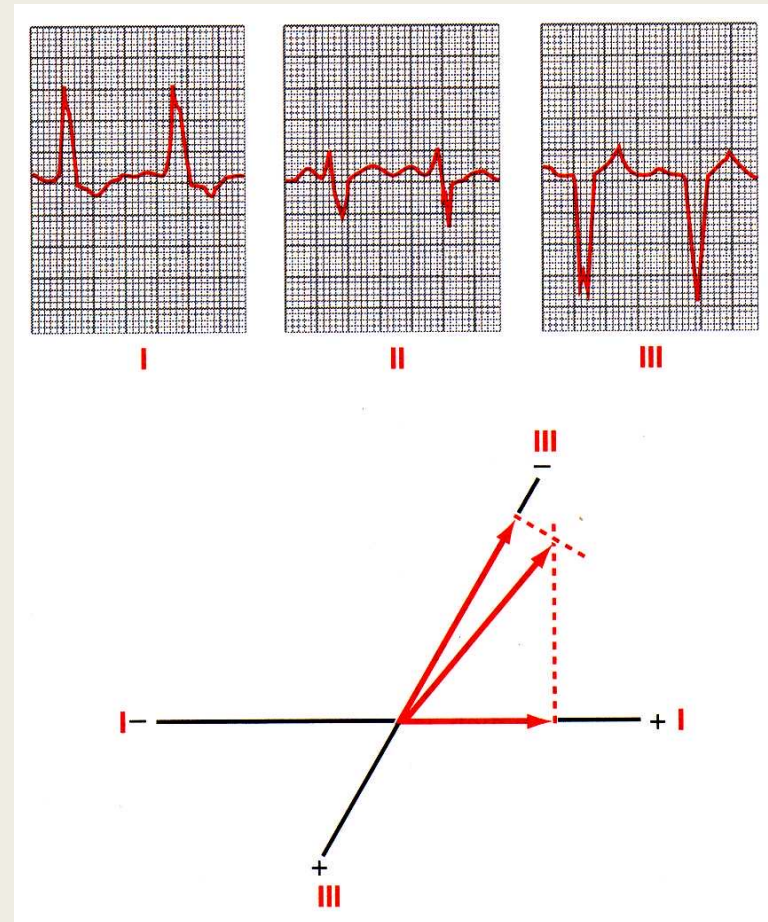
Factors Causing Electrical Axis Deviation (cont'd)



- Hypertrophy of right ventricle (right axis shift) caused by pulmonary hypertension, pulmonary valve stenosis, interventricular septal defect. All cause slightly prolonged QRS and high voltage.

