

# The normal and abnormal 2024 UPDATE

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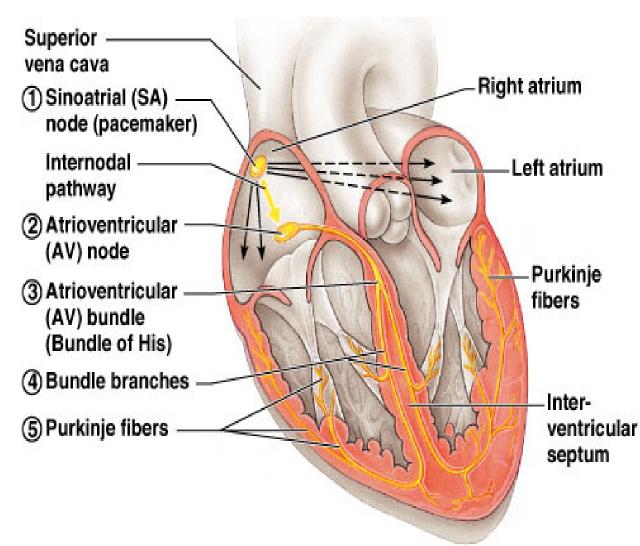
# The Cardiac Cycle

- 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

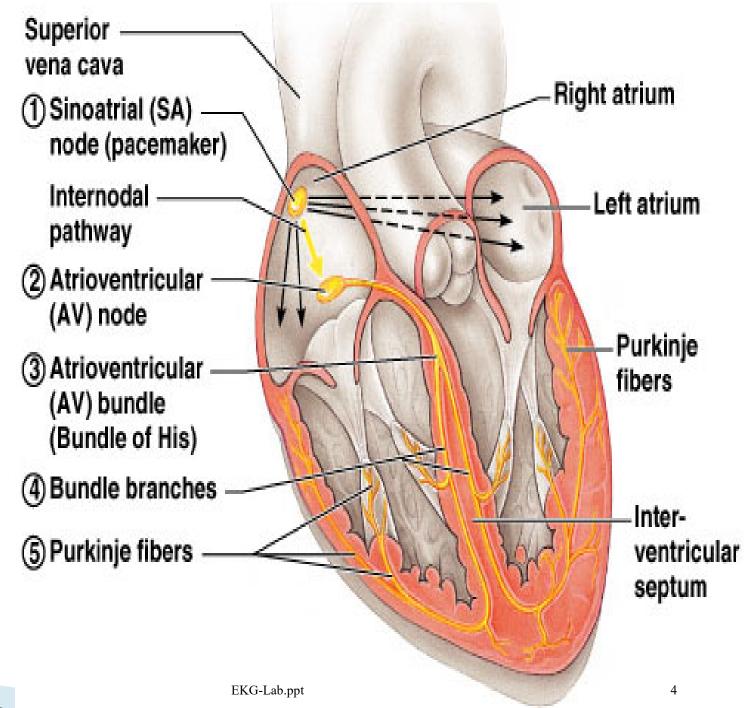
Conduction

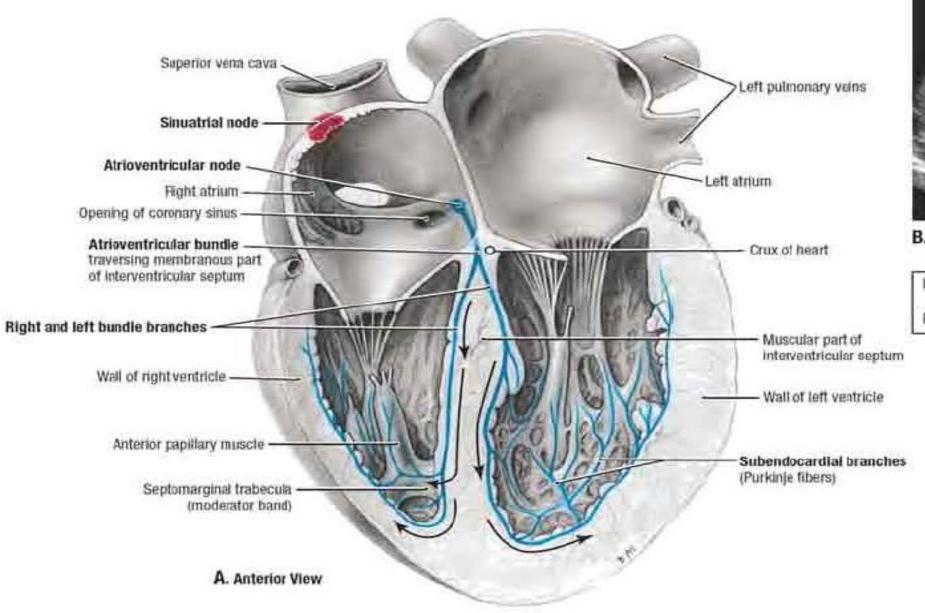
- Heart is autorhythmic
- Depolarization begins in sinoatrial (SA) node
- Spread through atrial myocardium
- Delay in atrioventricular (AV) node

