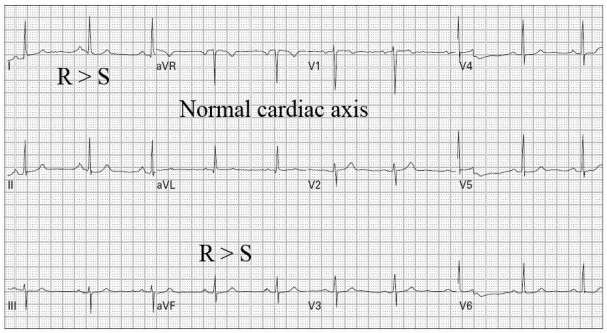


FIGURE 1.41 Normal and abnormal cardiac axis.

STEP 05 P WAVE

The normal P wave has a width of less than 3 small squares and amplitude of less than 2.5 small squares. In sinus rhythm, the P wave is normally upright in all leads except aVR. When the QRS complex is predominantly downward in lead aVL, the P wave may also be inverted normally. When you would like to identify the P wave look first at leads II, III, aVF, and leads V_5 and V_6. In these leads, the P waves should be upright. If the P wave is difficult to be seen in these leads, look then at other leads.
Abnormalities of P wave may take one of the following fashions:

Absence of P wave	This occurs in atrial fibrillation, atrial flutter, junctional ectopic beat, junctional escape beat, junctional tachycardia (SVT), idionodal rhythm, ventricular ectopic beat, ventricular escape beat, ventricular tachycardia, and idioventricular rhythm. It may also occur in hyperkalemia and in sinoatrial block (discussed later)
Inverted P wave in lead I	This may indicate dextrocardia or improper lead placement
Abnormal P wave shape	This may indicate atrial ectopic beat, atrial tachycardia, atrial escape beat, and atrial escape rhythm
Widened P wave (more than 2.5 small squares)	This may occur in atrial infarction, intra-atrial conduction defect, and left atrial enlargement

Now look especially at leads II and V_1 to diagnose atrial enlargement (FIGURE 1.42). Because the sinus node is located in the right atrium, the right atrium begins to depolarize before the left atrium and finishes earlier as well. Therefore, the first part of the P wave predominantly represents right atrial depolarization and the second part represents left atrial depolarization. The P wave should be upright in lead II and biphasic in lead V_1 when you are looking at these leads. The criteria designed to diagnose atrial enlargement via the EKG are as follows:

Peaked tall P wave	This indicates right atrial enlargement. In this condition, the P waves has an amplitude exceeding 2.5 mm in the inferior leads (P-pulmonale) with no change in the duration of the P wave i.e. < 3 mm
Bifid notched P wave	This indicates left atrial enlargement. The amplitude of the terminal (negative) component of the P wave may be increased and must descend at least 1 mm below the isoelectric line in lead V_1 giving the appearance of notched P wave (P-mitrale). The duration of the P wave is increased, and the terminal (negative) portion of the P wave must be at least one small square in width.

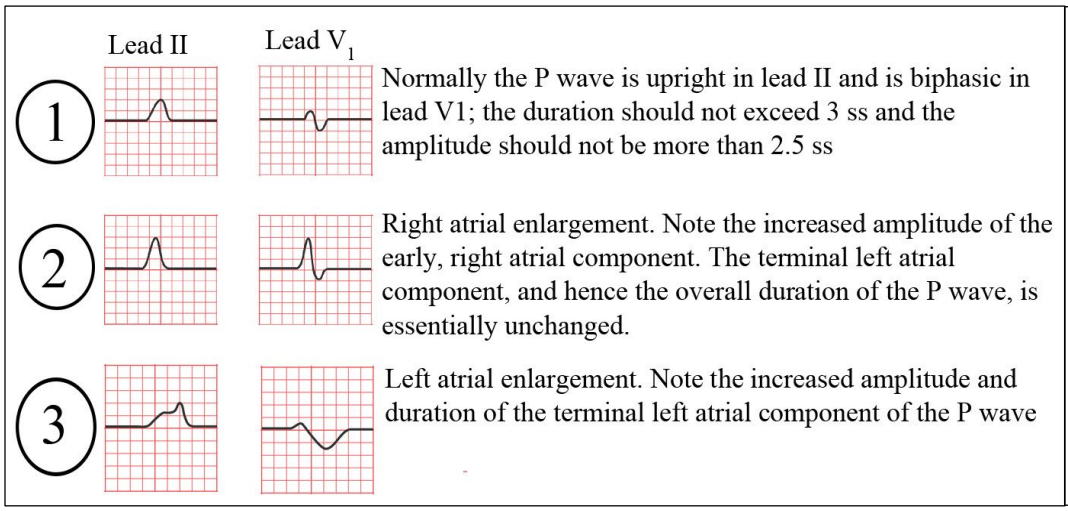


FIGURE 1.42 Atrial enlargements.

STEP 06 PR SEGMENT

The PR segment is the straight line running from the end of the P wave to the start of the QRS complex. It therefore measures the time from the end of atrial depolarization to the start of ventricular depolarization. It should be isoelectrical (FIGURE 1.43). Depressed PR interval is a sensitive sign for acute pericarditis. It may also encounter in ventricular hypertrophy and chronic lung disease.

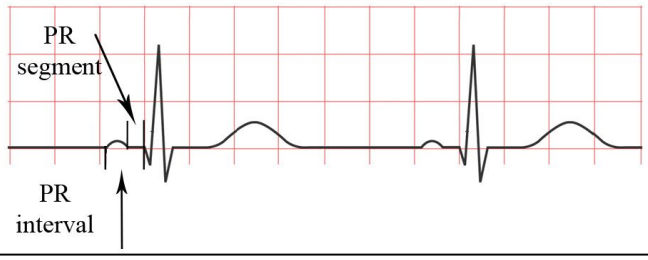


FIGURE 1.43 PR segment and interval.

STEP 07 PR INTERVAL

The PR interval is measured from the start of the P wave to the beginning of the QRS complex (FIGURE 1.43). In sinus rhythm, the PR interval ranges from 120-200 ms (represented by 3 – 5 mm or 3 – 5 small squares). A PR interval of less than 3 small squares indicates pre-excitation syndrome (i.e. electrical conduction occurs more quickly than usual) and that of more than 5 small squares indicates a conduction block (i.e. electrical conduction occurs more slowly than usual).

SHORT PR INTERVAL: PRE-EXCITATION SYNDROME

The depolarization normally starts at the SA node and then spreads through atrial muscle fibers. While depolarization spreads through AV node, there is a physiological delay represented by the 3 – 5 small squares (i.e. the PR interval). In pre-excitation, there is an accessory (or extra) conduction pathway which connects the atria with the ventricles or the atria with the His bundle and these are Wolff-Parkinson-White (WPW) syndrome and Lown-Ganong-Levine (LGL) syndrome respectively. In both syndromes, the accessory conduction pathways act as short circuits, allowing the atrial wave of depolarization to bypass the AV node and activate the ventricles prematurely.

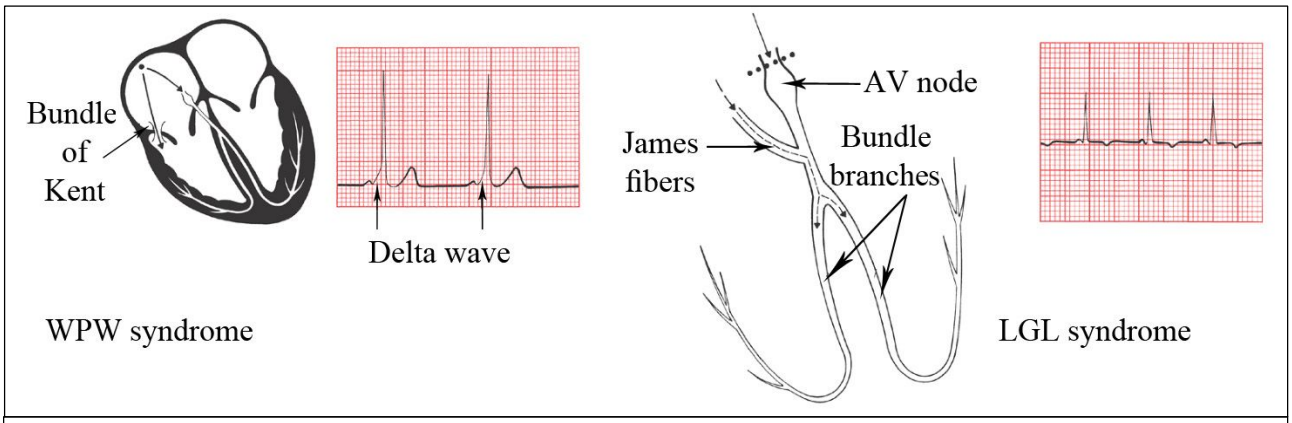


FIGURE 1.44 Mechanism of WPW and LGL syndrome origin.

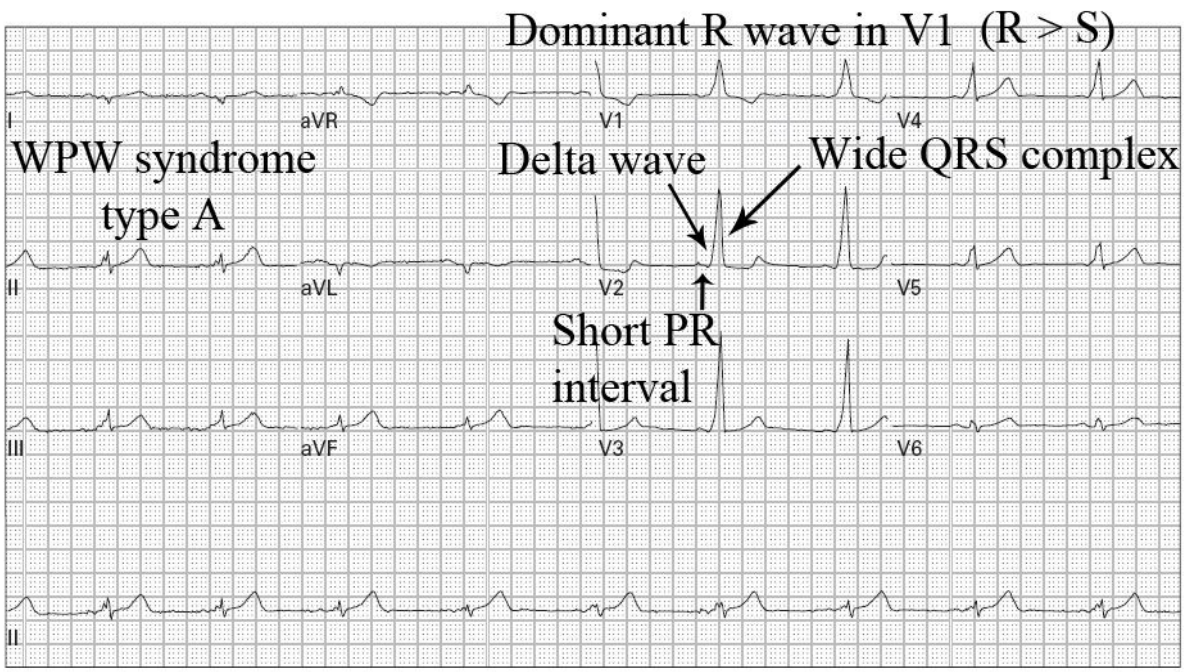


FIGURE 1.45 WPW syndrome type A.

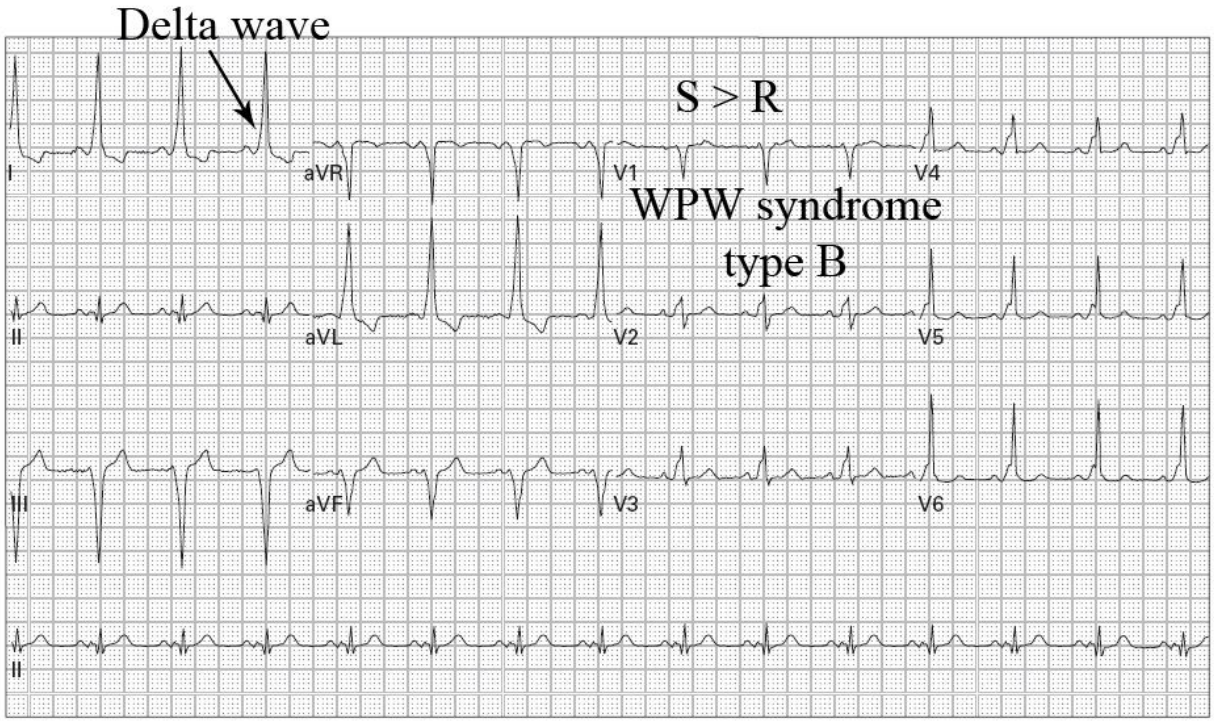


FIGURE 1.46 WPW syndrome type B.

In WPW syndrome there is an accessory conducting pathway (called bundle of Kent) which connects either right atrium with right ventricle (WPW type B) or left atrium with left ventricle (WPW type A), by passing the normal delay at AV node, thus ventricular depolarization occurs early and the PR interval is short. Early ventricular depolarization causes a slurred upstroke of the QRS complex called delta wave (FIGURE 1.44). WPW syndrome type A has a dominant R wave in V_1 (FIGURE 1.45) while WPW type B has no such (FIGURE 1.46). LGL syndrome is due to an AV node bypass that connects the atrium to the His bundle (FIGURE 1.44) and is called James fibers. The EKG reveals only short PR interval with normal shape QRS complex (FIGURE 1.47). Both syndromes can be associated with the development of tachyarrhythmia. In WPW syndrome, depolarization can spread down the normal pathway and back (retrogradely) up through the accessory pathway to reactivate the atria and so cause a tachycardia. The ventricles are therefore depolarized in the normal way, producing narrow QRS complexes with P waves sometimes visible just after

each QRS complexes. This is called orthodromic tachycardia which is the most common form of tachycardia in WPW syndrome (FIGURE 1.48) and is similar to junctional tachycardia. Alternatively, depolarization can pass down the accessory pathway and retrogradely upward the His bundle. The ventricles are then depolarized through the accessory pathway; producing broad complex tachycardia with P waves may or may not be seen. This is called an antidromic tachycardia (FIGURE 1.48). This type of tachycardia is similar to ventricular tachycardia described earlier. The onset of atrial fibrillation may produce very rapid ventricular rates because the by pass pathway lacks the rate limiting properties of the normal AV node (FIGURE 1.48). When re-entry and therefore tachycardia occurs in LGL syndrome, the QRS complexes remain narrow, with appearance similar to that of a junctional tachycardia. Tachycardia due to the WPW and LGL are grouped together under the term atrioventricular re entrant (AVRT) tachycardia.

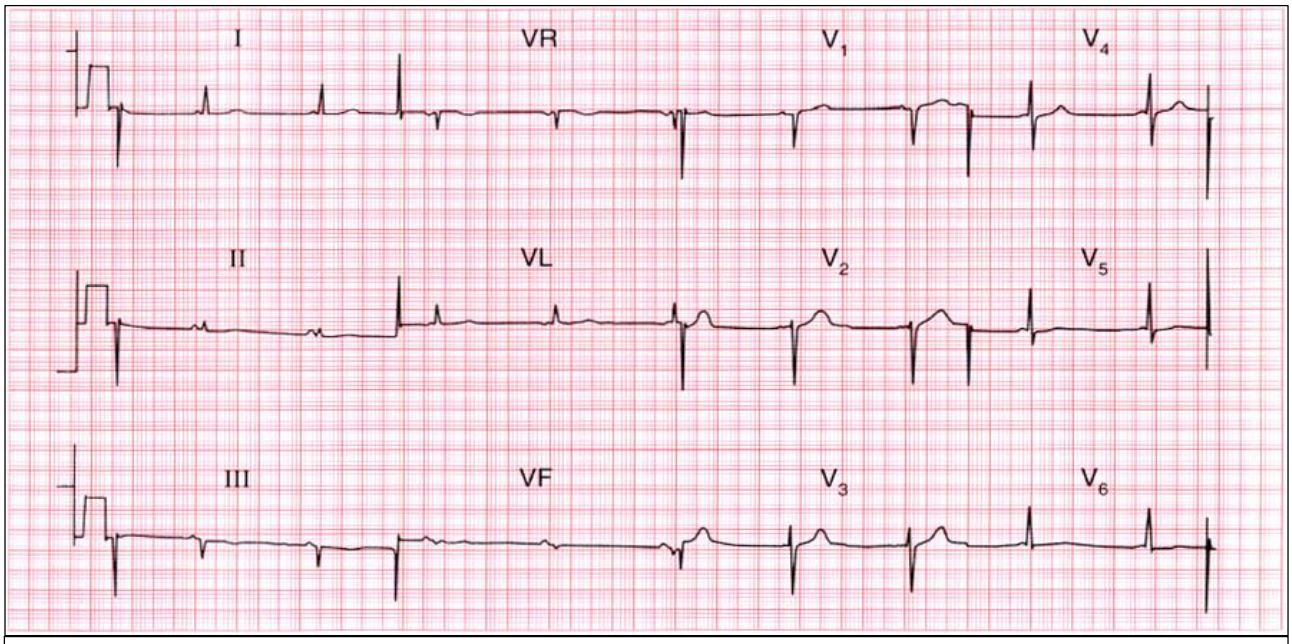


FIGURE 1.47 Lown-Ganong-Levine syndrome.

