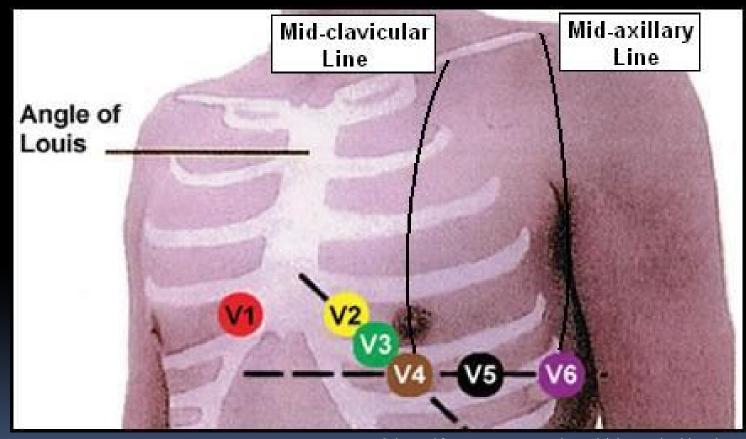
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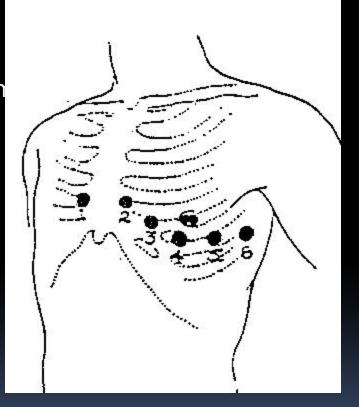
Precordial Leads



Adapted from: www.numed.co.uk/electrodepl.html

ad Placement

- = 4th intercostal space, right border of sternun
- = 4th intercostal space, left border of sternum
- = midway between V2 and V4
- = 5th intercostal space, midclavicular line
- = anteroaxillary line at level of V4
- = midaxillary line at level of V4 and V5 ctrocardiography



The ECG reading

- • Paper is in 1mm intervals (horizontal and
- vertical)
- Every 5mm the line is accentuated
- • **Speed of the record** = 25mm/sec
- 5mm distance = 0.2sec
- 1mm distance = 0.04sec
- 1 sec = 5 bold lines = 25mm=1 large box
- Calibration
- 1.0mV=10mm vertical deflection on the grid

Heart Rate (measurement strategies)

• When the HR is irregular – mark off a 6sec time period on the grid (30 heavy lines), count the number of QRS complexes in that interval and multiply by 10

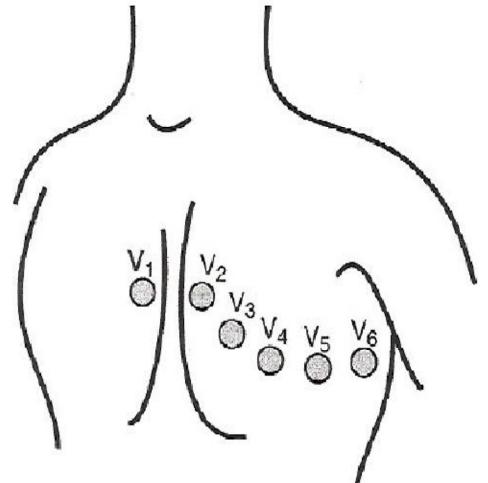
 When the HR is regular – measure the RR interval between two successive heart beats then
 divide this value into 1500 (there are 1500 mm in

1 minute)

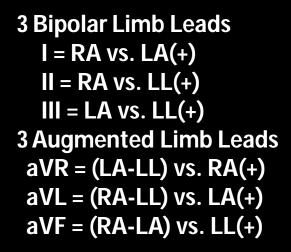
BPM = 1500 / RR interval (msec)

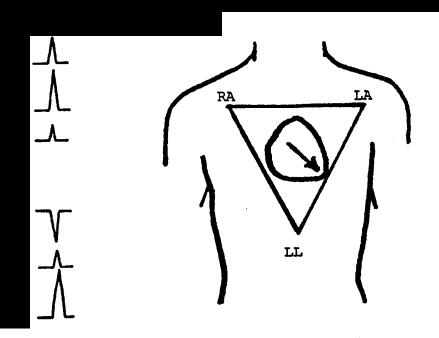
ad Placement

V1 – Right Sternal Border – 4th ICS
V2 – Left Sternal Border – 4th ICS
V3 Midway Between V2 and V4
V4 Midclavicular line – 5th ICS
V5 Anterior Axillary line – 5th I
V6 Mid axillary line – 5th ICS

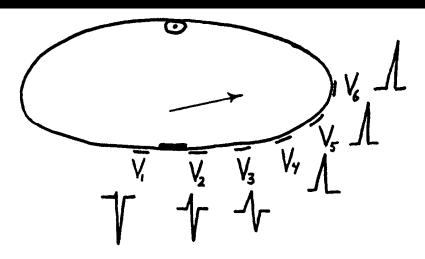


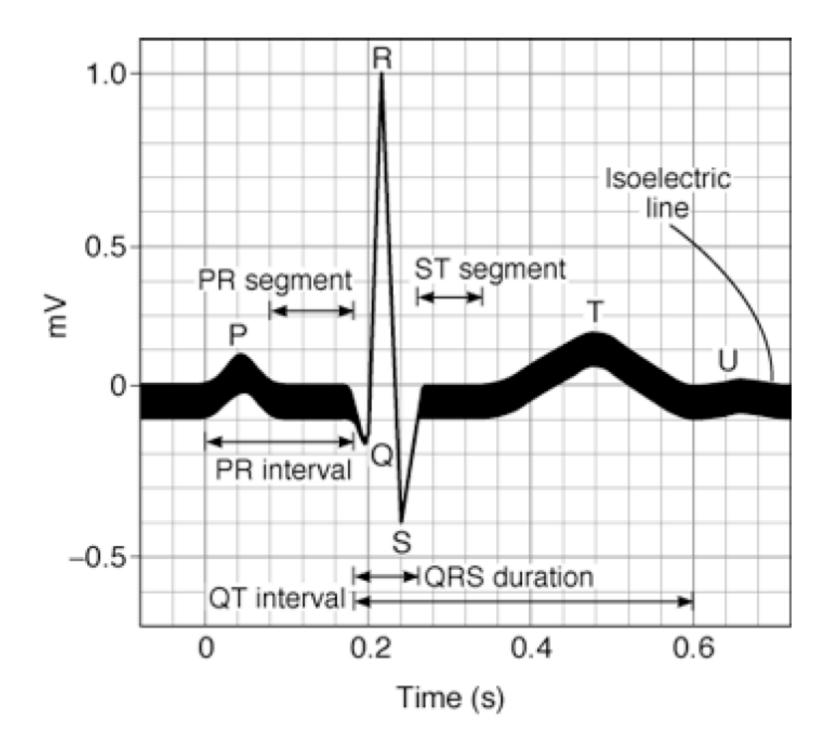
ECG Recordings: (QRS vector---leftward, inferiorly and posteriorly





6 Precordial (Chest) Leads: Indifferent electrode (RA-LA-LL) vs. chest lead moved from position V_1 through position V_6 .

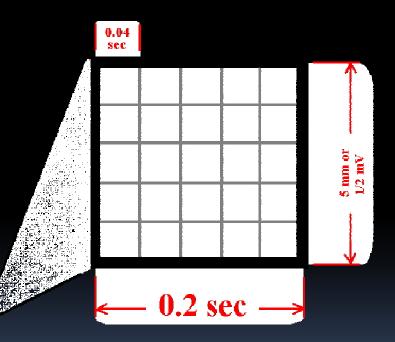


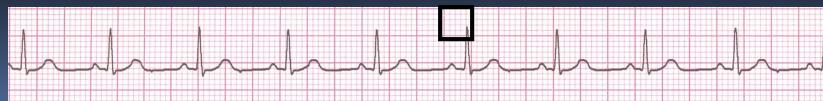


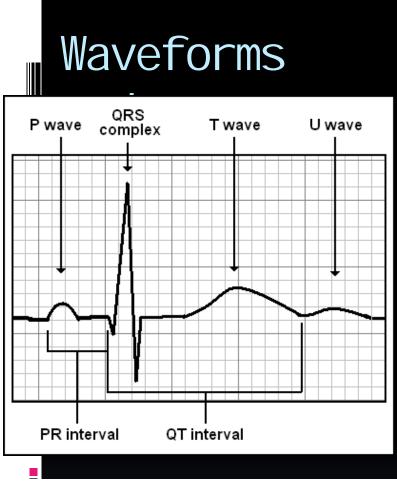
The ECG Paper

Horizontally

- One small box 0.04 s
- One large box 0.20 s
- Vertically
 - One large box 0.5 mV







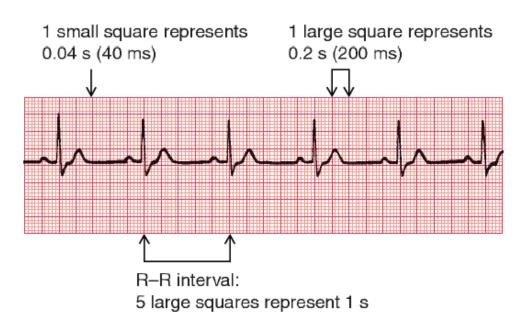


Fig. 1.4 Relationship between the squares on ECG paper and time. Here, there is one QRS complex per second, so the heart rate is 60 beats/min

Q waves

- Can occur normally in several leads
 - Normal Q waves called physiologic
- Physiologic Q waves
 - .04 sec (40ms)
- Pathologic Q
 - >.04 sec (40ms)

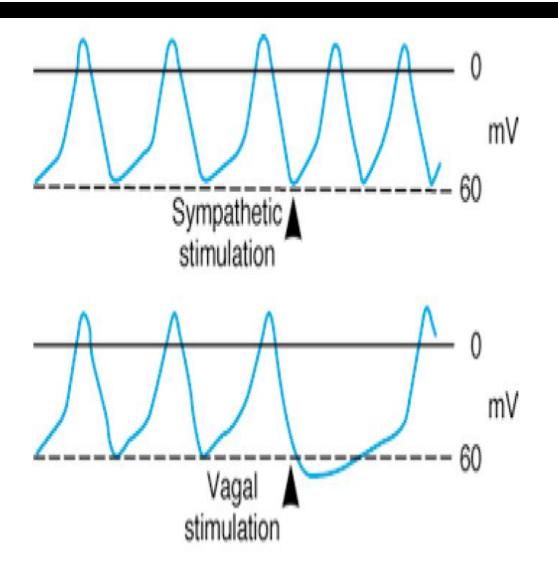


Figure 28-3. Effect of sympathetic (noradrenergic) and vagal (cholinergic) stimulation on the membrane potential of the SA node.

