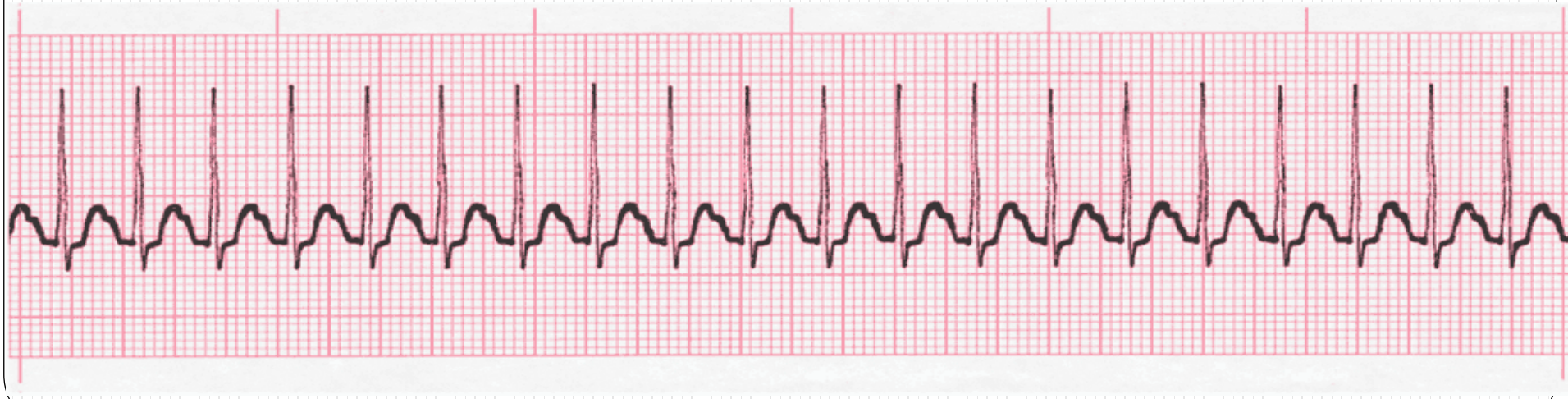
ECG Abnormalities

Dr. Tamara Alqudah



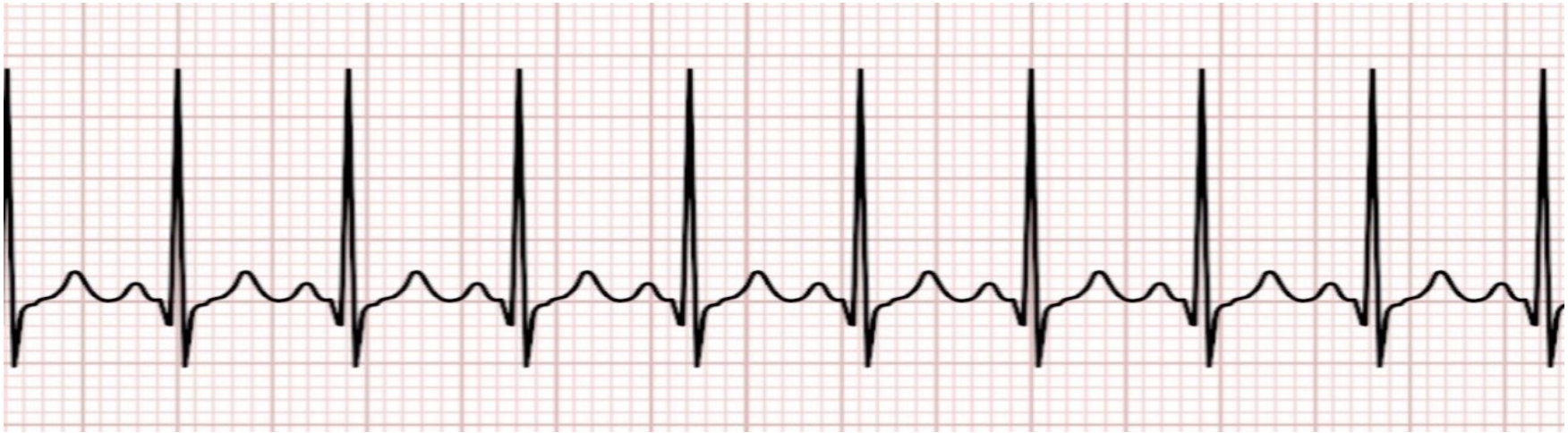
Arrhythmias

- An abnormal heart rhythm, the abnormality occurs in the rate or the regularity of heart beat.
- 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

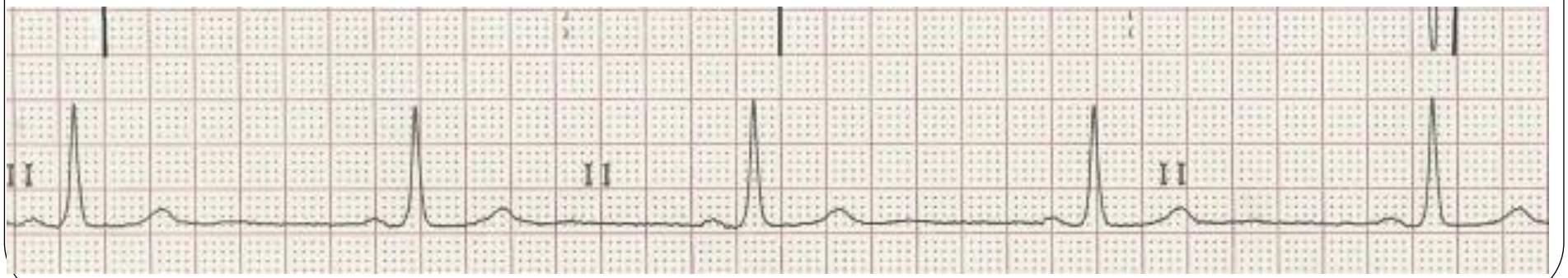
Sinus Tachycardia

- Fast heart rate above 100 beats per minute (B.P.M) usually caused by increased sympathetic tone in physiological stress.
- Other causes:
- Fever, Pain, Dehydration, Hypovolemia, Anemia



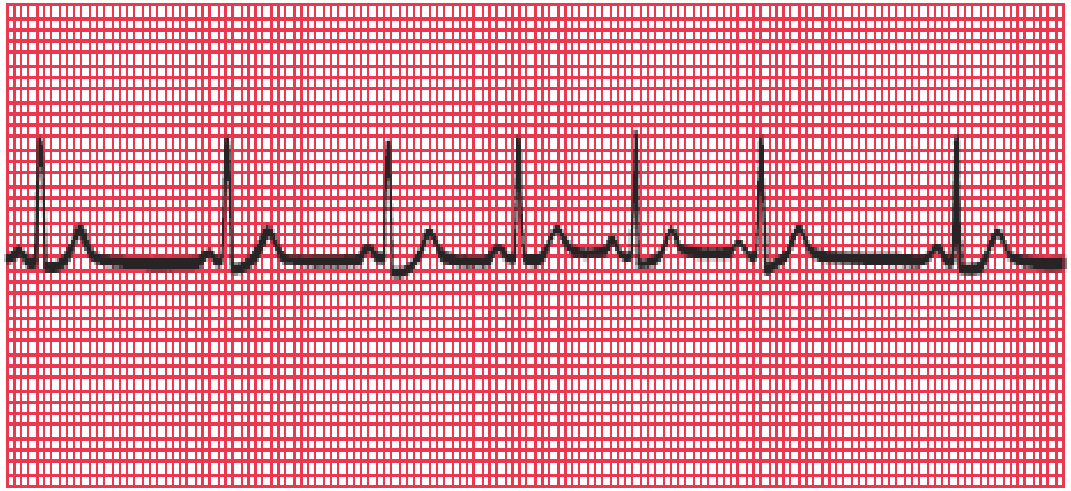
Sinus Bradycardia

- Slow heart rate below 60 B.P.M
- Seen normally in athletes at rest
- Vagal stimulation can trigger bradycardia



Sinus Arrhythmia

- The heart rate increases with inspiration & decreases with expiration this is especially evident in young people
- If the deviation exceeds 0.12 seconds in the R-R interval its sinus arrhythmia
- This condition is benign, common in young, healthy adults and children.



Note

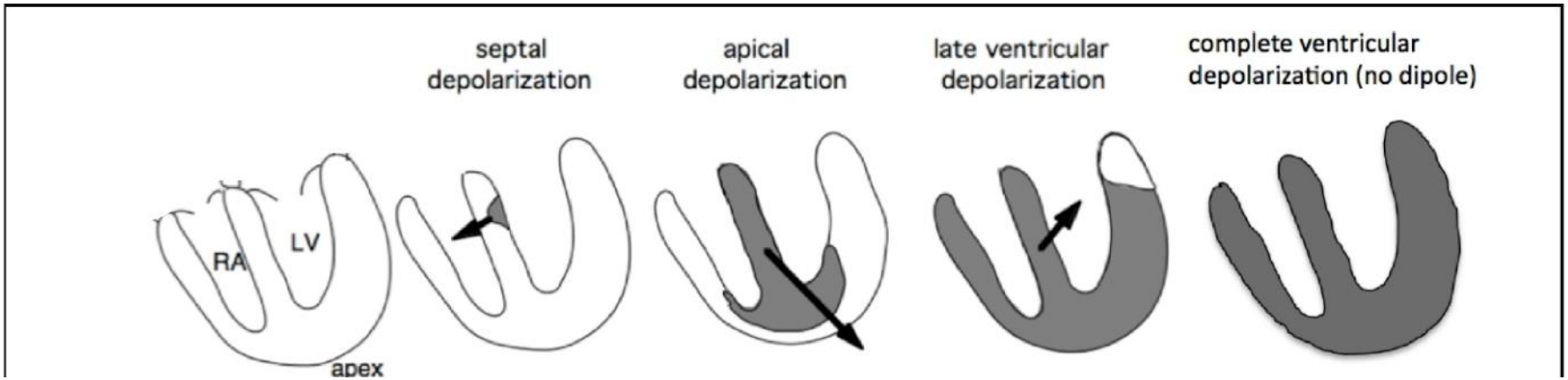
- One P wave per QRS complex
- Constant PR interval
- Progressive beat-to-beat change in the R–R interval