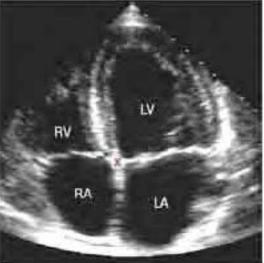


# Depolarization and Impulse Conduction

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

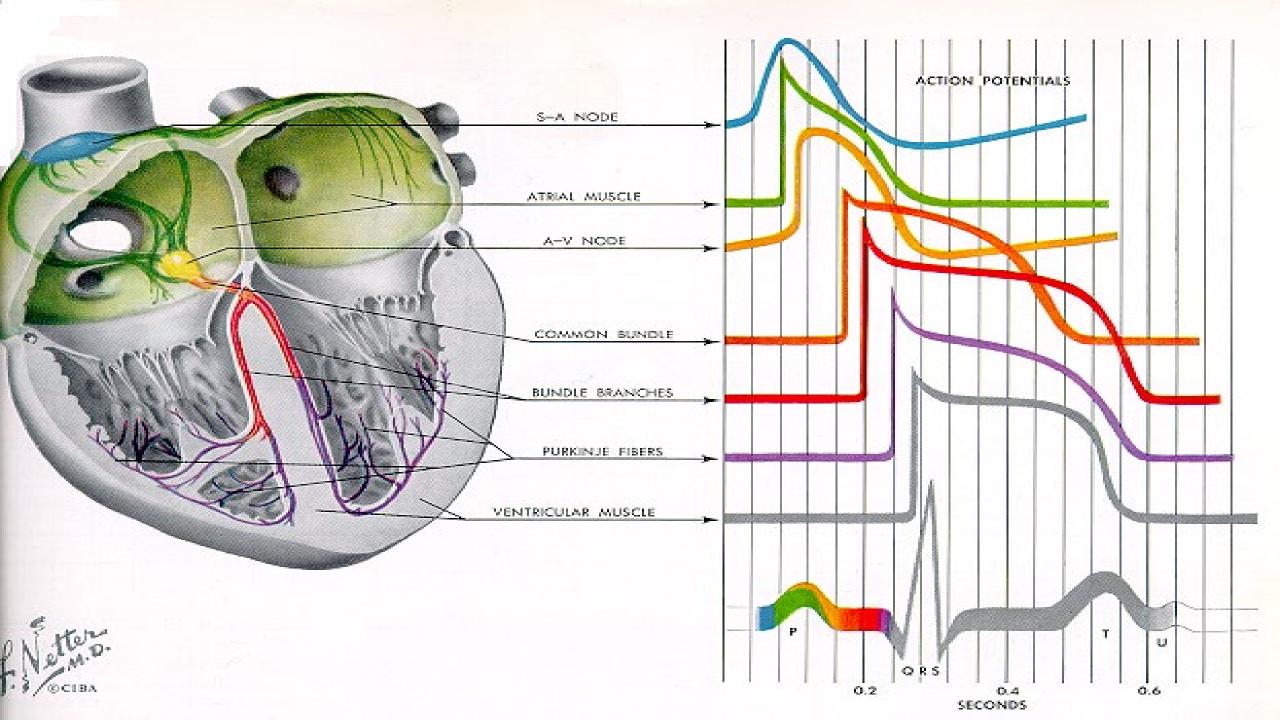






B. Echocardiogram, Apical Four-chamber View

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



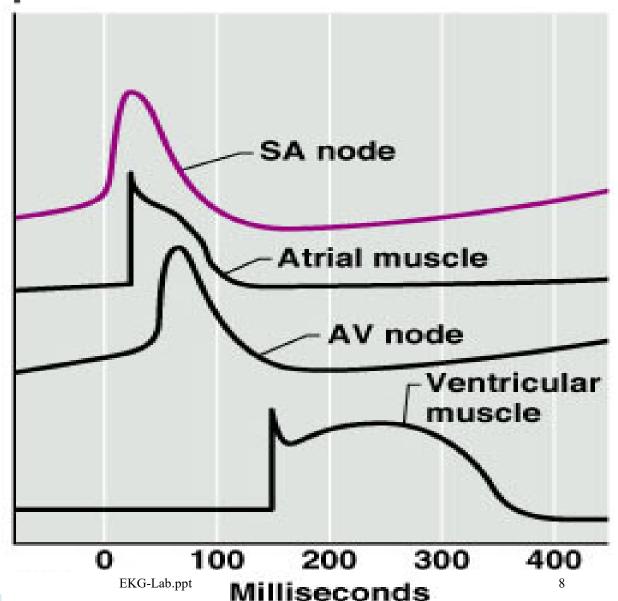
# Arrhythmia Formation

Arrhythmias can arise from problems in the:

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

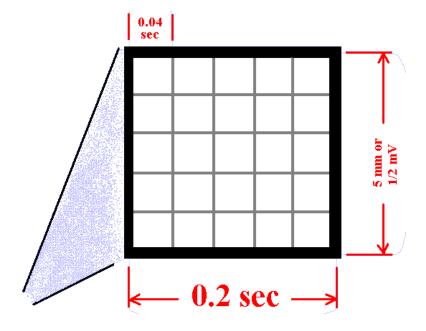
# Depolarization and Impulse Conduction

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



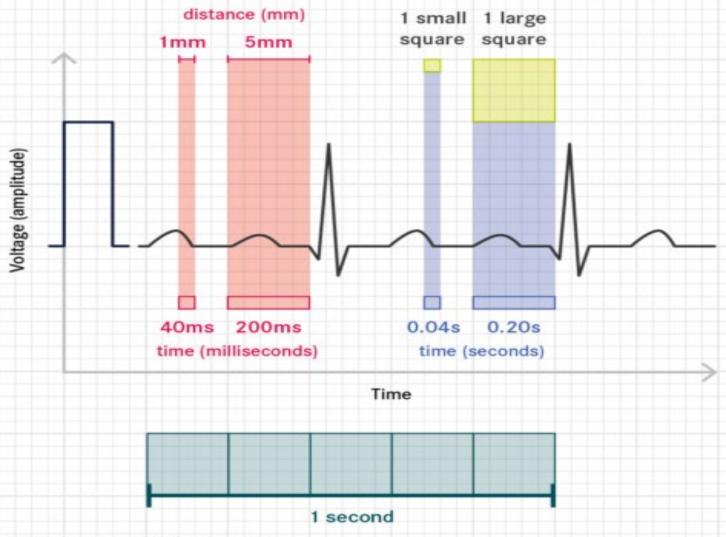
# The ECG Paper

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

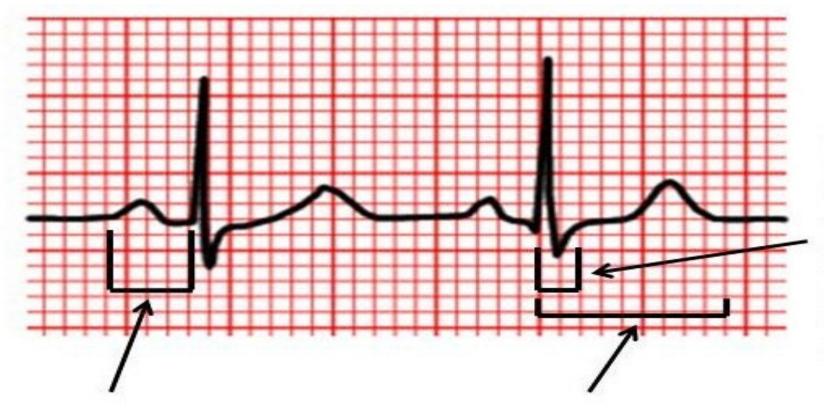




#### **Understanding an ECG**



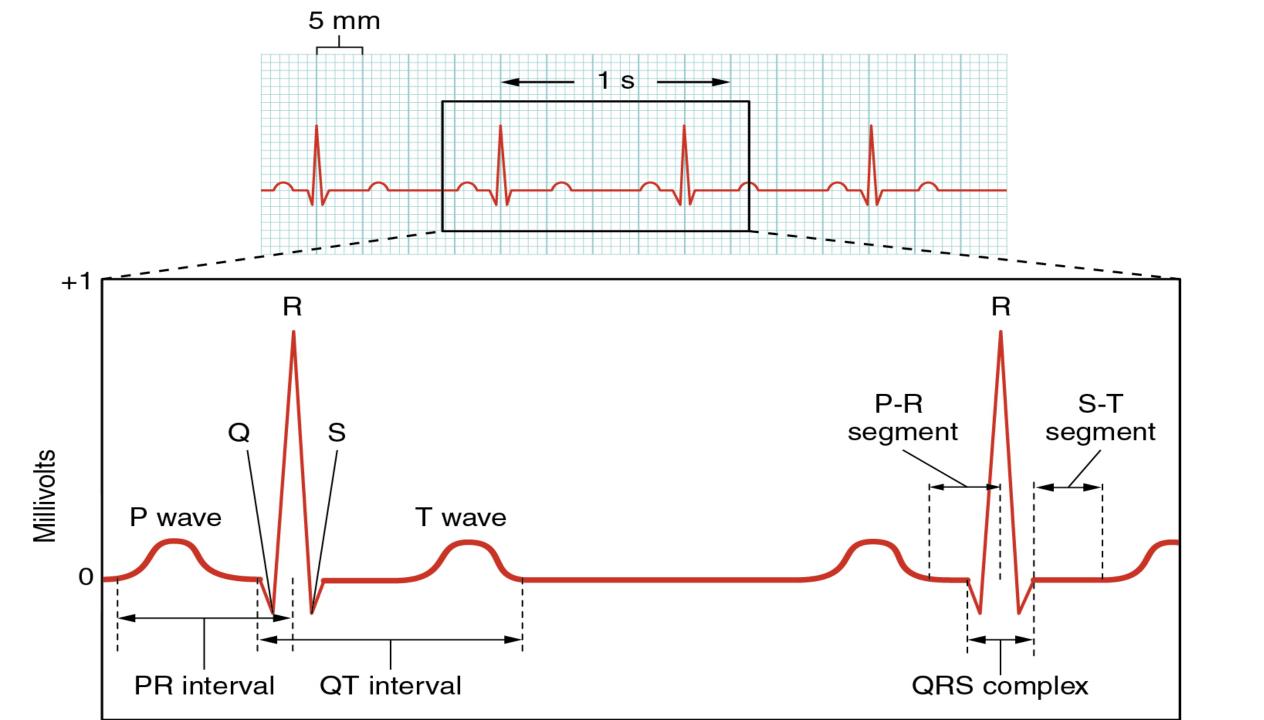
Standard calibrations: 25 mm/sec 10 mm/mV



QRS Interval:
measures 2 small
squares (2 mm)
which represents
a QRS interval of
0.08 seconds. The
normal QRS
interval is <0.12
seconds.

PR Interval:
measures 4 small
squares (4mm)
which represents a
PR interval of 0.16
seconds. The normal
PR interval is <0.2
seconds.

QT Interval: measures 9 small squares (9 mm) which represents a QT interval of 0.36 seconds. The normal QT interval is rate dependent, and reported as a corrected QT interval. Corrected QT interval = (observed QT interval) / (square root of RR interval). In general the QT interval is normal when it is less than half of the RR interval.