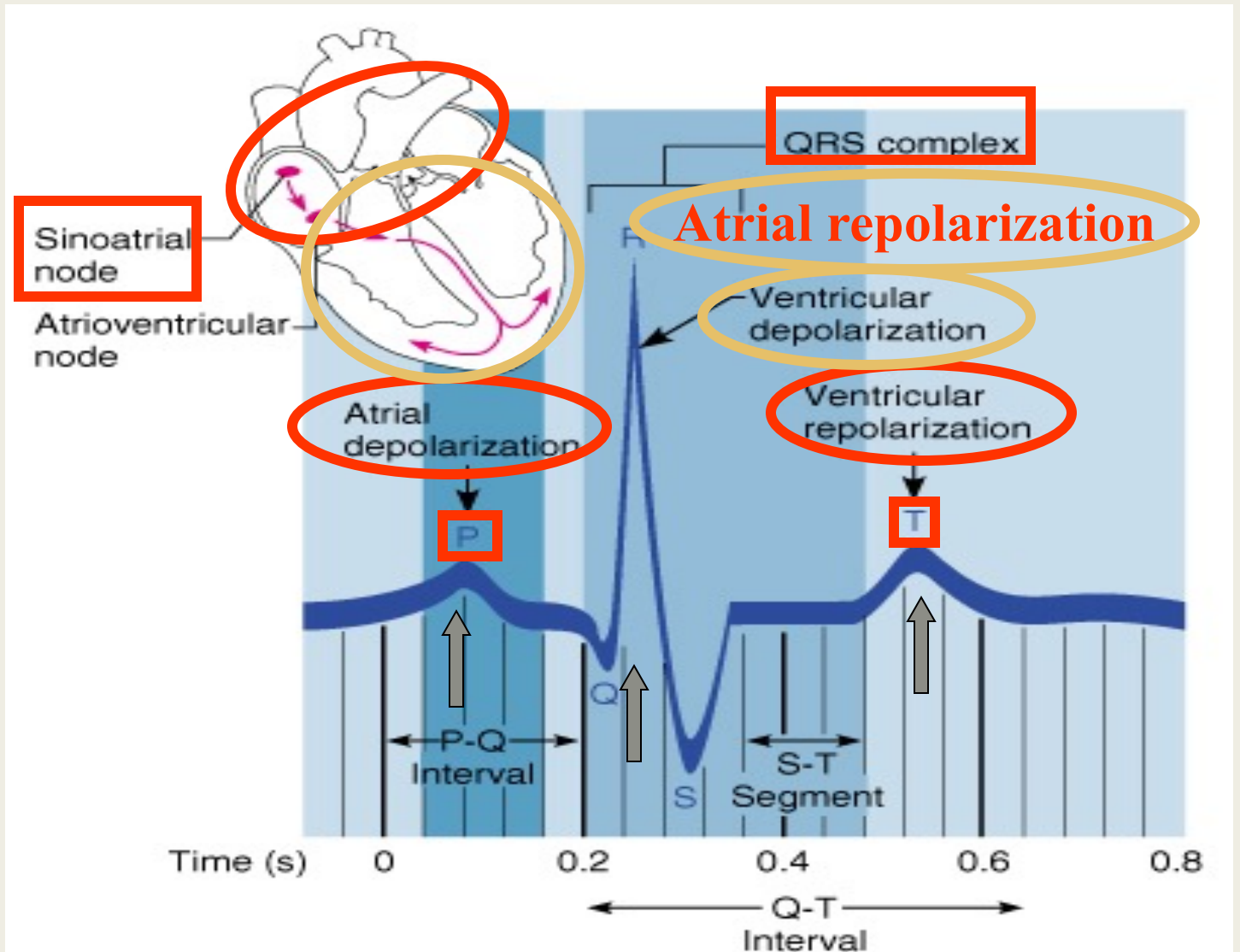
Factors Causing Electrical Axis Deviation ...cont'd

- Bundle branch block-**Left** bundle branch block causes **left** axis shift because **right** ventricle depolarizes much faster than **left** ventricle. QRS complex is prolonged. By the same token **Right** bundle branch block causes **right** axis deviation.