Ventricular fibrillation

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


septal depolarization
apical depolarization
late ventricular depolarization
complete ventricular depolarization (no dipole)
RA
LV
apex

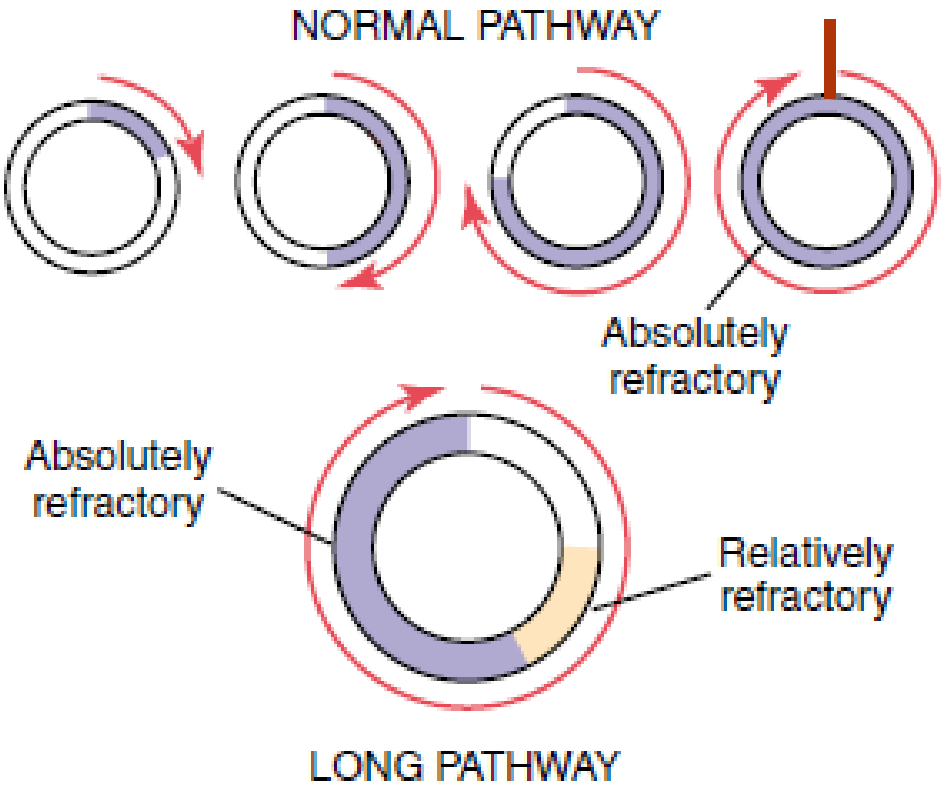


Figure 13-15 Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

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

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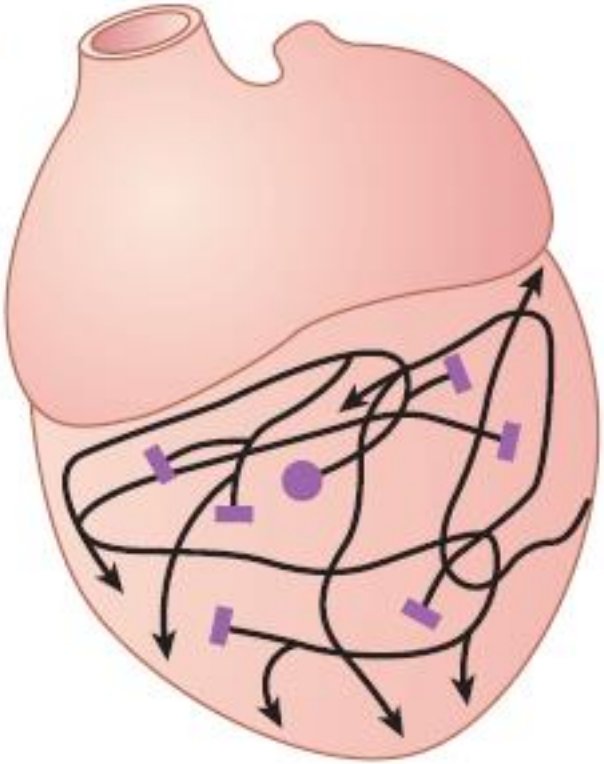
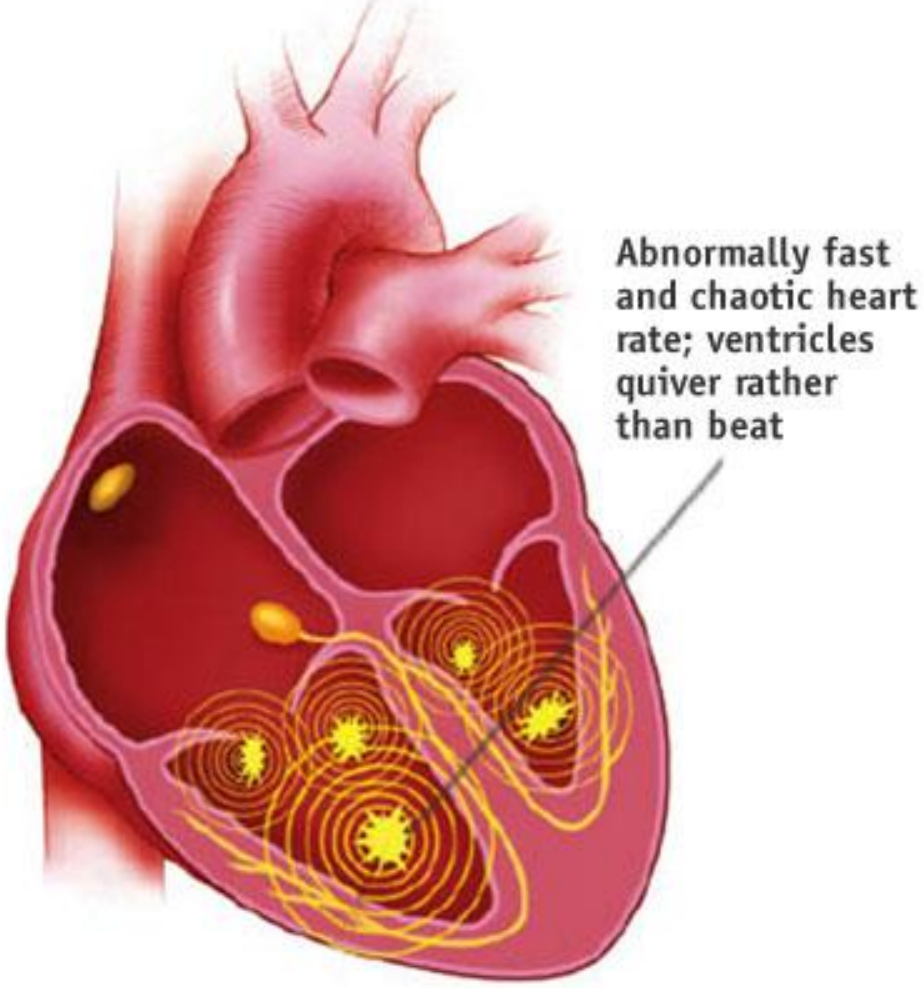
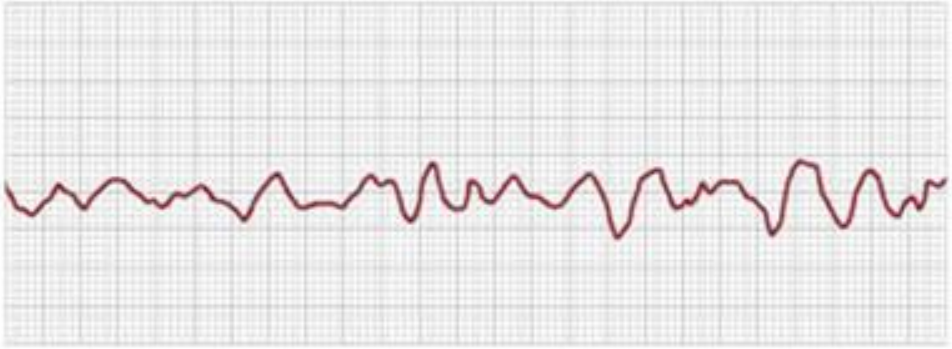




