EKG The normal and abnormal

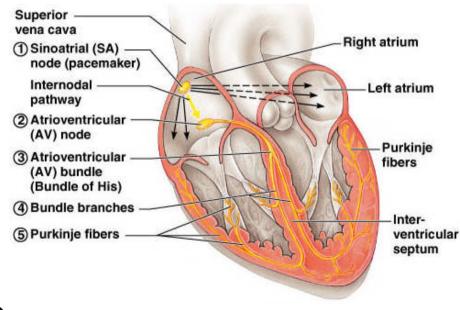
D.HAMMOUDI .MD

The Cardiac Cycle

- Heart at rest
 - Blood flows from large veins into atria
 Passive flow from atria into ventricles
- Atria (R & L) contract simultaneously
 - Blood forced into ventricles
- Ventricles (R & L) contract simultaneously
 - Atrioventricular valves close → "lubb" sound
 - Blood forced into large arteries
- Ventricles relax
 - Semilunar valves close → "dub" sound
- Heart at rest

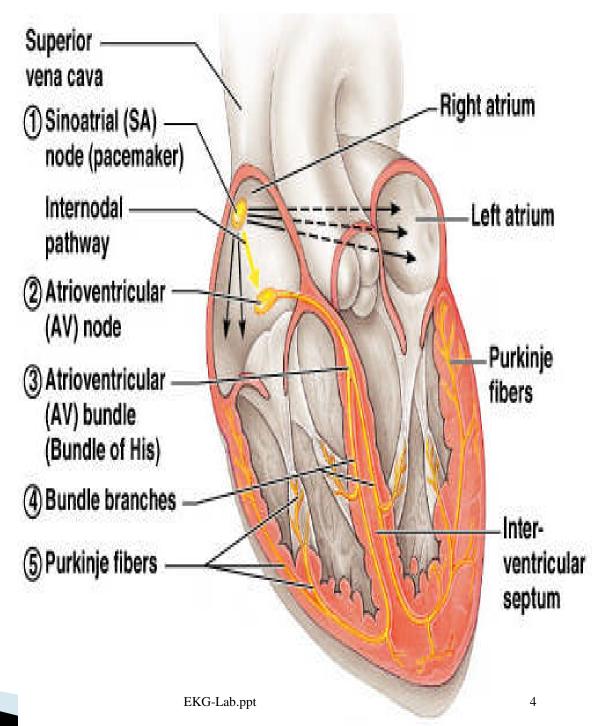
Depolarization and Impulse Conduction

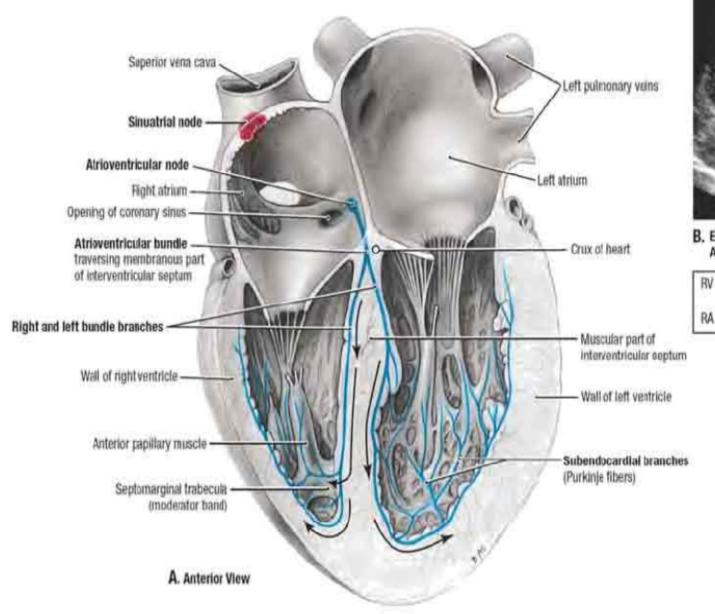
- Heart is autorhythmic
- Depolarization begins in sinoatrial (SA) node
- Spread through atrial myocardium
- Delay in atrioventricular (AV) node



Depolarization and Impulse Conduction

- Spread from atrioventricular (AV) node
 - AV bundle
 - Bundle branches
 - Purkinje fibers

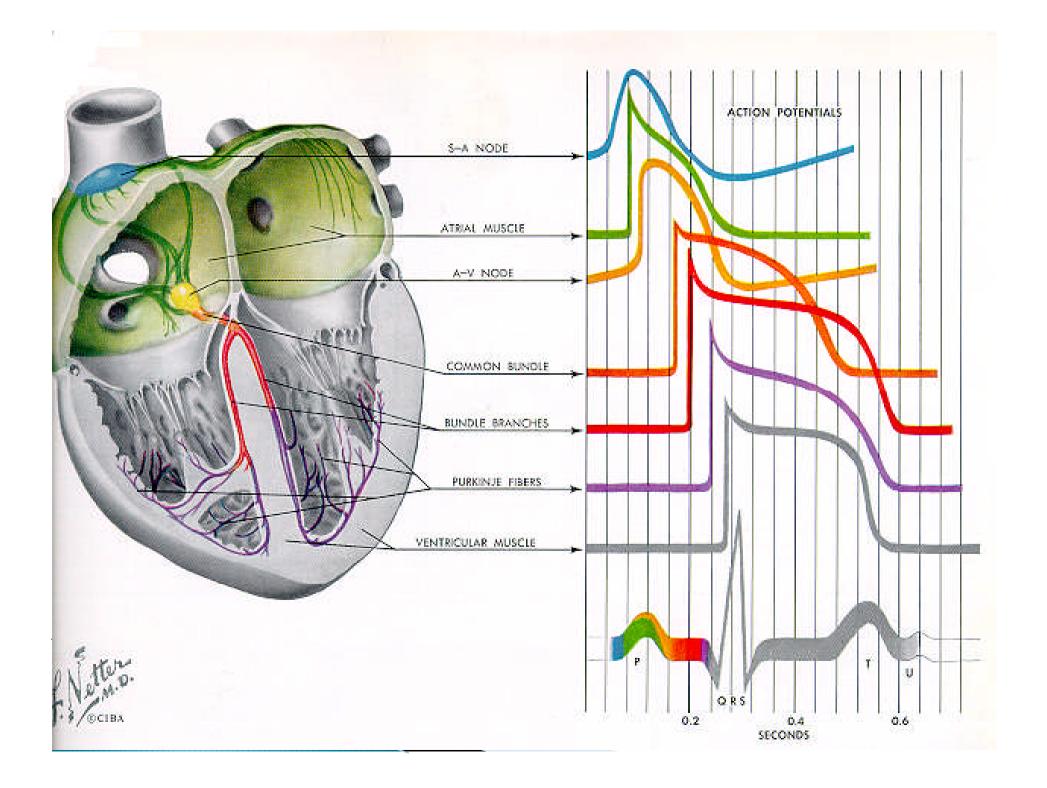






B. Echocardiogram, Apical Four-chamber View

RV Right ventricle LV Left ventricle x Crux of heart
RA Right atrium LA Left atrium



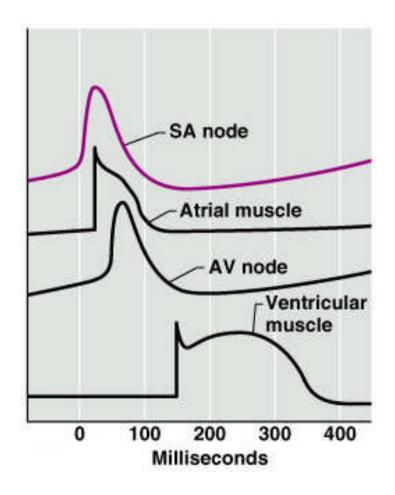
Arrhythmia Formation

Arrhythmias can arise from problems in the:

- Sinus node
- Atrial cells
- AV junction
- Ventricular cells

Depolarization and Impulse Conduction

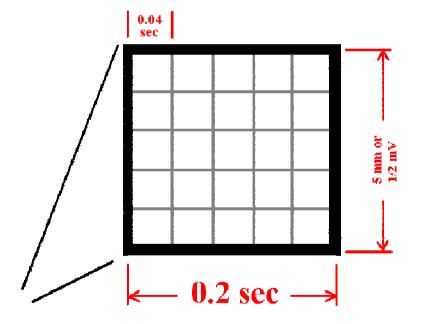
Depolarization in SA node precedes depolarization in atria, AV node, ventricles



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The ECG Paper

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





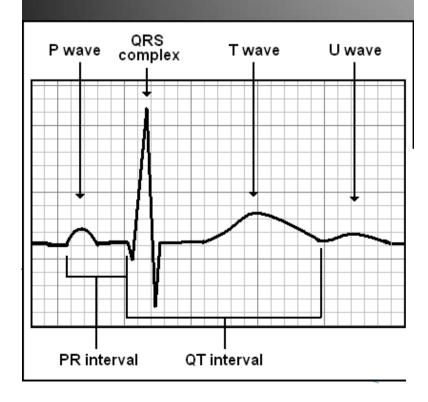
The ECG Paper (cont)



- Every 3 seconds (15 large boxes) is marked by a vertical line.
- This helps when calculating the heart rate.

NOTE: the following strips are not marked but all are 6 seconds long.

Waveforms and Intervals



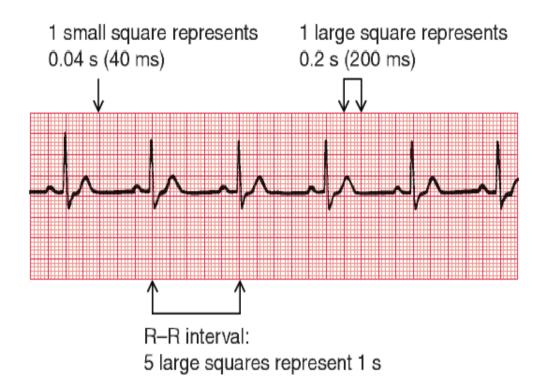
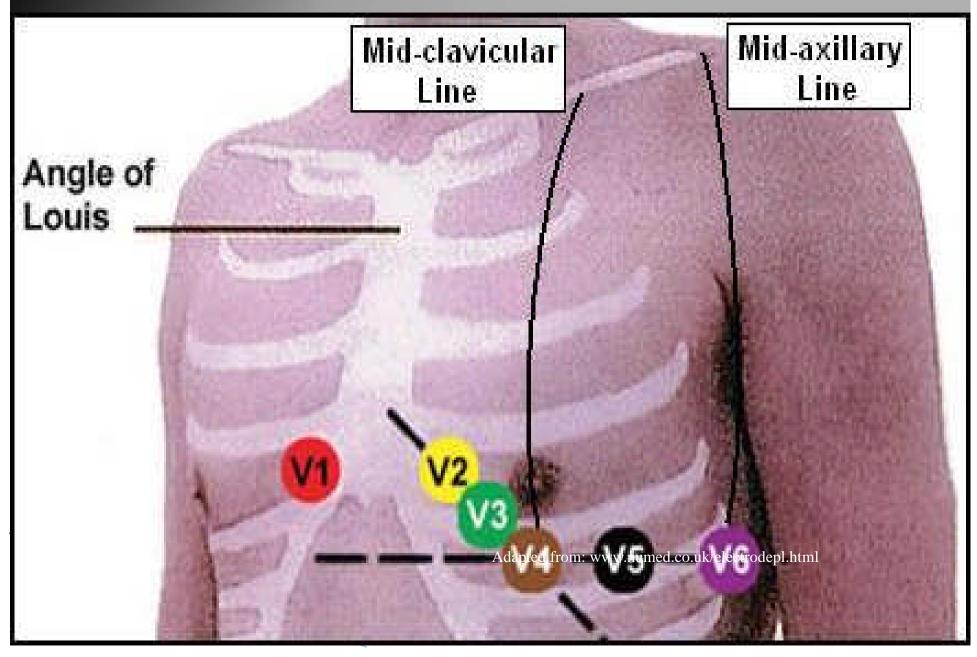