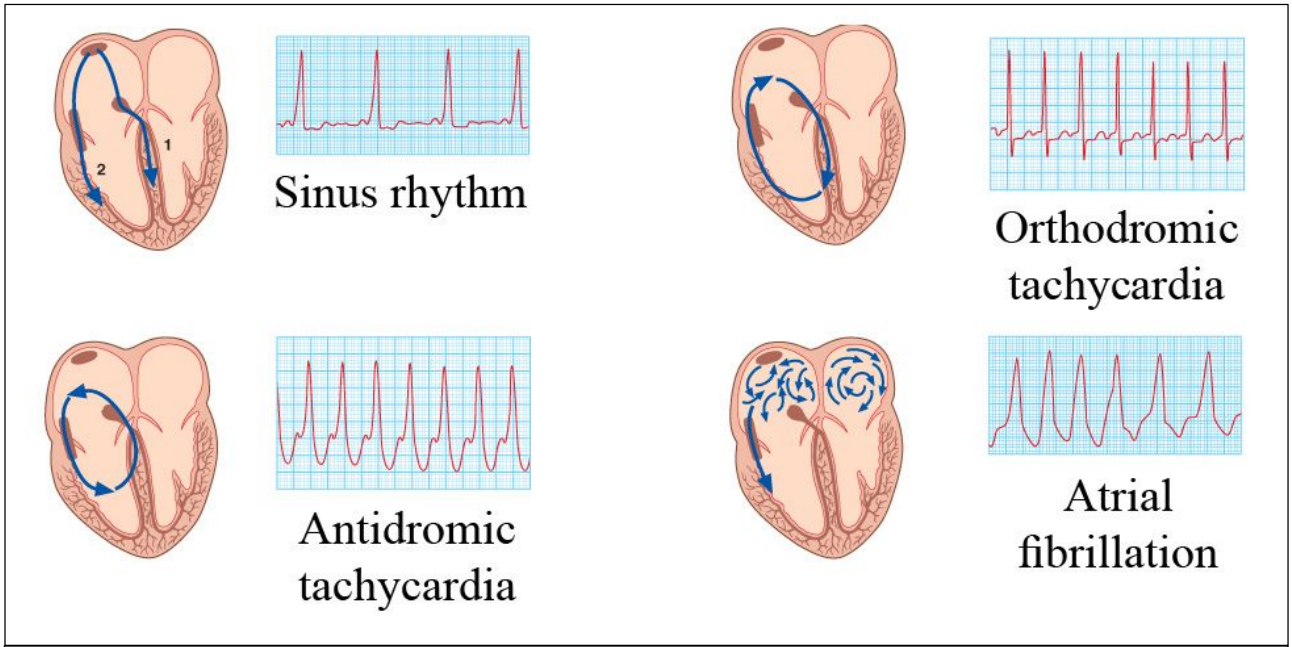


FIGURE 1.48 WPW syndrome. When the ventricles are depolarized through the AV node (1) the ECG is normal. When the ventricles are depolarized through the accessory conducting tissue (2) the ECG shows a very short PR interval and a wide QRS complex.

PROLONGED PR INTERVAL: CONDUCTION BLOCK

If the PR interval is more than 200 ms (i.e. > 5 small squares) it indicates a conduction defect (block) e.g. first degree heart block. Conduction defect may take one of the following forms:

01 SINOATRIAL BLOCK

In sinoatrial block, the SA node depolarizes normally, but the depolarization fails to penetrate the atrium. The EKG appearance reveals no P QRS T, but the atrium must have been depolarized because the next P wave appears at the predicted time. This should be differentiated from sinus arrest (sinus pause) that means loss of SA node activity. In sinus arrest the expected P wave does not appear until after two or three normal intervals and then not at the predicated time (FIGURE 1.49).

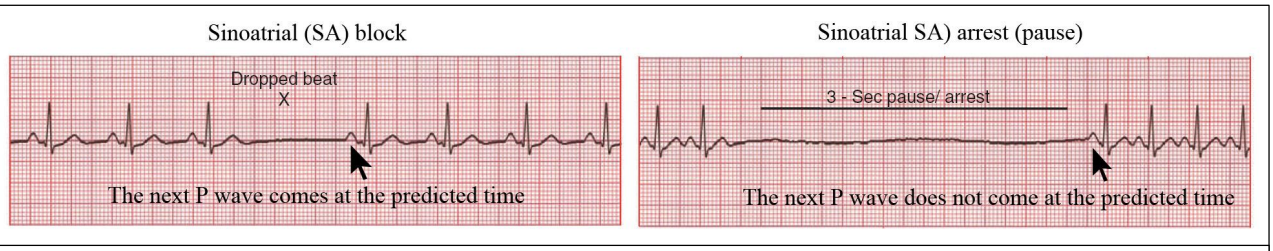
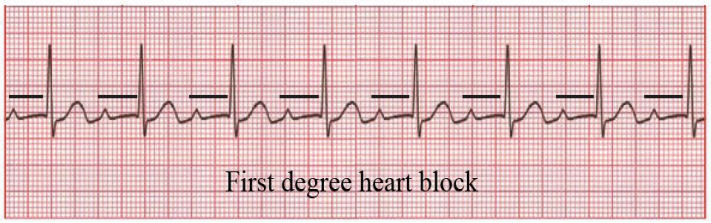


FIGURE 1.49 Sinoatrial block and sinoatrial arrest.

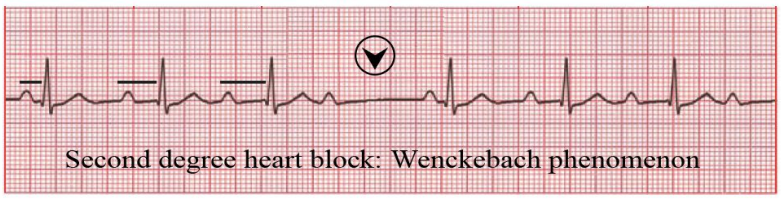
02 CONDUCTION PROBLEMS IN THE AV NODE AND HIS BUNDLE

I. FIRST DEGREE HEART BLOCK (FIGURE 1.50)
When each atrial depolarization is followed by ventricular depolarization, but atrioventricular conduction is slow, the PR interval on the surface EKG is prolonged (> 5 ss) and the first degree heart block is said to be present. First degree heart block had fixed prolonged PR interval.

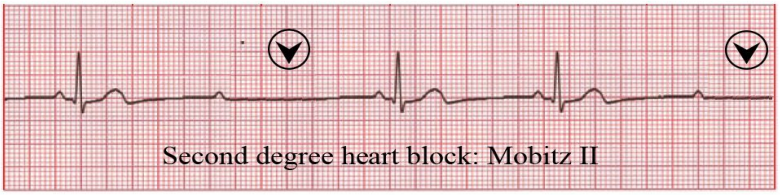


This type of heart block is characterized by fixed prolonged PR interval.

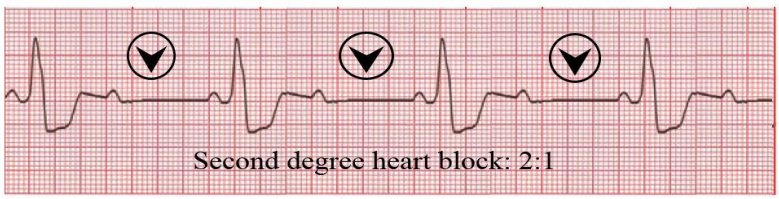
FIGURE 1.50 First degree heart block.



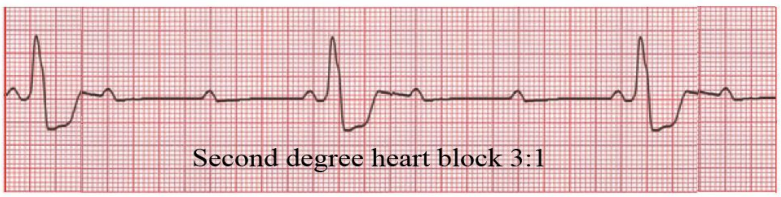
The PR interval becomes progressively longer untill one P wave is totally blocked and produces no QRS complex. After a pause, during which the AV node recovers, this cycle is repeated.



Most beats are conducted but occasionally a P wave is not followed by a QRS complex.



Conduction rate here (ratio of P waves to QRS complexes) is 2: 1



Conduction rate here (ratio of P waves to QRS complexes) is 3:1

FIGURE 1.51 Second degree heart block.

II. SECOND DEGREE HEART BLOCK (FIGURE 1.51)
When atrial depolarization intermittently fails to induce ventricular depolarization, second degree heart block exists. There are three varieties. Mobitz type I (Wenckebach phenomenon) describes progressive lengthening of the PR interval with each beat till a P wave is not conducted and is not followed by a QRS complex; Mobitz type II is present when most beat are conducted, but occasionally a P wave is not followed by a QRS complex; Type 2:1 is present when alternate P waves are not conducted. This block may be 2:1 or 3:1 depending on the relation between P wave and QRS complex.

III. THIRD DEGREE (COMPLETE) HEART BLOCK (FIGURE 1.52)
Complete heart block is said to occur when atrial contraction is normal, but no beats are conducted to the ventricles. Complete heart block results either from His bundle disease or from bilateral bundle branch block. A narrow QRS complexes indicate that the rhythm originates within the His bundle itself below the block, but a wide QRS complex indicates that ventricular depolarization originates in the Purkinje system. The EKG in third-degree heart block shows P waves marching across the rhythm strip at their usual rate (60 to 100 waves per minute) but bearing no relationship to the QRS complexes that appear at a much slower escape rate (30 to 40 waves per minute). The QRS complexes appear either narrow or wide.

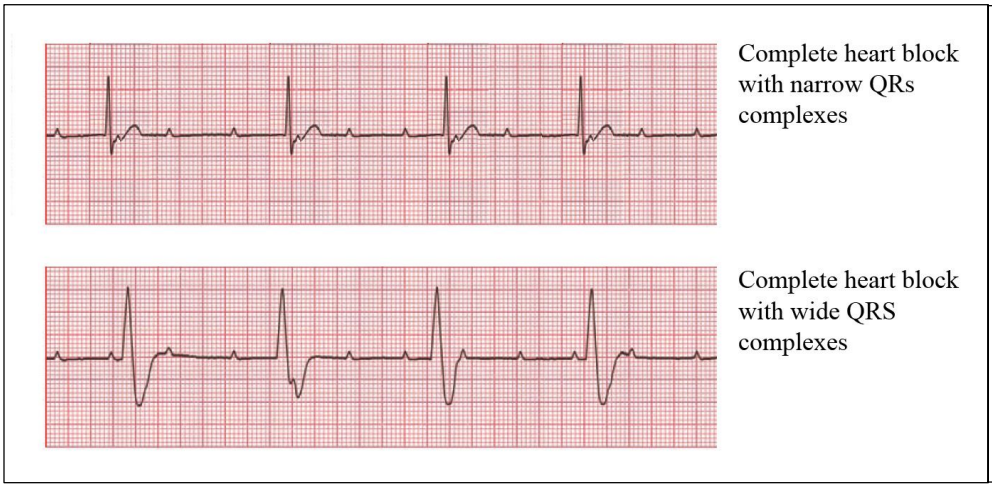


FIGURE 1.52 Third degree heart block. Look at the P wave which has no relation to the QRS complexes i.e. it comes before, within, or after.

It is important to remember that AV dissociation is not synonymous with complete heart block. AV dissociation refers to any circumstance in which the atria and ventricles beat independently of each other. This situation occurs in heart block, ventricular tachycardia, and sometimes junctional escape rhythm.

IV. BUNDLE BRANCH BLOCK
When the His bundle conducts normally, but one of the bundle branches is blocked, the PR interval is normal, but QRS complex is widened because of the late depolarization of the part of the ventricle normally supplied by the bundle branch which is blocked. The characteristic appearances of right bundle branch block (RBBB) include (FIGURE 1.53) dominant R wave in V_1, wide QRS complexes (greater than 0.12 seconds or three small squares), an RSR′ pattern in V_1 and V_2 (rabbit ears) with ST segment depression and T wave inversion, and lastly deep and wide S waves in V_6.

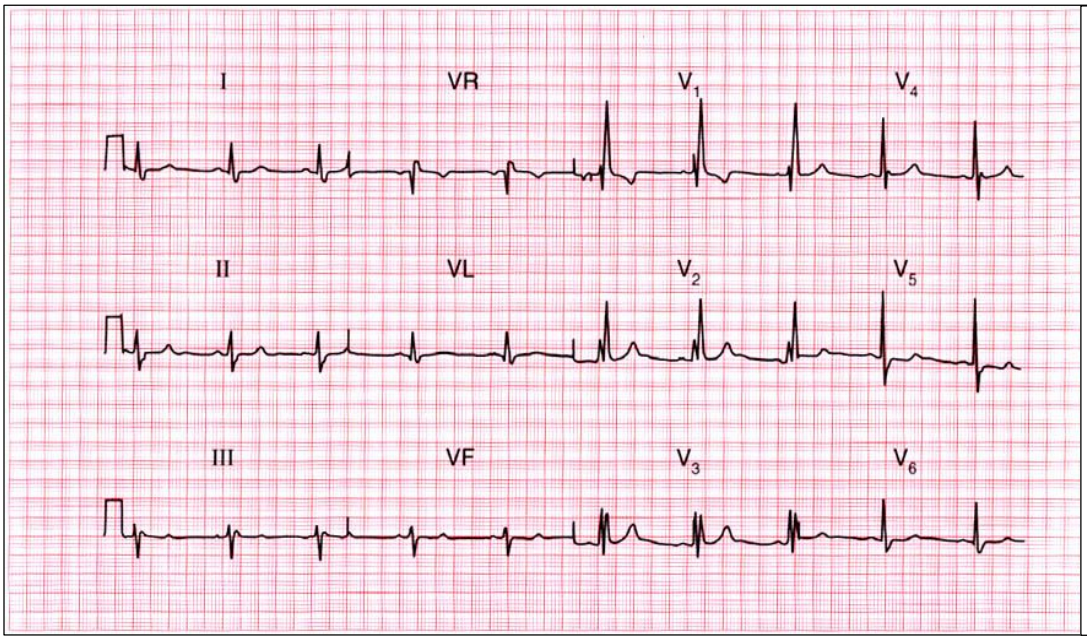


FIGURE 1.53 Right bundle branch block.

The characteristic appearance of left bundle branch block (LBBB) includes (FIGURE 1.54) wide QRS complex (wider than 0.12 seconds or wider than three small squares), loss of septal Q waves, broad or notched R wave (M shape) with prolonged upstroke of the QRS complex in the lateral leads (I, II, VL, and V_5-V_6) with ST segment depression and T wave inversion, reciprocal changes in V_1 and V_2, and lastly left axis deviation may be present.