Ventricular Fibrillation ECG



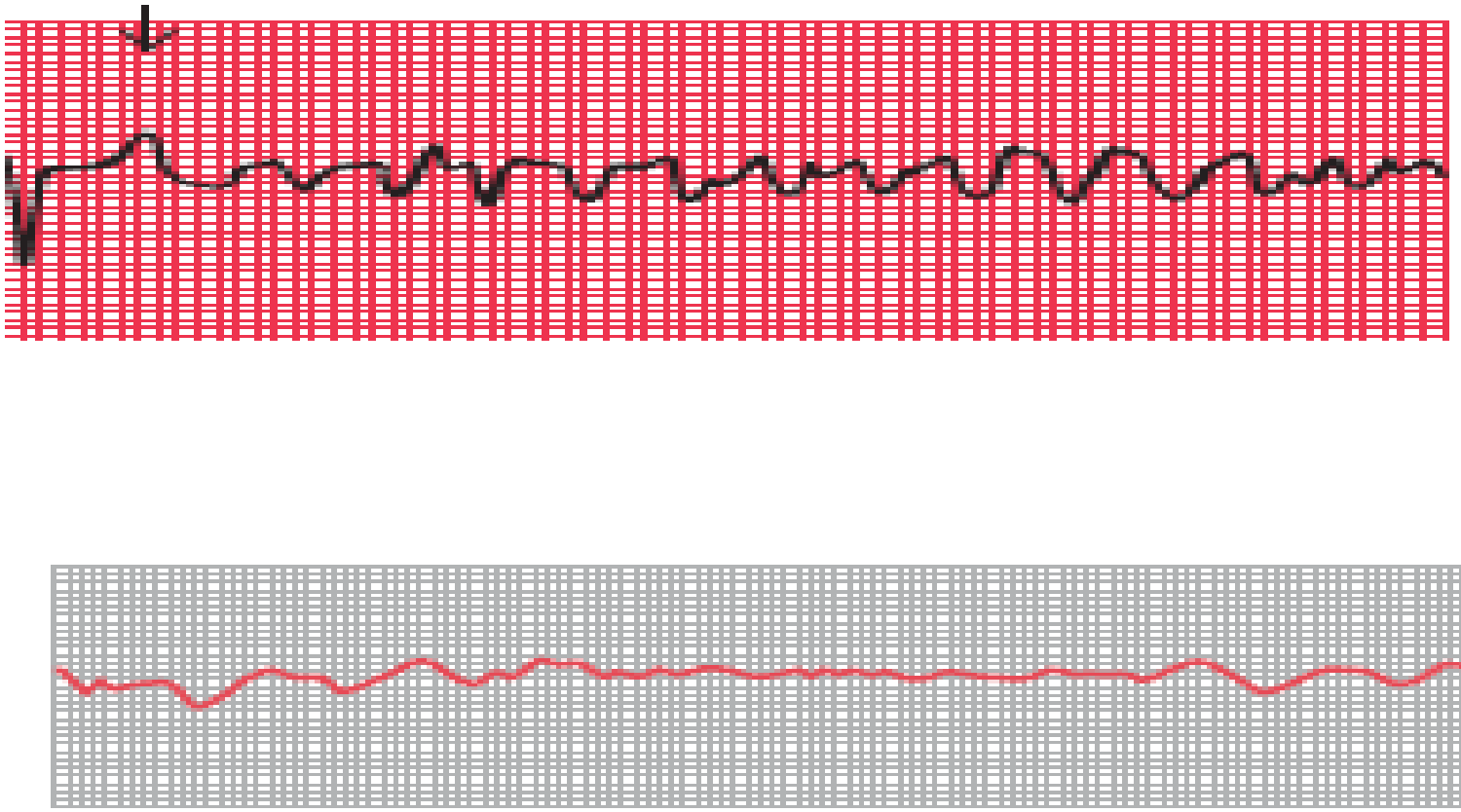
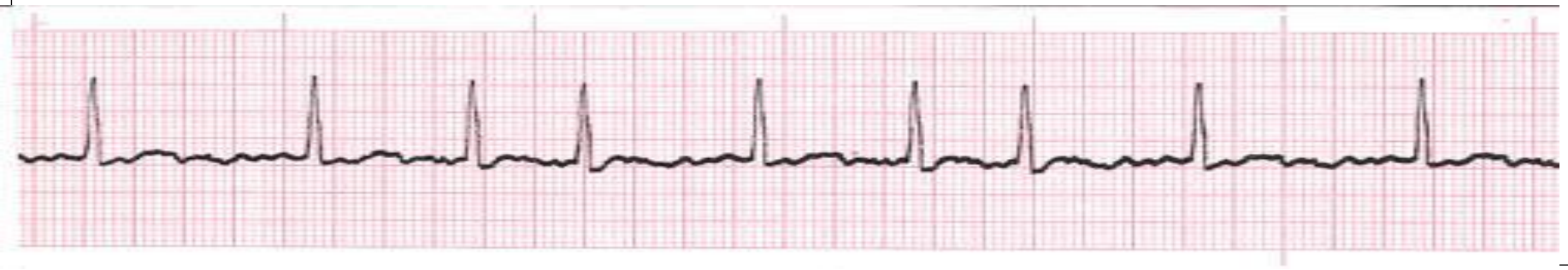


Figure 13-17 Ventricular fibrillation (lead II).

- 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.

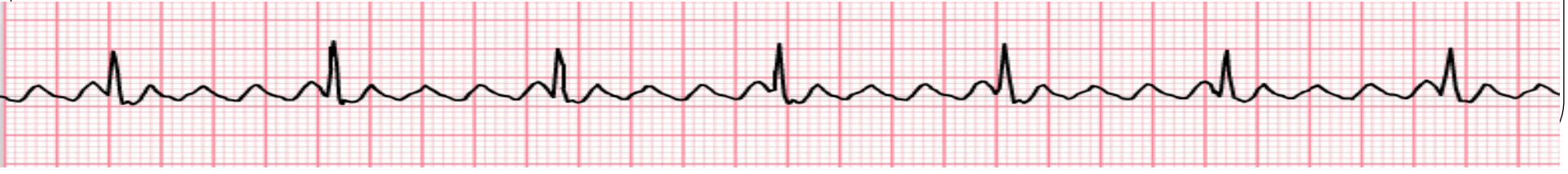
Atrial fibrillation

- 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-T 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



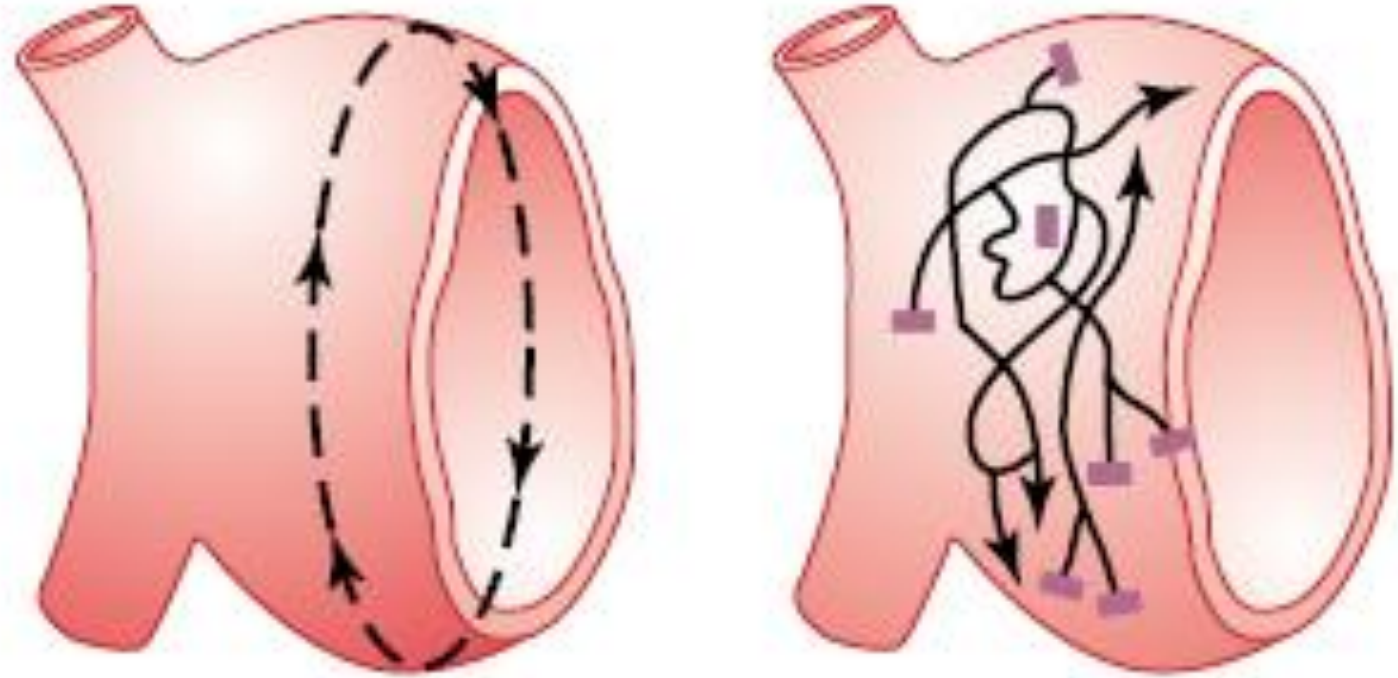


Figure 13-20 Pathways of impulses in atrial flutter and atrial fibrillation.

Atrioventricular (AV) Block

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

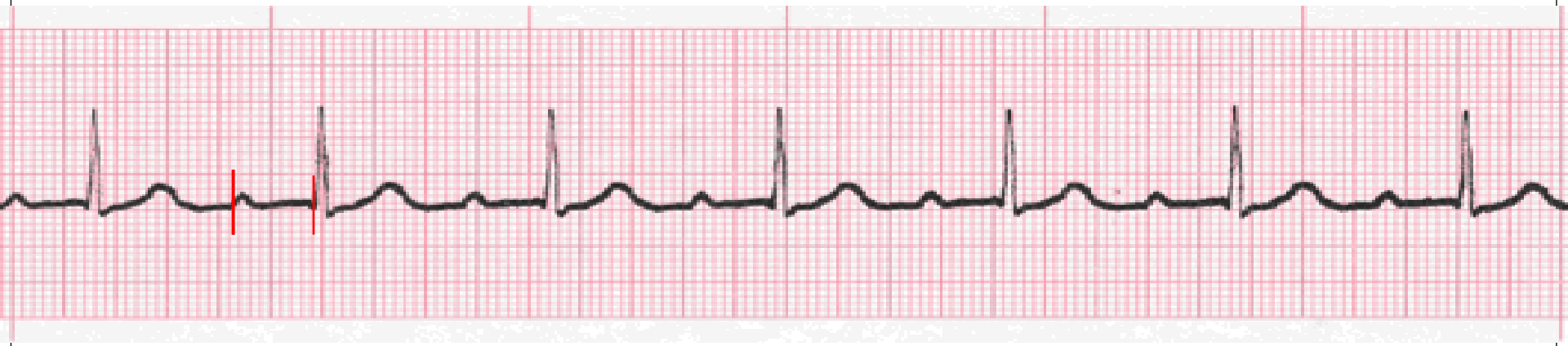
1. Ischemia of the A-V node or A-V bundle fibers
2. Compression of the A-V bundle by scar tissue or by calcified portions of the heart.
3. Inflammation of the A-V node or A-V bundle
4. Extreme stimulation of the heart by the vagus nerves

Types of AV block

- First degree heart block
- Second degree heart block
- Third degree heart block
- Stokes- Adams Syndrome

First Degree Heart Block

- when the PR interval increases to greater than 0.22 second, 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.



