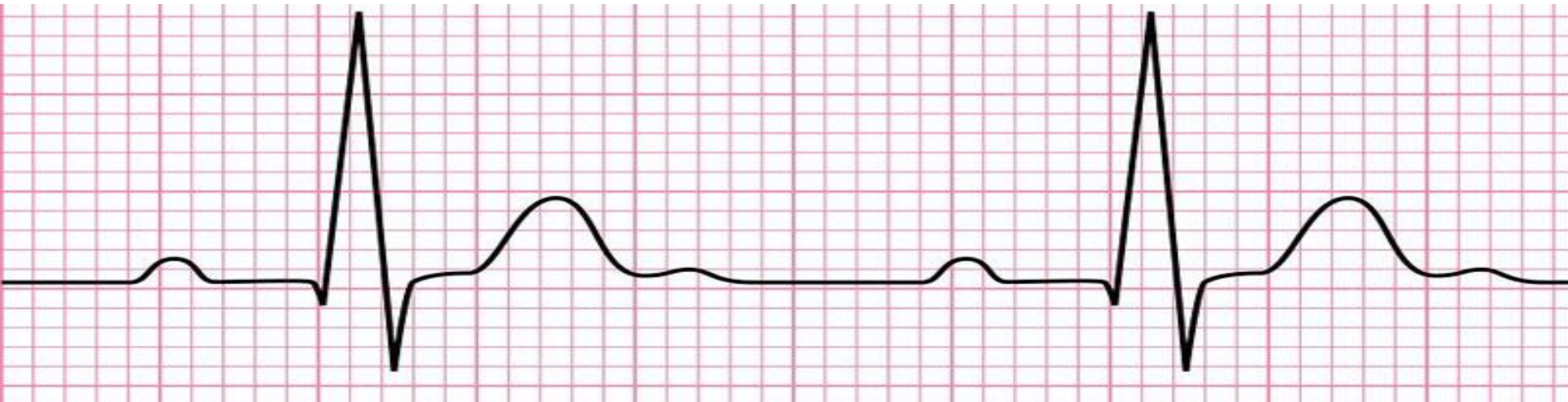
THE ECG

Dr. Tamara Alqudah



ECG changes in IHD

ECG is very useful for diagnosing IHD and locating the affected areas. But the diagnosis requires more than an ECG.

Angina pectoris

Myocardial infarction

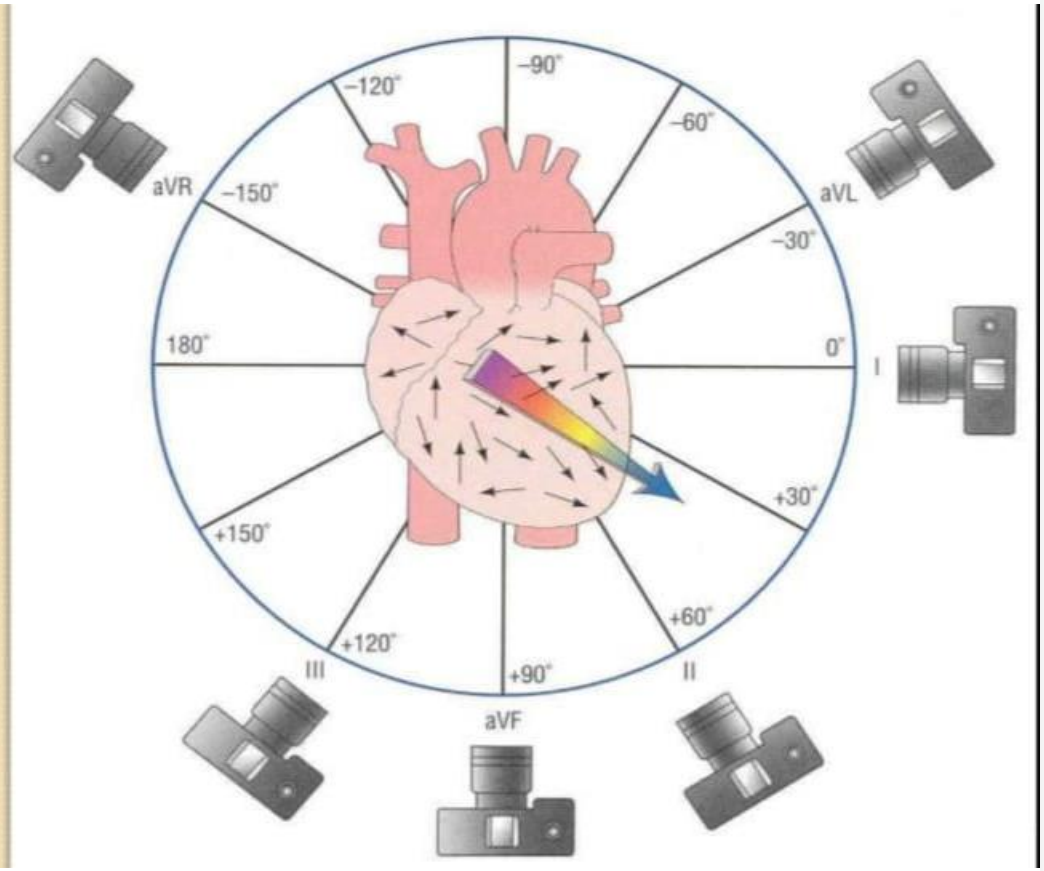
- ST segment elevation myocardial infarction (STEMI)
- Non-ST segment elevation myocardial infarction (NSTMI)

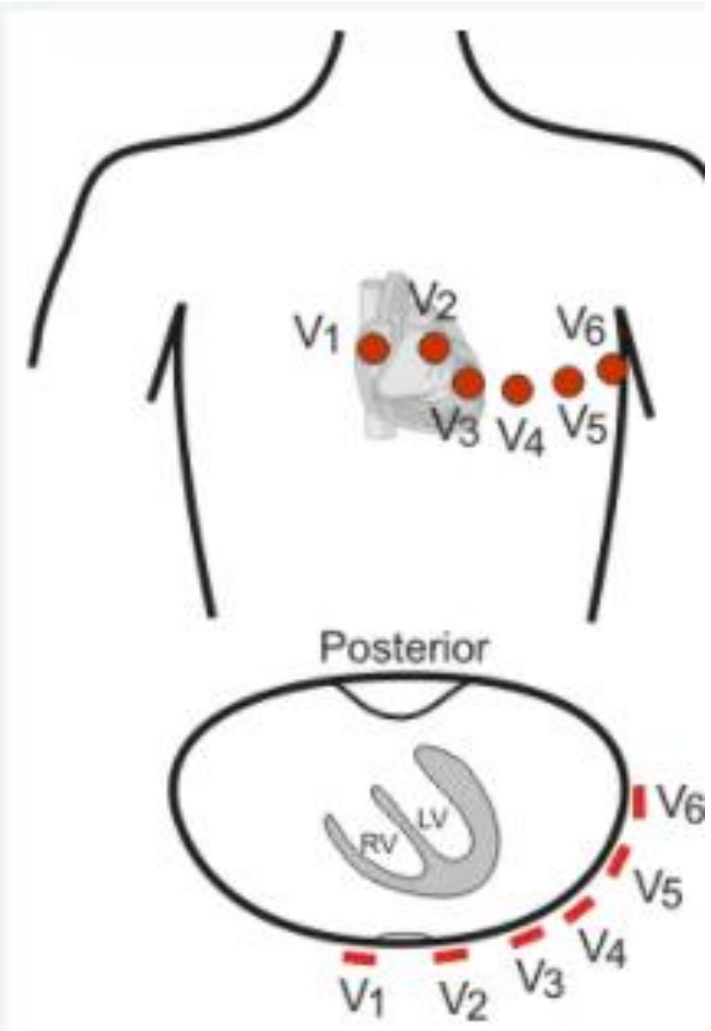
ST segment changes

- One important ECG feature is ST segment depression or elevation

- To be considered significant , more than 1 mm of ST segment elevation or depression in at least two contiguous limb leads (e.g. I and VL; III and VF), or more than 2 mm of ST segment elevation or depression in at least two contiguous chest leads