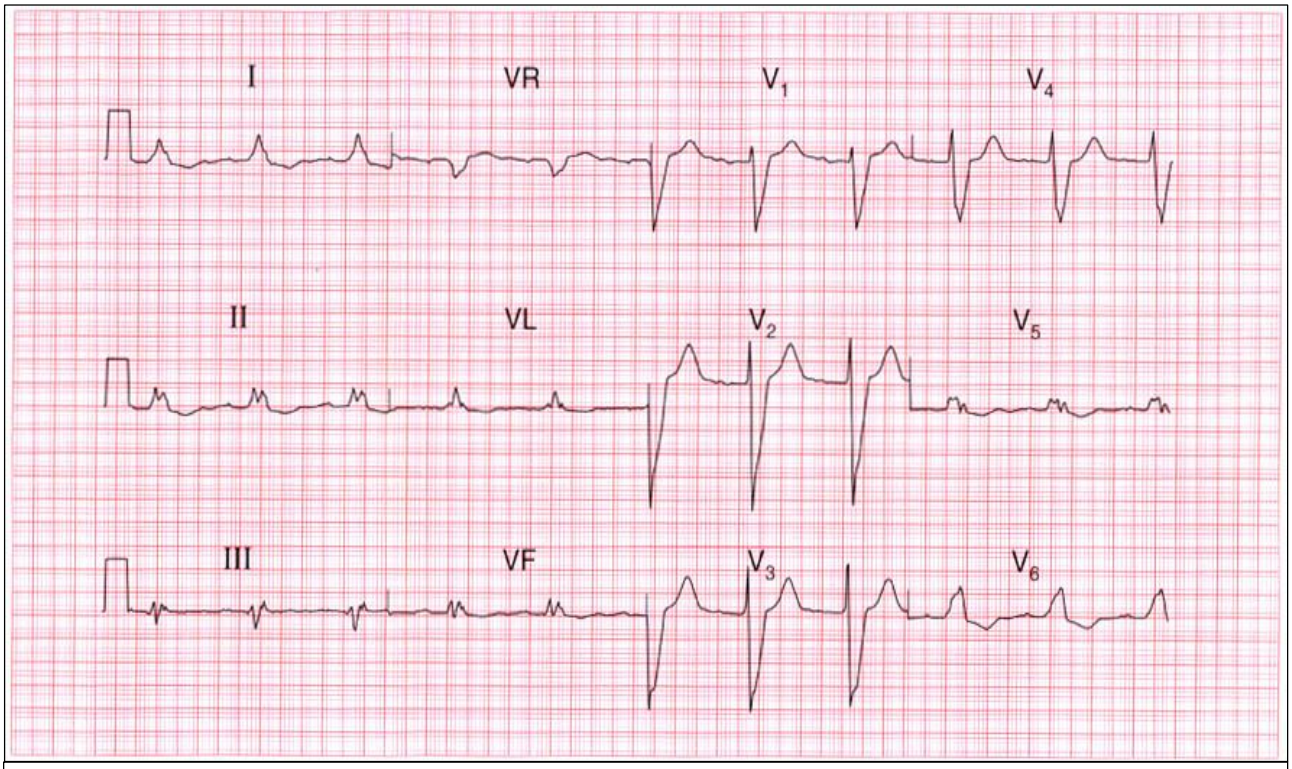


FIGURE 1.54 Left bundle branch block.

Remember that a dominant R wave in V_1 (i.e. longer R wave than S wave in V_1) is the most discriminative feature and it indicates right bundle branch block. Both right and left bundle branch block can be intermittent or fixed. In some individuals, the ventricles conduct normally at slow heart rates, but, above when the heart rate accelerates, bundle branch block develops. This occurs because the descending impulse in tachycardia finds one of the branches is still in its refractory period. This is called rate-dependant bundle branch block. Causes of bundle branch block are shown in BOX 1.5.

BOX 1.5 Causes of bundle branch block

RIGHT BUNDLE BRANCH BLOCK	LEFT BUNDLE BRANCH BLOCK
Coronary artery disease	Coronary artery disease
Right ventricular hypertrophy or strain pattern e.g. pulmonary embolism	Hypertension
Congenital heart disease e.g. ASD	Aortic valve disease
Normal variant	Cardiomyopathy

When there is QRS widening greater than 0.12 seconds (three small squares) without any other criteria for either bundle branch block, the term used is nonspecific intraventricular conduction delay. Partial or incomplete RBBB is characterized by normal QRS complex duration, but with an RSR′ pattern in V_1. It is quite common in healthy people. A diagnosis of incomplete LBBB may be made if the QRS duration is greater than 0.10 with notching of the R wave in V_5 or V_6.

V. FASCICULAR BLOCK

The right bundle branch block is a single structure, but the left bundle branch divides into two fascicles; anterior and posterior. Block at the anterior fascicle (left anterior fascicular block or left anterior hemi block) causes extreme left axis deviation while block at the posterior fascicle (left posterior fascicular block or left posterior hemi block) may cause extreme right axis deviation. Because of this anatomy, LBBB can be called bifascicular block and RBBB is called monofascicular block. Bifascicular block includes RBBB and left anterior fascicular block (FIGURE 1.55) or RBBB with left posterior fascicular block, or LBBB. If first degree heart block is added to the bifascicular block, trifascicular block is said to be present (FIGURE 1.56). When RBBB is present with LBBB (or left anterior and posterior hemi block), complete heart block is likely to occur.

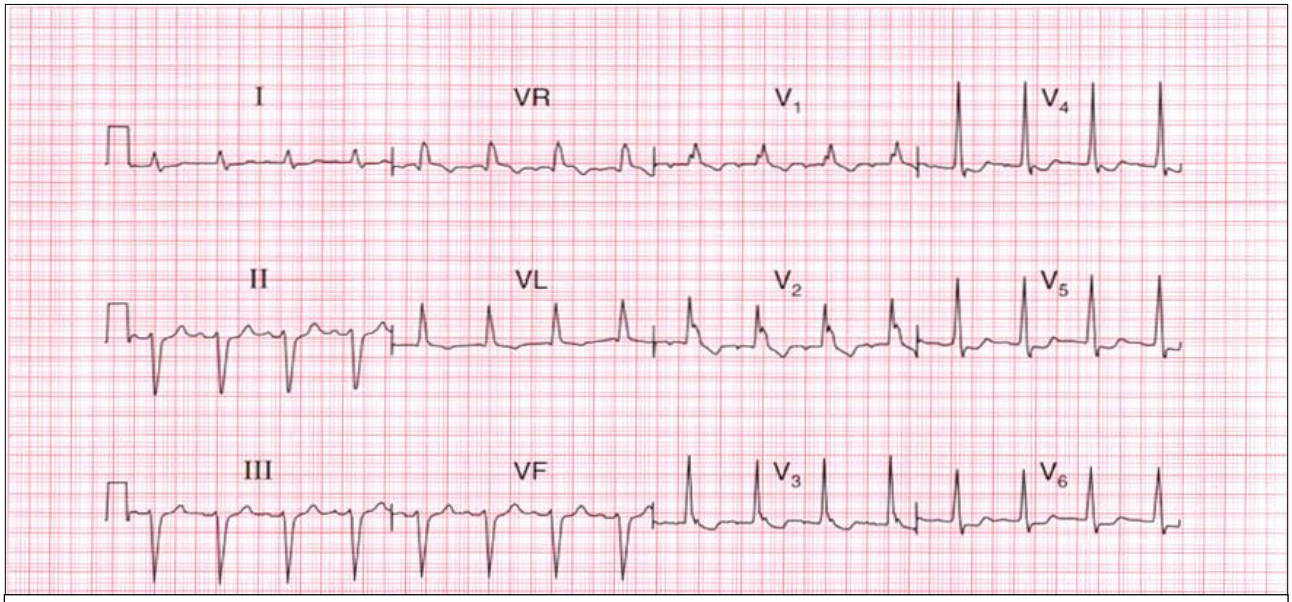


FIGURE 1.55 RBBB and left anterior hemiblock. This combination is called bifascicular block.

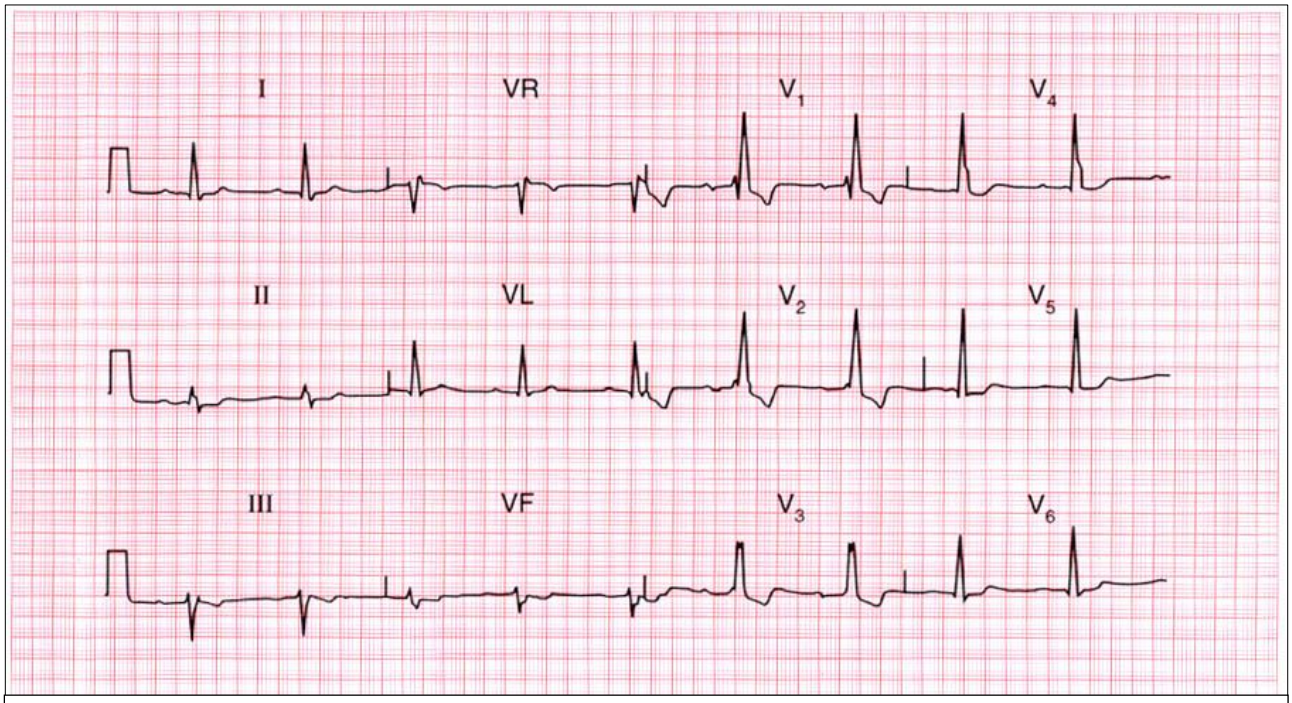
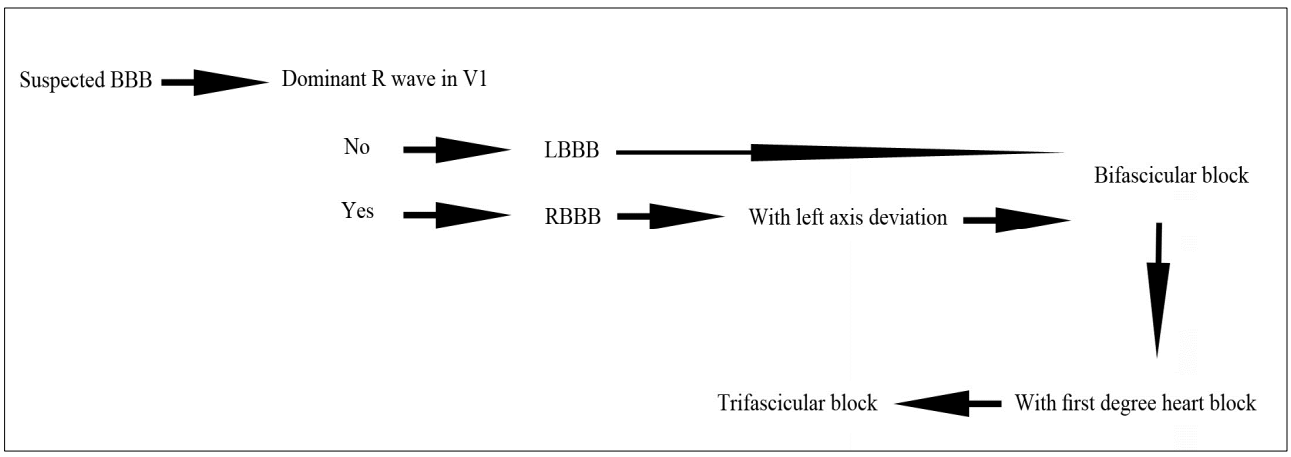


FIGURE 1.56 RBBB, left anterior hemiblock, and first degree heart block. This combination is called trifascicular block.

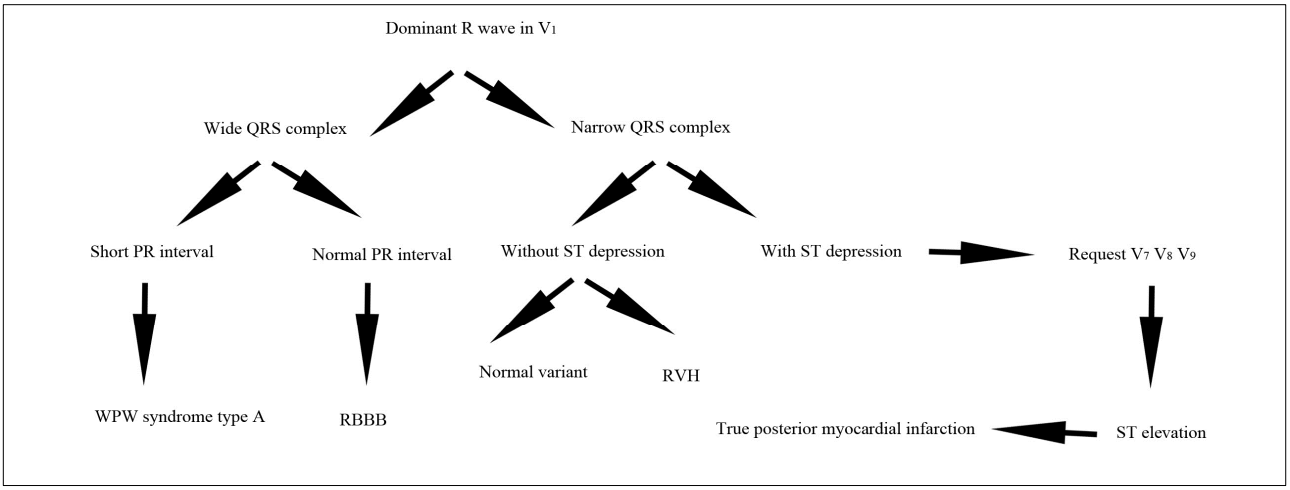
An approach to suspected bundle branch block is shown here:



STEP 08 QRS COMPLEX

In the normal chest leads, the QRS complexes start as a negative wave in lead V_1 and V_2 and then ended as a positive lead in V_5 and V_6; the transition point where R and S waves are equal in the chest lead over the interventricular septum is normally at V_3 or V_4; an RSR′ pattern in V_1 is a normal acceptable variant provided that the duration is less than three small squares (partial RBBB); the normal width of the QRS is less than three small squares; R wave is smaller than S wave in V_1; R wave in V_6 is less than five large squares; R wave in V_5 or V_6 plus S wave in V_1 or V_2 is less than seven large squares. There may be small thin Q waves (less than 1 small square in width and less than 3 small squares in depth or less than 1/4 of the corresponding height of R wave) in the lateral leads: I, VL, V_5-V_6 or in lead III, but not VF. These Q waves are called septal Q waves. More than this value one should consider them pathological until proves otherwise. Abnormalities in the precordial QRS leads may take one of the following forms:

1. Wide QRS (more than three > small squares) may indicate bundle branch block, WPW syndrome, hyperkalemia, and ventricular source (e.g. ventricular ectopic beats and tachycardia, ventricular escape beat and rhythm), or wide complex tachycardia.
2. Tall (dominant) R wave in V_1 may occur in right ventricular hypertrophy, WPW syndrome type A, right bundle branch block, or true posterior myocardial infarction. This finding could be normally seen in certain individuals. An approach to dominant R wave in V_1 is shown here



Right ventricular hypertrophy (FIGURE 1.57) is seen in leads V_1 – V_4. The Sokolow-Lyon criteria for right ventricular hypertrophy adds the R wave amplitude in V_1 to the S wave amplitude in lead V_5 or V_6; a sum of 1.1 (11 small squares) mV or greater implies right ventricular hypertrophy (RVH). There is thus dominant R wave in V_1 and in severe cases there is inversion of T waves (with/without ST depression) in V_1 and V_2 and sometimes V_3 or even V_4. This is called right ventricular strain pattern.

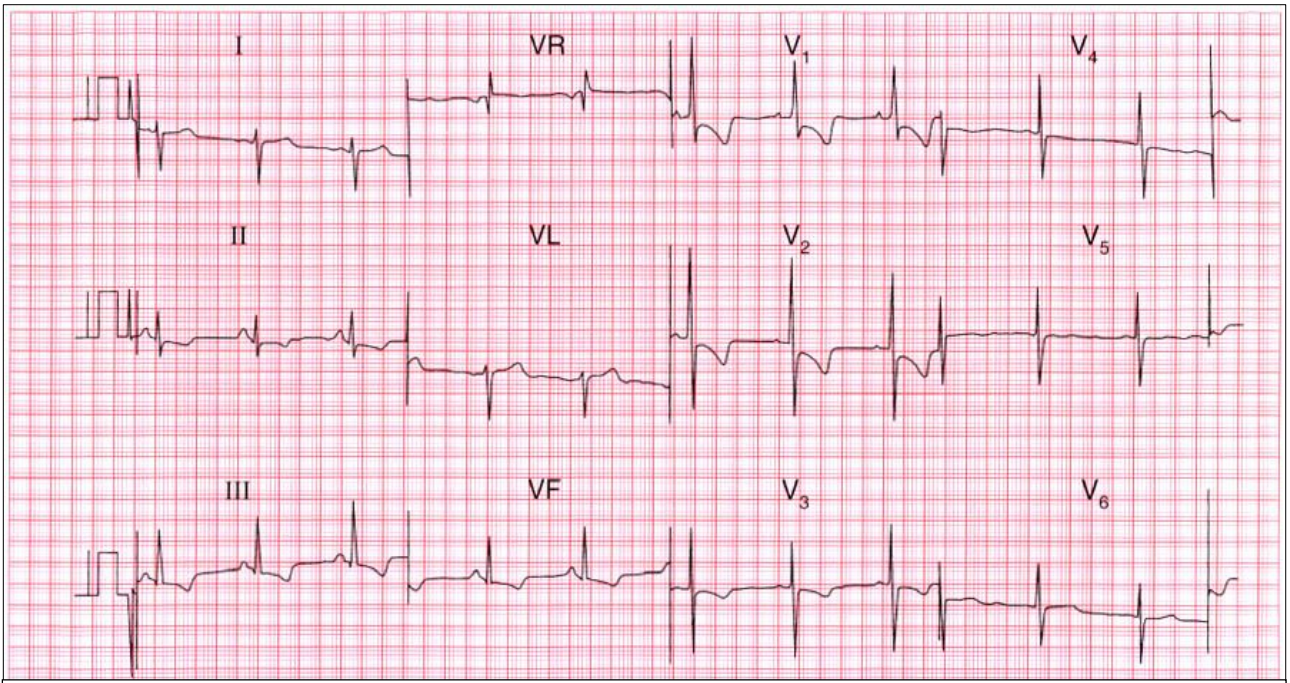


FIGURE 1.57 Right ventricular hypertrophy with strain pattern.