PR interval here is 0.32 seconds

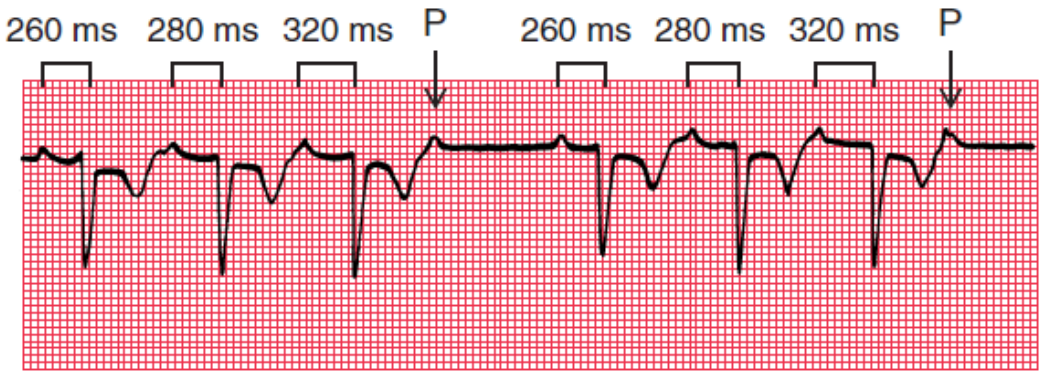
Second Degree Block

- 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 two types of second-degree A-V block:

1. Type I (Wenckebach periodicity)
2. Type II (Fixed ratio blocks)

Type I (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.

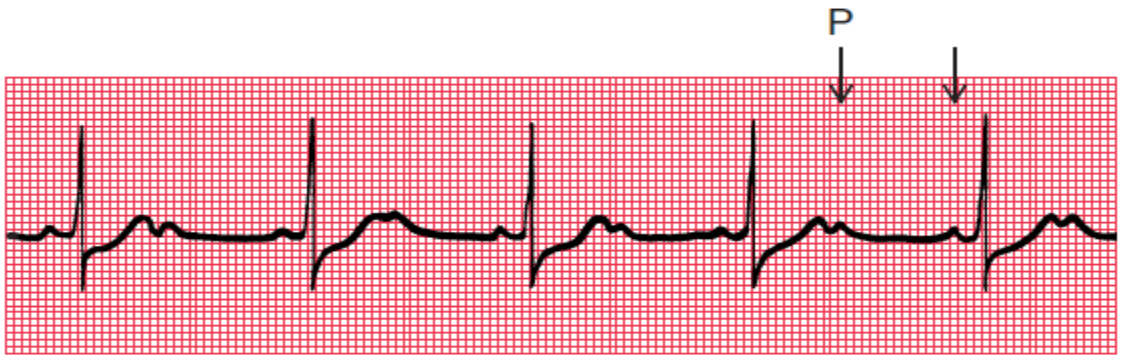


- Progressive lengthening of the PR interval
- One nonconducted P wave
- Next conducted beat has a shorter PR interval than the preceding conducted beat

Type II (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) ,or one conducted atrial beat and then two (3:1) or three (4:1) non-conducted beats.
- Caused by an abnormality of the bundle of His-Purkinje system and may require implantation of a pacemaker

Second degree heart block (2:1 type)



Note

- Two P waves per QRS complex
- Normal, and constant, PR interval in the conducted beats

Third degree block

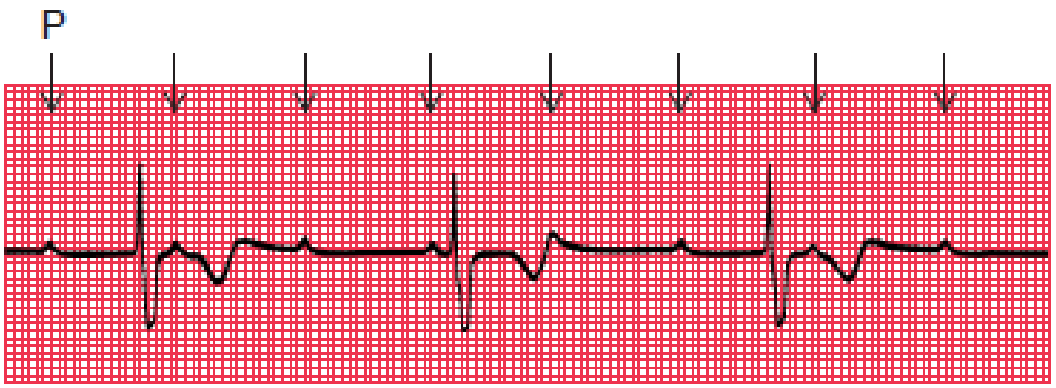
- Occurs with complete block of the impulse from the atria into the ventricles.

- 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.

Third degree heart block



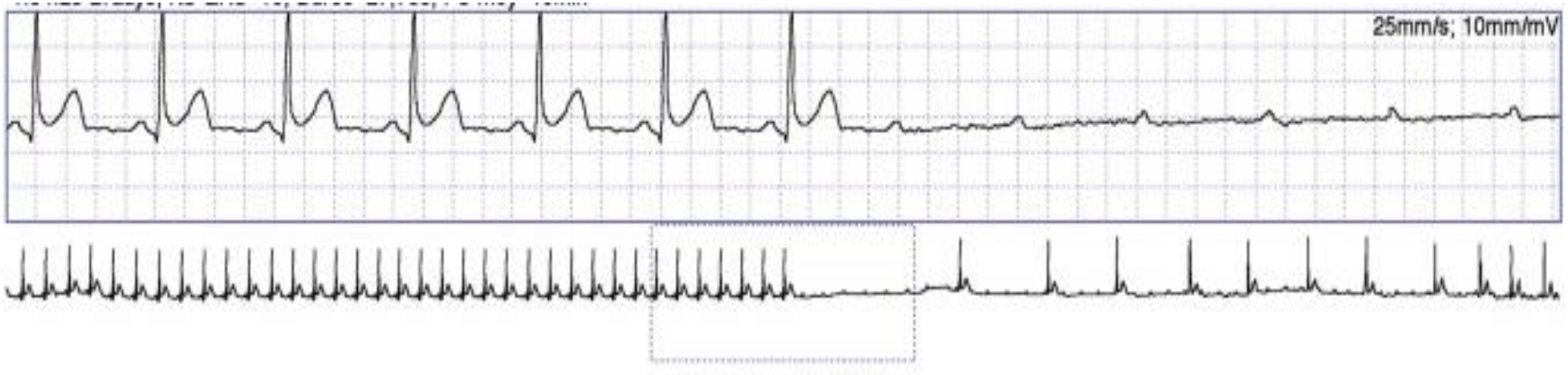
Note

- P wave rate 90/min
- No relationship between P waves and QRS complexes
- QRS complex rate 36/min
- Abnormally shaped QRS complexes, because of abnormal spread of depolarization from a ventricular focus

