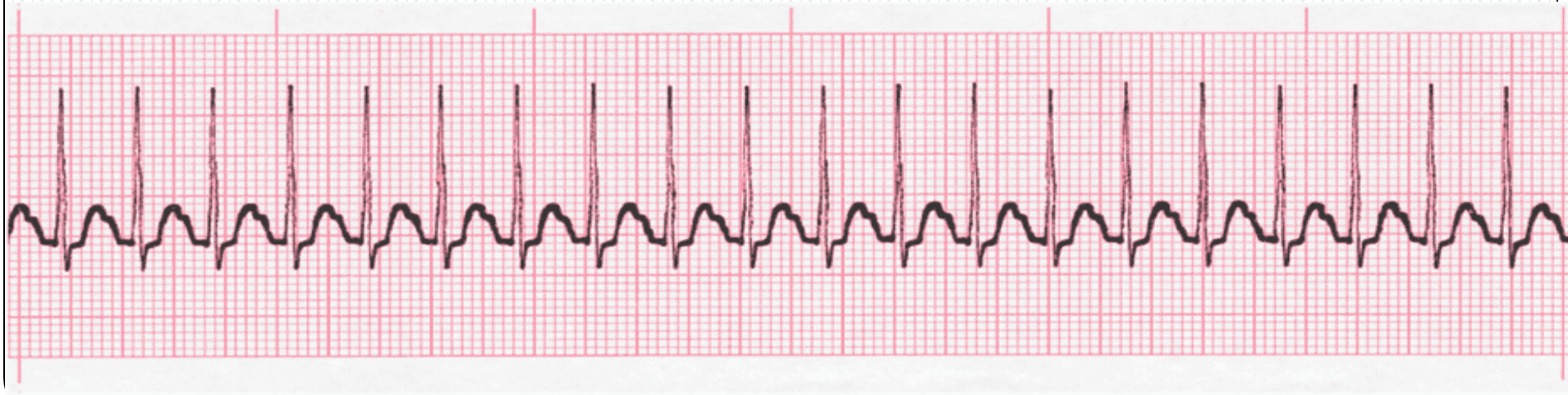
1. This ECG belongs to Huda Jamal a 40 year old female patient
2. The heart rate is 70 B.P.M. (65,68,75). When there is a variation in RR interval count the number of R waves in 6 sec(30 large squares) and multiply the result by 10
3. Normal sinus rhythm, the R-R interval has little variation (0.12 sec) there is a P wave before every QRS complex. The P waves all look alike in the same lead.
4. Cardiac axis is normal. the angle is 51degrees.
5. P wave duration is 0.08 sec, maximum height is 2 mm (Lead II)
6. QRS complex has a duration of .08 sec. Maximum voltage in limb leads is 1 mV (in lead II) and 1.6 mV in chest leads (V4). There is normal R wave progression in chest leads. No pathological Q waves.
7. T wave is upright in all leads except aVR & V1 which is normal. T wave has normal voltage in limb & chest leads
8. PR interval=0.16 sec , QT interval= 0.36 sec .QTc=0.39sec (use the average RR interval 0.87 sec)
9. ST segment is isoelectric in all leads.

ECG Abnormalities

Dr. Tamara Alqudah



- When we interpret an ECG we compare it instantaneously with the normal ECG and normal variants stored in our memory; these memories are stored visually in the posterior parts of the cerebrum and intellectually in the frontal parts. If these reservoirs contain only question marks, we are as helpless and lost in our interpretation as someone wandering through a snowstorm without a compass. It is important therefore to fill, or reactivate, these reservoirs

Cardiac abnormalities

- Arrhythmias
- Conduction abnormalities
- Myocardial infarction and Angina pectoris
- Ventricular hypertrophy

Arrhythmias

- An abnormal heart rhythm, the abnormality occurs in the rate or the regularity of heart beat due to abnormalities in the cardiac excitatory and conductive system.

- Abnormal Sinus Rhythm
- Ventricular fibrillation
- Atrial fibrillation
- Atrial flutter

Fig. 3.2

Points where cardiac rhythms can begin

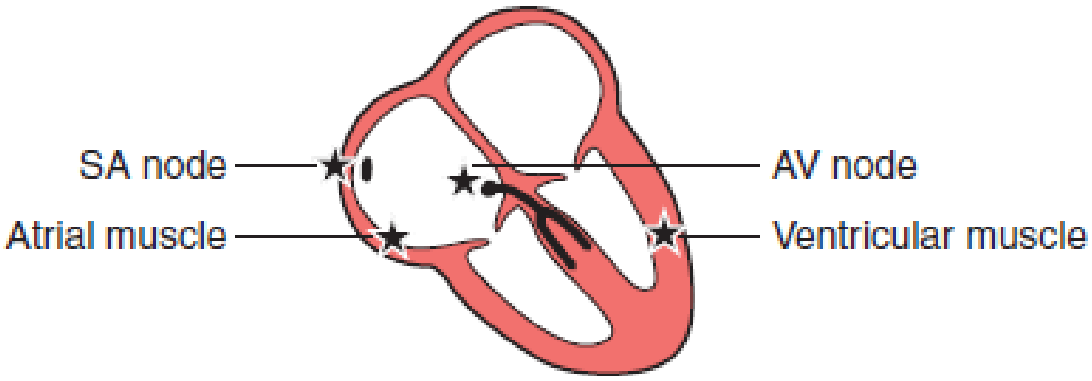
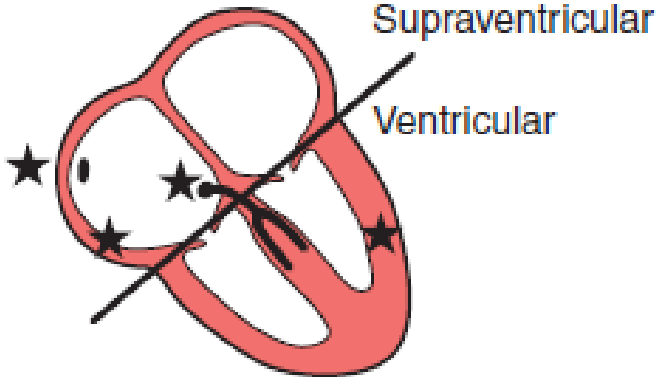


Fig. 3.3

Division of abnormal rhythms into supraventricular and ventricular

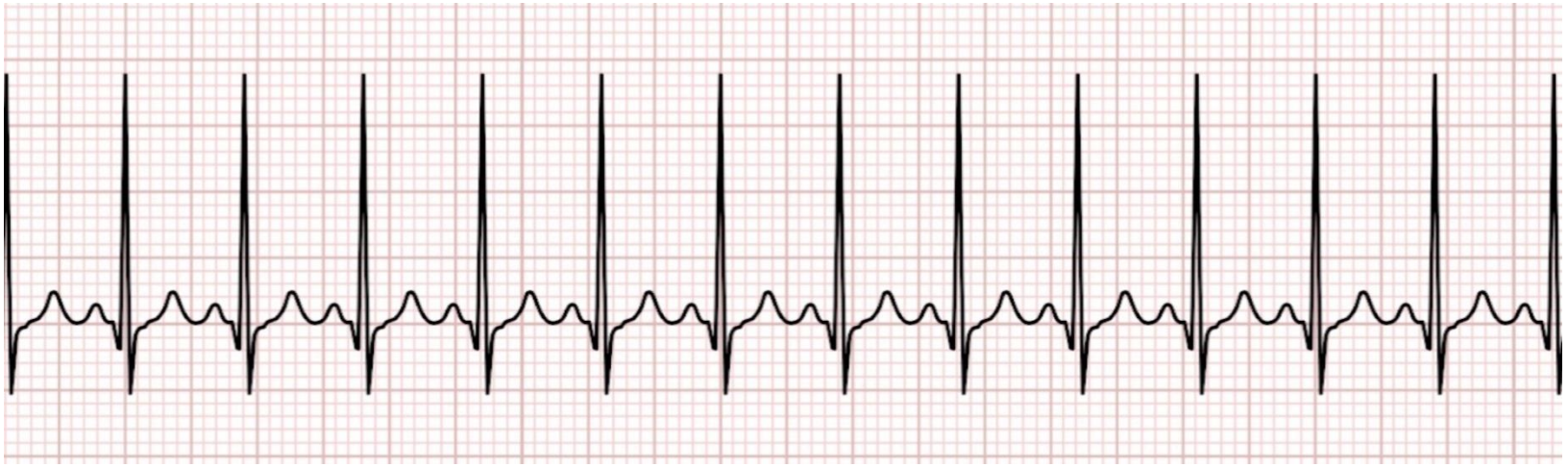


Abnormal sinus Rhythm

- Abnormal sinus rhythm means there is an irregularity in the heart rhythm, originating at the sinus node. Types :
- Sinus Tachycardia
- Sinus Bradycardia
- Sinus Arrhythmia

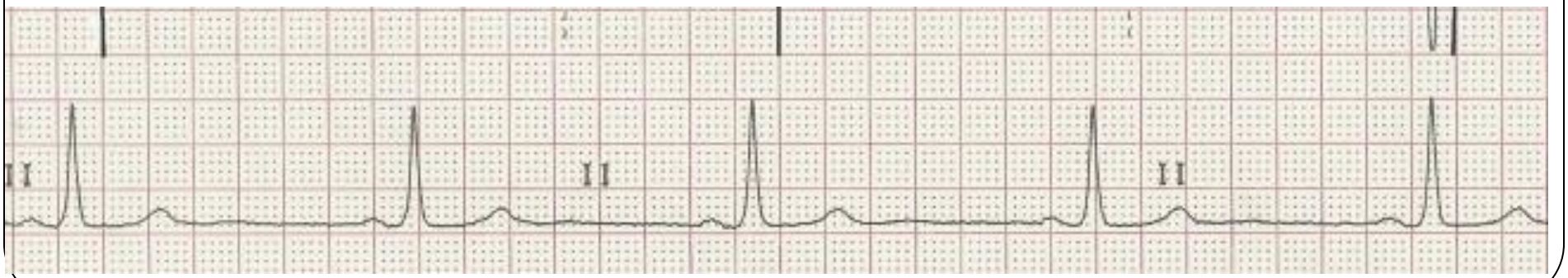
Sinus Tachycardia

- Fast heart rate above 100 beats per minute (B.P.M)
- Causes:
  - Fever
  - Sympathetic stimulation



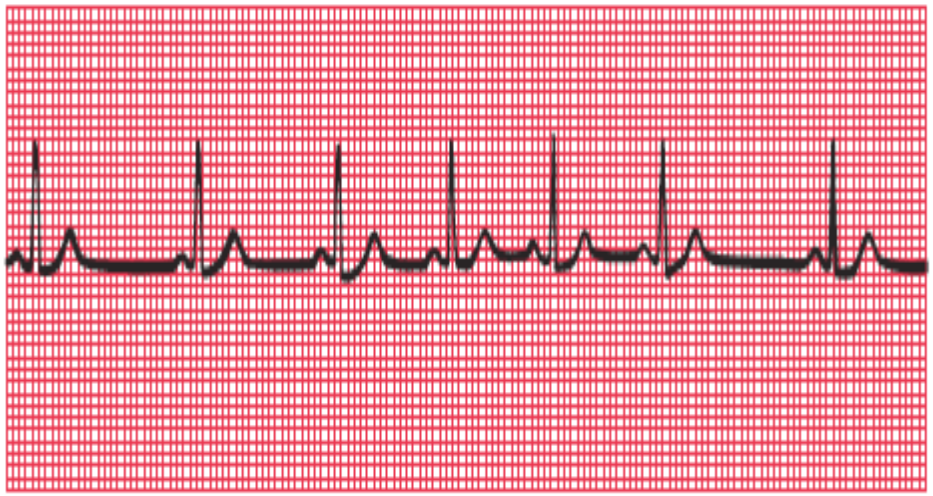
Sinus Bradycardia

- 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 due to decreased vagal tone & decreases with expiration this is especially evident in young people
- If the deviation exceeds 0.12 seconds 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