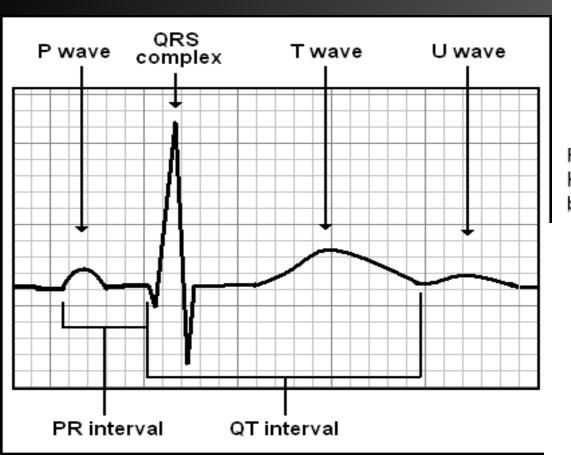
#### The ECG Paper (cont)



- 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



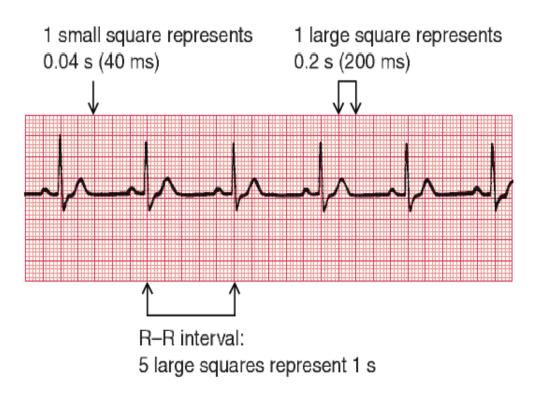
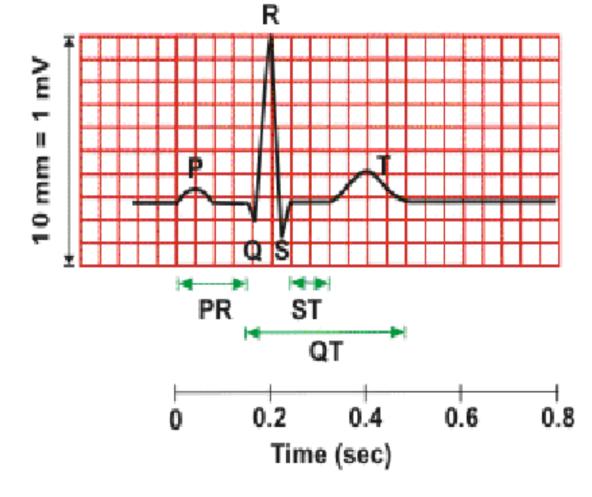


Fig. 1.4 Relationship between the squares on ECG paper and time. Here, there is one QRS complex per second, so the heart rate is 60 beats/min

#### Q waves

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

Component	Characteristics
Heart Rate	60-100 bpm
PR Interval	0.12–0.20 sec
QRS Interval	0.06–0.10 sec
QT Interval	Less than half of the R-R interval
ST segment	0.08 sec



P wave (0.08 - 0.10 s)

QRS (0.06 - 0.10 s)

P-R interval (0.12 - 0.20 s) Q-T<sub>C</sub> interval (≤ 0.44 s)\*

$$*QT_c = QT/\sqrt{RE}$$

Suppose we measured the height of the P wave to be 2 mm. Now we'll have to convert this measurement from millimeters into millivolts. It is a standard for each machine that 10 mm = 1 mV. Thus we can set up the following:

Amplitude of the P wave = 
$$2 \frac{\text{mm}}{10 \frac{\text{mm}}{\text{mm}}} = 0.2 \text{mV}$$

To measure the duration of an interval, we must measure its width in millimeters. Suppose we measure the width of the PR interval to be 4 mm. We'll have to convert this measurement from millimeters into seconds. The machine is set so that 25 mm of paper is used per 1 second. Thus we can set up the following:

Duration of the PR interval = 
$$4 \frac{\text{mm}}{\text{25} \frac{\text{mm}}{\text{mm}}} = 0.16 \text{ seconds}$$

To determine atrial rate, we begin by measuring the **PP interval**, the distance from the start of one P wave to the start of the next. During this span we will have one P wave and thus one atrial depolarization and one atrial contraction. Suppose the PP interval was 30 mm long. We can then set up the following:

$$\frac{1 \text{ P wave}}{30 \text{ mm}} \times \frac{1 \text{ atrial contraction}}{1 \text{ P wave}} \times \frac{25 \text{ mm}}{1 \text{ sec}} \times \frac{60 \text{ sec}}{1 \text{ min}} = \frac{50 \text{ atrial contractions}}{1 \text{ minute}}$$

We would now assume that the ventricular rate was also 50 contractions per minute. We could verify this by measuring the **RR interval**, the distance from the peak of one R wave to the peak of the next. If it was also 30 mm long, we could set up the following:

$$\frac{1 \text{ QRS}}{30 \text{ mm}} \times \frac{1 \text{ ventricular contraction}}{1 \text{ QRS}} \times \frac{25 \text{ mm}}{1 \text{ sec}} \times \frac{60 \text{ sec}}{1 \text{ min}} = \frac{50 \text{ ventricular contractions}}{1 \text{ minute}}$$

ECG Manifestations: Electrolyte Imbalance
Diercks DB et al. J Emerg Med. 2004; 27(2); 153-60.
Rosen's EM: Concepts and Clinical Practice, 7th ed, 2010. Goldberger, AL. Clinical Electrocardiography: A Simplified Approach, 7th ed, 2006.

