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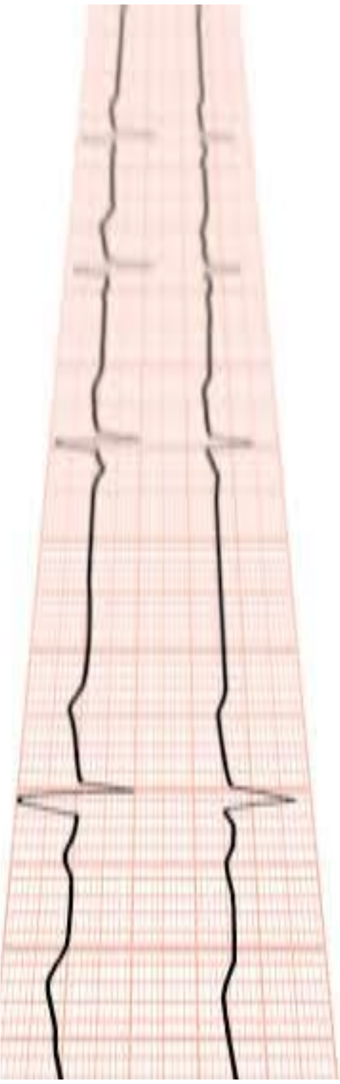

RAJOOJ'S CLINICAL EKG

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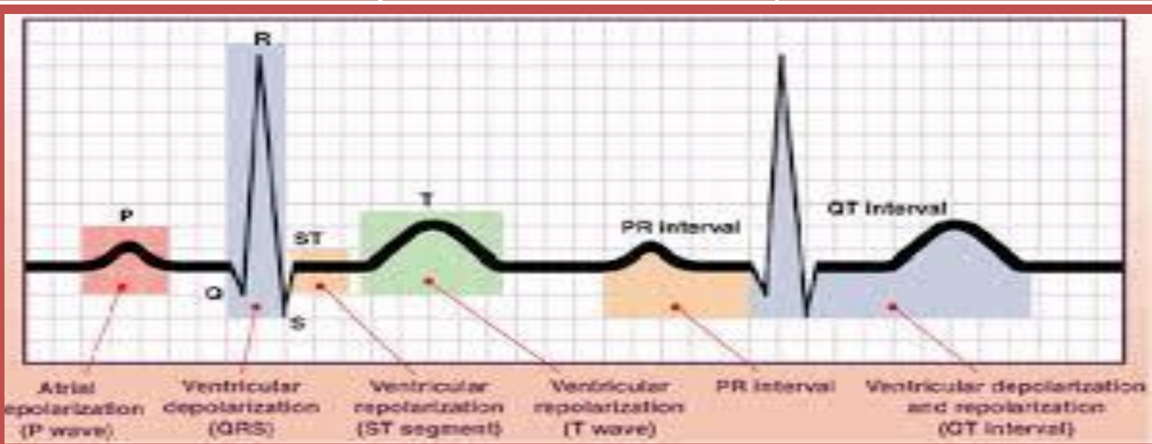
Fourth Edition 2015



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PREFACE TO THE FOURTH EDITION

This book is specially designed to meet the need of undergraduate students and resident doctors. It can however be read as a basic guide by postgraduate students. This book explains the techniques, indications and interpretations of the EKG. The aim of this book is to help the medical undergraduates and resident doctors efficiently acquire the practical means of the EKG interpretation and to provide them with the sound-up-to date information about this interpretation since the ability of doctors to accurately interpret the EKG is crucial.

A. RAJOOJ H.

INTRODUCTION

Contraction of cardiac muscles is accompanied by electrical activity called depolarization that can be detected by electrodes attached to the surface of the body via the EKG (ECG). EKG is used to detect cardiac ischemia, infarction, heart block, and myocardial hypertrophy. It may also give information about electrolyte disturbances and the toxicity of certain drugs.

RECORDING AN EKG

To obtain a 12-lead EKG, four wires (electrodes) are attached to the four limbs (FIGURE 1.1 The left view) and six wires are attached at different locations on the chest (FIGURE 1.1 The right view).

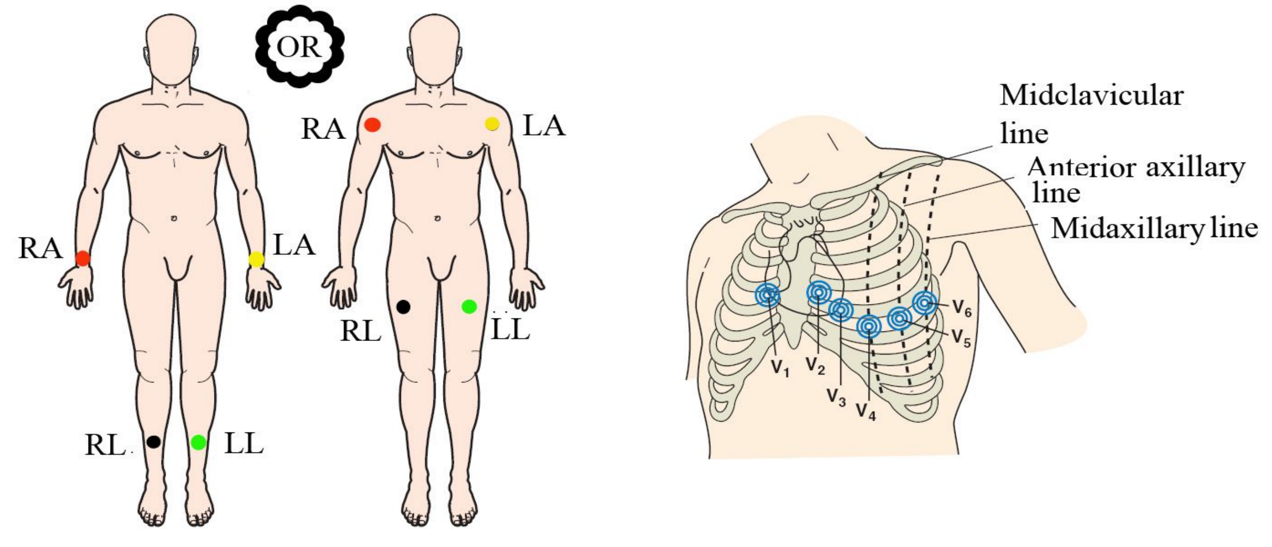


FIGURE 1.1 ECG connections. RA refers to right arm, LA refers to left arm, RL refers to right leg, and LL refers to left leg.

The total of ten wires provides 12 leads; six limb leads and six chest (precordial) leads. Limb wires are placed on the right arm (usually red in color), left arm (usually yellow in color), right leg (usually black in color), and left leg (usually green in color). With only these four electrodes, six leads are viewed. These are the three bipolar limb leads (I, II, and III) and the three unipolar leads (VR, VL, and VF). The three unipolar limb leads (FIGURE 1.2 A) are connected to a central terminal which is electrically neutral (connected to the right leg). The signal recorded from the electrode on the left arm is augmented (designated by the letter a) relative to the central terminal and is therefore designated lead aVL. Similarly augmented signals are obtained from the right arm (aVR) and left foot (aVF). The bipolar leads; I, II and III (FIGURE 1.2 B) are generated by subtraction of the signals from two adjacent leads. Lead I is the left arm minus the right arm, lead II is the left leg minus right arm and lead III is the left leg minus left arm.

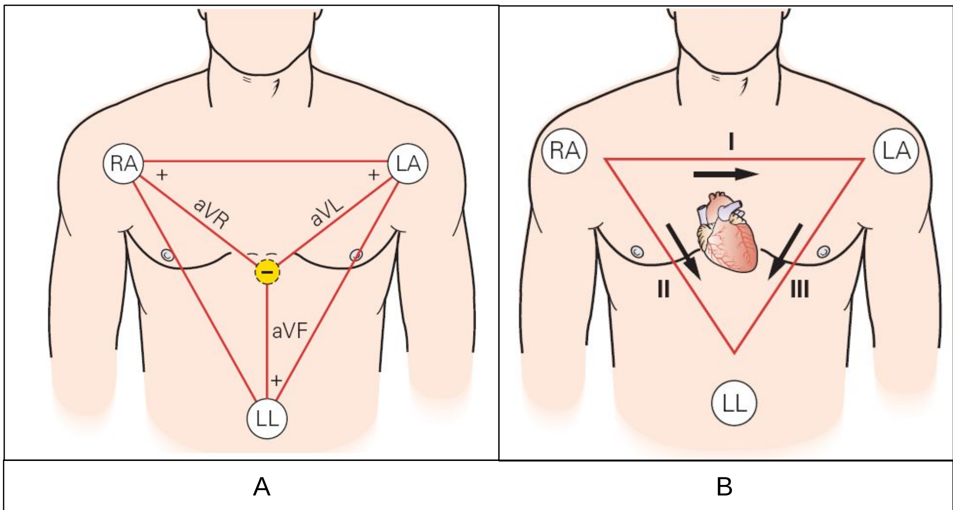


FIGURE 1.2 A and B demonstrate positions of limb leads. RA means right arm, LA means left arm, and LL means left leg (foot)

The six unipolar chest leads V_1-V_6 are attached to anterior chest wall (FIGURE 1.1 The right view) in the following positions:

V_1	This lead is placed at the right fourth intercostal space
V_2	This lead is placed at the left fourth intercostal space
V_3	This lead is placed between V_2 and V_4
V_4	This lead is placed at the apex beat (left fifth intercostal space, midclavicular line)
V_5	This lead is placed at the left fifth intercostal space anterior axillary line
V_6	This lead is placed at left fifth intercostal space mid axillary line

Thus leads V_1 and V_2 look at the right ventricle, V_3-V_4 look at the interventricular septum and the anterior wall of the left ventricle, and V_5 –V_6 look at the anterior and lateral walls of the left ventricle.

TIME AND SPEED OF THE EKG

EKG machines record changes in electrical activity by drawing a trace on a moving paper strip. All EKG machines run at a standard rate and use paper with standard sized squares (FIGURE 1.3). The horizontal scale represents time, such that, at a standard paper speed of 25 mm/sec, each small square (1mm) represents 0.04 second and each large square (5 mm) represents 0.2 second. There are therefore five large squares per second (25 small squares) and 300 per minute. The vertical scale represents amplitude (10 mm = 1mV).

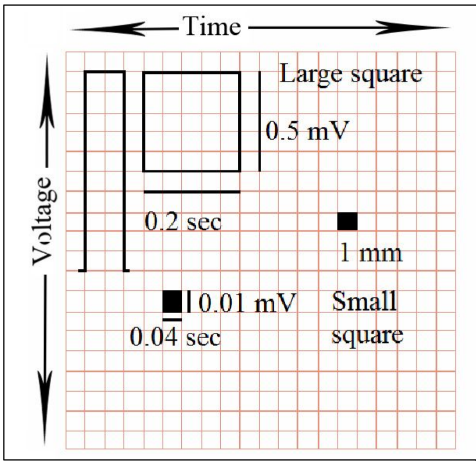


FIGURE 1.3 ECG speed and time.

ORIGIN OF THE EKG DEFLECTIONS

P wave is the deflection produced by atrial activation; QRS complex is the complex of deflections produced by ventricular activation; T wave is the deflection produced by ventricular repolarization (FIGURE 1.4).

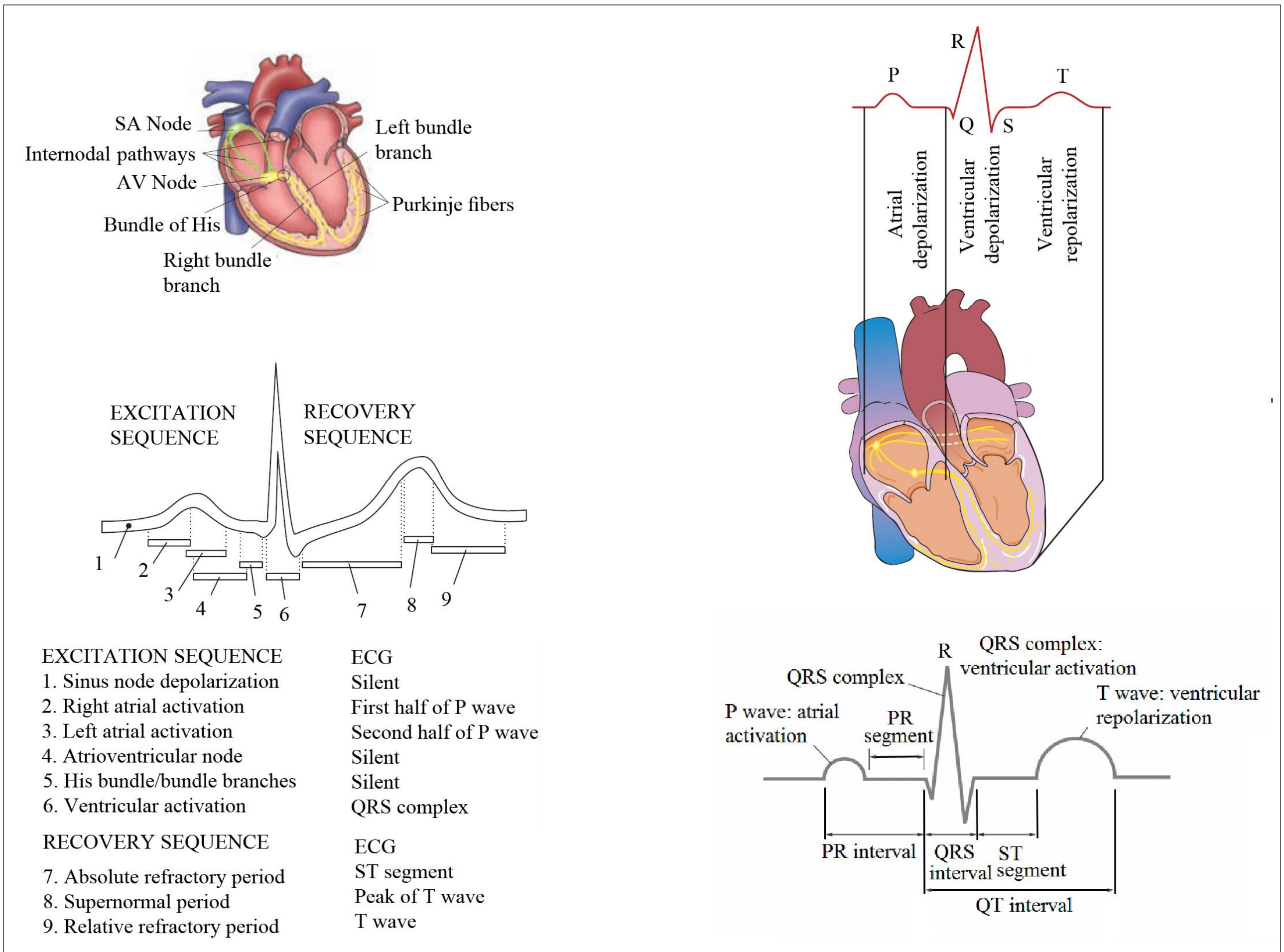


FIGURE 1.4 Origin of the ECG deflections.