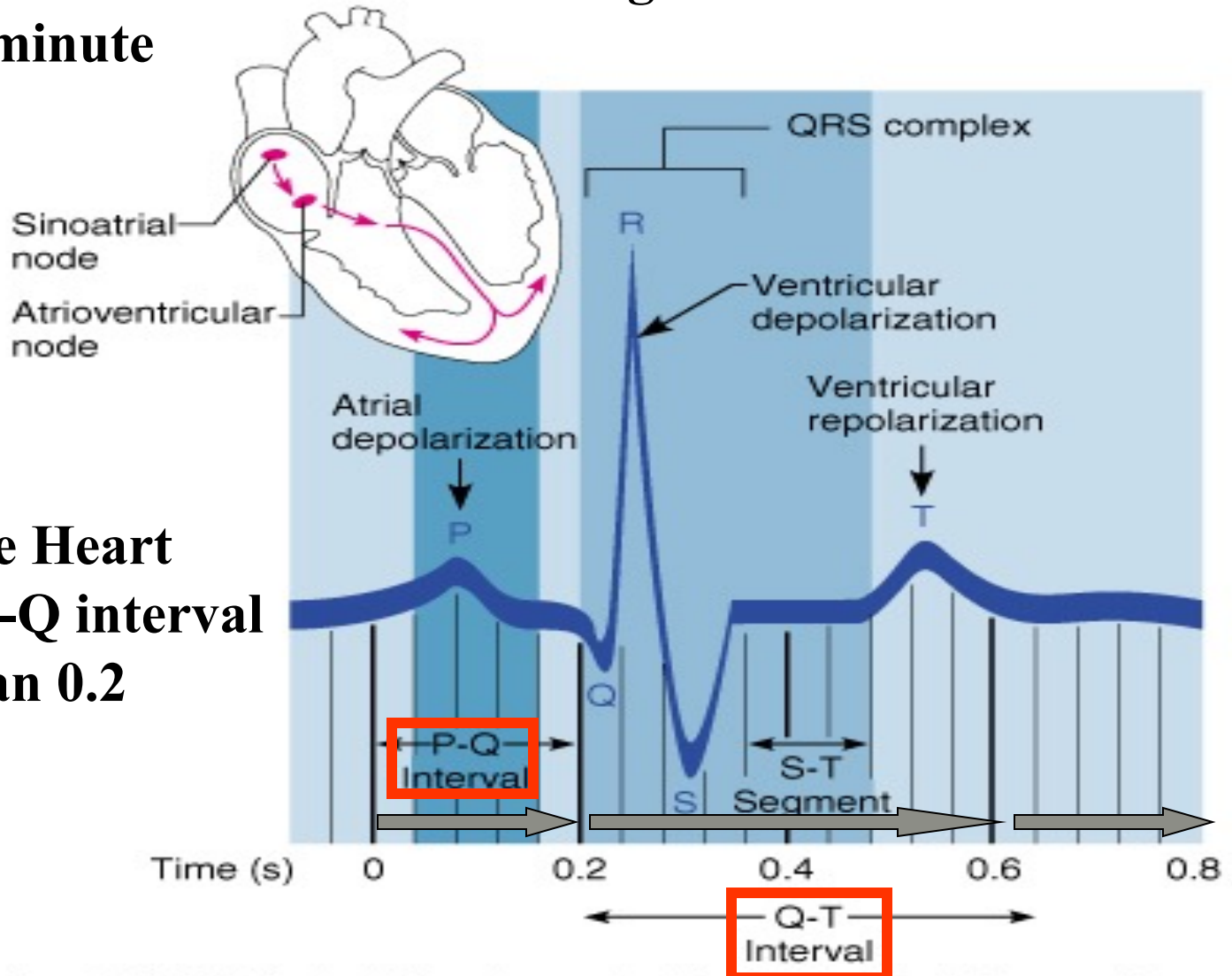
ECG Deflection Waves

(Pacemaker)



ECG Deflection Waves

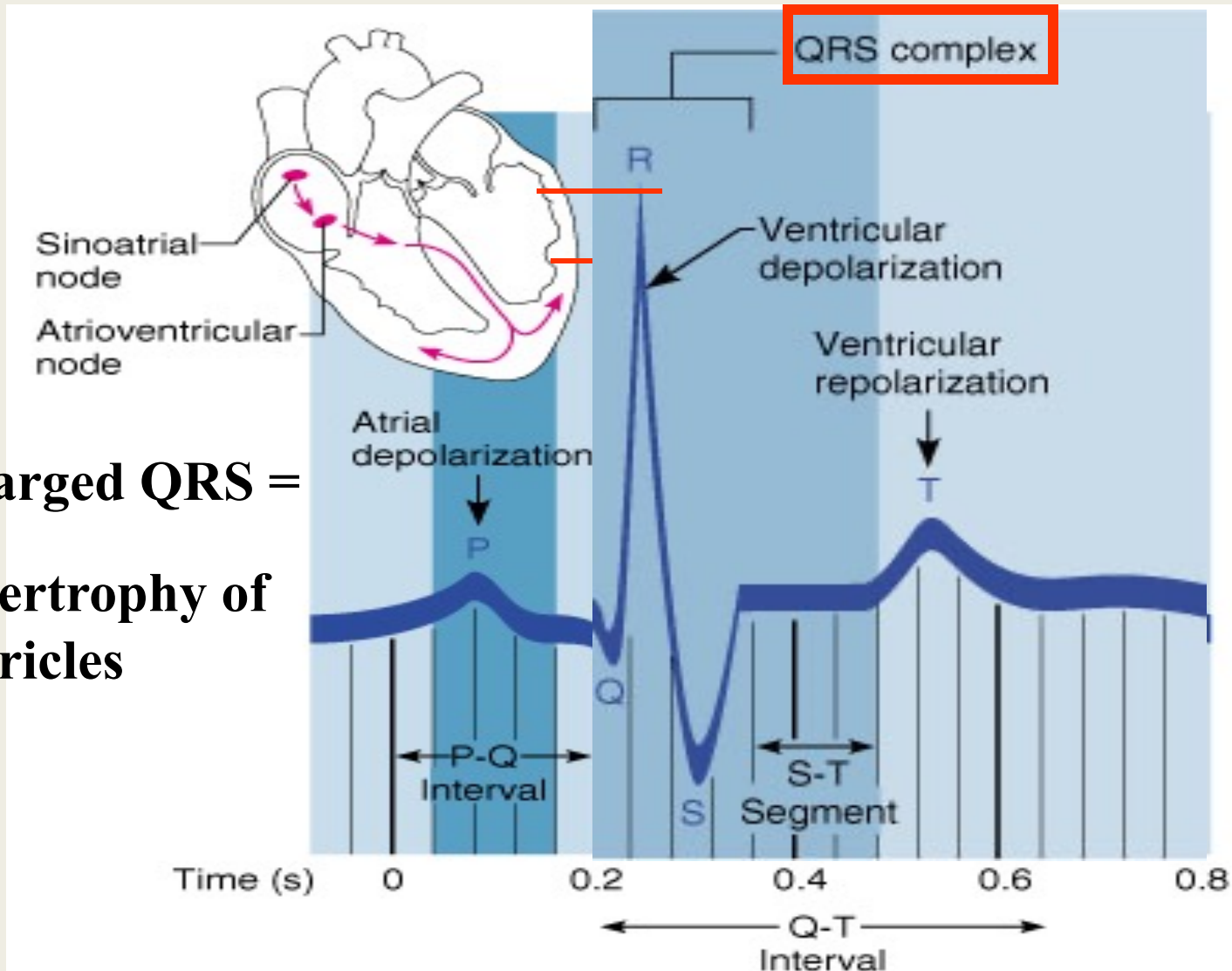
60 seconds \div 0.8 seconds = resting heart rate of 75 beats/minute