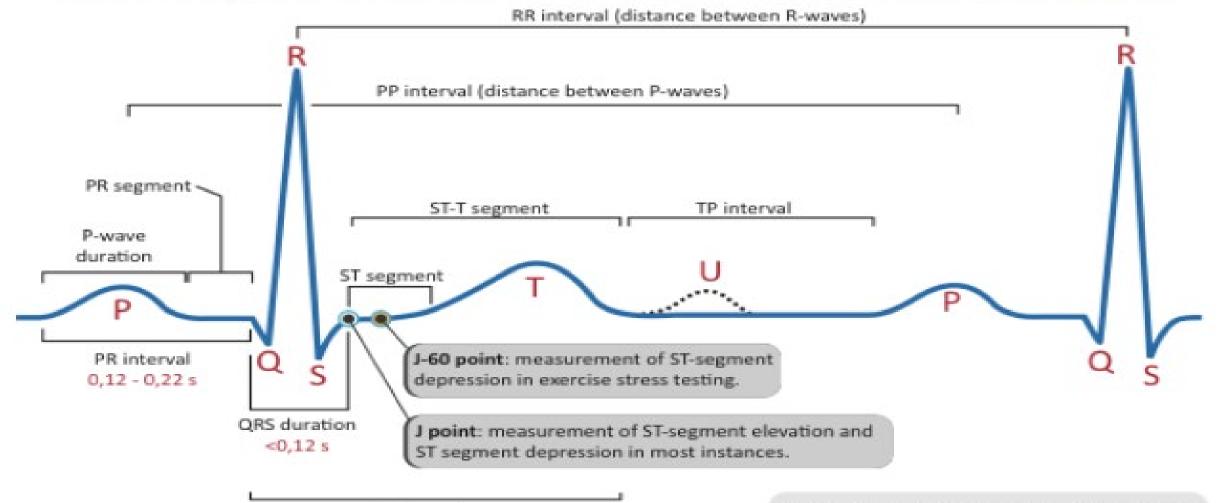
PR interval	Short	Prolonged	
-	(Think pre-excitation syndromes	High K	
	such as Wolff-Parkinson-White)	Low Ca	
QRS duration	Narrow	Wide (>100 msec)	
	Low K	High K	
	Low Ca	High Ca	
	Normal		
QTc interval	Short (<350 msec)	Prolonged (>440 msec)	
	High Ca	Low K	
	,700	Low Ca	
ST segment	Depressed	Elevated	
	Low K	High K	
	High Ca		
T wave	Peaked/tall	Flattened	
	High K	Low K	
U wave	Absent	Present	
	Normal	Low K	
		Low Ca	
Heart rate	Slow	Fast	
	(bradydysrhythmia, nodal block)	(tachydysrhythmia)	
	High K	Low K	
	High Ca	Low Ca	

	Low	High	
Са	<ul> <li>QTc prolonged (hallmark)</li> <li>U wave</li> <li>Heart blocks, ventricular dysrhythmias, torsades de pointes</li> </ul>	<ul> <li>QTc shortened (hallmark)</li> <li>ST segment depression and shortening</li> <li>QRS widening</li> <li>Rare: bradydysrhythmias, bundle branch blocks, high degree AV blocks</li> </ul>	
К	Early to late findings:  T wave: decreased amplitude T wave: flat or inverted ST segment depression U wave QTc prolonged (at risk for VT or torsades de pointes)	Early to late findings:  T wave: tall, then "peaked" (symmetrical)  P wave flattening  PR interval prolonged  QRS widening  Nodal blocks, escape beats  Sine wave: fusion of QRS and T wave > VF or asystole	

Mg derangements: Nonspecific ECG findings; often co-exist with Ca derangements. • Classic teaching: Low Mg level --> QTc prolongation --> torsades de pointes

7/22/2024

#### Waves, intervals and durations on the ECG



QT duration

Corrected QT duration men: ≤ 0,45 s Corrected QT duration women: ≤ 0,47 s The reference level for measuring ST-segment deviation (depression or elevation) is not the TP interval. The correct reference level is the PR segment. This level is also called baseline level or isoelectric level.

#### **EKG Leads**

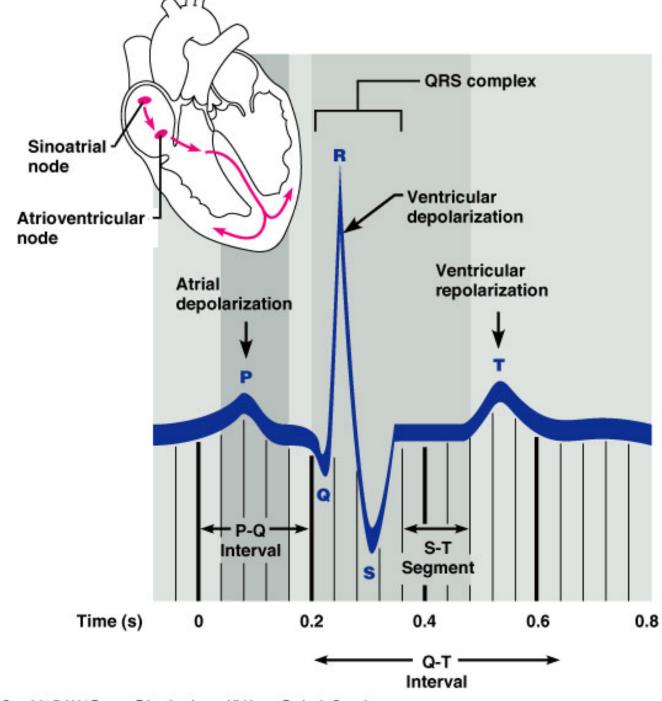
The standard EKG has 12 leads:

- 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.