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

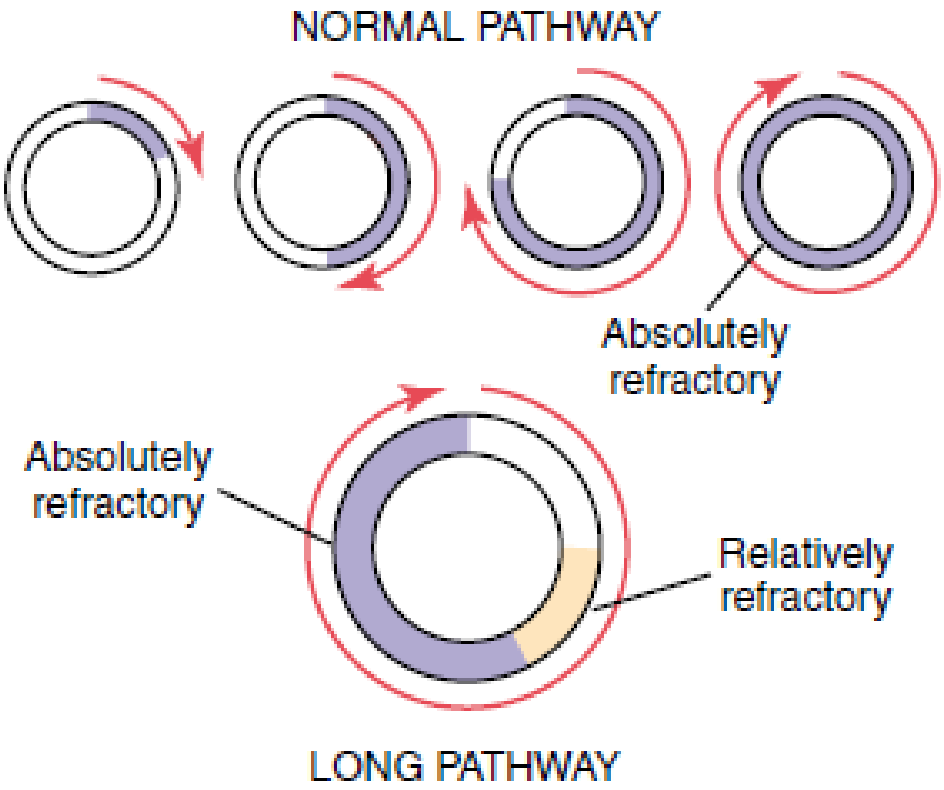
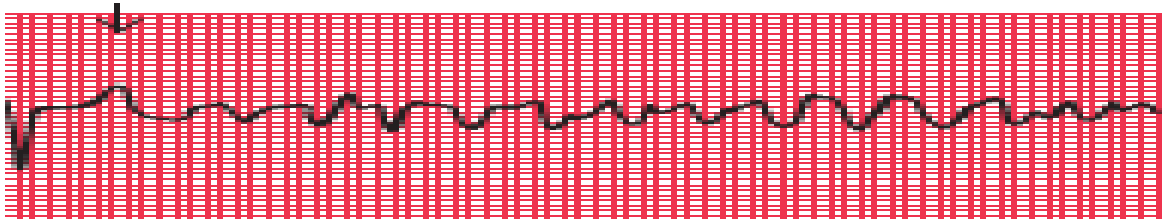


Figure 13-15 Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

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 like SVT



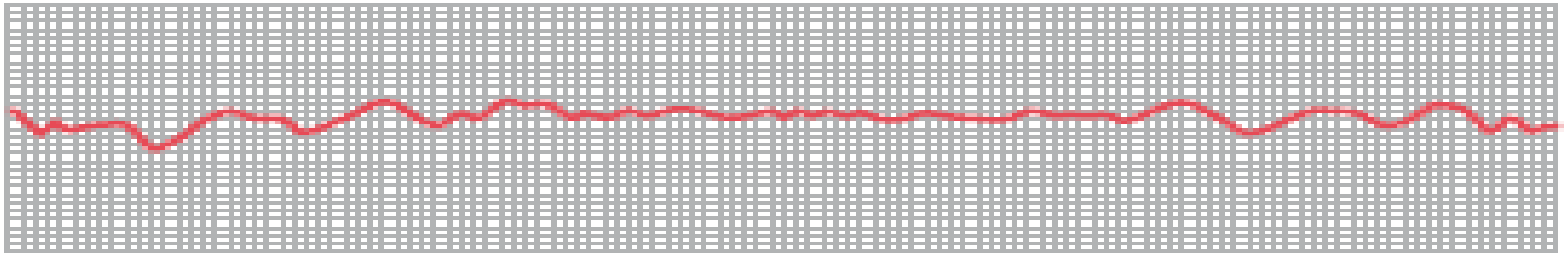
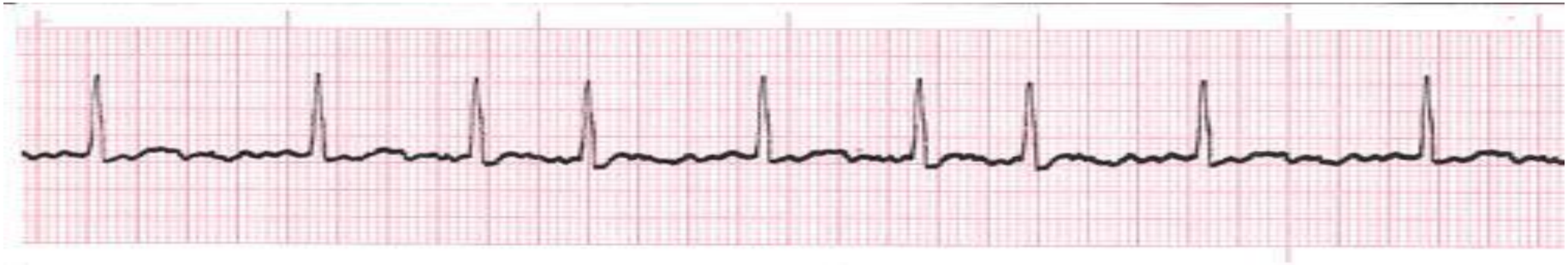


Figure 13-17 Ventricular fibrillation (lead II).

- 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.

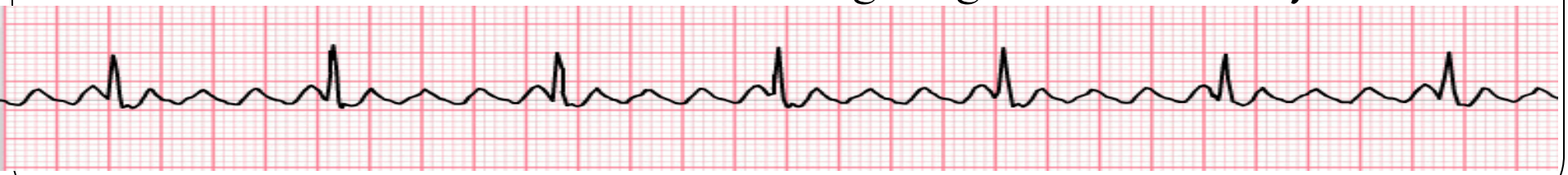
Atrial fibrillation

- The 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

- Type of supraventricular tachycardia 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



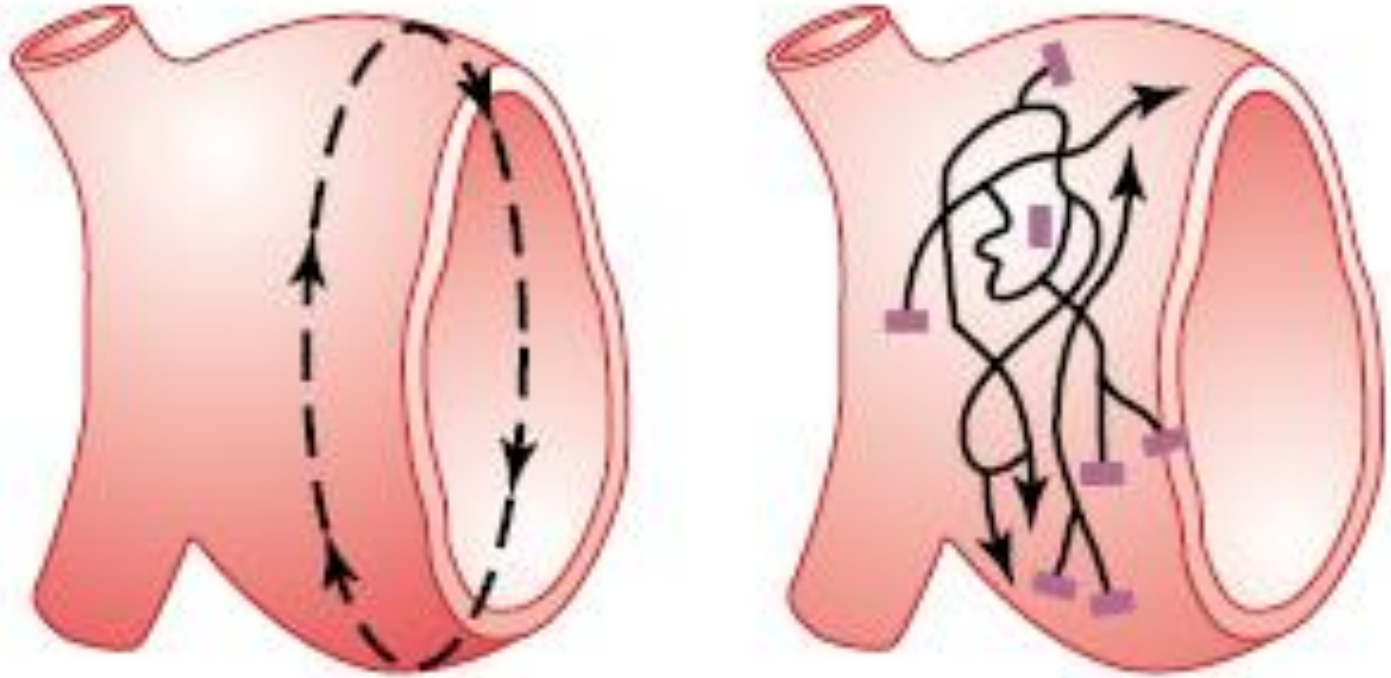


Figure 13-20 Pathways of impulses in atrial flutter and atrial fibrillation.

Conduction Abnormalities

- Conduction problems in the AV node and His bundle (Atrioventricular Block)
- Conduction problems in the right and left bundle branches – bundle branch block

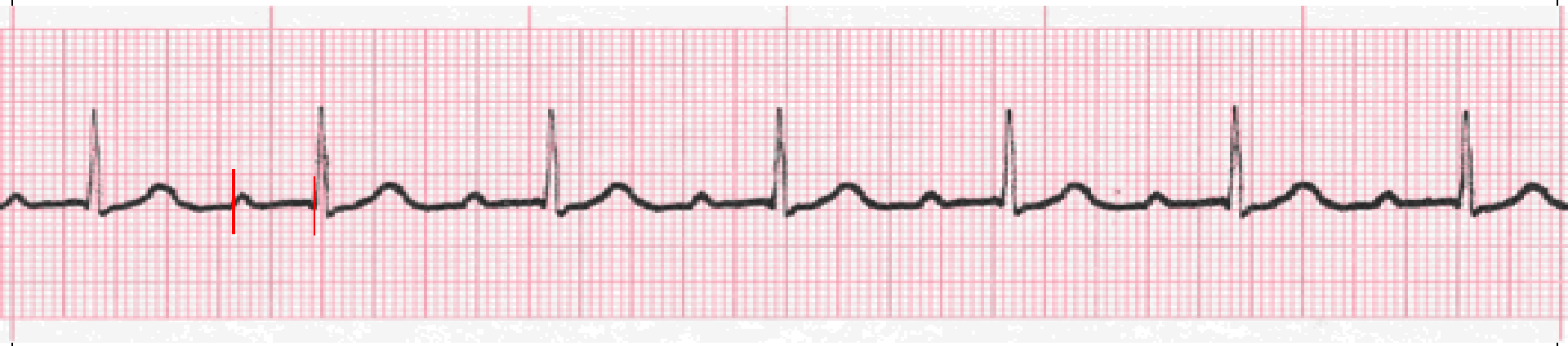
Atrioventricular Block

- Results from conditions that can either decrease the rate of impulse conduction in the AV bundle or block the impulse entirely. Like Ischemia of the AV node or AV bundle, compression of the AV bundle by a scar tissue or extreme vagal stimulation.