- ST segment changes occur in the leads corresponding to the part of the heart that is damaged:
- Leads V1-V4 with anteroseptal wall infarction
- Lead aVL, I, V5 & V6 with lateral wall infarction
- Leads II, III and aVF with inferior wall infarction.


aVR
−120°
−90°
−60°
aVL
−150°
−30°
180°
0°
I
+150°
+30°
+120°
+60°
III
+90°
II
aVF


V1
V2
V3
V4
V5
V6
Posterior
RV
LV
V6
V5
V4
V3
V1
V2

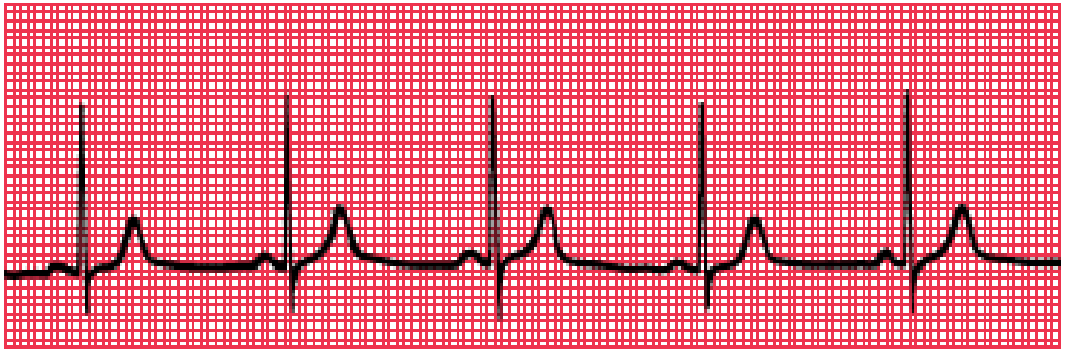
Angina pectoris

- With angina ECG changes are noticed while the patient is in pain, once the pain has resolved the ECG returns to normal.
- Depression of the ST segment, is usually a sign of ischaemia.
- When the ECG is normal at rest, ST segment depression may be induced by making the patient exercise, this test is called stress ECG

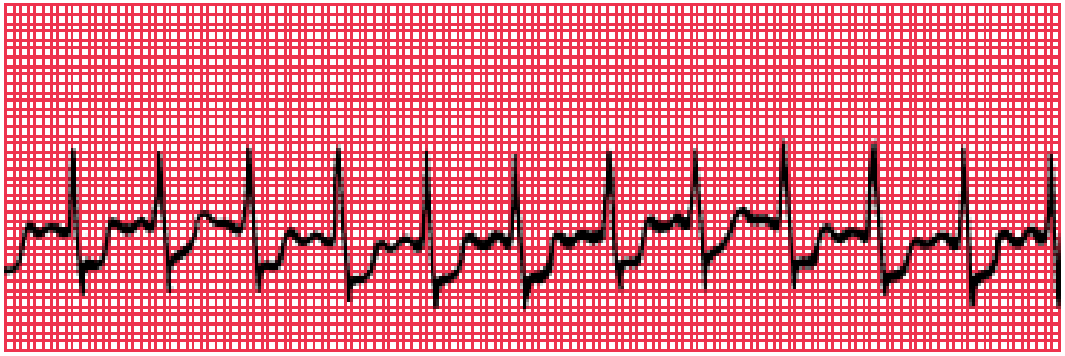
Fig. 4.14

Exercise-induced ischaemic changes

Rest:



Exercise:



Note

- In the upper (normal) trace, the heart rate is 55/min and the ST segments are isoelectric
- In the lower trace, the heart rate is 125/min and the ST segments are horizontally depressed

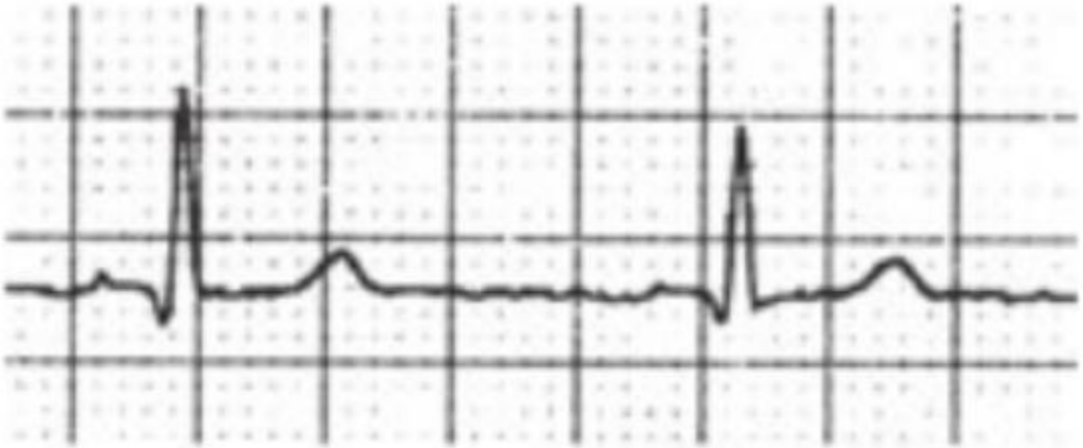
ST segment elevation myocardial infarction (STEMI)

- The earliest ECG changes are ST segment elevations and hyperacute T waves.

- Large and transmural MI might produce permanent pathological Q waves within the first day of infarction.
- Q waves are considered pathological if:
  - > 0.04 sec in duration
  - > 2 mm deep

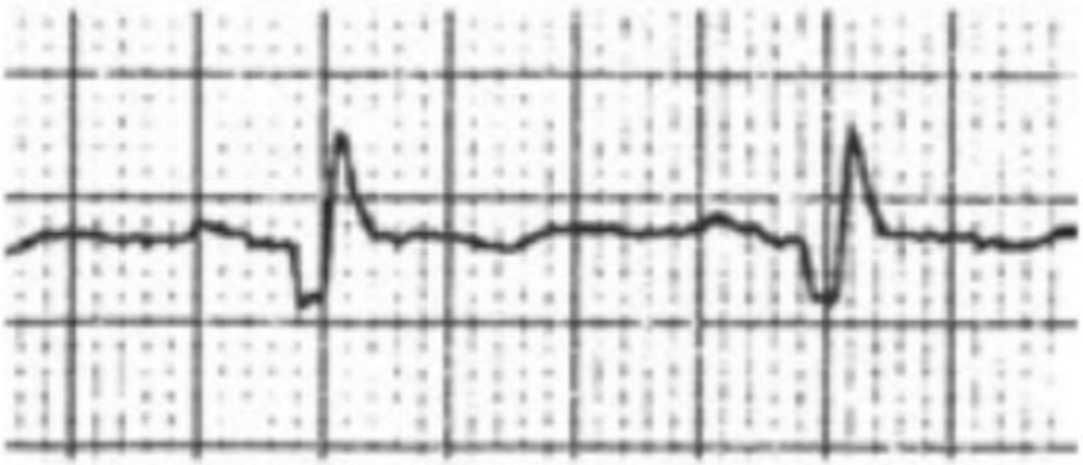
- Hours or days later ST segments return to the baseline and deep T wave inversions appear in the leads that previously showed ST elevations.