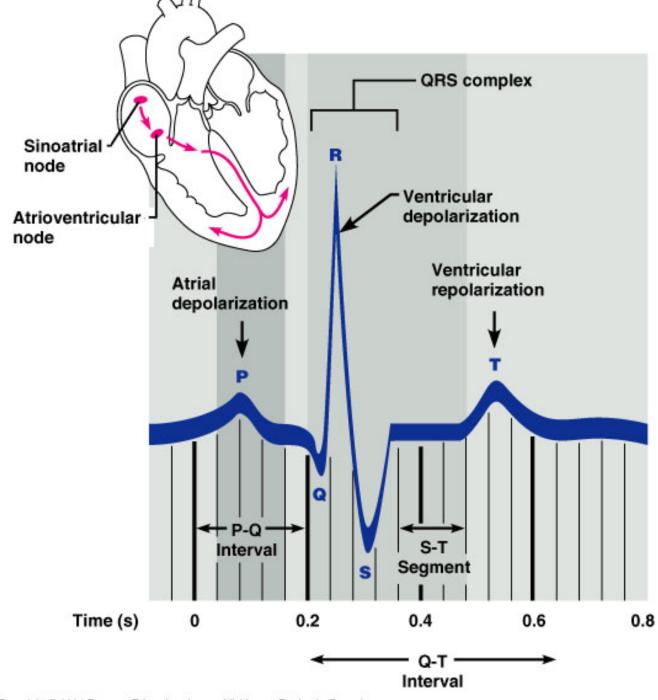
# Electrocardiogram

- 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



# Electrocardiogram

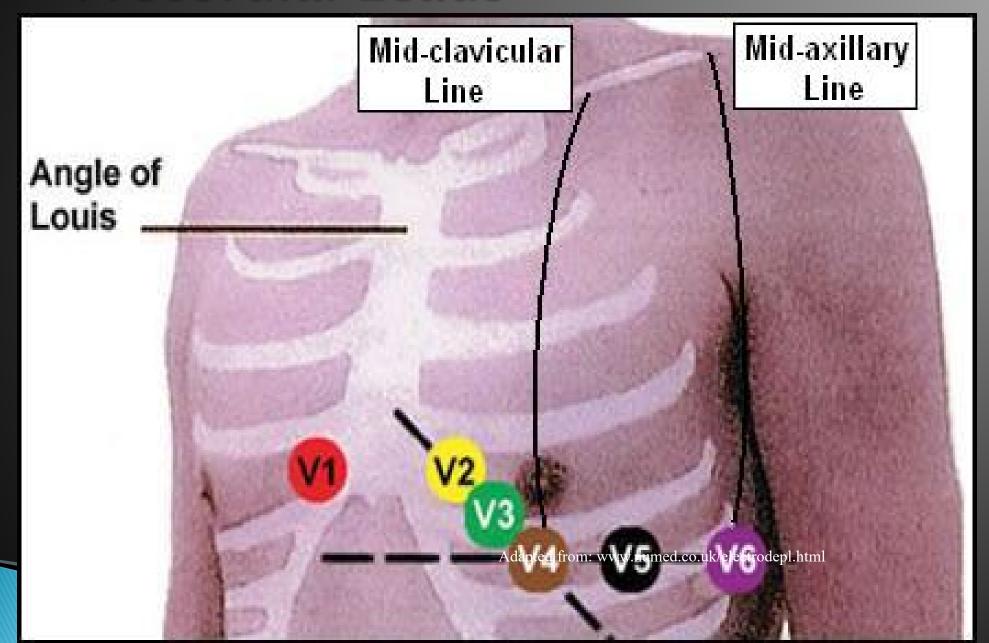
- P-Q interval
  - Time atria depolarize & remain depolarized
- Q-T interval
  - Time ventricles depolarize & remain depolarized



#### **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

# Precordial Leads



#### Lead Placement

V1 = 4th intercostal space, right border of sternum

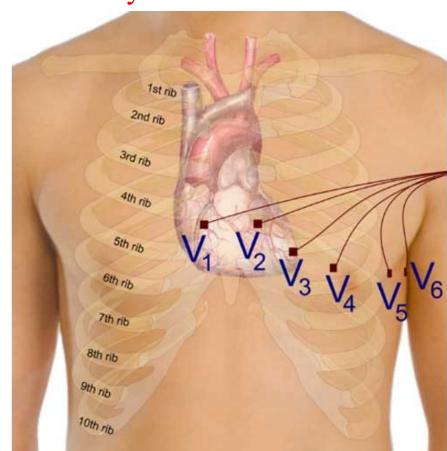
V2 = 4th intercostal space, left border of sternum

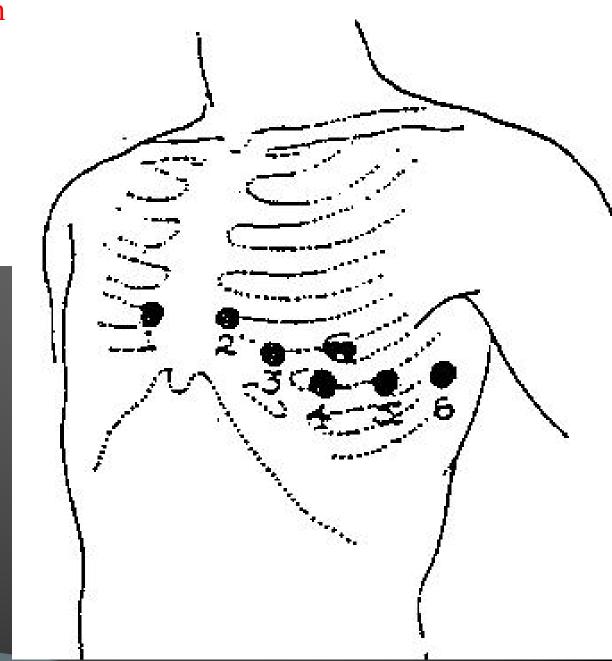
V3 = midway between V2 and V4

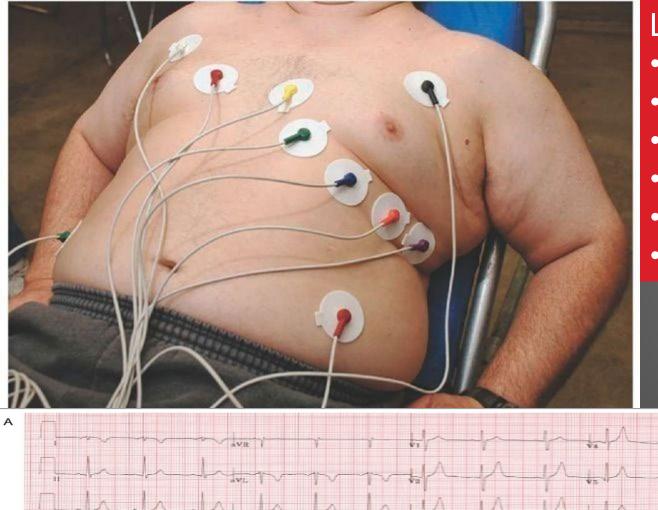
V4 = 5th intercostal space, midclavicular line