- 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

- Sometimes the depolarization wave completely fails to pass through the AV node or the bundle of His. When this occurs intermittently, 'second degree heart block' is said to exist
- The underlying causes of second degree heart block are the same as those of first degree block
- Types:

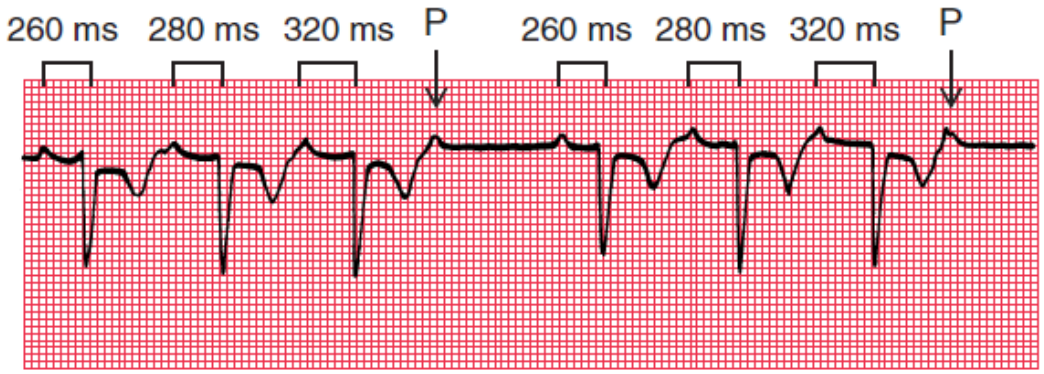
1. Mobitz type 1
2. Mobitz type 2
3. Fixed ratio blocks

Mobitz type 1

- 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

Fig. 2.3

Second degree heart block (Wenckebach (Mobitz type 1))



Note

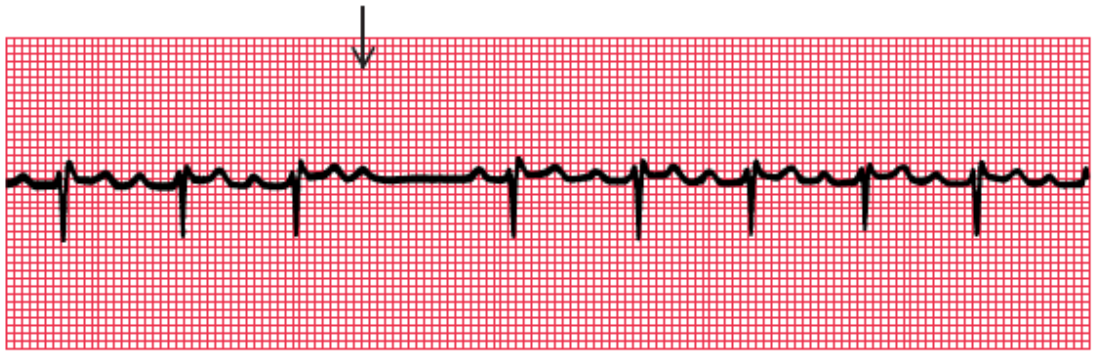
- Progressive lengthening of the PR interval
- One nonconducted P wave
- Next conducted beat has a shorter PR interval than the preceding conducted beat

Mobitz type 2

- Most beats are conducted with a constant PR interval, but occasionally there is an atrial depolarization without a subsequent ventricular depolarization.

Fig. 2.4

Second degree heart block (Mobitz type 2)



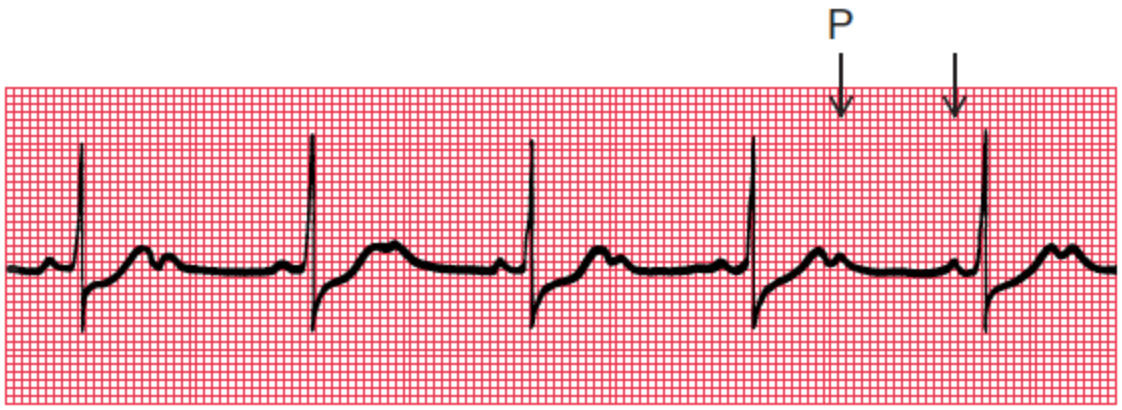
Note

- PR interval of the conducted beats is constant
- One P wave is not followed by a QRS complex

Fixed ratio blocks

- 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.

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

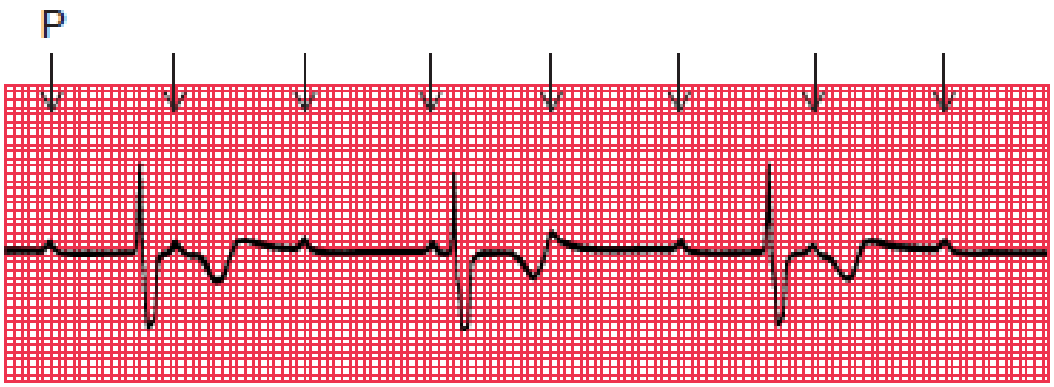
- Occurs with complete block of the impulse from the atria into the ventricles.

- 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



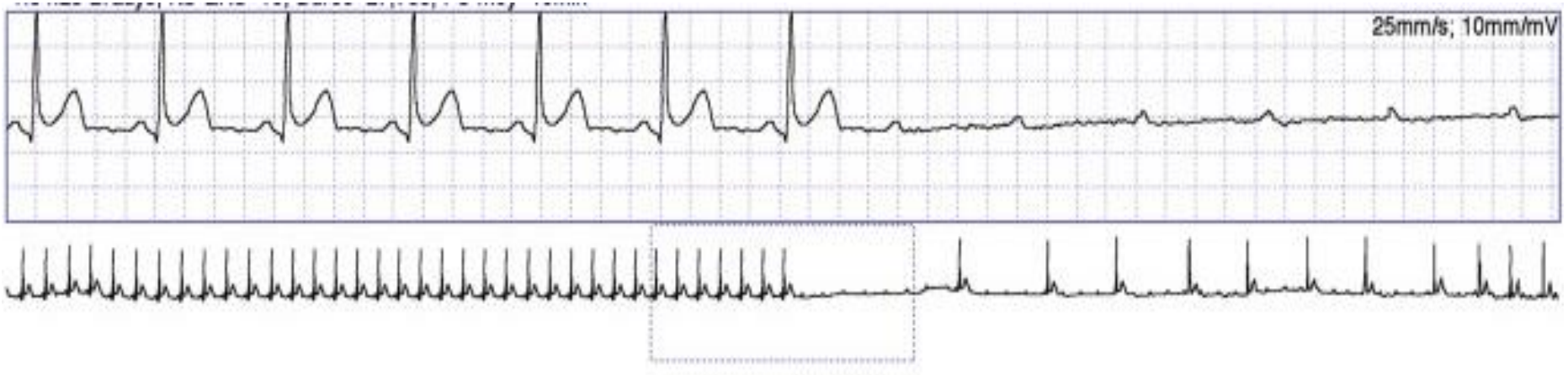
Note

- 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.



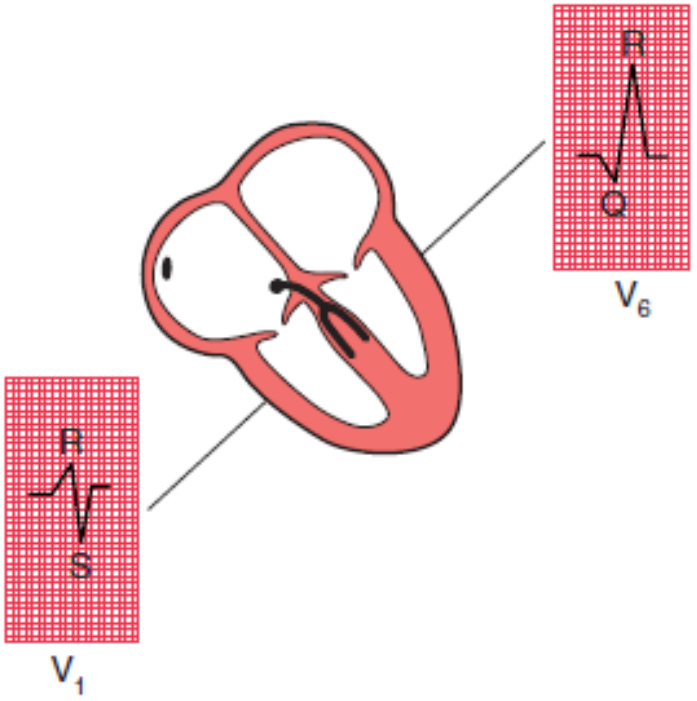
Data obtained from Holter monitor

Left bundle branch block LBBB

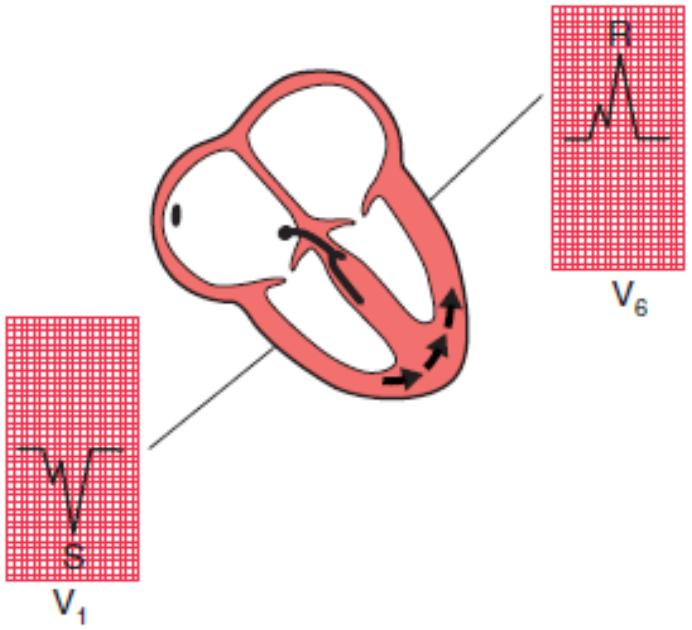
- If conduction down the left bundle branch fails, the septum becomes depolarized from right to left, causing a small Q wave in lead V1, and an R wave in lead V6
- Best seen in lead V6, where there is a broad QRS complex with a notched top, which resembles the letter 'M' and is therefore known as an 'M' pattern .The complete picture, with a 'W' pattern in lead V1, is often not fully developed.
- The QRS complex is wide >0.12sec
- T wave inversion occurs in some lateral leads
- Left axis deviation might be present