A



Normal Q Waves

B



Pathologic Q Waves

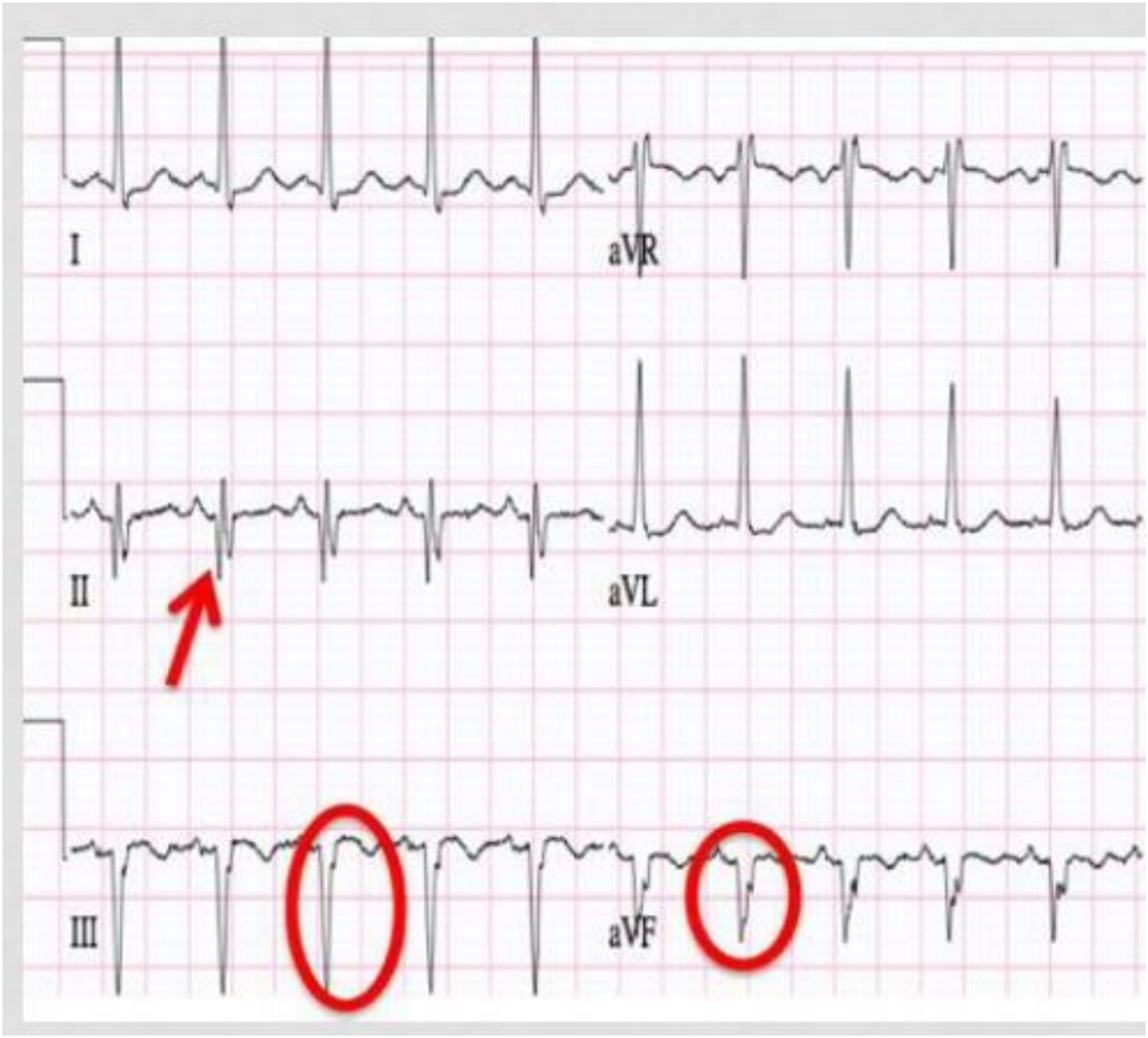
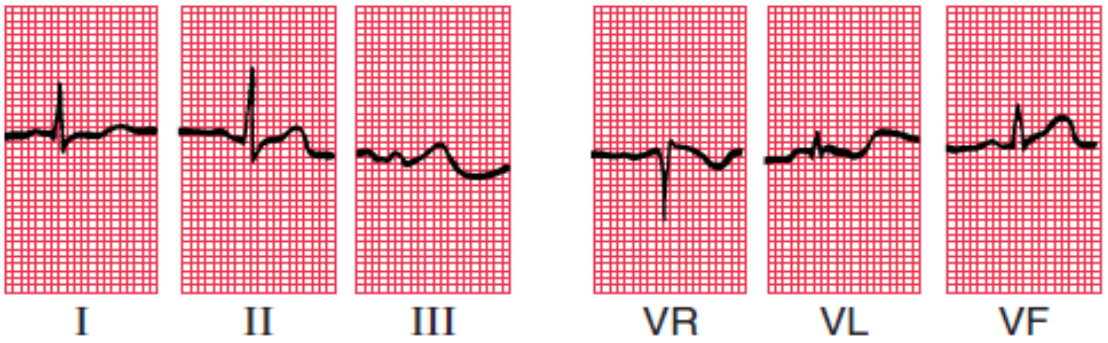

I
aVR
II
aVL
III
aVF

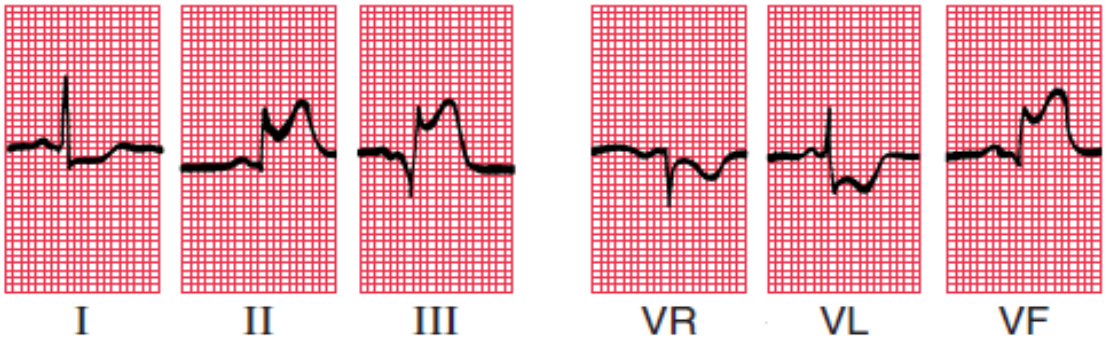
Fig. 4.15

Development of inferior infarction

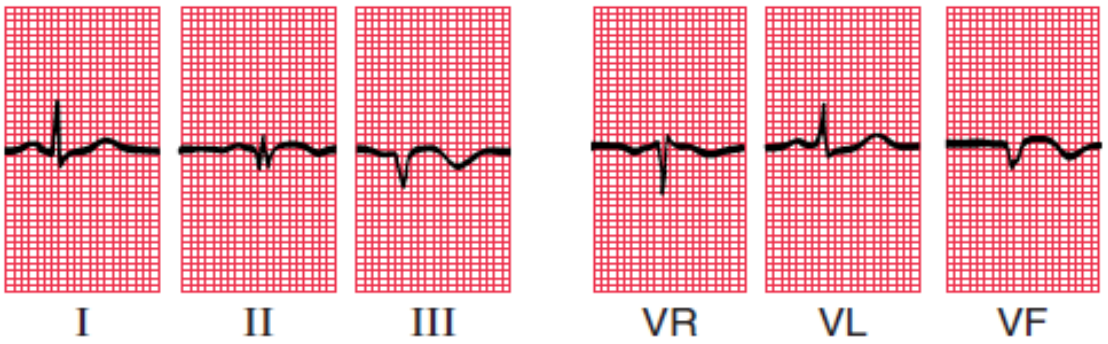
1 h after onset of pain:



6 h after onset of pain:



24 h after onset of pain:

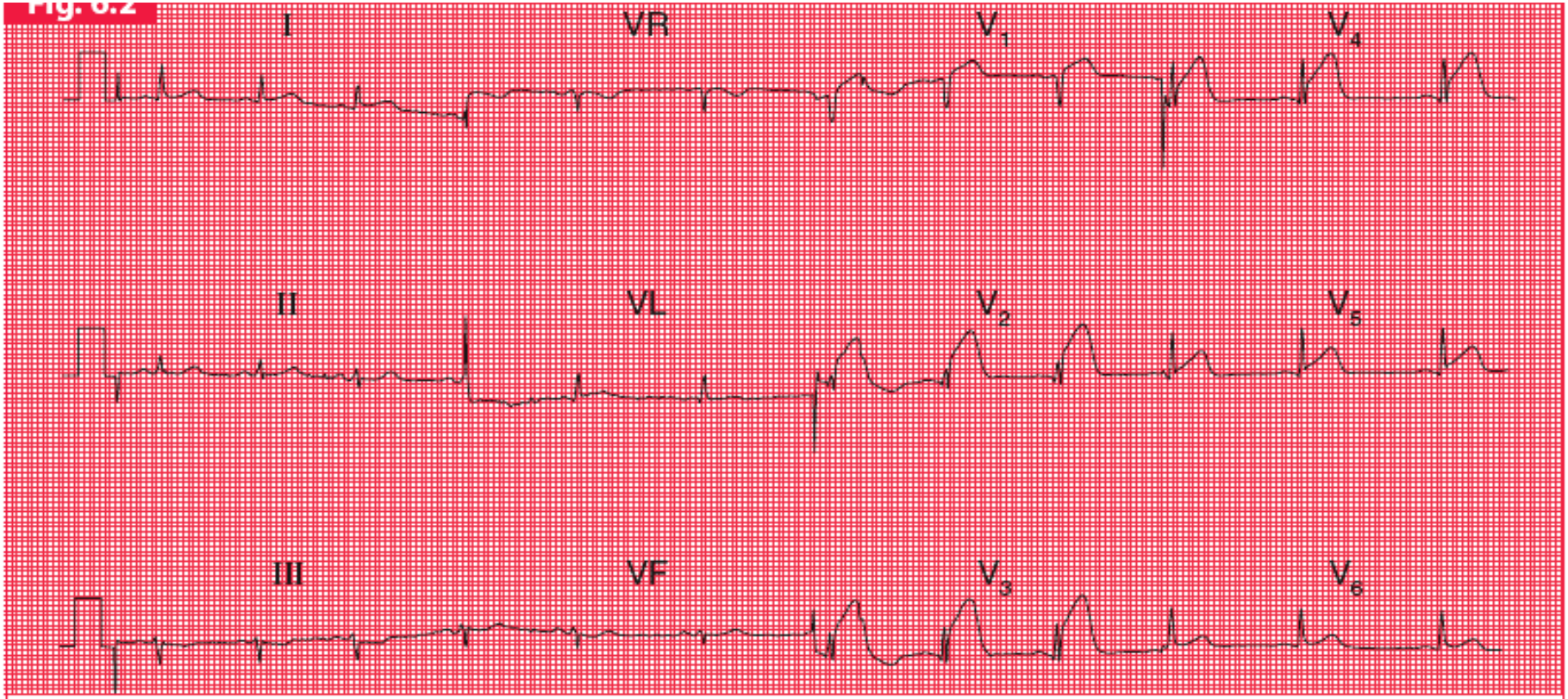


Note

- Three ECGs have been recorded over 24 h, and have been arranged horizontally
- Sinus rhythm with a normal cardiac axis in all three ECGs
- The first record is essentially normal
- 6 h after the onset of pain, the ST segments have risen in leads II, III and VF, and the ST segment is depressed in leads I, VR and VL. A Q wave has developed in lead III
- 24 h after the onset of pain, a small Q wave has appeared in lead II, and more obvious Q waves can be seen in leads III and VF. The ST segments have returned to the baseline, and the T waves are now inverted in leads III and VF

Non-ST segment elevation myocardial infarction (NSTMI)

- Associated with ST segment depression and T wave inversion in the leads corresponding to the site of myocardial damage



ST segment elevation in acute anterior ST segment elevation myocardial infarction

Note

- Sinus rhythm, rate 75/min
- Normal axis
- Normal QRS complexes
- ST segments elevated in leads V_1–V_5
- Normal T waves

Arrhythmias

- An abnormal heart rhythm.
- Might be associated with tachycardia or bradycardia
- The causes of the cardiac arrhythmias are usually one or a combination of the following abnormalities in the rhythmicity-conduction system of the heart:

1. Abnormal rhythmicity of the pacemaker
2. Shift of the pacemaker from the sinus node to another place in the heart
3. Blocks at different points in the spread of the impulse through the heart
4. Abnormal pathways of impulse transmission through the heart
5. Spontaneous generation of spurious impulses in almost any part of the heart

Types of arrhythmias

- Abnormal sinus rhythms
  - Sinus tachycardia
  - Sinus Bradycardia
  - Sinus arrhythmia
- Atrioventricular block
  - First degree heart block
  - Second degree heart block
  - Third degree heart block
  - Stokes-Adams Syndrome
- Ventricular fibrillation
- Atrial fibrillation
- Atrial flutter

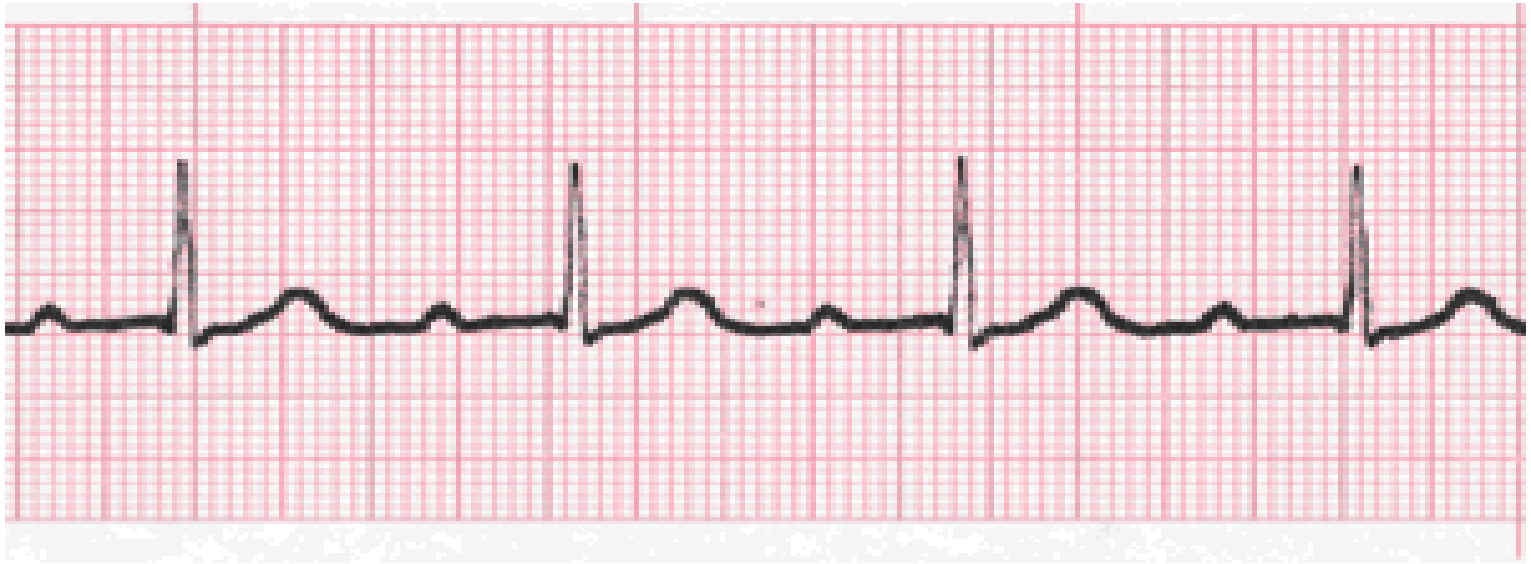
Atrioventricular (AV) Block

- Results from conditions that can either decrease the rate of impulse conduction in the AV bundle or block the impulse entirely:

1. Ischemia of the A-V node or A-V bundle fibers
2. Compression of the A-V bundle by scar tissue or by calcified portions of the heart.
3. Inflammation of the A-V node or A-V bundle
4. Extreme stimulation of the heart by the vagus nerves

First Degree Heart Block

- When the PR interval increases to greater than 0.2 sec, the P-R interval is said to be prolonged and the patient is said to have first-degree heart block.
- Caused by coronary artery disease, acute rheumatic carditis, digoxin toxicity or electrolyte disturbances.



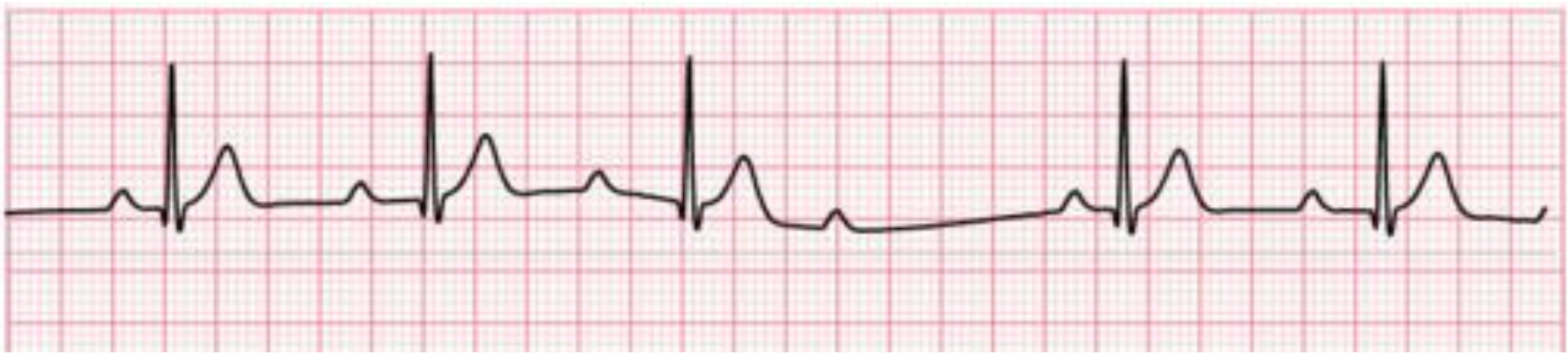
Second Degree Block

- Happens when conduction through the A-V bundle is slowed enough to increase the P-R interval to 0.25 to 0.45 second
- The action potential is sometimes strong enough to pass through the bundle into the ventricles and sometimes not strong enough to do so.
- So occasionally there will be “dropped beats” ; an atrial P wave but no QRS-T wave.
- There are different types of second-degree A-V block:

1. Wenckebach periodicity
2. Fixed ratio blocks

Wenckebach periodicity

- Progressive lengthening of the PR interval and then failure of conduction of an atrial beat, followed by a conducted beat with a shorter PR interval and then a repetition of this cycle.
- A type I block is almost always caused by abnormality of the A-V node. In most cases no specific treatment is needed.

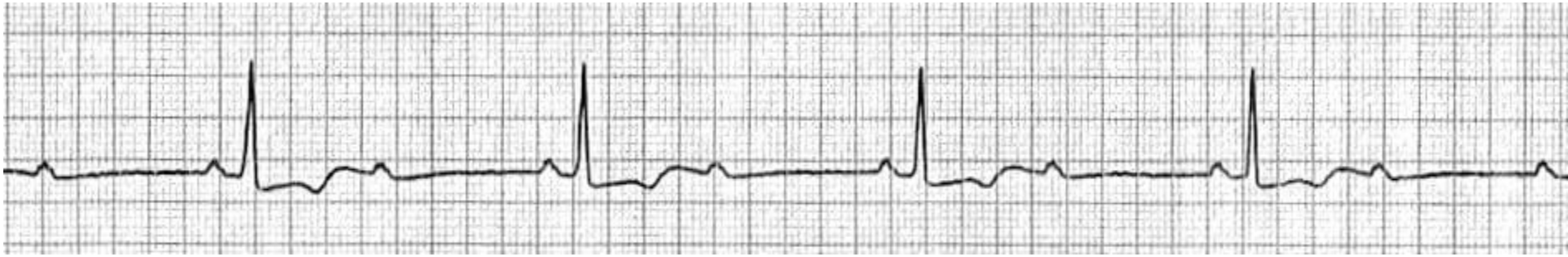


Fixed ratio blocks

- There is usually a fixed number of non-conducted P waves for every QRS complex.
- **Alternate conducted and non-conducted atrial beats(2:1)**

At other times, rhythms of 3 : 2 or 3 : 1 may develop.

- Caused by an abnormality of the bundle of His-Purkinje system



Third degree block

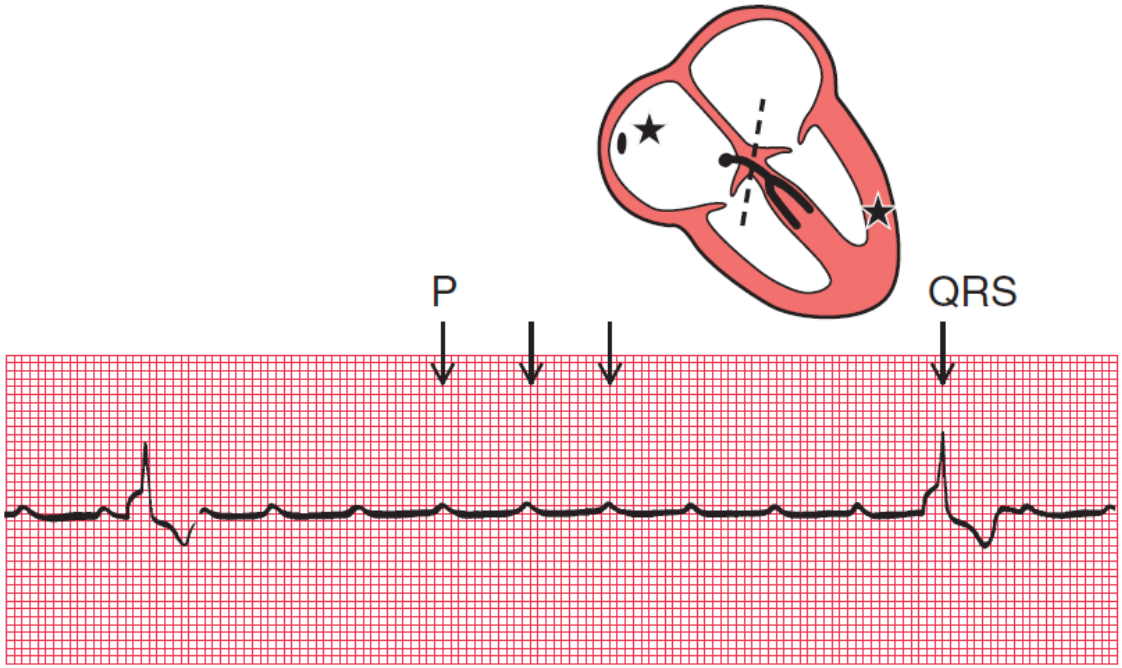
- Occurs with complete block of the impulse from the atria into the ventricles.