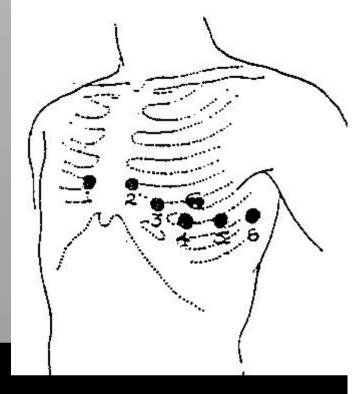
Fig. 1.4 Relationship between the squares on ECG paper and time. Here, there is one QRS complex per second, so the heart rate is 60 beats/min

Q waves

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

Precordial Leads





Lead Placement

V1 = 4th intercostal space, right border of sternum

V2 = 4th intercostal space, left border of sternum

 $\overline{V3} = \overline{\text{midway between V2 and V4}}$

V4 = 5th intercostal space, midclavicular line

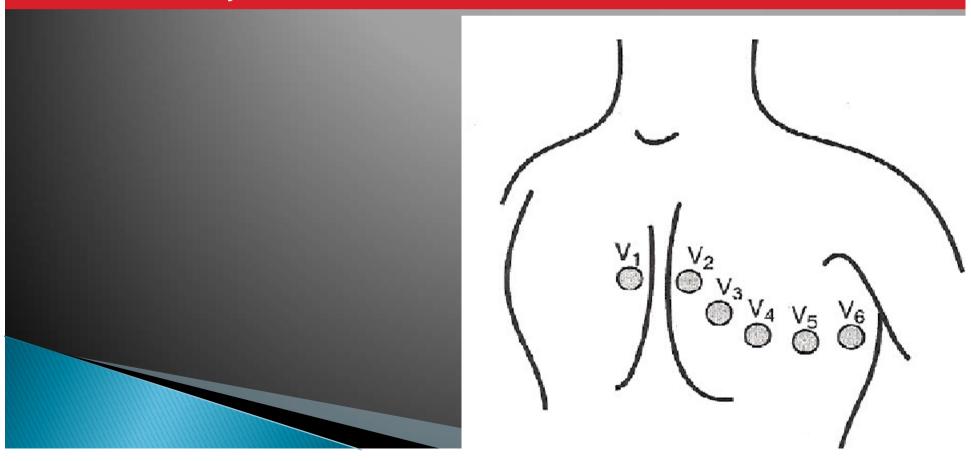
V5 = anteroaxillary line at level of V4

V6 = midaxillary line at level of V4 and V5

Electrocardiography

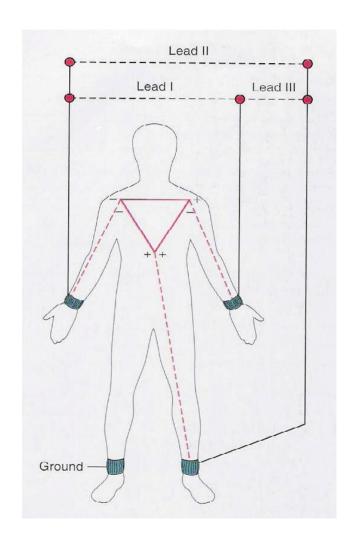


- V1 Right Sternal Border 4th ICS
- V2 Left Sternal Border 4th ICS
- V3 Midway Between V2 and V4
- V4 Midclavicular line 5th ICS
- V5 Anterior Axillary line 5th ICS
- V6 Mid axillary line 5th ICS

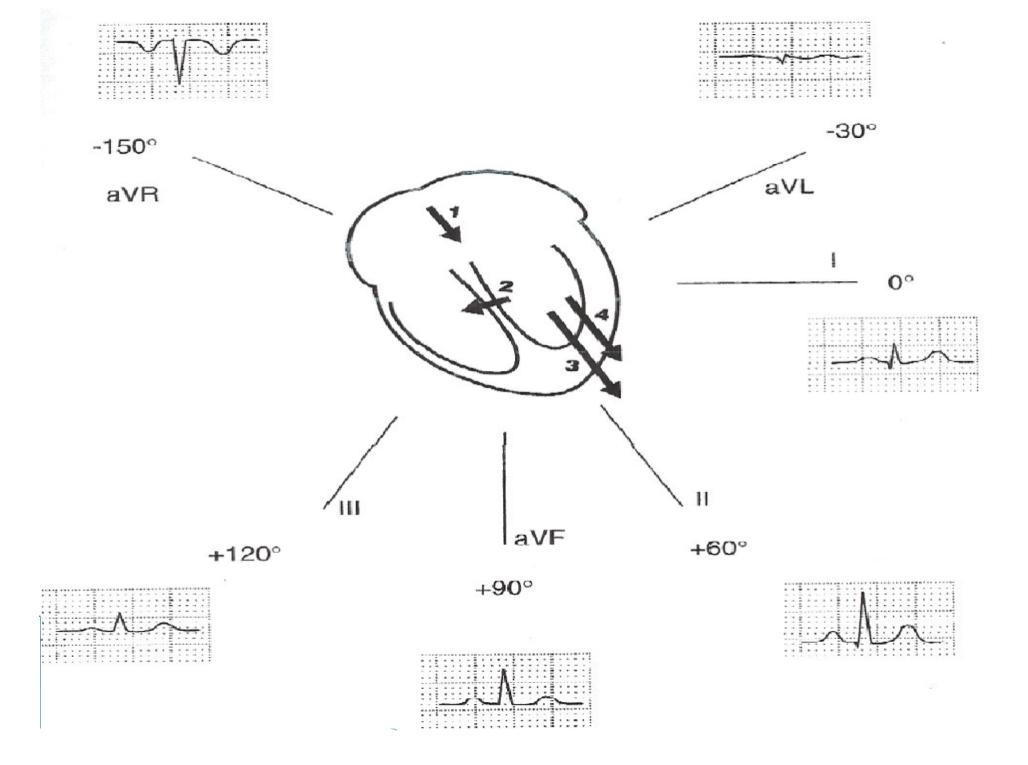


Electrocardiogram

- Einthoven's triangle
 - Three standard limb leads
 - Voltage differences between corners of triangle
 - We will use "Lead II"
 - Right shoulder to left leg



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

3 Bipolar Limb Leads

I = RA vs. LA(+)

II = RA vs. LL(+)

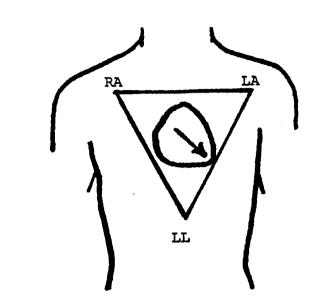
III = LA vs. LL(+)

3 Augmented Limb Leads

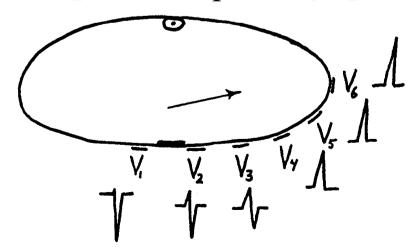
aVR = (LA-LL) vs. RA(+)

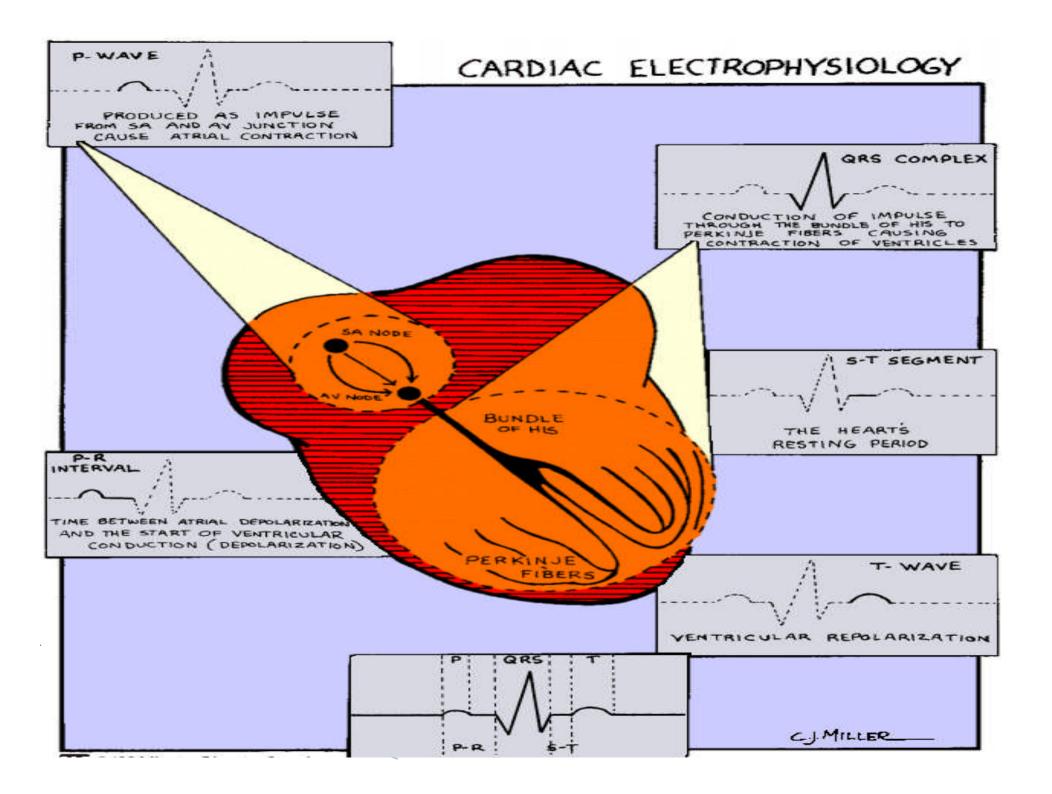
aVL = (RA-LL) vs. LA(+)

aVF = (RA-LA) vs. LL(+)



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





EKG Leads

The standard EKG has 12 leads:

- 3 Standard Limb Leads
- 3 Augmented Limb Leads
- 6 Precordial Leads

The axis of a particular lead represents the viewpoint from which it looks at the heart.

Electrocardiogram

P wave

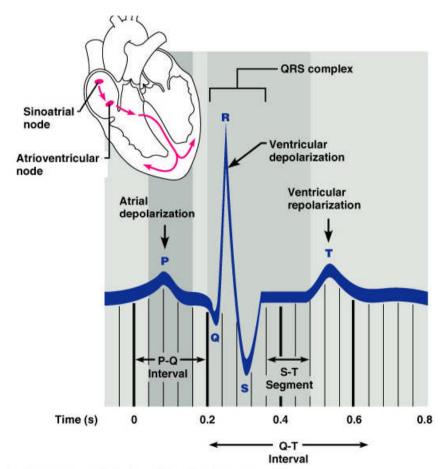
- Depolarization of atria
- Followed by contraction

QRS complex

- 3 waves (Q, R, & S)
- Depolarization of ventricles
- Followed by contraction

T wave

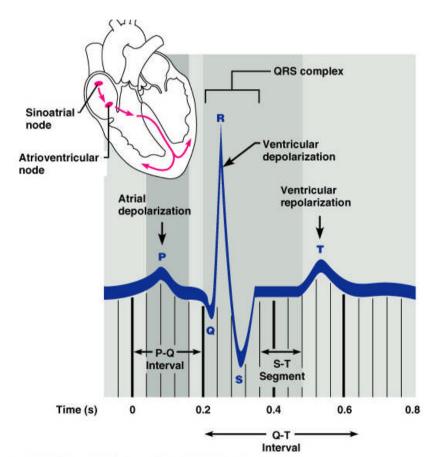
 Repolarization of ventricles



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Electrocardiogram

- ▶ P-Q interval
 - Time atria depolarize & remain depolarized
- Q-T interval
 - Time ventricles depolarize & remain depolarized



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Intervals

- P wave atrial depolarization
- **PR interval time from sinoatrial** node (S-A) to atrioventricular node (A-Vnode)
- QRS Complex ventricular depolarization
- ST Segment beginning of ventricular repolarization
- T Wave later stages of ventricular repolarization
- U Wave final component of ventricular repolarization
- RR Interval represents the time for one complete cardiac cycle

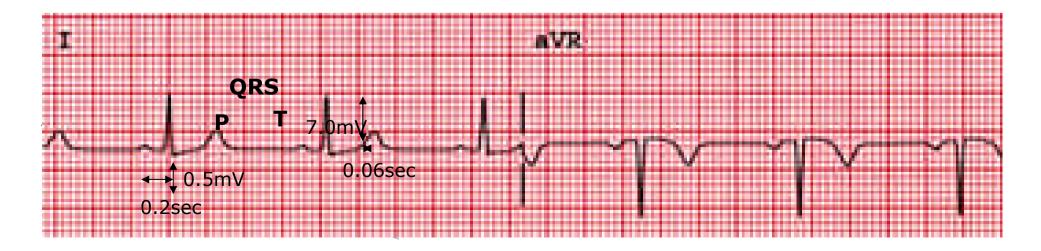
- A positive wave form (QRS mainly above the baseline) results from the wave of depolarization moving towards the positive end of the lead.
 - e.g.
- A negative waveform (QRS mainly below the baseline) is when a wave of depolarization is moving away from the positive electrode (towards the negative end of the lead).
- EKG paper has 1 millimeter small squares – so height and depth of wave is measured in millimeters.