EKG WAVES NOMNECLATURE

Components of the QRS complex are designed as follows (FIGURE 1.5):

I Q wave is the initial downward deflection (if present) followed by an R wave

II R wave is the first upward deflection whether preceded by a Q wave or not

III S wave is the downward deflection following the R wave; S wave is only considered when it touches the base line

IV R′ wave is the second upward deflection (if present)

V S′ wave is a subsequent negative deflection following the first S wave (if present)

VI QS is a single negative deflection represent the entire QRS complex

VII Capital letters (Q, R, and S) refer to relatively large waves (over 1 large square); small letters (q, r, s) refer to relatively small waves (under 1 large square)

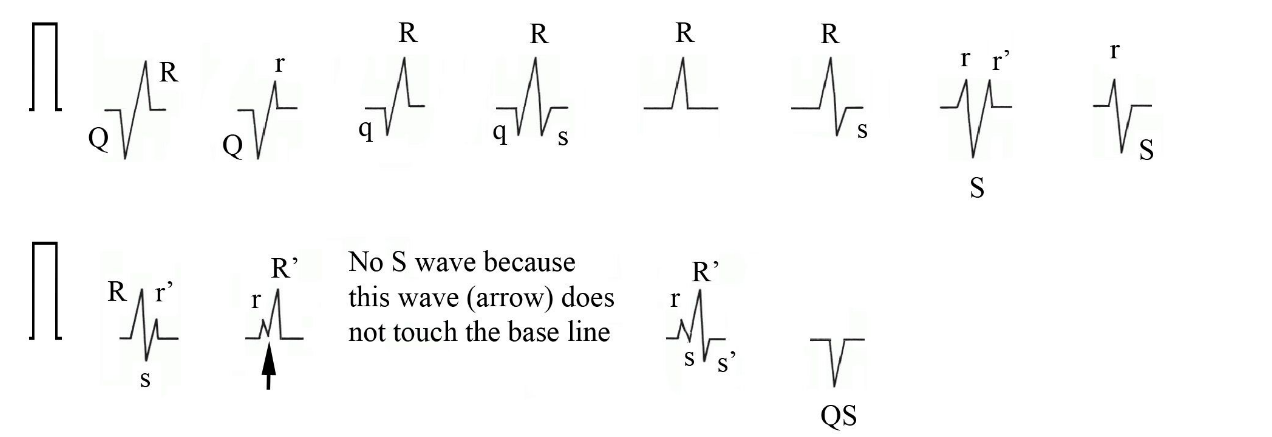


FIGURE 1.5 Names of the ECG waves.

TRY TO NAME THE FOLLOWING QRS COMPLEXES

QRS COMPLEXES IN LIMB LEADS

EKG is arranged so that when a depolarization wave spreads towards a lead, the stylus moves upwards i.e., R wave is larger than S wave and when it spreads away from the lead, the stylus moves downwards that is to say S wave is larger than R wave. If QRS complex is predominantly upward or positive (R wave > S wave), depolarization is mainly toward that lead. If the QRS complex is predominantly downward or negative (S wave > R wave), depolarization is mainly away from that lead. When depolarization wave is moving at right angles to the lead, R wave and S wave are of equal size. Seen from the front, depolarization wave normally spreads through the ventricles from 11 to 5 o'clock (FIGURE 1.6) so that the aVR is normally mainly downward (negative). Thus when the cardiac axis is normal, the QRS complex are predominantly upright in leads I, II, and III (FIGURE 1.6).

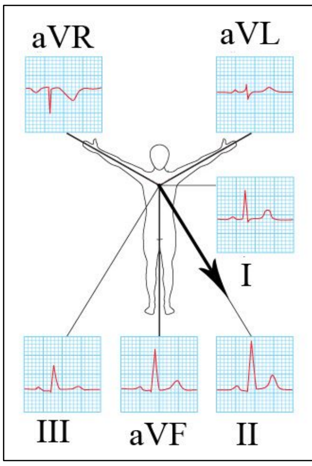


FIGURE 1.6 Direction of the normal depolarization waves in limb leads.

QRS COMPLEXES IN CHEST LEADS

Depolarization of the interventricular septum occurs first (FIGURE 1.7) and moves from the left to the right thus generating an initial negative deflection in V_6 (Q wave) and an initial positive deflection in V_1 (R wave). The second phase of depolarization causes an activation of the body of the left ventricle, which creates a large positive deflection or R wave in V_6 with reciprocal changes in V_1. The third and final phase of depolarization involves the right ventricle and produces a small negative deflection or S wave in V_6. The QRS complex in the chest leads shows progression from V_1 where the QRS is predominantly downward (negative) to V_6 where it is predominantly upward (positive). The point at which R wave and S wave are of equal size is called the transition point which indicates the position of the interventricular septum. This point is normally at V_3/V_4 (FIGURE 1.7).

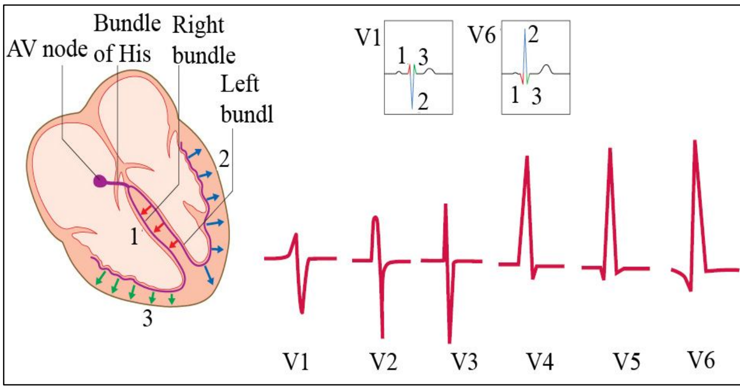


FIGURE 1.7 The phases of depolarization and resultant electrical pictures.

EKG DESCRIPTION AND INTERPRETATION

EKG should be read in two fundamental steps; description and then interpretation. The description of the EKG should always be performed in an orderly fashion as shown in BOX 1.1.

BOX 1.1 Steps for EKG description

STEP 01	Technical quality	STEP 07	PR interval
STEP 02	Cardiac rhythm	STEP 08	QRS complex
STEP 03	Heart rate	STEP 09	ST segment
STEP 04	Cardiac axis	STEP 10	T wave
STEP 05	P wave	STEP 11	QT interval
STEP 06	PR segment	STEP 12	Additional waves

STEP 01 TECHNICAL QUALITY OF THE EKG

This step is undertaken under three headings. Look first at the speed which should be 25 mm/s and look then to the amplitude which is normally 10 mm/s. Both these data are usually provided in the bottom of the EKG paper although some devices provide them in the upper part of the EKG paper (FIGURE 1.8). The third step involves looking at the aVR lead. The depolarization wave normally spreads through the ventricles from 11 o'clock to 5 o'clock thus the deflections in aVR are normally mainly downward i.e., negative. Negative, neutral and positive aVR leads are shown in FIGURE 1.9.

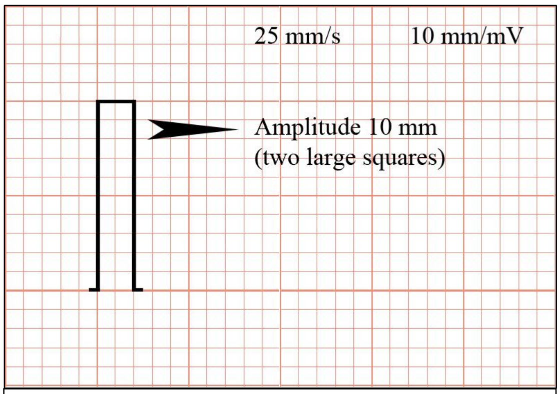


FIGURE 1.8 Normal ECG speed and amplitude. You can slow the speed of the ECG machine and reduce the amplitude.

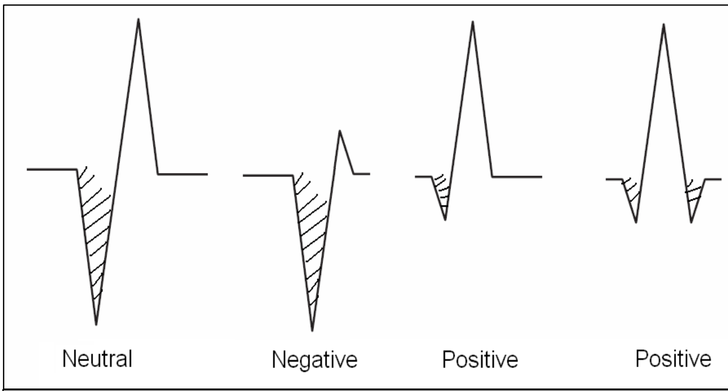


FIGURE 1.9 Calculation of the QRS net area. The area below the isoelectric line (hatched) is subtracted from the area above the line.

If the aVR deflections are upward (positive), you should think about three possibilities; reversed limb leads placement (limb leads have been incorrectly attached to the limbs), dextrocardia, and right ventricular hypertrophy/strain or right bundle branch block.

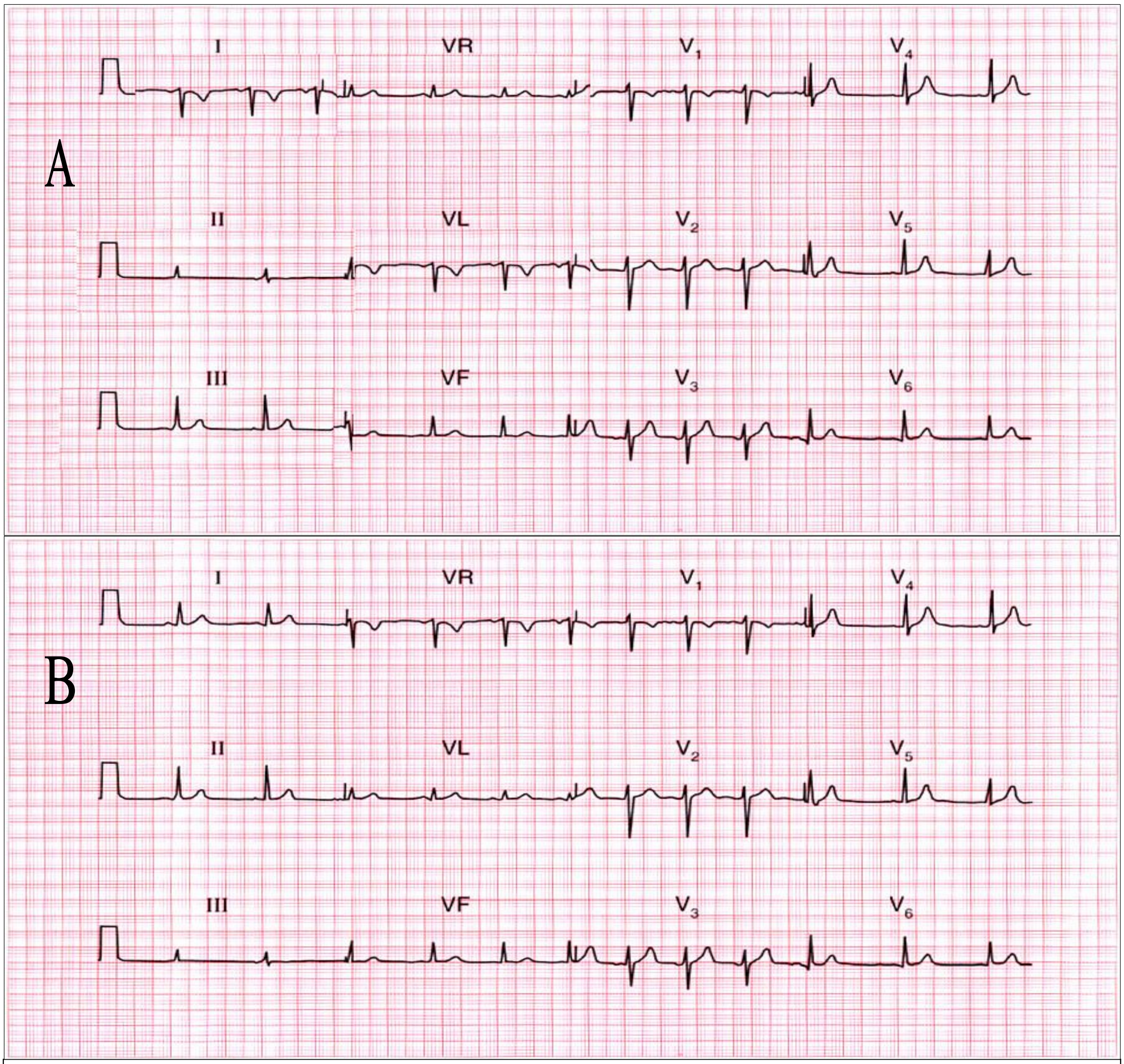


FIGURE 1.10 Reversed limb leads (A) and proper lead placement (B). Look at the aVR in view A and look again at view B when the limb leads are properly placed. Lead aVR is interchanged with lead aVL; lead II is interchanged with III; lead I become negative; and lead aVF is unchanged. All these changes are reversed when the leads are properly placed (view B).