- The ventricles spontaneously establish their own signal, usually originating in the AV node, AV bundle or purkinji fibers. Therefore, the P waves become dissociated from the QRS-T complexes.
- Third-degree block is characterized by:

1. Regular P-P interval
2. Regular R-R interval
3. Lack of an apparent relationship between the P waves and QRS complexes
4. Atrial rate is higher than ventricular rate

- Occur as an acute phenomenon in patients with myocardial infarction or it may be chronic, usually due to fibrosis around the bundle of His.

Complete heart block



Note

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

The A-V nodal fibers, when not stimulated by SA node discharge at an intrinsic rhythmical rate of 40 to 60 times per minute, and the Purkinje fibers discharge at a rate somewhere between 15 and 40 times per minute

Ventricular Fibrillation

- The most serious of all cardiac arrhythmias, if not stopped within 1 to 3 minutes, is almost invariably fatal
- Results from cardiac impulses that have gone berserk within the ventricular muscle mass, stimulating first one portion of the ventricular muscle, then another portion, then another, and eventually feeding back onto itself to re-excite the same ventricular muscle over and over never stopping.
- The ventricular muscle contraction is not coordinated. So no pumping of blood occurs.
- Caused by

1. Sudden electrical shock of the heart
2. Ischemia of the heart muscle, of its specialized conducting system, or both.
3. Other forms of arrhythmia

Phenomenon of Re-entry

- When the normal cardiac impulse in the normal heart has travelled through the extent of the ventricles, it has no place to go because all the ventricular muscle is in refractory period and cannot conduct the impulse farther. Therefore, that impulse dies, and the heart awaits a new action potential to begin in the sinus node.
- Under some circumstances, however, this normal sequence of events does not occur. This initiates **re-entry** and lead to "circus movements," which in turn cause ventricular fibrillation. Causes:

1. A long pathway, typically occurs in dilated hearts.
2. Decreased rate of conduction, frequently results from blockage of the Purkinje system, ischemia of the muscle or high blood potassium levels.
3. A shortened refractory period commonly occurs in response to various drugs, such as epinephrine

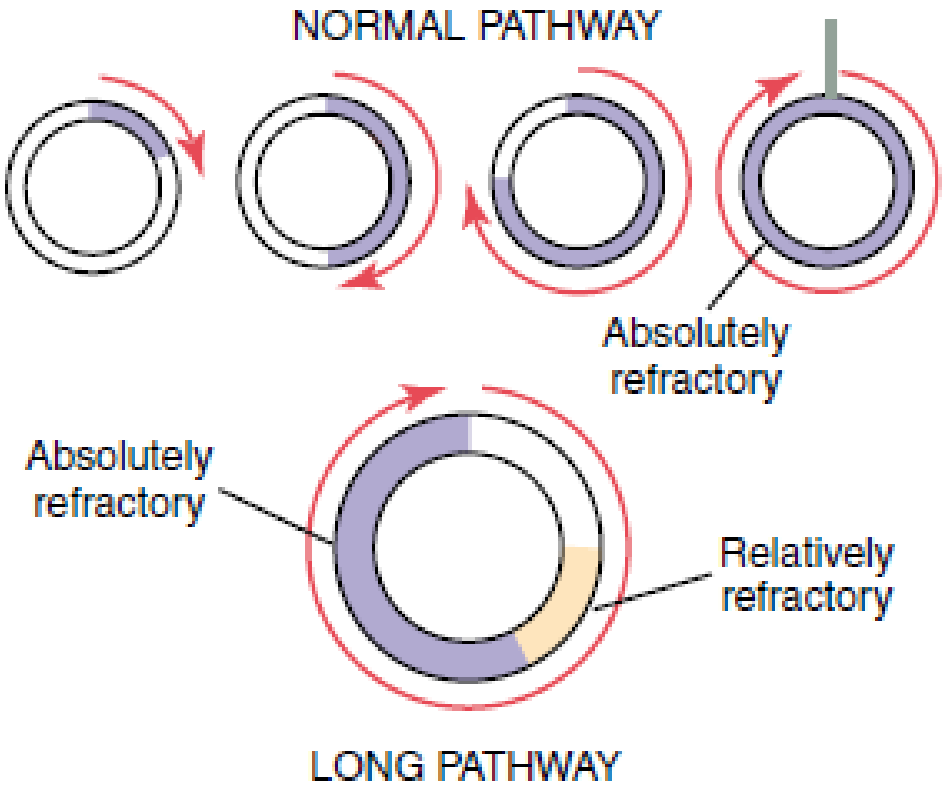


Figure 13-15 Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

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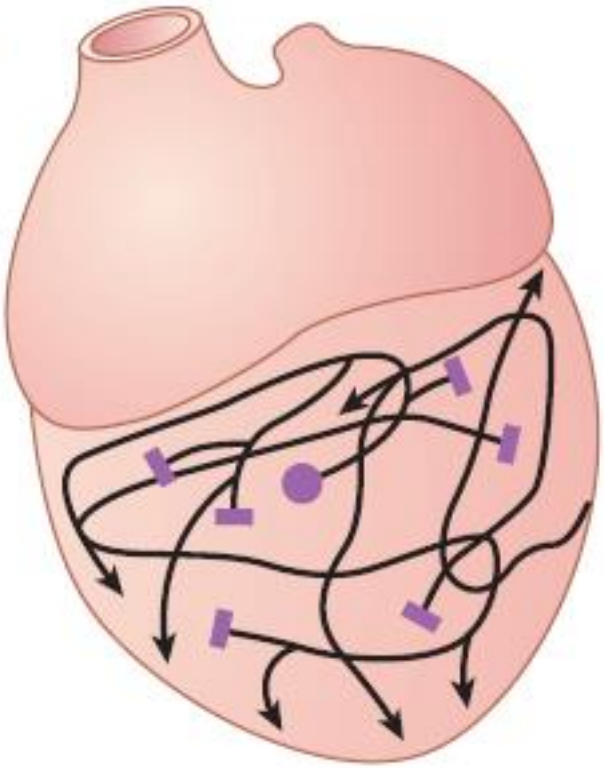
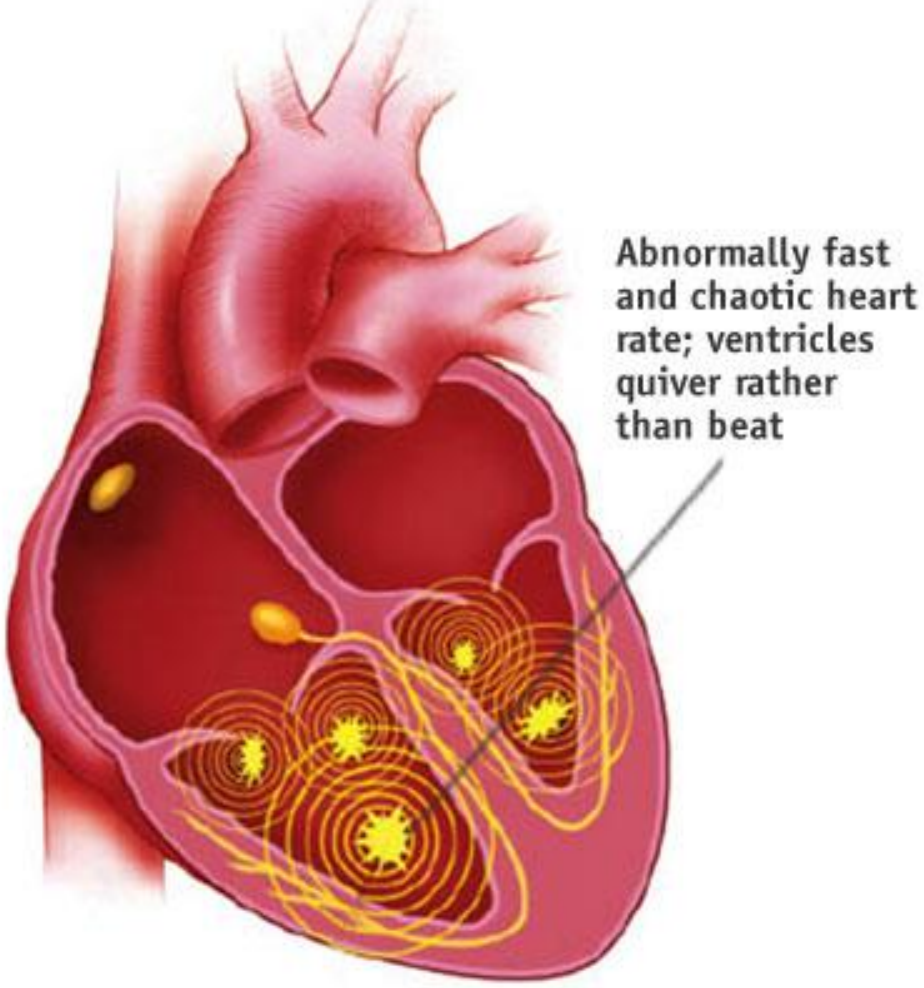
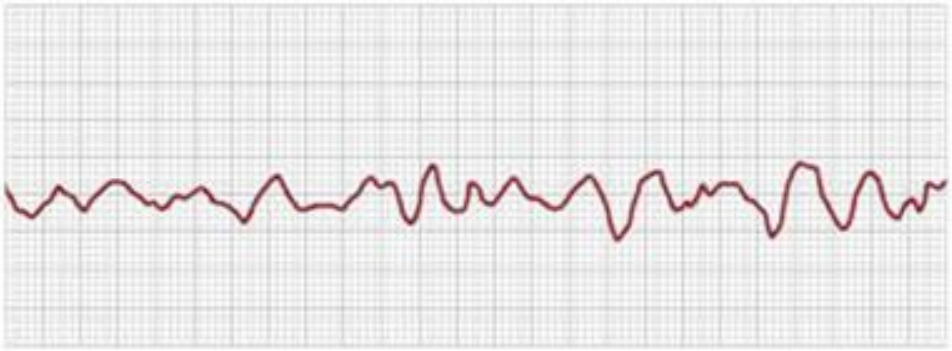