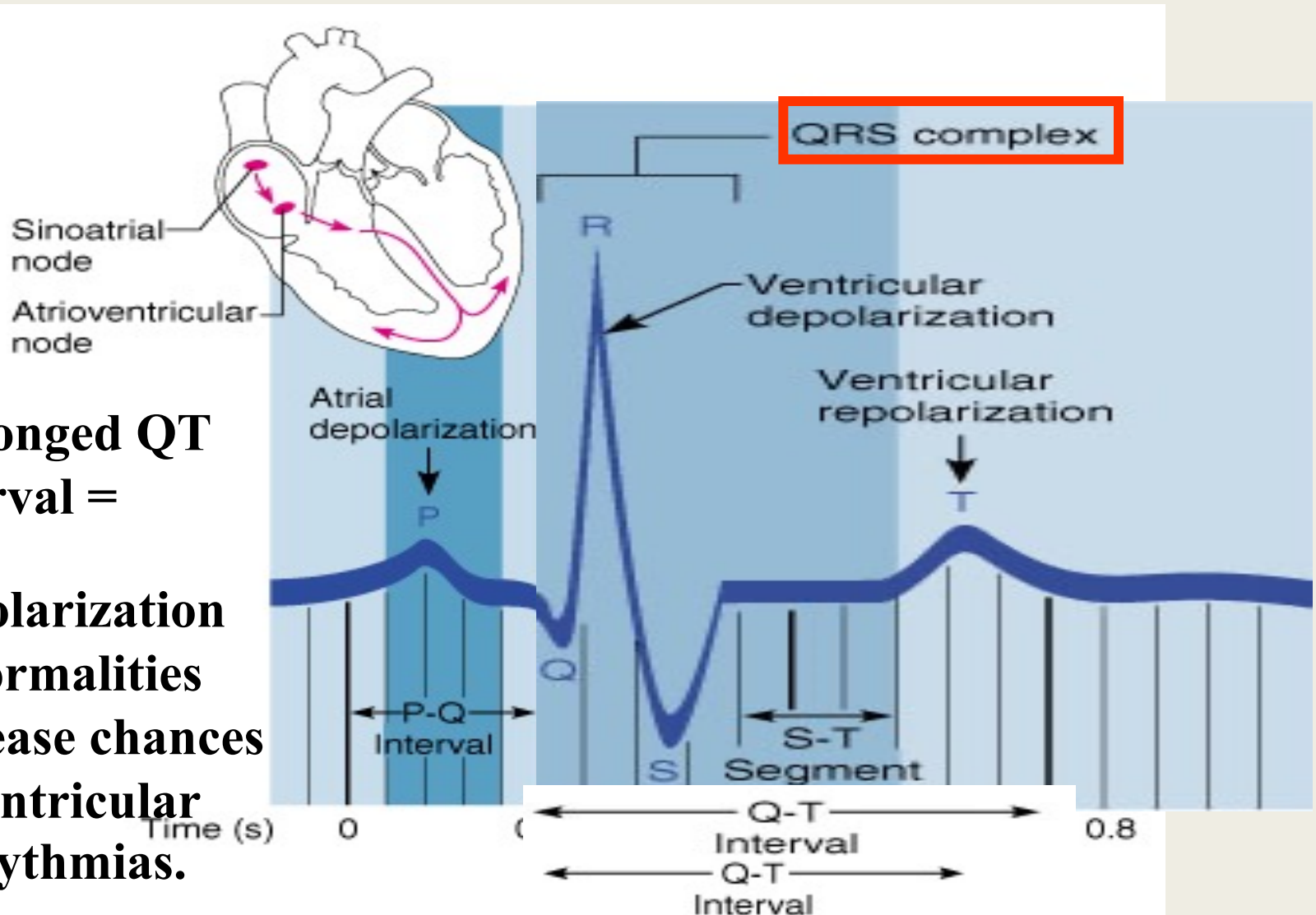
1st Degree Heart Block = P-Q interval longer than 0.2 seconds.