10 mm = 1.0 mVolt

- Horizontal axis is time.
 - 0.04 seconds for 1 mm (1 small box).
 - 0.2 seconds for 1 large box = 5 small boxes = 5 x .04 seconds.

Normal EKG

Positive QRS in Lead I. Negative QRS in Lead aVR. R wave = 7-8 mm high in Lead I = 7-8mV. QRS wave = 0.06 seconds long in Lead I.



Rhythm Summary



Rate

Regularity

P waves

PR interval

QRS duration

Interpretation?

90-95 bpm

regular

normal

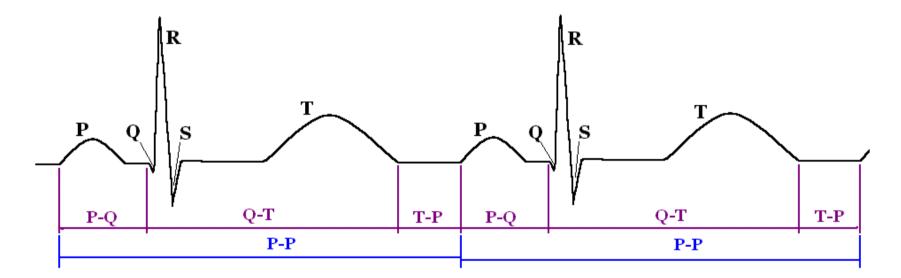
0.12 s

0.08 s

Normal Sinus Rhythm

Electrocardiogram

- Intervals show timing of cardiac cycle
 - P-P = one cardiac cycle
 - P-Q = time for atrial depolarization
 - Q-T = time for ventricular depolarization
 - T-P = time for relaxation



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- What these intervals represents?
- P wave
- PR interval –
- QRS Complex -
- • ST Segment –
- T Wave -
- • U Wave -
- RR Interval -

- Intervals
- P wave atrial depolarization
- PR interval time from sinoatrial node (S-A) to atrioventricular node (A-Vnode)
- QRS Complex ventricular depolarization
- ST Segment beginning of ventricular repolarization
- T Wave later stages of ventricular repolarization
- • U Wave final component of ventricular repolarization
- RR Interval represents the time for one complete cardiac cycle

- Lead Placement
- ▶ V1 =
- ▶ V2 =
- ▶ V3 =
- ▶ V4 =
- ▶ V5 =
- ▶ V6 =

- Lead Placement
- V1 = 4th intercostal space, right border of sternum
- V2 = 4th intercostal space, left border of sternum
- V3 = midway between V2 and V4
- V4 = 5th intercostal space, midclavicular line
- V5 = anteroaxillary line at level of V4
- V6 = midaxillary line at level of V4 and V5

Anatomic Groups (Septum)

l	aVR	V ₁	V ₄
Lateral	None	Septal	Anterior
II	aVL	V ₂	V ₅
Inferior	Lateral	Septal	Lateral
III	aVF	V ₃	V ₆
Inferior	Inferior	Anterior	Lateral

Anatomic Groups (Anterior Wall)

l	aVR	V ₁	V ₄
Lateral	None	Septal	Anterior
II	aVL	V ₂	V ₅
Inferior	Lateral	Septal	Lateral
III	aVF	V ₃	V ₆
Inferior	Inferior	Anterior	Lateral

Anatomic Groups (Lateral Wall)

l	aVR	V ₁	V ₄
Lateral	None	Septal	Anterior
II	aVL	V ₂	V ₅
Inferior	Lateral	Septal	Lateral
III	aVF	V ₃	V ₆
Inferior	Inferior	Anterior	Lateral

Anatomic Groups (Inferior Wall)

l	aVR	V ₁	V ₄
Lateral	None	Septal	Anterior
II	aVL	V ₂	V ₅
Inferior	Lateral	Septal	Lateral
III	a∨F	V ₃	V ₆
Inferior	Inferior	Anterior	Lateral

Anatomic Groups (Summary)

l	a∀R	V ₁	V ₄
Lateral	None	Septal	Anterior
II	aVL	V ₂	V ₅
Inferior	Lateral	Septal	Lateral
III	a∀F	V ₃	V ₆
Inferior	Inferior	Anterior	Lateral

Rule of 300

Take the number of "big boxes" between neighboring QRS complexes, and divide this into 300. The result will be approximately equal to the rate

Although fast, this method only works for regular rhythms.

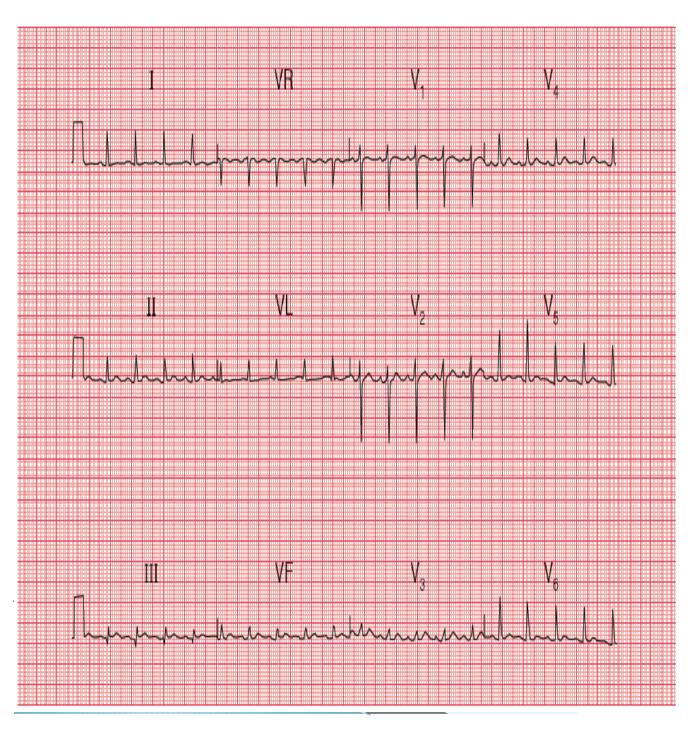


Fig. 1.23 12-lead ECG: example 1

Description

- Sinus rhythm, rate 110/min
- Normal PR interval (140 ms)
- Normal QRS duration (120 ms)
- Normal cardiac axis
- Normal QRS complexes
- Normal T waves (an inverted T wave in lead VR is normal)

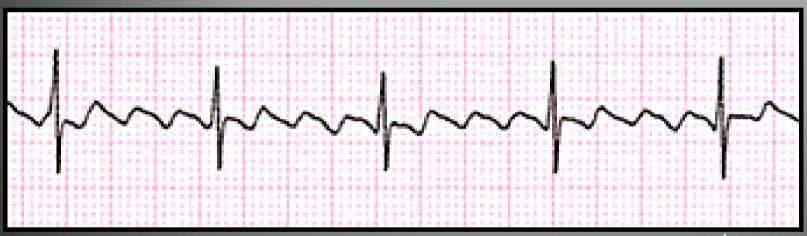
Interpretation

Normal ECG



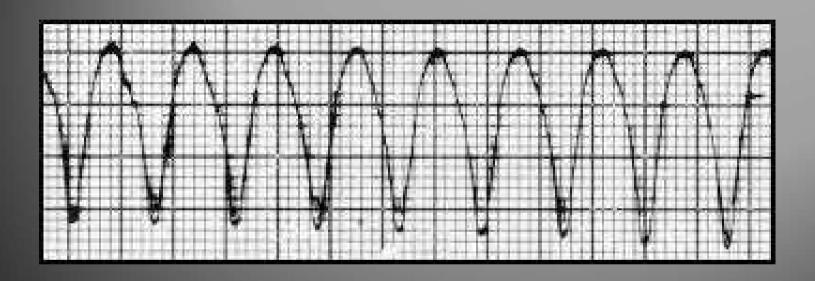
www.uptodate.com

(300 / 6) = 50 bpm



www.uptodate.com

 $(300 / \sim 4) = \sim 75 \text{ bpm}$



(300 / 1.5) = 200 bpm

The Rule of 300

It may be easiest to memorize the following table:

# of big boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50

10 Second Rule

As most EKGs record 10 seconds of rhythm per page, one can simply count the number of beats present on the EKG and multiply by 6 to get the number of beats per 60 seconds.

This method works well for irregular rhythms.

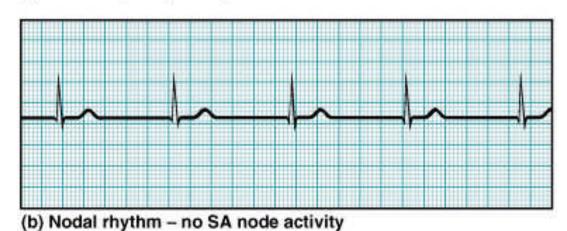


The Alan E. Lindsay ECG Learning Center; http://medstat.med.utah.edu/kw/ecg/

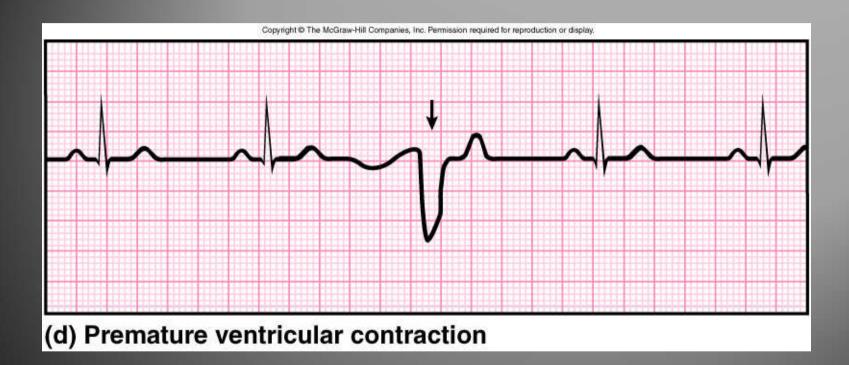
 $33 \times 6 = 198 \text{ bpm}$

ECGs, Normal and Abnormal



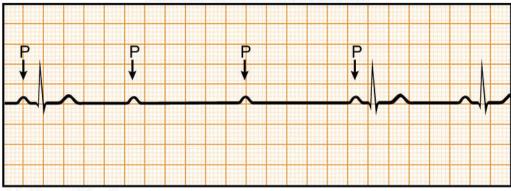


ECGs, Abnormal



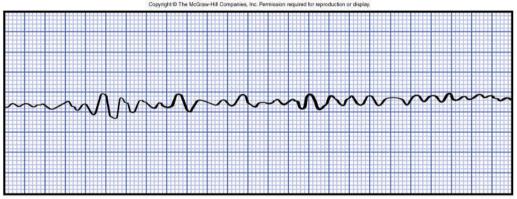
Extrasystole: note inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.