In both reversed limb leads and dextrocardia, lead I will show negative P wave and negative QRS and T wave, aVR is reversed with aVL (aVR becomes aVL i.e. positive aVR and negative aVL), lead III is reversed with lead II, and lastly the aVF is unchanged. The difference between the two conditions is that in reversed limb lead (FIGURE 1.10) the precordial leads remain normal (progression from predominantly negative QRS complexes to positive QRS complexes) while in dextrocardia (FIGURE 1.11) the precordial leads will show poor R wave progression from V_1 through V_6 (precordial leads remain negative). Moreover, if the EKG of a patient with dextrocardia is repeated with the limb leads reversed and the chest leads are placed to the right side of the chest, the EKG becomes like that of a normal person. The approach to a positive aVR is shown in FIGURE 1.12.

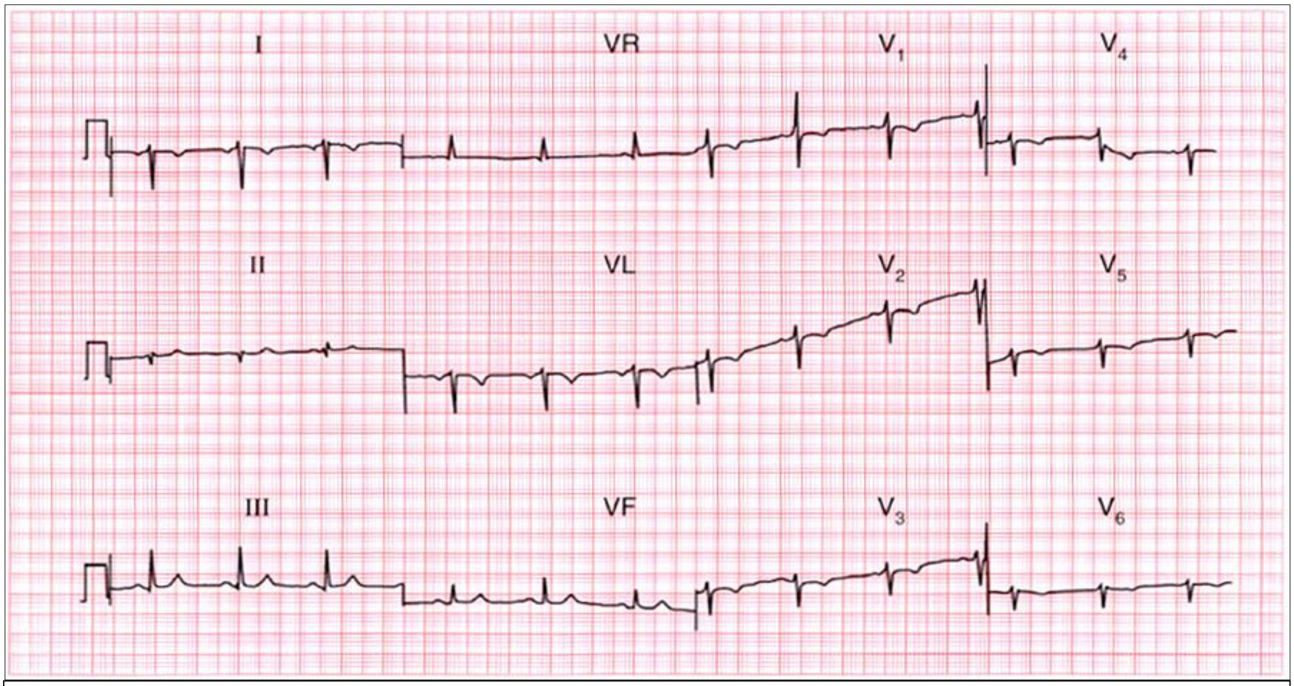


FIGURE 1.11 A case of dextrocardia. Look at aVR which is positive, P wave in V_1 which is inverted, and lack of normal chest leads progression.

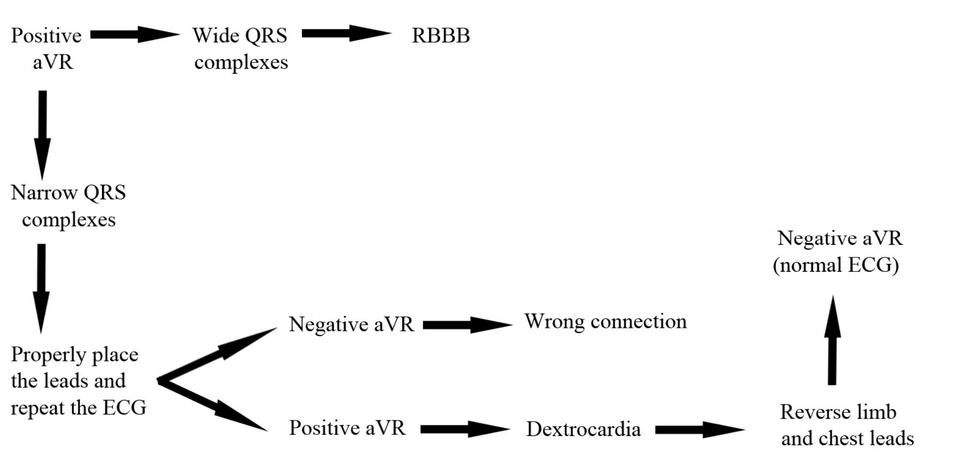


FIGURE 1.12 Approach to a positive aVR.

STEP 02 THE RHYTHM

When an EKG is described, the rhythm could be either regular or irregular depending on the R-R interval (FIGURE 1.13). Causes of irregular rhythm are shown in BOX 1.2.

BOX 1.2 Causes of irregular rhythm

Sinus arrhythmia	Atrial fibrillation
Frequent ectopic beats	Atrial flutter with variable response
Frequent escape beats	Second degree heart block with variable response

Infrequent escape beats and extra systoles do not disorganize the EKG pattern therefore the only cause for completely irregular rhythm is atrial fibrillation (AF). Extreme form of sinus arrhythmias and atrial rhythm can cause irregular rhythm, however both of these conditions have P wave while in AF there is no P wave.

FIGURE 1.13 Look at the regularity of the RR interval in the first example and the irregularity of RR interval in the second example.

ARRYTHMIAS

When the depolarization is begun at atrial muscle (atrial rhythm), the region around atrioventricular node (nodal or junctional rhythm) or ventricular muscle (ventricular rhythm), an arrhythmia is said to be present (FIGURE 1.14). In supraventricular rhythm, depolarization spreads normally into the ventricles, whether it has begun in the sinoatrial node, atrial muscle or the AV node. The QRS complex is thus normally shaped. Ventricular repolarization is also normal so the T wave is of a normal shape (FIGURE 1.14).
In ventricular rhythm, depolarization spreads relatively slowly through the ventricular muscle, so the QRS complex is wide and abnormal. Repolarization is also abnormal so the T wave is often inverted (FIGURE 1.14). When the activation of the atria or ventricles is totally disorganized, fibrillation is said to occur.

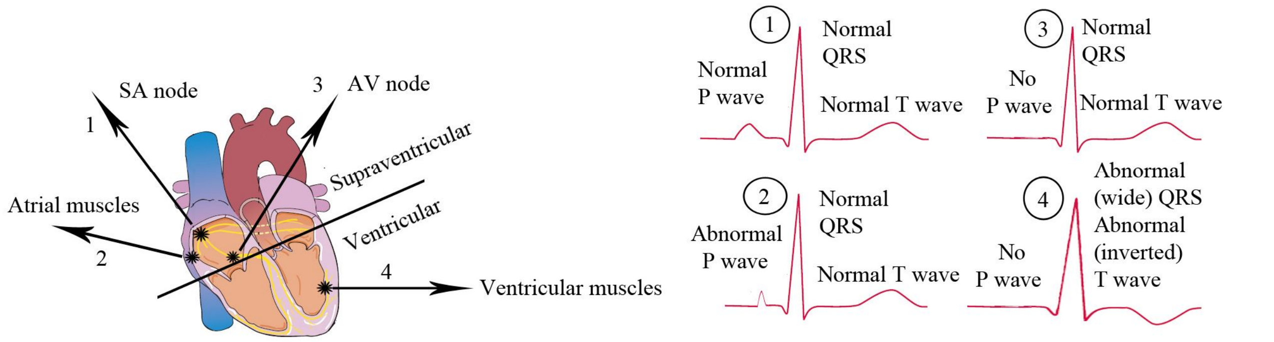


FIGURE 1.14 Origin and configuration of cardiac beats.

Arrhythmia can occur singly earlier than it should and is called single early beat (extra-systole, ectopic beat, or premature beat), or singly later than it should and is called late beat, or as sustained early beats (tachycardia or tachyarrythmia), or as sustained late beats (escape rhythm). Thus arrhythmia includes:

I. SINGLE EARLY BEAT (ECTOPIC BEAT)

Any part of the heart can be depolarized earlier than it should and the accompanying heart beat is called extrasystoles or ectopic beat or premature contraction. Ectopic beats can be classified according to their origin into supraventricular (atrial or junctional) or ventricular (FIGURE 1.14). As mentioned above supraventricular extra-systoles have normally shaped QRS and T waves, but ventricular extrasystoles have abnormally shaped QRS complex and T waves. Atrial ectopic beat had abnormal P wave; junctional (mid-junctional) ectopic beat had no P wave (it is usually buried inside the QRS and is difficult to be seen). The QRS complex and the T wave are the same of those of the sinus rhythm. Ventricular ectopic beat had no P wave, abnormal QRS complex and abnormally shaped T wave. The difference thus between supraventricular and ventricular beats is simply realized by the shape of that beat.

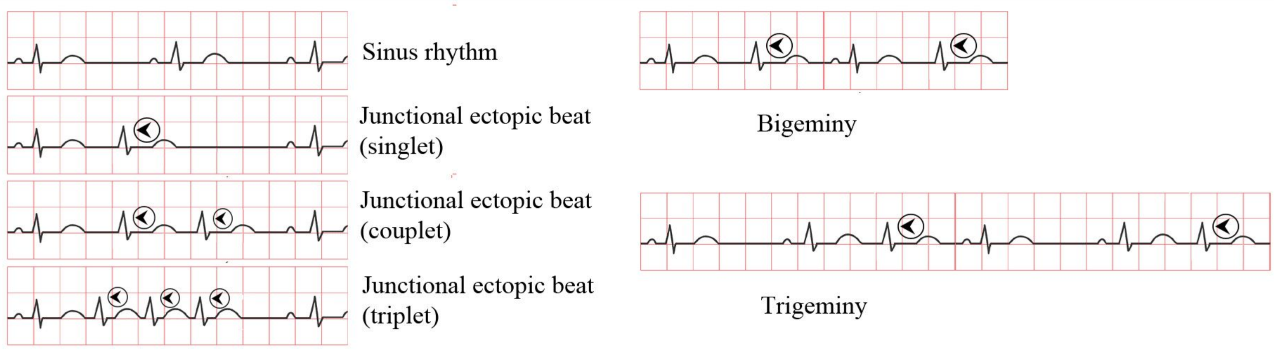
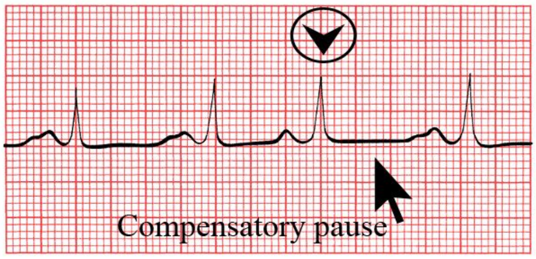
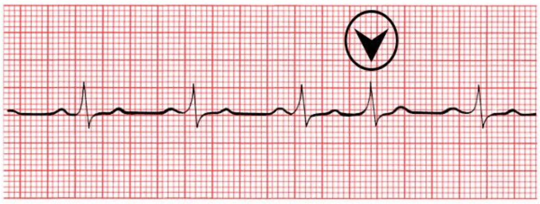


FIGURE 1.15 Nomenclature of ectopic and escape beats.