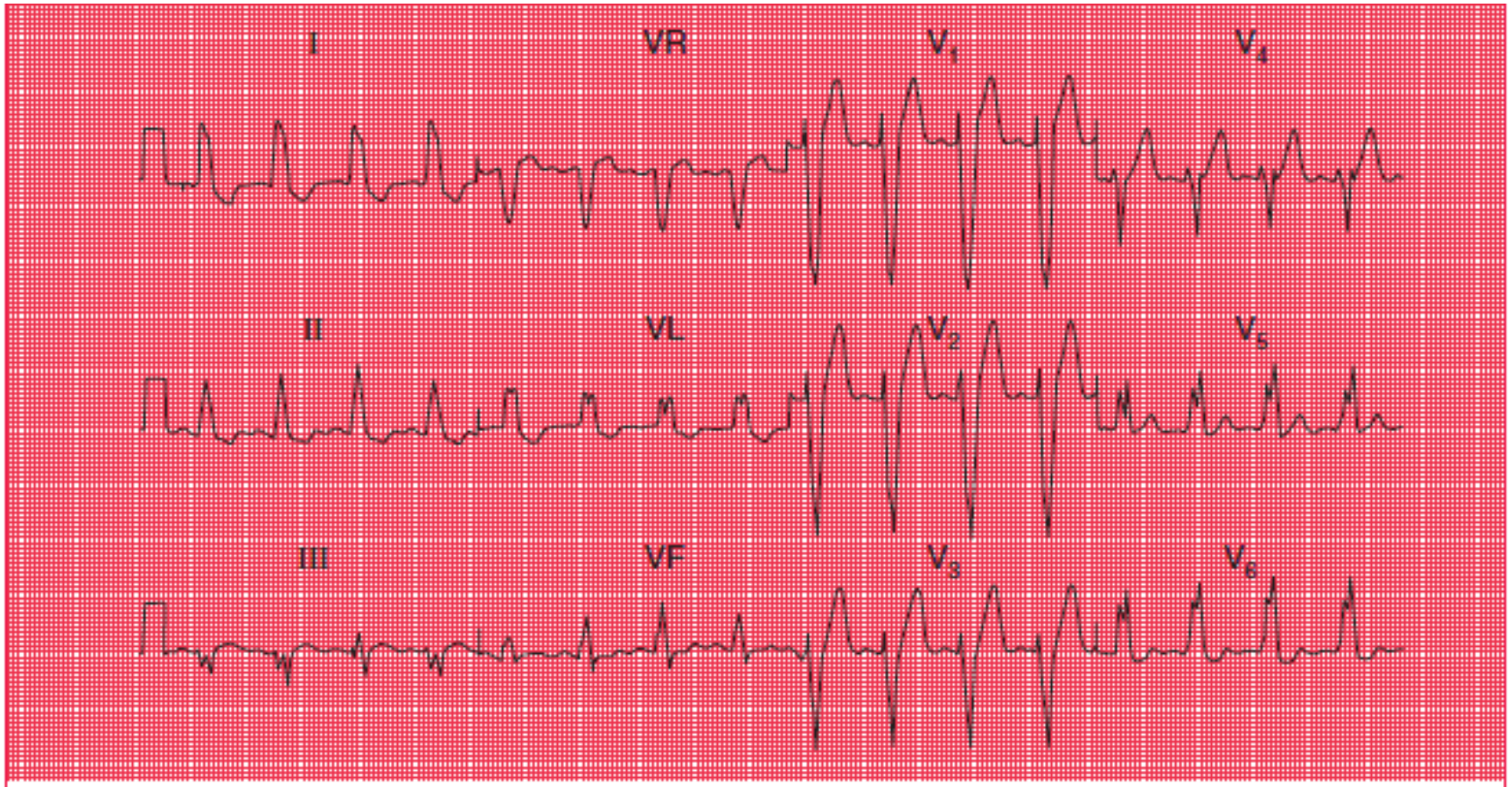
Fig. 1.20

Shape of the QRS complex: third stage



Conduction in left bundle branch block: third stage





Sinus rhythm with left bundle branch block

Note

- Sinus rhythm, rate 100/min
- Normal PR interval
- Normal cardiac axis
- Wide QRS complexes (160 ms)
- M pattern in the QRS complexes, best seen in leads I, VL, V_5 and V_6
- Inverted T waves in leads I, II, VL

Right bundle branch block RBBB

- In RBBB, no conduction occurs down the right bundle branch but the septum is depolarized from the left side as usual
- Best seen in lead V1, where there is an RSR1 pattern
- It takes longer than normal for excitation to reach the right

ventricle .The right ventricle therefore depolarizes after the left. This causes a second R wave in V1 and a wide, slurred S wave in lateral leads

- The QRS complex is wide >0.12 sec.
- T wave inversion might be seen in V1-V3
- Right axis deviation might be present

Fig. 1.20

Shape of the QRS complex: third stage

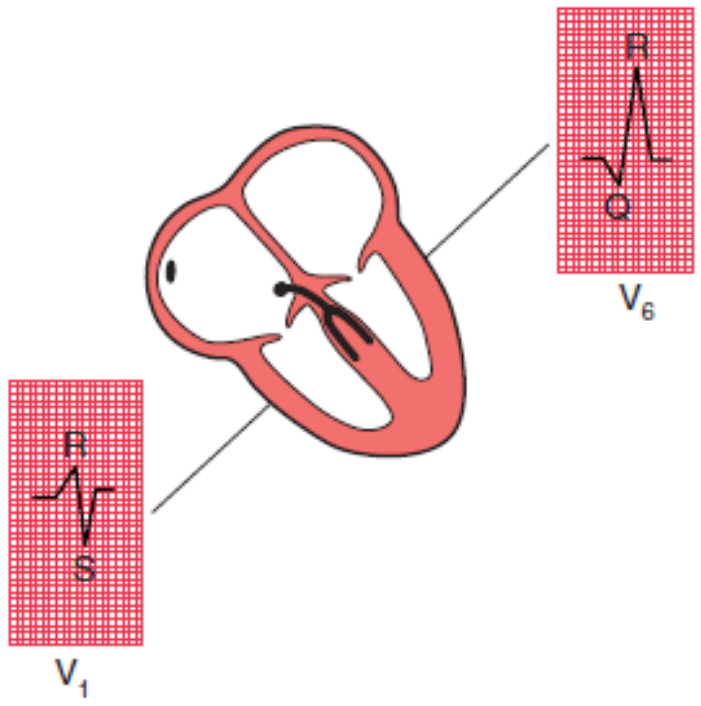
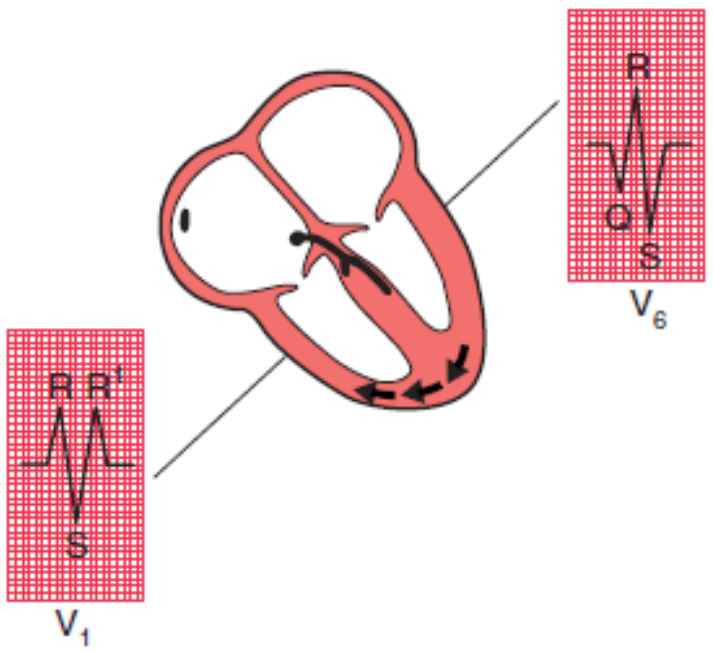
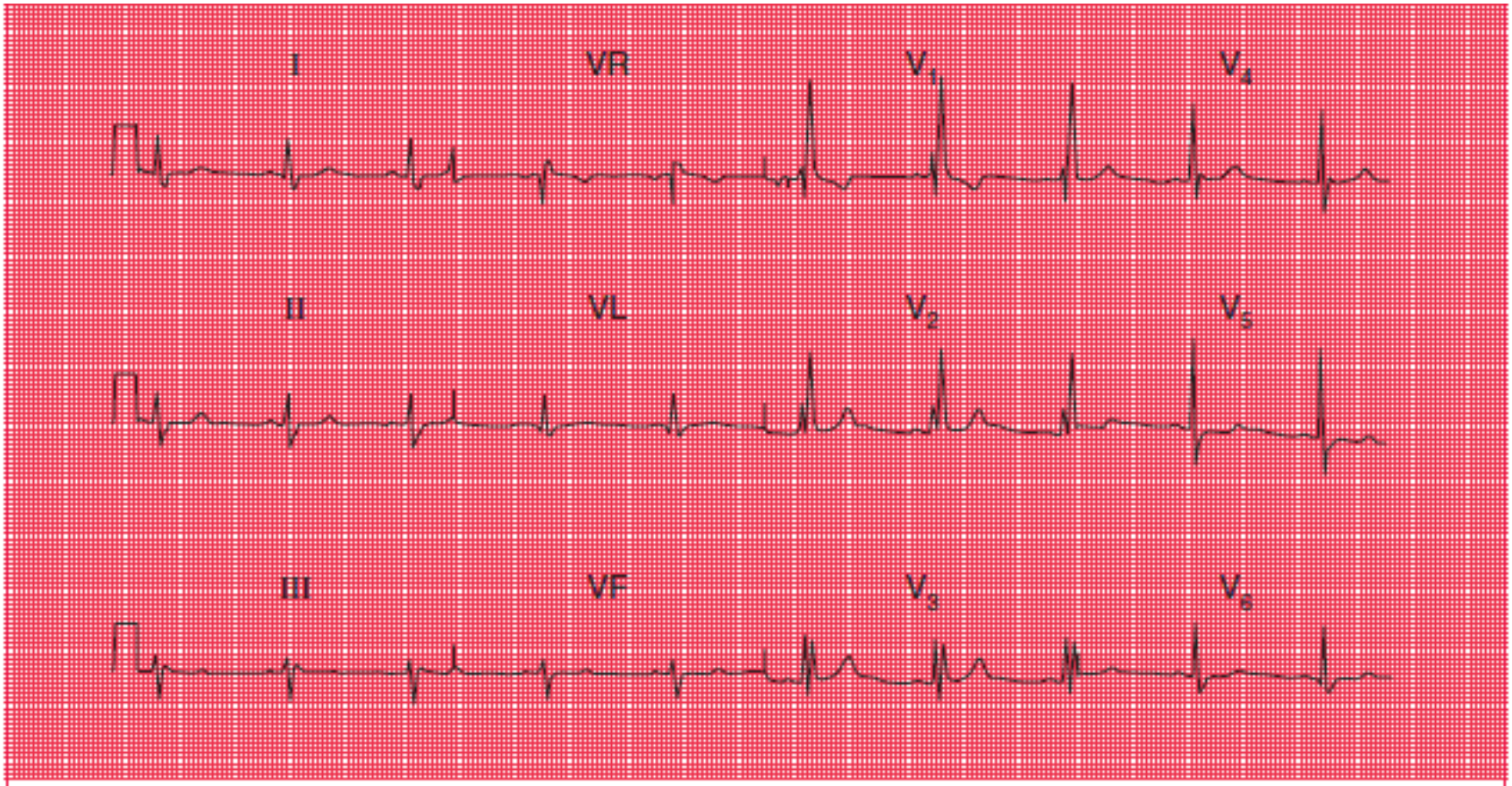