ECGs, Ahnormal



(c) Heart block

Arrhythmia: conduction failure at AV node



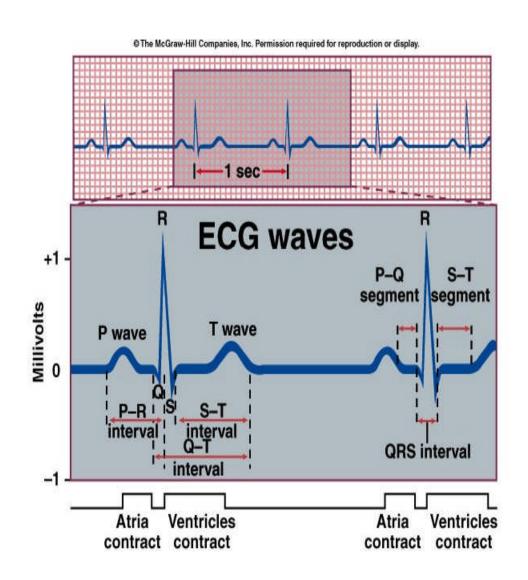
(e) Ventricular fibrillation

No pumping action occurs

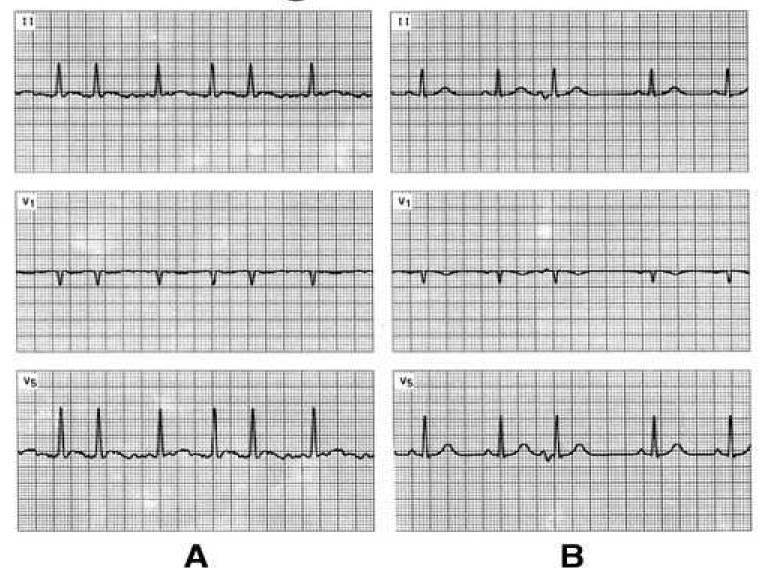
Interpretation of ECG

Normal Sinus Rhythm

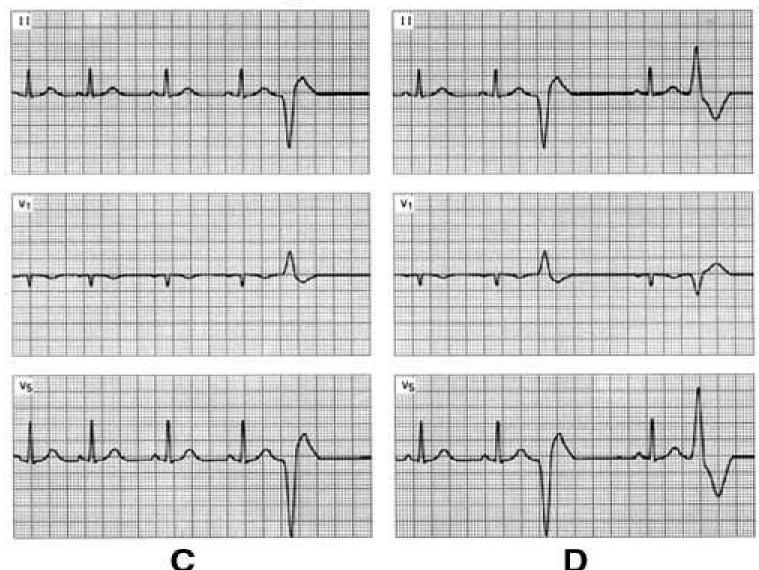
- ▶ Rate: 60–100 b/min
- Rhythm: regular
- P waves: upright in leads I, II, aV_F
- PR interval: < .20 s</p>
- ightharpoonup QRS: < .10 s



Irregular ECGs

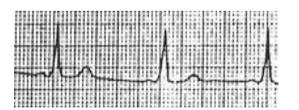


Irregular ECGs



PR interval

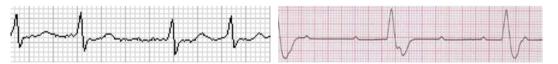
< 0.12 s	0.12-0.20 s	> 0.20 s
High catecholamine states Wolff-Parkinson-White	Normal	AV nodal blocks





QRS complex

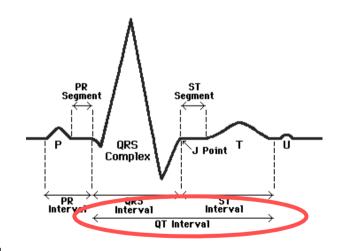
≤ 0.10 s	0.10-0.12 s	> 0.12 s
Normal	Incomplete bundle branch block	Bundle branch block PVC Ventricular rhythm



QT interval

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

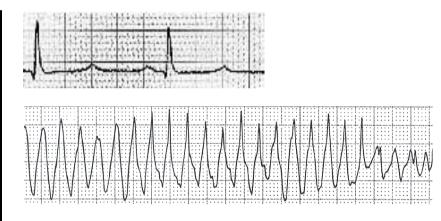
The faster the heart beats, the faster the ventricles repolarize so the shorter the QT interval. Therefore what is a "normal" QT varies with the heart rate. For each heart rate you need to calculate an adjusted QT interval, called the "corrected QT" (QTc):

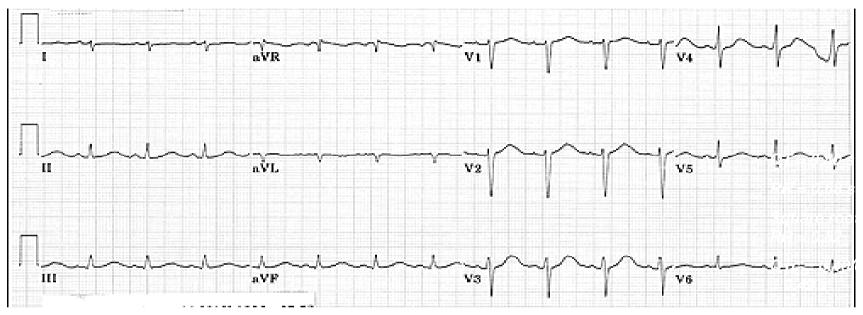


QTc = QT / square root of RR interval

QTc interval

< 0.44 s	> 0.44 s
Normal	Long QT





PR interval?

0.16 seconds

QRS 0.08 width? seconds

0.08 QTc 0.49 seconds interval? seconds

Interpretation of intervals?

Normal PR and QRS, long

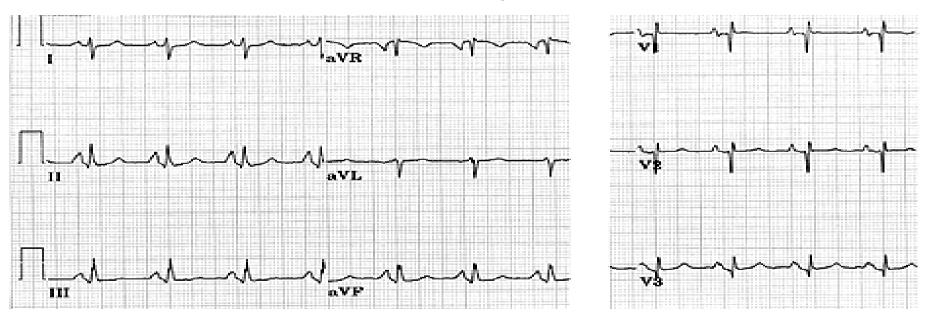
QT

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

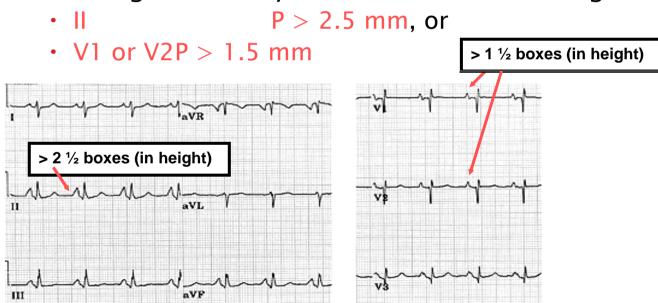
• Take a look at this ECG. What do you notice about the P waves?



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

Right atrial enlargement

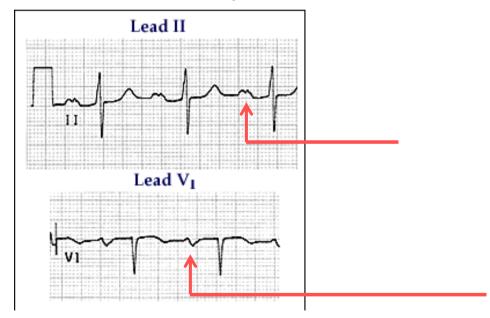
To diagnose RAE you can use the following criteria:



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?

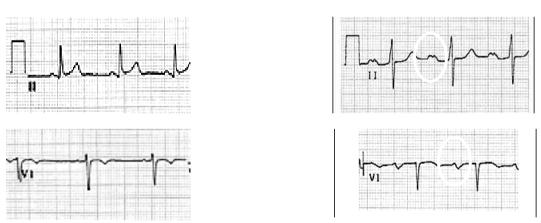


The P waves in lead II are notched and in lead V1 they have a deep and wide negative component.

Left atrial enlargement

- To diagnose LAE you can use the following criteria:
 - > 0.04 s (1 box) between notched peaks, or
 - V1

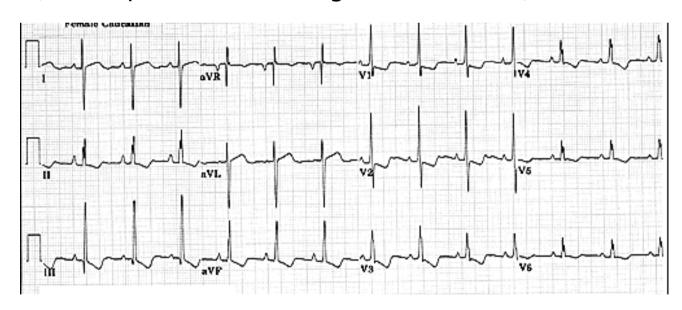




A common cause of LAE is LVH from hypertension.

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.