ECG Deflection Wave irregularities

**Enlarged QRS =
Hypertrophy of
ventricles**



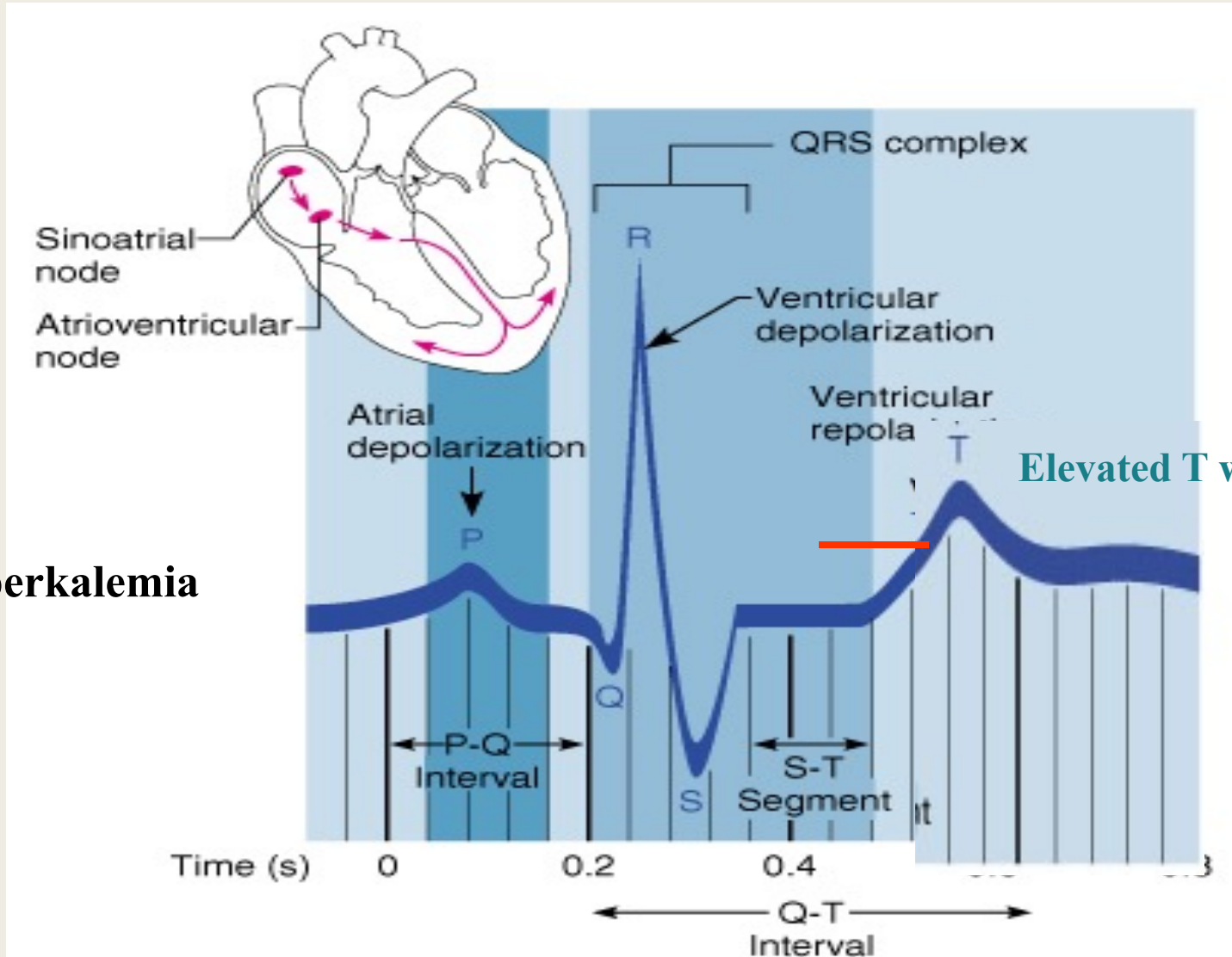
ECG Deflection Wave Irregularities