Ventricular Fibrillation ECG



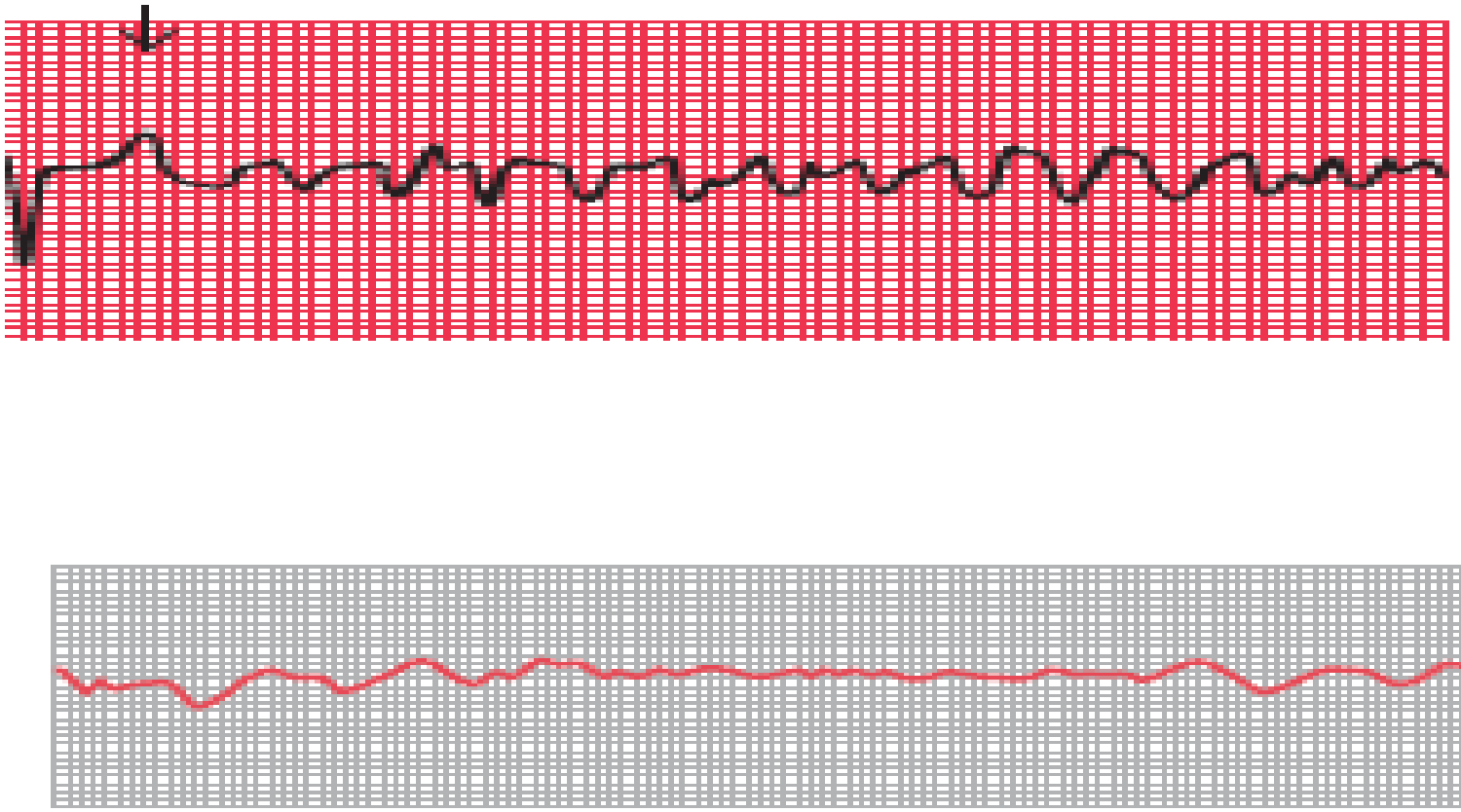


Figure 13-17 Ventricular fibrillation (lead II).

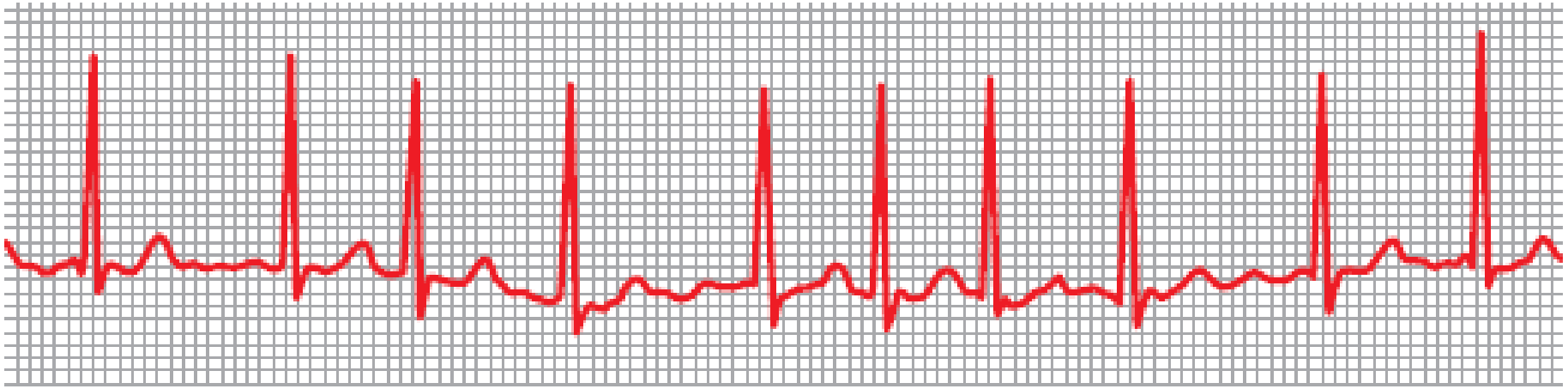
- ECG is bizarre and shows no regular rhythm of any type.
- Voltages of the waves in the ECG are usually about 0.5 millivolt when ventricular fibrillation first begins, but they decay rapidly.