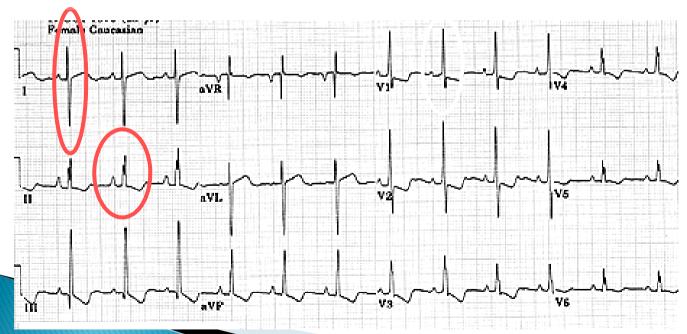
Right ventricular hypertrophy

- 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

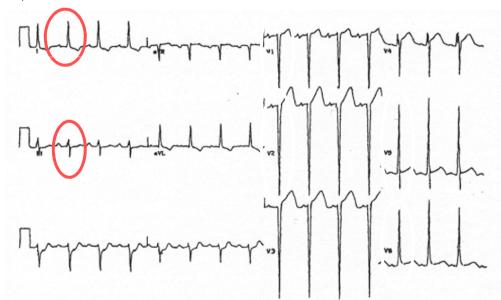
- To diagnose RVH you can use the following criteria:
 - Right axis deviation, and
 - V1 R wave > 7mm tall



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)?

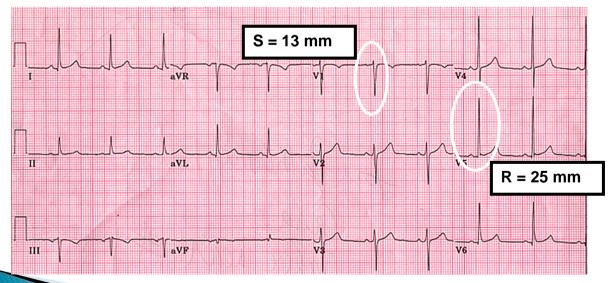


There is left axis deviation (positive in I, negative in II) and there are tall R waves in V5, V6 and

about Charles in 1/1 1/2

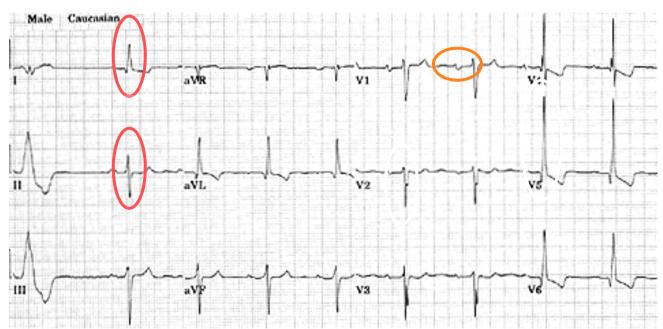
Left ventricular hypertrophy

- 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



A common cause of LVH is hypertension.

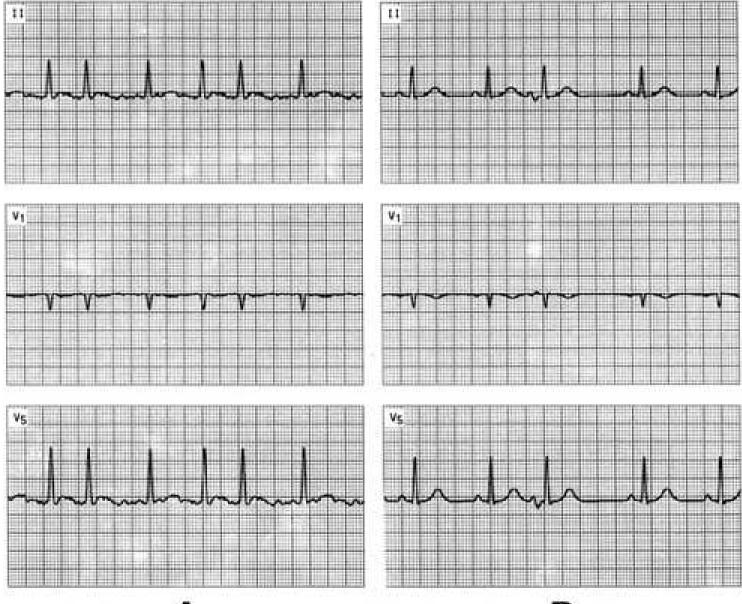
A 63 yo man has longstanding, uncontrolled hypertension. Is there evidence of heart disease from his hypertension? (Hint: There a 3 abnormalities.)



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

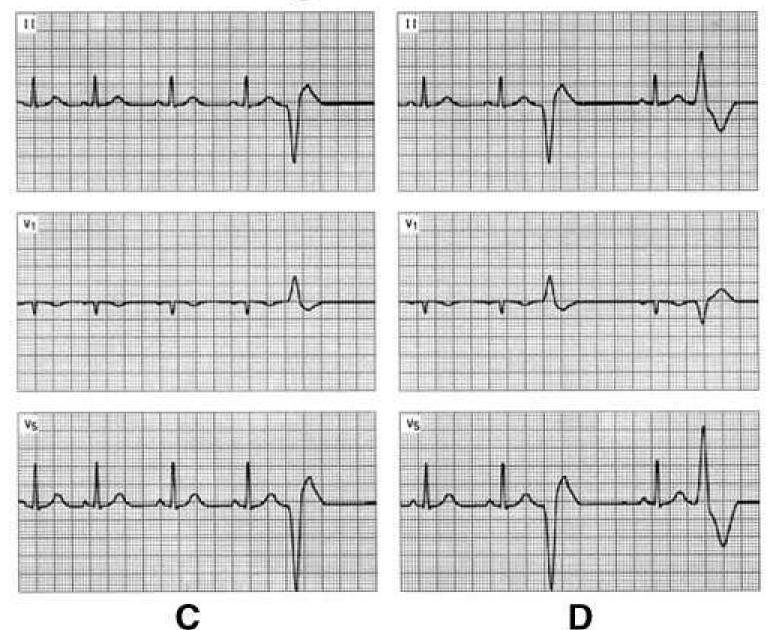
Irregular ECGs

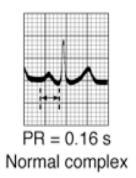


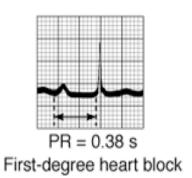
A

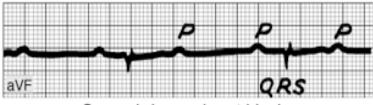
В

Irregular ECGs

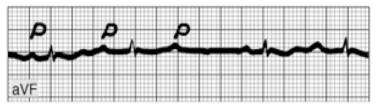




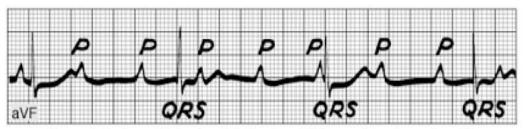




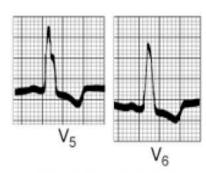
Second-degree heart block (2:1 heart block)



Second-degree heart block (Wenckebach phenomenon)



Complete heart block. Atrial rate, 107; ventricular rate, 43



Two V leads in left bundle branch block

Figure 28-11. Heart block.

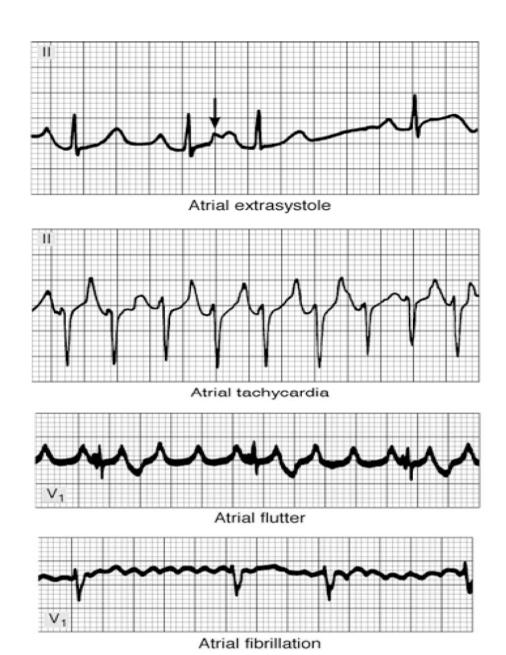


Figure 28-13. Atrial arrhythmias. The illustration shows an atrial premature beat with its P wave superimposed on the T wave of the preceding beat (arrow); atrial tachycardia; atrial flutter with 4:1 AV block; and atrial fibrillation with a totally irregular ventricular rate. (Tracings reproduced, with 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.)

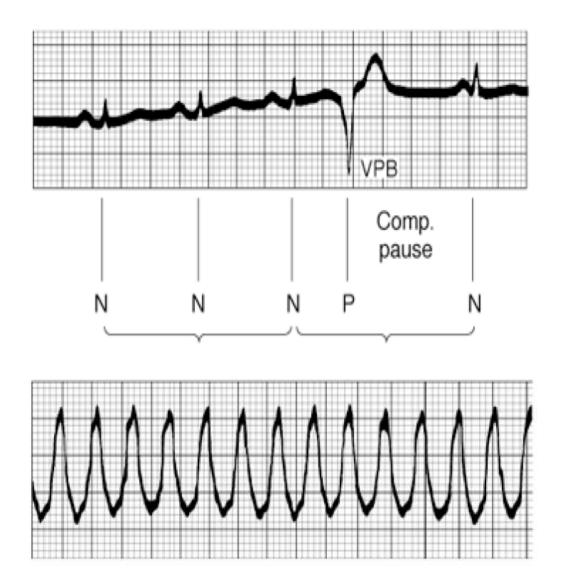


Figure 28-14. Top: Ventricular premature beats (VPB). The lines under the tracing illustrate the compensatory pause and show that the duration of the premature beat plus the preceding normal beat is equal to the duration of two normal beats. **Bottom:** Ventricular tachycardia.

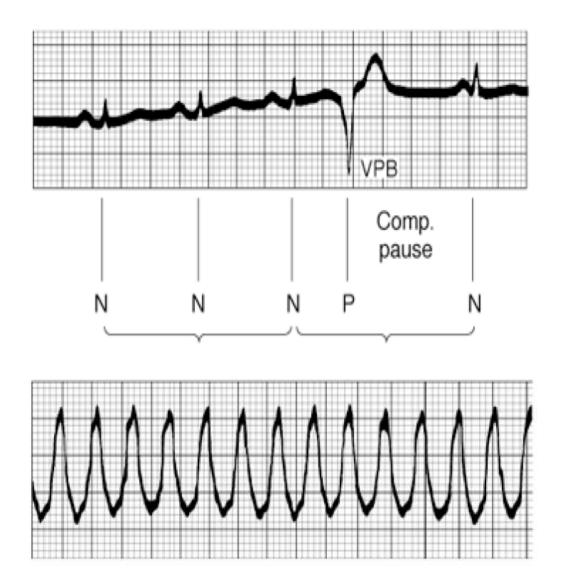
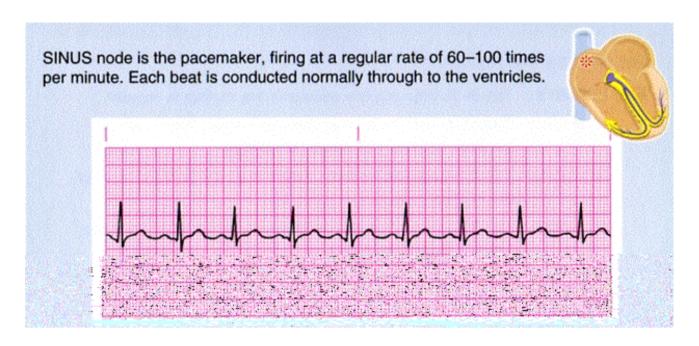
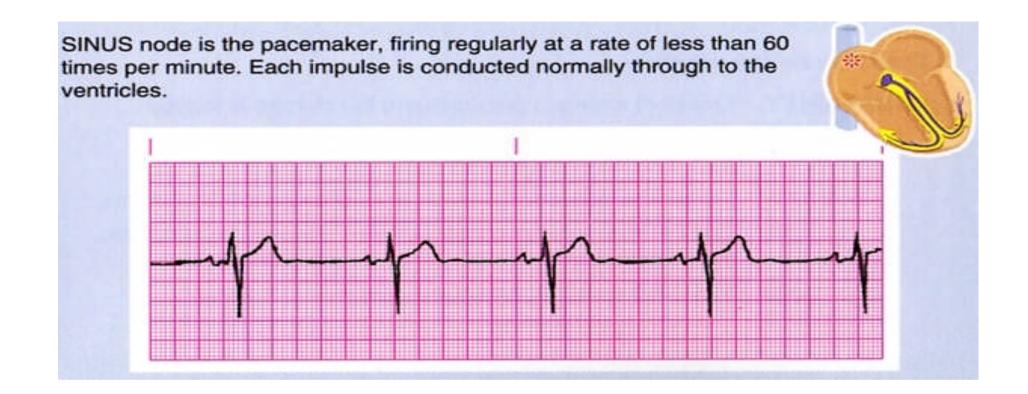


Figure 28-14. Top: Ventricular premature beats (VPB). The lines under the tracing illustrate the compensatory pause and show that the duration of the premature beat plus the preceding normal beat is equal to the duration of two normal beats. **Bottom:** Ventricular tachycardia.