Prolonged QT Interval =

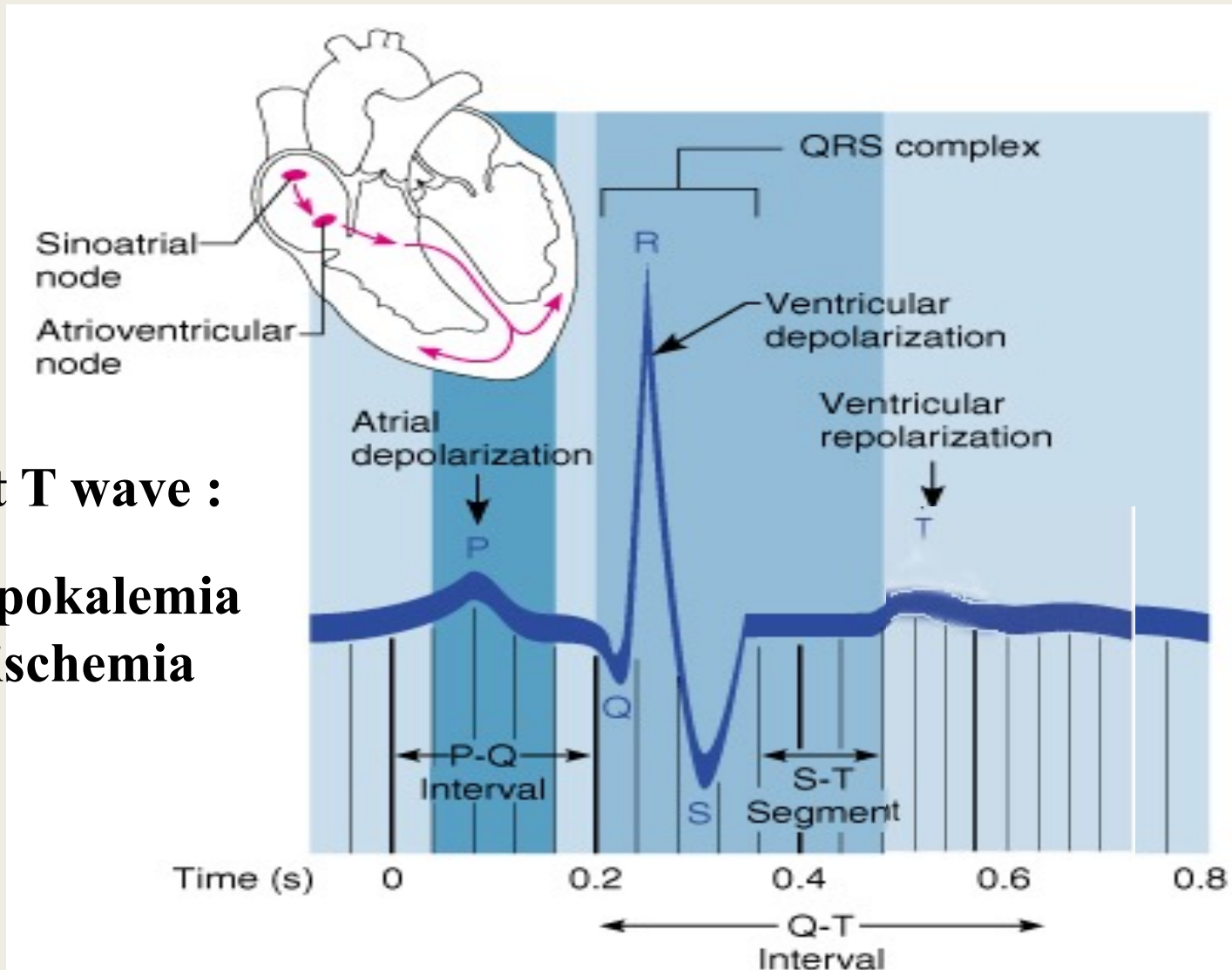
Repolarization abnormalities increase chances of ventricular arrhythmias.