These ectopic beats can be unifocal that is identical in morphology, arising from a single ectopic focus or multifocal that is of varying morphology arising from multiple foci. When two successive ectopic beats had been arisen, they called couplet ectopic beats (FIGURE 1.15) and called triplet ectopic beats if they are three. When more than three successive ectopic beats are present, the resultant electrical activity is called rhythm. Moreover bigeminy means alternate ectopic and sinus beat while trigeminy means alternate ectopic and two sinus beats. The same principle can be applied for quadrigeminy. R on T phenomenon means occurrence of an ectopic beat at the peak of T wave of the preceding beat (FIGURE 1.16).



The third beat is an atrial premature beat. The P wave is shaped differently from other and the beat is clearly premature. The beat comes early (premature) and is followed by a compensatory pause.

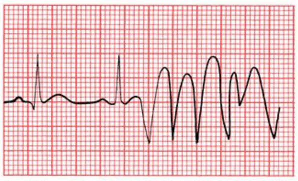


The fourth beat is a junctional premature beat. These is no P wave preceding the premature QRS.

The third beat is a premature ventricular beat. This beat has no P wave, wide QRS, and abnormal T wave.

Ventricular bigeminy. The ectopic beat comes alternatingly after each sinus beat.

The first and the fourth beats are sinus in origin. The other three beats are ventricular ectopic beats. They are variable in shape i.e. multifocal

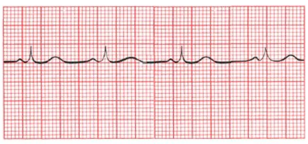


A ventricular premature beat falls on the preceding T wave (R on T phenomenon) and initiates a run of ventricular tachycardia

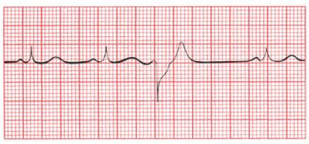
FIGURE 1.16 Examples of ectopic beat.

II. SINGLE LATE BEAT (ESCAPE BEAT)

The EKG appearance of an escape beat arising in either the atrial muscle, the nodal or junctional region, or the ventricular muscle, is the same as that of the corresponding extra-systole. The difference is that ectopic beat comes early (than expected next sinus beat) and escape beat comes late (than expected next sinus beat). This is shown in FIGURE 1.17.



Sinus rhythm. Normal shaped P wave is followed by normal (narrow) QRS and normal up-right T wave. The R-R interval is regular.

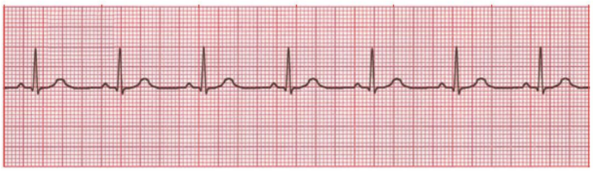


The third beat is a premature ventricular beat that occurs before the next normal beat

The third beat occurs late, after a prolonged pause. This is called ventricular escape beat.

FIGURE 1.17 An example showing the difference between the ectopic beat and the escape beat. The shape is similar but the time of occurrence is different.

TRY TO IDENTIFY THE CHARACTERS OF THE FOLLOWIGN BEATS



What is the underlying rhythm?
Is there any abnormality?
Is it regular or irregular rhythm?

What do we call these beats (arrows)?
Are they ectopic or escape beats?
Are they unifocal or multifocal?

What do we call these beats (arrows)?
Are they ectopic or escape beats?

What do we call these beats (arrows)?
Are they ectopic or escape beats?
Are they unifocal or multifocal?

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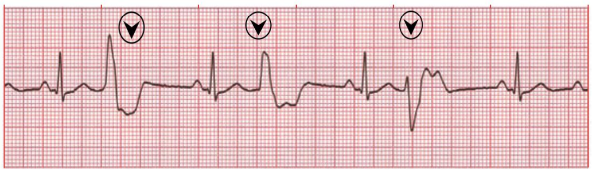

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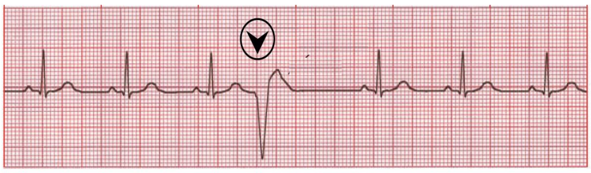
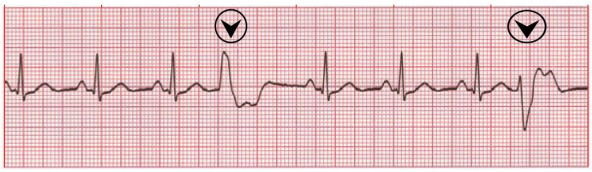
What do we call these beats (arrows)?
Are they ectopic or escape beats?

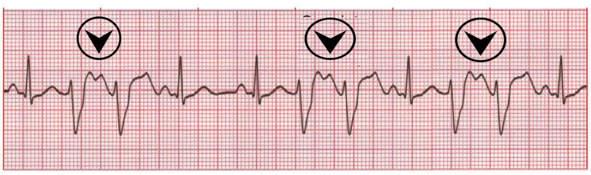
What do we call these beats (arrows)?
Are they ectopic or escape beats?
Are they unifocal or multifocal?
Are they bigeminy, trigeminy, or quadrigeminy?



What do we call this beat (arrow)?
Is it ectopic or escape beat?
Describe the P wave, QRS complex, and the T wave of this beat.



What do we call these beats (arrows)?
Are they ectopic or escape beats?
Are they unifocal or multifocal?
Are they bigeminy, trigeminy, or quadrigeminy?



What do we call these beats (arrows)?
Are they ectopic or escape beats?
Are they couplets or triplets?

III. SUSTAINED EARLY BEATS (TACHYCARDIA/TACHYARRHYTHMIA)

Tachyarrhythmia means sustained repetitive premature beats that can be supraventricular or ventricular. Foci in the atria, the junctional (AV nodal) region, and ventricles may fire repeatedly, causing a sustained tachycardia. The criteria already described for ectopic or escape beats can be used to decide the origin of the arrhythmia. Sustained tachycardia can be classified into two type according to the origin of the beats; supraventricular and ventricular tachycardia. The mechanism is illustrated in FIGURE 1.18.

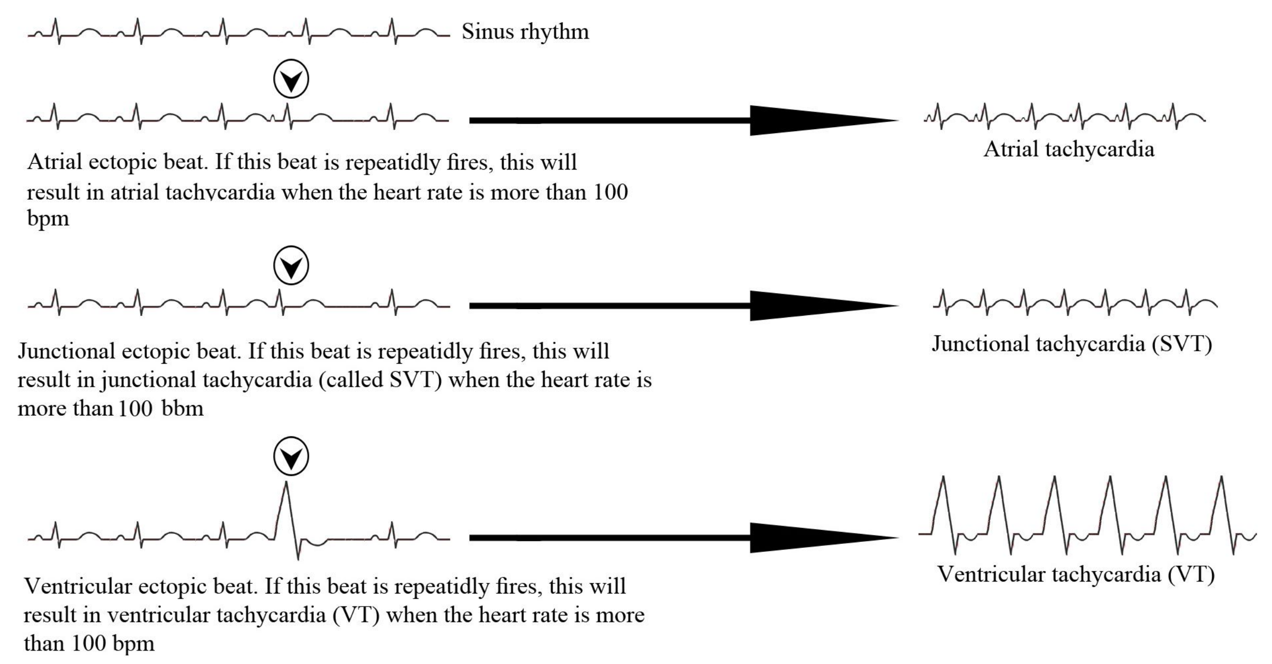


FIGURE 1.18 Mechanism of tachyarrhythmia.

1 SUPREVENTRICULAR TACHYCARDIA (NARROW COMPLEX TACHYCARDIA)

The rhythm here is originated from a region above the ventricles and therefore called supraventricular. This tachycardia includes sinus tachycardia, atrial tachycardia, atrial flutter, atrial fibrillation, and junctional (AV nodal) tachycardia. Because QRS complexes are normal in all these types, the term narrow complex tachycardia is sometimes used. Sinus tachycardia (FIGURE 1.19) is characterized by normally shaped P wave, QRS complex, T wave and regular RR interval as well as normal PR interval.

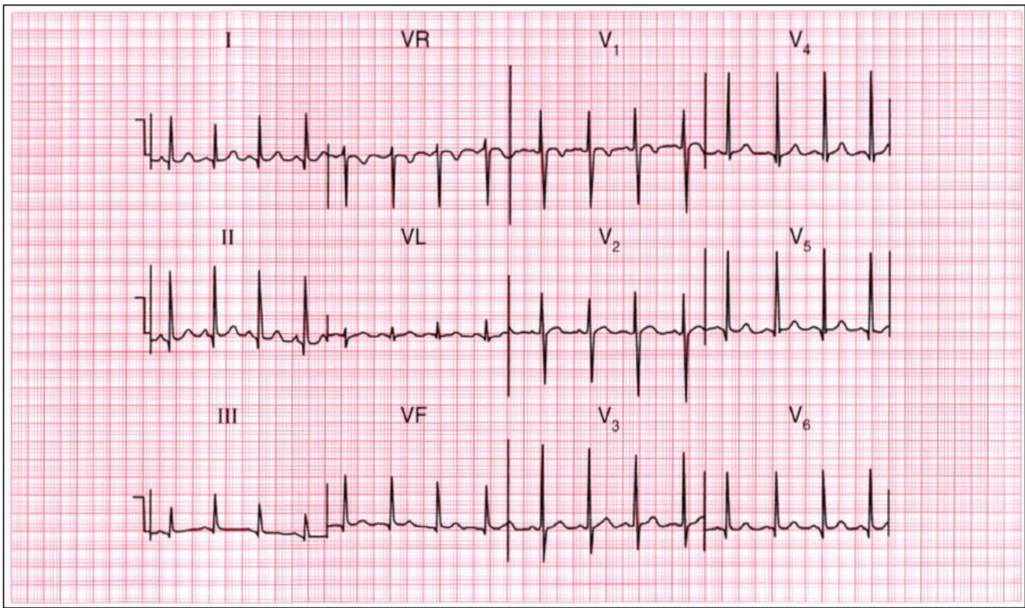


FIGURE 1.19 Sinus tachycardia. Look at the RR interval which is regular and the normally shaped P wave, QRS complexes, and the T wave.

The frequency of depolarization of the SA node is affected by the vagus nerve and inspiration causes its rate to increase and expiration causes its rate to decrease. The normal heart thus is little irregular and the term sinus arrhythmia is applied (FIGURE 1.20). This vagal effect diminishes with increasing age and lost in autonomic neuropathy.

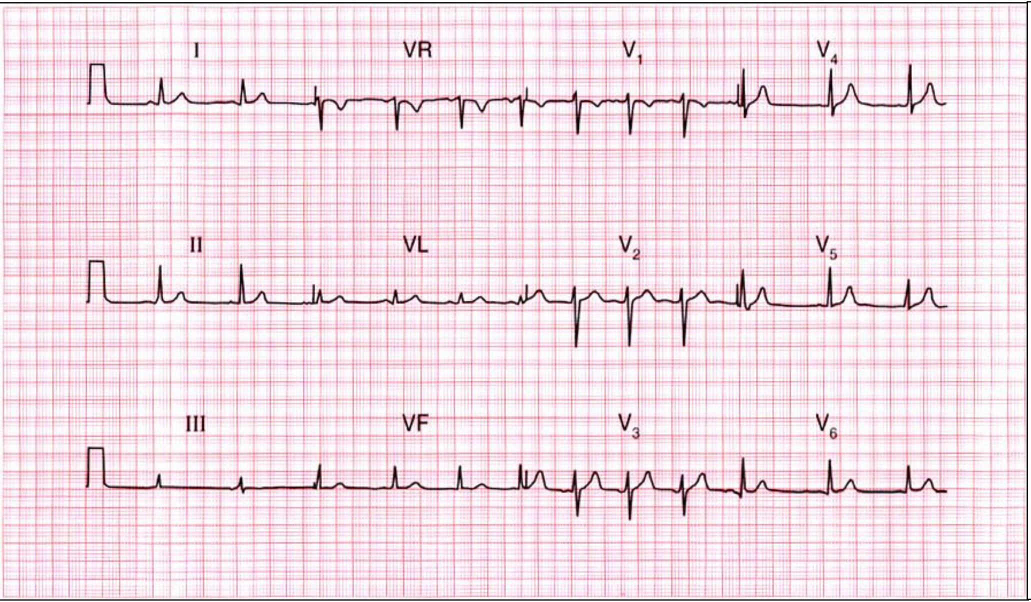


FIGURE 1.20 Sinus arrhythmia. Look at the RR interval which is irregular and the normally shaped P wave, QRS complexes, and the T wave.