Normal Sinus Rhythm



Sinus 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.



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.

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.

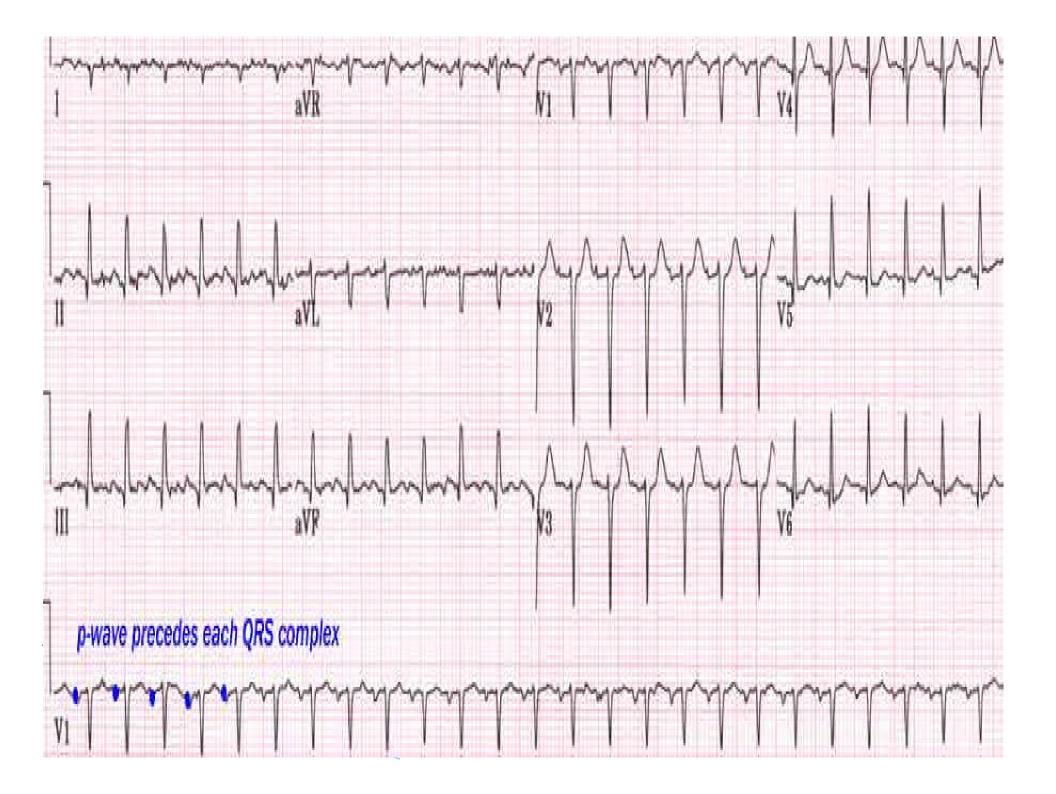
Here there is a focus other than the SA node that is firing, which causes a QRS.

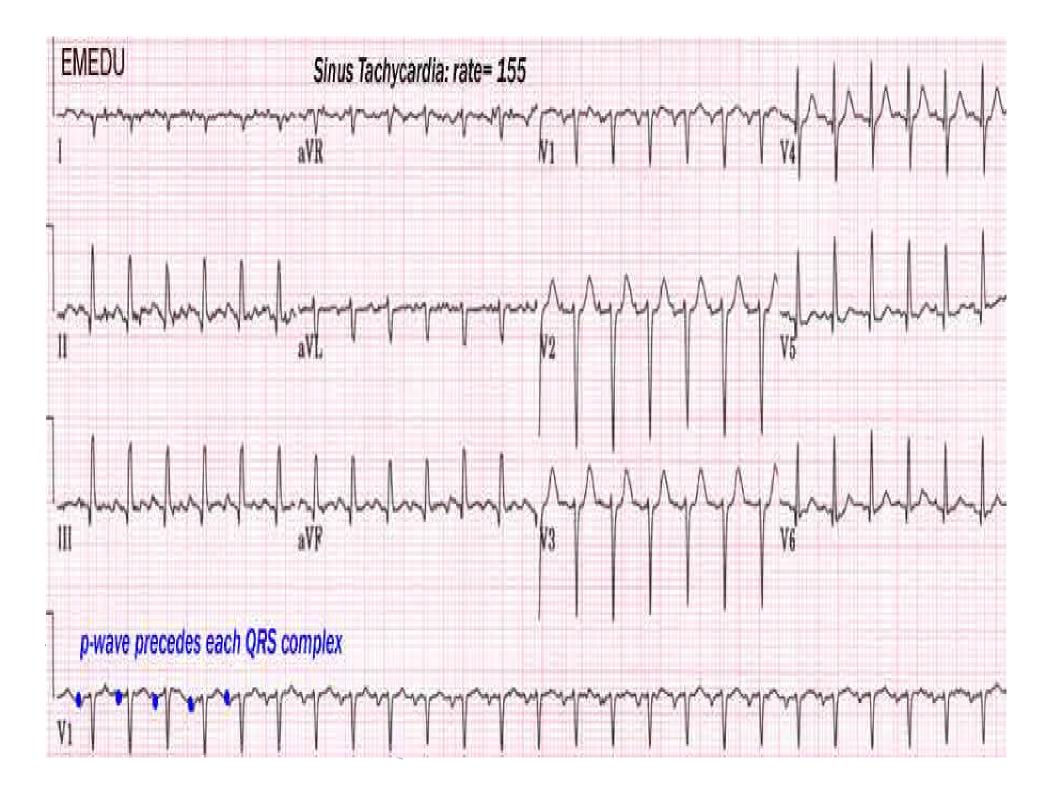
What is they underlying rate? 7*10 using 6-second. Or, 75 using 4 big boxes per QRS.

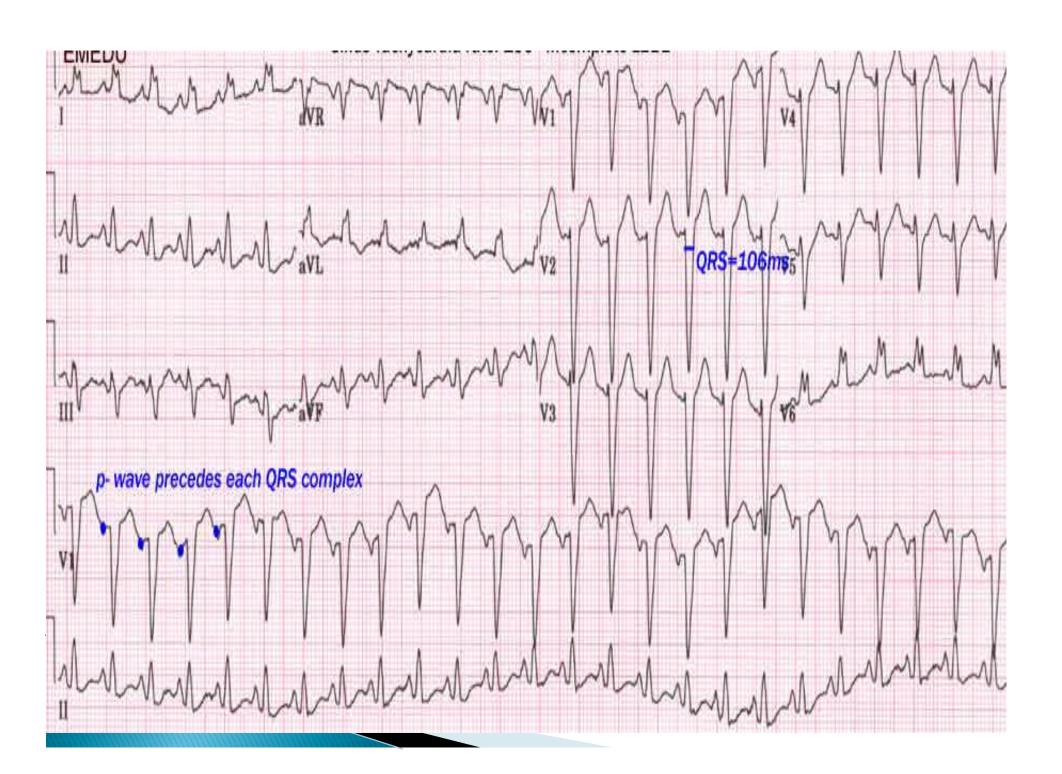
The PAC is the third QRS; you can see there is only about 1 big box between the 2nd and 3rd QRS. Usually the abnormal focus will cause a p-wave the looks different from the other p-waves (the SA node p-waves).

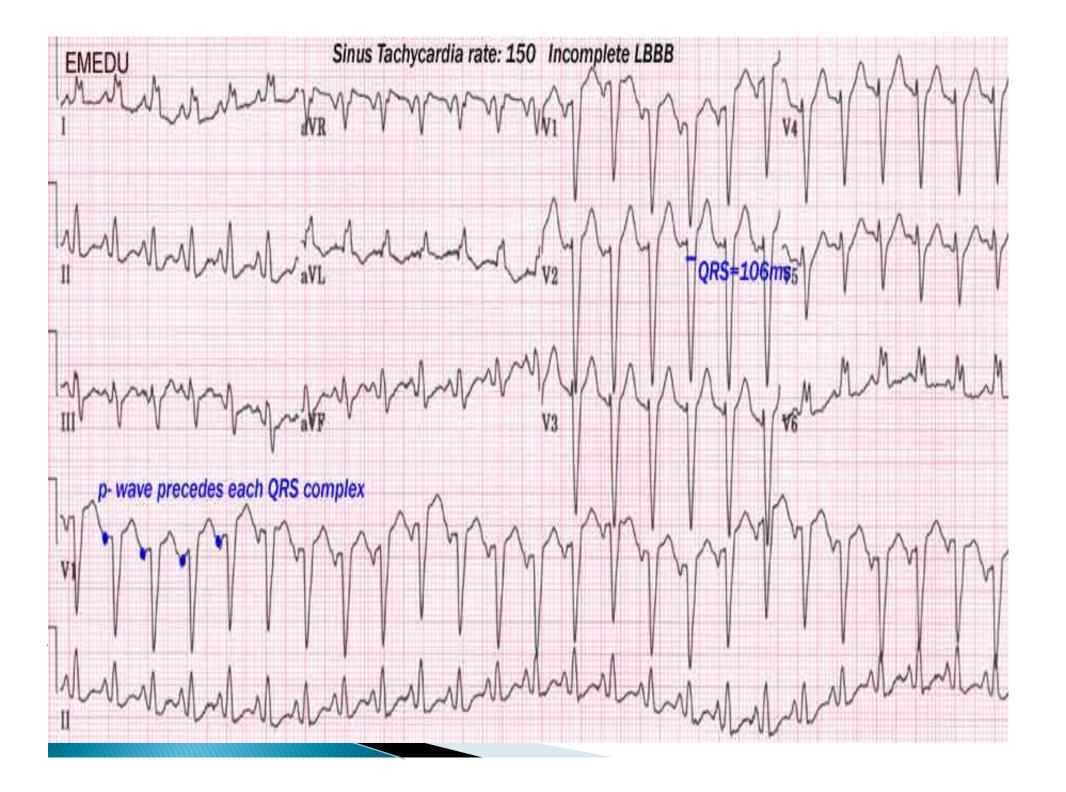
Here the aberrant atrial depolarization appears as a peaked p-wave in comparison.