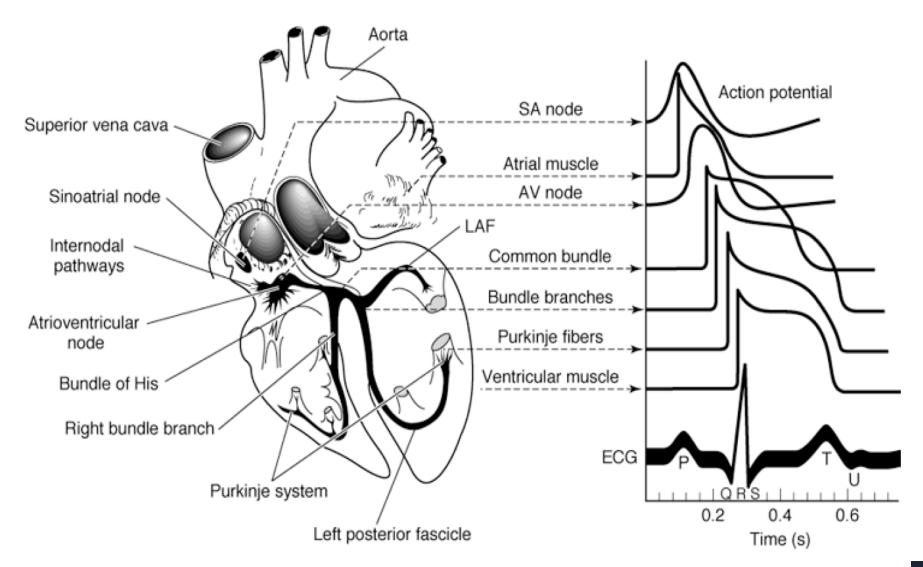
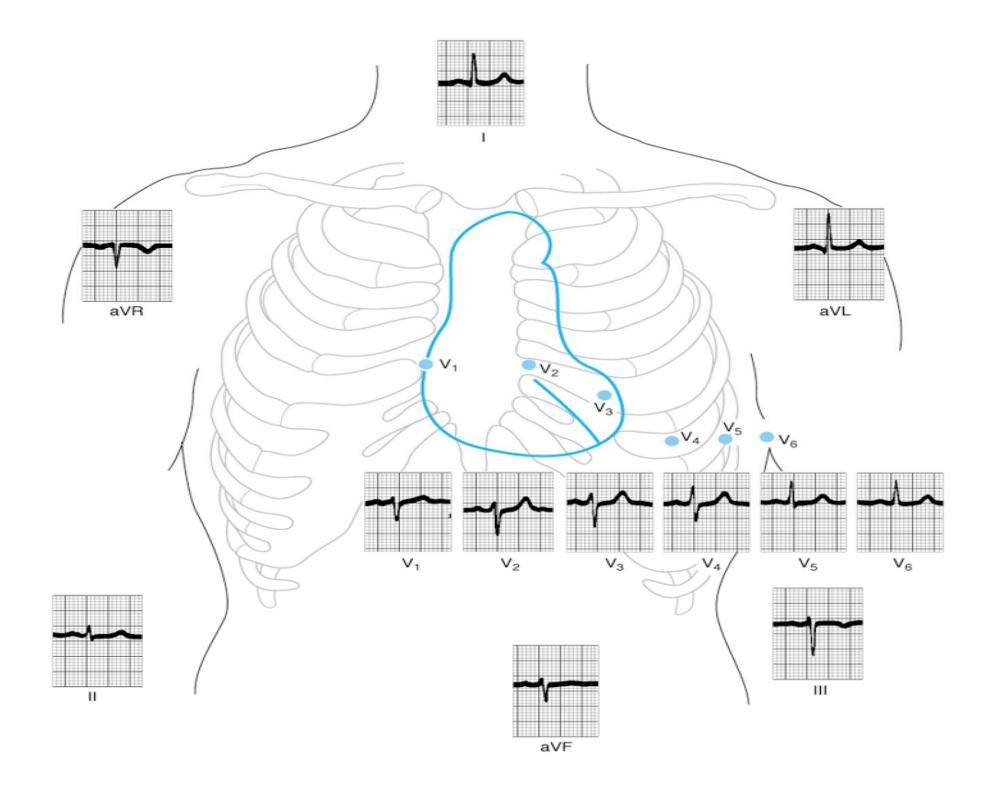


Figure 28-1. Conducting system of the heart. Typical transmembrane action potentials for the SA and AV nodes, other parts of the conduction system, and the atrial and ventricular muscles are shown along with the correlation to the extracellularly recorded electrical activity, ie, the electrocardiogram (ECG). The action potentials and ECG are plotted on the same time axis but with different zero points on the vertical scale. LAF, left anterior fascicle.



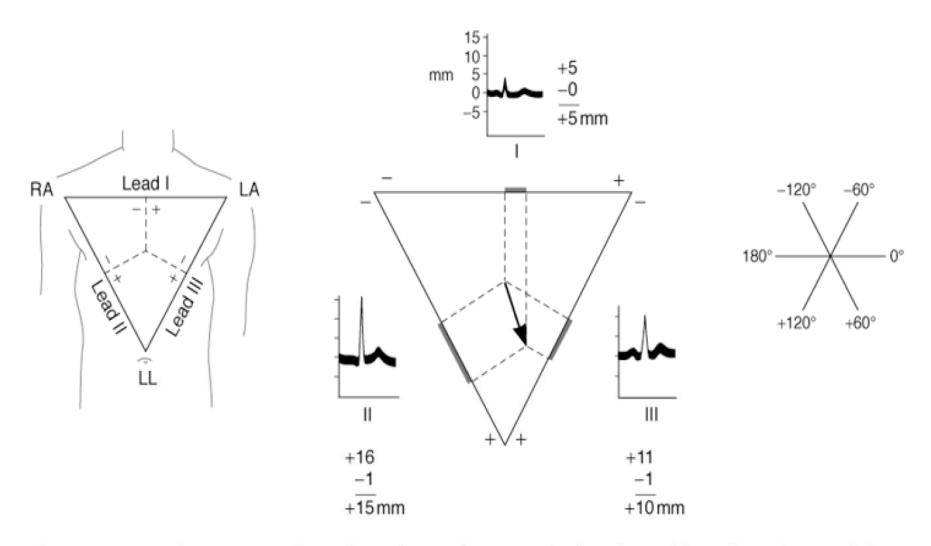


Figure 28-8. Cardiac vector. **Left:** Einthoven's triangle. Perpendiculars dropped from the midpoints of the sides of the equilateral triangle intersect at the center of electrical activity. RA, right arm; LA, left arm; LL, left leg. **Center:** Calculation of mean QRS vector. In each lead, distances equal to the height of the R wave minus the height of the largest negative deflection in the QRS complex are measured off from the midpoint of the side of the triangle representing that lead. An arrow drawn from the center of electrical activity to the point of intersection of perpendiculars extended from the distances measured off on the sides represents the magnitude and direction of the mean QRS vector. **Right:** Reference axes for determining the direction of the vector.

PR interval

< 0.12 s	0.12-0.20 s	> 0.20 s
High catecholamine states Wolff-Parkinson-White	Normal	AV nodal blocks

Wolff-Parkinson-White

1st Degree AV Block

QRS complex

NormalIncomplete bundle branch blockBundle branch blockVCVentricular rhythm	<u><</u> 0.10 s	0.10-0.12 s	> 0.12 s
	Normal		PVC



Incomplete bundle branch block

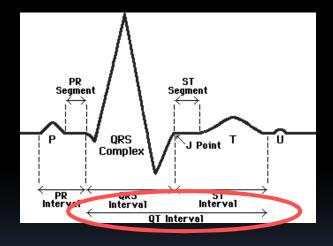
3rd degree AV block with ventricular escape rhythm

Remember: If you have a BBB determine if it is a right or left BBB. If you need a refresher see Module VI.

QT interval

The duration of the QT interval is proportionate to the heart rate.

The faster the heart beats, the faster the ventricles repolarize <u>so the shorter the</u> **QT interval.** Therefore what is a "normal" QT varies with the heart rate. For each heart rate you need to calculate an adjusted QT interval, called the "corrected QT" (QTc):



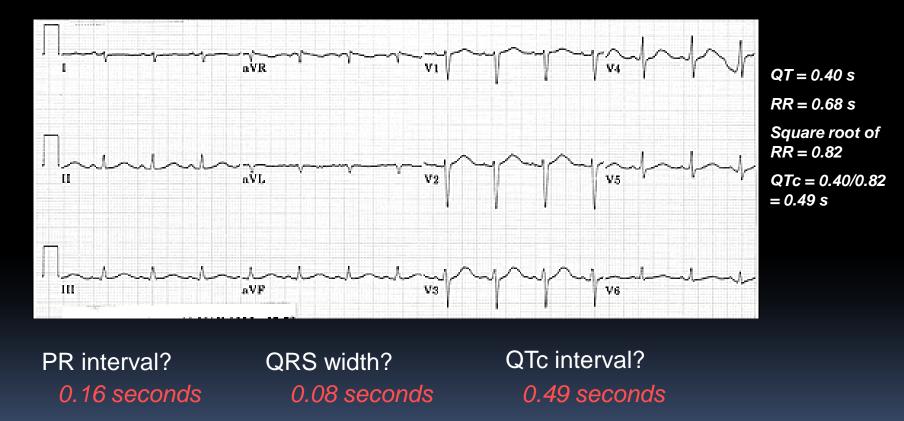
QTc = QT / square root of RR interval

QTc interval

< 0.44 s	> 0.44 s	Long QT
Normal	Long QT	

Torsades de Pointes

A prolonged QT can be very dangerous. It may predispose an individual to a type of ventricular tachycardia called Torsades de Pointes. Causes include drugs, electrolyte abnormalities, CNS disease, post-MI, and congenital heart disease.



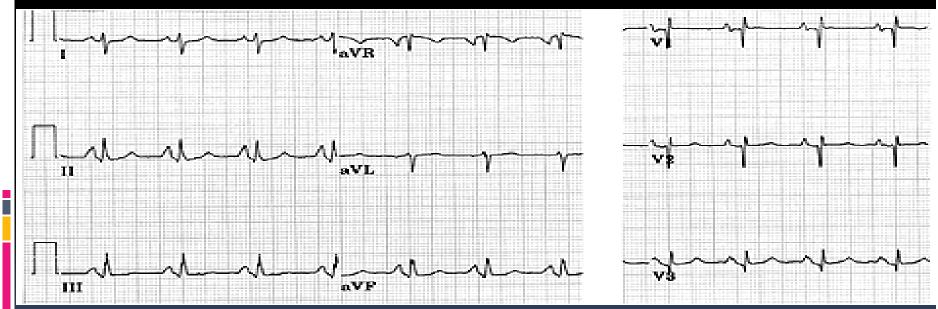
Interpretation of intervals? Normal PR and QRS, long QT

In this step of the 12-lead ECG analysis, we use the ECG to determine if any of the 4 chambers of the heart are enlarged or hypertrophied. We want to determine if there are any of the following:

- Right atrial enlargement (RAE)
- Left atrial enlargement (LAE)
- Right ventricular hypertrophy (RVH)
- Left ventricular hypertrophy (LVH)

Right atrial enlargement

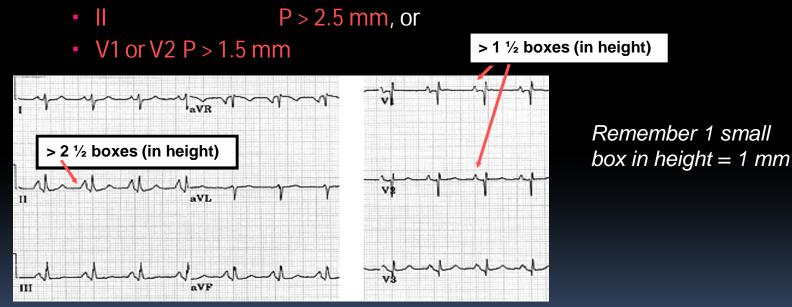
Take a look at this ECG. What do you notice about the P waves?



The P waves are tall, especially in leads II, III and avF. Ouch! They would hurt to sit on!!

Right atrial enlargement

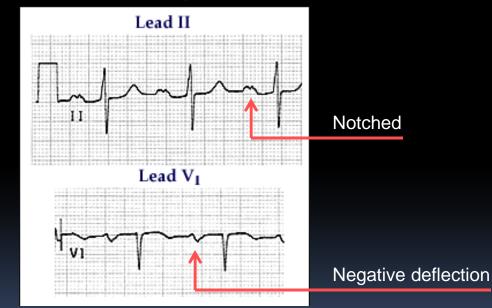
• To diagnose RAE you can use the following criteria:



A cause of RAE is RVH from pulmonary hypertension.

Left atrial enlargement

• Take a look at this ECG. What do you notice about the P waves?