In contrast to sinus rhythm, sinus arrhythmia had markedly variable RR interval, but the shape of the P wave, QRS complexes, and T wave is normal. The duration of the PR intervals are constant in both (FIGURE 1.21).

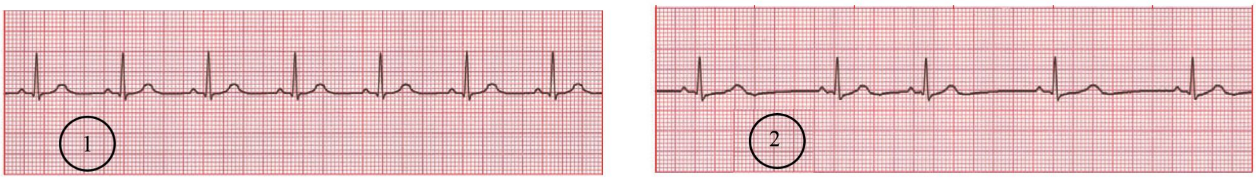


FIGURE 1.21 Look at the regular RR interval in the first example (sinus rhythm) and also look at the second example that had irregular RR interval (sinus arrhythmia).

Atrial tachycardia (FIGURE 1.22) is characterised by abnormally shaped P waves, normal shapes of the QRS complexes and the T waves.

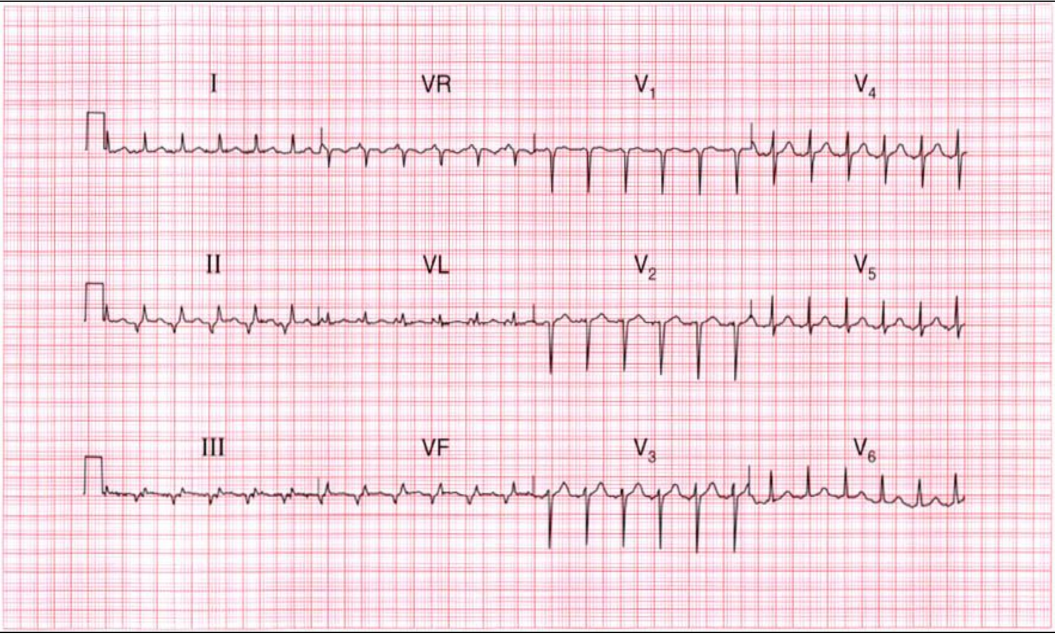


FIGURE 1.22 Atrial tachycardia. Look at the abnormally shaped P wave, normal QRS complexes, and normal T wave.

Any tachycardia arising above the AV junction that has a P wave configuration different from sinus rhythm is called atrial tachycardia. In general, impulses arising in the superior portion of the right or left atrium produce a positive P wave in the inferior leads (i.e., leads II, III, and aVF), whereas impulses arising in the lower or inferior portions result in negative P waves in the same leads. When the P wave configuration is uniform from beat to beat, the tachycardia is unifocal atrial tachycardia (FIGURE 1.22). Multifocal atrial tachycardia (MAT) is characterized by at least three distinct P wave morphologies and often at least three different PR intervals (FIGURE 1.23). Heart rate in atrial tachycardia is typically between 100-150 beats/min.

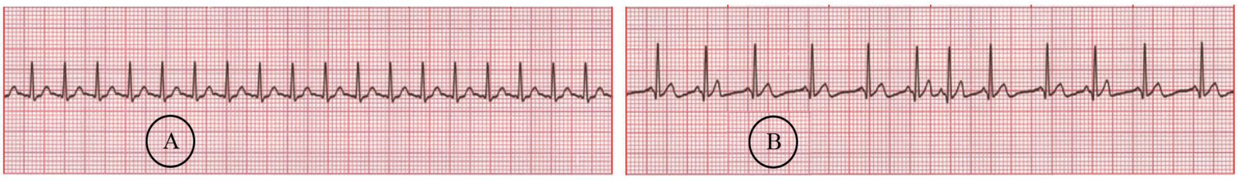


FIGURE 1.23 The differences between unifocal atrial tachycardia (A) and multifocal atrial tachycardia (B) is the presence of different shaped P waves (> 3) and different PR intervals. In both the P wave shape is different from that of sinus rhythm.

In junctional tachycardia (FIGURE 1.24), the atria contract faster than 120 beats per minute. It is characterized by absence of P waves, normal QRS complexes and T waves. This type of arrhythmia is called supraventricular tachycardia (SVT), or junctional tachycardia, or nodal tachycardia, or more recently AV nodal re-entry tachycardia (AVNRT).

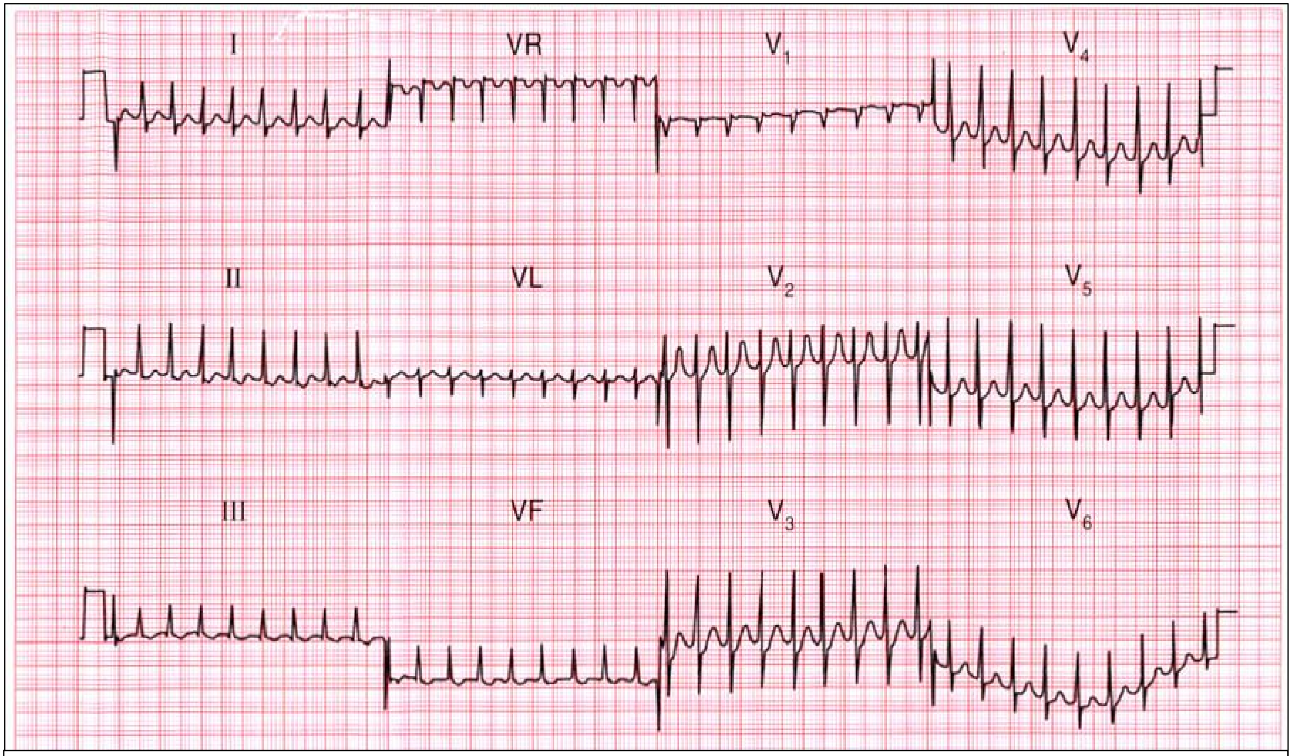


FIGURE 1.24 Junctional tachycardia (SVT) is characterised by absence of P wave and normal QRS complexes and T waves.

In junctional tachycardia, the P waves are frequently absent because they are buried in the preceding QRS complexes or the T waves. Some times however, the P wave may be seen within the QRS (FIGURE 1.25).

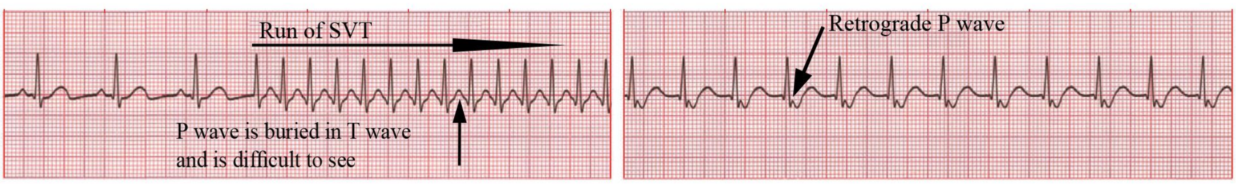


FIGURE 1.25 Two examples of junctional tachycardia (SVT). In the first example the P wave can not be seen and this is the usual clinical setting while in the second example, the P wave is seen after the QRS i.e. retrograde conduction.

Junctional tachycardia is termed high–junctional tachycardia when there are P waves that appear immediately before each QRS complexes and termed mid–junctional when these P waves appear within the QRS complexes and termed low–junctional tachycardia if these P waves appear after the QRS complexes as in FIGURE 1.25.

In atrial flutter (FIGURE 1.26), the atrial rate is greater than 250 beats per minute; the P waves form continuous saw-tooth line with normal QRS complexes and T waves.

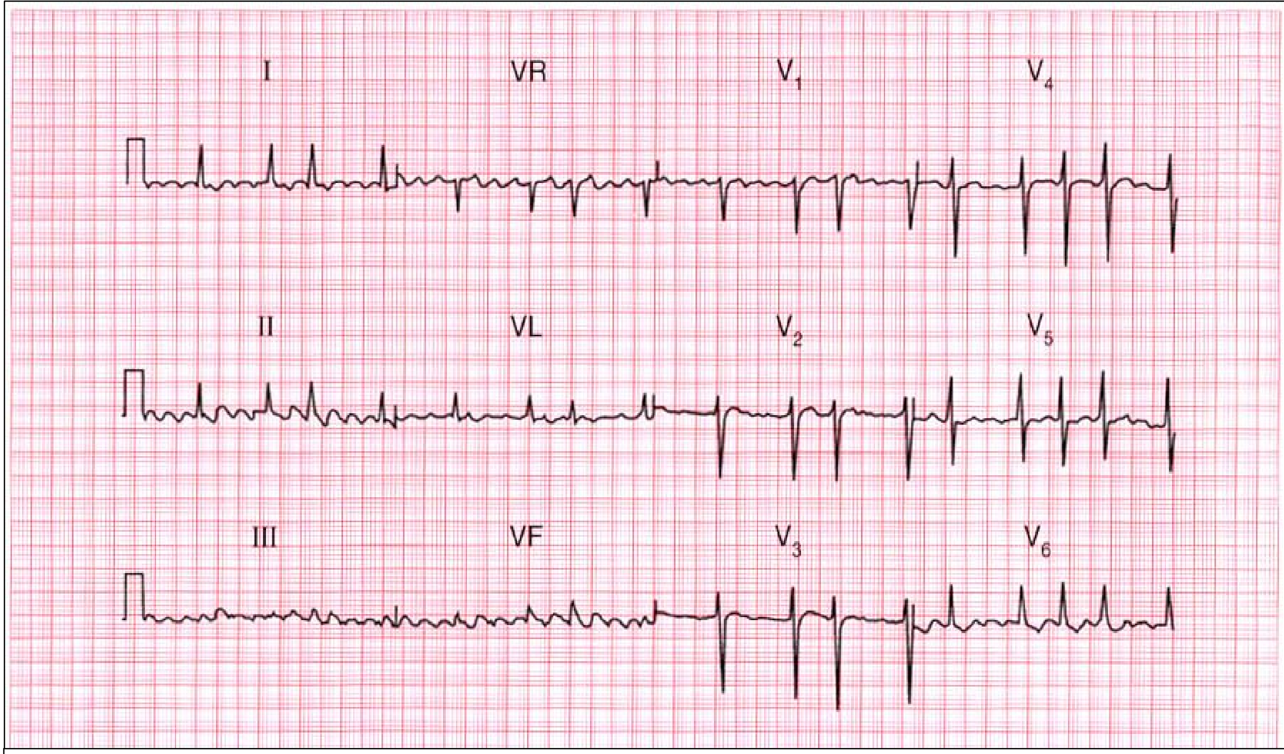
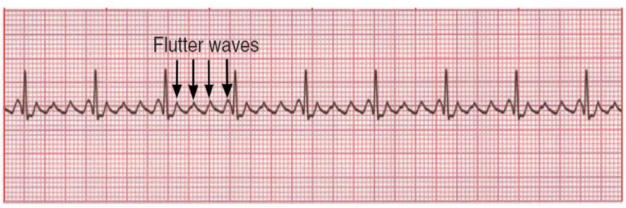


FIGURE 1.26 Atrial flutter with variable response i.e. the relation between F wave and QRS complex is variable.