V5 = anteroaxillary line at level of V4

V6 = midaxillary line at level of V4 and V5

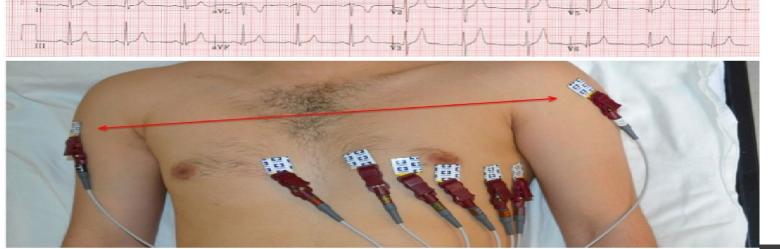


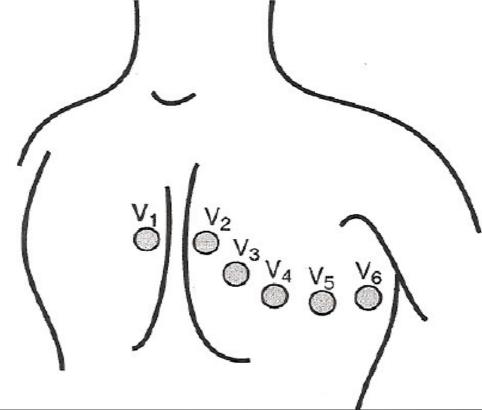




#### **Lead Placement**

- 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 ICS
- V6 Mid axillary line 5th ICS





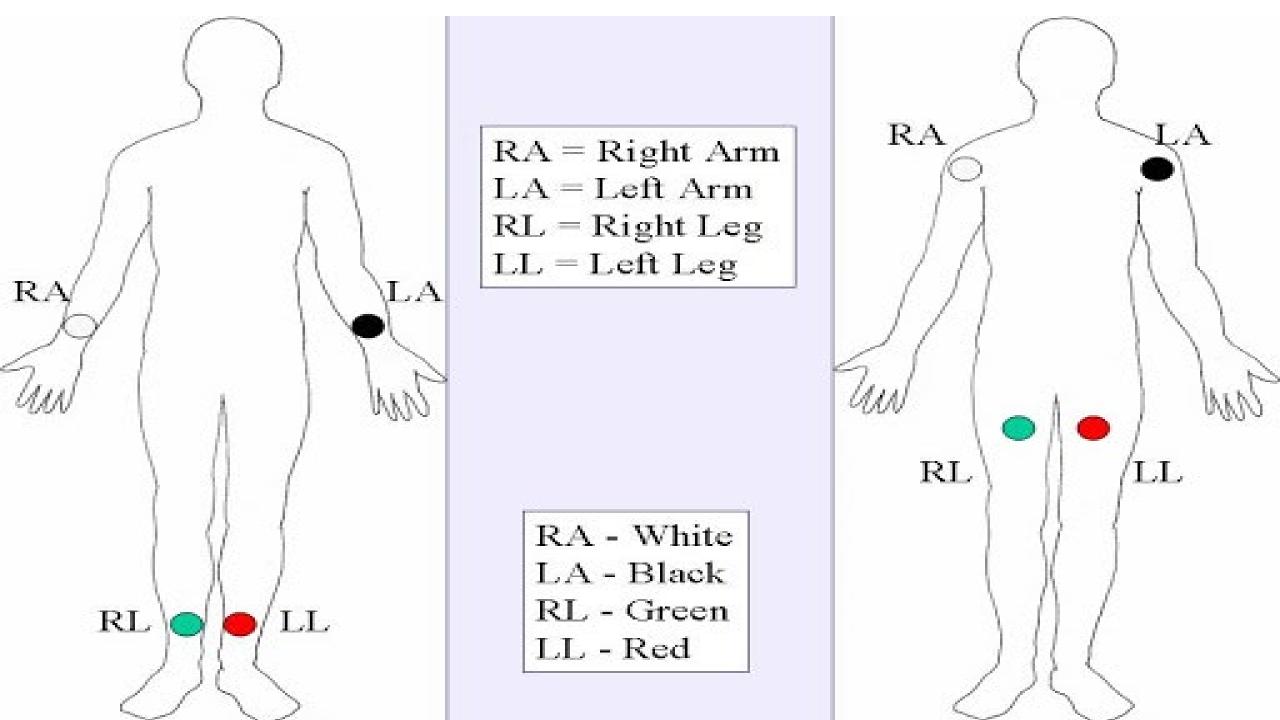
- Lead Placement
- ▶ V1 =
- ▶ V2 =
- ▶ V3 =
- ▶ V4 =
- ▶ V5 =
- ▶ V6 =