The P waves in lead II are notched and in lead V1 they have a deep and wide negative component.

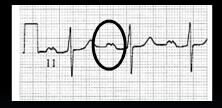
Left atrial enlargement

- To diagnose LAE you can use the following criteria:
 - II > 0.04 s (1 box) between notched peaks, or
 V1 Neg. deflection > 1 box wide x 1 box deep





Normal



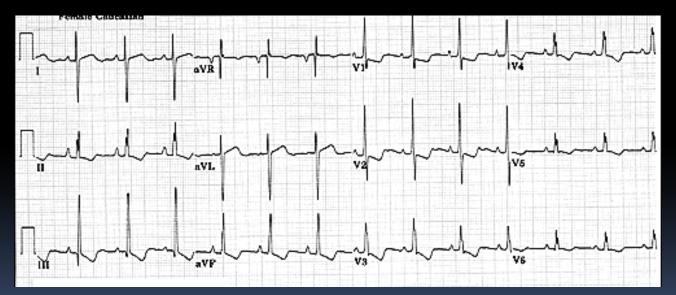




A common cause of LAE is LVH from hypertension.

Right ventricular hypertrophy

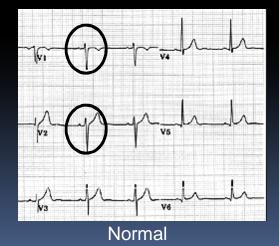
Take a look at this ECG. What do you notice about the axis and QRS complexes over the right ventricle (V1, V2)?

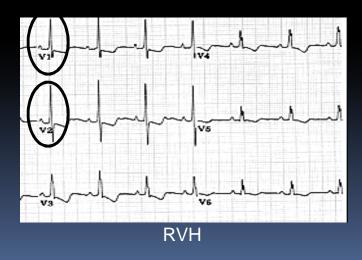


There is right axis deviation (negative in I, positive in II) and there are tall R waves in V1, V2.

Right ventricular hypertrophy

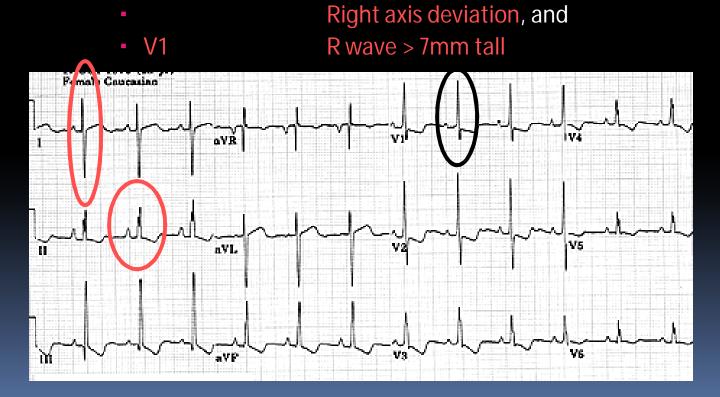
- Compare the R waves in V1, V2 from a normal ECG and one from a person with RVH.
- Notice the R wave is normally small in V1, V2 because the right ventricle does not have a lot of muscle mass.
- But in the hypertrophied right ventricle the R wave is tall in V1, V2.





Right ventricular hypertrophy

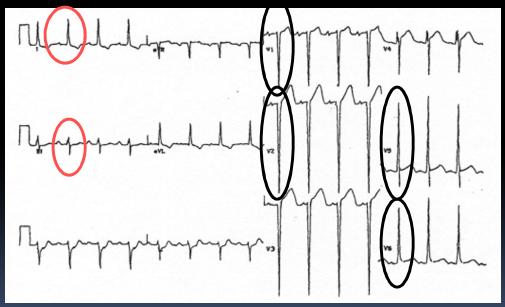
• To diagnose RVH you can use the following criteria:



A common cause of RVH is left heart failure.

Left ventricular hypertrophy

Take a look at this ECG. What do you notice about the axis and QRS complexes over the left ventricle (V5, V6) and right ventricle (V1, V2)?



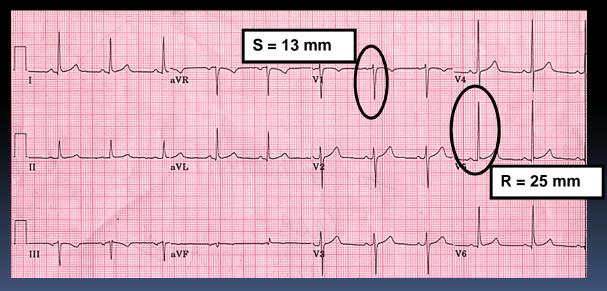
The deep S waves seen in the leads over the right ventricle are created because the heart is depolarizing left, superior and posterior (away from leads V1, V2).

There is left axis deviation (positive in I, negative in II) and there are tall R waves in V5, V6 and deep S waves in V1, V2.

Left ventricular hypertrophy

- To diagnose LVH you can use the following criteria*:
 - R in V5 (or V6) + S in V1 (or V2) > 35 mm, or
 - avL

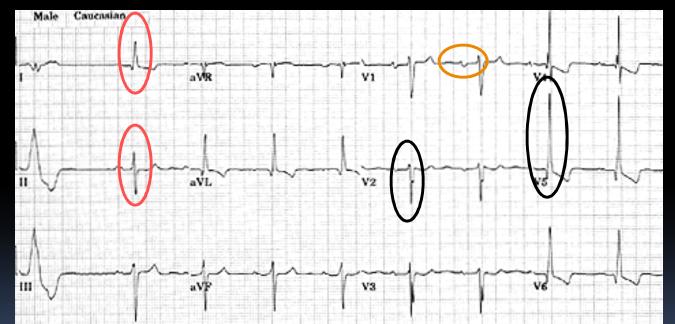
R > 13 mm



* There are several other criteria for the diagnosis of LVH.

A common cause of LVH is hypertension.

A 63 yo man has longstanding, uncontrolled hypertension. Is there evidence of heart disease from his hypertension? (*Hint: There a 3 abnormalities.*)



Yes, there is left axis deviation (positive in I, negative in II), left atrial enlargement (> 1 x 1 boxes in V1) and LVH (R in V5 = 27 + S in V2 = $10 \rightarrow$ > 35 mm).

When analyzing a 12-lead ECG for evidence of an infarction you want to look for the following:

Abnormal Q waves

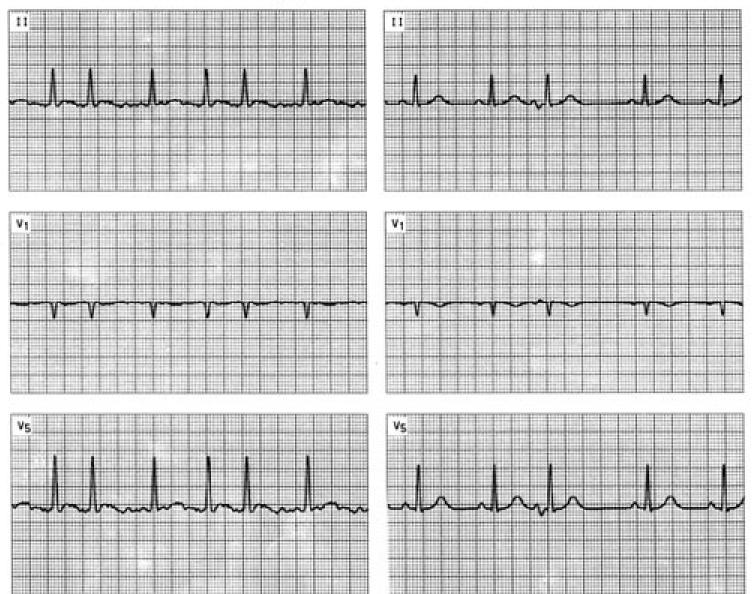
- ST elevation or depression
- Peaked, flat or inverted T waves

These topics were covered in Modules V and VI where you learned:

- ST elevation (or depression) of 1 mm in 2 or more contiguous leads is consistent with an AMI
- There are ST elevation (Q-wave) and non-ST elevation (non-Q wave) MIs

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Irregular ECGs

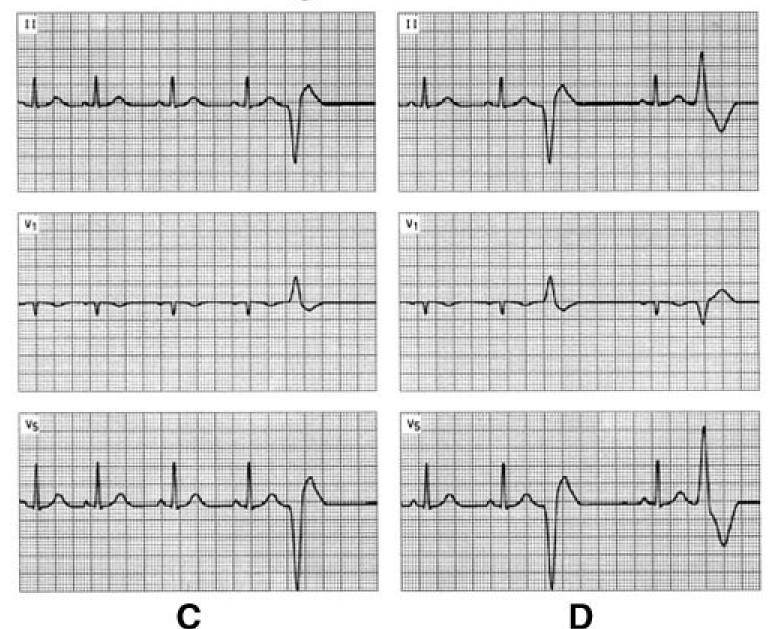


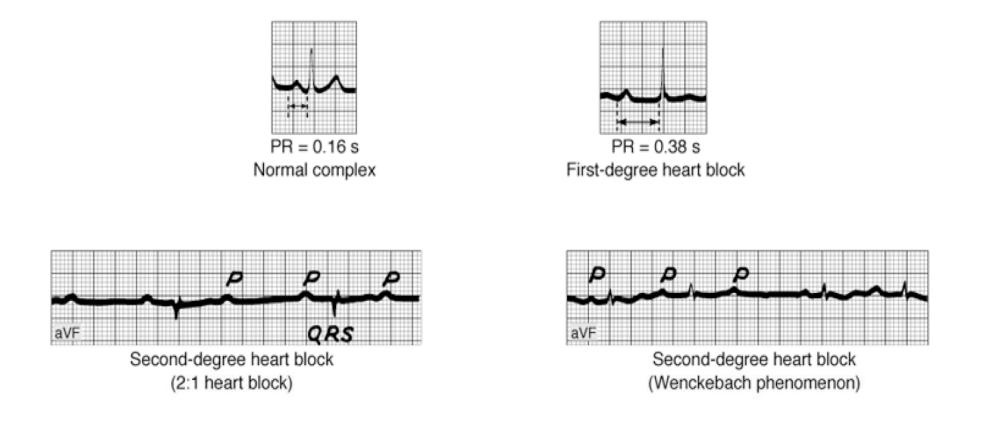
Δ

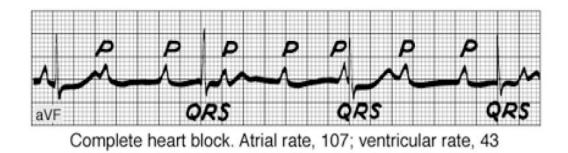
B

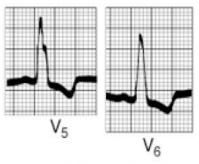
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Irregular ECGs









Two V leads in left bundle branch block

Figure 28-11. Heart block.