Atrial flutter circuit rotates in a clockwise or counterclockwise direction in the right atrium around the tricuspid valve annulus. Common atrial flutter (counterclockwise 80%), with a negative “saw-tooth” appearance in leads II, III, and aVF and positive in V_1, has a fairly uniform route of impulse propagation localized to the right atrium. Incidental left atrial activation produces a negative saw-tooth flutter wave in the inferior leads. A reverse of this direction in the circuit could cause a positive flutter wave in the same leads (uncommon or clockwise). As the AV node usually fails to conduct all the P waves, the relationship between P waves and QRS complexes is usually 1:1, 2:1, 3:1, or 4:1 (FIGURE 1.27); these P waves are called flutter waves. If the Ventricular rate is rapid and P waves can not be seen, carotid sinus pressure will usually increase the block in the AV node and make the saw tooth more obvious (FIGURE 1.28).



The relationship between the P waves (flutter waves) and the QRS complexes in this example is 4:1

FIGURE 1.27 Atrial flutter with fixed response 4:1. Look at the regular rhythm, flutter waves, and normal shape of QRS and T waves.

Carotid sinus massage produced temporary AV block and revealed the flutter waves

FIGURE 1.28 Atrial flutter becomes more apparent with carotid sinus massage.

In atrial fibrillation (FIGURE 1.29), the atrial muscle fibers independently contract (fibrillating) to produce no P wave and totally irregular base line. In atrial fibrillation, in contrast to atrial flutter, the QRS complexes are totally irregular. At certain times, atrial activity may become sufficiently synchronized to produce flutter waves pattern. This finding is called atrial flutter-fibrillation which behaves likes atrial fibrillation. Again carotid sinus pressure will usually make the saw-tooth appearance more obvious. Carotid sinus pressure may affect some of supraventricular tachycardia as shown in BOX 1.3.

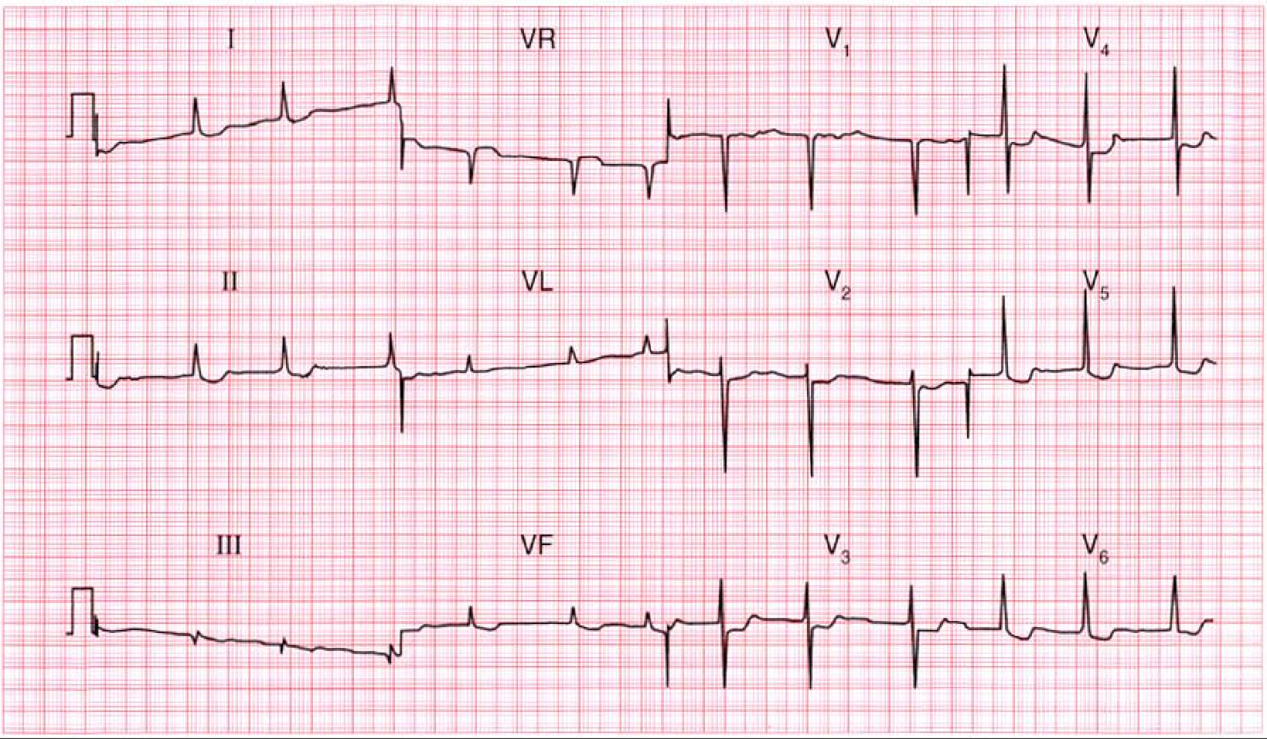
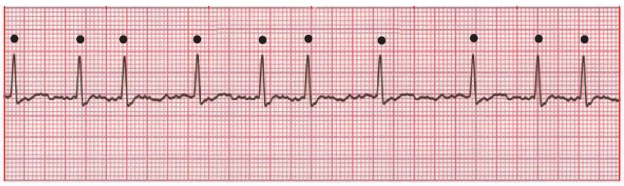


FIGURE 1.29 Atrial fibrillation. Look at the base line which lacks the P waves and at the rhythm which is irregular.

BOX 1.3 Effect of carotid sinus pressure on arrhythmia

TYPES OF ARRHYTHMIAS	EFFECTS OF CAROTIS SINUS PRESSURE
Sinus tachycardia	Slows
Atrial tachycardia	Abolished or no effect
Junctional tachycardia	Abolished or no effect
Atrial flutter	Increased AV block and reveal the flutter wave
Atrial fibrillation	No effect

Atrial fibrillation (AF) is described as rapid AF when the heart rate is fast or as slow AF when the heart rate is slow. Moreover, AF is described as fine (FIGURE 1.29) or coarse (FIGURE 1.30).



Irregular RR interval with absence of P waves are features of atrial fibrillation. The base line shows coarse activity and therfore called coarse atrial fibrillation

FIGURE 1.30 An ECG showing coarse atrial fibrillation.

2 VENTRICULAR TACHYCARDIA (WIDE COMPLEX TACHYCARDIA)

The rhythm here is originated from the ventricles and therefore called ventricular. Because of the widening in the QRS complexes, this tachycardia is called wide complex tachycardia. It includes ventricular tachycardia (VT), Torsades des pointes (TdP), and ventricular fibrillation (VF). In ventricular tachycardia (FIGURE 1.31) there is no P wave, abnormal wide QRS complexes, and abnormal T wave (as with the corresponding ventricular ectopic beat). Ventricular tachycardia is called monomorphic when all the QRS complexes have the same appearance, and polymorphic when they vary (FIGURE 1.32). A twisting polymorphic ventricular tachycardia is called torsade des pointes. The EKG (FIGURE 1.33) shows rapid irregular complexes that oscillate from an up right to an inverted position and seem to twist around the baseline as the mean QRS complex axis changes. This arrhythmia is usually non-sustained, but may degenerate into ventricular fibrillation. When the ventricular muscle fibers contract independently, no QRS complexes can be identified and the EKG is totally disorganized (FIGURE 1.34). This type of tachycardia is called ventricular fibrillation. The need to recognize and treat ventricular fibrillation (VF) quickly is one of the main foundations on which the policy of acute coronary care unit is built. If you realized such changes in the EKG, look at the patient as he /she may have lost consciousness.

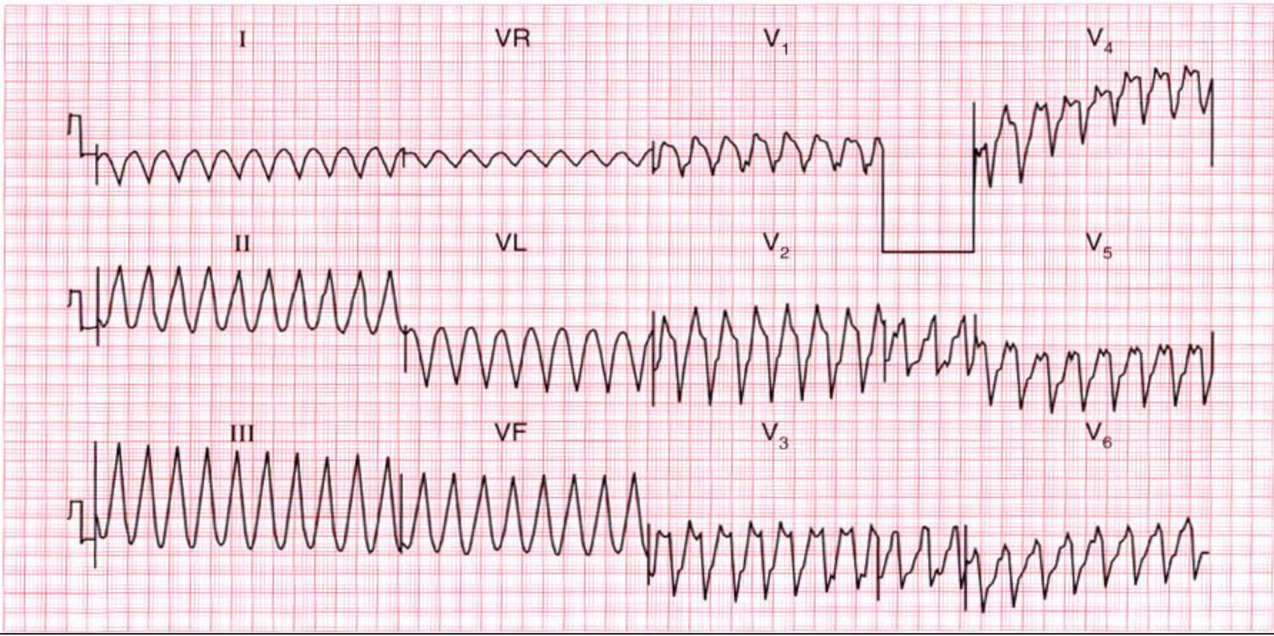


FIGURE 1.31 Ventricular tachycardia. Look at the regularity in the RR interval, absence of P wave, widening of the QRS complexes, and abnormality in T wave.

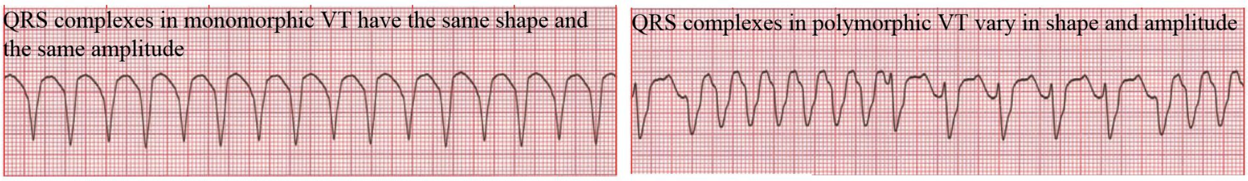
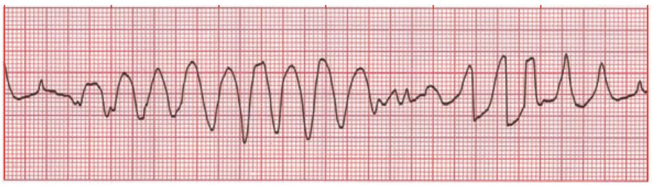


FIGURE 1.32 Ventricular tachycardia; monomorphic and polymorphic.



The french term of Torsade de Pointes means twisting of the points. This tachycardia had irregular rhythm and wide and bizarre QRS. The QRS reverses polarity and the strip shows a spindle effect.

FIGURE 1.33 Torsade de Pointes.

FIGURE 1.34 Ventricular fibrillation. Look at the end of the strip where VF started after an R on T phenomenon.