3. Tall R wave in V_5 or V_6 may indicate left ventricular hypertrophy (LVH). It is significant when there is voltage criteria (R waves in V_5 or V_6 is greater than 5 large squares or R wave in V_5 or V_6 plus S waves in V_1 or V_2 is greater than 7 large squares). Strain pattern is said to be present when inverted T waves (with/without ST depression) are seen in the lateral leads of I, VL, V_5-V_6 (FIGURE 1.58).

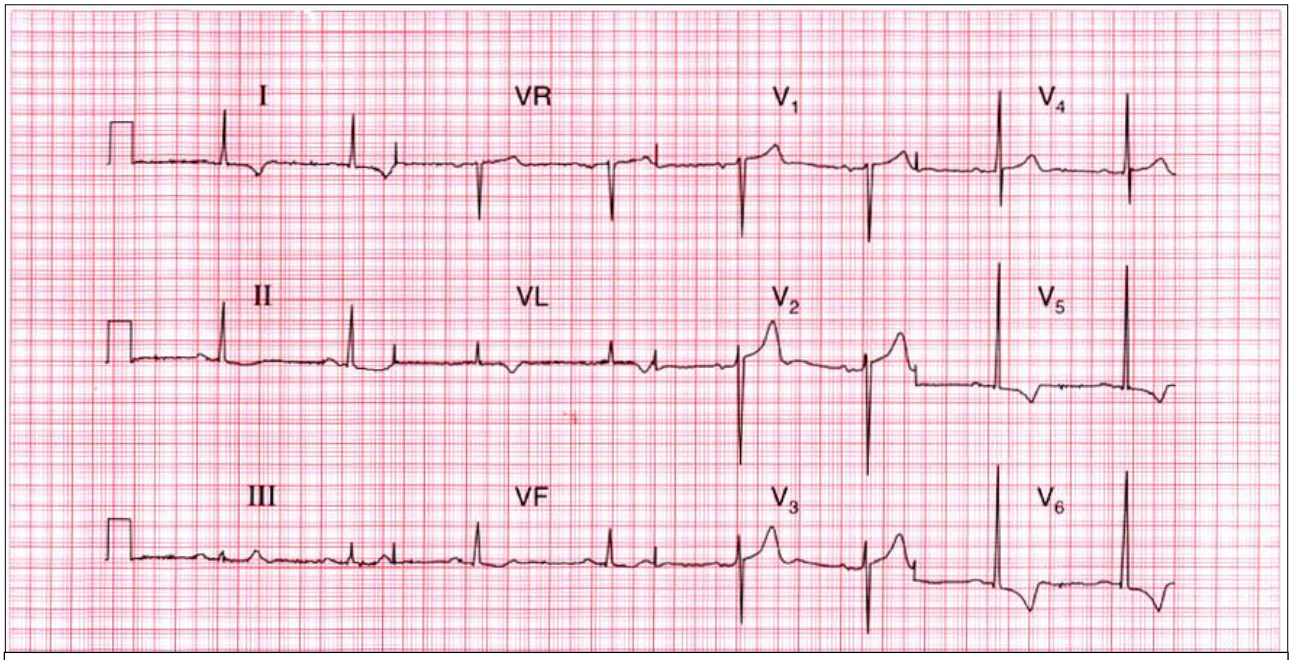


FIGURE 1.58 Left ventricular hypertrophy with strain pattern.

4. Low voltage QRS complex is present when QRS height is less than one large square in limb leads and less than two large squares in chest leads. This (FIGURE 1.59) may result from incorrect standardization of the EKG device, emphysema, obesity, pericardial effusion, hypopituitarism, and myxoedema. In pericardial effusion, QRS complexes are small and there may be an electrical alternans that is a changing axis with alternate beats caused by heart moving in a bag of fluid (FIGURE 1.60).

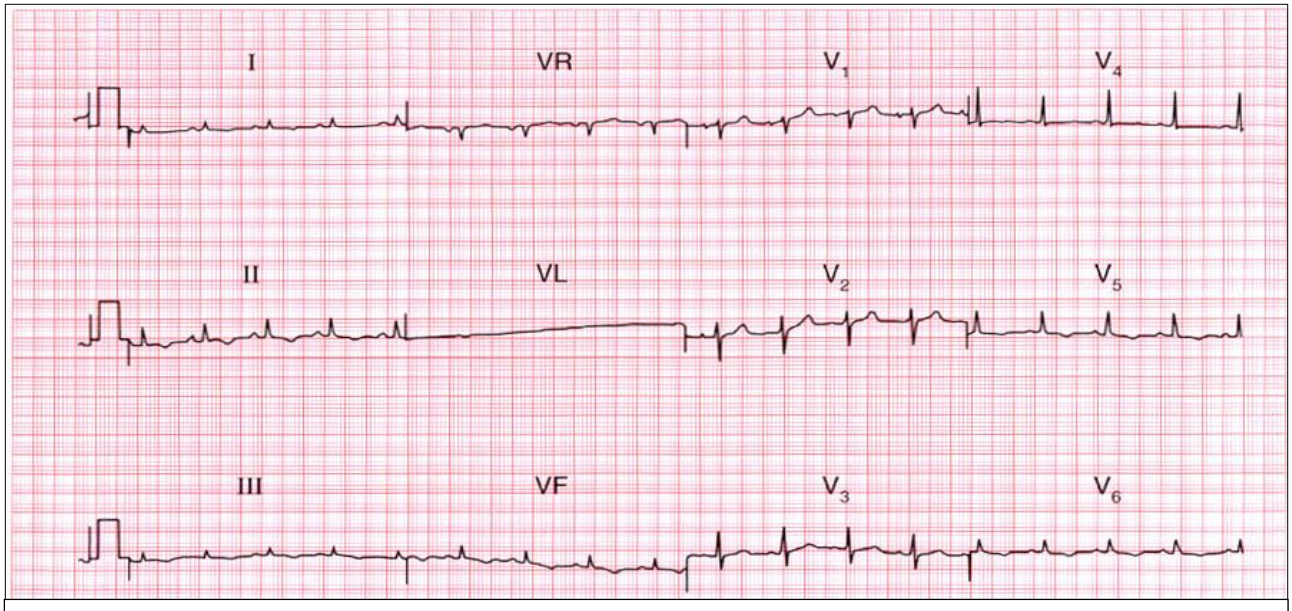


FIGURE 1.59 Low voltage QRS complex.

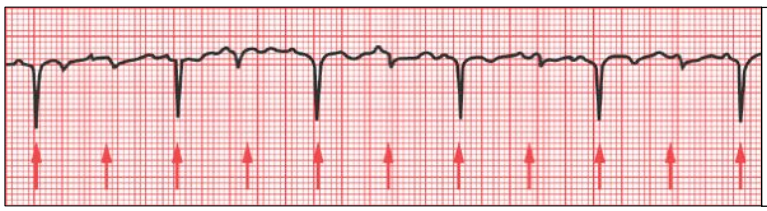


FIGURE 1.60 Electrical alternans. The arrows point to each QRS complex.

5. Shifting of the transition point from its normal site at V_3-V_4 to further point e.g., V_4-V_5 or V_5-V_6 may indicate chronic lung disease. This is called clockwise rotation of the heart and shown in FIGURE 1.61.

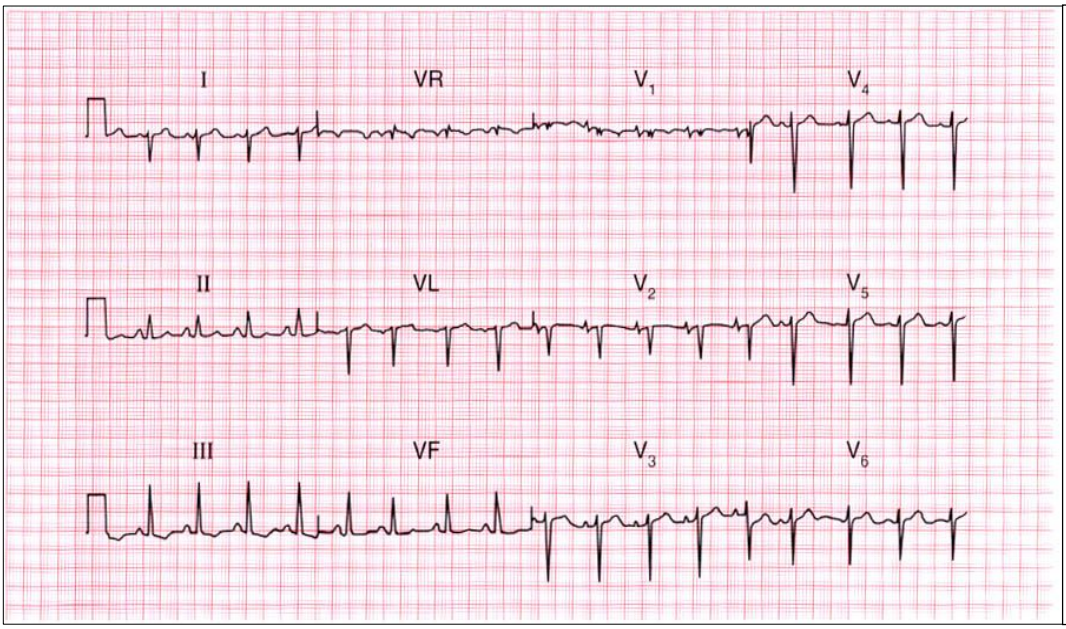


FIGURE 1.61 Shifting of the transition point. This is called clockwise rotation of the heart.

EKG

STEP 09 ST SEGMENT

ST segment is measured from the end of the QRS to the beginning of the T wave (FIGURE 1.4). It should be isoelectric (the same level as the EKG trace between beats that is between the T wave to the next P wave). Abnormalities of the ST segment may include elevation or depression.

ST SEGMENT ELEVATION

A raised ST segment may indicate acute myocardial infarction, left ventricular aneurysm, Prinzmetal's angina, brugada syndrome or pericarditis. It can be however a normal variant.
Horizontal ST segment elevation more than two small squares in chest leads and/or more than one small square in limb leads indicate acute myocardial infarction. The infracted (and even ischemic) areas of the myocardium can be localized through the leads showing EKG changes (FIGURE 1.62 and BOX 1.6).

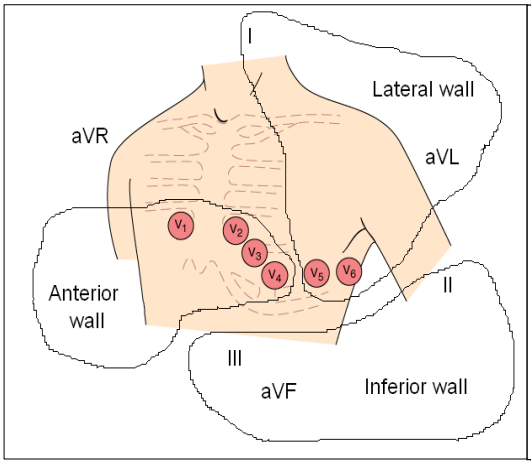


FIGURE 1.62 Localization of the main myocardial walls.

BOX 1.6 EKG localization of myocardial wall affected by infarction or ischemia

MYOCARDIAL INFARCTION	EKG CHANGES	CORONORY TERRITORY
Anterolateral	V_4 to V_6, lead I, and aVL	Left main stem
Septal	V_1 and V_2	Left anterior descending artery
Anterior	V_3 and V_4	Left anterior descending artery
Anteroseptal	V_1 to V_4	Left anterior descending artery
Lateral	I, aVL and V_1-V_6	Left circumflex
High lateral	Lead I and aVL	Left circumflex artery
Posterior	Dominant R wave in V_1	Usually left circumflex, also right coronary
Inferior	II, III and aVF	Right coronary artery
Right ventricular	ST segment elevation in V4R	Right coronary artery

Persistent ST segment elevation is quite common after an anterior myocardial infarction. It may indicate the development of a left ventricular aneurysm, but it is not a reliable evidence of this. The upward concave shape of the ST segment and unusual distribution of changes in pericarditis may help to distinguish pericarditis from myocardial infarction (FIGURE 1.63). The EKG in acute pericarditis typically evolves through four stages. In **stage 1**, there is widespread elevation of the ST segments, often with upward concavity (sometimes called smiling face), involving two or three standard limb leads and V_2 to V_6, with reciprocal depressions only in aVR and sometimes V_1, as well as PR-segment depression. Usually there are

no significant changes in QRS complexes. In **stage 2**, after several days, the ST segments return to normal and only then, or even later, do the T waves become inverted (**stage 3**). Ultimately, weeks or months after the onset of acute pericarditis, the EKG returns to normal in **stage 4**.

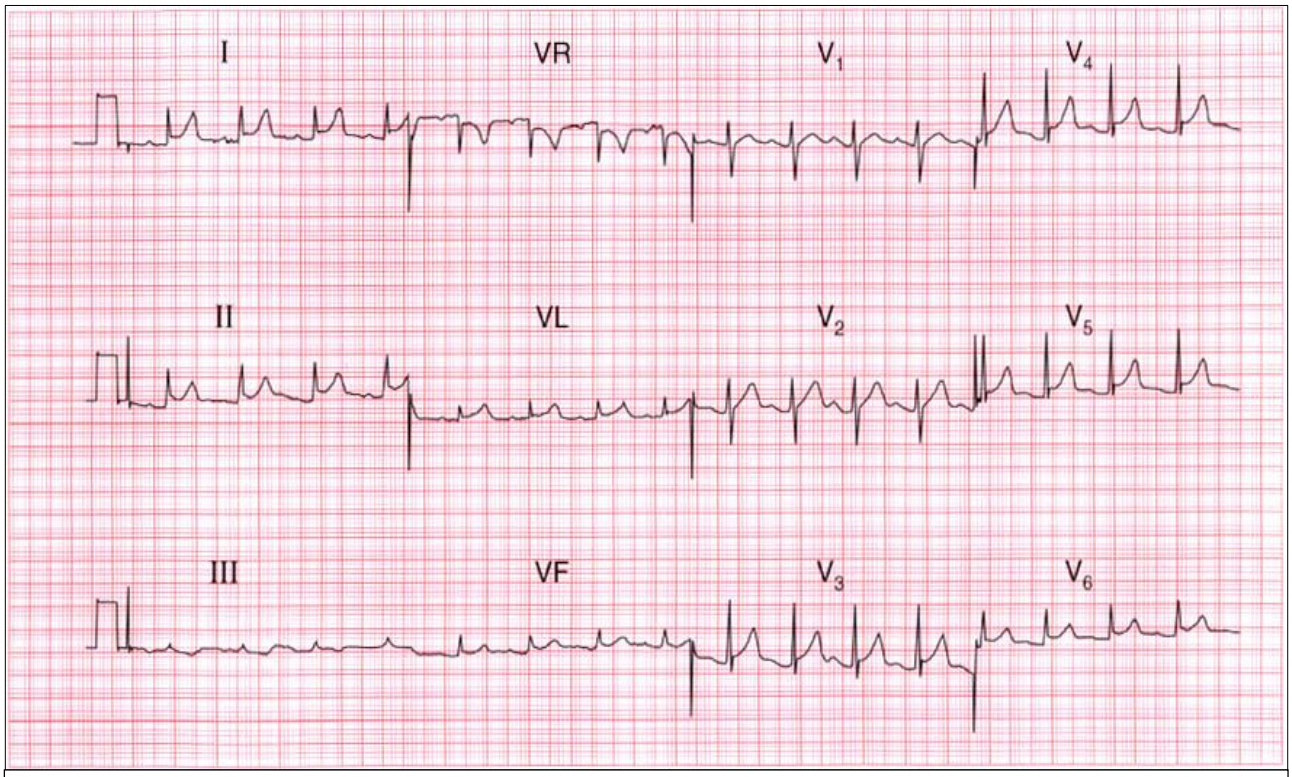


FIGURE 1.63 Stage 1 acute pericarditis. There are ST segment elevation in inferior and some of the anterior leads with reciprocal changes at the aVR and PR segment depression.

Prinzmetal's angina (FIGURE 1.64) occurs as a result of coronary artery spasm. It may be associated with reversible ST segment elevation without myocardial infarction. These EKG abnormalities can be transient at time of pain.

