Cardiovascular Physiology

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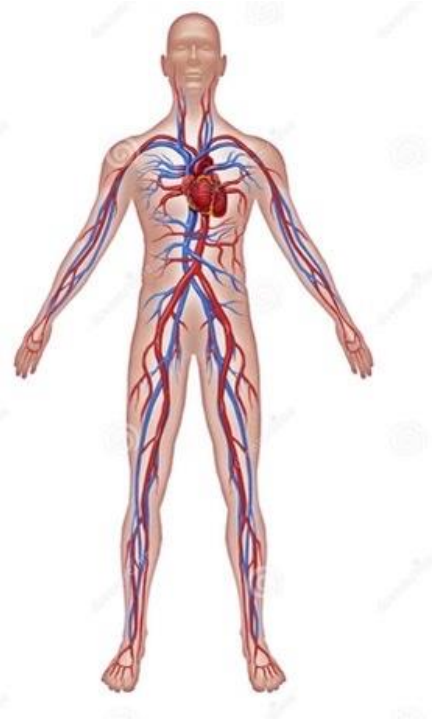
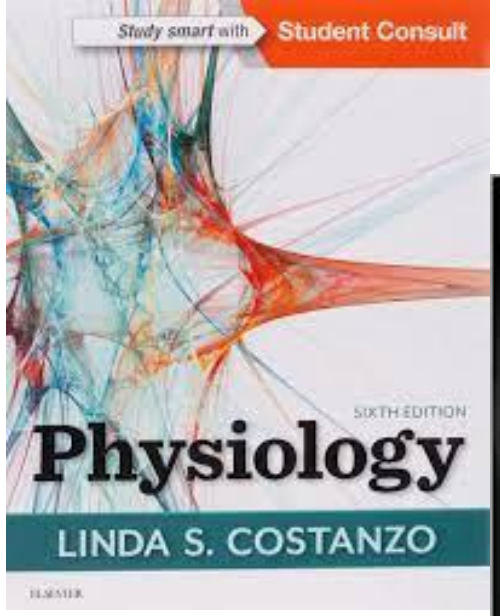
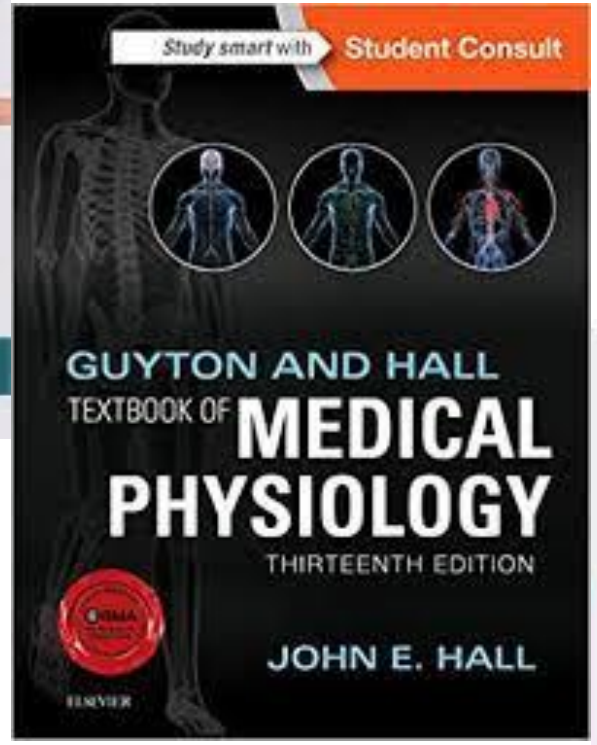
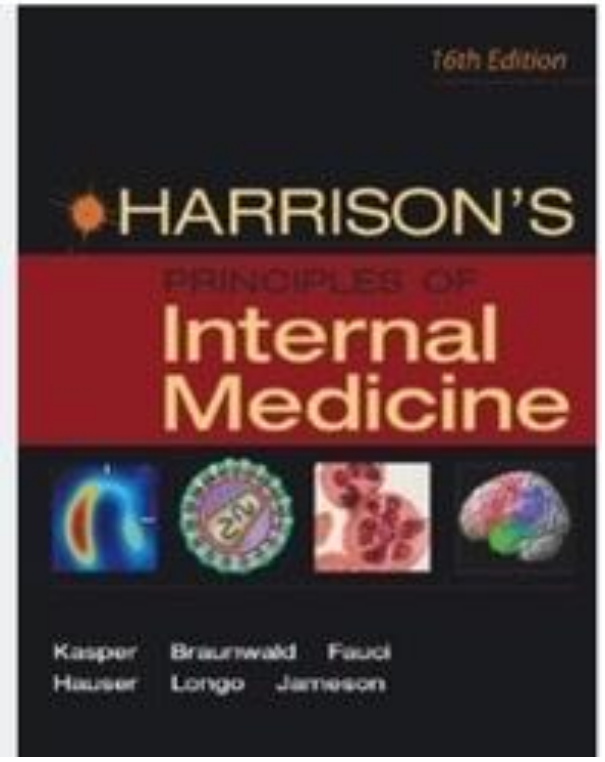
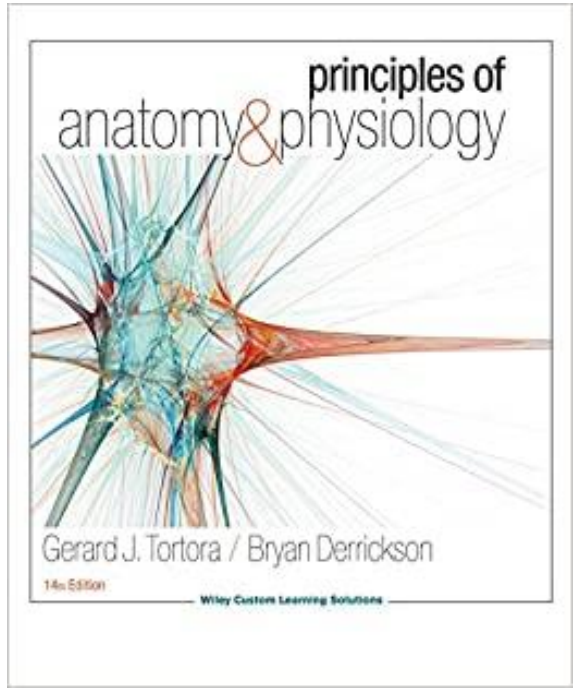


References



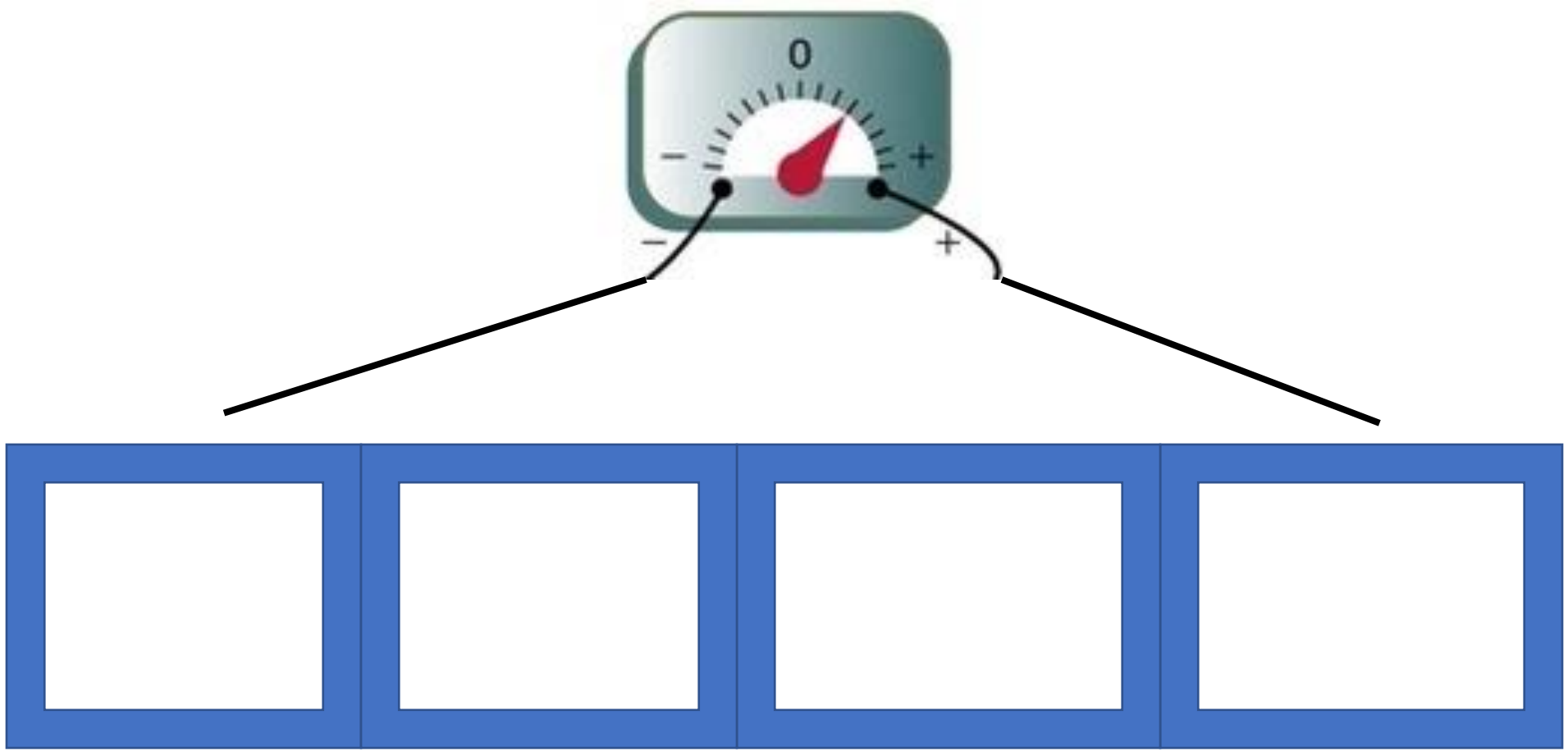






Ecgpedia.org

Electrocardiography


0
–
+
–
+

ECG basics

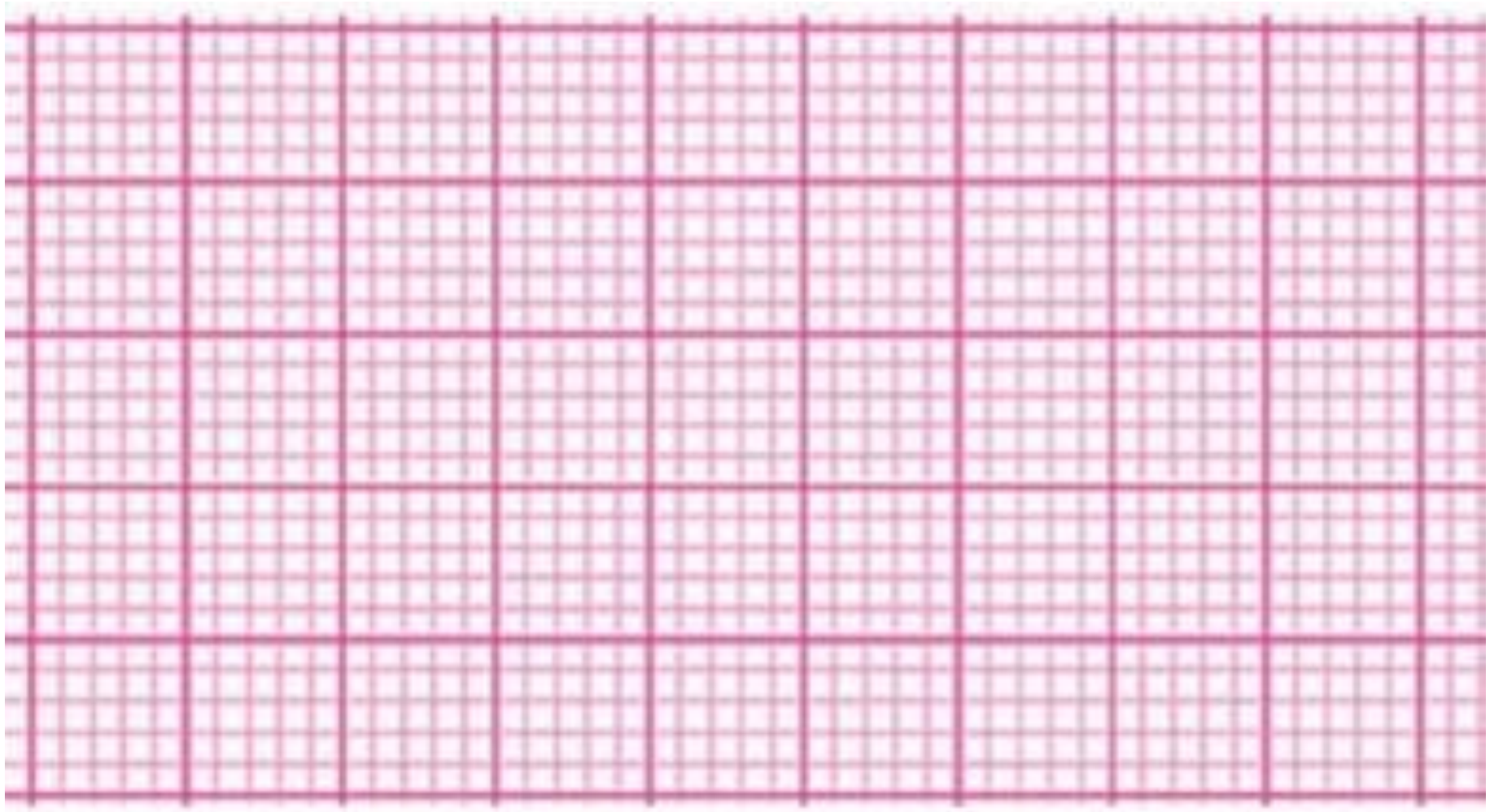
- Galvanometer measures potential difference between myocardial cells.
- no flow of ions, no deflection (straight line on ECG).
- no potential is recorded in the ECG when the ventricular muscle is either completely polarized or completely depolarized.
- Only when the muscle is partly polarized and partly depolarized does current flow from one part to another.