Ventricular tachycardia is easily recognized and differentiated from supraventricular tachycardia because VT had wide QRS complex (more than 3 mm) while SVT had normal (or narrow) QRS complexes. When the heart rate exceeds 180 bpm, there will be physiological widening in the QRS complexes (bundle branch block) or when the underlying basic EKG has wide QRS complexes, one should differentiate the rhythm whether it is SVT with block or VT. If you can not differentiate between the two, treat the patient as he/she is having ventricular tachycardia. The problem can be analyzed by looking at the following points:

01 Broad complex tachycardia occurring in the course of an acute myocardial infarction is almost always ventricular tachycardia

02 Response to the carotid sinus pressure or to intravenous adenosine infavor SVT with block

03 Presence of atrioventricular node dissociation is a pathognomonic feature for wide complex tachycardia to be ventricular tachycardia

04 The presence of one P wave per QRS complex suggest sinus rhythm and bundle branch block while if P waves are seen at a slower rate than QRS complex, the rhythm is ventricular

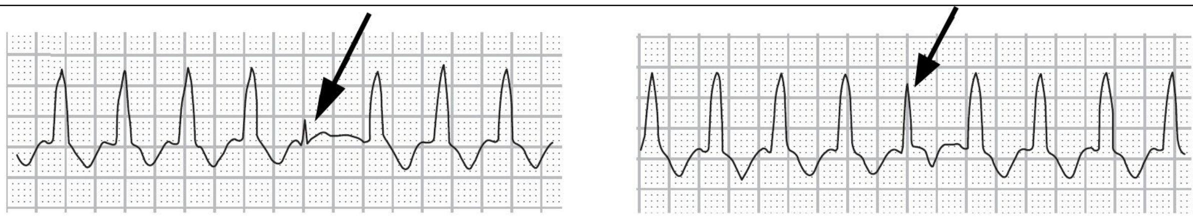
05 QRS complex duration if more than 160 ms (> 4 small squares), it is probably ventricular

06 QRS complex regularity. Ventricular tachycardia is usually regular; an irregularity means atrial fibrillation with block

07 QRS complex configuration. If the QRS complex in the chest leads all point either upward or downward (concordance), it is probably VT, but twisting QRS complexes indicate torsade de pointes

08 When the QRS complex show right bundle branch block (RBBB) pattern, abnormal conduction is more likely if the second R peak is higher than the first; VT is likely if the first R is higher

09 Presence of fusion and capture beats is a pathognomonic for ventricular tachycardia (FIGURE 1.35). In VT, there is independent atrial and ventricular activity. Occasionally a P wave is conducted to the ventricles through AV node. This may produce a normal sinus beat in the middle of the tachycardia (capture beat). However, more commonly the conducted impulse fuses with an impulse from the tachycardia (a fusion beat).



Capture beat (arrow) - a supraventricular impulse causes depolarization of the ventricles resulting in a narrow QRS complex

Fusion beat (arrow) - an intermediate width QRS complex resulting from a combination (or fusin) of supraventricular and ventricular impulses

FIGURE 1.35 Ventricular tachycardia; capture and fusion beats.

IV. SUSTAINED LATE BEATS (ESCAPE RHYTHM)

It is important not to try to suppress escape rhythm, because without it the heart might stop altogether. Normally the SA node controls the heart rate because it has the highest frequency of discharge, but if for any reason this fails, the region with the next highest intrinsic depolarization frequency will emerge as the pacemaker and set up an escape rhythm. The atria and the junctional region have automatic depolarization frequencies of about 50/min. If both the SA node and the nodal region fail to depolarize or if conduction to the ventricles fails, a ventricular focus may emerge, with a rate of 30-40/min. There are three escape rhythms (FIGURES 1.36, 1.37, and 1.38); atrial escape (idioatrial) rhythm, nodal or junctional (idionodal) rhythm, and ventricular (idioventricular) rhythm. The EKG appearance is the same for that of atrial, junctional, and ventricular tachycardia, but the difference is in the heart rate. Escape rhythms are not primary disorder, but are the response to problems higher in the conducing pathway. They are commonly seen in the acute phase of a heart attack. Atrial escape rhythm (FIGURE 1.39), also called idioatrial rhythm or wandering atrial pacemaker, has the same features of that of atrial tachycardia; the difference is in the heart rate. Junctional escape rhythm (FIGURE 1.39), also called idionodal rhythm, has the same features of that of junctional tachycardia; the difference is in the heart rate. It is usually associated with a rate of 40–60/minute and narrow QRS complexes. The heart rate may be enhanced and increased to 100/minute; in this stage it is called “accelerated junctional rhythm”. Ventricular escape rhythm (FIGURE 1.39), also called idioventricular rhythm, has the same features of that of ventricular tachycardia; the difference is in the heart rate. Ventricular escape rhythms are usually associated with rates 20–40/minute. The heart rate may be enhanced and increased to 100/minute; in this stage it is called “accelerated idioventricular rhythm”.

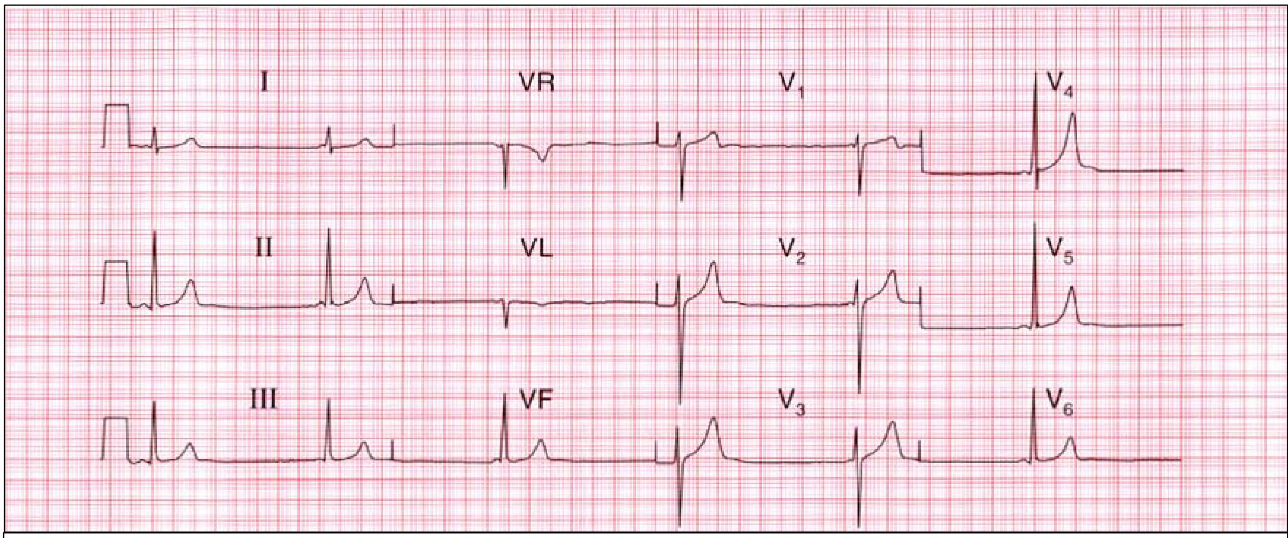


FIGURE 1.36 Idioatrial rhythm. It is also called wandering atrial pacemaker. The difference between atrial tachycardia and this rhythm is in the heart rate.

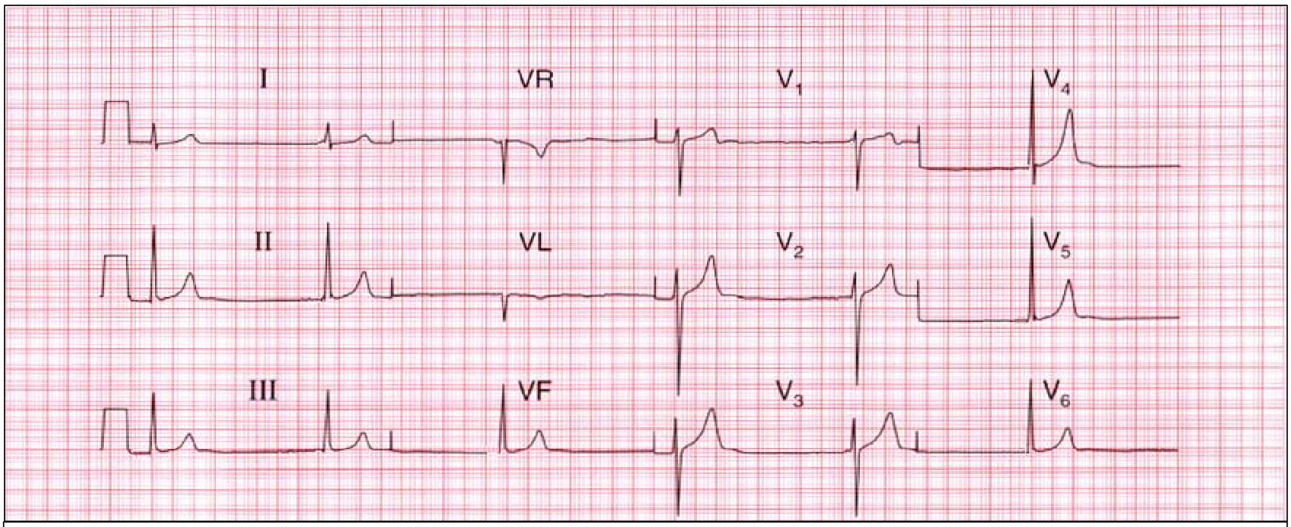


FIGURE 1.37 Idionodal rhythm. It is also called junctional escape rhythm. The difference between SVT and this rhythm is in the heart rate.

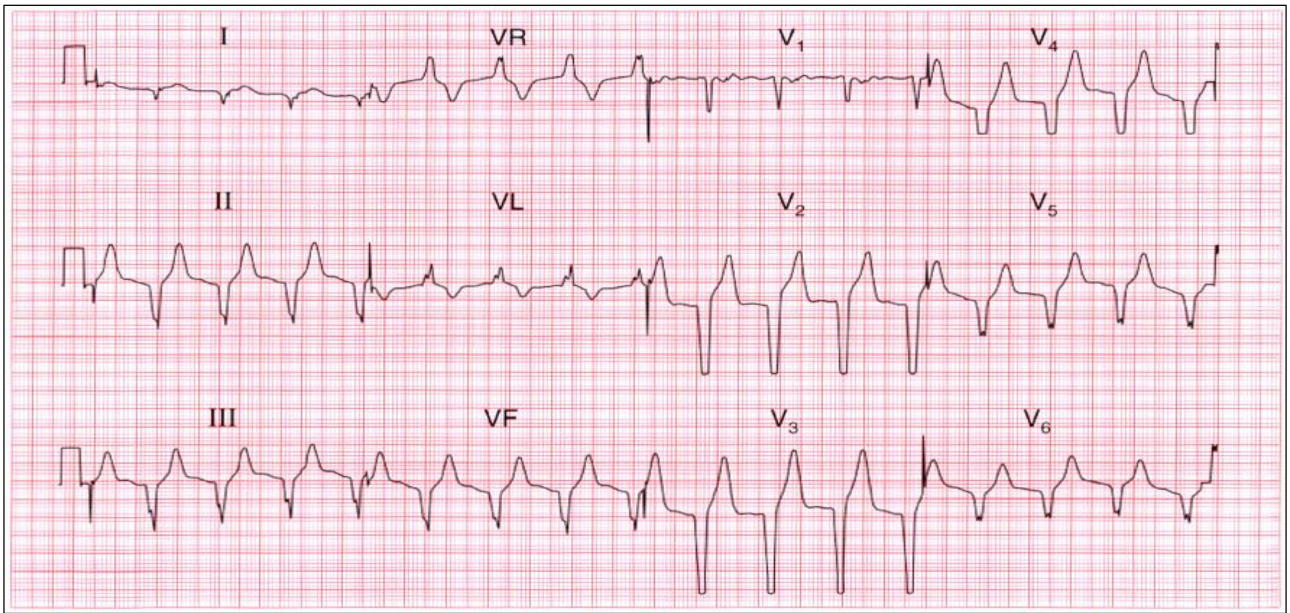


FIGURE 1.38 Idioventricular rhythm. It is also called ventricular escape rhythm. The difference between VT and this rhythm is in the heart rate.