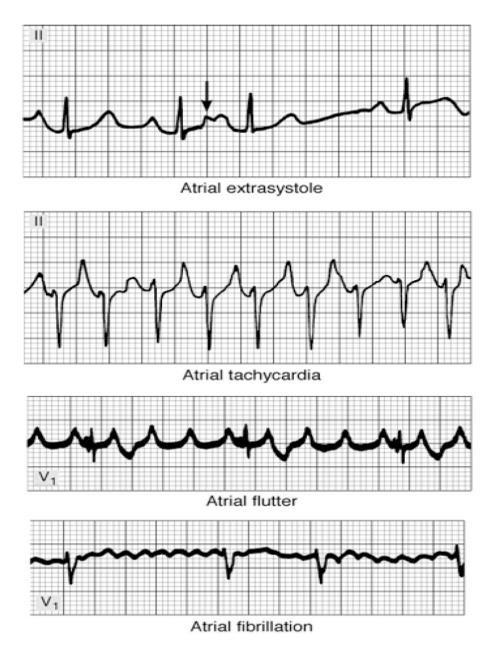


Figure 28-13. Atrial arrhythmias. The illustration shows an atrial premature beat with its P wave superimposed on the T wave of the preceding beat (arrow); atrial tachycardia; atrial flutter with 4:1 AV block; and atrial fibrillation with a totally irregular ventricular rate. (Tracings reproduced, with permission, from Goldschlager N, Goldman MJ: *Principles of Clinical Electrocardiography*, 13th ed. Originally published by Appleton & Lange. Copyright © 1989 by The McGraw-Hill Companies, Inc.)

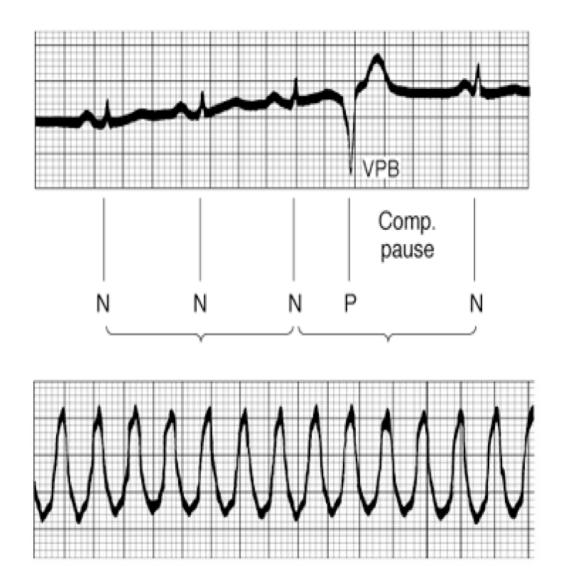


Figure 28-14. Top: Ventricular premature beats (VPB). The lines under the tracing illustrate the compensatory pause and show that the duration of the premature beat plus the preceding normal beat is equal to the duration of two normal beats. **Bottom:** Ventricular tachycardia.

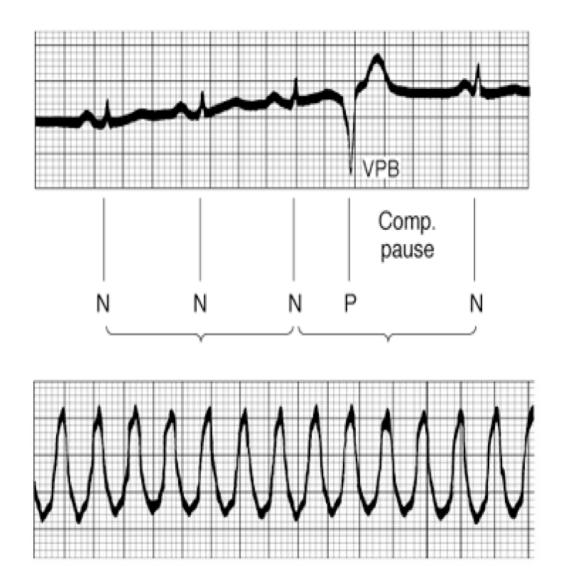
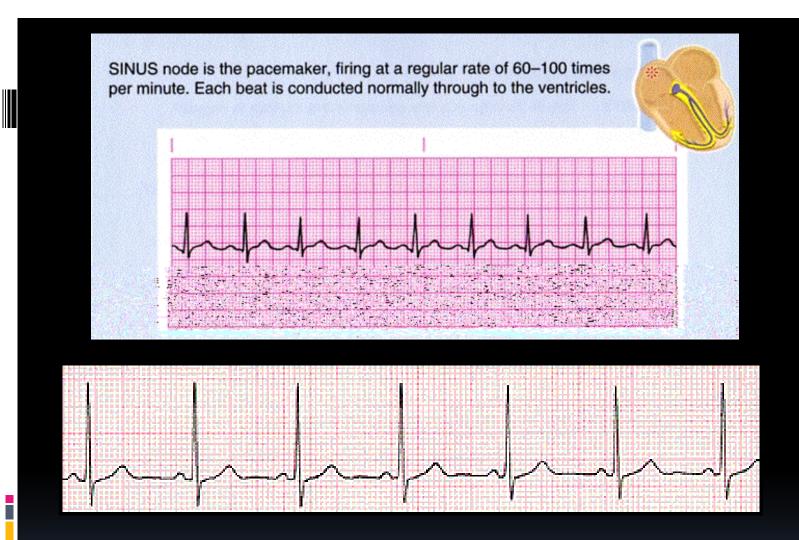
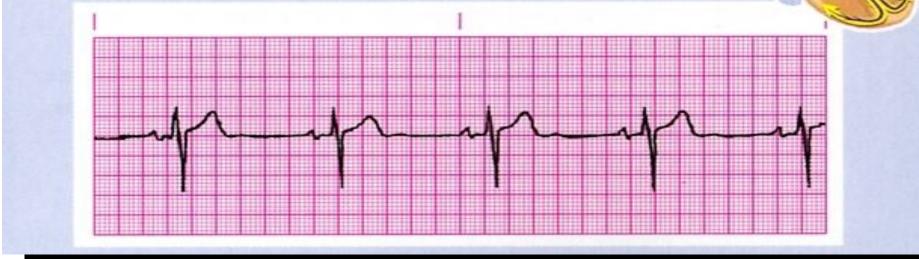


Figure 28-14. Top: Ventricular premature beats (VPB). The lines under the tracing illustrate the compensatory pause and show that the duration of the premature beat plus the preceding normal beat is equal to the duration of two normal beats. **Bottom:** Ventricular tachycardia.



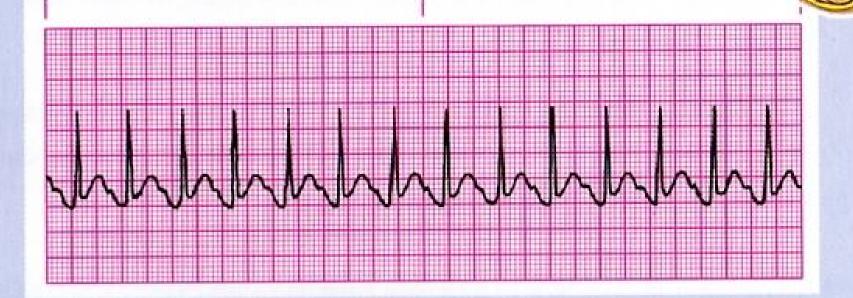
Normal Sinus Rhythm

SINUS node is the pacemaker, firing regularly at a rate of less than 60 times per minute. Each impulse is conducted normally through to the ventricles.



Sinus Bradycardia

SINUS node is the pacemaker, firing regularly at a rate of greater than 100 times per minute. Each impulse is conducted normally through to the ventricles.



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Sinus Tachycardia

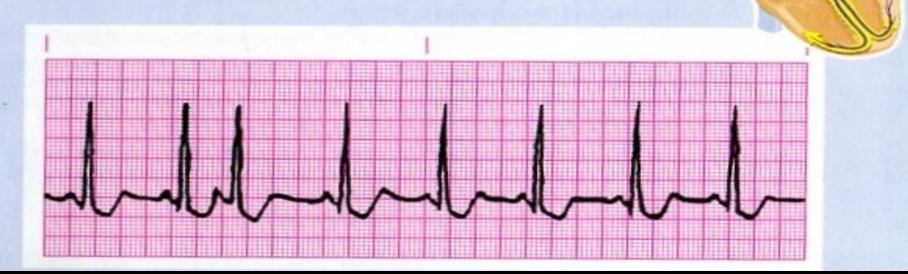
SINUS node is the pacemaker, but impulses are initiated in an irregular pattern. The rate increases as the patient breathes in and decreases as the patient breathes out. Each beat is conducted normally through to the ventricles.



놼

Sinus Arrhythmia

The pacemaker is an irritable focus within the ATRIUM that fires prematurely and produces a single ectopic beat. Conduction through to the ventricles is normal.



Premature atrial contraction acronym is PAC; sort of a misnomer as this is a premature atrial depolarization.

Some also call this a premature atrial beat (PAB), but again electrical depolarization does not always mean mechanical contraction.

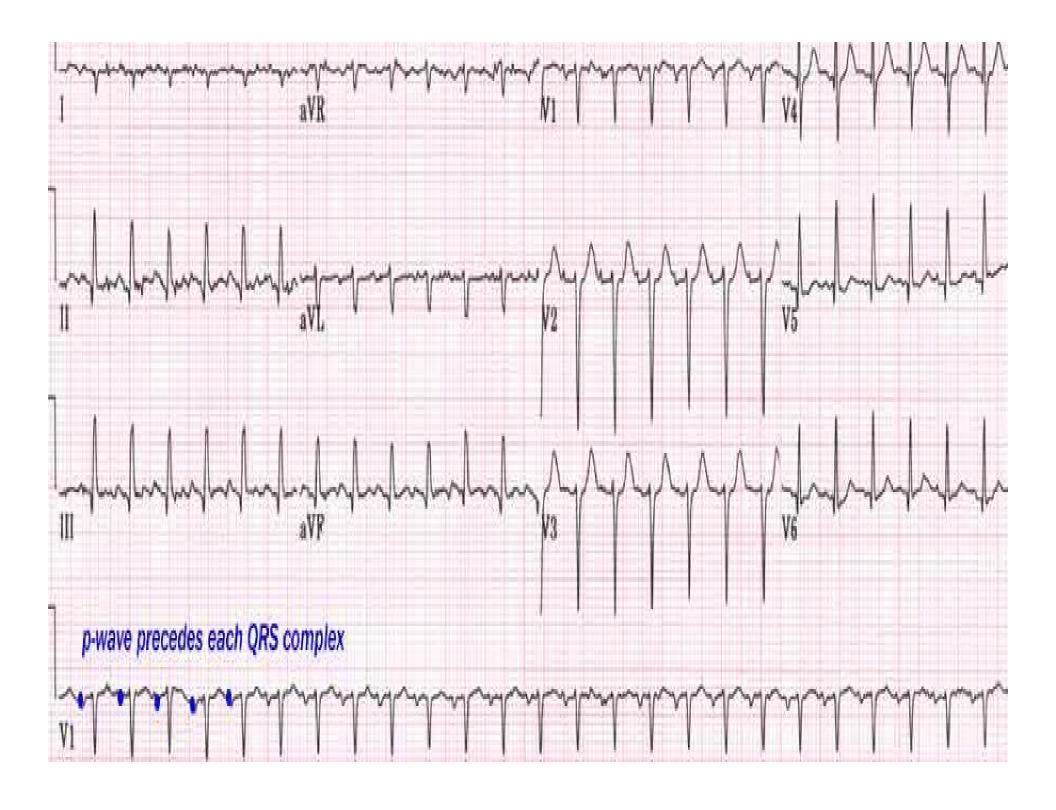
Here there is a focus other than the SA node that is firing, which causes a QRS.