ECG basics

Movement of current in relation to the electrodes:

- + potential (depolarization) toward + electrode = + deflection
- **-** potential (repolarization) toward – electrode = + deflection
- **-** potential toward + electrode = **-** deflection
- + potential toward – electrode = **-** deflection on ECG strip.

Voltage (mV)



Time (seconds)

ECG calibration

- Therefore, each 25 millimeters in the horizontal direction is 1 second, and each 5-millimeter segment, indicated by the dark vertical lines, represents 0.20 second.

- The 0.20-second intervals are then broken into five smaller intervals by thin lines, each of which represents 0.04 second.

ECG calibration

- the horizontal calibration lines are arranged so that 10 of the small line divisions upward or downward in the standard ECG represent 1 millivolt, with positivity in the upward direction and negativity in the downward direction.

- The vertical lines on the ECG are time calibration lines. A typical ECG is run at a speed of 25 millimeters per second, although faster speeds are sometimes used.

amplitude

- Changes in membrane potential in small parts of the heart such as SA node and AV node will not be detected by ECG.

- The amplitude of the deflection is determined by the tissue mass; the larger the mass (ventricle vs atrium), the larger the amplitude (voltage).

Time

- The speed of deflection is determined by the speed of conduction; faster in ventricular depolarization than atrial depolarization.

Wave voltage

- The recorded voltages of the waves in the normal ECG depend on the manner in which the electrodes are applied to the surface of the body and how close the electrodes are to the heart.

ECG leads

- Standard ECG has 12 leads, that help “looking” at the heart from different angles.

- Bipolar limb leads: 3 leads.
- Augmented unipolar limb leads: 3 leads.
- Chest leads: 6 leads.

- Limb leads: on vertical plane, chest leads: on horizontal plane.

- the direction from negative electrode to positive electrode is called the axis of the lead.

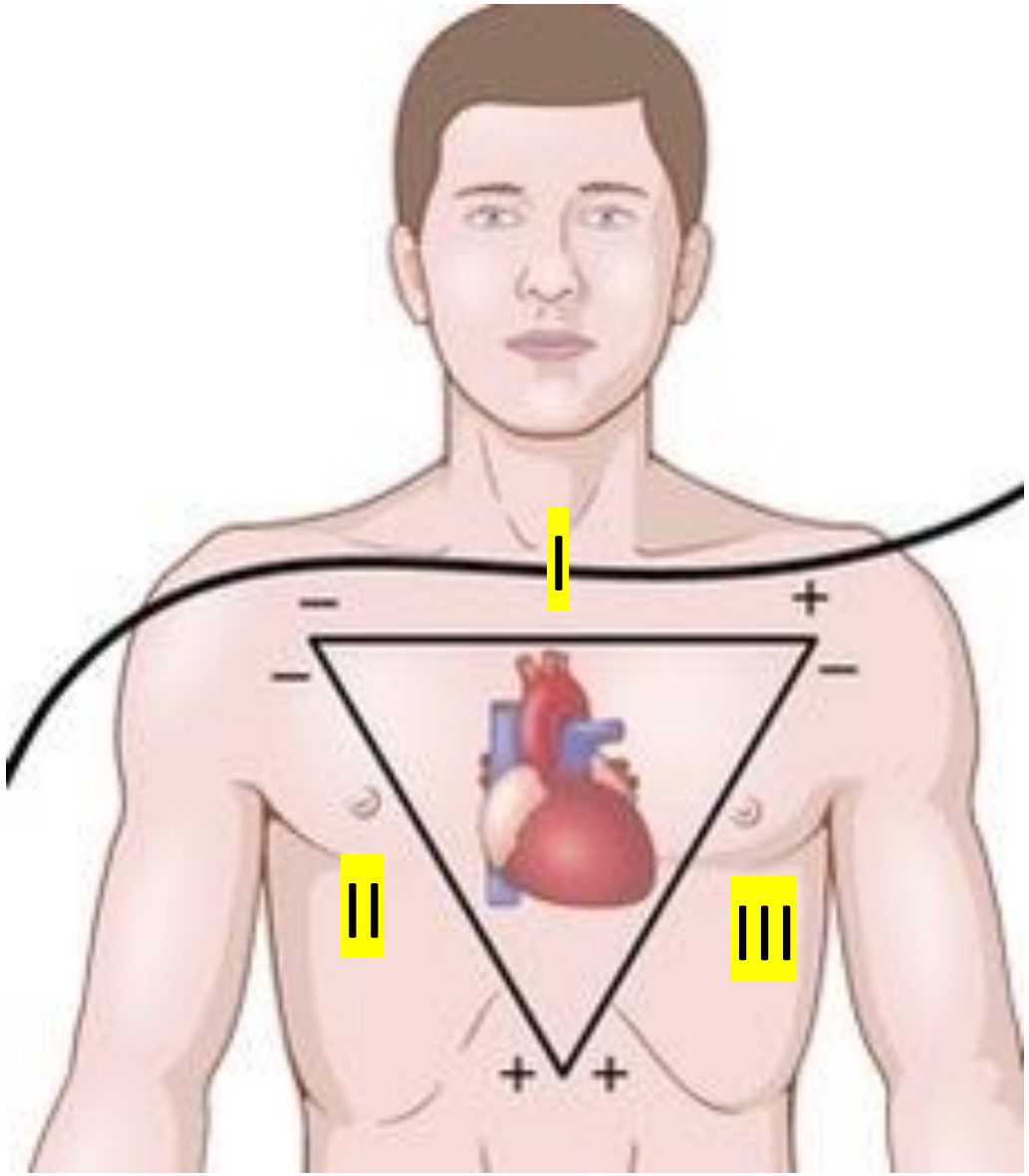
ECG limb leads

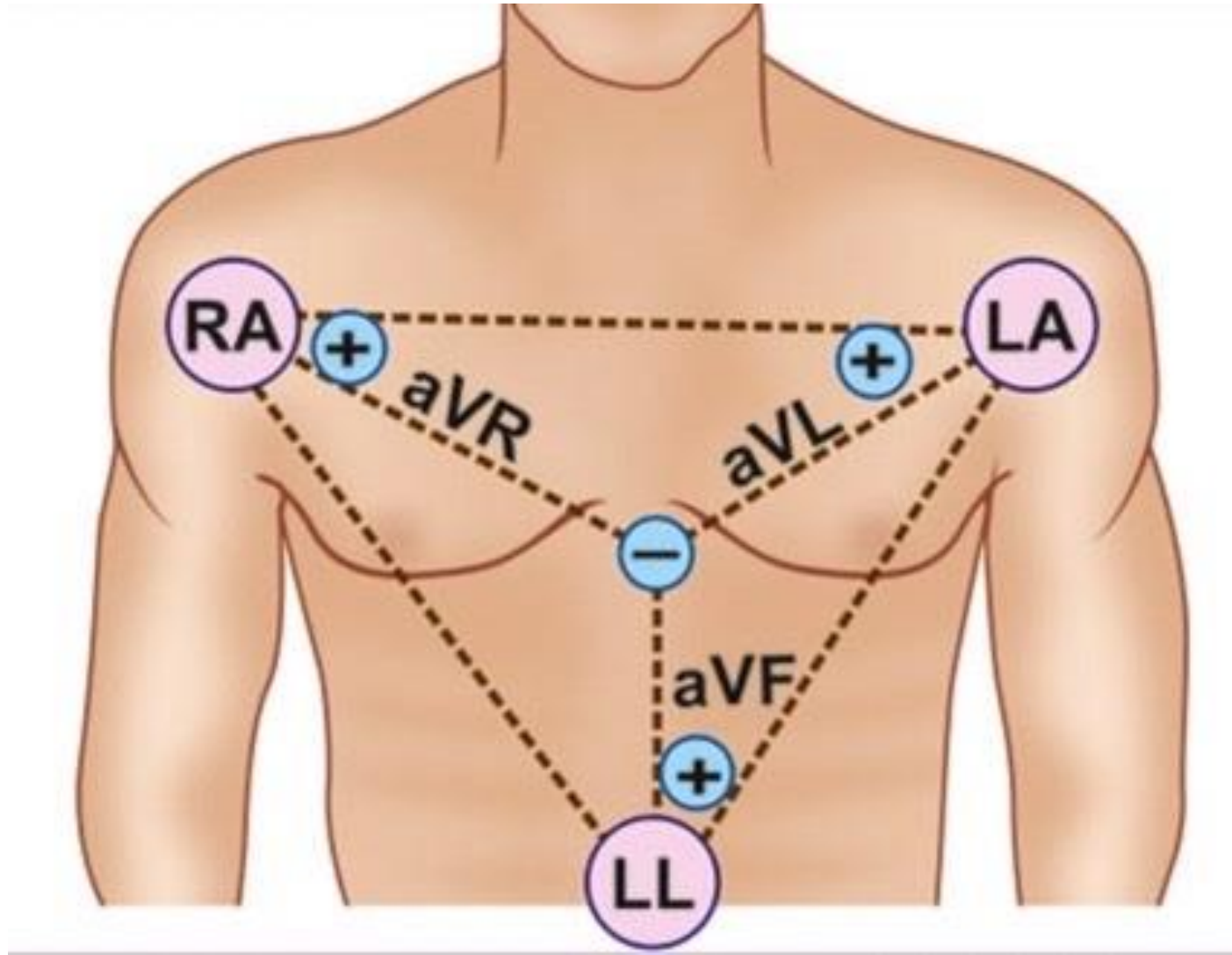
- Bipolar limb leads: bipolar means that the ECG is recorded from two electrodes located on different limbs.

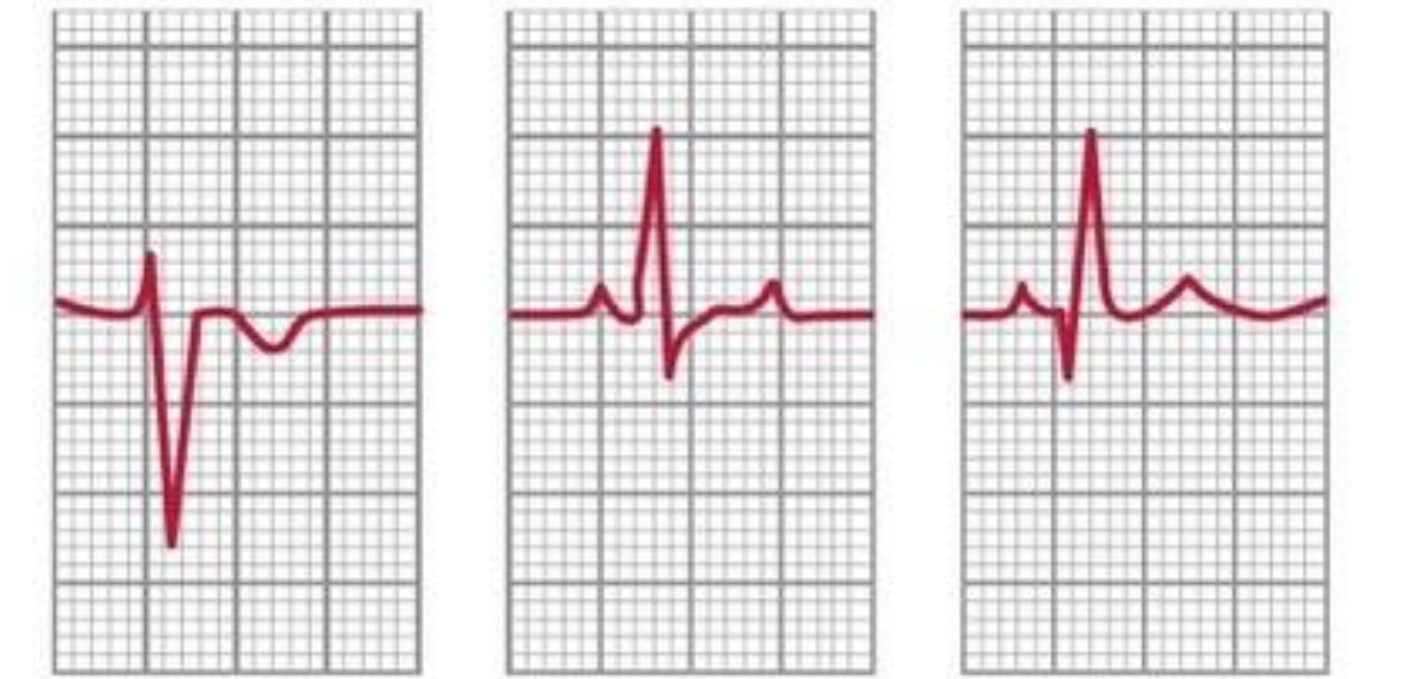
- Augmented unipolar limb leads: two of the limbs are connected through electrical resistances to the negative terminal of the electrocardiograph, and the third limb is connected to the positive terminal.

- The recording of the aVR lead is inverted.