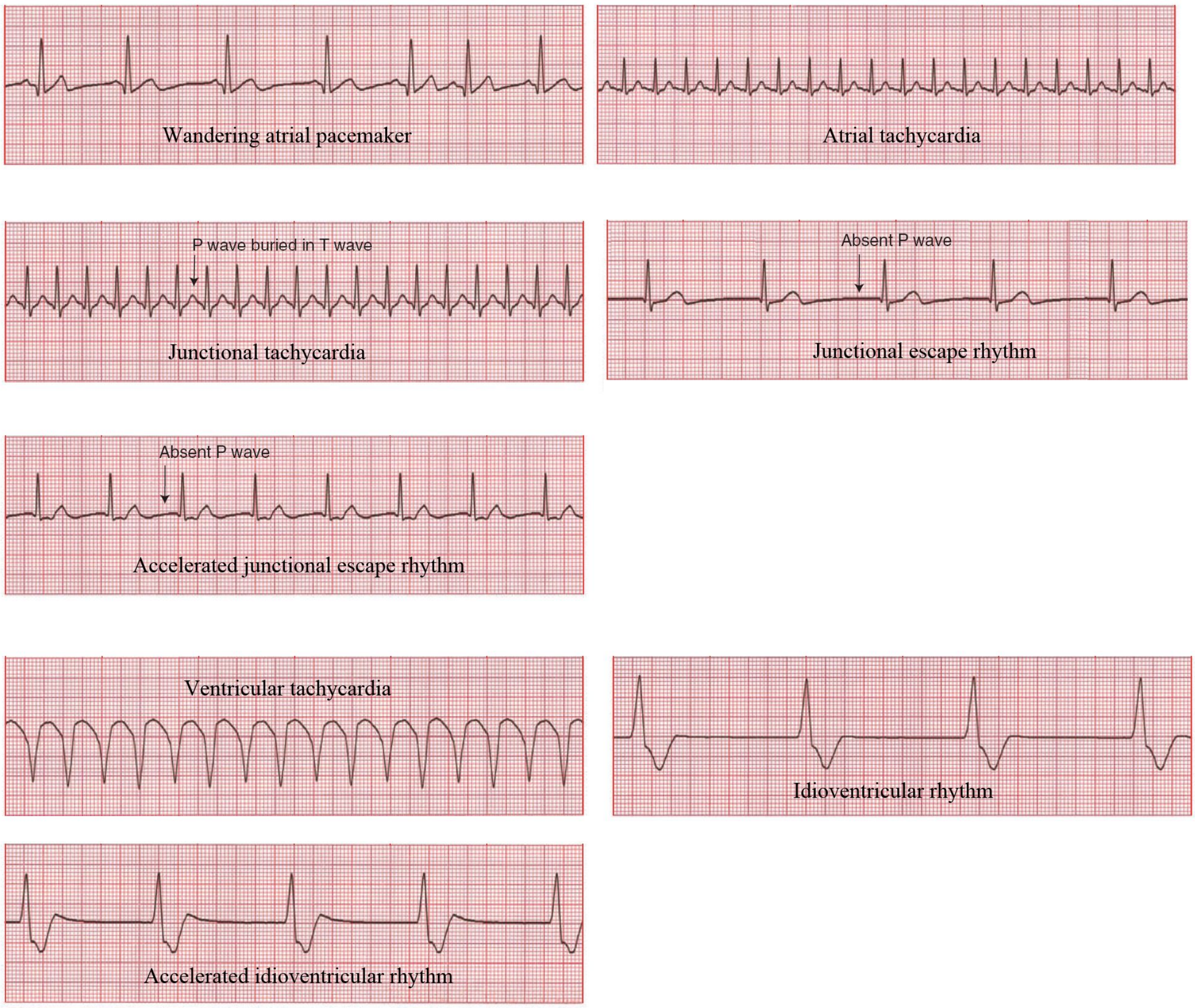
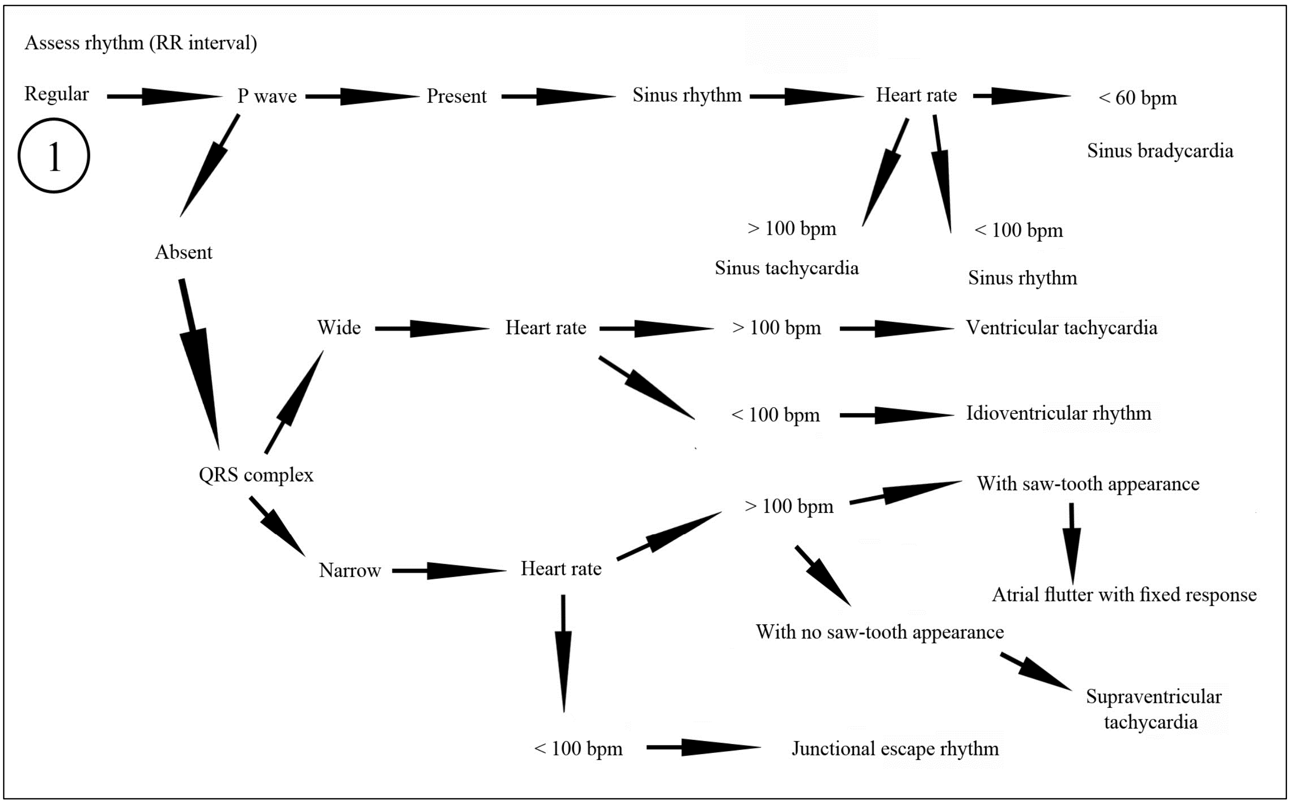
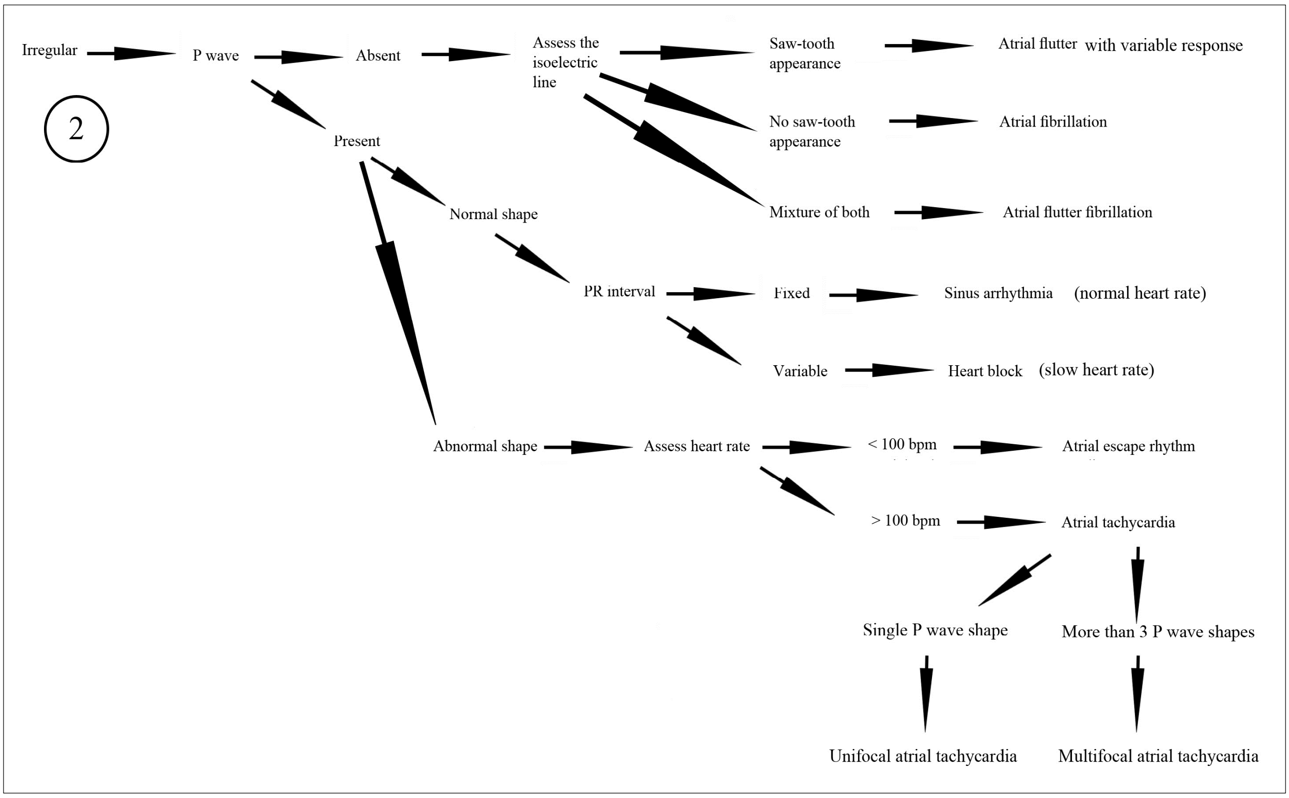


FIGURE 1.39 Tachycardia versus escape rhythm.

REVIEW OF ARRHYTHMIA

Narrow complex rhythm (in which the QRS complex is normal in width) includes sinus rhythm (and sinus arrhythmia), atrial rhythm (atrial tachycardia, atrial flutter, atrial fibrillation, atrial escape rhythm) and junctional rhythm (junctional tachycardia (SVT) and junctional escape rhythm). Wide complex rhythm (in which the QRS is more than three small squares) includes ventricular rhythm (ventricular tachycardia and Torsade de pointes) and narrow complex rhythm when the EKG is basically wide (e.g. bundle branch block). The following two approaches are recommended in assessing the heart rhythm:





STEP 03 HEART RATE

Heart rate can be estimated with regard to the regularity of the rhythm as follows:

I. Regular rhythm
Heart Rate = 300 ÷ number of large squares between beats. Or Heart Rate = 1500 ÷ number of small squares between beats.
II. Irregular rhythm
Heart Rate = number of R waves in 15 large X 20 squares

Heart rate above 100/minute is called tachycardia while below 60/minute is called bradycardia. If the rhythm is irregular heart rate is estimated (FIGURE 1.40) and the result should be given as a range. The commonest cause for irregularly irregular rhythm is atrial fibrillation.

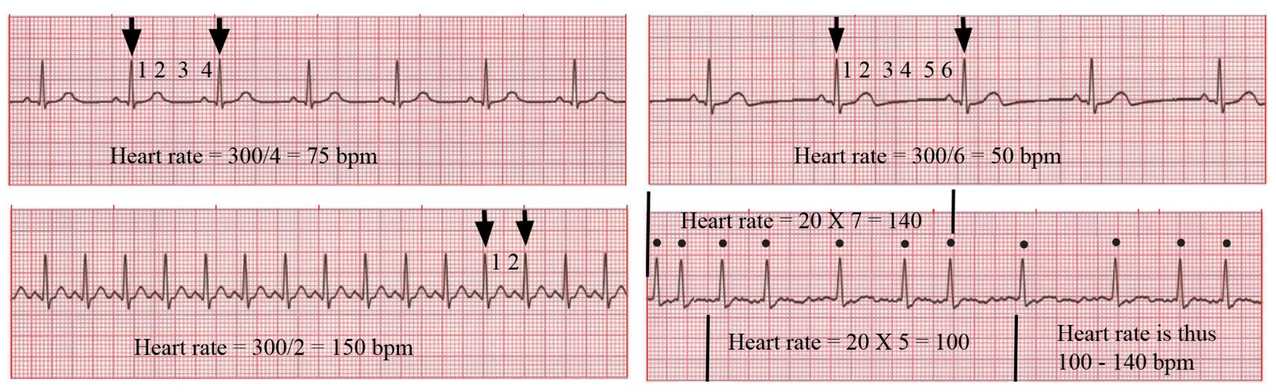


FIGURE 1.40 Heart rate estimation.

STEP 04 CARDIAC AXIS

The average deflection of depolarization waves in the ventricle spreads from 11 o'clock to 5 o'clock. It is therefore moving a way from aVR and towards other limb leads. This is called the cardiac axis (or cardiac vector) and in the normal heart the QRS complex is predominantly downward in aVR (that is, the S wave exceeds the R wave), but predominantly upward in the other limb leads (the R wave is bigger than any S wave that may be present). The direction of the QRS complex in leads I and aVF determines the cardiac axis as follows:

Lead I	Lead aVF	Interpretation
Positive	Positive	Normal
Positive	Negative	Left axis deviation
Negative	Positive	Right axis deviation
Negative	Negative	Extreme right axis deviation

Left axis deviation (FIGURE 1.41) is usually seen in short obese healthy individuals and right axis deviation (FIGURE 1.41) is usually seen normally in tall thin healthy individuals however other causes for left and right axis deviations (BOX 1.4) should also be sought.

BOX 1.4 Causes of axis deviation

LEFT AXIS DEVIATION	RIGHT AXIS DEVIATION
Normal in obese and short individuals	Normal in tall and thin individuals
Left ventricular hypertrophy	Right ventricular hypertrophy
Left anterior hemiblock	Left posterior hemi block (rare)
Anterior myocardial infarction	Anteriolateral myocardial infarction
Wolff-Parkinson-White syndrome (type A)	Wolff-Parkinson-White syndrome (type B)
Pregnancy	Pulmonary embolism