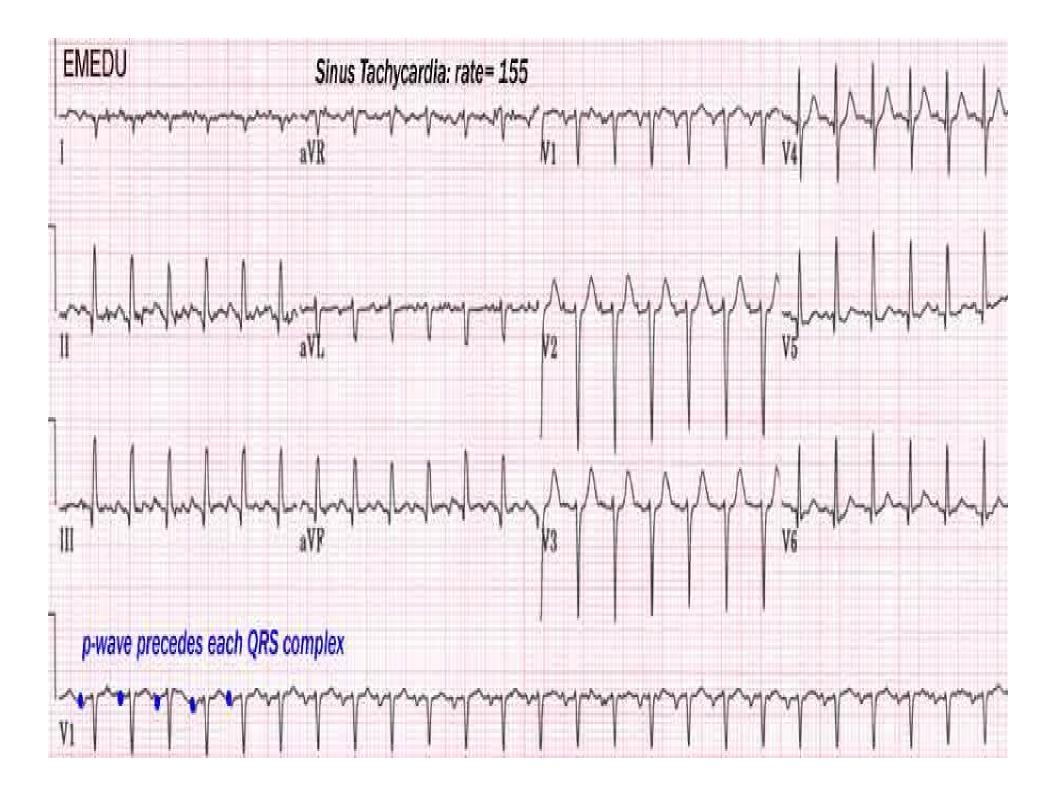
What is they underlying rate? 7*10 using 6-second. Or, 75 using 4 big boxes per QRS.

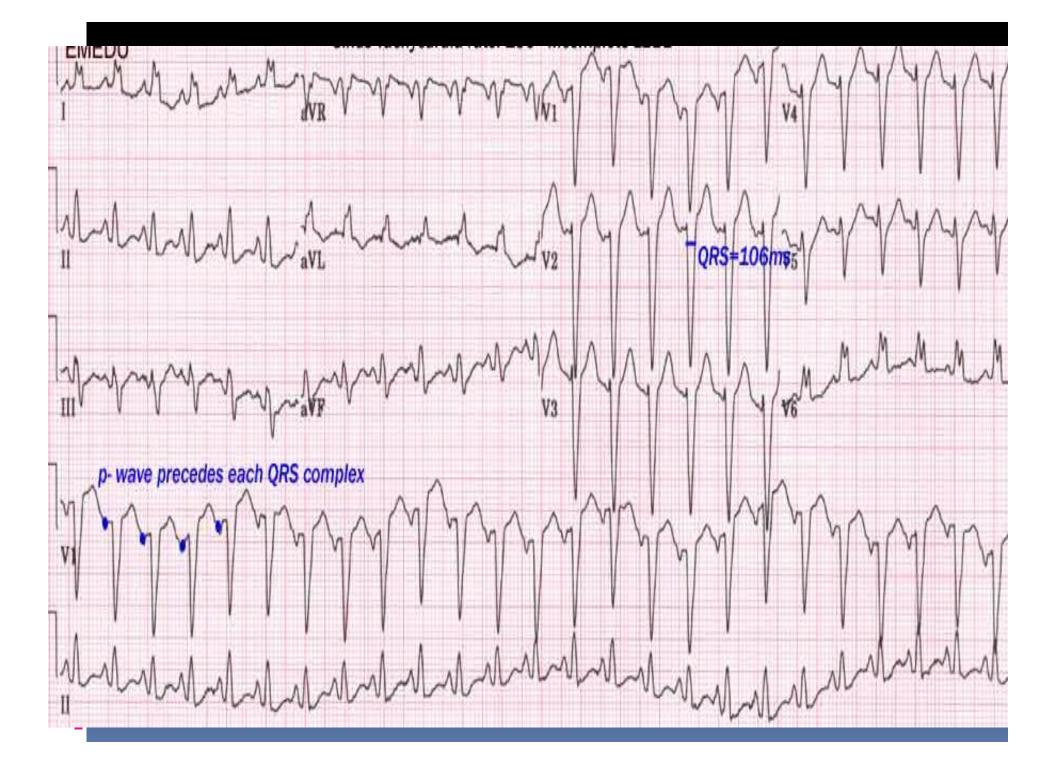
The PAC is the third QRS; you can see there is only about 1 big box between the 2nd and 3rd QRS. Usually the abnormal focus will cause a p-wave the looks different from the other p-waves (the SA node p-waves).

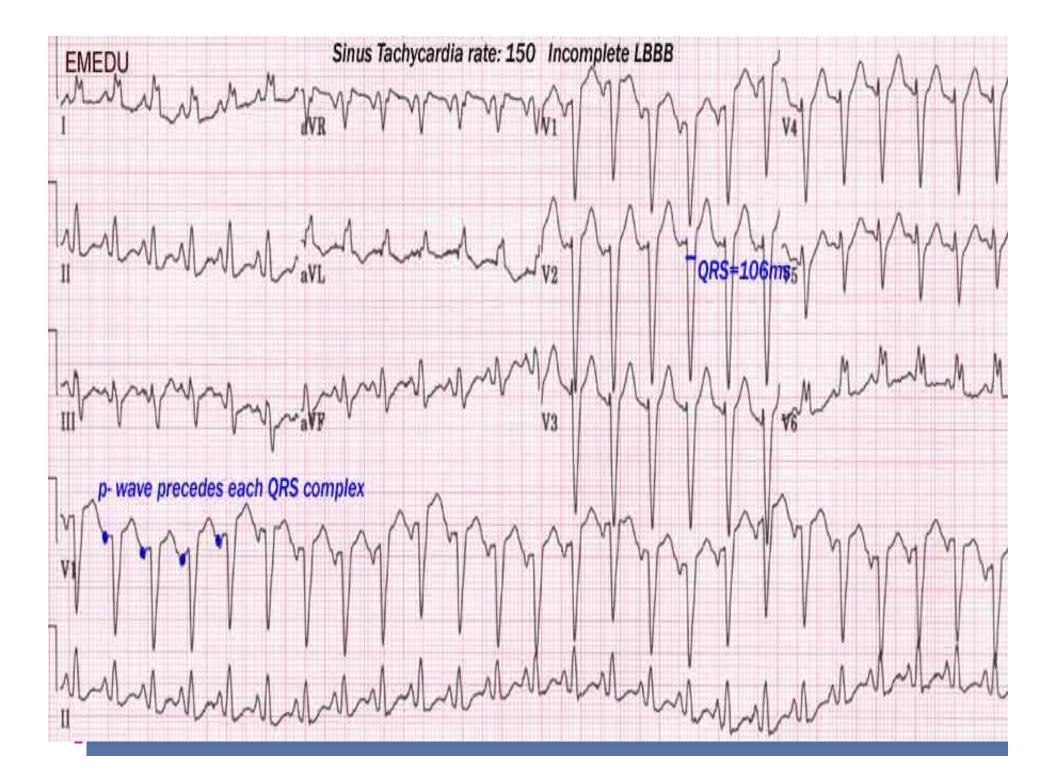
Here the aberrant atrial depolarization appears as a peaked p-wave in comparison.