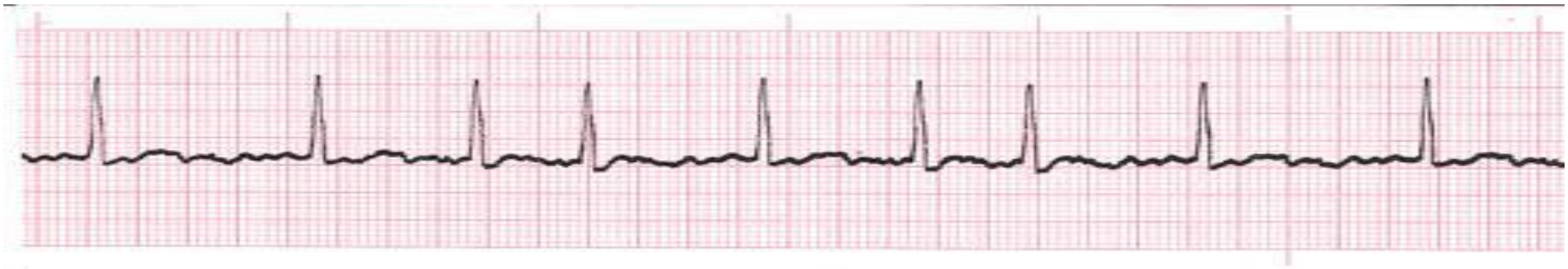
Atrial fibrillation

- The mechanism of atrial fibrillation is identical to that of ventricular fibrillation
- A frequent cause of atrial fibrillation is atrial enlargement

- The normal regular electrical impulses generated by the SA node are overridden by disorganized electrical impulses usually originating in the roots of the pulmonary veins.

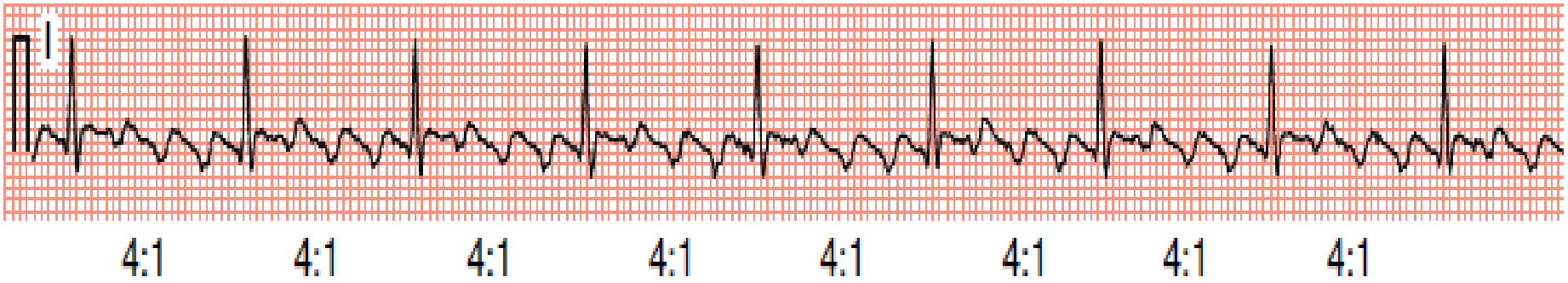
- On the ECG either no P waves are seen or only a fine, high frequency, very low voltage wavy record. The QRS complexes are normal in shape but are irregular



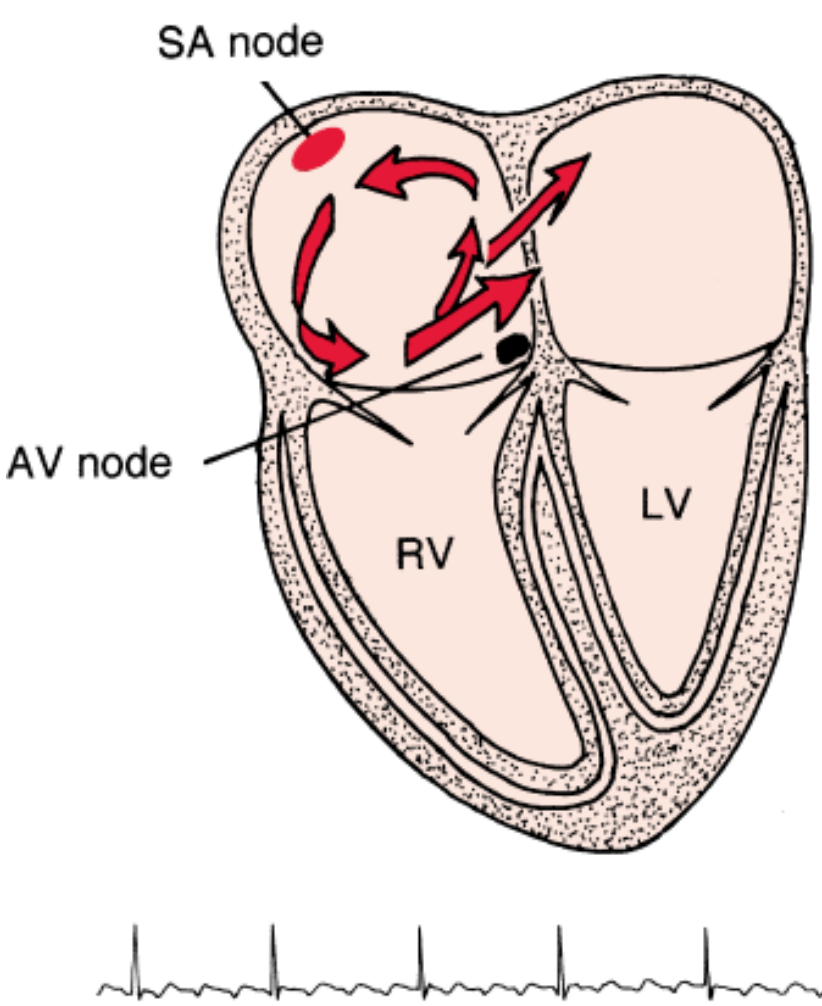


Atrial flutter

- Caused by a re-entry circuit within the right atrium.
- The electrical signal travels along a circular pathway within the right atrium, causing the atria to beat faster than the ventricles.
- Atrial rate is around 300 bpm (200-400)
- Ventricular rate is determined by the AV conduction ratio. The commonest AV ratio is 2:1, resulting in a ventricular rate of ~150 bpm.
- P waves are strong (saw tooth appearance)
- QRS-T complex follows an atrial P wave only once for every two to three beats of the atria, giving a 2:1 or 3:1 rhythm



ATRIAL FLUTTER



ATRIAL FIBRILLATION