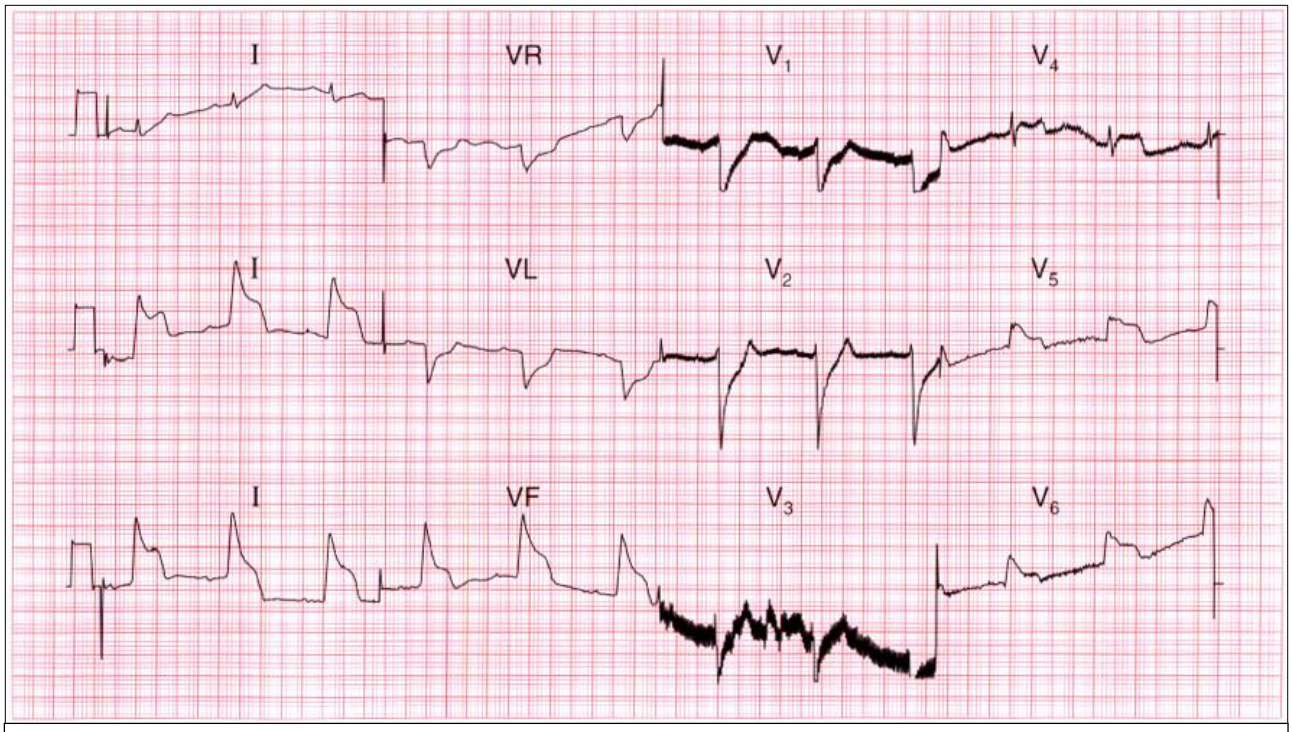


FIGURE 1.64 Prinzmetal's angina. It is characterized by reversible ST segment elevation.

The EKG appearance of Brugada syndrome (FIGURE 1.65) between attacks superficially resembles that associated with partial RBBB, with an RSR′ pattern in leads V_1 and V_2. However the ST segment in these leads is raised and there is no wide S wave in V_6 as there in RBBB.

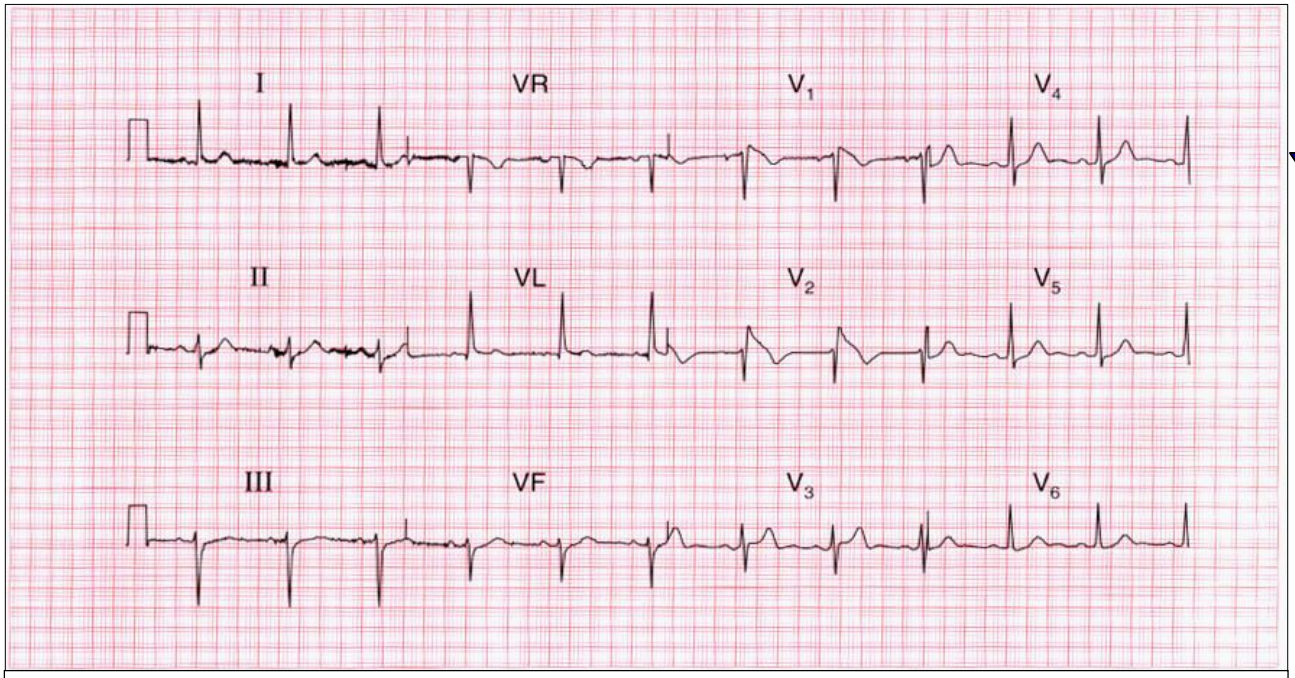


FIGURE 1.65 Brugada syndrome.

Myocardial infarction can be divided into two types on the basis of their associated EKG findings into:

1. ST segment elevation myocardial infarction
2. Non ST segment elevation myocardial infarction

ST SEGMENT ELEVATION MYOCARDIAO INFARCTION (STEMI)

This type is also called full thickness myocardial infarction, transmural myocardial infarction, or Q – wave myocardial infarction. The serial evolutions of EKG changes (FIGURE 1.66) in this type include:

1. Symmetrically peaked (hyperacute) T waves that resolve after several minutes as the characteristic ST segment elevation develops (WITHIN SECONDS).
2. Acute ST segment elevation that indicates the current of injury (WITHIN MINUTES).
3. Progressive loss of R wave, development of Q wave, resolution of the ST segment elevation and terminal T wave inversion (WITHIN HOURS).
4. Deep Q wave and T wave inversion (WITHIN DAYS).
5. Old or established myocardial infarction is characterized by persistent Q waves and less marked T waves (WITHIN WEEKS OR MONTHS).
6. In the setting of acute MI there may be ST segment depression in some leads, and these are called reciprocal changes.

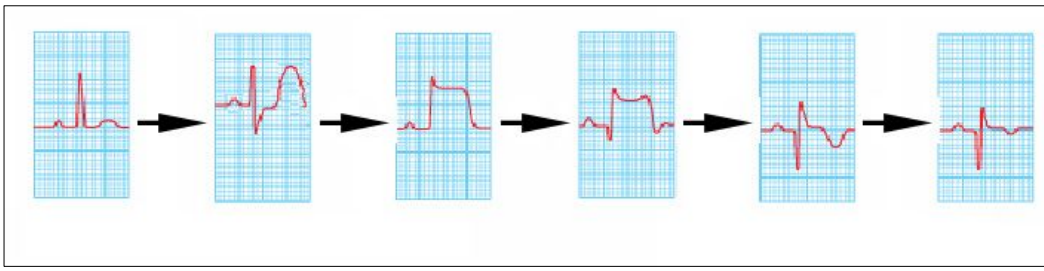


FIGURE 1.66 Serial evolution of STEMI.

During the initial stage, total occlusion of an epicardial coronary artery produces ST-segment elevation. Most patients initially presenting with ST-segment elevation ultimately evolve Q waves on the EKG. However, Q waves in the leads overlying the infarct zone may vary in magnitude and even appear only transiently depending on the reperfusion status of the ischemic myocardium. A small proportion of patients initially presenting with ST-segment elevation will not develop Q waves when the obstructing thrombus is not totally occlusive, obstruction is transient, or if a rich collateral network is present. A minority of patients who present initially without ST-segment elevation may develop a Q-wave myocardial infarction. For these reasons terms such as Q-wave myocardial infarction, non-Q-wave myocardial infarction, transmural myocardial infarction, and nontransmural myocardial infarction, have been replaced by STEMI and NSTEMI. Examples of STEMI are shown in FIGURES 1.67, 1.68, and 1.69.

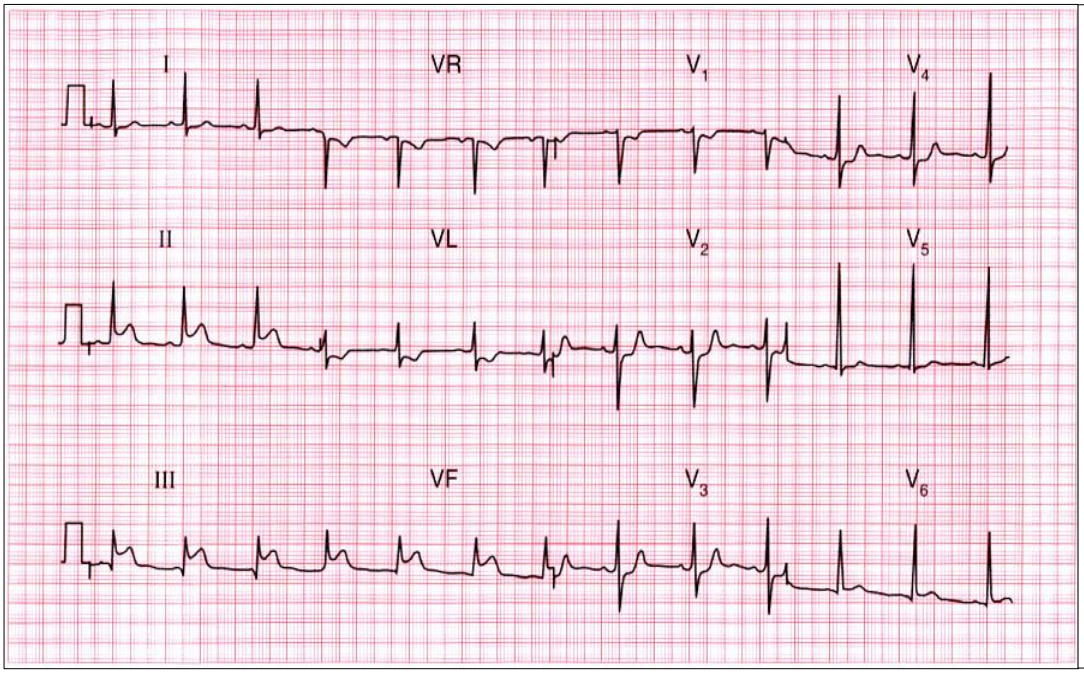


FIGURE 1.67 Inferior wall STEMI. There are ST segment elevation in leads II, III, and aVF with reciprocal changes in V_1-V_4.

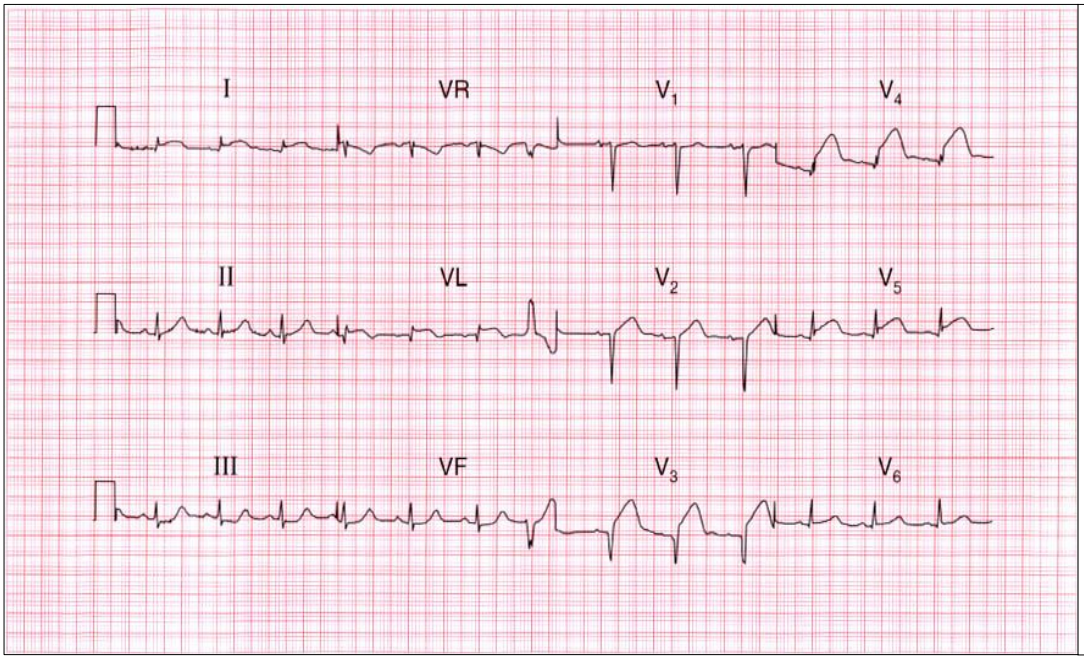


FIGURE 1.68 Anterior wall STEMI. There are ST segment elevation in leads V_3 and V_4 with Q waves.

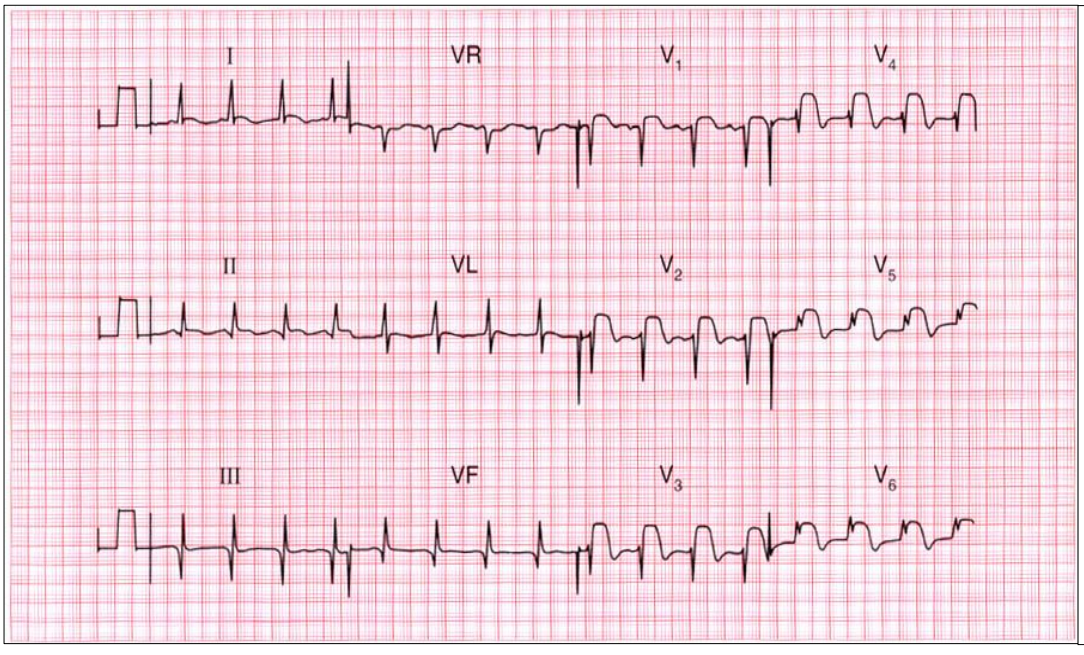


FIGURE 1.69 Anterolateral STEMI with old inferior STEMI. There is ST segment elevation in V_1 – V_6.

Infarction of the posterior wall of the left ventricle does not cause ST segment elevation or Q waves in standard leads, but can be diagnosed by presence of the reciprocal changes in the form of ST segment depression and a tall R wave in leads V_1-V_4 (FIGURE 1.70).