Fig. 2.11

Conduction in right bundle branch block: third stage





Sinus rhythm with right bundle branch block

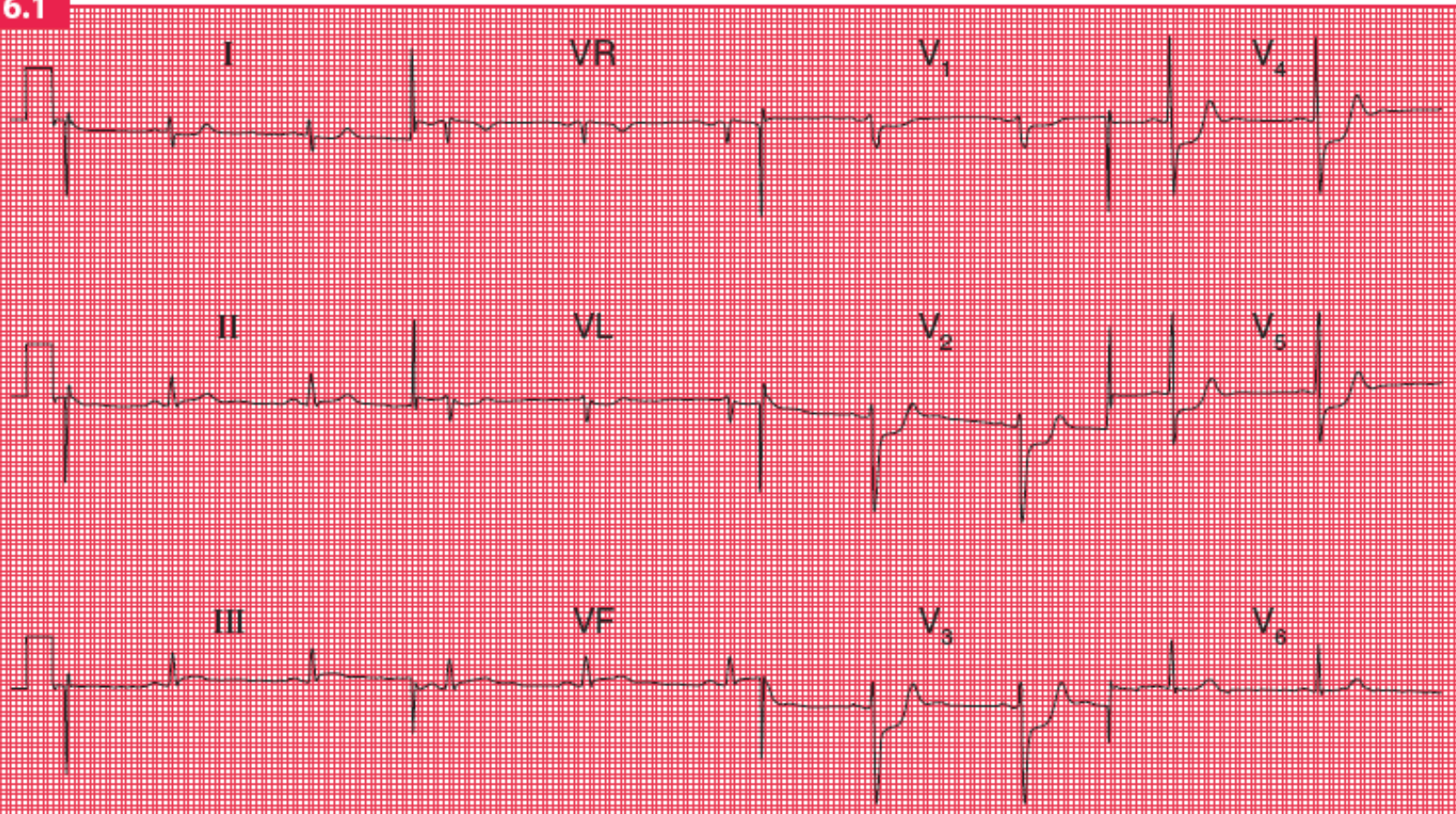
Note

- Sinus rhythm, rate 60/min
- Normal PR interval
- Normal cardiac axis
- Wide QRS complexes (160 ms)
- RSR[1] pattern in lead V_1 and deep, wide S waves in lead V_6 ,I & II
- Normal ST segments and T waves

Angina pectoris

- ECG changes are noticed while the patient is in pain, once the pain has resolved the ECG returns to normal.
- Horizontal depression of the ST segment, is usually a sign of ischaemia as opposed to infarction.
- 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. 6.1



ST segment depression in unstable angina

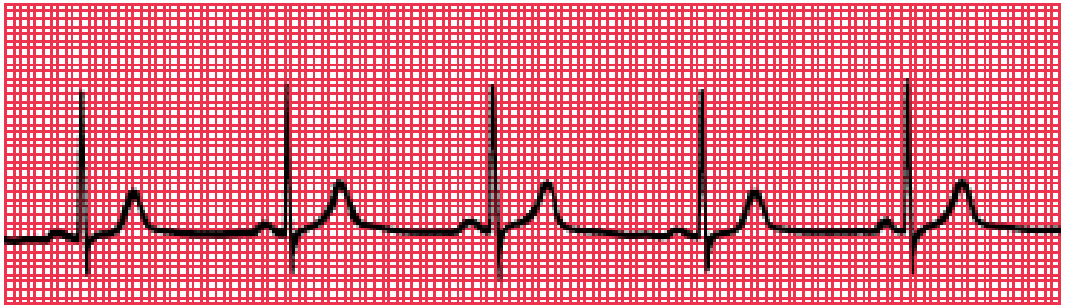
Note

- Sinus rhythm, rate 60/min
- Normal axis
- Normal QRS complexes
- ST segments depressed horizontally in leads V2-V5
- Normal T waves

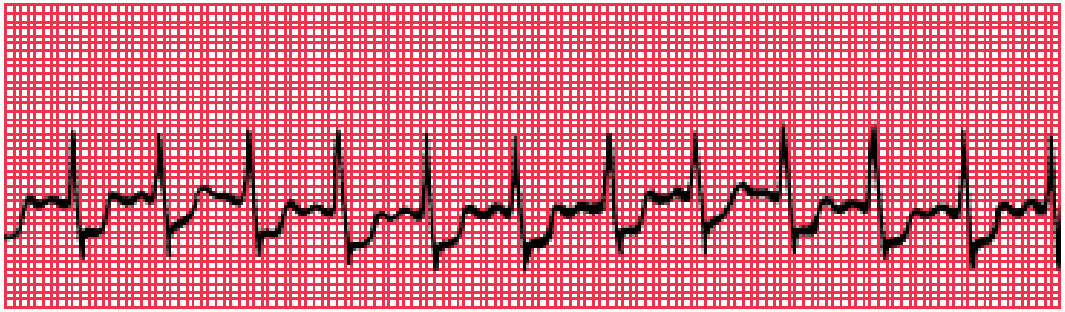
Fig. 4.14

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

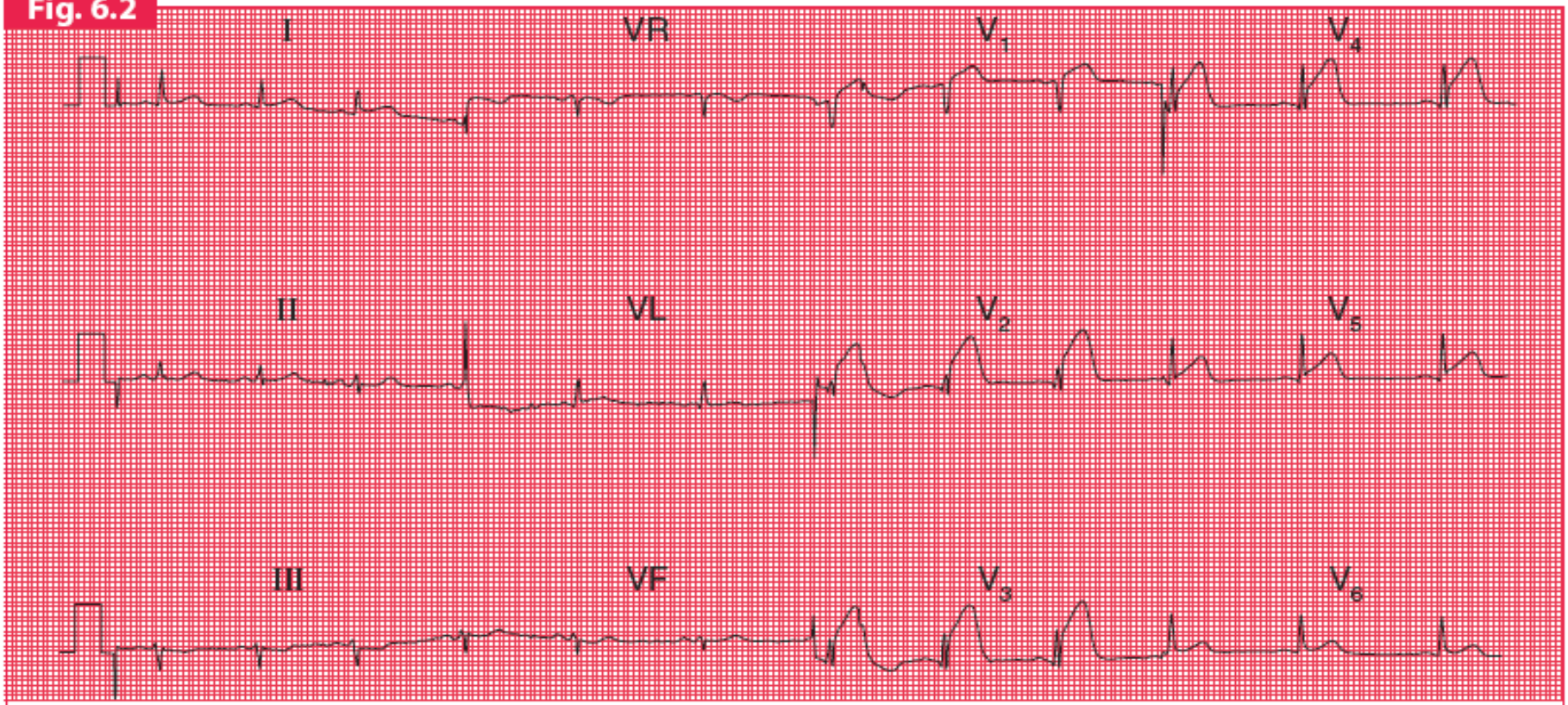
ECG changes seen in Myocardial infarction (MI)

- ECG is very useful for diagnosing MI and locating areas of infarction.
- The earliest sign of infarction is ST segment elevation
- ST segment elevation occurs in the leads corresponding to the part of the heart that is damaged:
- Leads V1-V4 with anterior wall infarction,
- 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 in at least two contiguous limb leads (e.g. I and VL; III and VF), or more than 2 mm of ST segment elevation in at least two contiguous precordial leads