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

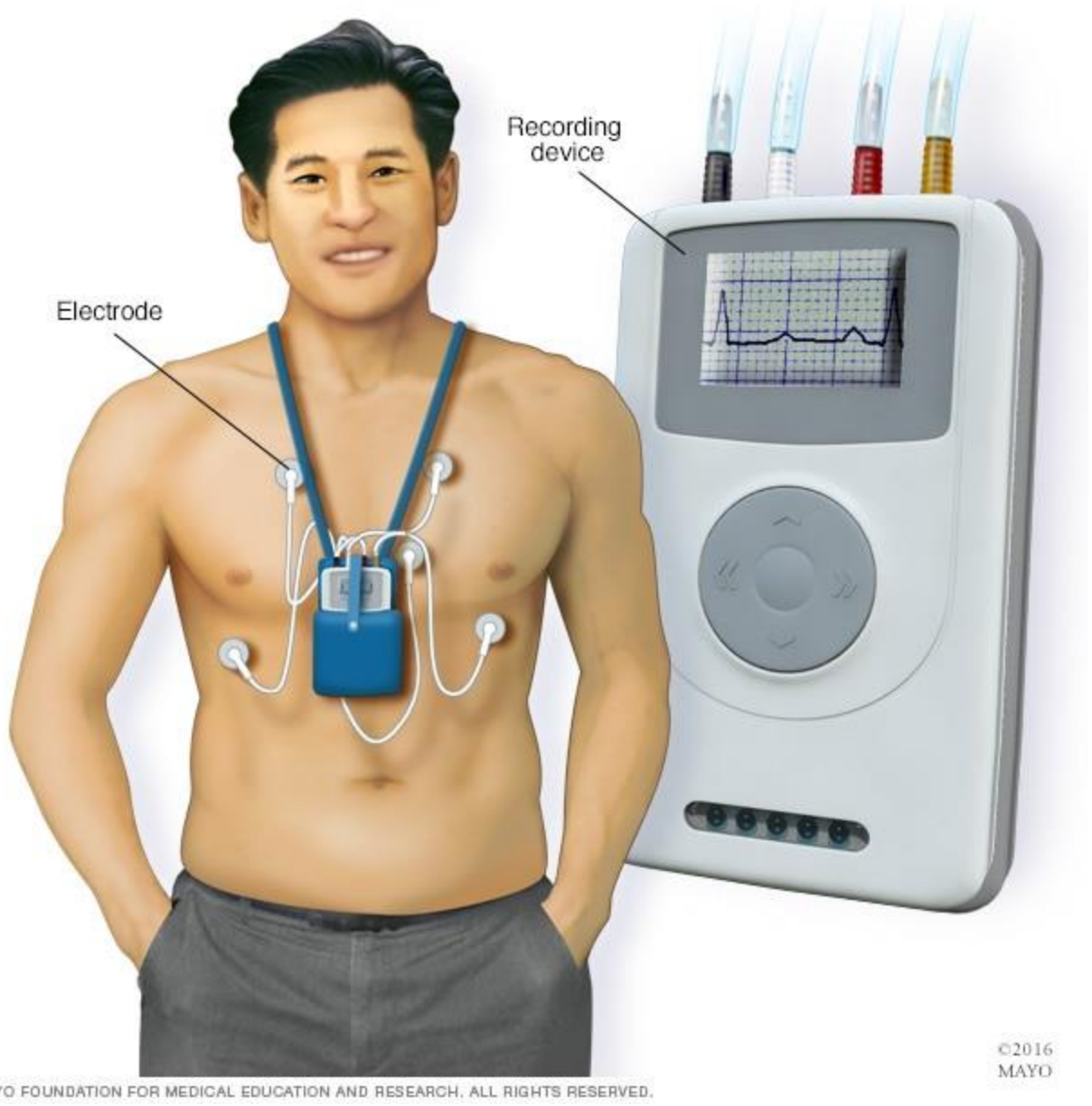
Stokes- Adams Syndrome

- The total block comes and goes
- The duration of block may be a few seconds, a few minutes, a few hours, or even weeks
- **Overdrive suppression** prevents the ventricles from beating until after a delay of 5 to 30 seconds. During this time the patient will faint.



Data obtained from Holter monitor

Holter monitor



Ventricular Conditions That Cause Axis Deviation

- Change in the Position of the Heart in the Chest.
- Hypertrophy of One Ventricle
- Bundle branch block

Change in the Position of the Heart in the Chest

- Conditions that cause left angulation of the heart and left axis deviation:

1. Deep expiration
2. Lying down
3. Obesity

- Conditions that cause right angulation of the heart and leright axis deviation:

1. Deep inspiration
2. Standing up

Hypertrophy of One Ventricle

- When one ventricle greatly hypertrophies, the axis of the heart shifts toward the hypertrophied ventricle, because:

1. Greater quantity of muscle exists on the hypertrophied side of the heart which allows generation of greater electrical potential on that side.
2. More time is required for the depolarization wave to travel through the hypertrophied ventricle. Consequently, the normal ventricle becomes depolarized in advance of the hypertrophied ventricle.

Bundle branch block

- Left bundle branch block
- When the left bundle branch is blocked, cardiac depolarization spreads through the right ventricle greatly ahead of the left ventricle. This leads to left axis deviation and widening of the QRS complex.

- Right bundle branch block
- When the right bundle branch is blocked, cardiac depolarization spreads through the left ventricle greatly ahead of the right ventricle. This leads to right axis deviation and widening of the QRS complex

Conduction in left bundle branch block: third stage

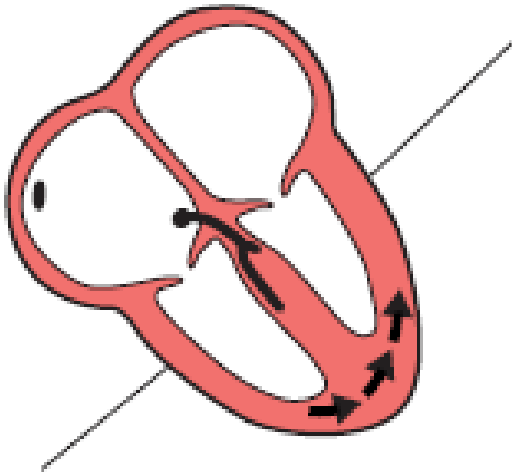
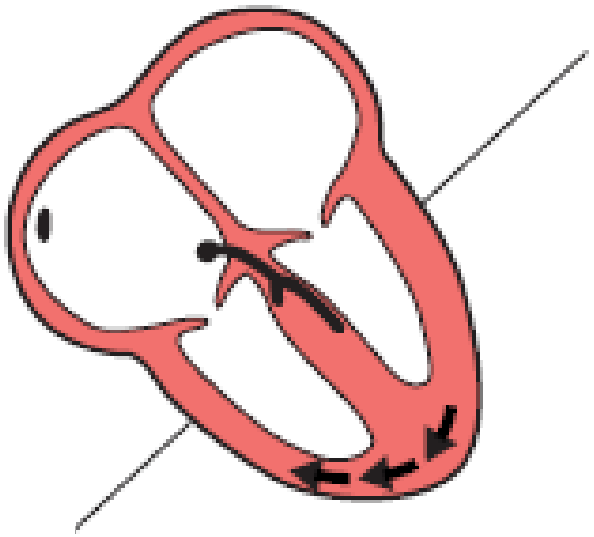


Fig. 2.11

Conduction in right bundle branch block: third stage



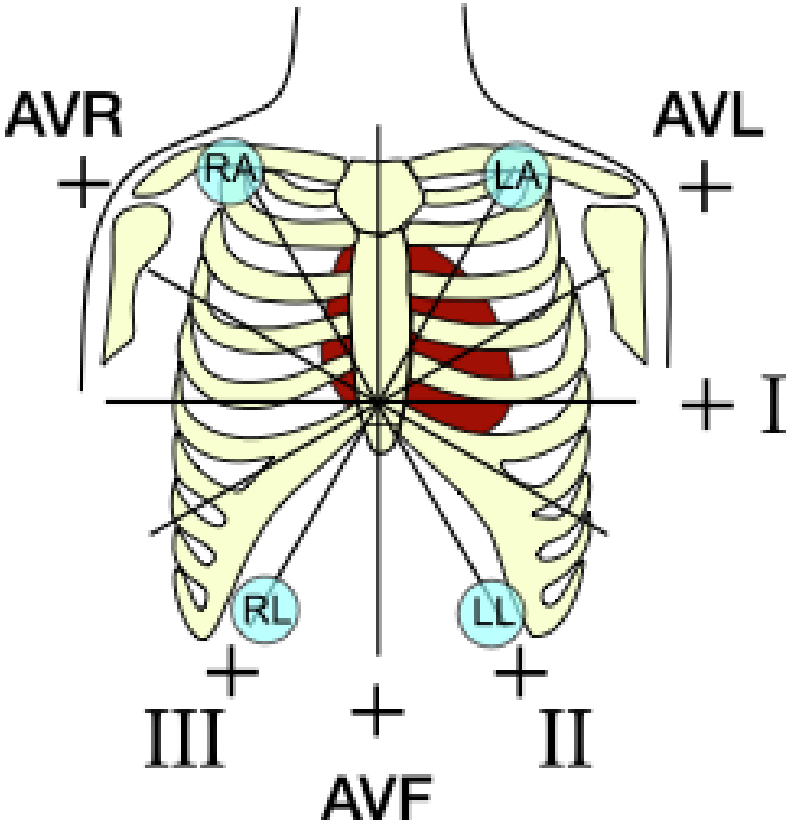
ECG changes seen in Myocardial infarction (MI) and Angina pectoris

- ECG is very useful for diagnosing MI & Angina pectoris and locating the affected areas.

- The earliest sign of MI is ST segment changes & it occurs in the leads corresponding to the part of the heart that is damaged:
- Leads V1-V3 with anterior wall infarction (right ventricle & the septum),
- Lead aVL, I, V5 & V6 with lateral wall infarction
- Leads II, III and aVF with inferior wall infarction.
- 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

- In case of myocardial infarction, within a day or so, the ST segments return to the baseline.
- Without proper & prompt treatment the T waves in the affected leads become inverted, and Q waves develop within 24 hours. These ECG changes are usually permanent
- In angina ECG changes are noticed while the patient is in pain, once the pain has resolved the ECG returns to normal.
- ST segment changes are very common with angina.
- When the ECG is normal at rest, ST segment changes may be induced by making the patient exercise, this test is called stress ECG