Premature Atrial Contraction









What do you think?



A: normal

B: sinus tachycardia

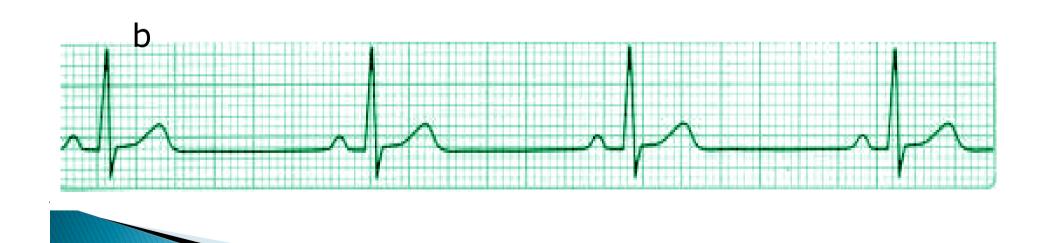




What do you think?

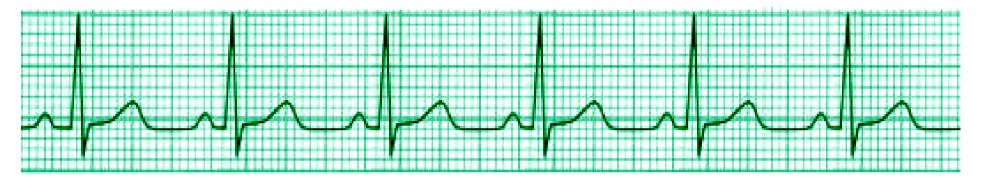
a





A: normal

a B:sinus bradycardia









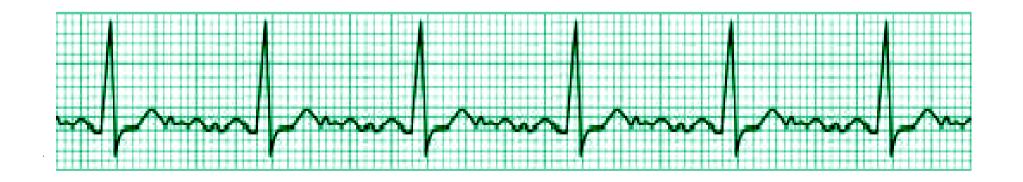
Normal



Atrial Flutter



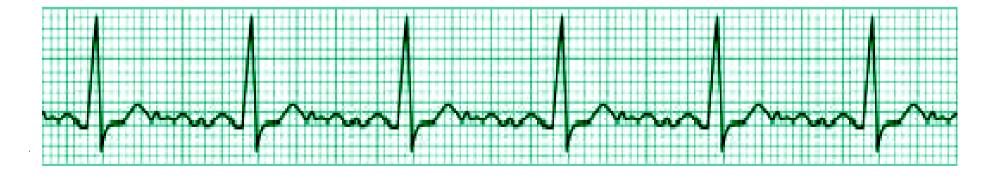


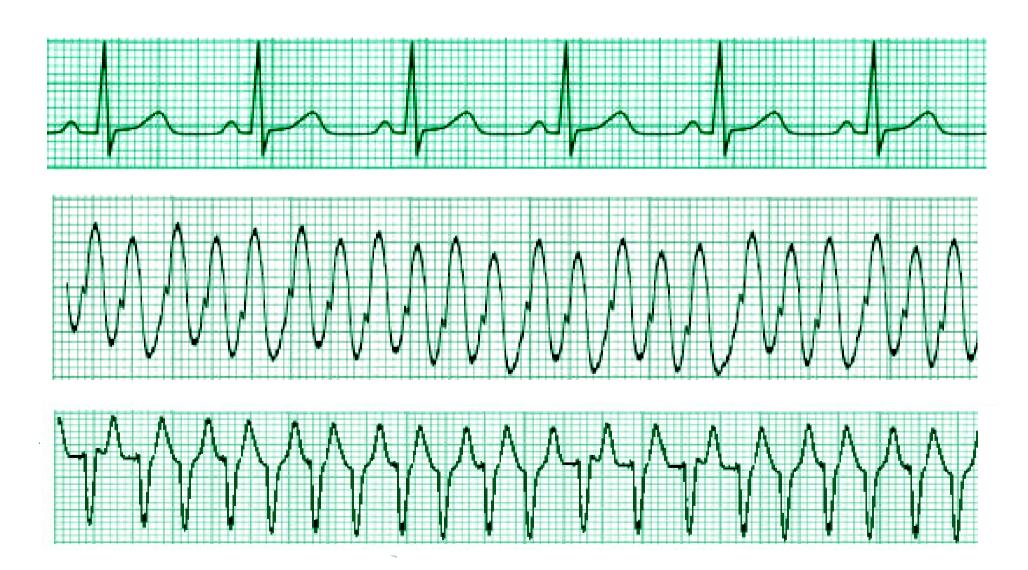


Normal



Atrial Fibrillation

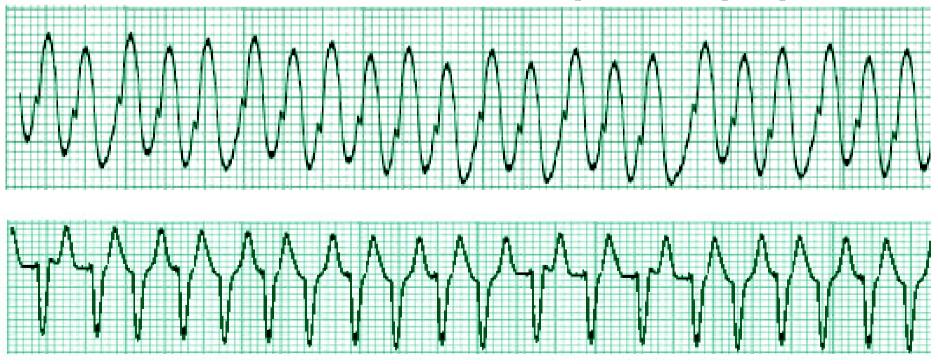


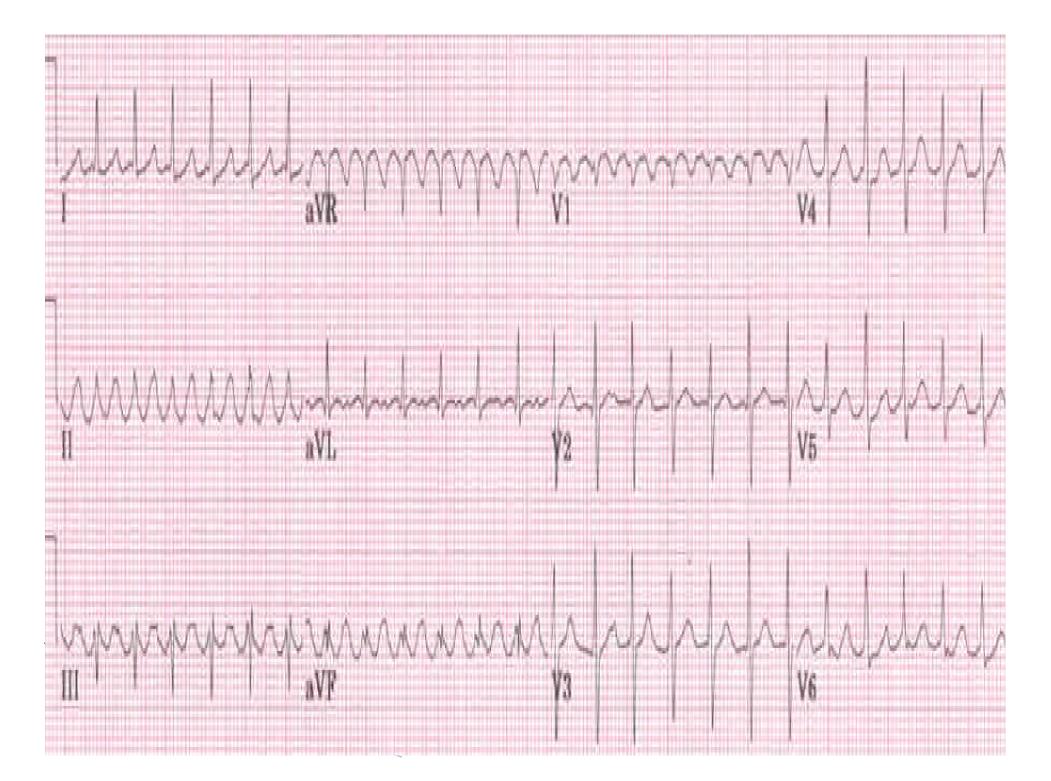


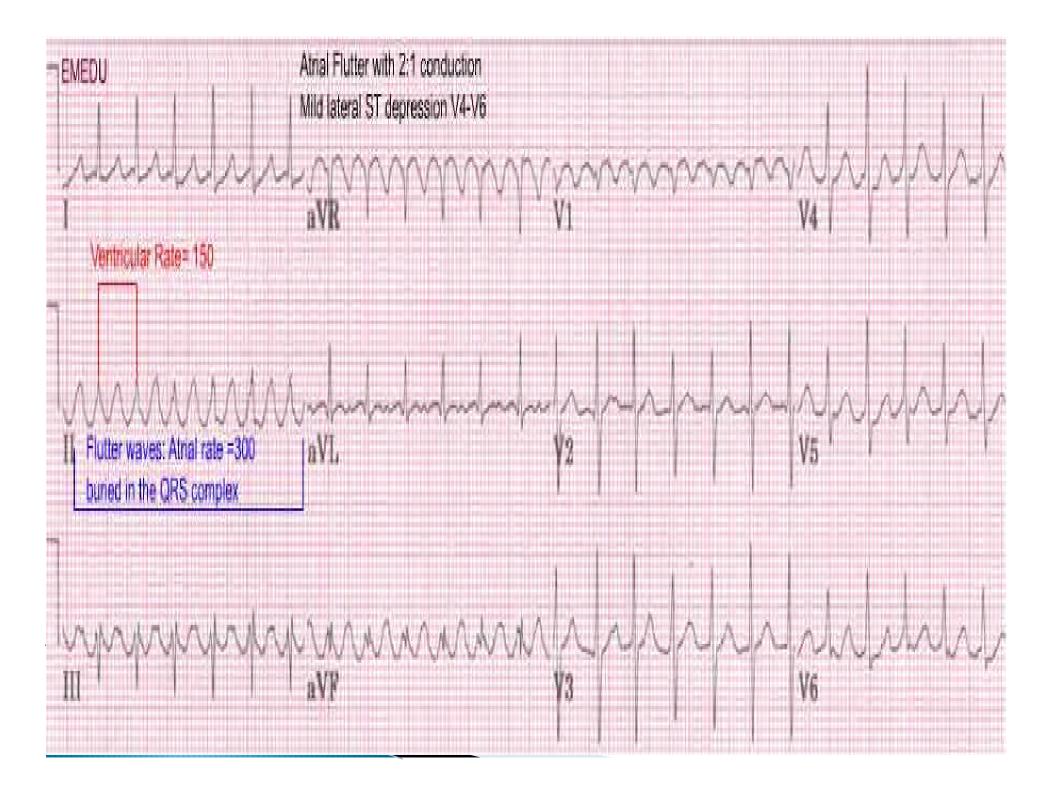
Normal



Ventricular Tachycardia (VT)