ECG Deflection Wave Irregularities



Hyperkalemia

ECG Deflection Wave Irregularities



Flat T wave :
Hypokalemia
or ischemia

Increased Voltages in Standard Bipolar Limb Leads

- ❖ If sum of voltages of Leads I-III is greater than 4 mV, this is considered to be a high voltage EKG.
- ❖ Most often caused by increased ventricular muscle mass (hypertension, marathon runner).
- ❖ If sum is less than 1.5mv this is considered to be a low voltage EKG.

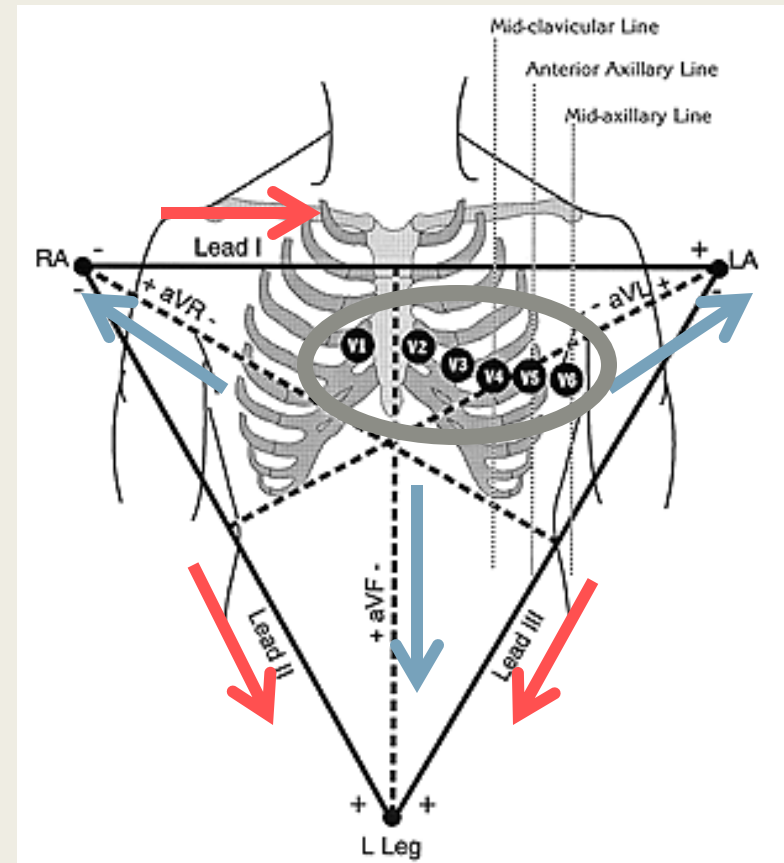
Decreased Voltages in Standard Bipolar Limb Leads

- ❏ Cardiac muscle abnormalities (old infarcts causing decreased muscle mass, low voltage EKG, and prolonged QRS).
- ❏ Conditions surrounding heart (fluid in pericardium, pleural effusions, emphysema).
- ❏ In these cases the tissues around the heart become better insulators with more resistance to electrical currents.

The 12-Leads

The 12-leads include:

- 3 Limb leads (I, II, III)
- 3 Augmented leads (aVR, aVL, aVF)
- 6 Precordial leads (V_1 - V_6)



Thank You

اللهم احرس أهل غزة بعينك التي لا تنام، واجبر كسرهم،
واشف مرضاهم، وتقبل شهادتهم. اللهم انصر الإسلام
والمسلمين في فلسطين وأعزهم، اللهم اجعل النصر قريباً
واجعل الفرَجَ يأتيهم.