The 6 Limb Leads



The 6 Left Chest Leads

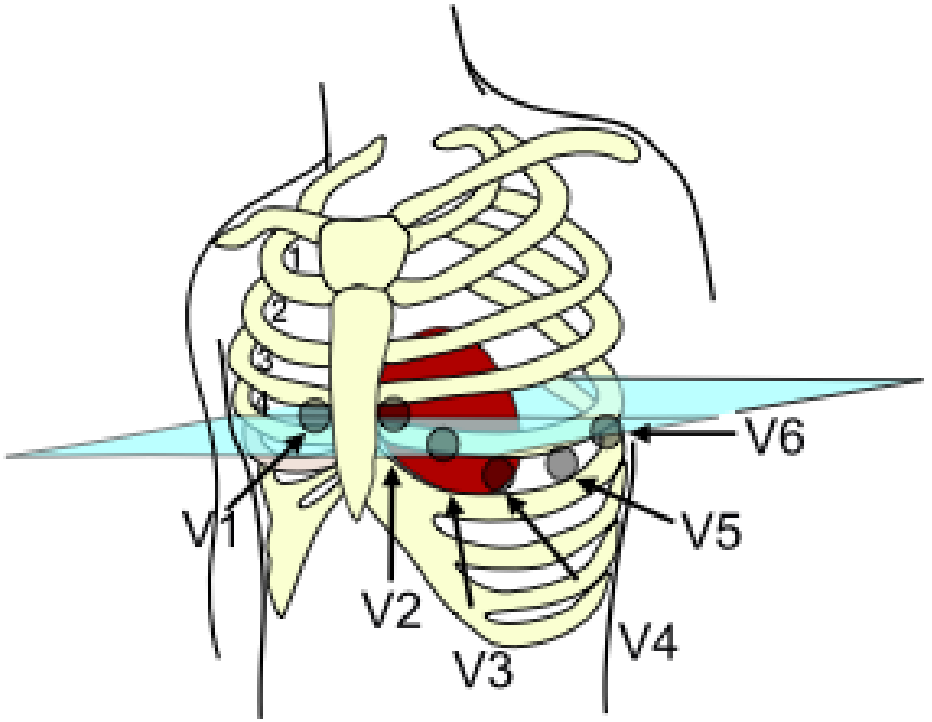
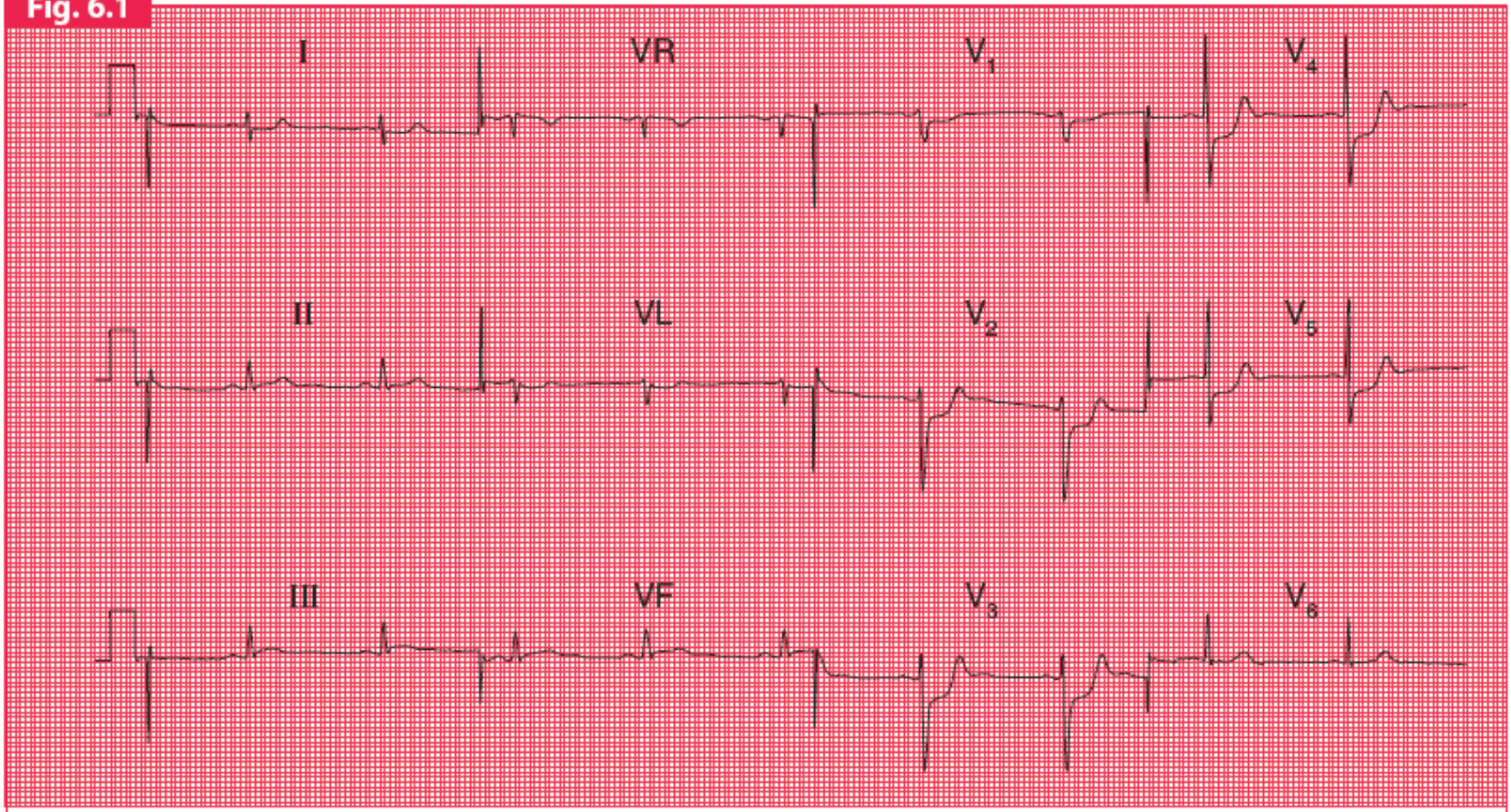


Fig. 6.1



ST segment depression in unstable angina

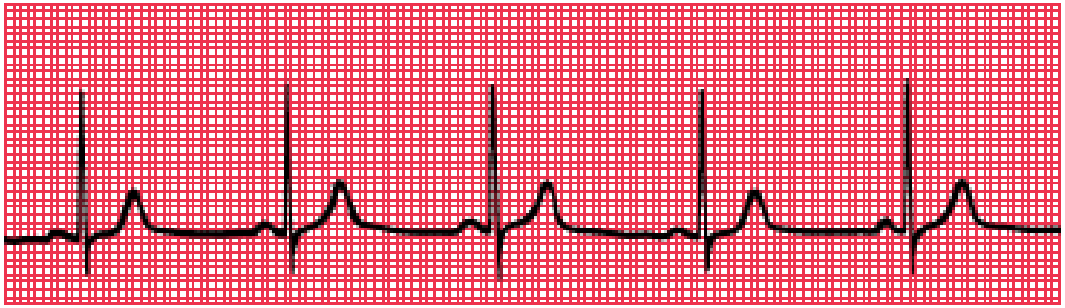
Note

- Sinus rhythm, rate 60/min
- Normal axis
- Normal QRS complexes
- ST segments depressed horizontally in leads V_3–V_5
- Normal T waves

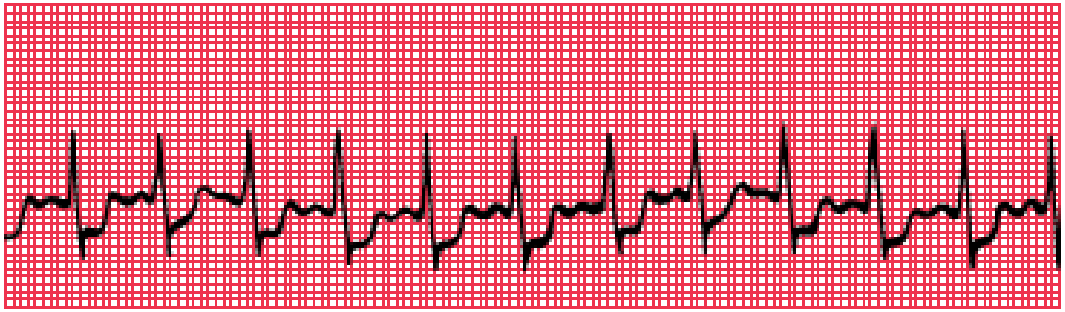
Fig. 4.14

Exercise-induced ischaemic changes

Rest:



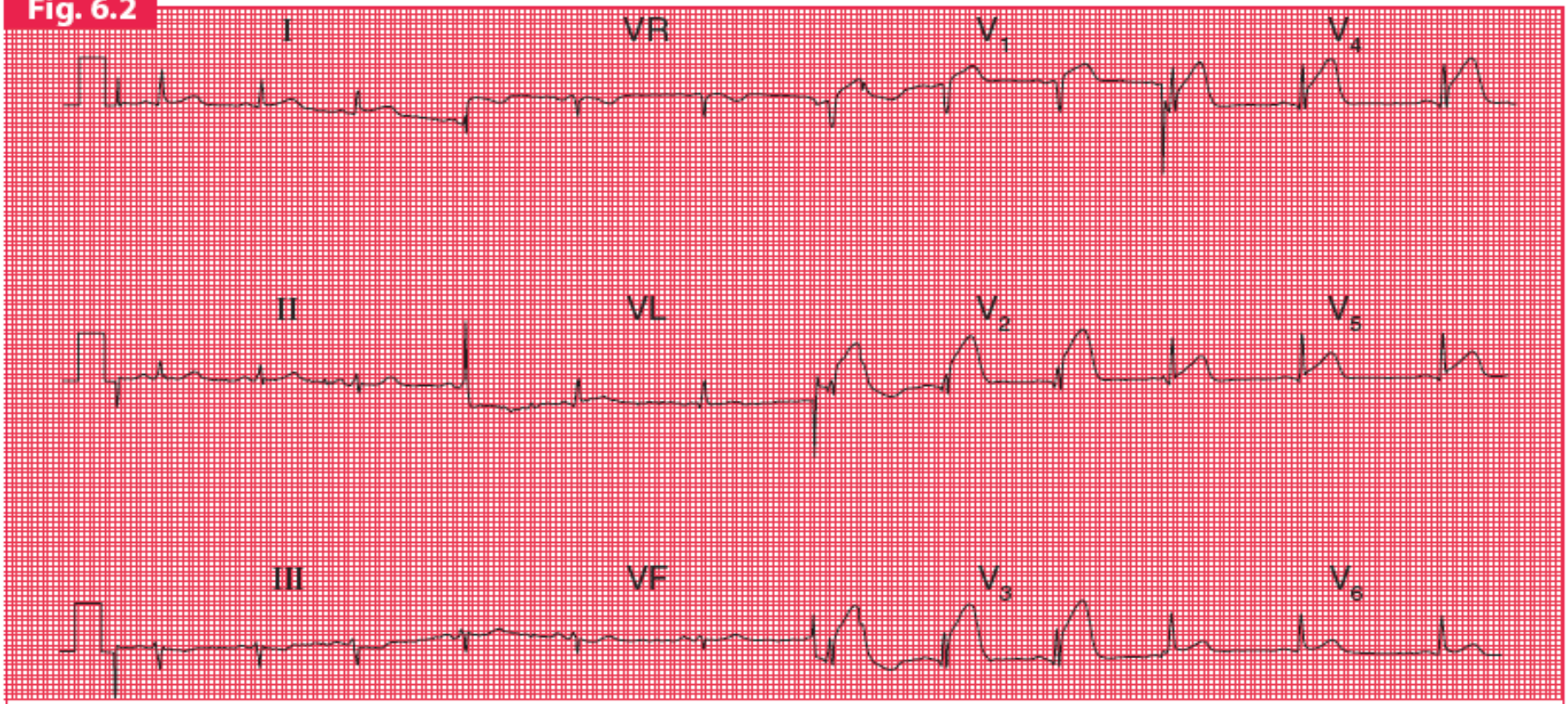
Exercise:



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

Fig. 6.2



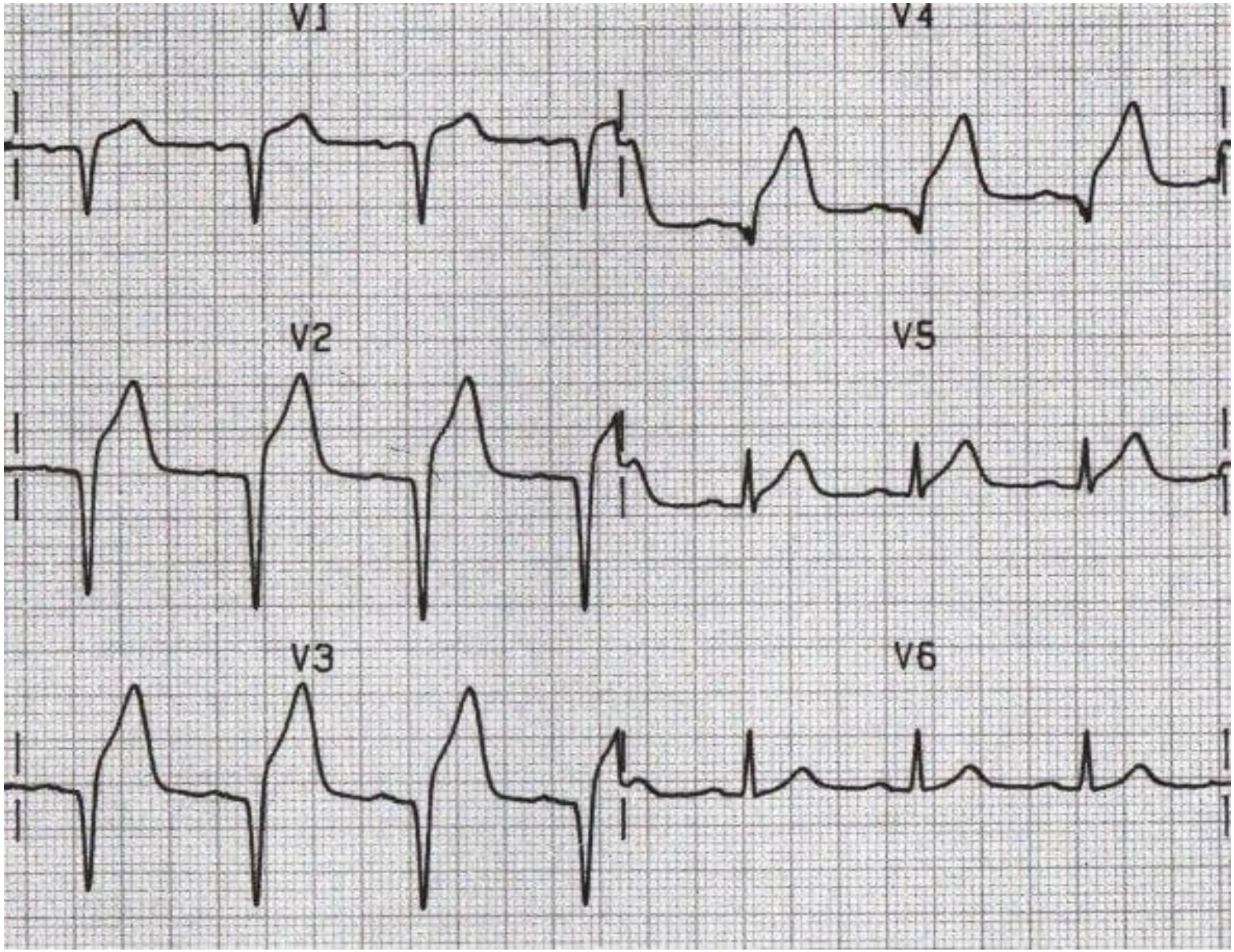
ST segment elevation in acute anterior ST segment elevation myocardial infarction

Note

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

Pathological Q wave

- Q waves are considered pathological if:
  - > .04 sec
  - > 2 mm deep
  - > 25% of depth of QRS complex
- **Pathological Q waves usually indicate ongoing or <u>prior myocardial infarction.</u>**