Einthoven’s triangle



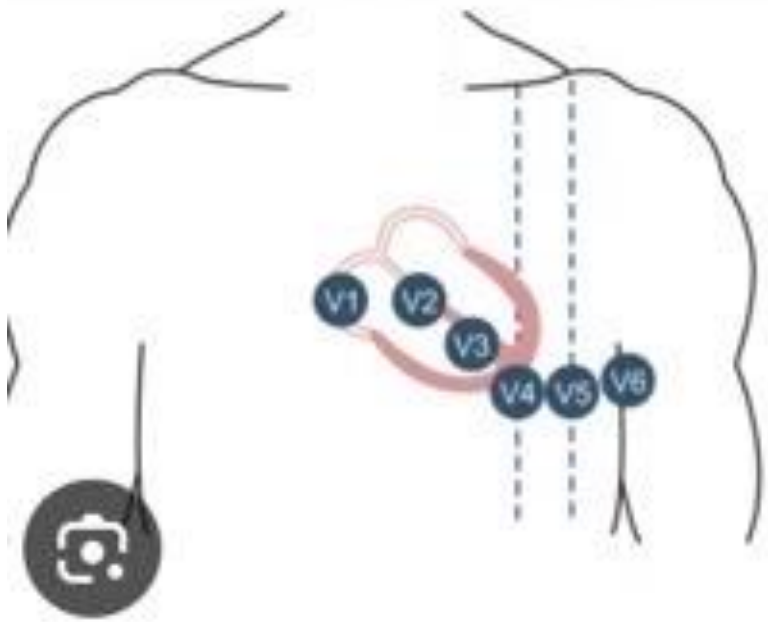

RA
LA
aVR
aVL
aVF
LL

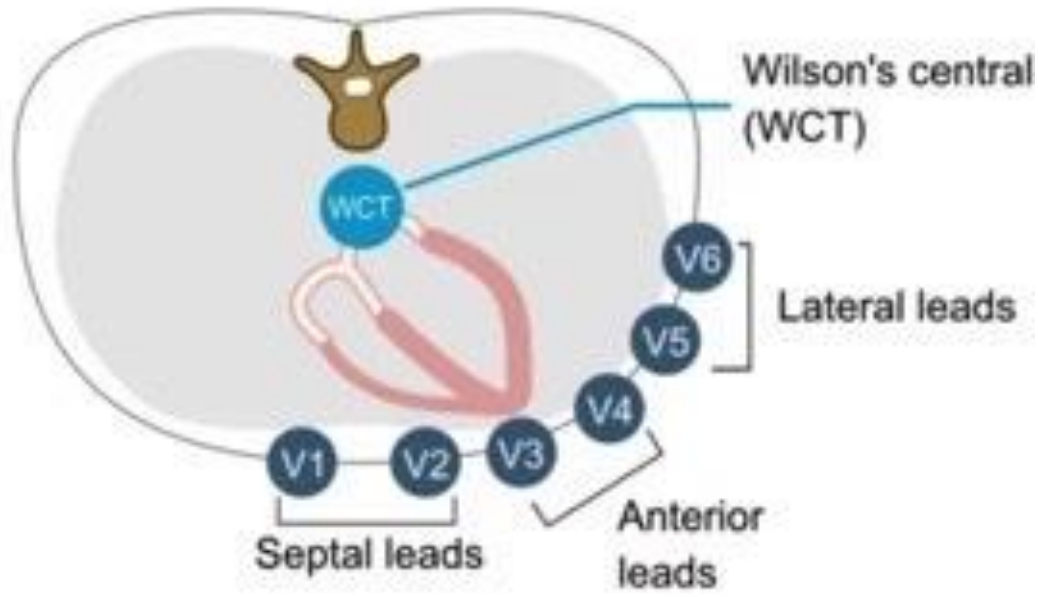

aVR
aVL
aVF

Chest leads

- Because the heart surfaces are close to the chest wall, each chest lead records mainly the electrical potential of the cardiac musculature immediately beneath the electrode.

- relatively minute abnormalities in the ventricles, particularly in the anterior ventricular wall, can cause marked changes in the ECGs recorded from individual chest leads.

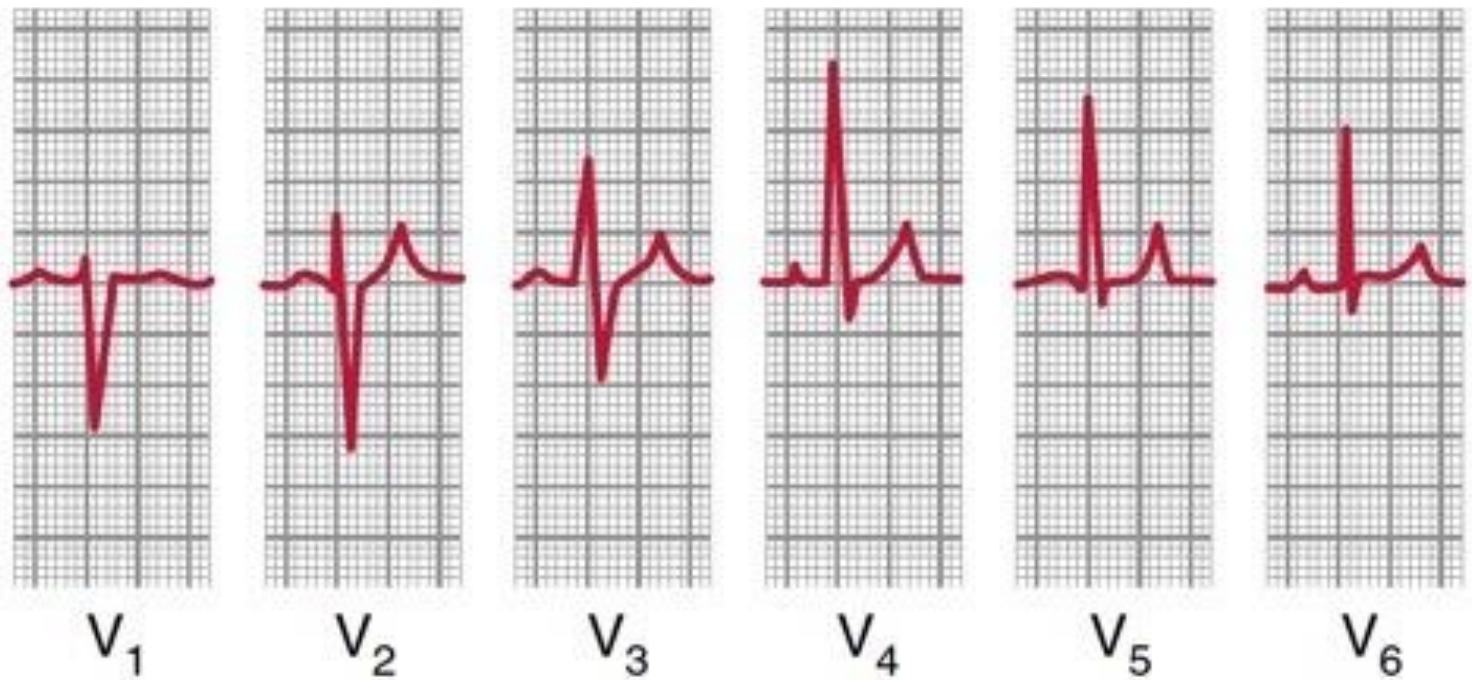

V1
V2
V3
V4
V5
V6


Wilson's central
(WCT)
WCT
V6
V5
V4
V3
V2
V1
Lateral leads
Septal leads
Anterior
leads

Chest leads

- In leads V1 and V2, the QRS recordings of the normal heart are mainly negative (near the base).

- Conversely, the QRS complexes in leads V4, V5, and V6 are mainly positive (near the apex).


V1
V2
V3
V4
V5
V6

Projected vectors

- To determine how much of the voltage in a vector will be recorded in a lead, a line perpendicular to the axis of the lead is drawn from the tip of the vector to the lead axis, and a so-called projected vector is drawn along the lead axis.

- The arrow of the projected vector points toward the positive or the negative end of the lead axis.

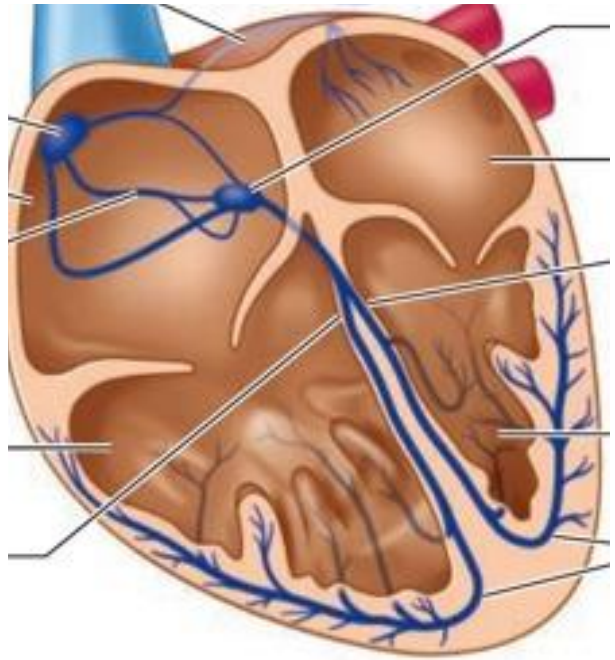
Vectorial analysis of ECG

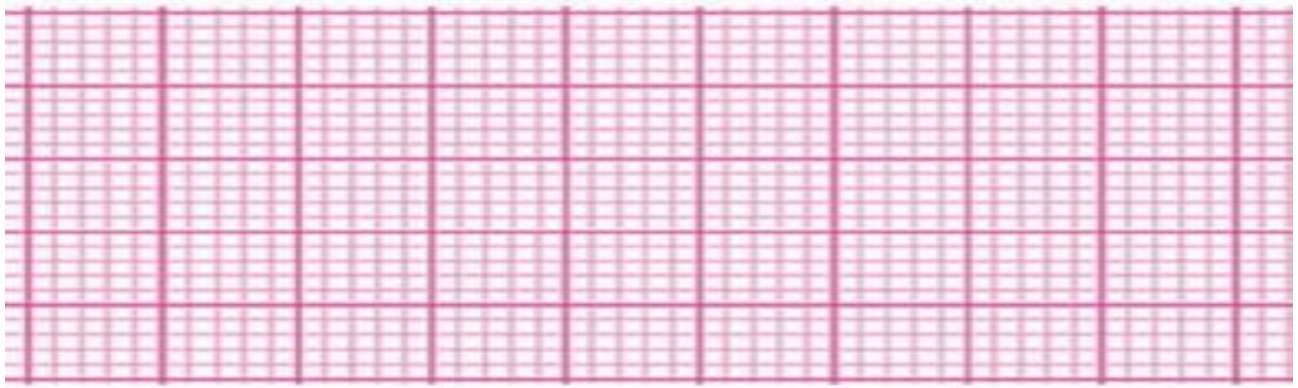
- when the vector in the heart is in a direction almost perpendicular to the axis of the lead, the voltage recorded in the ECG of this lead is very low.

- when the heart vector has almost exactly the same axis as the lead axis, essentially the entire voltage of the vector will be recorded.

Vectorial analysis of ECG

- heart current flows in a particular direction in the heart at a given instant during the cardiac cycle.
- A vector is an arrow that points in the direction of the electrical potential generated by the current flow.
- Also, by convention, the length of the arrow is drawn proportional to the voltage of the potential.





ECG waves

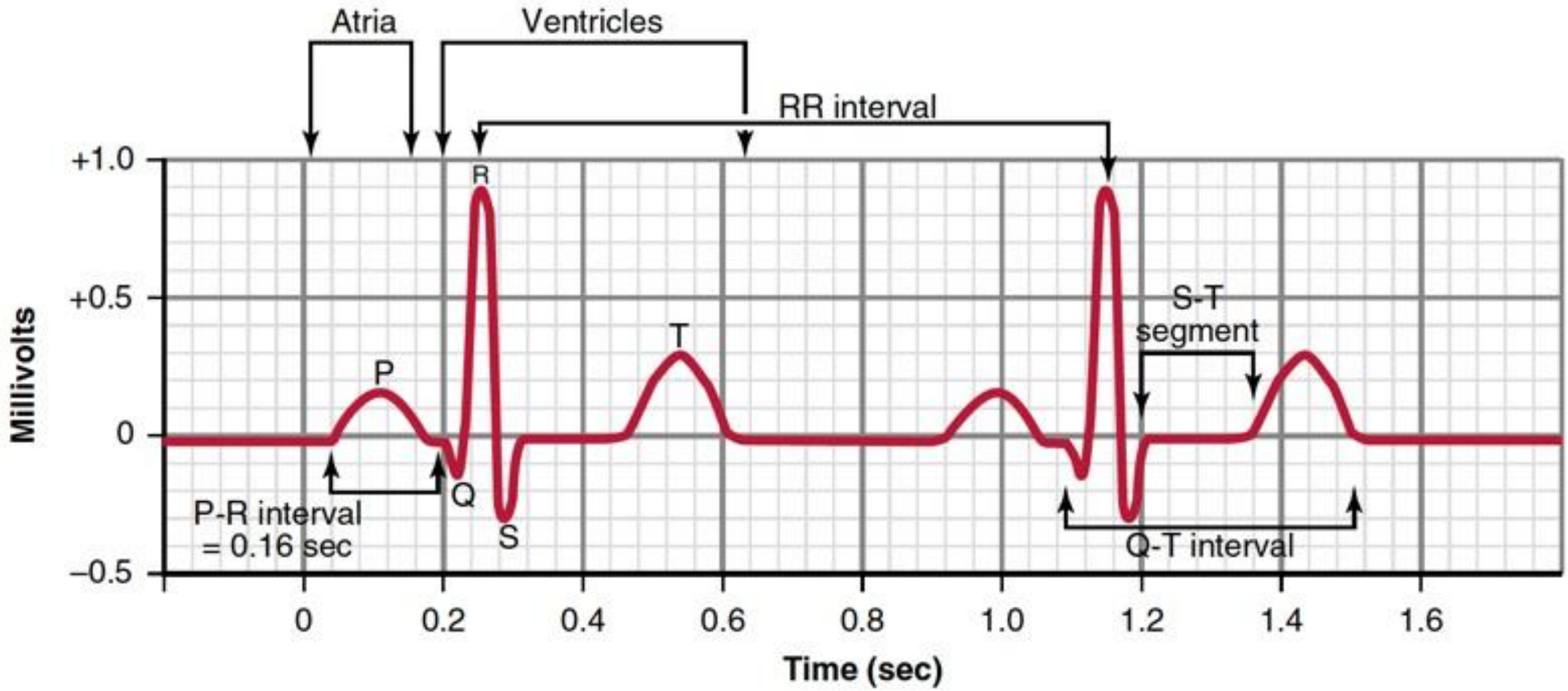
- Depolarization waves:
- Atria (P wave)
- Ventricle (QRS complex)

- Repolarization waves:
- Atria (not shown on ECG, low voltage and occur simultaneously with QRS)
- Ventricles (T wave)

ECG segments and intervals

- P-R segment
- S-T segment

- P-R interval
- Q-T interval


Atria
Ventricles
RR interval
+1.0
+0.5
0
–0.5
Millivolts
R
P
T
S-T
segment
Q
S
P-R interval
= 0.16 sec
Q-T interval
0
0.2
0.4
0.6
0.8
1.0
1.2
1.4
1.6
Time (sec)

Atrial depolarization and repolarization

- Spread of depolarization through the atrial muscle is much slower than in the ventricles because the atria have no Purkinje system.