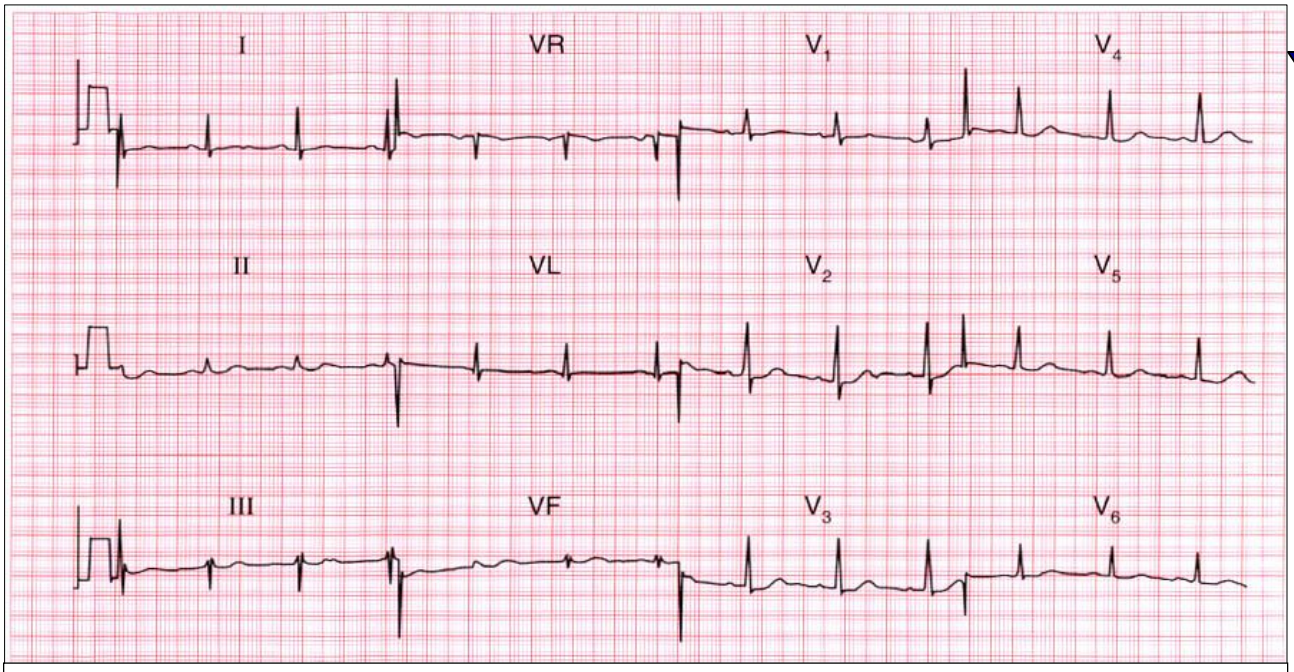


FIGURE 1.70 Probable posterior wall myocardial infarction.

Moreover, posterior infarction can be diagnosed by placing the chest leads on the back of the left side of the chest to obtain V_7, V_8, and V_9 (FIGURE 1.71).

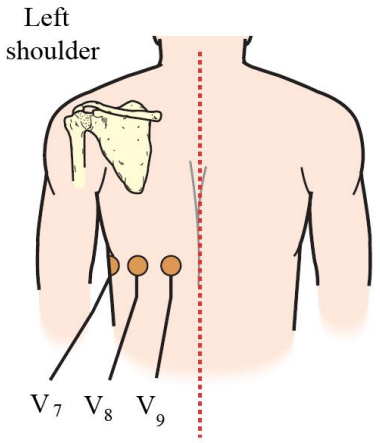


FIGURE 1.71 Posterior leads are placed as follows:
V_7 is placed at the fifth intercostal space posterior axillary line.
V_8 is placed at the posterior fifth intercostal space in left midscapular line.
V_9 is placed directly between V_8 and spinal column at posterior fifth of left ventricle intercostal space.

Inferior infarction may involve the right ventricle. This may be identified by recording from right ventricular leads (FIGURE 1.72). The classic clinical presentation involves a triad of hypotension, clear lung fields, and elevated JVP. The diagnosis is assisted by obtaining right precordial EKG leads (FIGURE 1.72), which are routinely indicated for inferior acute myocardial infarction. Acute ST segment elevation of at least 1 mm (0.1 mV) in one or more of leads V_4R to V_6R is both sensitive and specific (>90%) for identifying acute right ventricular injury, and Q or QS waves effectively identify right ventricular infarction.

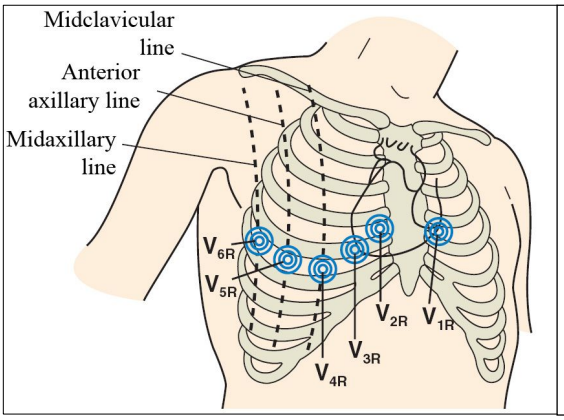


FIGURE 1.72 Right precordial leads are placed at sites corresponding to left precordial leads as follows:
V_1R is placed at the fourth intercostal space to left of sternum.
V_2R is placed at the fourth intercostal space to right of sternum.
V_3R is placed directly between V_2R and V_4R.
V_4R is placed at the fifth intercostal space at right midclavicular line.
V_5R is placed level with V_4R at right anterior axillary line.
V_6R is placed level with V_5R at right midaxillary line.

Sometimes it is perfectly normal for the ST segment to be elevated following an S wave in leads V_2-V_5 (FIGURE 1.73). This is called high take off ST segment and represent an early repolarization of the ventricles. As always, normal variety should be diagnosed by exclusion of other serious causes.

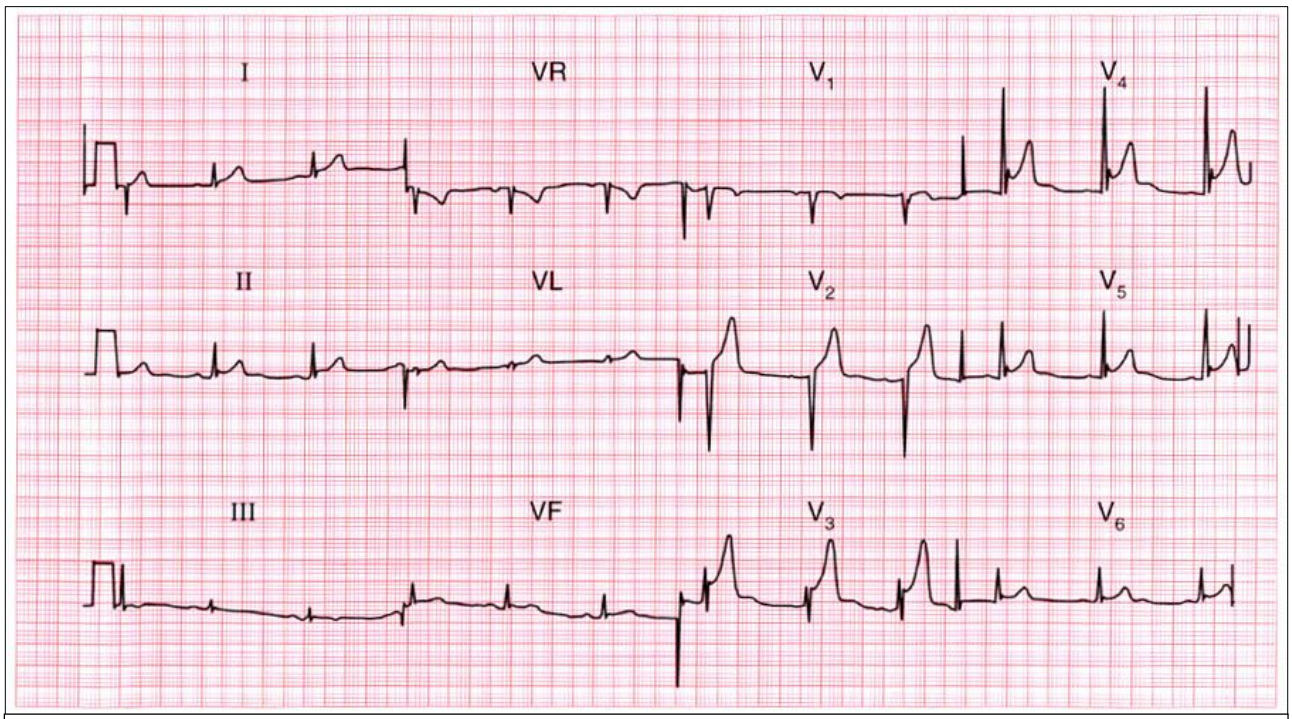


FIGURE 1.73. High take – off ST segment elevation.

NON ST SEGMENT MYOCARDIAL INFARCTION (NSTEMI)

This type is called non ST segment elevation myocardial infarction (NSTEMI), non Q wave myocardial infarction, partial thickness myocardial infarction, or subendocardial myocardial infarction. This type is characterized by deep symmetrical T wave inversion together with a reduction in the height of the R waves in leads facing the infracted area (FIGURE 1.74).

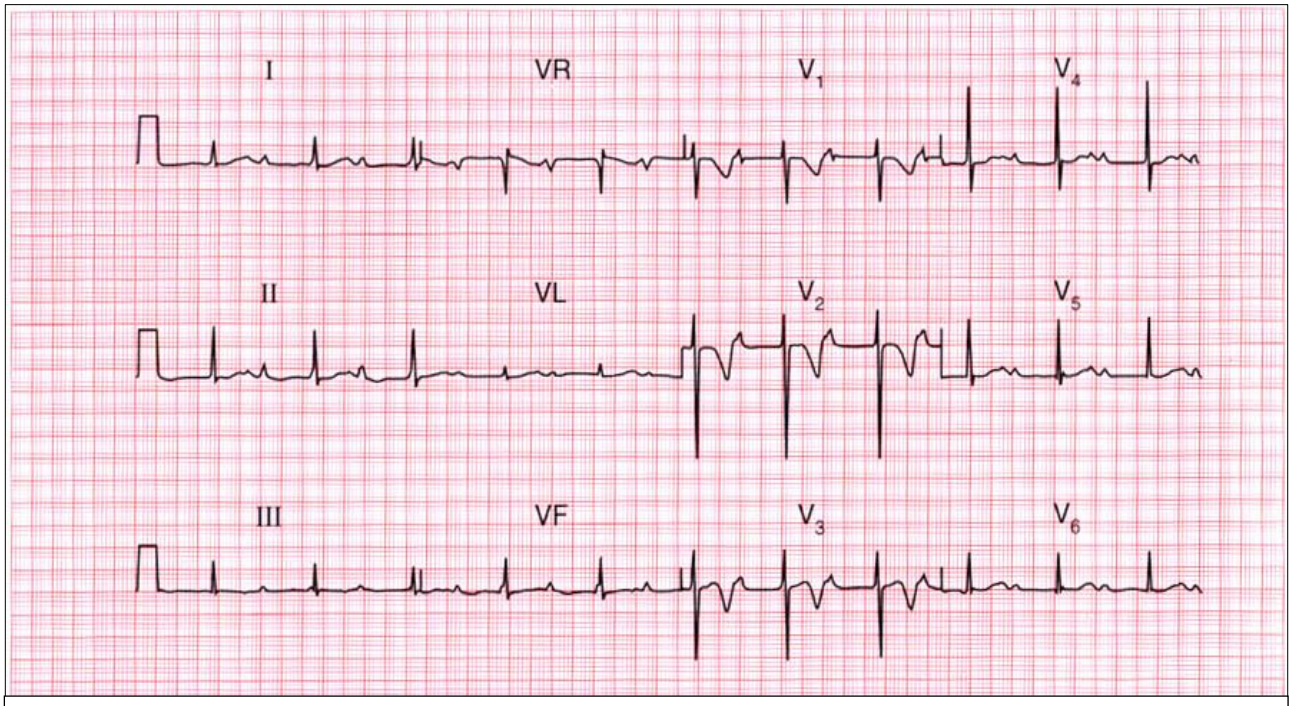


FIGURE 1.74 Anterior wall NSTEMI.

Sometimes one may encounter more than one infarction which may imply multi-vessel disease. This type of presentation is called double wall infarction (FIGURE 1.75).

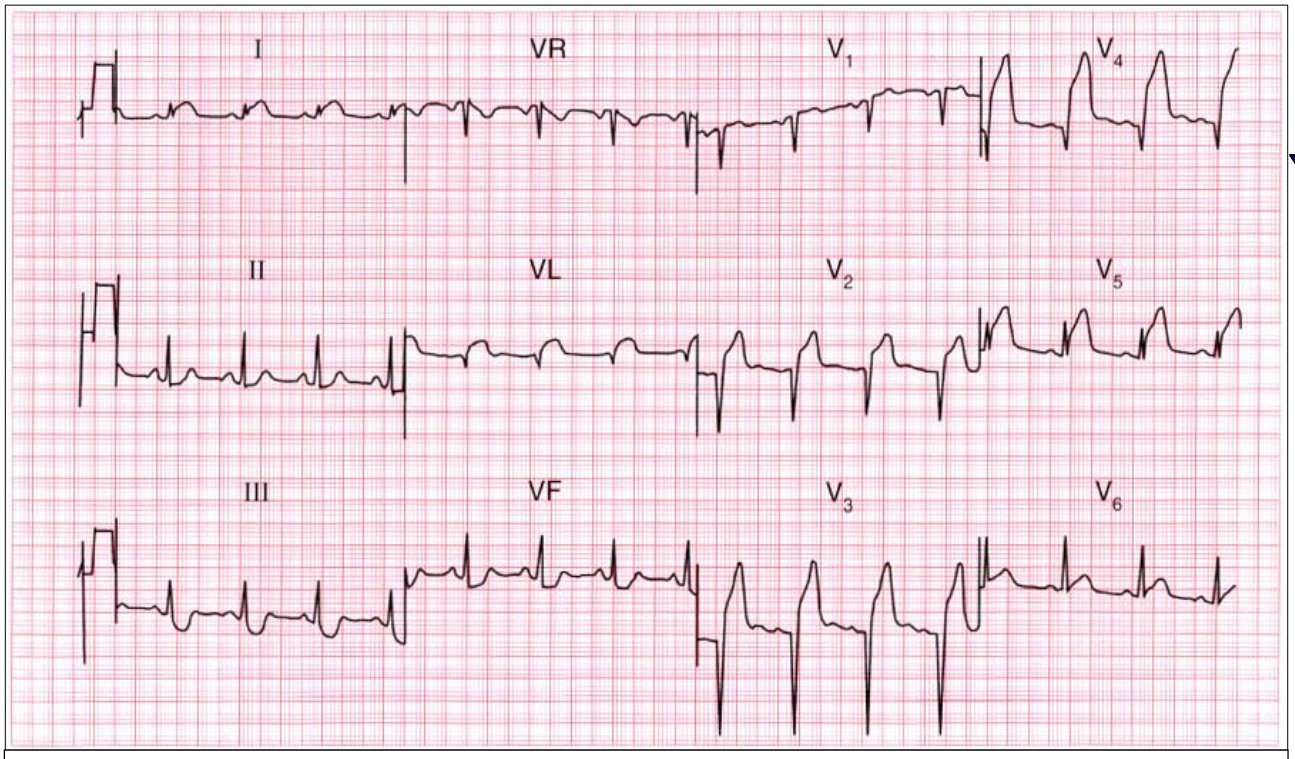


FIGURE 1.75 Acute anterolateral myocardial infarction and inferior ischemia.

STEMI AND BBB

The presence of RBBB usually does not mask typical ST-T wave or Q wave changes, except for rare cases of isolated true posterior acute myocardial infarction. LBBB usually causes disorganized EKG pattern and makes changes due to myocardial infarction more difficult. However a patient admitted with ischemic chest pain and EKG shows LBBB that is known to be new; it can be assumed that an acute infarction has occurred. Certain EKG patterns, although relatively insensitive, suggest acute myocardial infarction if present in the setting of LBBB. These are:

1. ST segment elevation of 1 mm or more in leads with a positive QRS complex.
2. ST segment elevation of 5 mm or more associated with a negative QRS complex.
3. R wave regression from V_1 to V_4.
4. ST segment depression of 1 mm or more in leads V_1, V_2, or V_3.
5. Q waves in two of leads I, aVL, V_5, V_6.

ST SEGMENT DEPRESSION

Normally ST segment may be depressed in lead III, but not aVF and often the segment slopes upward. On the other hand, digoxin causes down sloping depression of the ST segment (FIGURE 1.76). This finding of down slopping ST segment depression is called reverse tick sign.

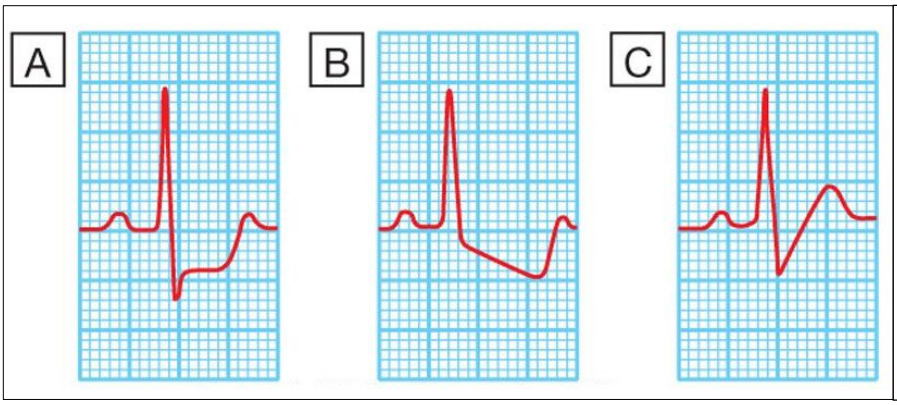


FIGURE 1.76 Varieties of ST segment depression.
A. Planar ST depression is usually indicative of myocardial ischemia.
B. Down-sloping depression usually indicates myocardial ischemia or digoxin therapy.
C. Up-sloping depression may be a normal finding.

Horizontal (Planar) depression of the ST segment more than two small squares indicates ischemia or even more than one small square in patient with chest pain. The EKG changes are best seen in the leads which face the ischemic area (FIGURES 1.77 and 1.78).