- 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

Fig. 6.2



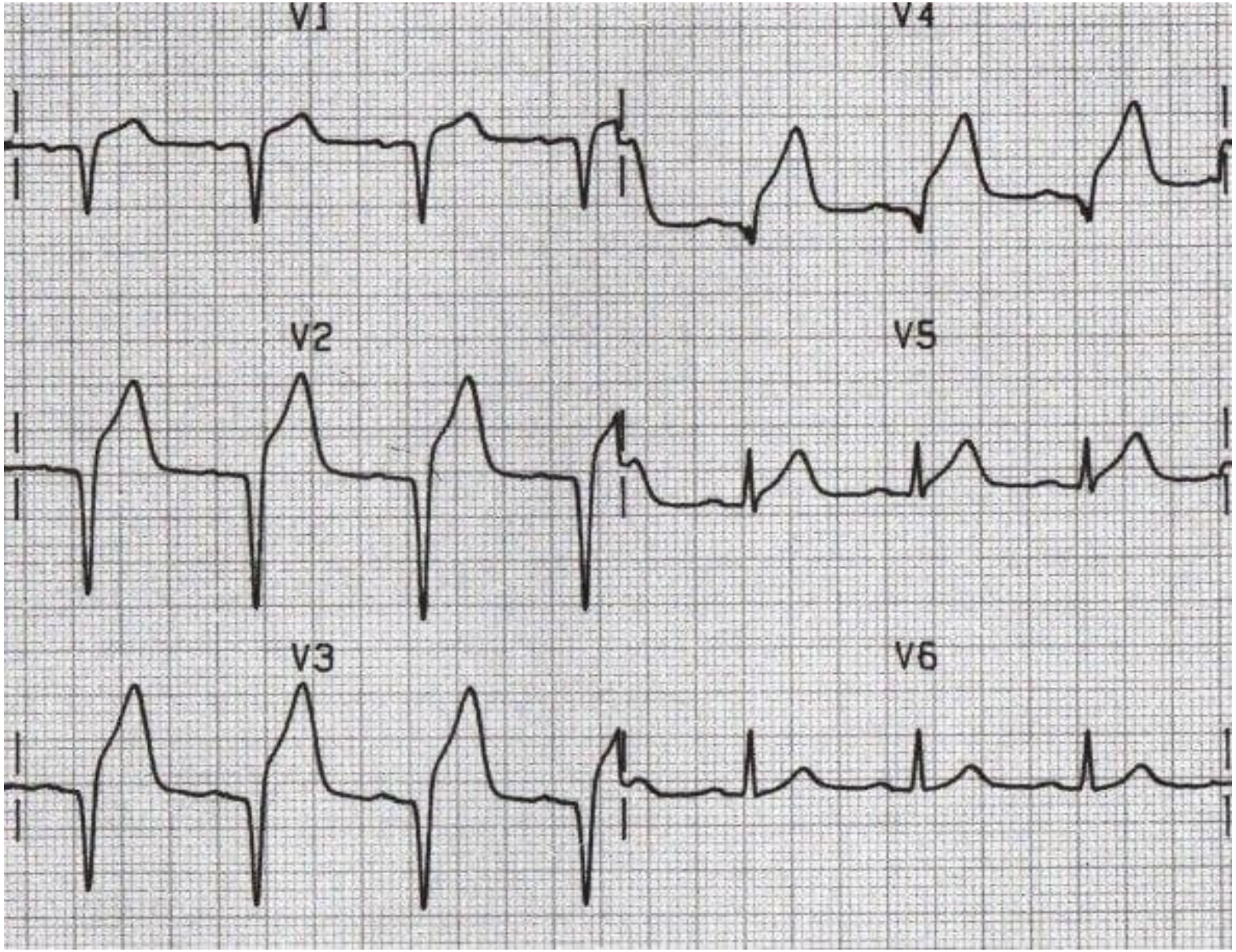
ST segment elevation in acute anterior ST segment elevation myocardial infarction

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>**

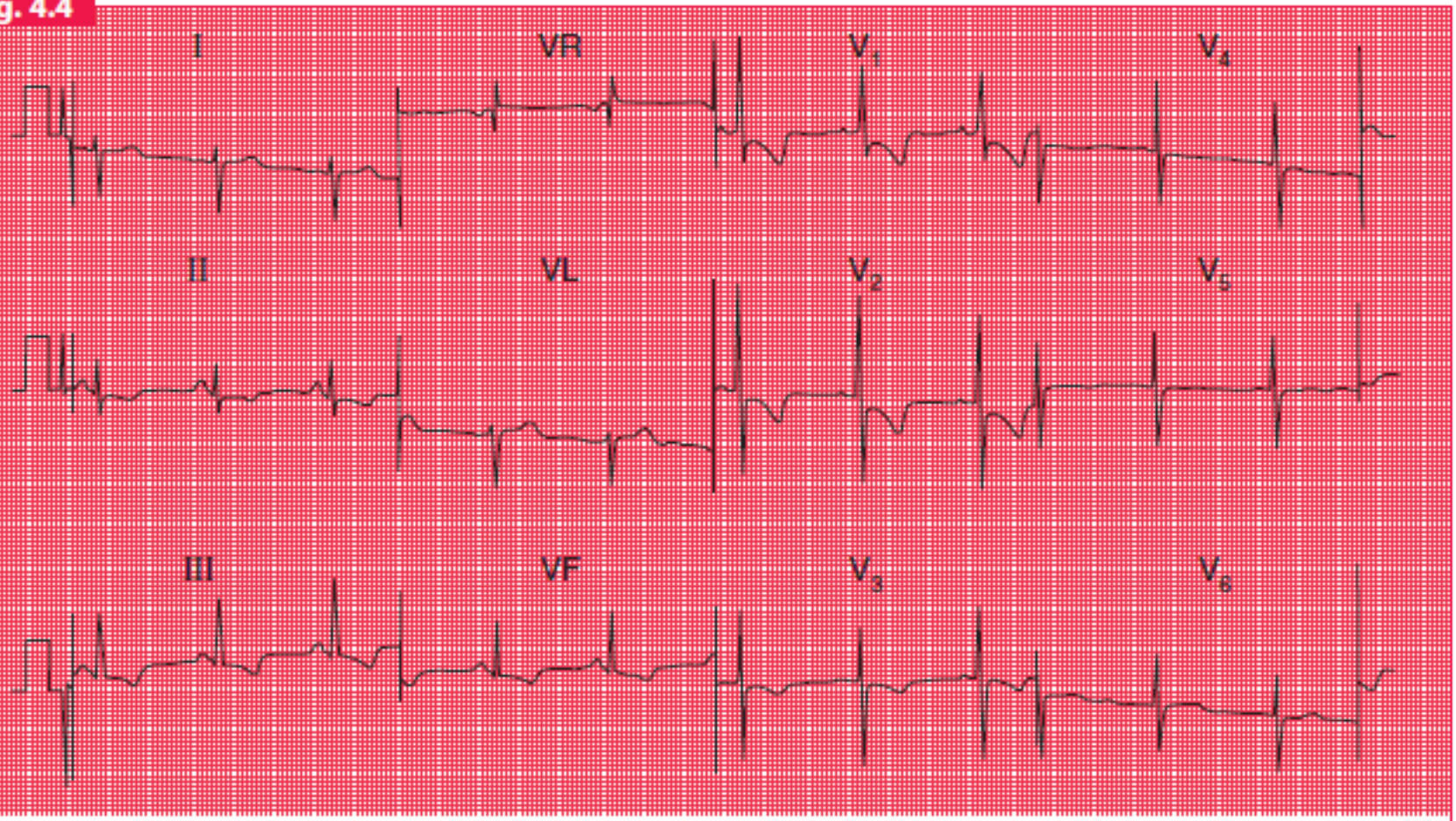


Pathological Q waves in V1-V4

Right Ventricular hypertrophy

- Best seen in the right ventricular leads (especially V1).
- The height of the R wave exceeds the depth of the S wave in V1
- Deep S wave in lead V6
- Right axis deviation
- Inversion of the T waves might be seen
- P pulmonale might be seen

Fig. 4.4



Severe right ventricular hypertrophy

Note

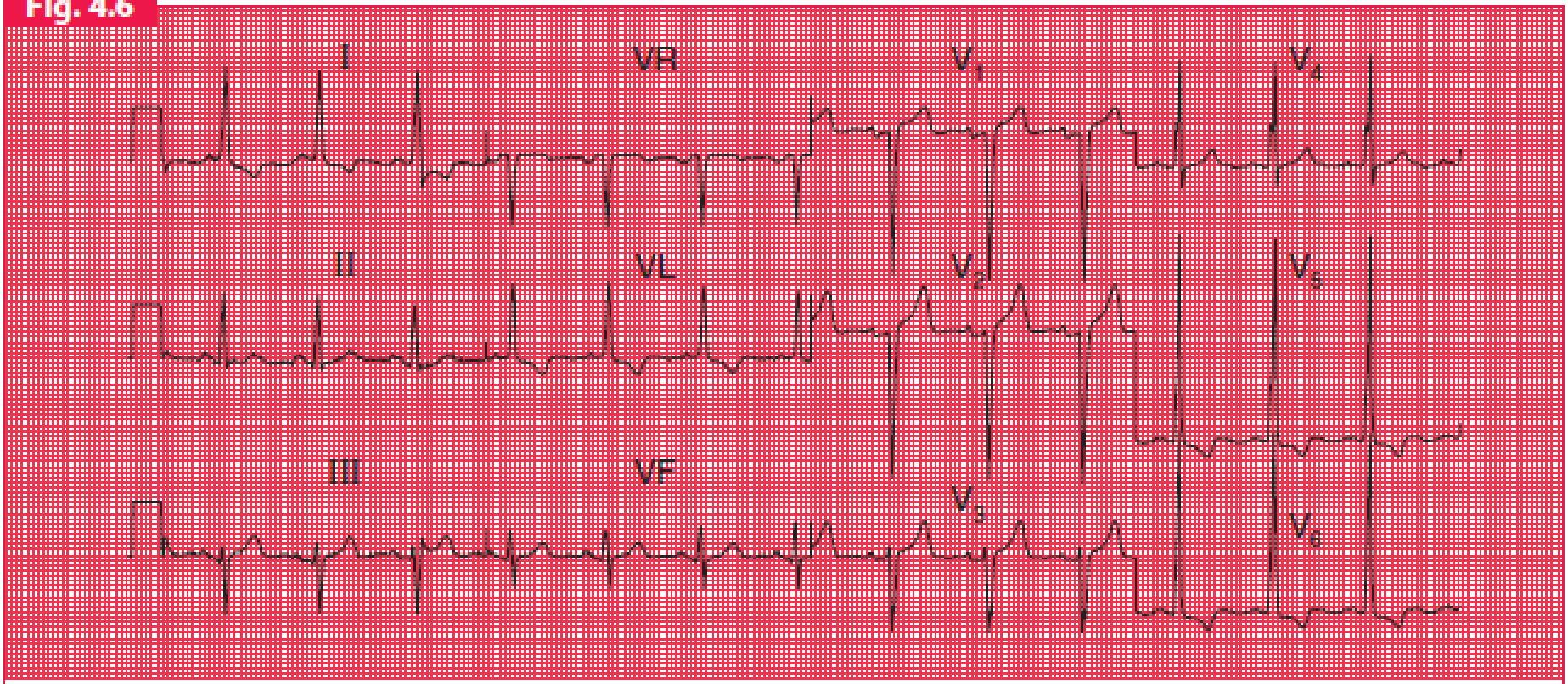
- Sinus rhythm, rate 63/min
- Right axis deviation (deep S waves in lead I)
- Dominant R waves in lead V_1
- Inverted T waves in leads II, III, VF and V_1–V_3

Left ventricular hypertrophy

- A tall R wave (greater than 25 mm- 30 mm) in lead V5 or V6
- A deep S wave in lead V1 or V2
- With significant hypertrophy, inverted T waves are seen in lateral leads.
- Left axis deviation occasionally occurs

✓ It is difficult to diagnose minor degrees of left ventricular hypertrophy from the ECG.