- the area in the atria that also becomes repolarized first is the sinus nodal region, the area that had originally become depolarized first (atrial T wave).

- Therefore, the atrial repolarization vector is backward to the vector of depolarization.

Ventricular depolarization

- When the cardiac impulse enters the ventricles through the atrioventricular bundle, the first part of the ventricles to become depolarized is the left endocardial surface of the septum.
- Then, depolarization spreads rapidly to involve both endocardial surfaces of the septum

Ventricular repolarization

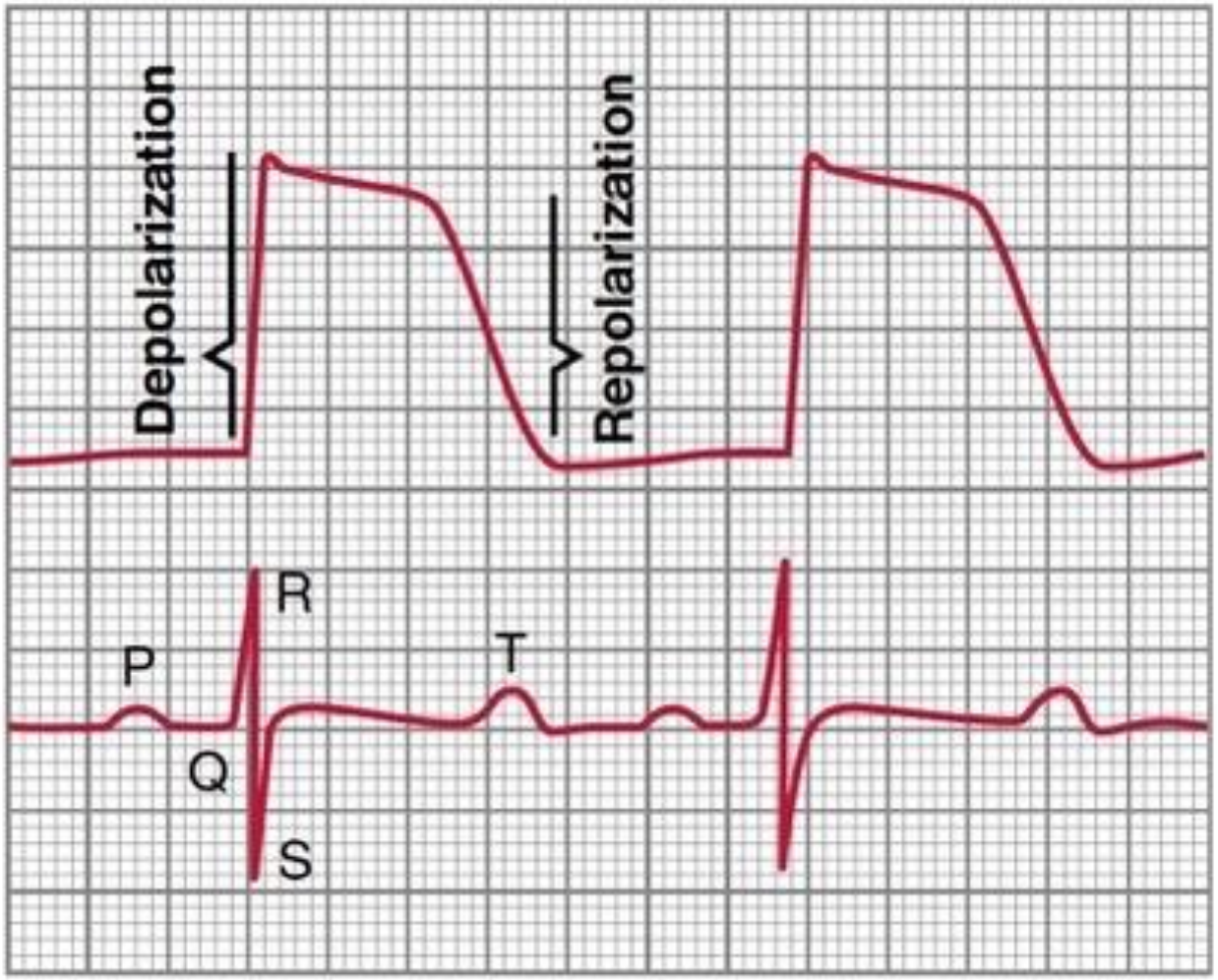
- Because the septum and endocardial areas of the ventricular muscle depolarize first, it seems logical that these areas should repolarize first as well.
- However, this is not the usual case, because the septum and other endocardial areas have a longer period of contraction than most of the external surfaces of the heart.

- Therefore, the greatest portion of ventricular muscle mass to repolarize first is the entire outer surface of the ventricles, especially near the apex of the heart.
- The endocardial areas, conversely, normally repolarize last.
- This sequence of repolarization is postulated to be caused by the high blood pressure inside the ventricles during contraction, which greatly reduces coronary blood flow to the endocardium, thereby slowing repolarization in the endocardial areas.

Ventricular repolarization

- The process of ventricular repolarization extends over a long period, about 0.15 second.

- For this reason, the T wave in the normal ECG is:
- prolonged wave,
- the voltage of the T wave is less than the voltage of the QRS complex.



T wave compared to QRS

Einthoven’s law

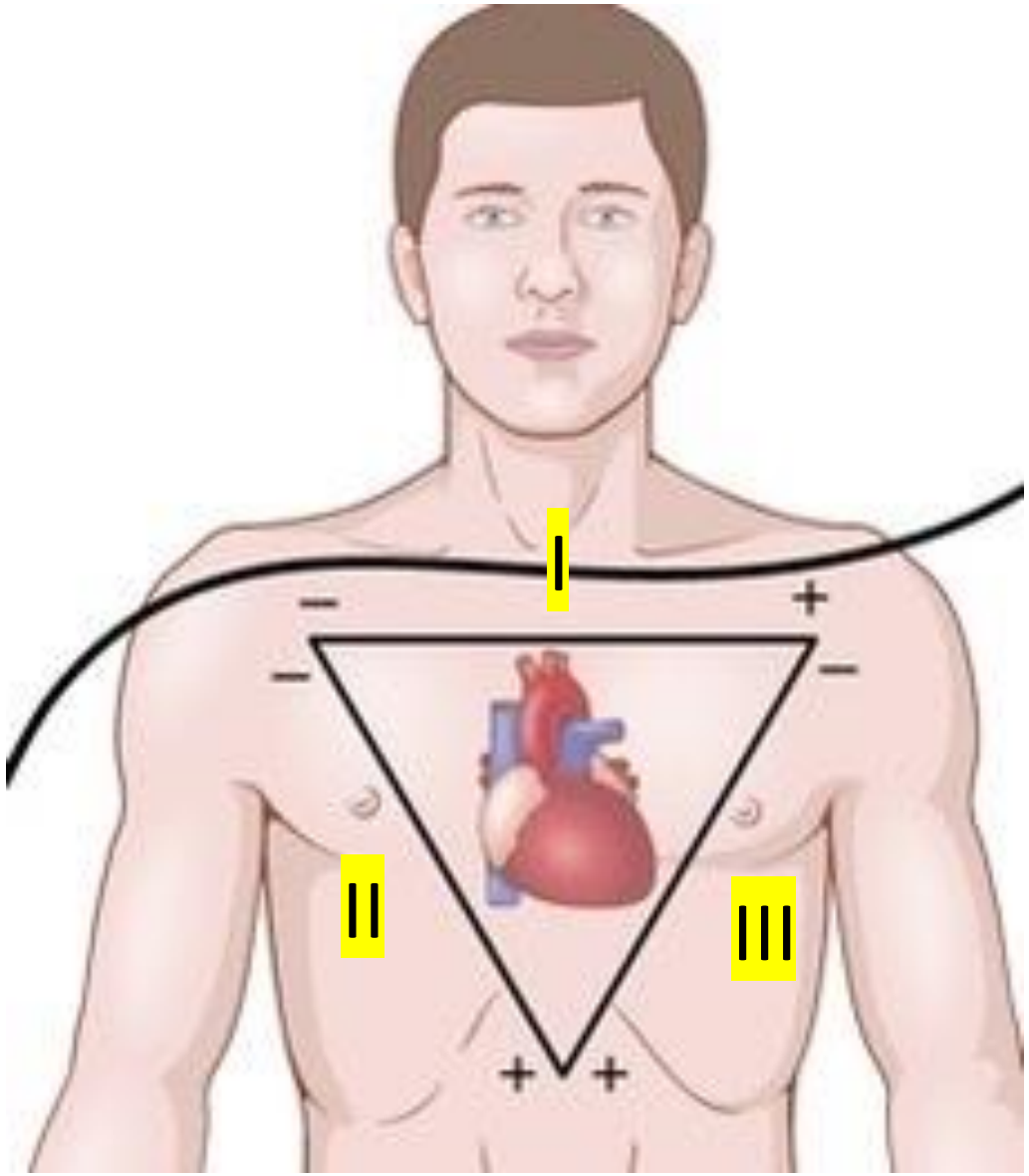
- if the ECGs are recorded simultaneously with the three limb leads, the sum of the potentials recorded in leads I and III will equal the potential in lead II:

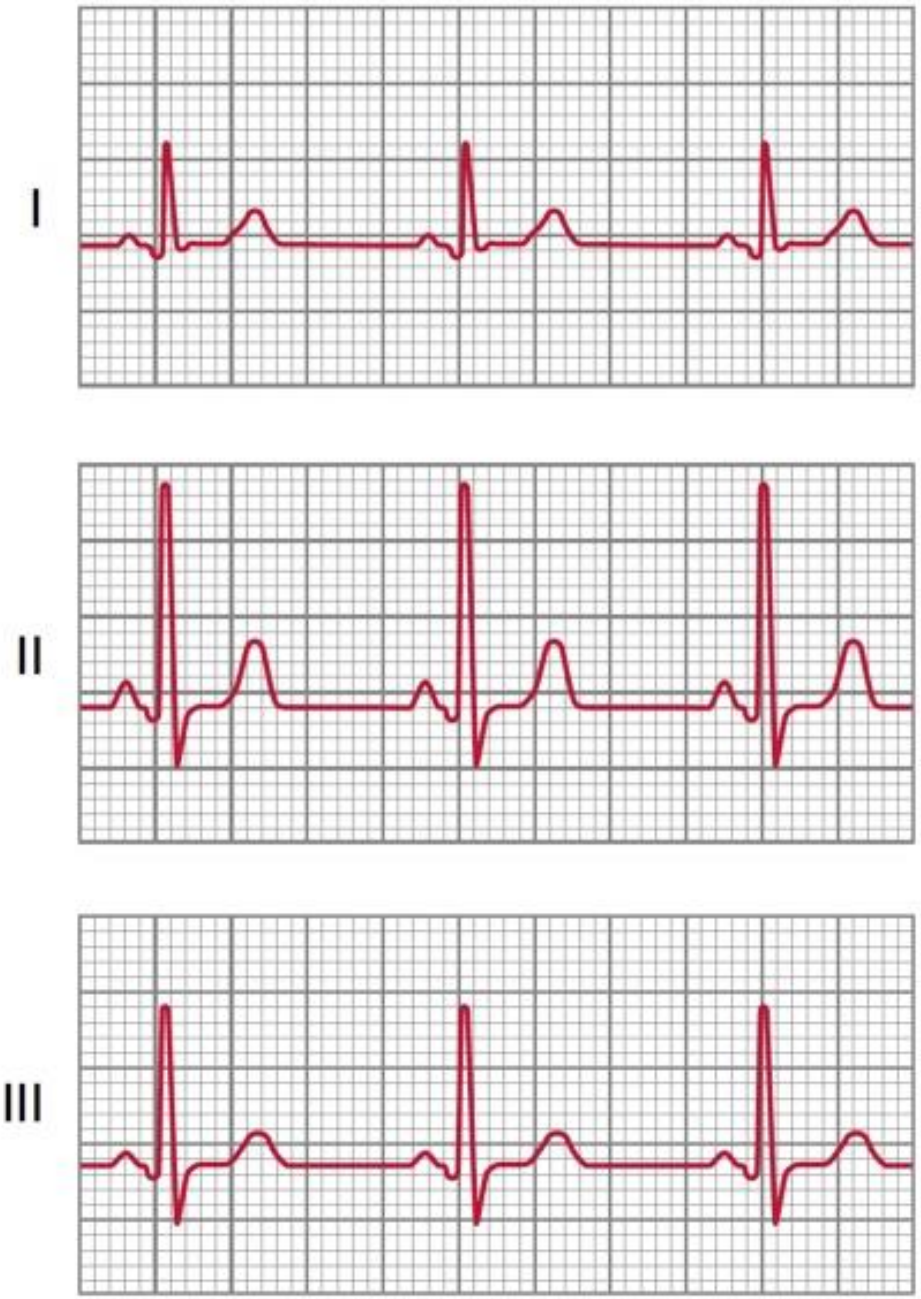
- Lead I potential+ Lead III potential = Lead II potential