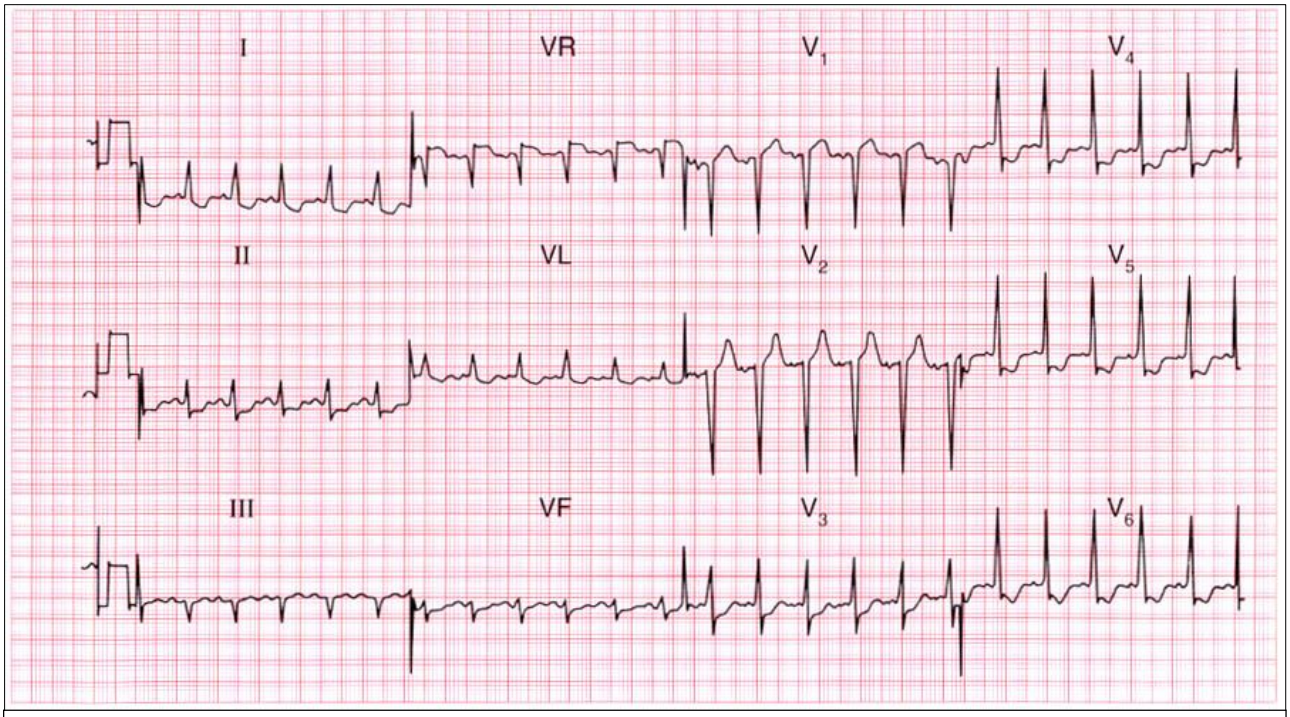


FIGURE 1.77 Anterolateral myocardial ischemia. There is horizontal ST segment depression involving the anterior leads and down sloping of the lateral leads. Lead aVF shows up – slopping ST segment depression.

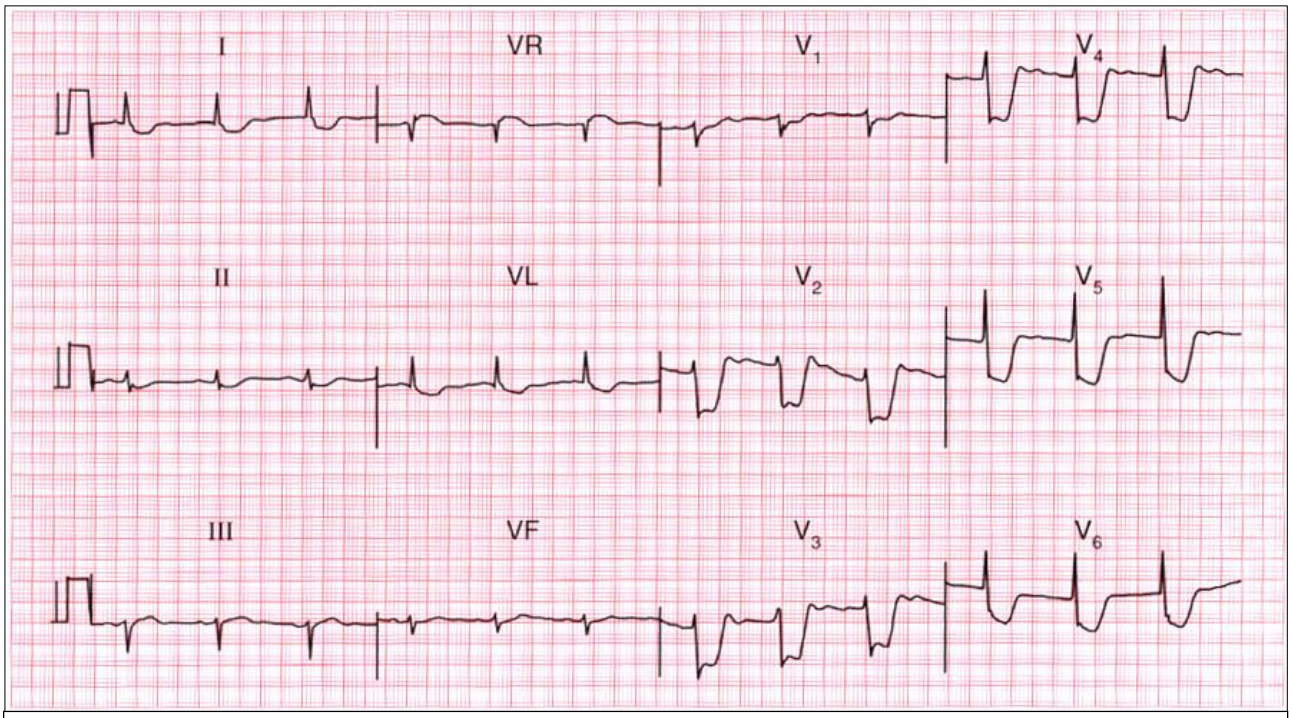


FIGURE 1.78 Severe anterolateral ischemia.

STEP 10 T WAVE

T wave is the most variable part of the EKG. Normally the T wave has the following characteristics:

1. Inverted in aVR
2. Inverted in aVL provided that the P wave is also inverted
3. Inverted in lead III, but not aVF
4. Inverted in V_1, V_2 in young and V_3 in black people

The T wave could be inverted, flattened or peaked according to the pathology. One should consider the following clinical settings:

Acute myocardial infarction	Hyperacute (peaked) T wave
Established STEMI	Terminal T wave inversion
NSTEMI	Deep symmetrical T wave inversion
Ischemia	Inverted T wave
Left or right ventricular hypertrophy	T wave inversion (strain pattern)
Bundle branch block	T wave inversion
Pulmonary embolism	T wave inversion
Hyperkalemia	Peaked T wave
Hypokalemia	Flat and prolonged
Hypertrophy cardiomyopathy	Deep T wave inversion mimics infarction called (pseudoinfarct pattern)
Subarachnoid hemorrhage	T wave inversion (cerebral T wave)

Many minor degrees of ST segment and T wave abnormalities such as T wave flattening are usually of no great significance and are best reported as non specific ST–T changes. However if these T wave changes are associated with ischemic chest pain, or elevated cardiac enzymes, or are new and deep (more than three small squares) one should considered them significant (FIGURE 1.79).

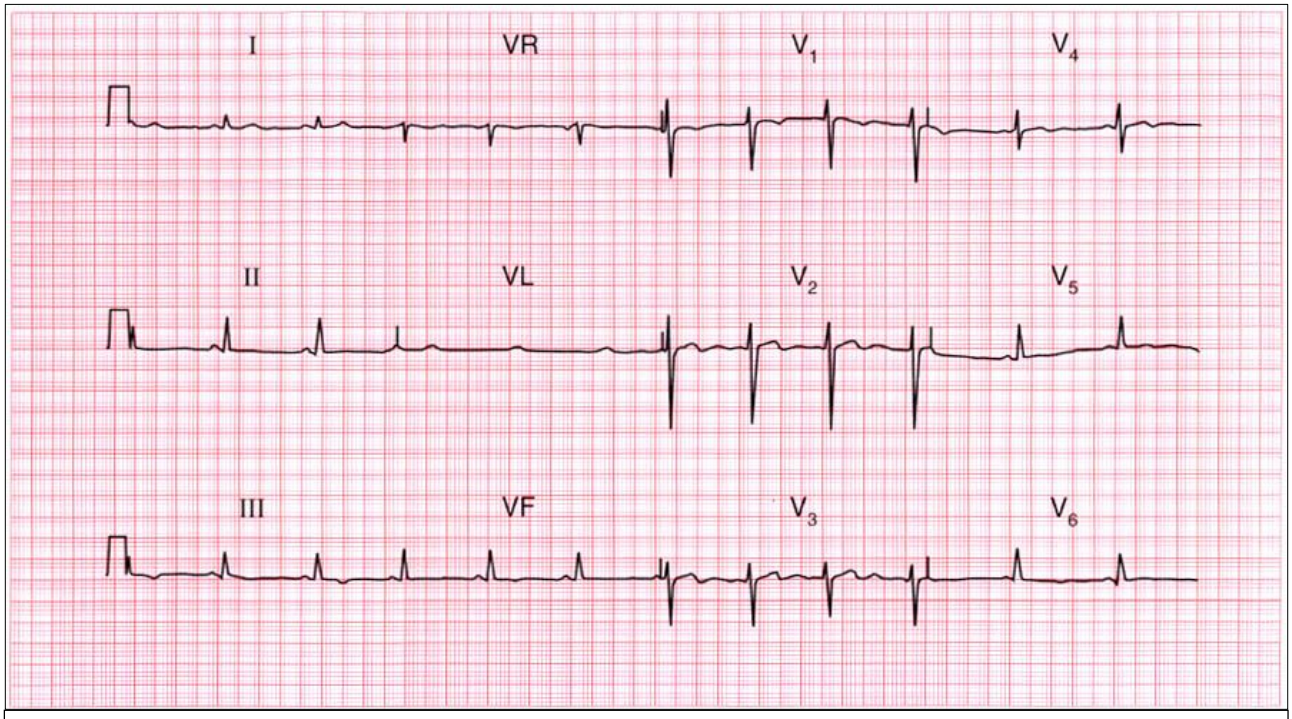


FIGURE 1.79 Anterior and inferior wall ischemia.

The EKG in pulmonary embolism is non specific and may show sinus tachycardia (most common) or features of right axis deviation, right ventricular hypertrophy, or right bundle branch block. A large S wave in lead I and a deep Q wave in lead III as well as inverted T wave in lead III may also be seen (FIGURE 1.80). This pattern is called $S_1Q_3T_3$. Unlike an inferior infarction, in which Q waves are usually seen in at least two of the inferior leads, the Q waves in an acute pulmonary embolus are generally limited to lead III.

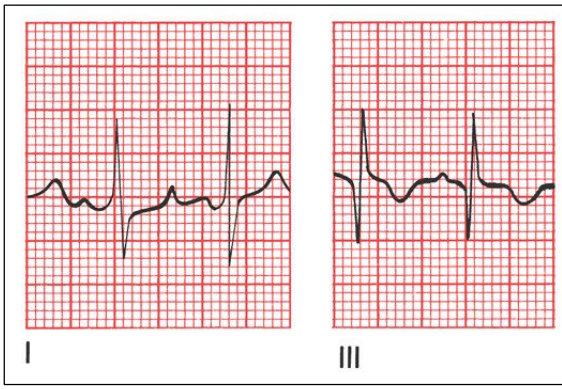


FIGURE 1.80 $S_1Q_3T_3$ of massive pulmonary embolism.

Hyperkalemia produces a progressive evolution of changes in the EKG that can culminate in ventricular fibrillation and death. As the potassium begins to raise, the T waves across the entire 12-lead EKG begin to peak (FIGURE 1.81 A). This effect can easily be confused with the peaked T waves of an acute myocardial infarction. One difference is that the changes in an infarction are confined to those leads overlying the area of the infarct, whereas in hyperkalemia, the changes are diffuse. With a further increase in the serum

potassium, the PR interval becomes prolonged, and the P wave gradually flattens and then disappears (FIGURE 1.81 B). Ultimately, the QRS complex widens until it merges with the T wave, forming a sine wave pattern (FIGURE 1.81 C). Ventricular fibrillation may eventually develop.

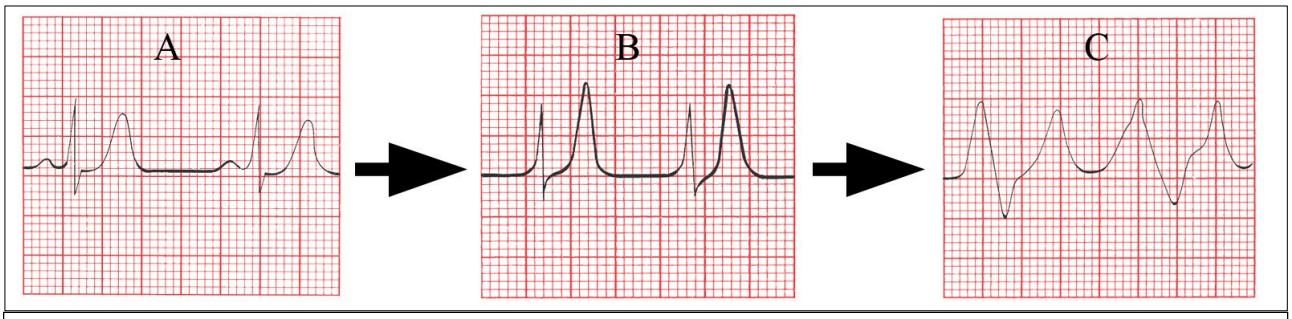


FIGURE 1.81 Changes of hyperkalemia.

With hypokalemia, the EKG may show ST segment depression, flattening of the T wave, and appearance of a U wave (FIGURE 1.82).

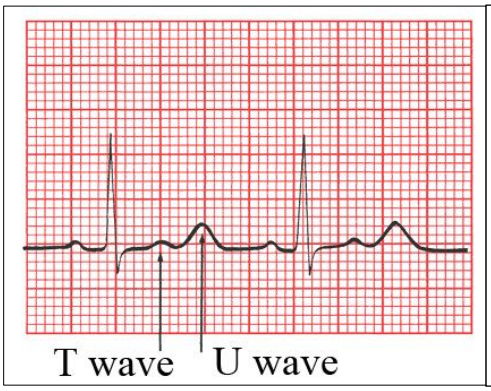


FIGURE 1.82 Changes of hypokalemia.

In hypertrophic cardiomyopathy, EKG usually shows left ventricular hypertrophy, often with prominent septal Q waves that can be misdiagnosed as indicative of infarction (FIGURE 1.83).

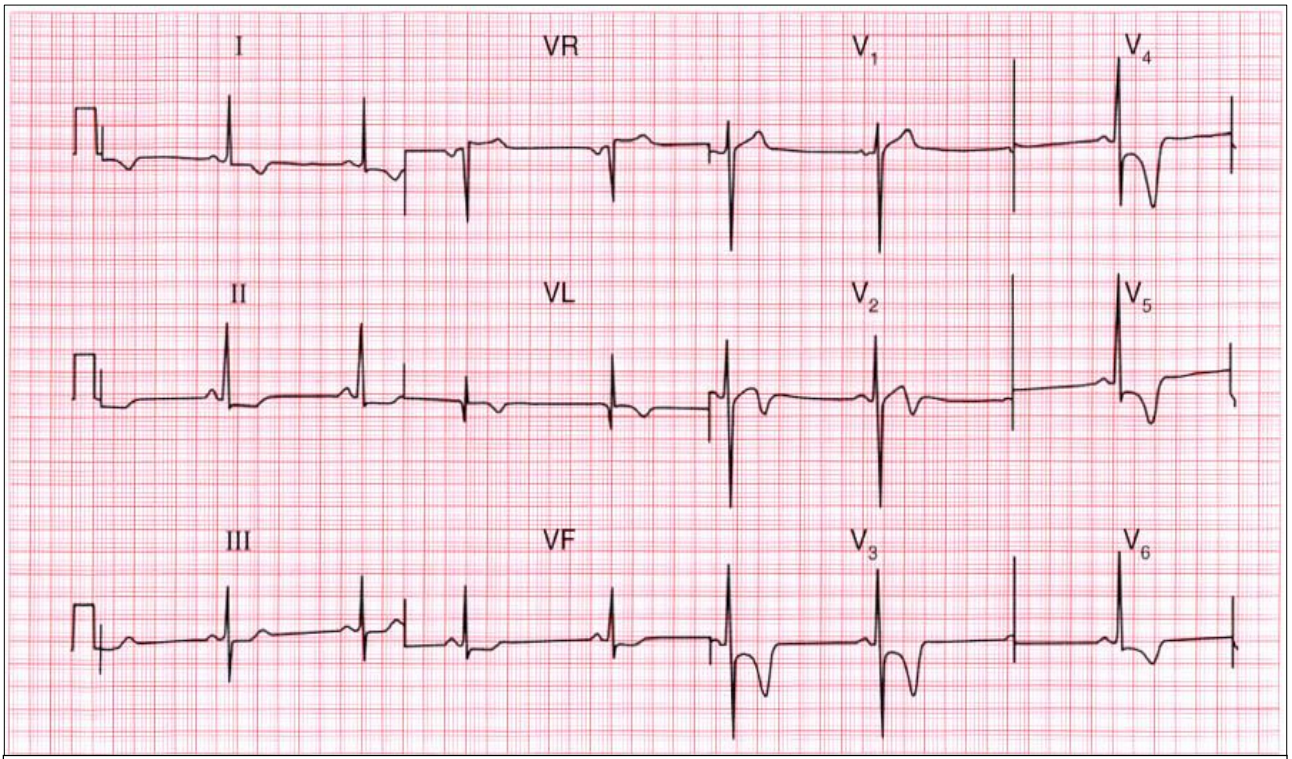


FIGURE 1.83 Hypertrophic cardiomyopathy.