Fig. 4.6

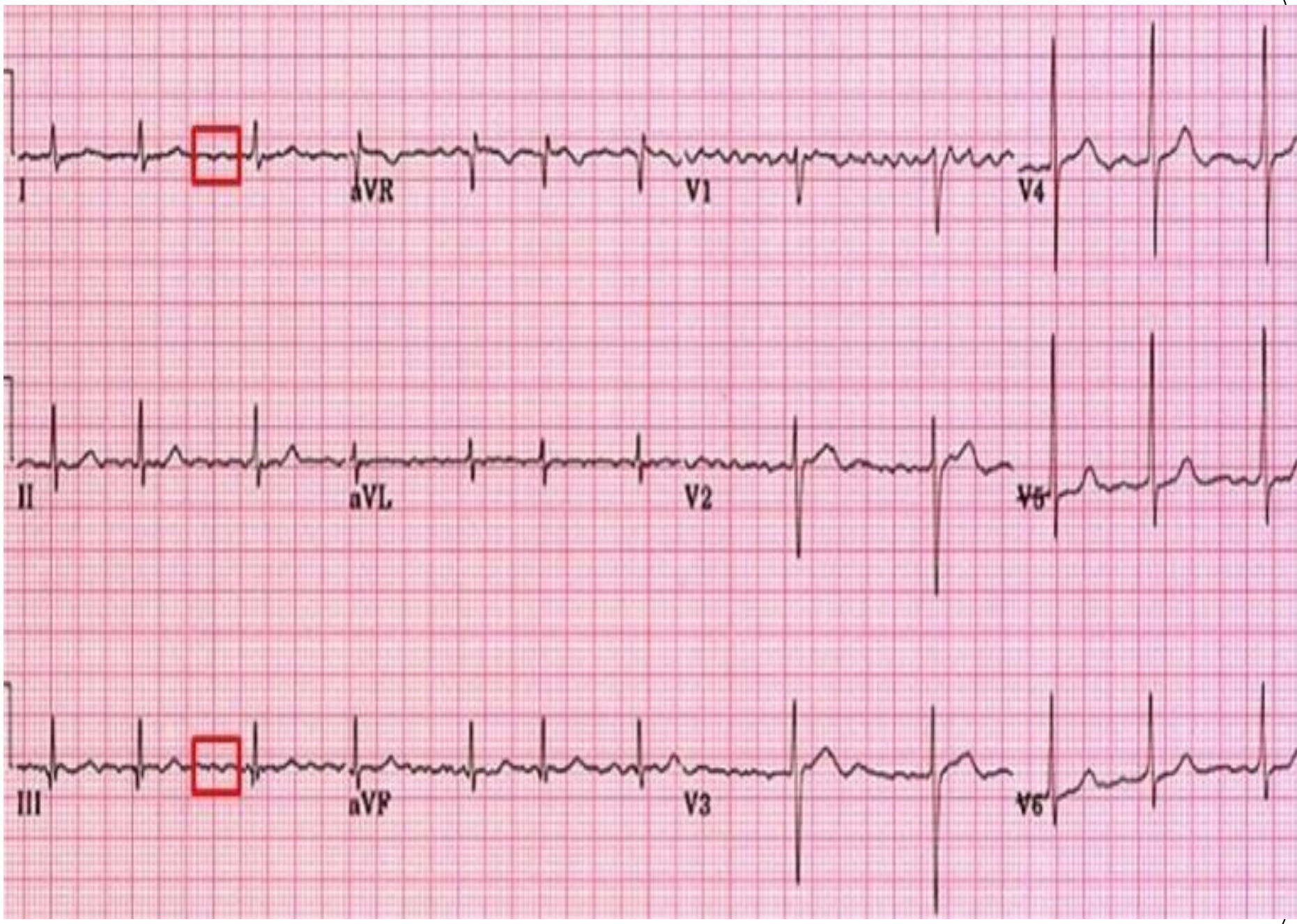


Left ventricular hypertrophy

Note

- Sinus rhythm, rate 83/min
- Normal axis
- Tall R waves in leads V_5–V_6 (R wave in lead V_5, 40 mm) and deep S waves in leads V_1–V_2
- Inverted T waves in leads I, VL and V_5–V_6

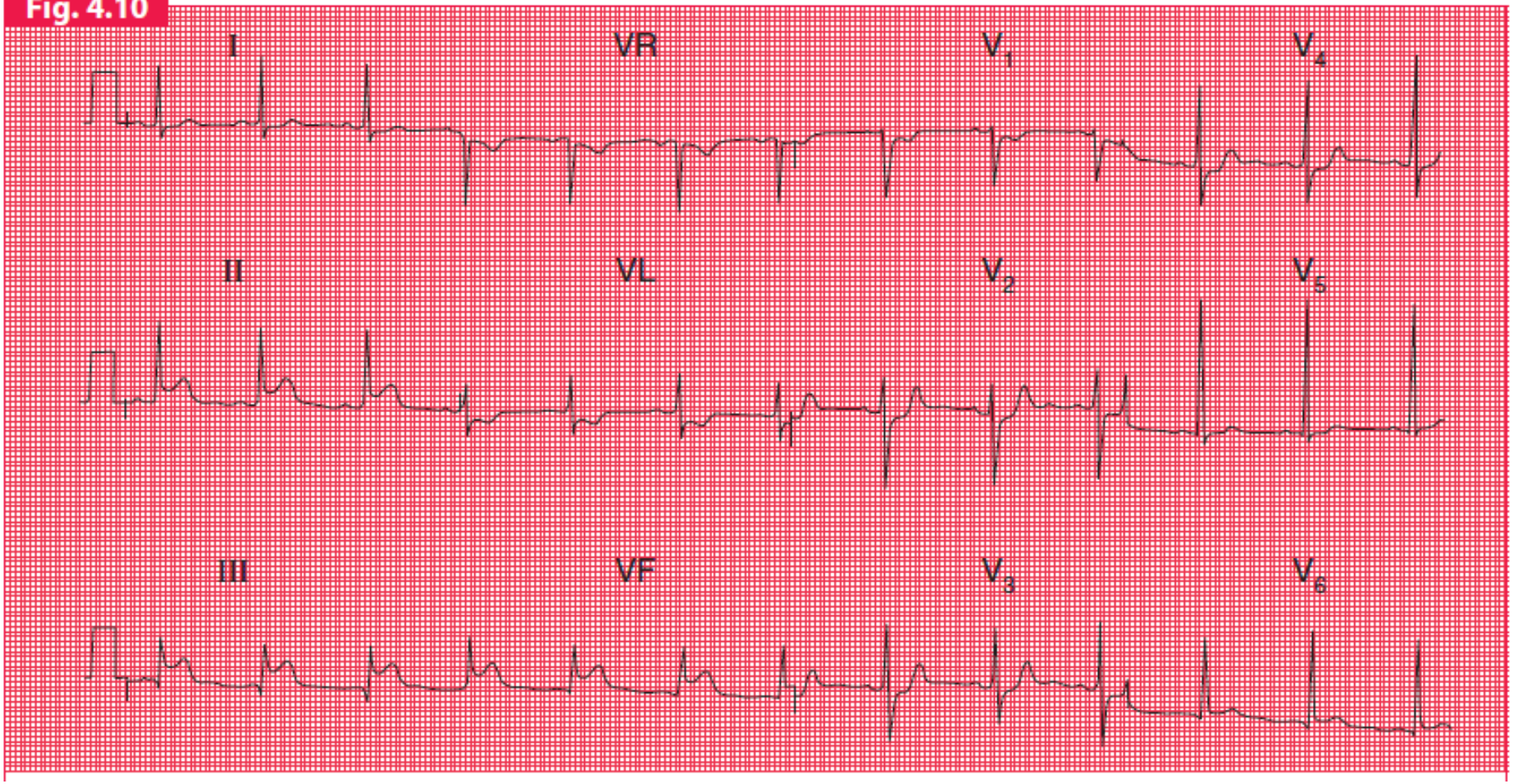
Abnormal ECGs


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

- P waves are replaced by rapidly quivering small deflection of variable amplitude
- Irregularly irregular ventricular rate
- Narrow QRS complexes

- **Atrial Fibrillation**

Fig. 4.10

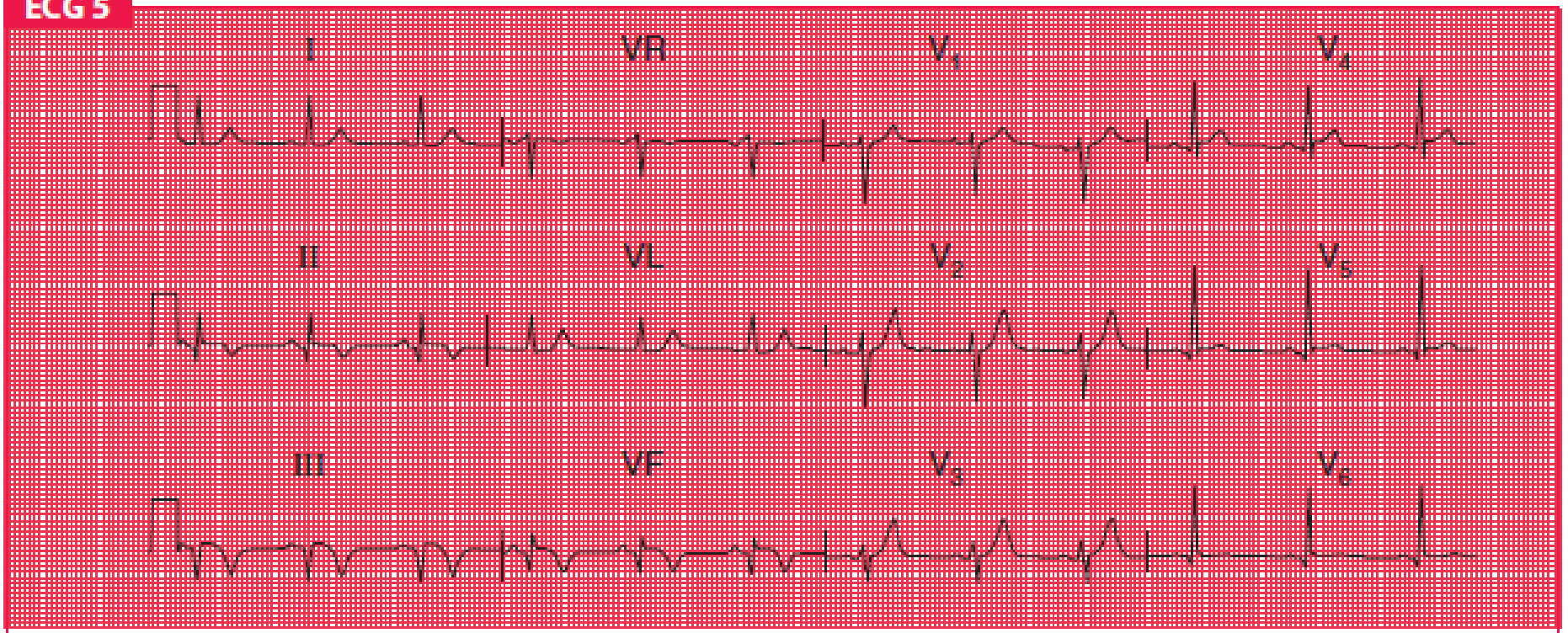


- Sinus rhythm, rate 70/min
- Normal axis

- Normal QRS complexes
- Raised ST segments in leads II, III and VF
- Inverted T waves in lead VL (abnormal) and in lead V_1 (normal)