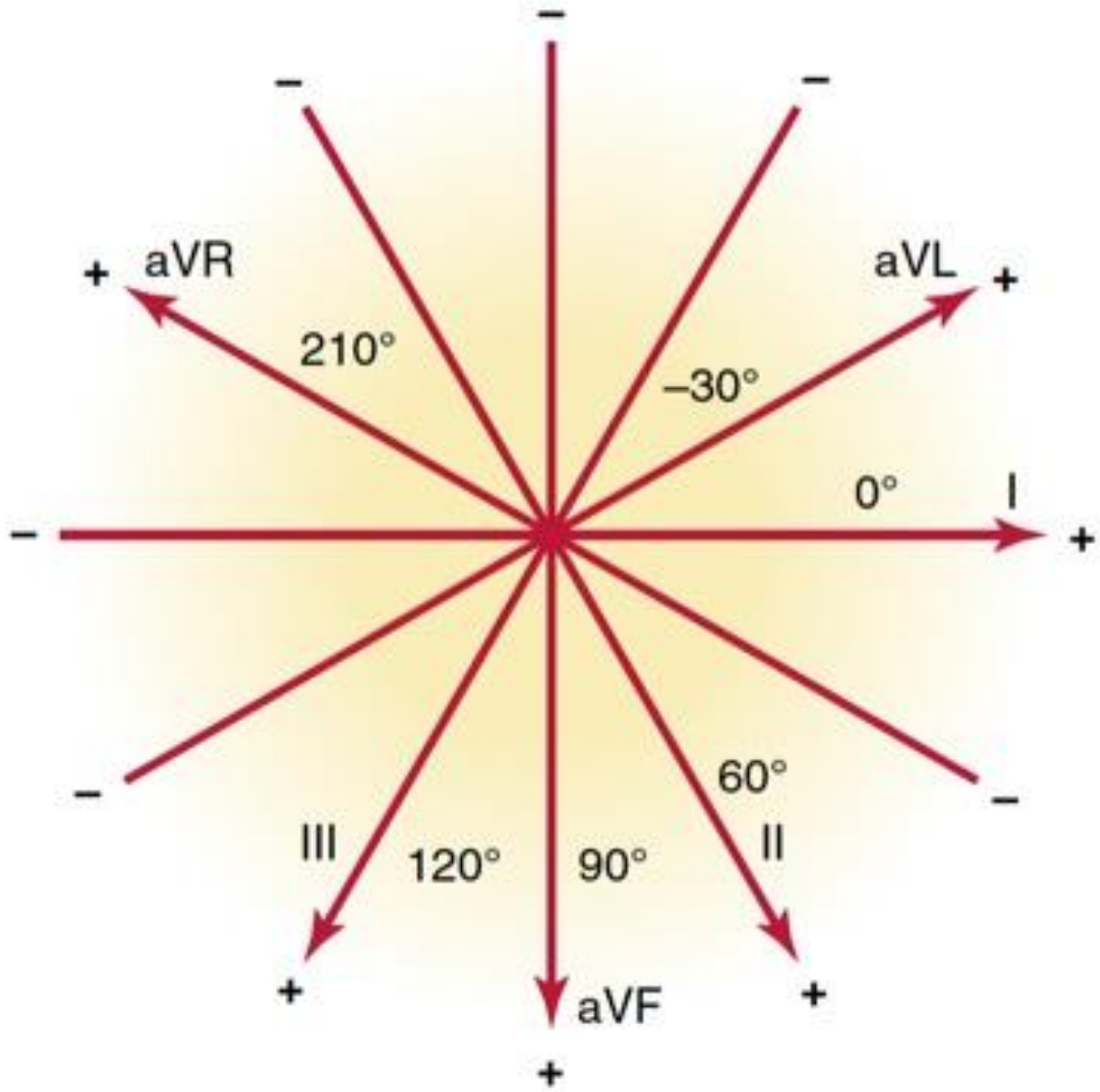
- Mathematically, this principle, called Einthoven’s law, holds true at any given instant while the three “standard” bipolar ECGs are being recorded.

- Note, however, that the positive and negative signs of the different leads must be observed when making this summation.

Einthoven's triangle




I
II
III


aVR
aVL
210°
–30°
0°
I
60°
III
120°
90°
II
aVF

Electrical Axis of QRS

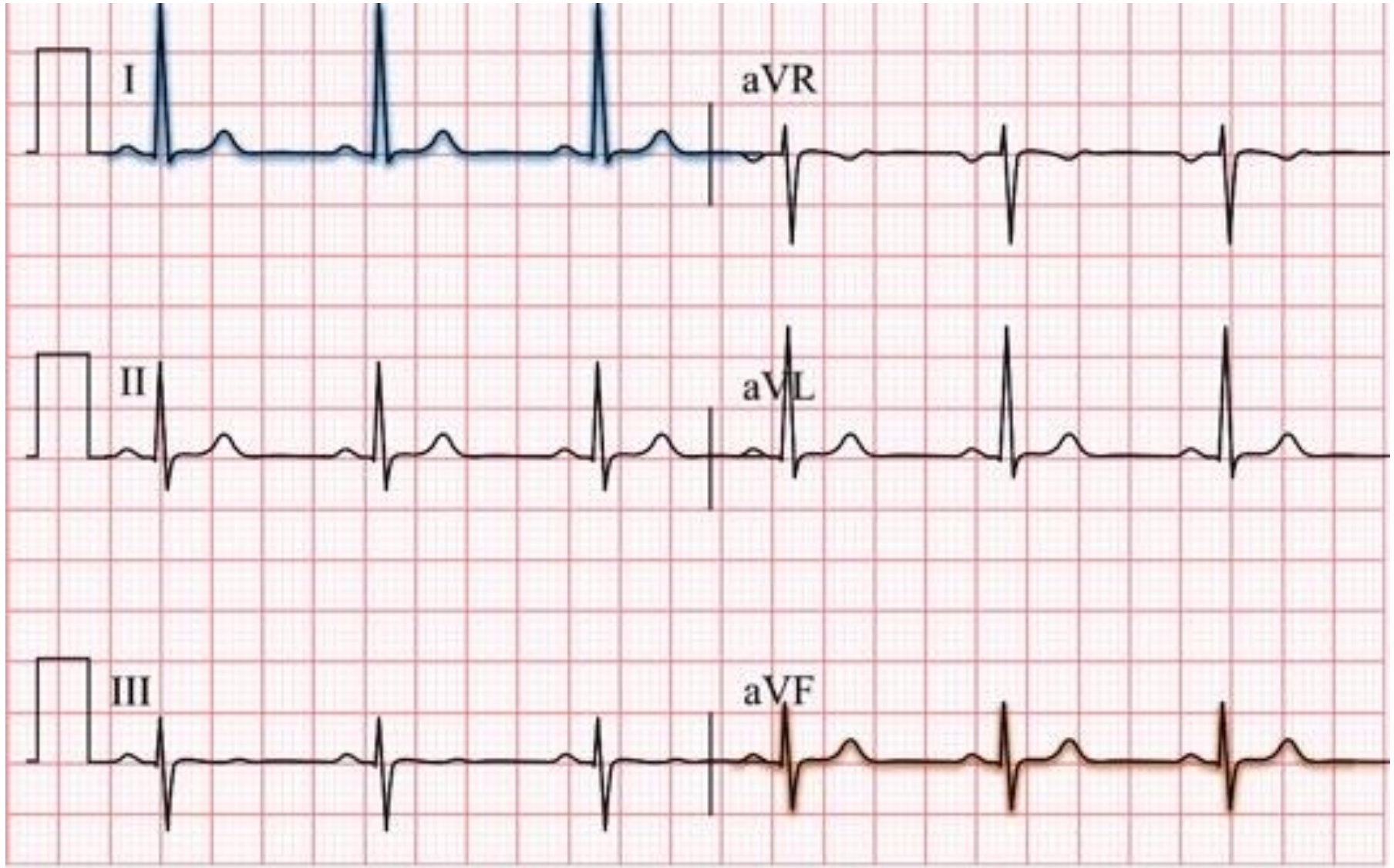
- The approximate average potential generated by the ventricles during depolarization is represented by the length of this mean QRS vector, and the mean electrical axis is represented by the direction of the mean vector.

- Thus, the orientation of the mean electrical axis of the normal ventricles is 59 degrees

Normal electrical axis of QRS

- Although the mean electrical axis of the ventricles averages about 59 degrees, this axis can swing, even in a normal heart, from about - 20 degrees to about +100 degrees (in other references -30 to +90).

- The causes of the normal variations are mainly anatomical differences in the Purkinje distribution system or in the musculature itself of different hearts.


I
aVR
II
aVL
III
aVF

BBB

- the lateral walls of the two ventricles depolarize at almost the same instant because both the left and right bundle branches of the Purkinje system transmit the cardiac impulse to the two ventricular walls at almost the same time.
- However, if only one of the major bundle branches is blocked, the cardiac impulse spreads through the normal ventricle before it spreads through the other ventricle. Therefore, depolarization of the two ventricles does not occur, even nearly at the same time, and the depolarization potentials do not neutralize each other. As a result, axis deviation occurs

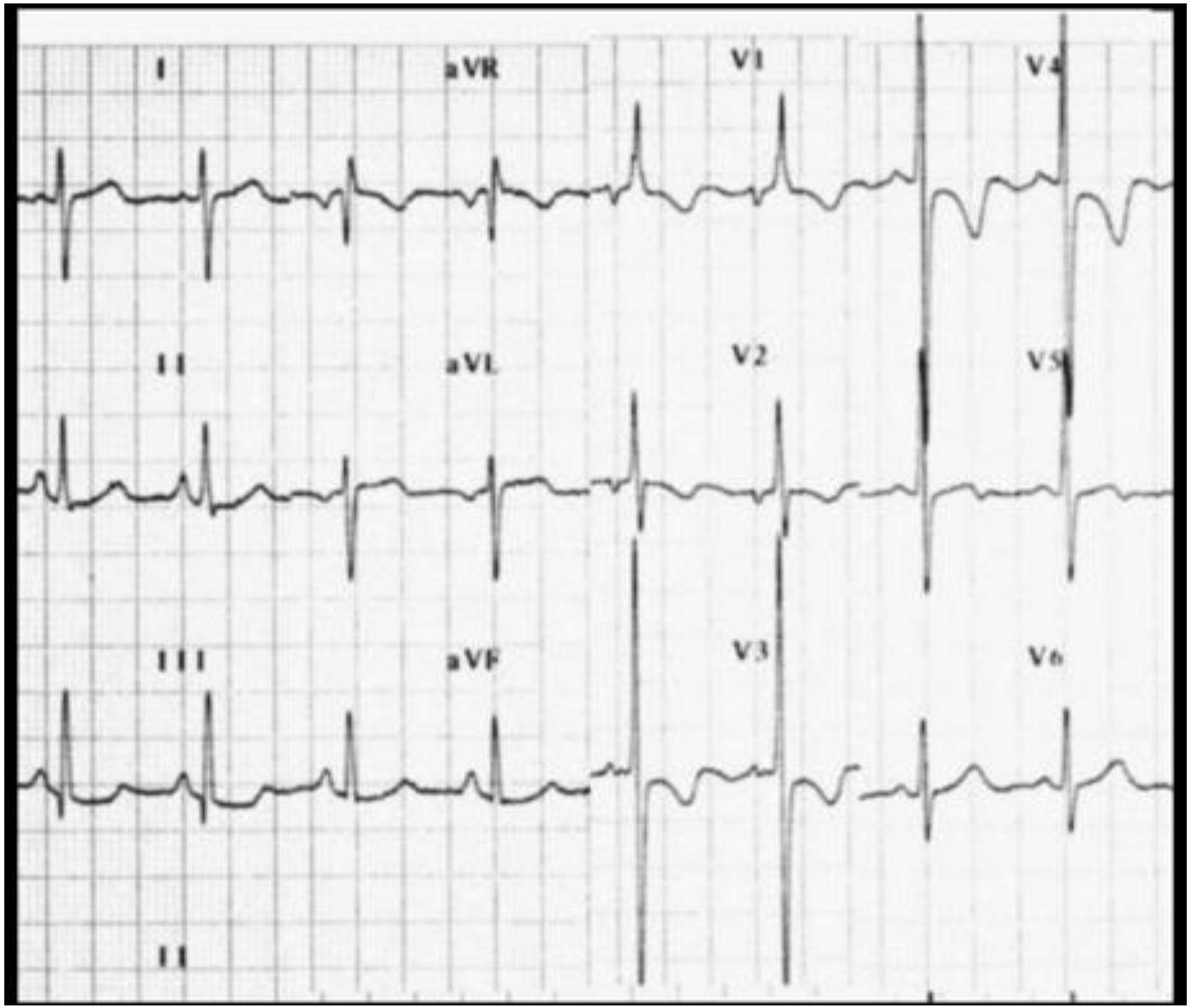
Ventricular hypertrophy

- When one ventricle hypertrophies greatly, the axis of the heart shifts toward the hypertrophied ventricle for two reasons.
- First, there is more muscle on the hypertrophied side of the heart than on the other side, which allows for the generation of greater electrical potential on that side.
- Second, more time is required for the depolarization wave to travel.

Right Axis Deviation

- (1) at the end of deep inspiration,
- (2) when a person stands up,
- (3) normally in tall thin people.