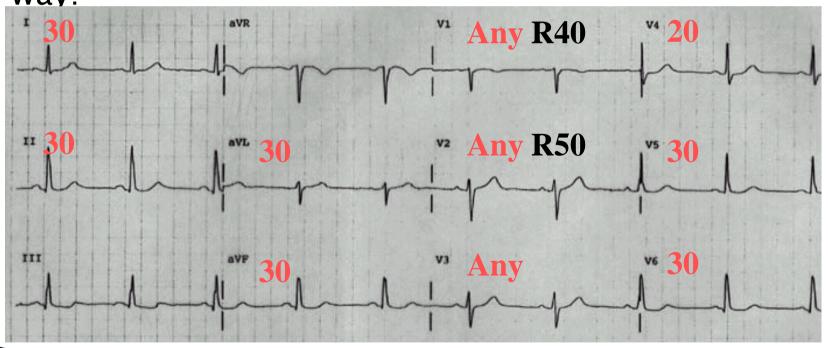
Rate Rhythm Axis Intervals Hypertrophy Infarct

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

Rate Rhythm Axis Intervals Hypertrophy Infarct

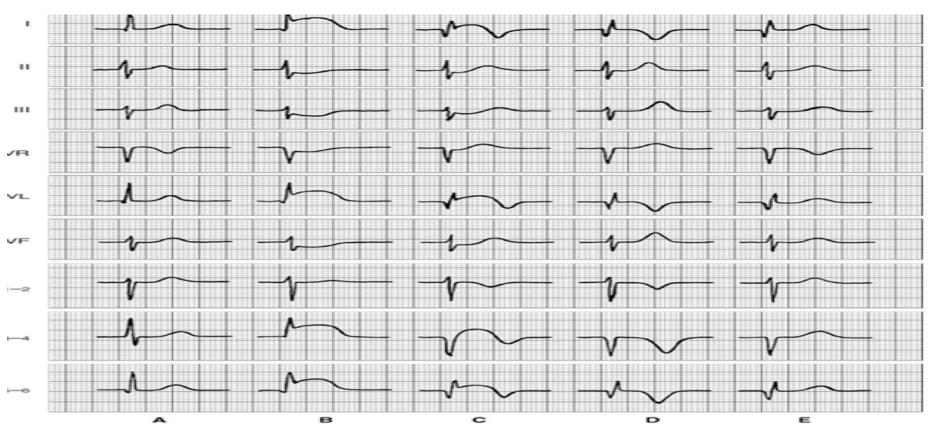
This mantra corresponds to the ECG in the following way:



SUMMARY Rate Rhythm Axis Intervals Hypertrophy Infarct

To summarize:

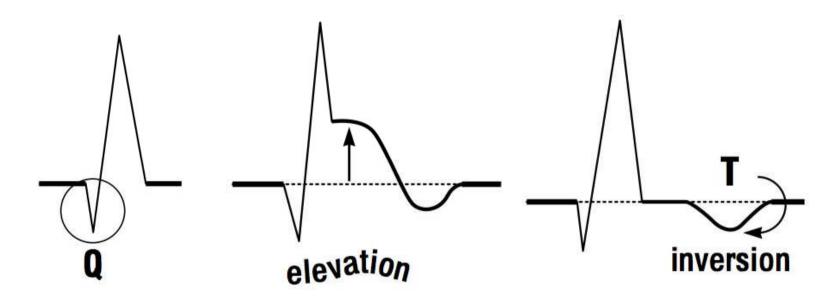
- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
 - _
 - _
 - _



A: Normal tracing. **B:** Very early pattern (hours after infarction): ST segment elevation in I, aVL, and V_{3-6} ; 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

Anterior

Anterolateral

Lateral

High lateral

Inferior

Inferolateral

True posterior

Leads

 $I, V_2, V_3, \text{ and } V_4$

I, aVL, V5, and V6

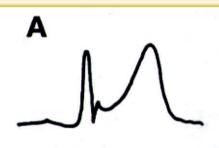
V₅ and V₆

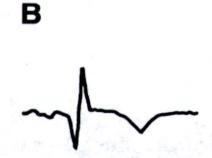
I and aVL (often with V₅, V₆)

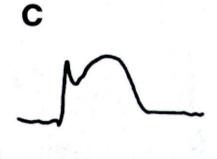
II, III, and aVF

II, III, aVF, and V6

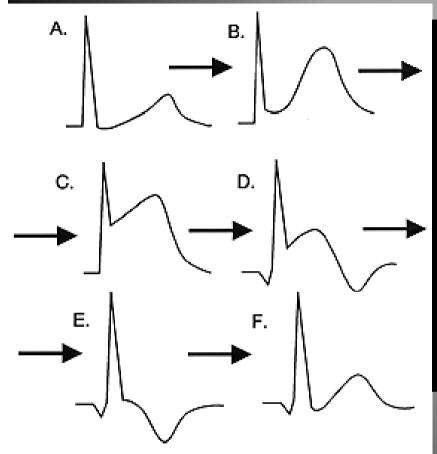
Reciprocal changes in V₁ and V₂







MI Location



Evolution of Acute MI

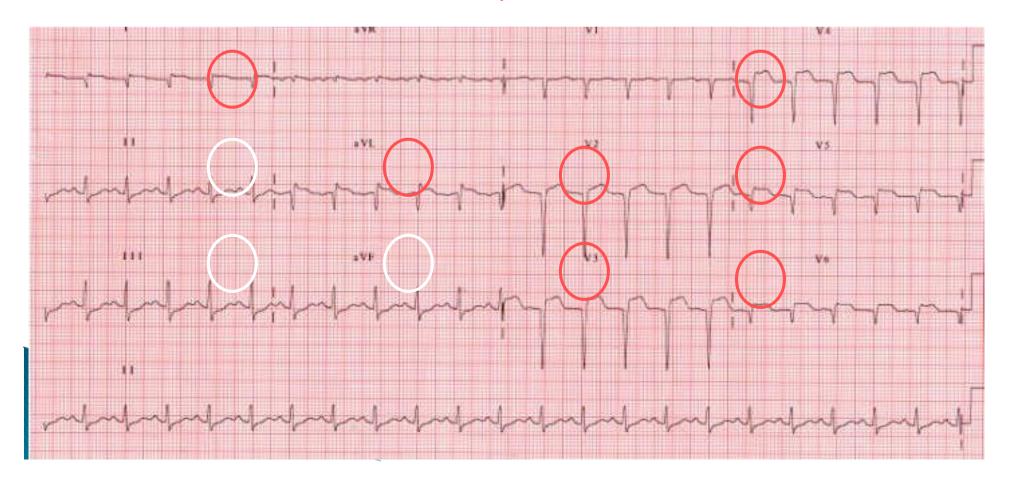
Myocardial involvement EKG leads Anterior V2, V3, V4 (at least 2) Anteroseptal V1, V2, V3 (+V4) **Anterolateral V4, V5, V6 (+V3, +V2) Extensive Anterior** V1 through V6 (all) Lateral **V5**, **V6** (+**I**, +**aVL**) **High lateral** I, aVL **Inferior** II, III, aVF (at least 2) Inferolateral as above, +V6 (+V5) **Posterior** V1, V2 (*recip. changes) **Inferoposterolateral Combine above 3 items Right Ventricular** V4R, +V3R and/or

V5R

SUMMARY Rate Rhythm Axis Intervals Hypertrophy Infarct

Infarct: Is the ST elevation or depression?

Yes! Elevation in V2-V6, I and avL. Depression in II, III and avF.



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

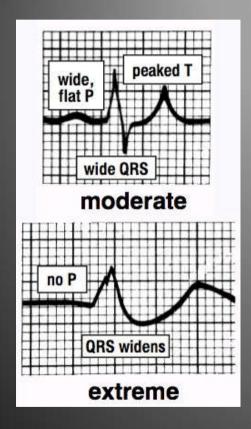
DEPRESSED ST

- 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
- Toxicity of digitalis, quinidine

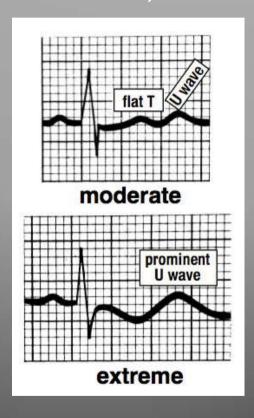
ST Segment Depression

Elexperior & Druglypokalemia

- High K+
- Peaked T



- Low K+
- Flat T, U Wave





Normal tracing (plasma K⁺ 4–5.5 meg/L). PR interval = 0.16 s; QRS interval = 0.06 s; QT interval = 0.4 s (normal for an assumed heart rate of 60).

Hyperkalemia (plasma K+ ±7.0 meg/L). The PR and QRS intervals are within normal limits. Very tall, slender peaked T waves are now present.



Hyperkalemia (plasma K+ ±8.5 meg/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+ ± 3.5 meg/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.



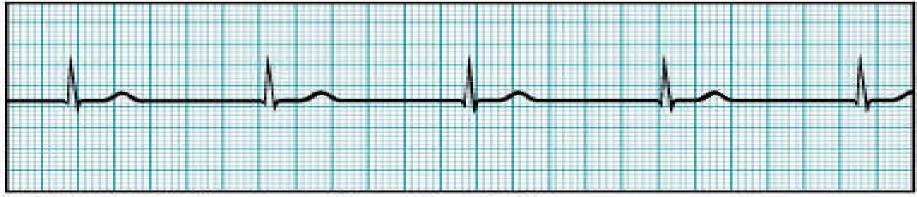
Hypokalemia (plasma K⁺ ±2.5 meg/L). The PR interval is lengthened to 0.32 s; the ST segment is depressed; the T wave is inverted; a prominent U wave is seen. The true QT interval remains normal.

Figure 28-20. Correlation of plasma K^+ level and the ECG, assuming that the plasma Ca^{2+} 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.)

ECGs, Normal and Abnormal



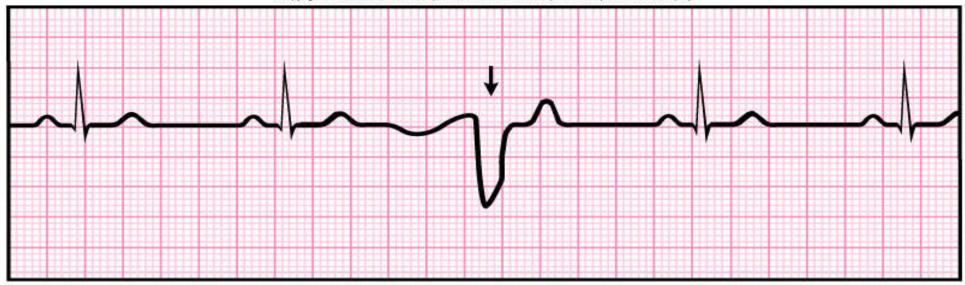
(a) Sinus rhythm (normal)



(b) Nodal rhythm - no SA node activity

ECGs, Abnormal

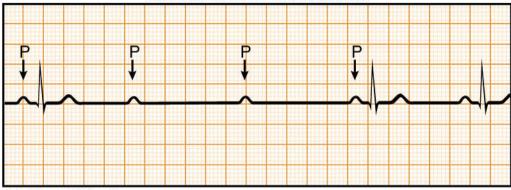
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(d) Premature ventricular contraction

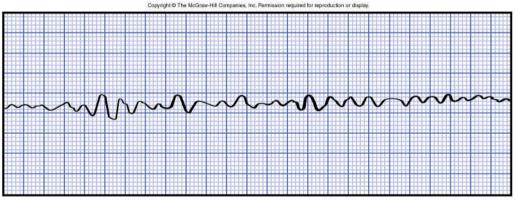
Extrasystole: note inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.

ECGs, Ahnormal



(c) Heart block

Arrhythmia: conduction failure at AV node



(e) Ventricular fibrillation

No pumping action occurs