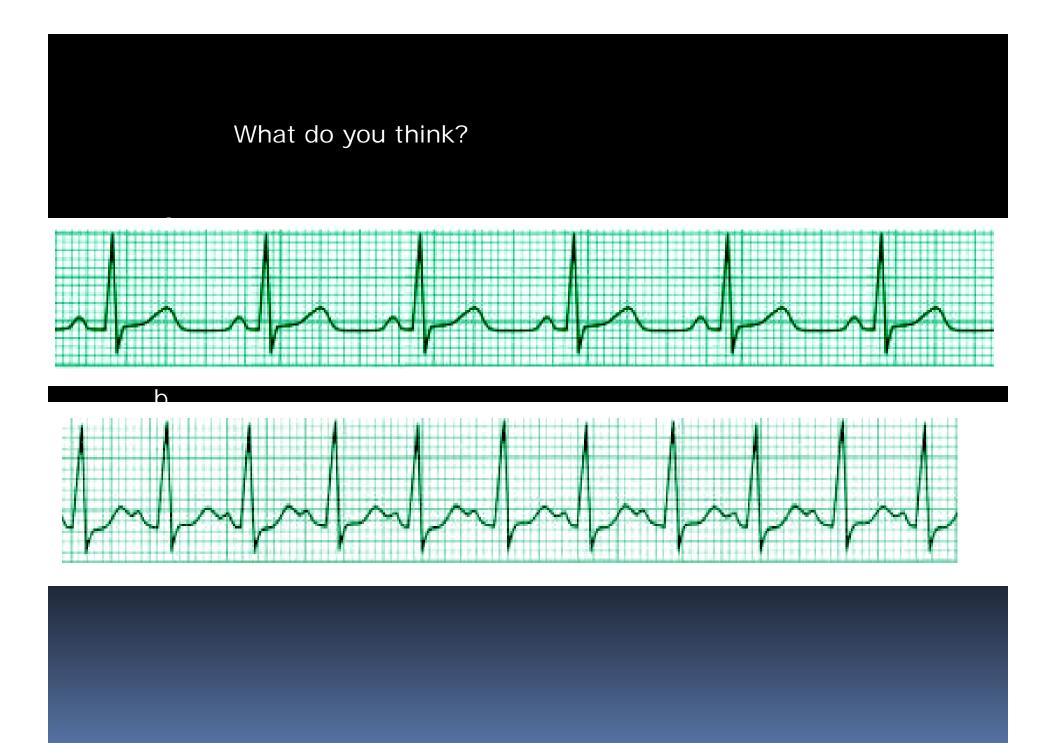
Premature Atrial Contraction

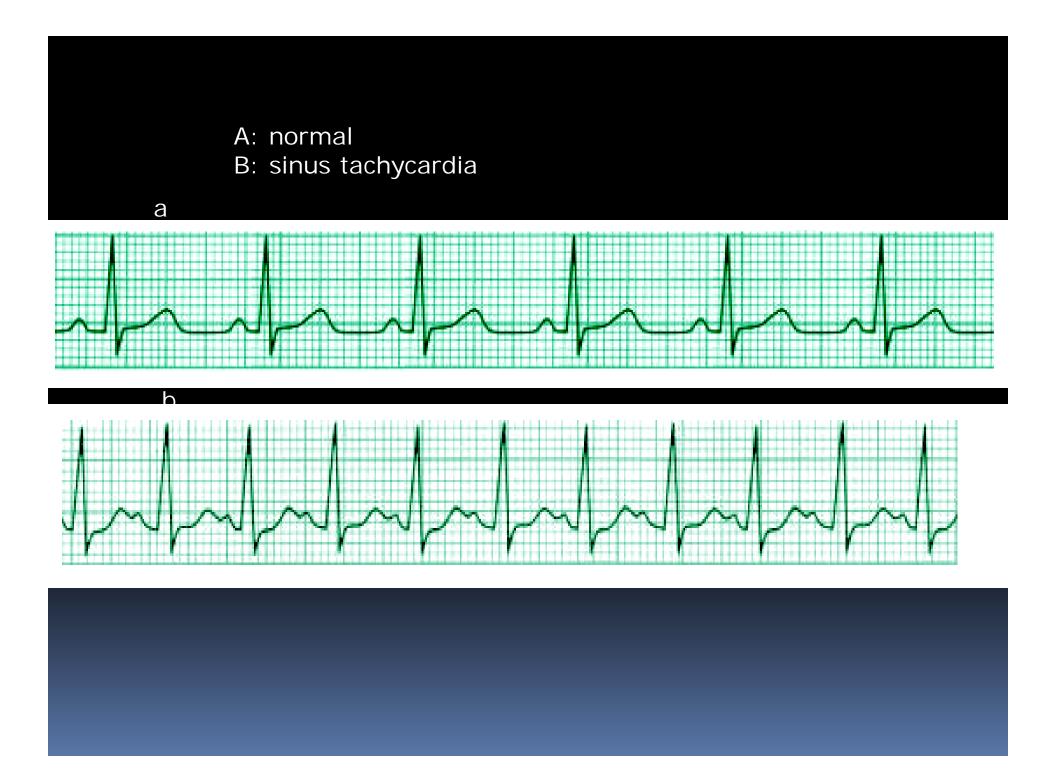


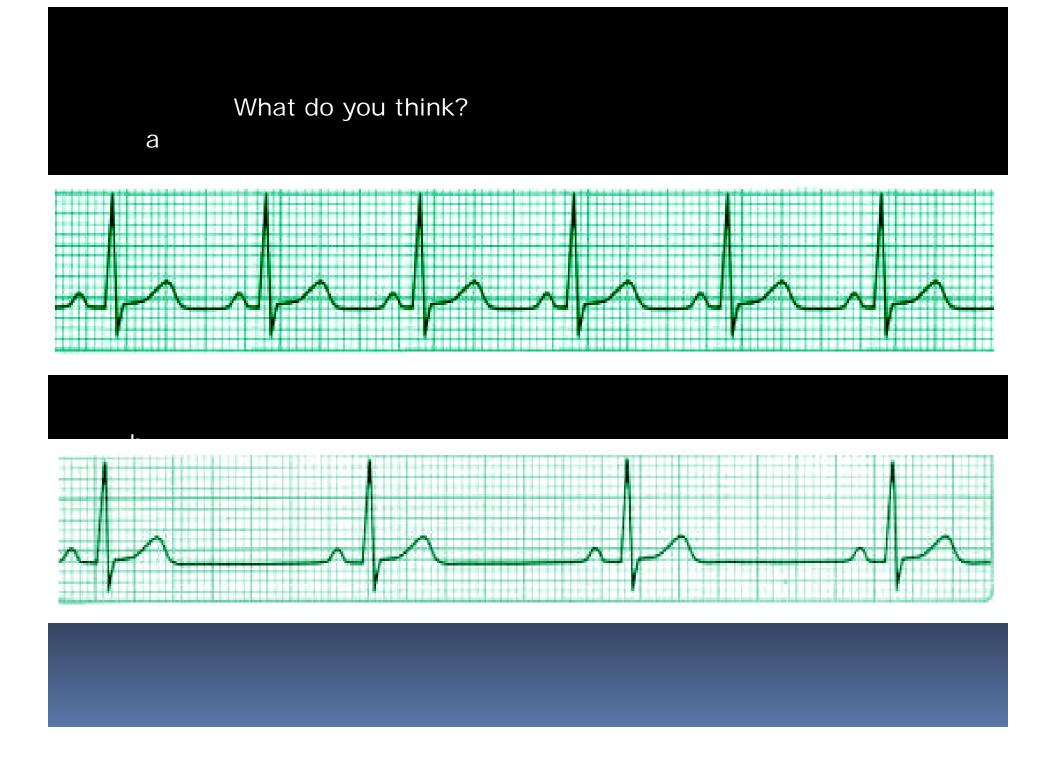


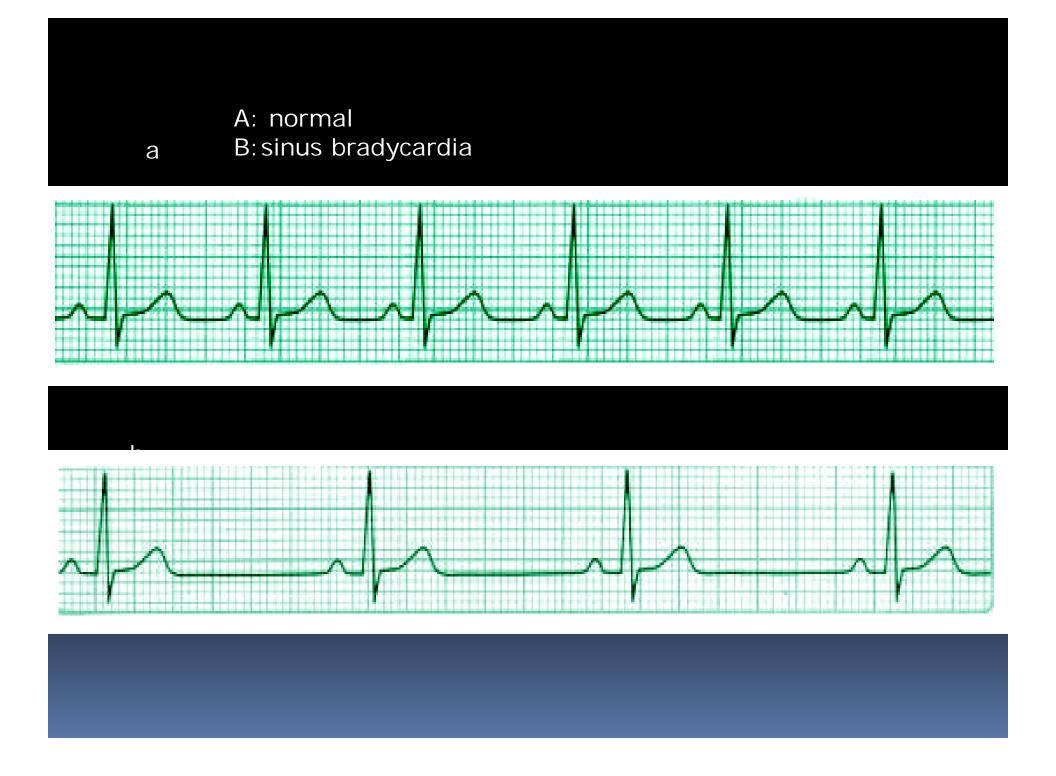


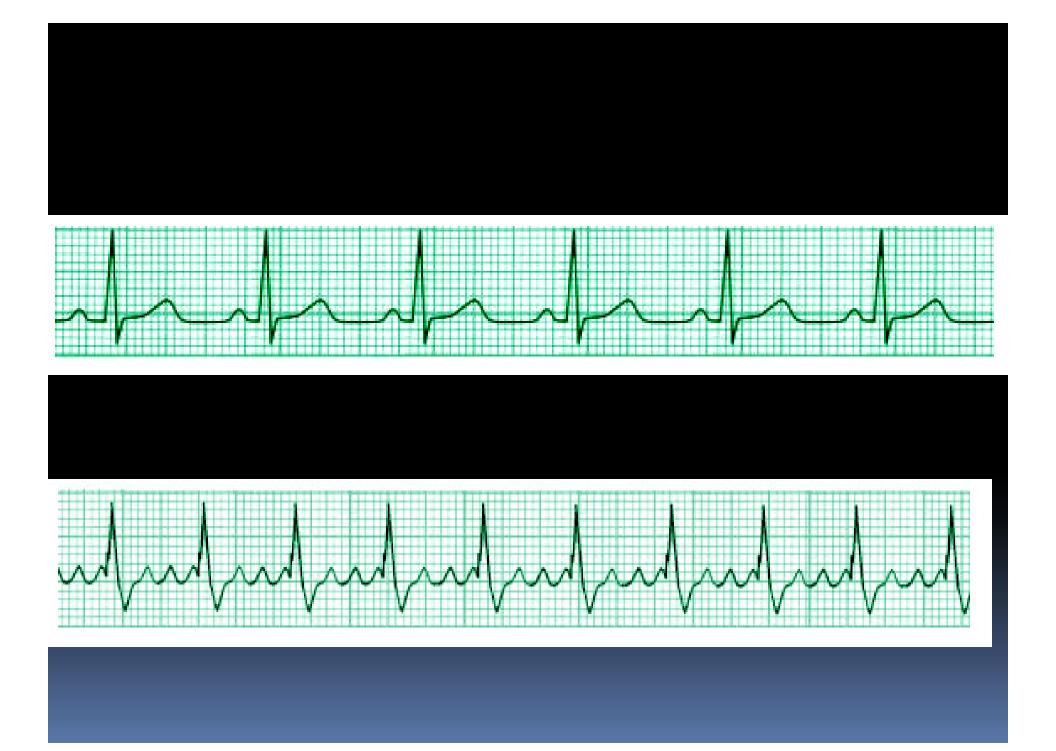


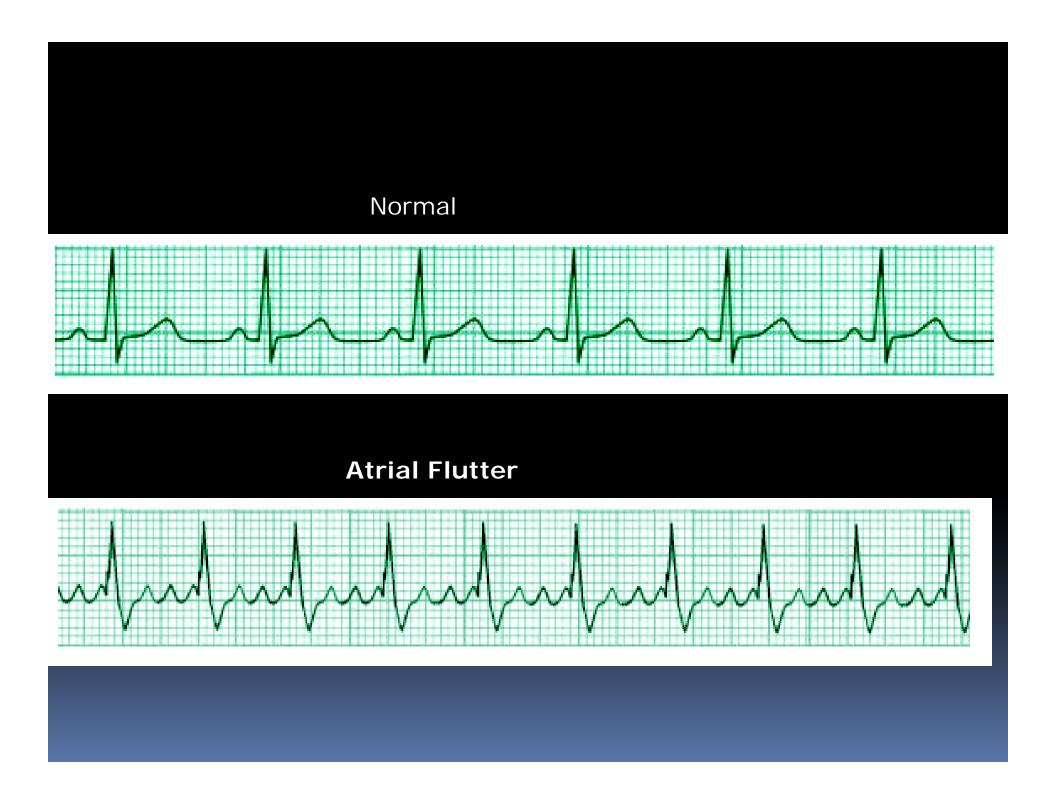






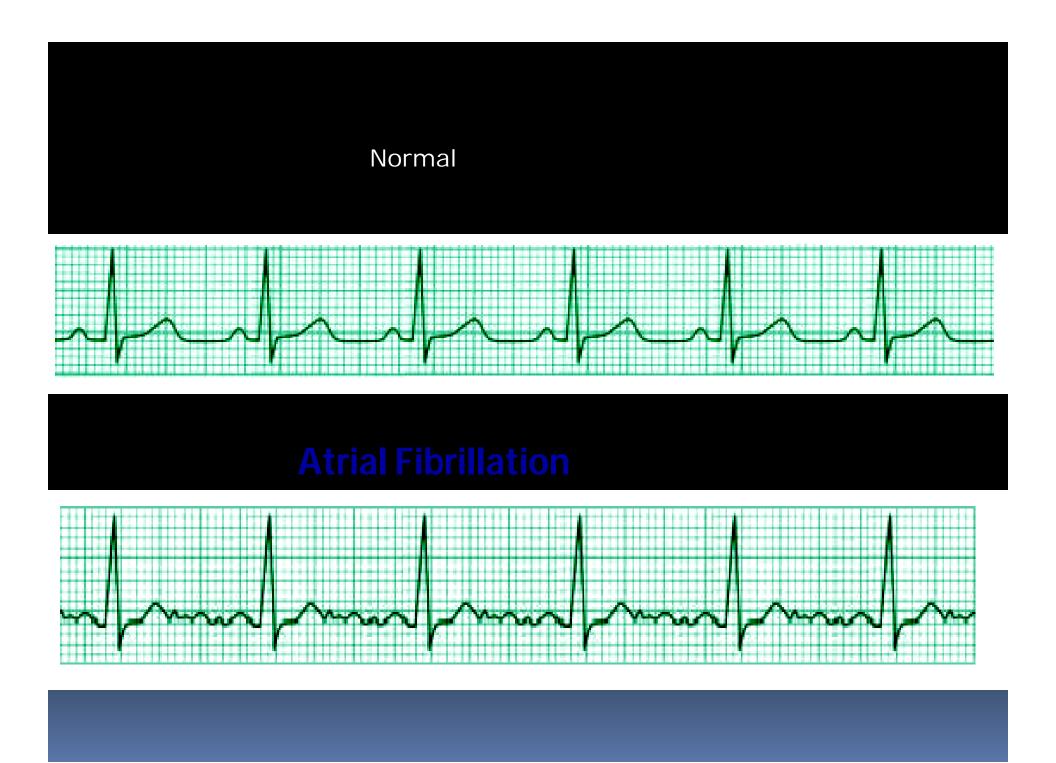








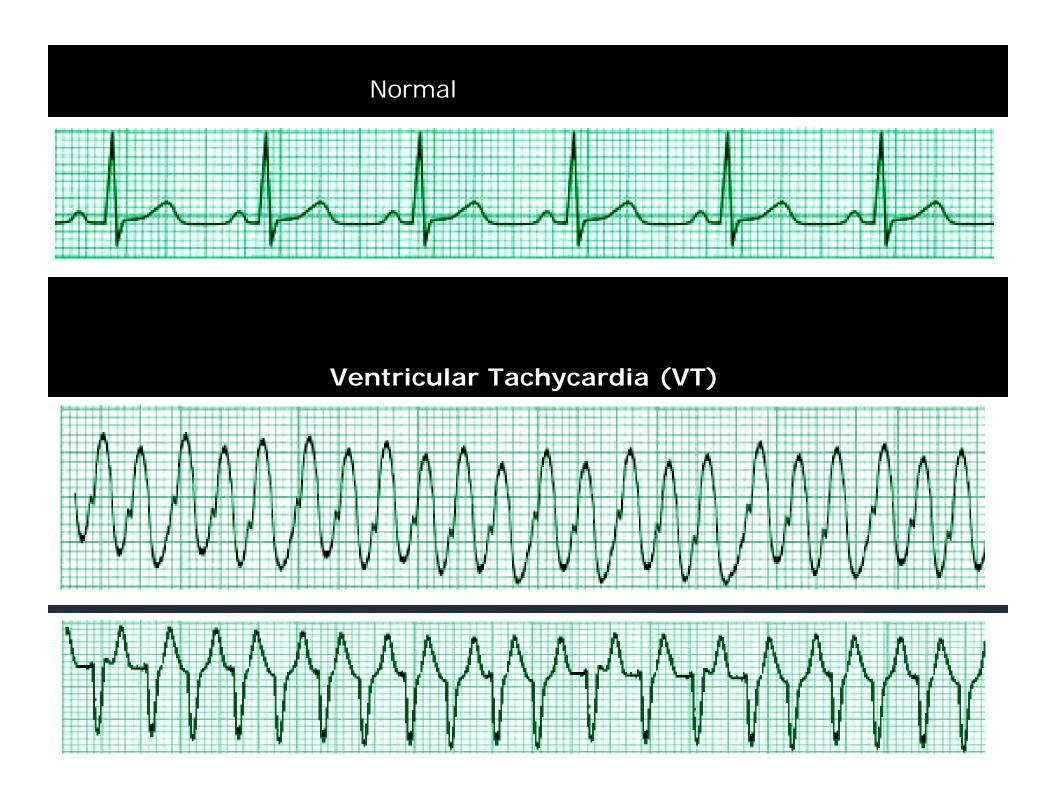


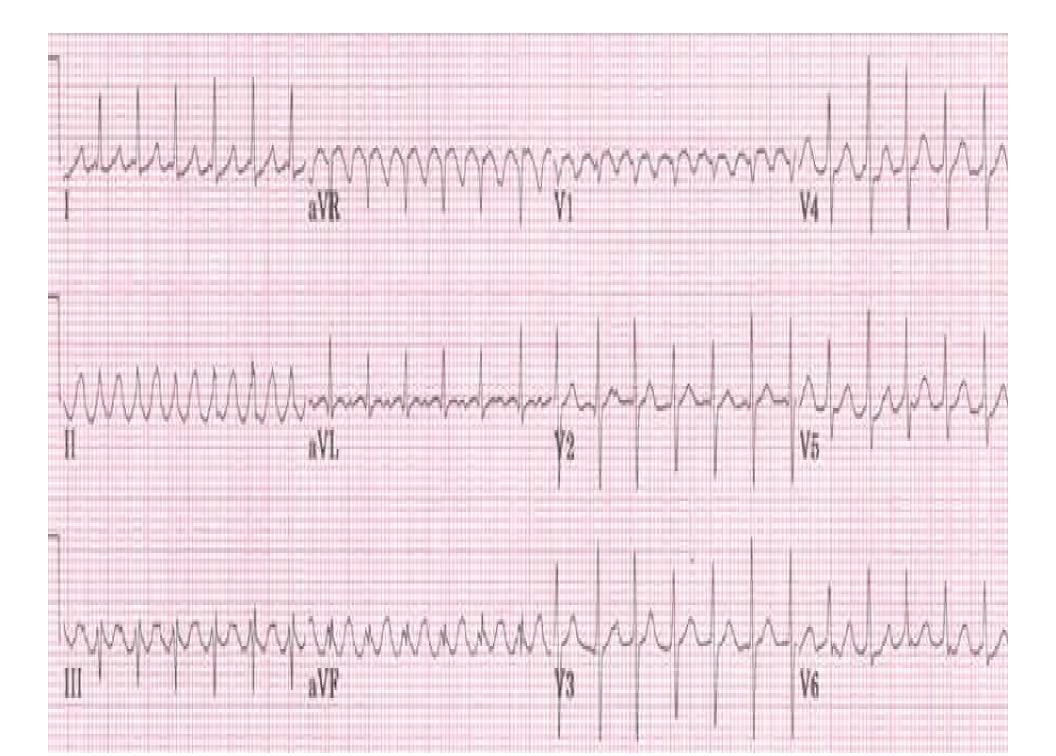


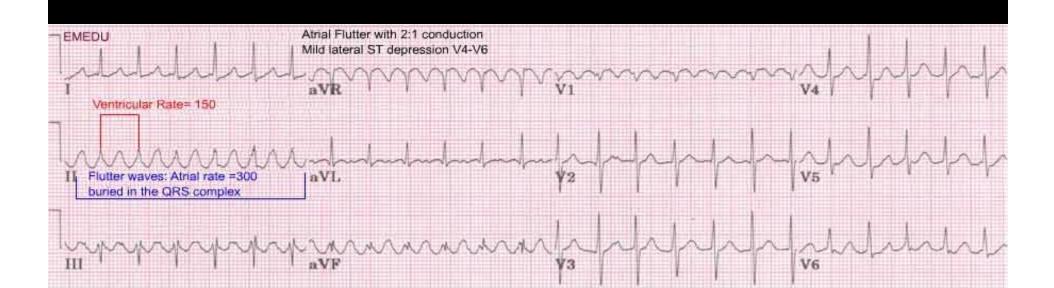












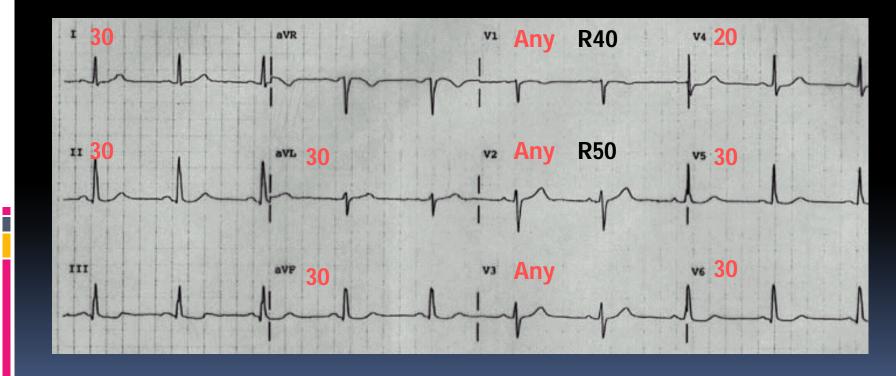
Rate Rhythm Axis Intervals Hypertrophy