- (4) Right Ventricular Hypertrophy.
- (5) Right bundle branch block (RBBB)


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

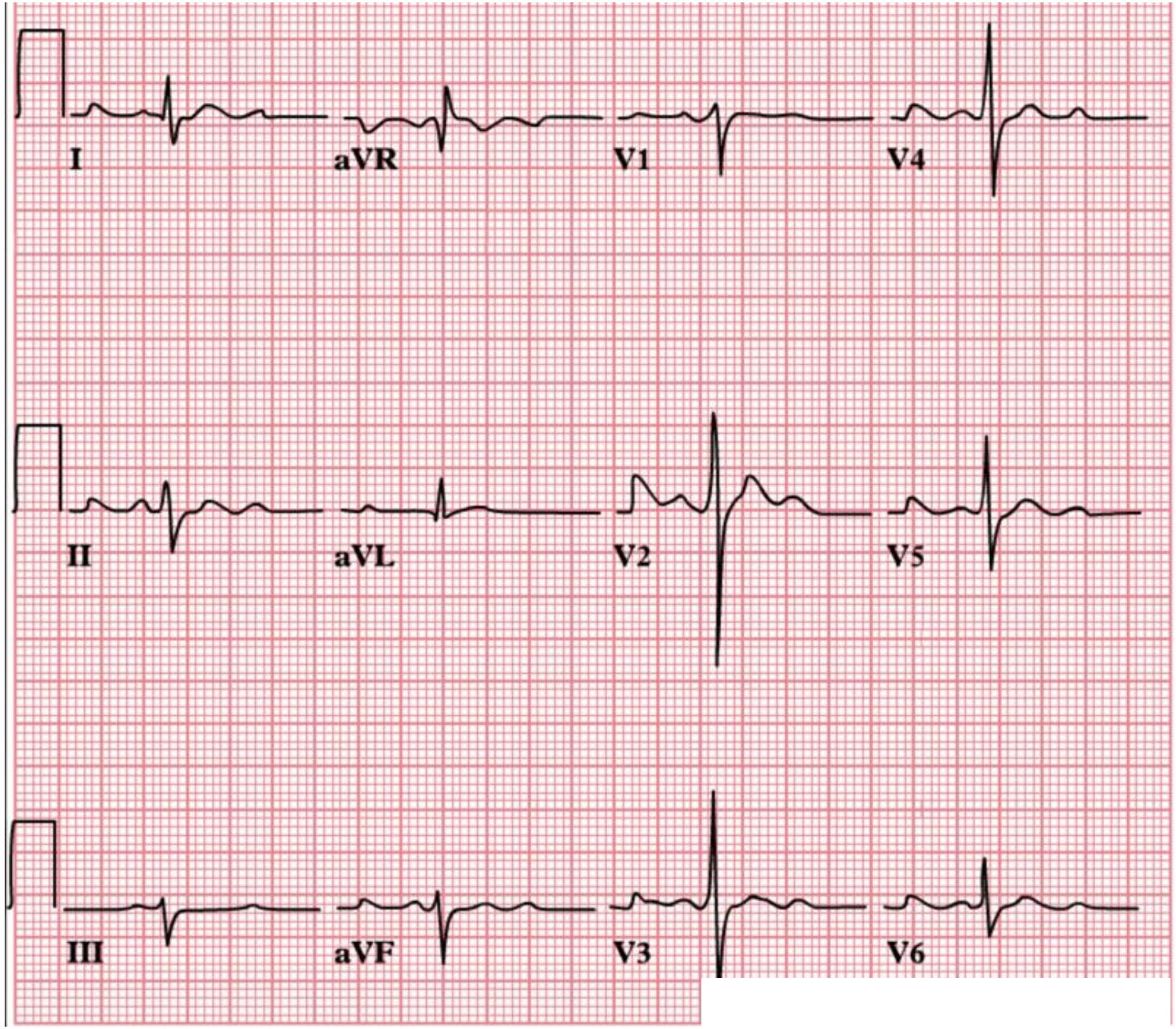
Left Axis Deviation

- (1) at the end of deep expiration,
- (2) when a person lies down, because the abdominal contents press upward against the diaphragm,
- (3) quite frequently in obese people, whose diaphragms normally press upward against the heart all the time as a result of increased visceral adiposity.

- (4) LVH
- (5) LBBB

LBBB

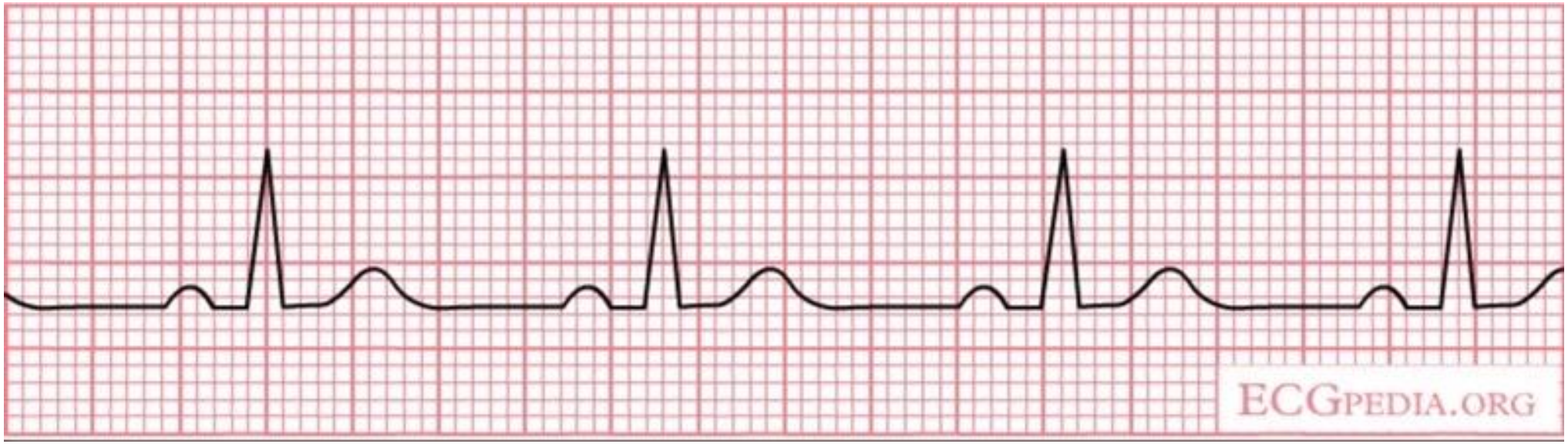
- Left axis deviation
- Prolonged QRS
- This extremely prolonged QRS complex differentiates bundle branch block from axis deviation caused by hypertrophy.

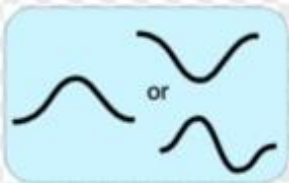
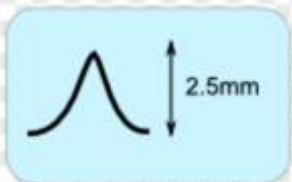
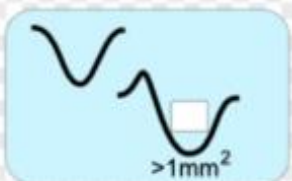

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Systematic approach to ECG

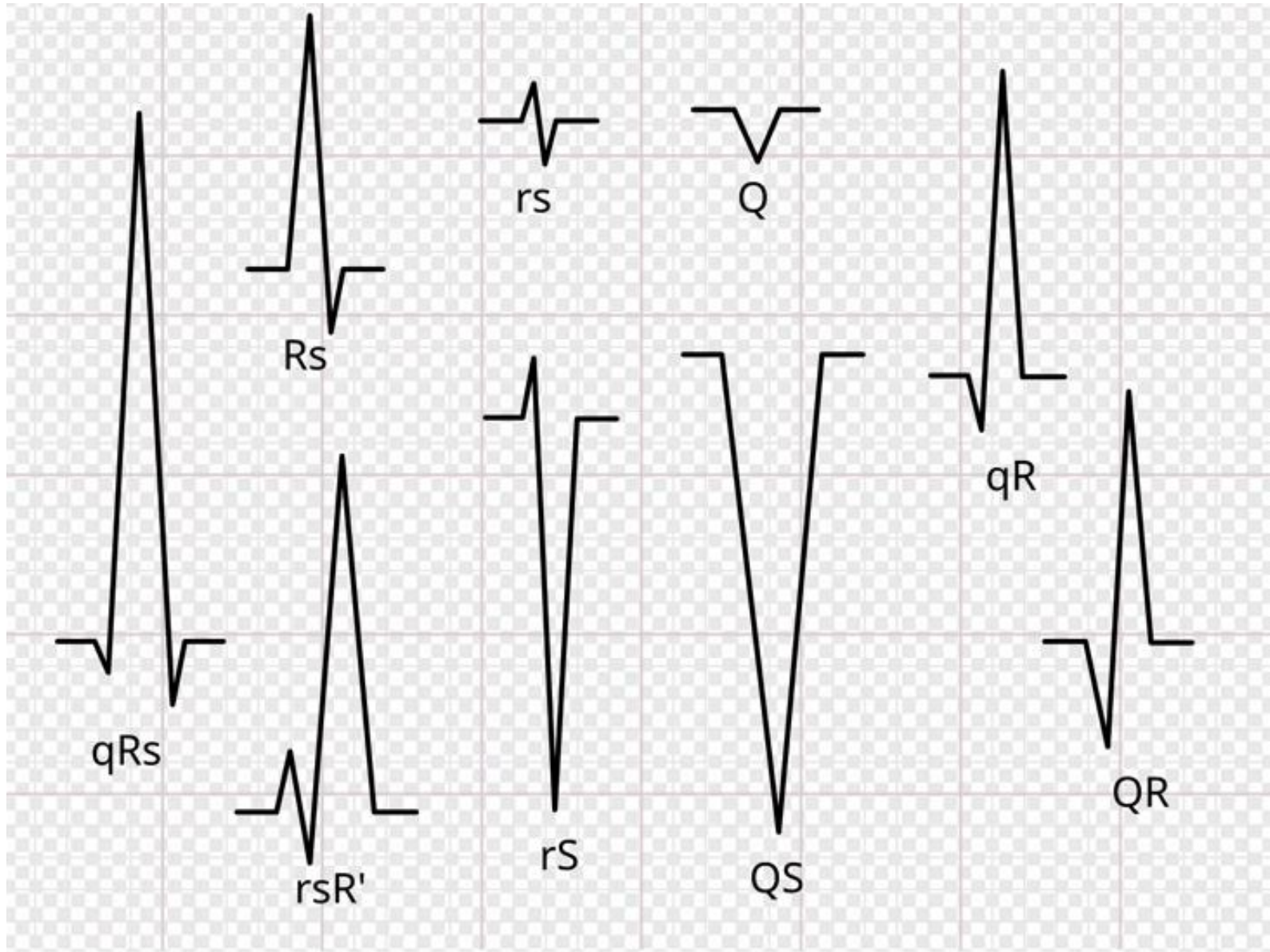
- 1. standardization: calibration, lead placement, artifact….
- 2. heart rate
- 3. rhythm
- 4. P wave
- 5. PR interval
- 6. QRS complex (time, voltage, axis, precordial R wave progression
- 7. QT interval
- 8. abnormal Q wave
- 9. ST segment
- 10. T wave
- 11. U wave

Heart rate and rhythm

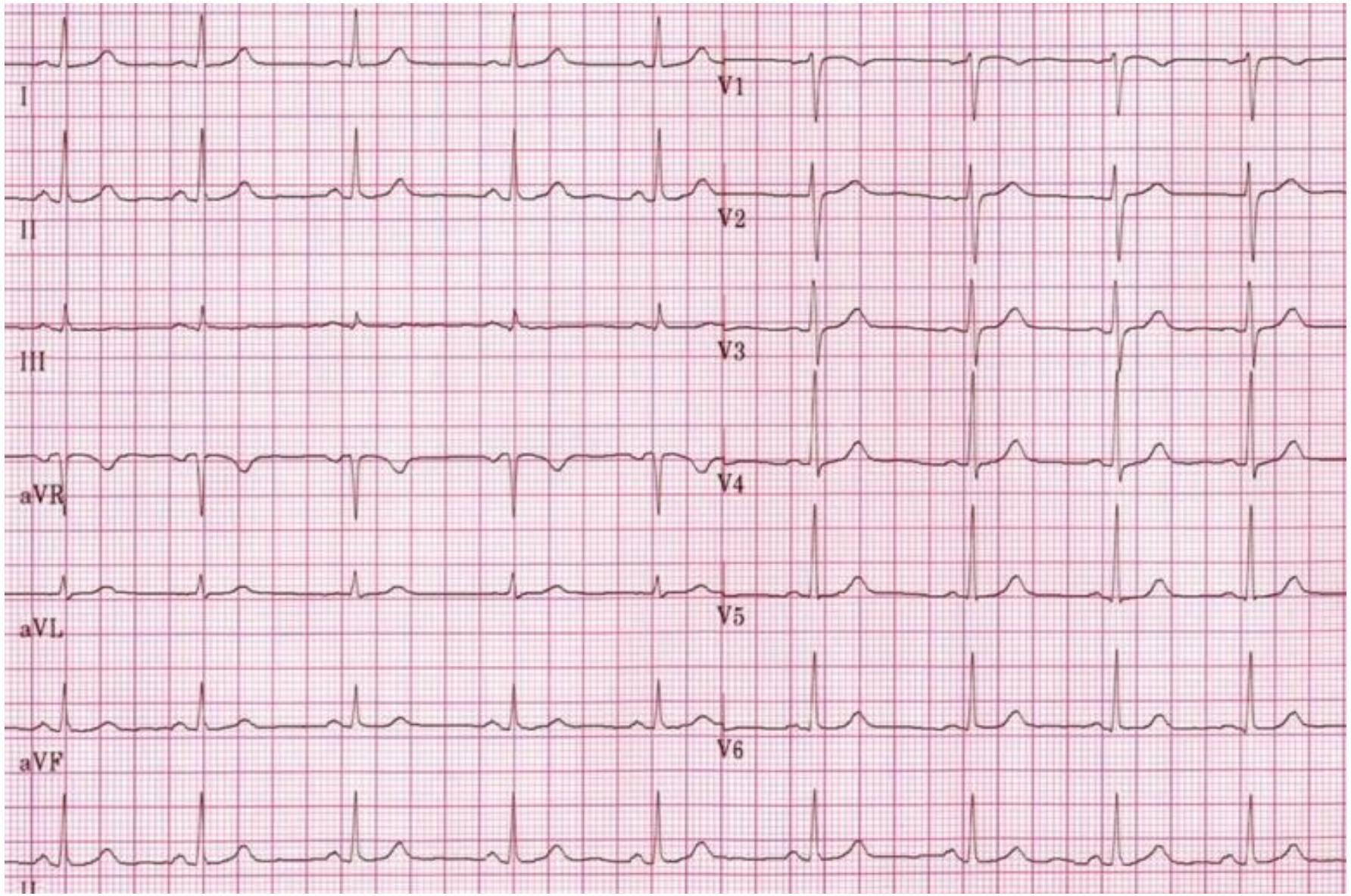


Condition	P Wave Morphology	
	Lead II	Lead V1
Normal Sinus Rhythm		
Right atrial enlargement (= **P** **P**ulmonale)		
Left Atrial Enlargement (=**P** **M**itrale)		