The EKG in subarachnoid hemorrhage frequently shows ST-segment and T-wave changes similar to those associated with cardiac ischemia (FIGURE 1.84). Prolonged QRS complex, increased QT interval, and prominent "peaked" or deeply inverted symmetric T waves are usually secondary to the intracranial hemorrhage. There is evidence that structural myocardial lesions produced by circulating catecholamines and excessive discharge of sympathetic neurons may occur after subarachnoid hemorrhage, causing these EKG changes and a reversible cardiomyopathy sufficient to cause shock or congestive heart failure. The sympathetic nerves themselves appear to be injured by direct toxicity from the excessive catecholamine release. An asymptomatic troponin elevation is common. Serious ventricular dysrhythmias are unusual.

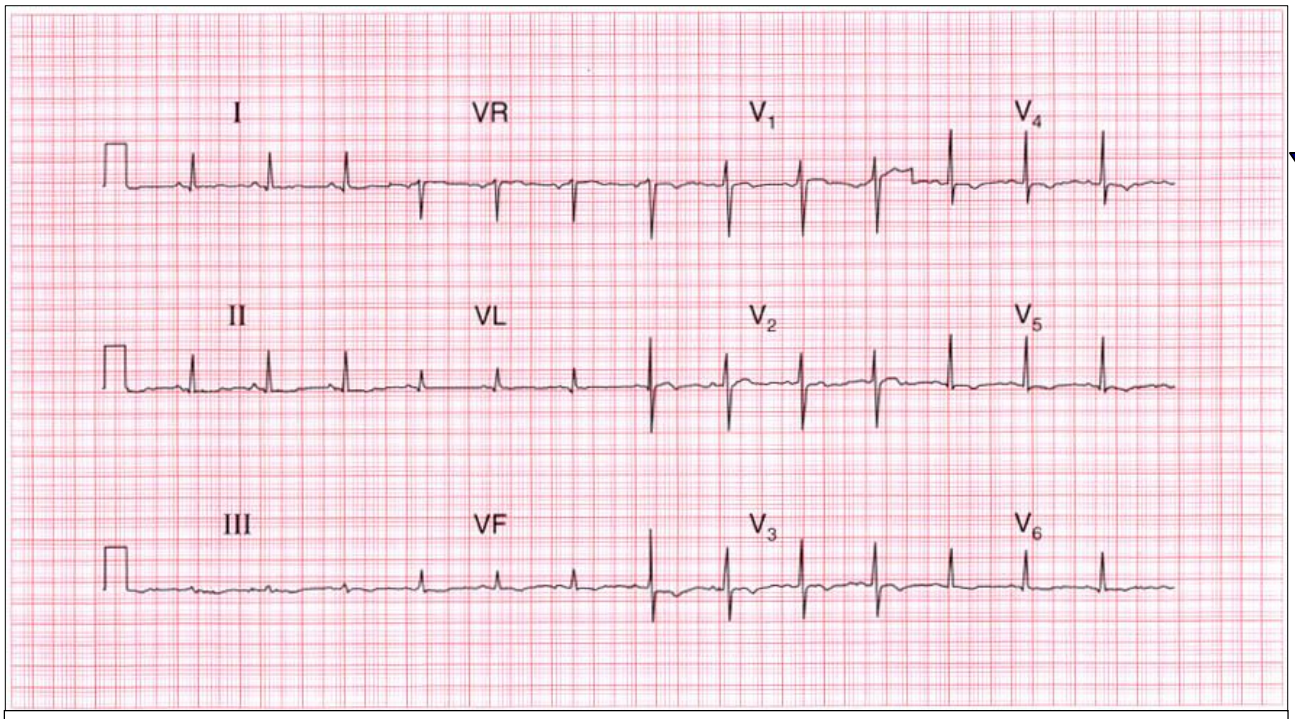


FIGURE 1.84 Subarachnoid hemorrhage.

STEP 11 QT INTERVAL

The QT interval is measured from the start of the QRS complex to the end of the T wave. It varies with heart rate (the faster the heart rate, the shorter is the QT interval), gender and time of day. There are several different ways of correcting for heart rate, but the simplest one is Bazett's formula. In this, the corrected QT interval, QTc, is calculated as (QTc = QT interval / √R-R interval)
Corrected QT intervals are considered long if greater than 440 msec in men (11 small squares) and 450–460 msec in women (11.5 small squares), but in practice long QT is considered when the QT interval is more than two large squares (FIGURE 1.85).

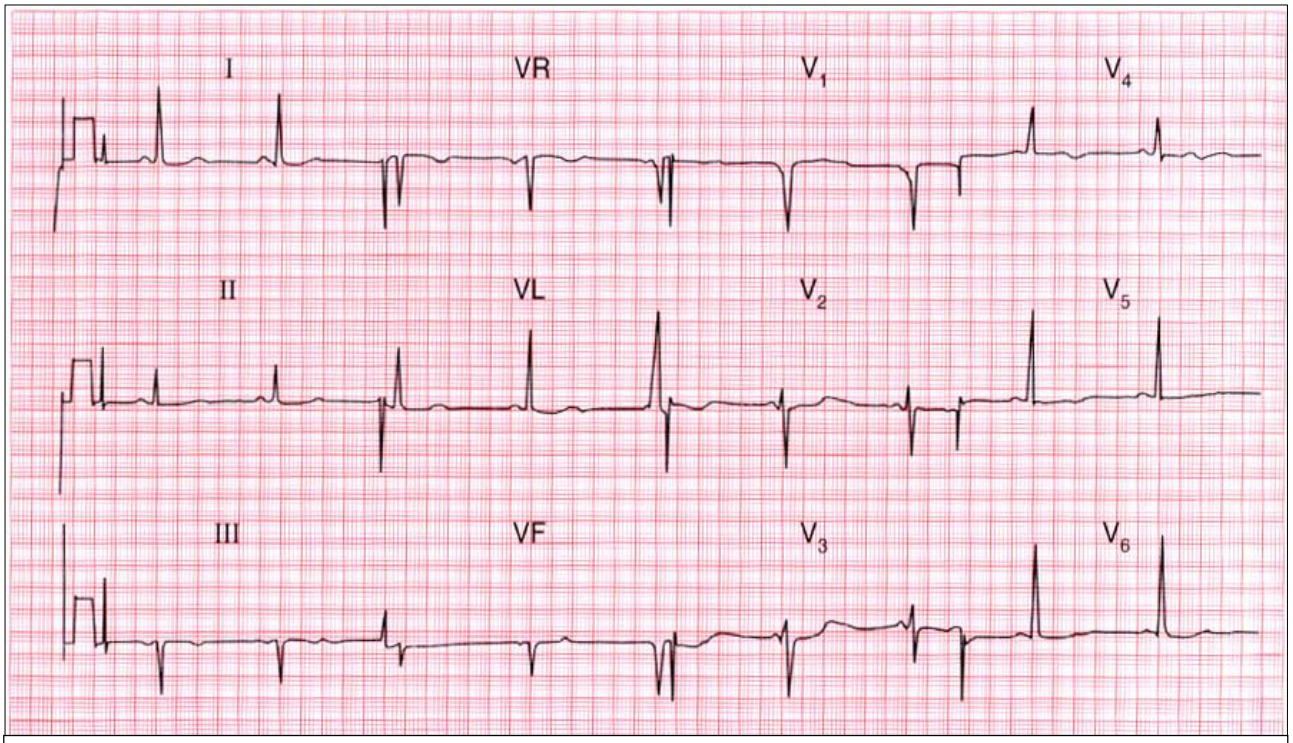


FIGURE 1.85 Markedly prolonged QT interval.

Short QT syndrome is considered when QT interval is less than 300 msec (7.5 small squares). Causes of abnormal QT are shown in BOX 1.7.

BOX 1.7 Causes of abnormal QT interval

PROLONGED QT INTERVAL

1. Congenital

Jervell-Lange-Nelson syndrome
Romano-ward syndrome

2. Drugs

Disopyramide
Amiodarone
Sotolol
Procainamide
Tricyclic antidepressant
Erythromycin

3. Electrolyte abnormalities

Hypokalemia
Hypocalcemia
Hypomagnesemia

SHORT QT INTERVAL

Hyperkalemia
Hypercalcemia
Digoxin therapy

STEP 12 ADDITIONAL WAVES

The normal U wave is a small, rounded deflection ≤1 mm that follows the T wave and usually has the same polarity as the T wave. It is thought to be due to depolarization of the interventricular (Purkinje) conduction system. It is commonly seen in normal subjects in the anterior chest leads. It may be seen with bradycardia and left ventricular hypertrophy. An abnormal increase in U-wave amplitude is most commonly due to drugs (e.g., amiodarone, sotalol, procainamide, and disopyramide) or to hypokalemia (FIGURE 1.82). Very prominent U waves are a marker of increased susceptibility to the torsades de Pointe type of ventricular tachycardia. Inversion of the U wave in the chest leads is abnormal and may be a subtle sign of ischemia.
Osborne or J wave is a small hump seen at the end of the QRS complex and is a characteristic of hypothermia (FIGURE 1.86). It may however be seen in normal subjects.

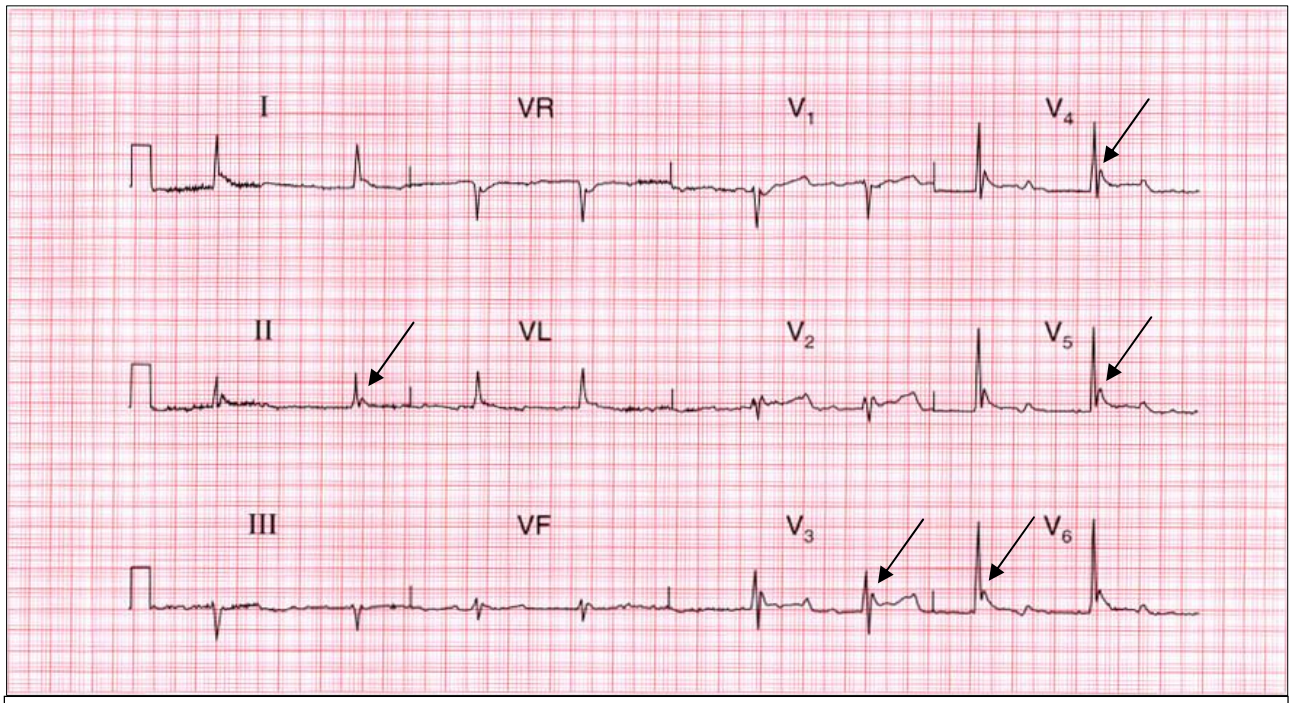


FIGURE 1.86 Osborne waves.

An epsilon wave (e wave) refers to the terminal notching of the QRS complexes in V_1–V_3. When it is distinct and appears separated from the QRS complex, it is referred to as an epsilon wave (FIGURE 1.87) and suggests right ventricular dysplasia.

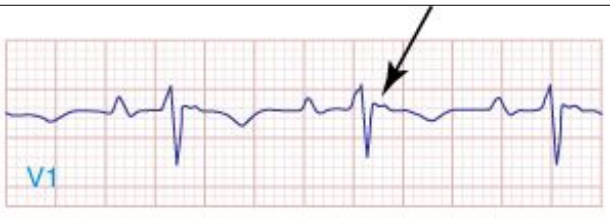


FIGURE 1.87 Epsilon waves.

Spikes whether atrial or ventricular is a sharp pacing preceded the P wave if the atrium is paced or the QRS complex if the ventricle is paced (FIGURE 1.88).
Spikes are indicators for the presence of the pacemaker. In atrial pacing, the QRS complex remains normal. In ventricular pacing, the QRS complex is wide and abnormal (FIGURE 1.89).

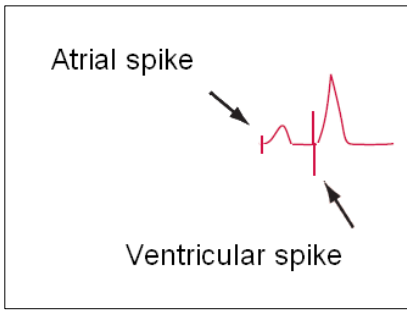


FIGURE 1.88 Atrial and ventricular spikes.

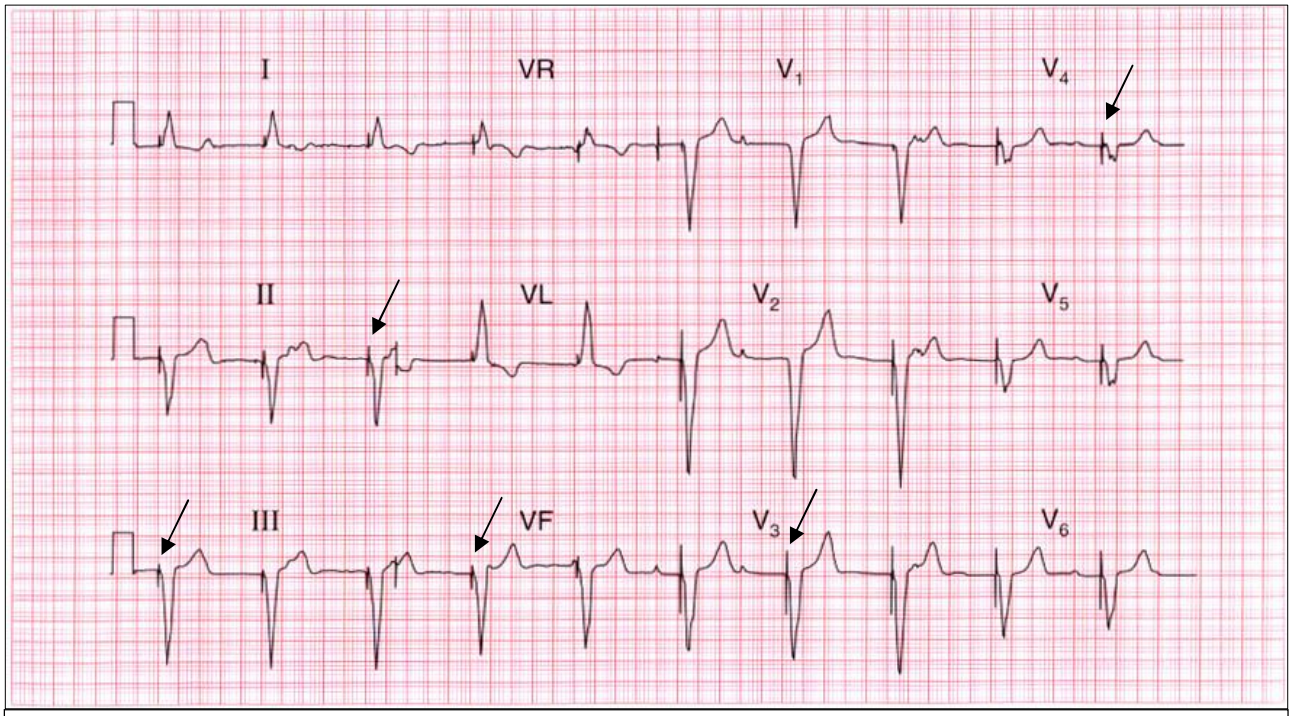


FIGURE 1.89 Artificial pacemaker with ventricular pacing.

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