Tip: One way to determine if Q waves (and R waves) are abnormal is by looking at the width and using the following mantra (read red downwards):

Any	Any Q wave in V1
Any	Any Q wave in V2
Any	Any Q wave in V3
20	A Q wave \geq 20 msec in V4 (i.e. 0.02 sec or $\frac{1}{2}$ width of a box)
30	A Q wave <u>></u> 30 msec in V5
30	A Q wave \geq 30 msec in V6
30	A Q wave <u>></u> 30 msec in I
30	A Q wave <u>></u> 30 msec in avL
30	A Q wave <u>></u> 30 msec in II
30	A Q wave \geq 30 msec in avF
R40	A R wave <u>></u> 40 msec in V1
R50	A R wave <u>></u> 50 msec in V2

Rate Rhythm Axis Intervals Hypertrophy

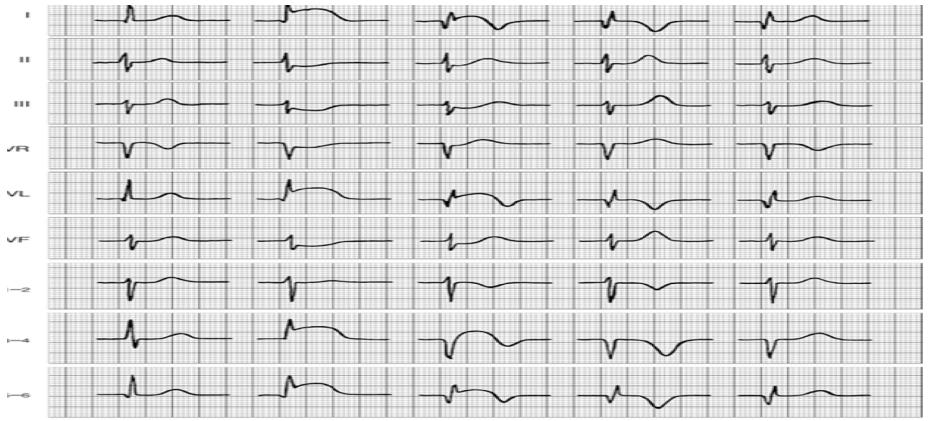
This mantra corresponds to the ECG in the following way:



SUMMARY Rate Rhythm Axis Intervals Hypertrophy Infarct

To summarize:

- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine **QRS AXIS**
 - Normal
 - Left axis deviation
 - Right axis deviation
 - Right superior axis deviation

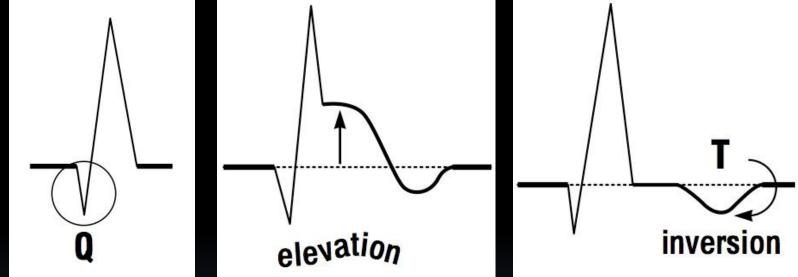


A: Normal tracing. B: very early pattern (nours after infarction): Si segment elevation in 1, avL, and V₃₋₆; reciprocal ST depression in II, III, and aVF. C: Later pattern (many hours to a few days): Q

waves have appeared in I, aVL, and V_{5-6} . QS complexes are present in V_{3-4} . This indicates that the major transmural infarction is underlying the area recorded by V_{3-4} ; ST segment changes persist but are of lesser degree, and the T waves are beginning to invert in the leads in which the ST segments are elevated. **D**: Late established pattern (many days to weeks): The Q waves and QS complexes persist, the ST segments are isoelectric, and the T waves are symmetric and deeply inverted in leads that had ST elevation and tall in leads that had ST depression. This pattern may persist for the remainder of the patient's life. **E**: Very late pattern: This may occur many months to years after the infarction. The abnormal Q waves and QS complexes persist. The T waves have gradually returned to normal. (Reproduced, with permission, from Goldschlager N, Goldman MJ: *Principles of Clinical Electrocardiography*, 13th ed. Originally published by Appleton & Lange. Copyright © 1989 by The McGraw-Hill Companies, Inc.)

- Significant Q wave = Necrosis
- ST elevation = Injury

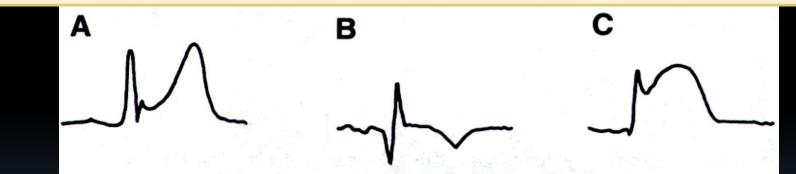
T wave inversion = Ischemia



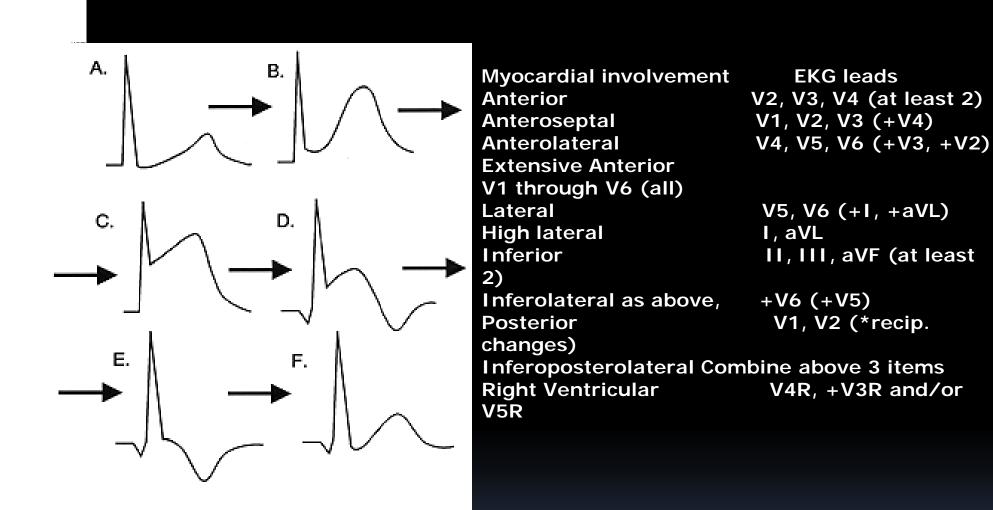
Myocardial Infarction

Location of Myocardial Ischemia/ Infarction

Location	Leads
Anterior	I, V_2 , V_3 , and V_4
Anterolateral	I, aVL, V_5 , and V_6
Lateral	V_5 and V_6
High lateral	I and aVL (often with V_5 , V_6)
Inferior	II, III, and aVF
Inferolateral	II, III, aVF, and V ₆
True posterior	Reciprocal changes in V_1 and V_2



MI Location



Evolution of Acute MI

ELEVATION

- Electrolytes
- Left bundle branch block
- Early repolarization
- Ventricular hypertrophy
- Aneurysm
- Treatment (pericardiocentesis)
- Injury (acute MI, contusion)
- Osborne waves (hypothermia)
- Nonocclusive vasospasm

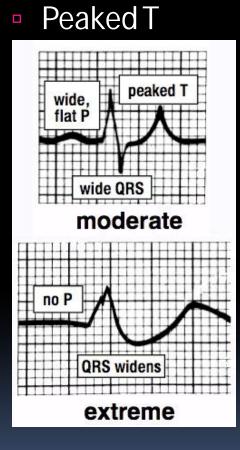
ST Segment Elevation

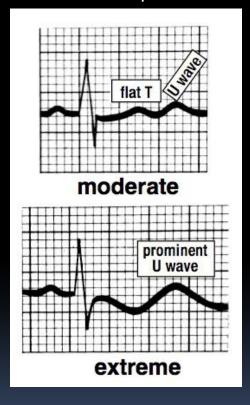
DEPRESSED ST

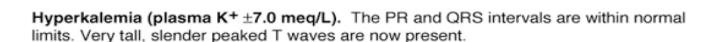
- Drooping valve (mitral valve prolapse)
- Enlargement or LV with strain
- Potassium loss (hypokalemia)
- Reciprocal ST depression (inferior MI)
- Embolism (PE)
- Subendocardial ischemia
- Subendocardial infarct
- Encephalon hemorrhage
- Dilated cardiomyopathy
- Shock
- ST Segment Depression

High K+ High K+

Flat T, U Wave







Hyperkalemia (plasma K⁺ ±8.5 meq/L). There is no evidence of atrial activity; the QRS complex is broad and slurred and the QRS interval has widened to 0.2 s. The T waves remain tall and slender. Further elevation of the plasma K⁺ level may result in ventricular tachycardia and ventricular fibrillation.

Hypokalemia (plasma K⁺ \pm3.5 meq/L). PR interval = 0.2 s; QRS interval = 0.06 s; ST segment depression. A prominent U wave is now present immediately following the T. The actual QT interval remains 0.4 s. If the U wave is erroneously considered a part of the T, a falsely prolonged QT interval of 0.6 s will be measured.

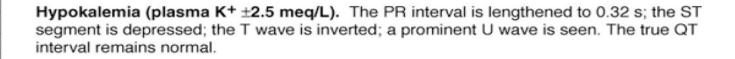
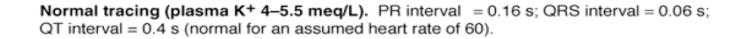


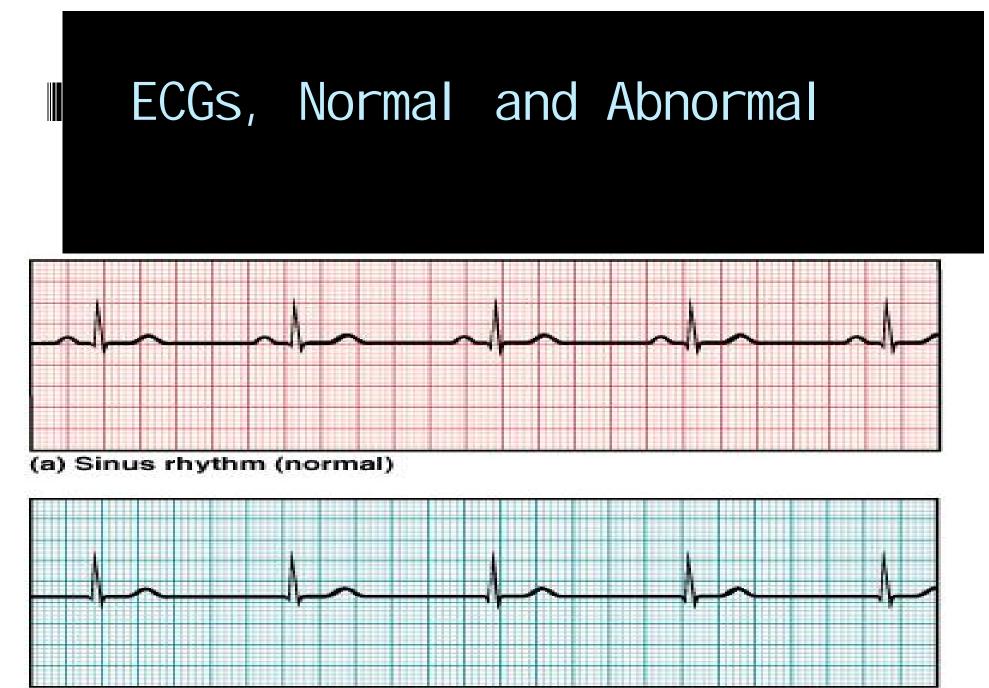
Figure 28-20. Correlation of plasma K⁺ level and the ECG, assuming that the plasma Ca²⁺ level is normal. The diagrammed complexes are left ventricular epicardial leads. (Reproduced, with permission, from Goldman MJ: *Principles of Clinical Electrocardiography*, 12th ed. Originally published by Appleton & Lange. Copyright © 1989 by The McGraw-Hill Companies, Inc.)







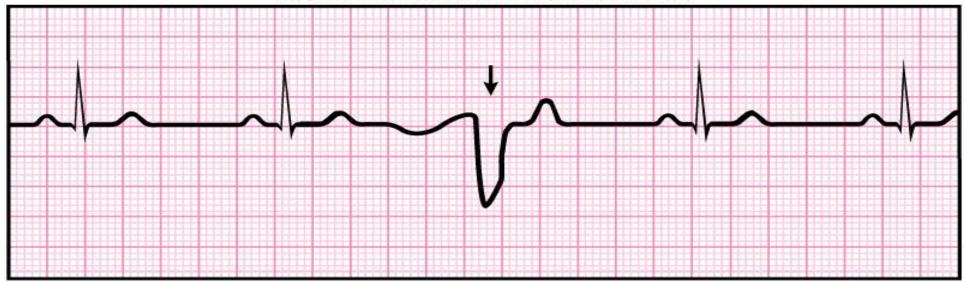




(b) Nodal rhythm - no SA node activity

ECGs, Abnormal

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(d) Premature ventricular contraction

Extrasystole : note inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.