ECG PEDIA.ORG

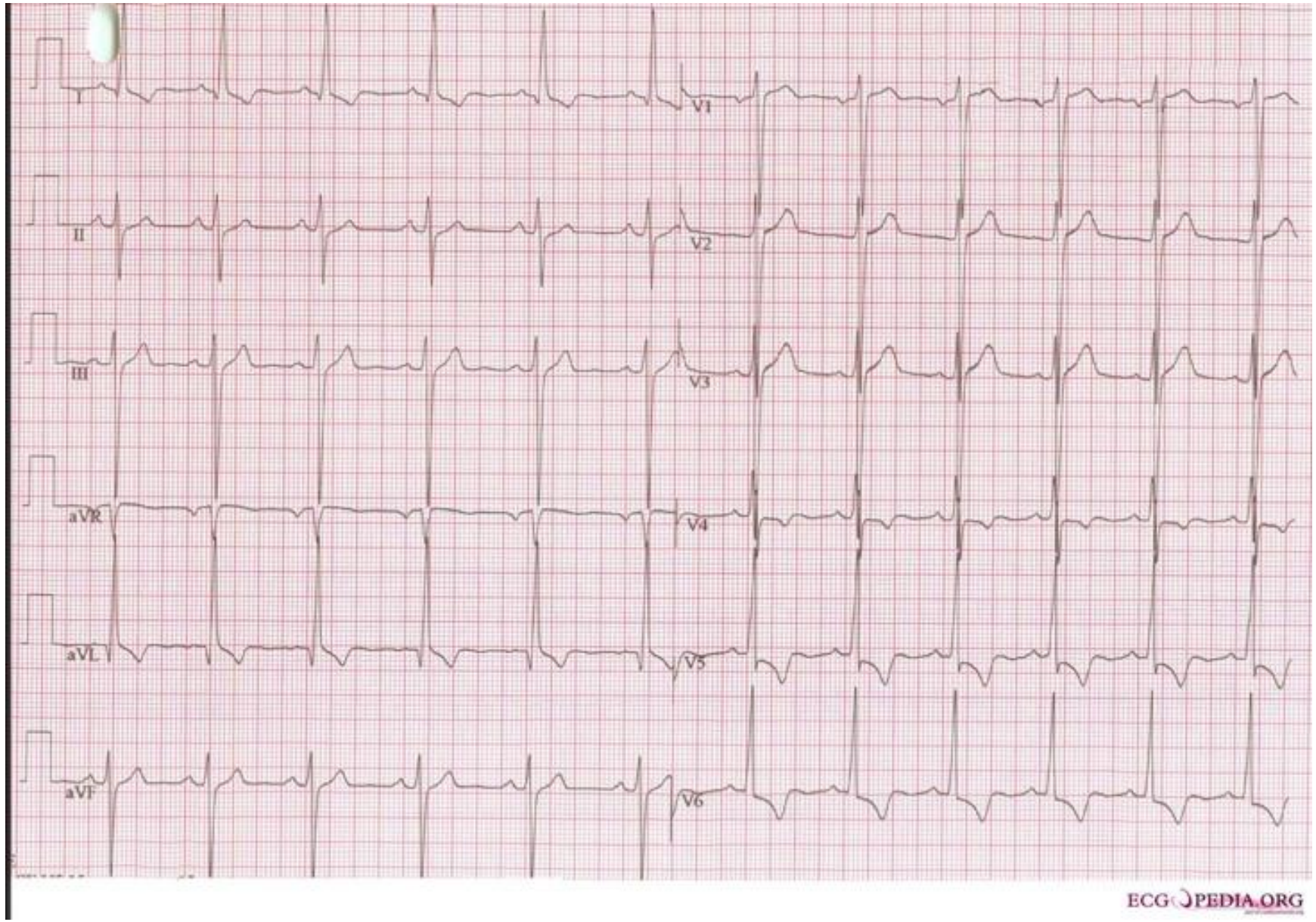

rs
Q
Rs
qR
qRs
rsR'
rS
QS
QR

Normal R propagation



High voltage QRS

- The cause of high-voltage QRS complexes is usually increased muscle mass of the heart, which ordinarily results from hypertrophy of the muscle in response to excessive load on one part of the heart or the other.
- For example, the left ventricle hypertrophies when a person has high systemic arterial blood pressure. The increased quantity of muscle generates increased electricity around the heart.


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
ECGPEDIA.ORG

Low voltage QRS

- One of the most common causes of decreased voltage of the QRS complex is a series of old myocardial infarctions with resultant diminished muscle mass. This condition also causes the depolarization wave to move through the ventricles slowly and prevents major portions of the heart from becoming massively depolarized all at once.
- Consequently, this condition causes some prolongation of the QRS complex, along with the decreased voltage.
- Infiltrative myocardial diseases cause low ECG voltage. For example, in cardiac amyloidosis.

Extra-cardiac causes of Low voltage QRS

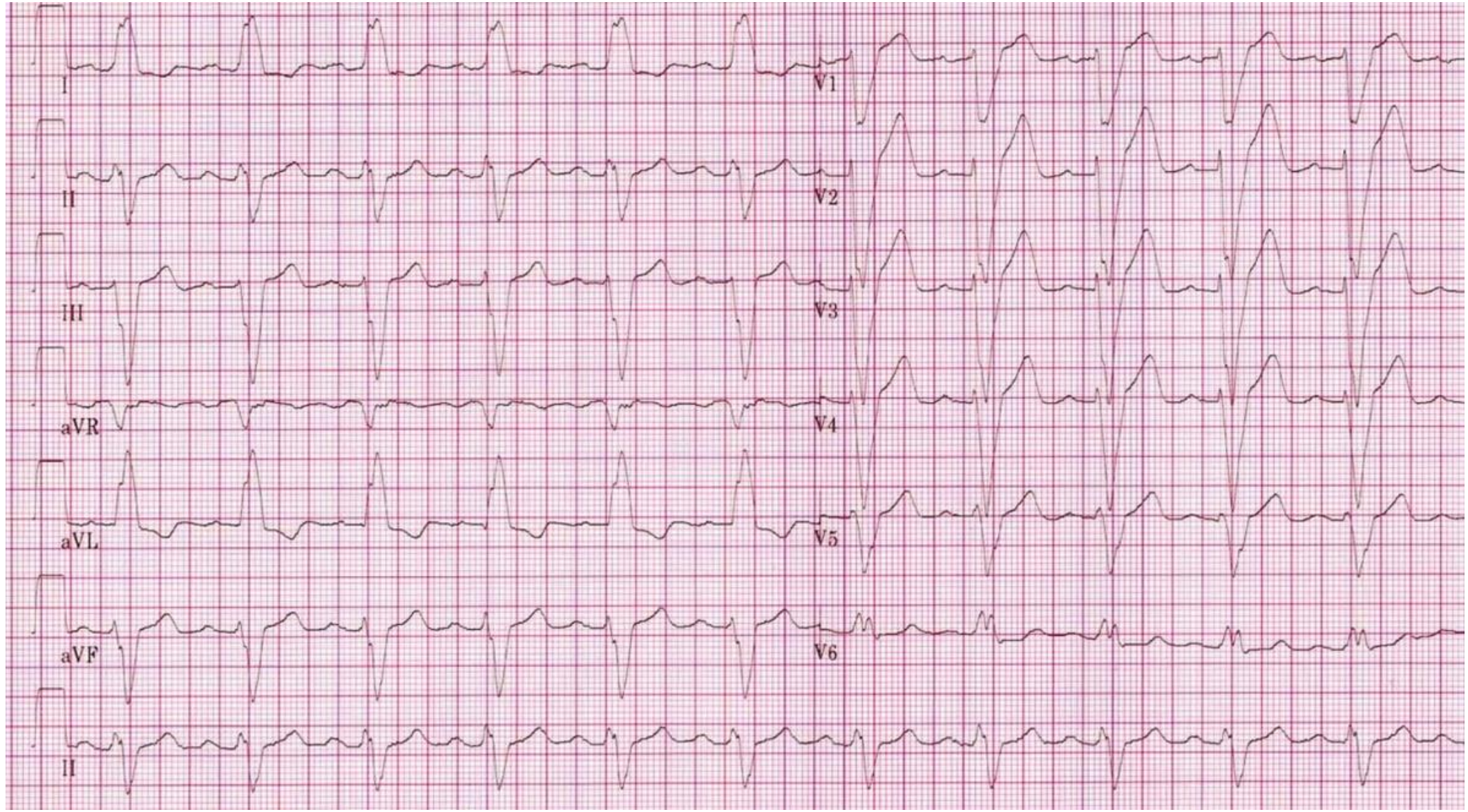
- One of the most important causes of decreased voltage in electrocardiographic leads is excessive fluid in the pericardium (pericardial effusion).
- Pleural effusion.
- Pulmonary emphysema.

Prolonged QRS

- The QRS complex lasts as long as depolarization continues to spread through the ventricles—that is, as long as part of the ventricles is depolarized and part is still polarized.
- Therefore, prolonged conduction of the impulse through the ventricles always causes a prolonged QRS complex.
- Such prolongation often occurs when one or both ventricles are hypertrophied or dilated because of the longer pathway that the impulse must then travel.

Prolonged QRS

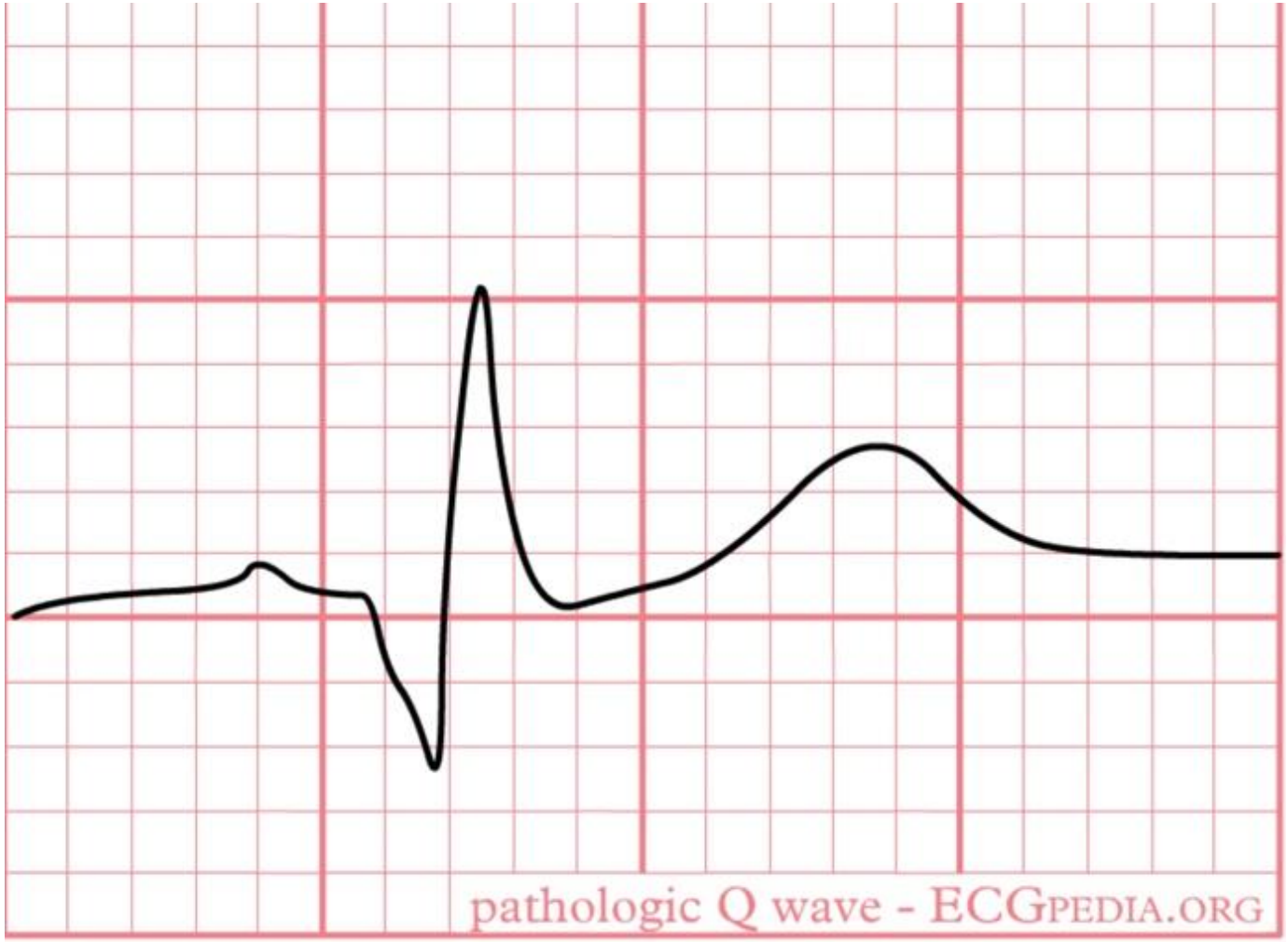
- When the Purkinje fibers are blocked, the cardiac impulse must then be conducted by the ventricular muscle instead of through the Purkinje system.
- This action decreases the velocity of impulse conduction to about one-third of normal, and causes prolonged QRS.
- In general, a QRS complex is considered to be abnormally long when it lasts more than 0.09 second.