Acute inferior infarction

ECG 5

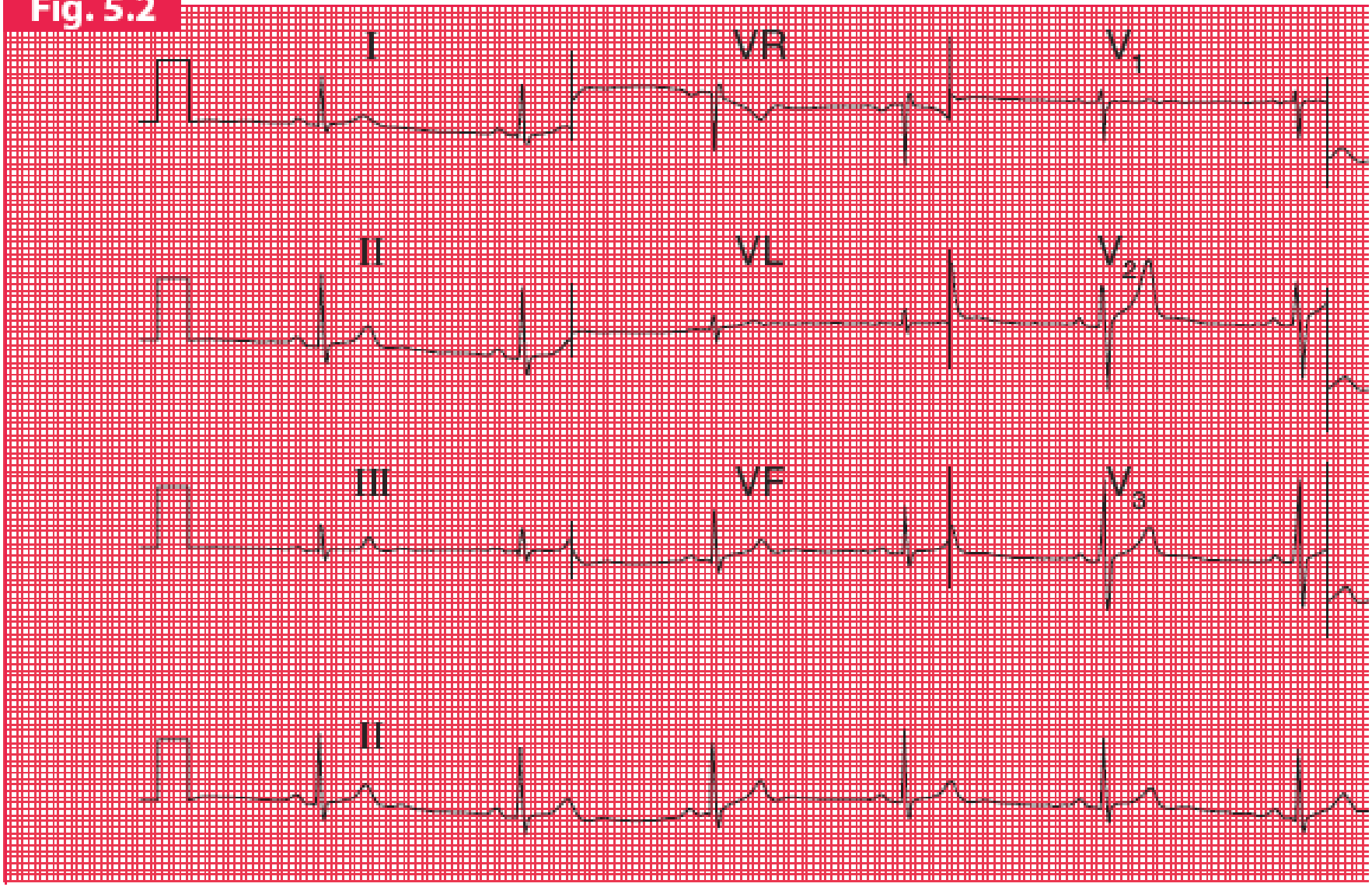


ECG from a 60-year-old man who had had severe chest pain 2 days earlier

- Sinus rhythm
- Normal PR interval
- Normal axis
- QRS complex has Q waves in leads II, III and VF
- ST segment isoelectric
- T waves inverted in leads II, III and VF.

Old inferior myocardial infarction

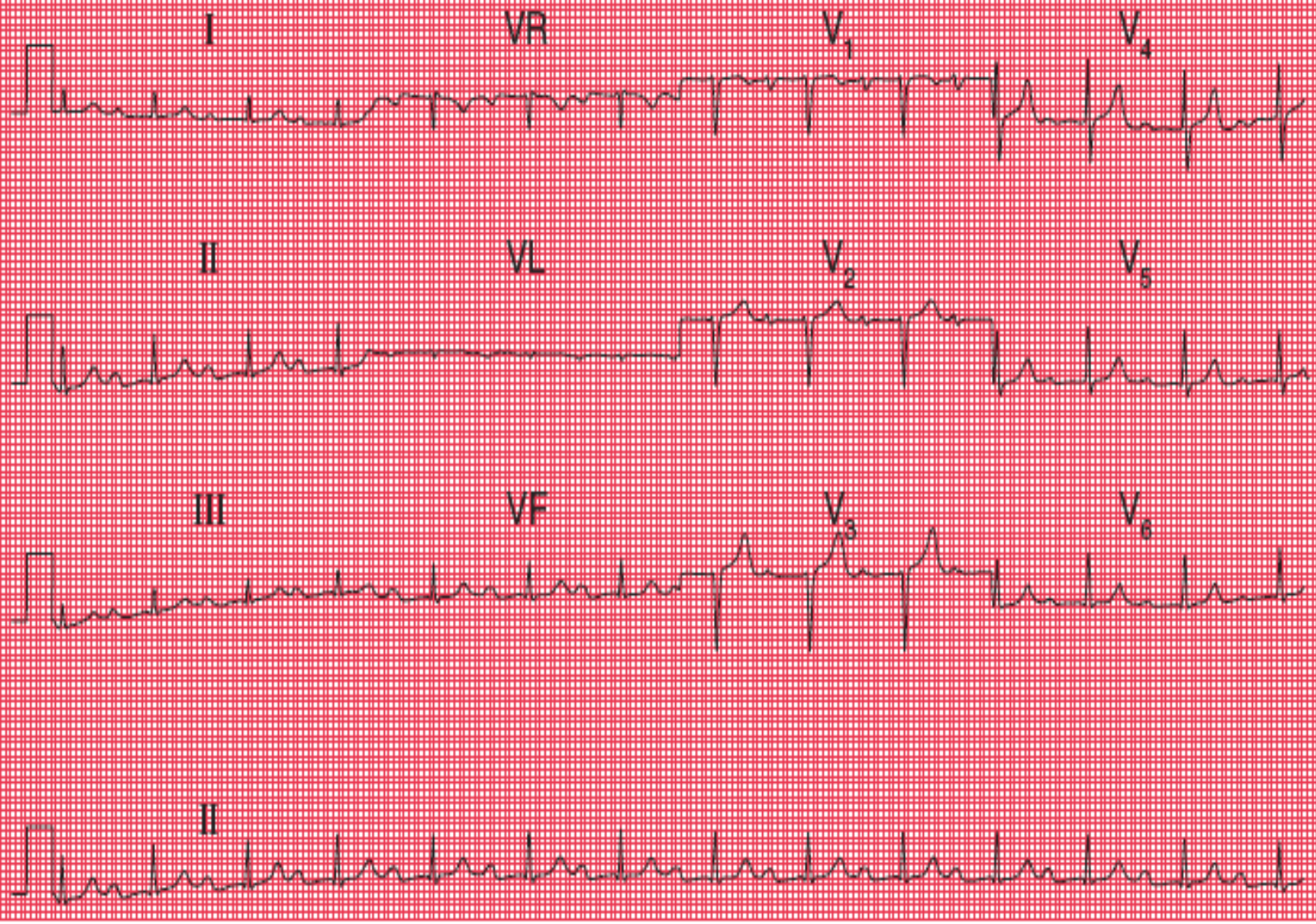
Fig. 5.2



- Sinus rhythm, rate 47/min
- Normal QRS complexes, ST segments and T waves

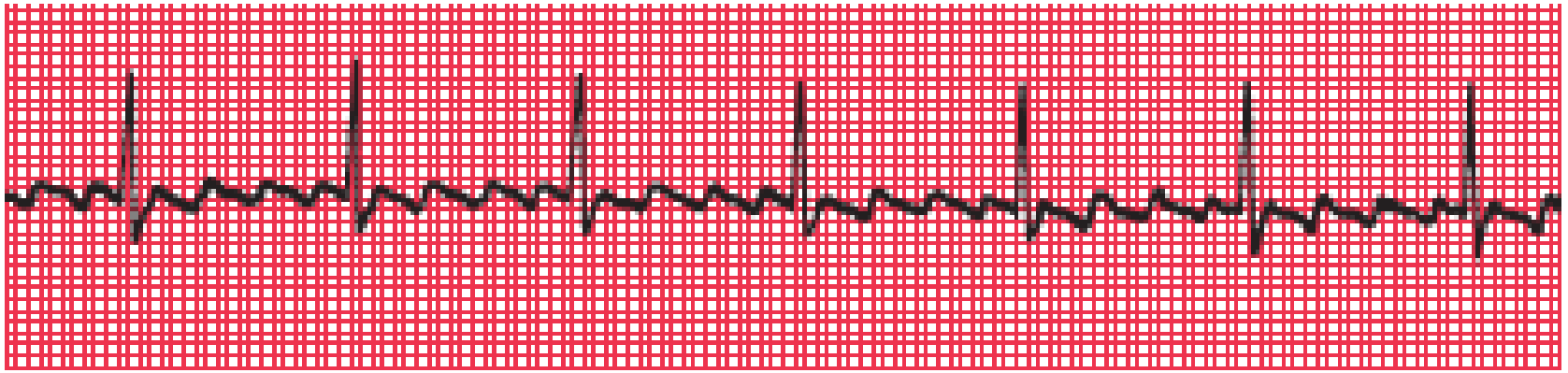
Sinus bradycardia in an athlete

Fig. 5.8



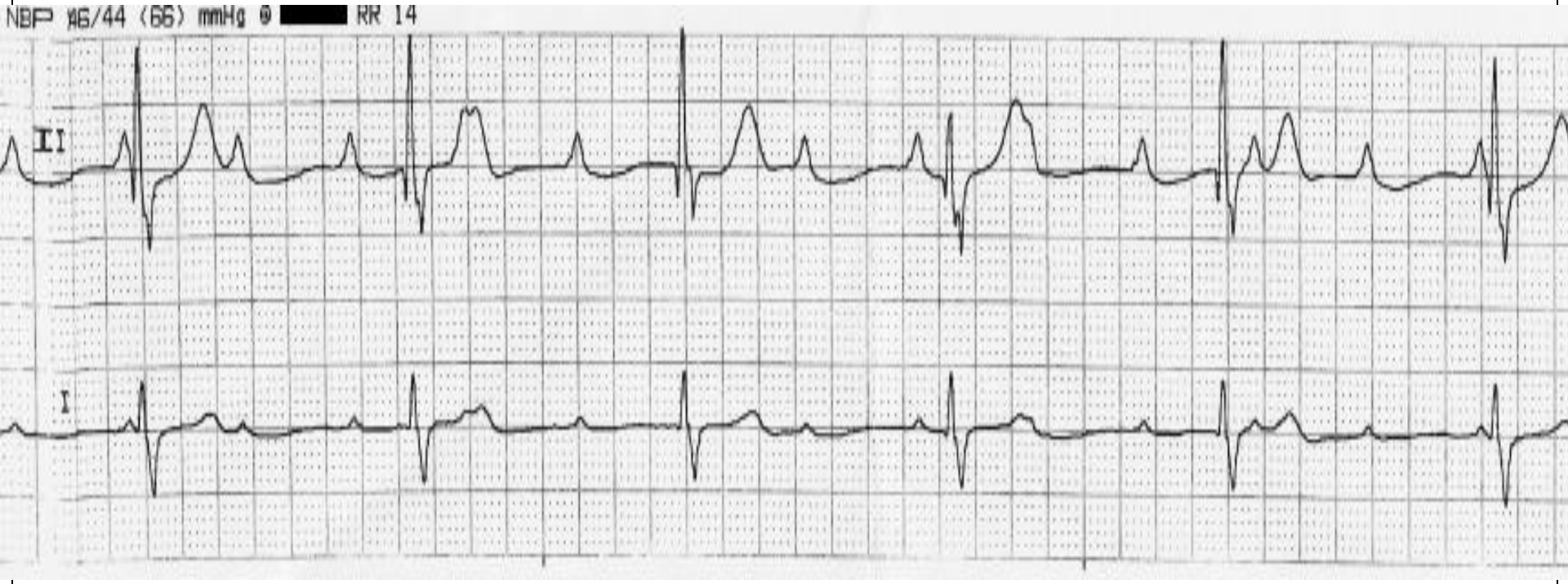
- Sinus rhythm, rate 80/min
- PR interval prolonged at 336 ms
- Constant PR interval in all beats
- Loss of the R wave in lead V_3 could indicate an old anterior infarction, otherwise QRS complexes, ST segments and T waves are normal

First degree heart block



- P waves can be seen at a rate of 300/min, giving a 'sawtooth' appearance
- There are four P waves per QRS complex (arrowed)
- Ventricular activation is perfectly regular at 75/min

Atrial flutter



- Atrial rate 100 b.p.m
- Ventricular rate 40 b.p.m
- No relationship between the P waves and QRS complexes

3rd degree block

Thank You

BEST OF LUCK