Pathological Q waves in V1-V4

Abnormal ECGs

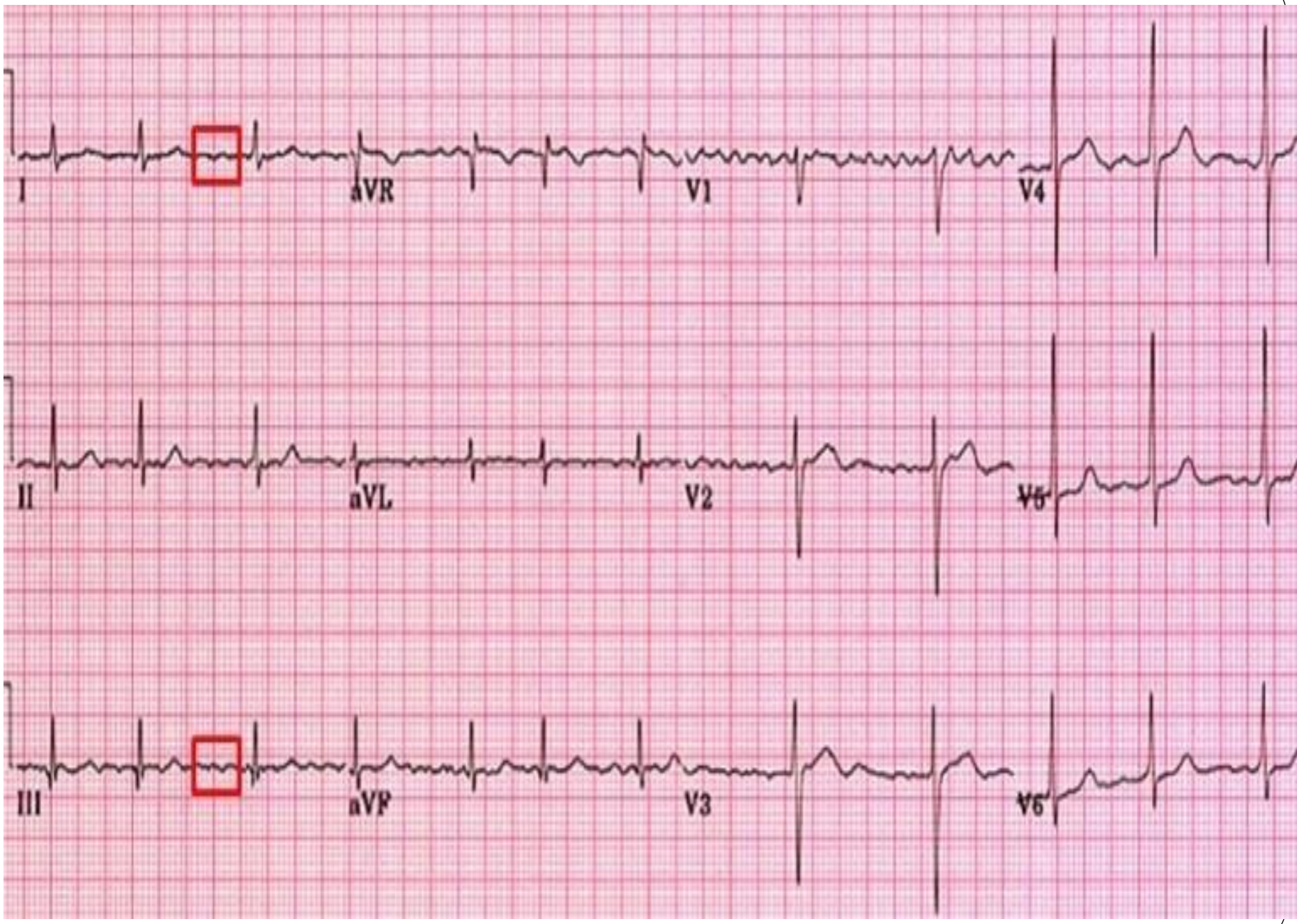
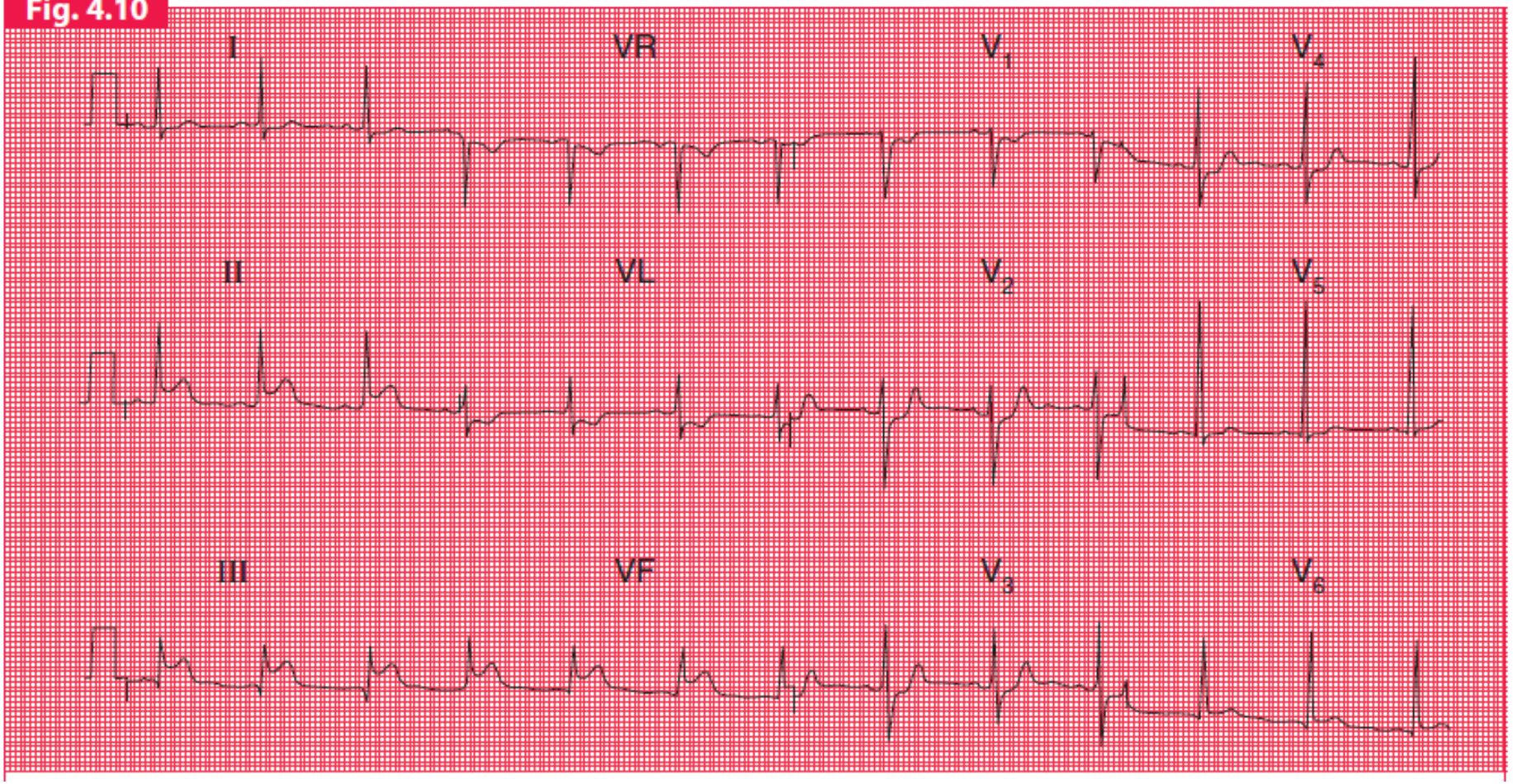
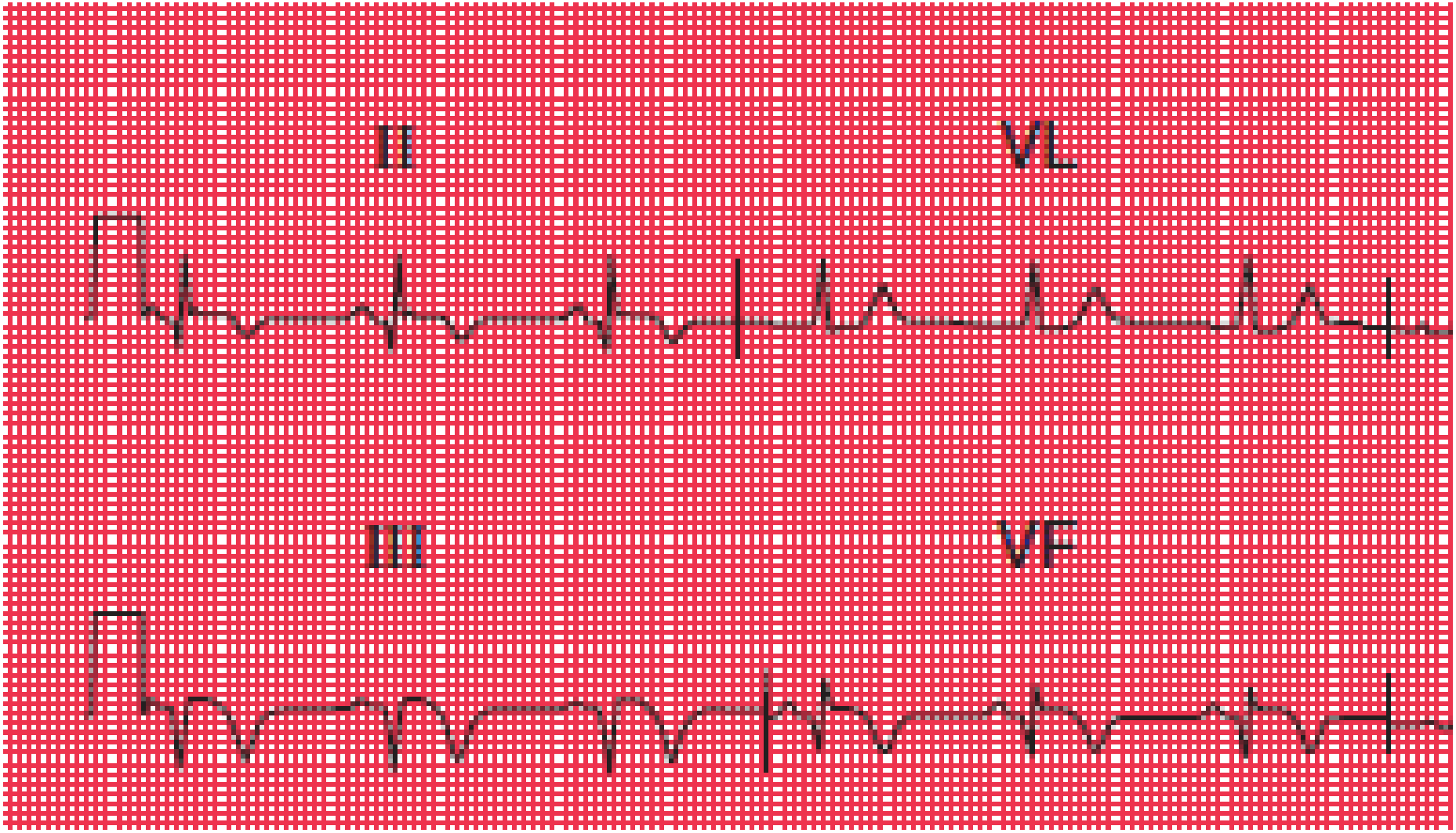

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Fig. 4.10

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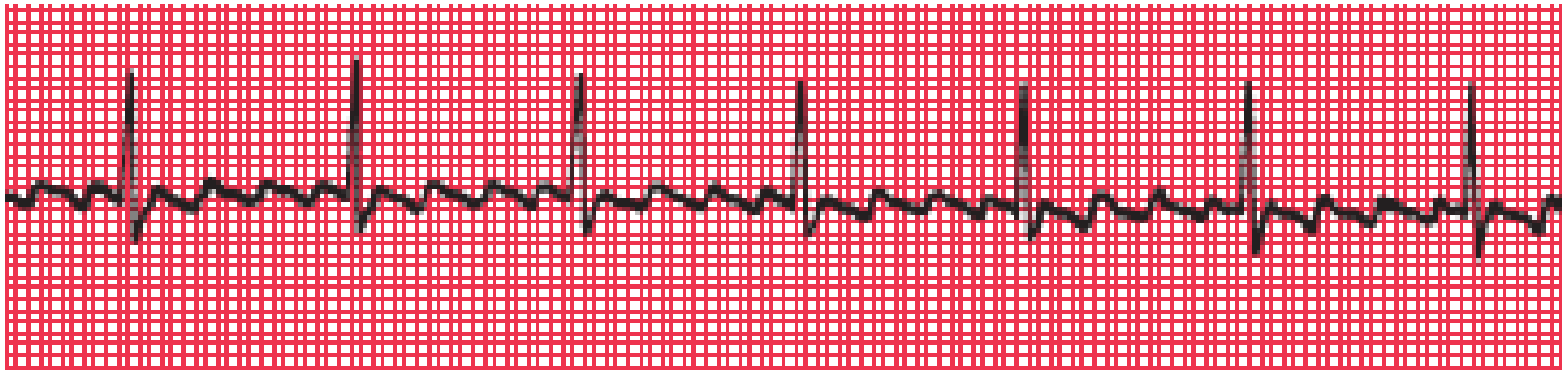

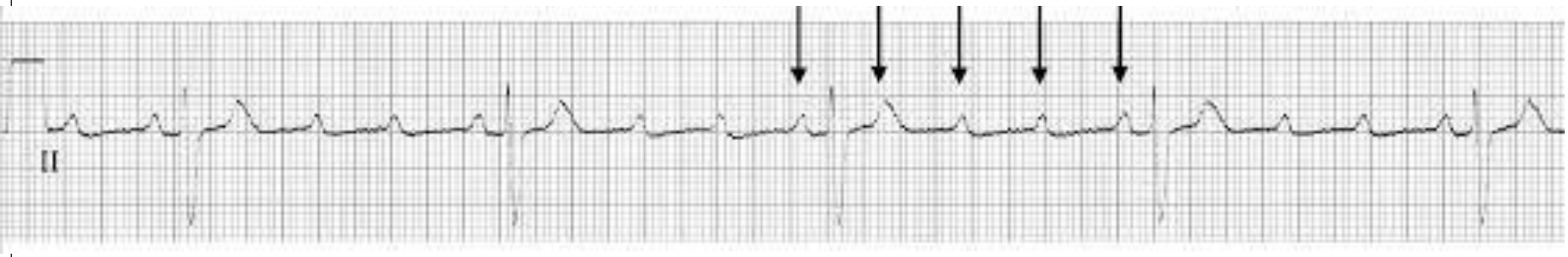
II

Thank You

BEST OF LUCK