I
II
III
aVR
aVL
aVF
II
V1
V2
V3
V4
V5
V6

Bizarre QRS

- Bizarre patterns of the QRS complex are usually caused by two conditions:
- (1) destruction of cardiac muscle in various areas throughout the ventricular system, with replacement of this muscle by scar tissue.
- (2) multiple small local blocks in the conduction of impulses at many points in the Purkinje system.
- As a result, cardiac impulse conduction becomes irregular, causing rapid shifts in voltages and axis deviations.
- This irregularity often causes double or even triple peaks in some of the electrocardiographic leads

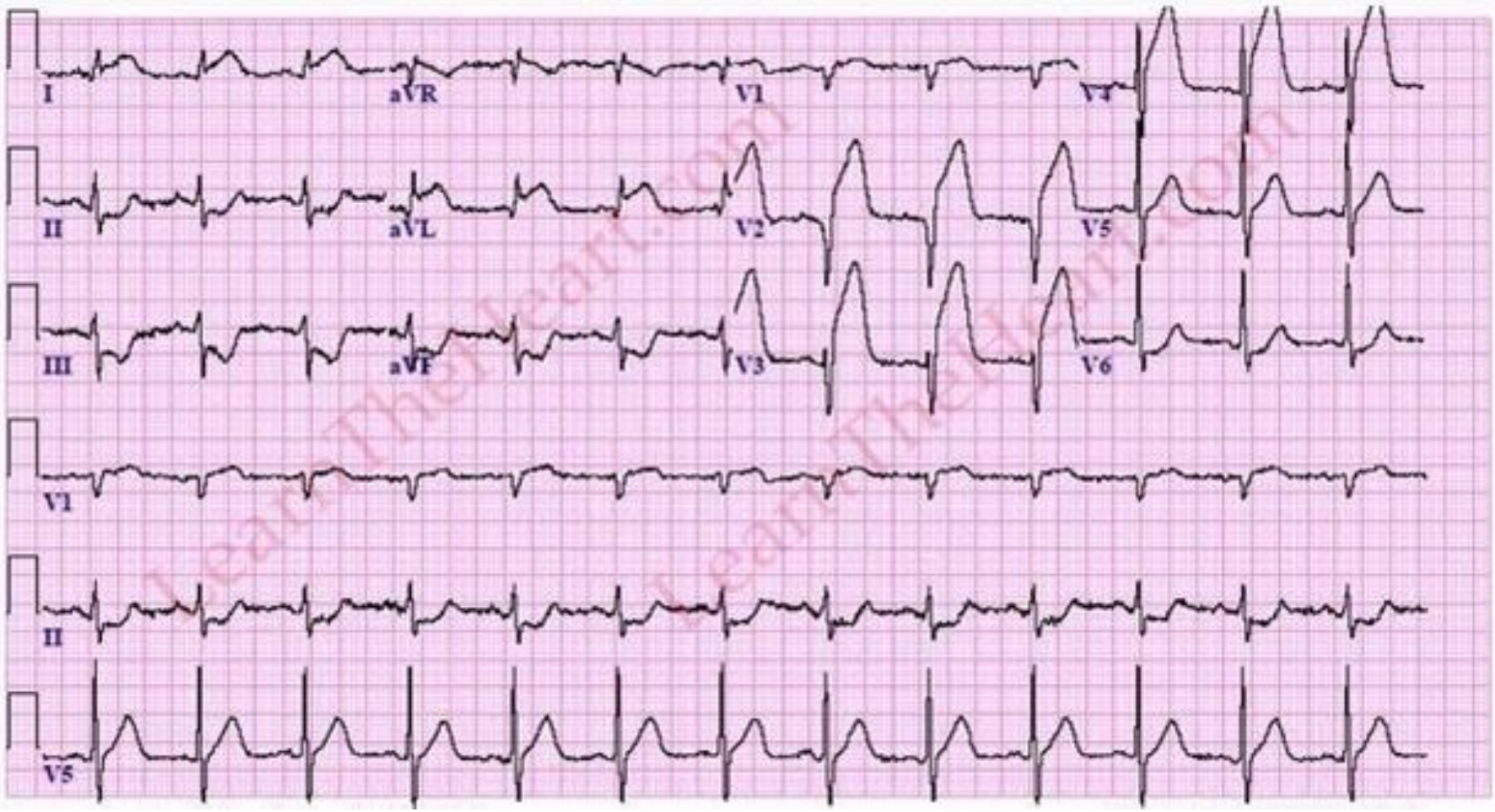
pathological Q wave



Myocardial ischemia

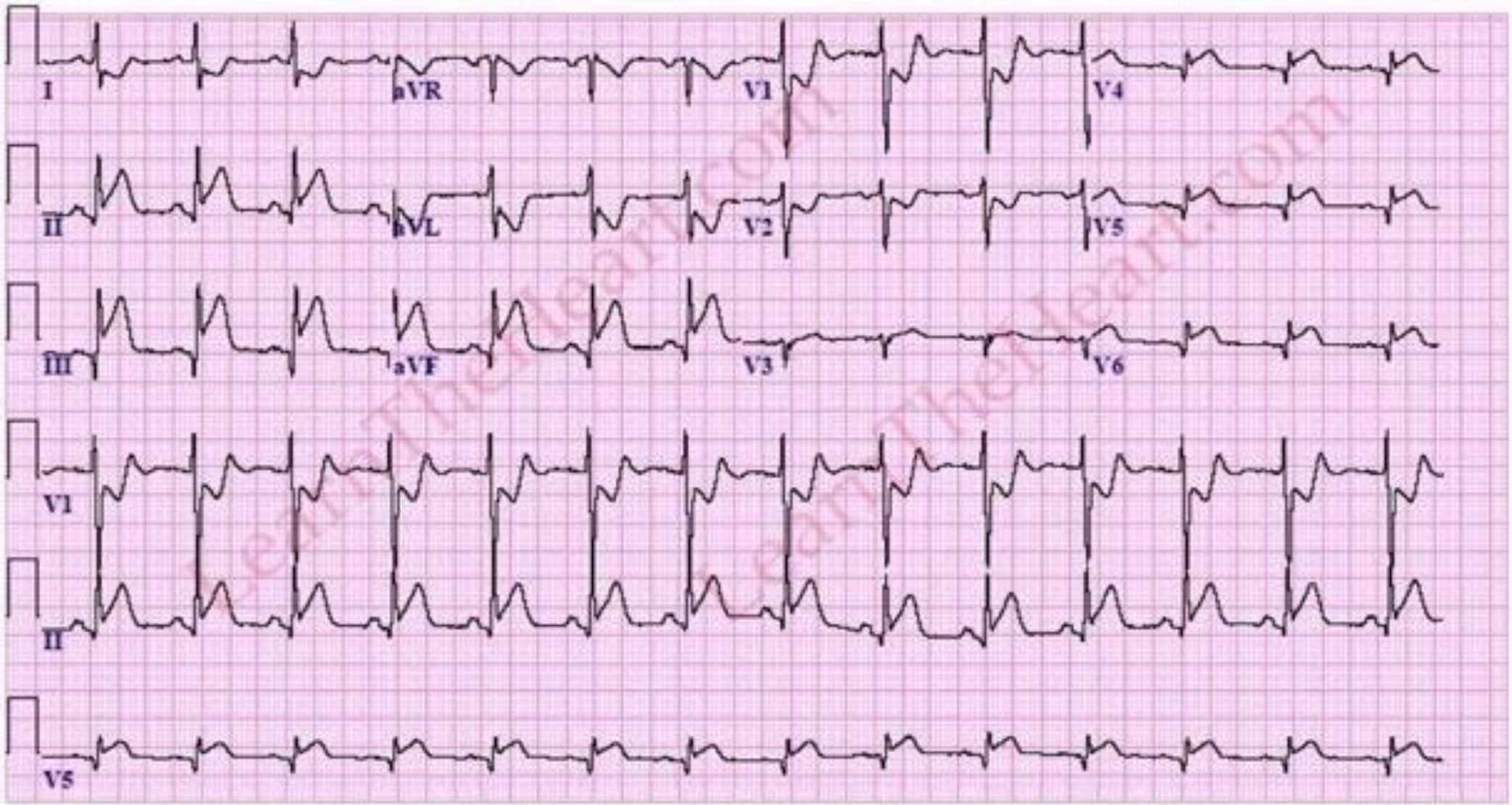
- When the ST elevation occurs as the earliest sign of acute MI, they are typically followed by T inversion (often correlated to prolonged repolarization and QT lengthening) within hours to days, followed by Q waves in the same lead distribution.

- Depending on the severity and duration of ischemia, ST elevation may resolve completely within minutes or followed by T wave inversion for days.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

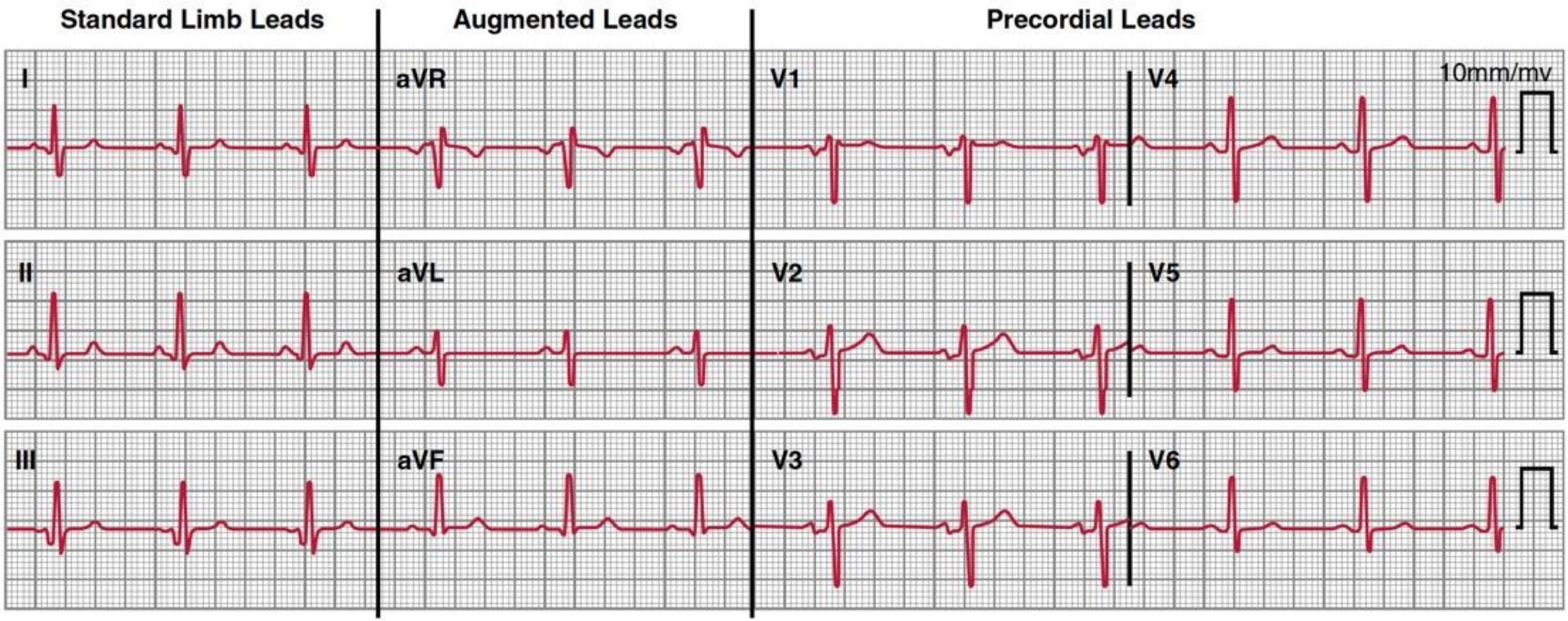
Myocardial ischemia

- Acute transmural anterior wall (including lateral and apical) ischemia is reflected by ST elevation or tall T wave in one or more of leads V1 to V6, and lead I and aVL.
- Inferior wall ischemia produces changes in lead II, III, and aVF.
- Posterior wall ischemia may be indirectly recognized by reciprocal ST depression in lead V1-V3.
- Right ventricular ischemia produces ST elevation in right sided chest leads.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

ECG values

- Heart rate: if regular rhythm: 300/ no. of large squares between 2 consecutive Rs.
- PR interval normal 0.12- 0.20 s : AV delay.
- QRS normal < = 0.10 s: ventricular depolarization.
- QT interval normal < =0.44 s: ventricular depolarization and repolarization, varies inversely with heart rate, so corrected: QTc= QT/ R-R square root.


Standard Limb Leads
Augmented Leads
Precordial Leads
I
aVR
V1
V4
10mm/mv
II
aVL
V2
V5
III
aVF
V3
V6

Thank you