#### 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



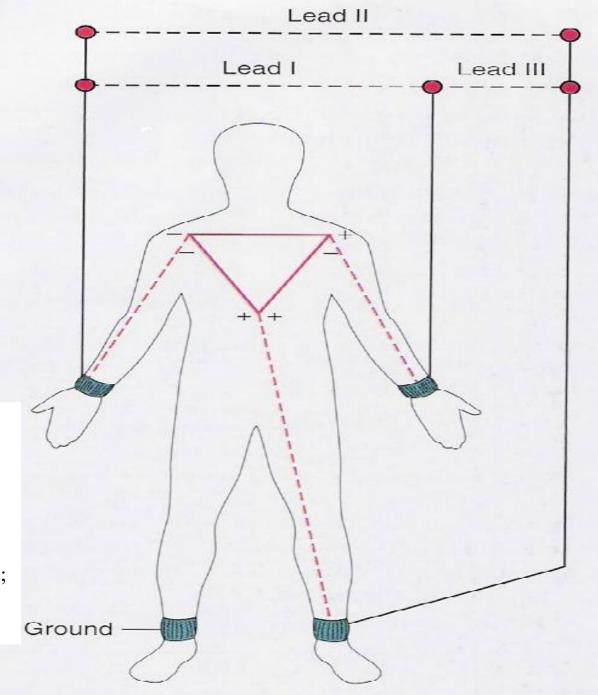


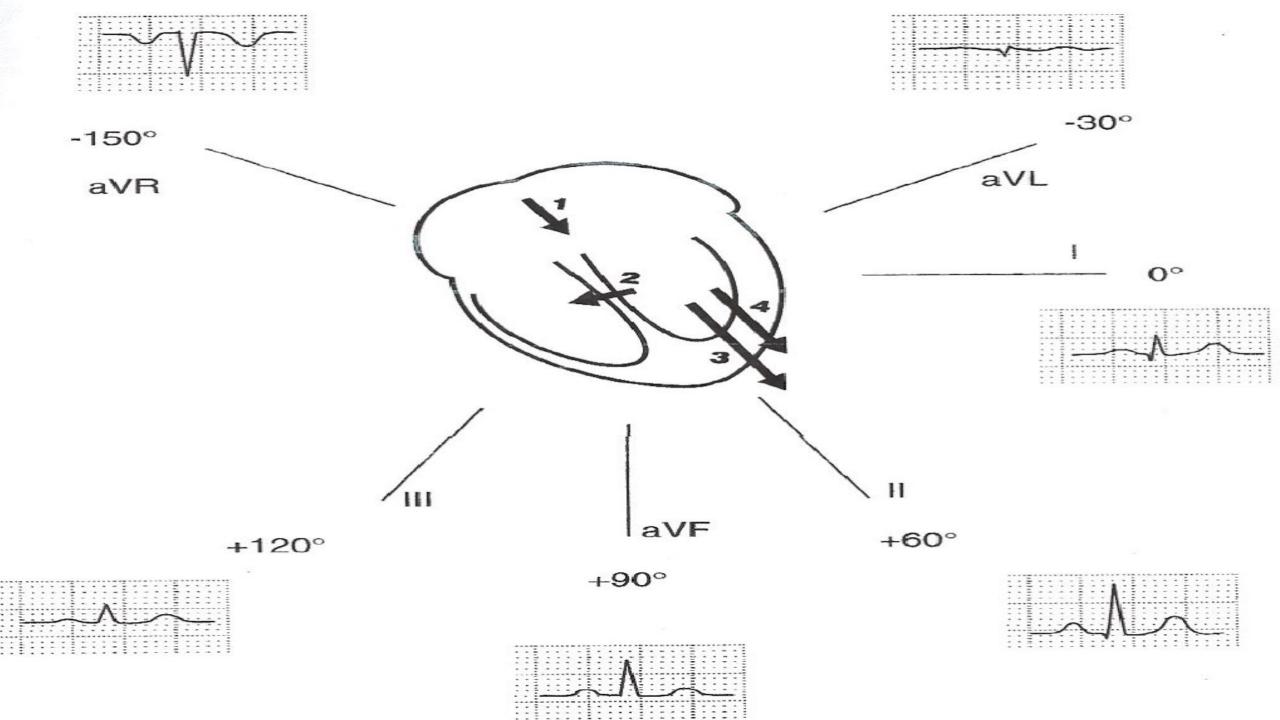
#### Electrocardiogram

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

Triangle and Einthoven's law: the three bipolar leads form what is called the Einthoven's triangle (named so after the inventor of the electrocardiogram). These leads maintain a mathematical proportion explained by the Einthoven's law, which says: II = I + III.

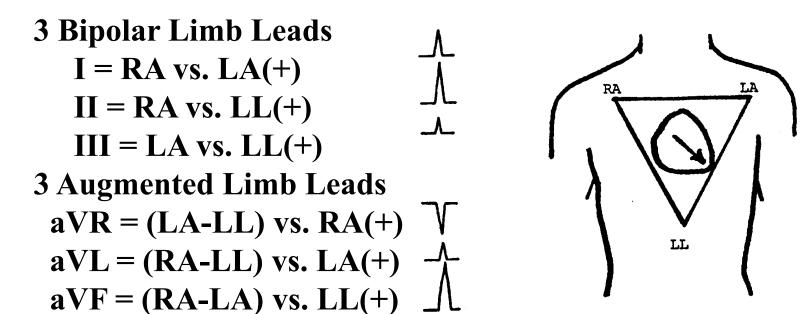
This law is of great value when Interpreting an electrocardiogram. It allows us to determine whether the limb electrodes are correctly placed; if the position of any electrode is altered, this law would not hold, thus allowing us to realise the EKG is not correctly done



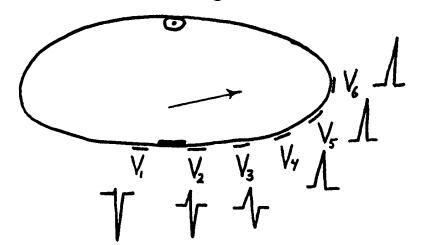


QRS Deflection			Axis
Lead I	Lead aVF	Lead II	
		NA	Normal
+		+	Normal
+		<u> </u>	LAD
	+	NA	RAD
-	<b>-</b>	NA	Extreme Axis

#### 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  $V_6$ .



- 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.

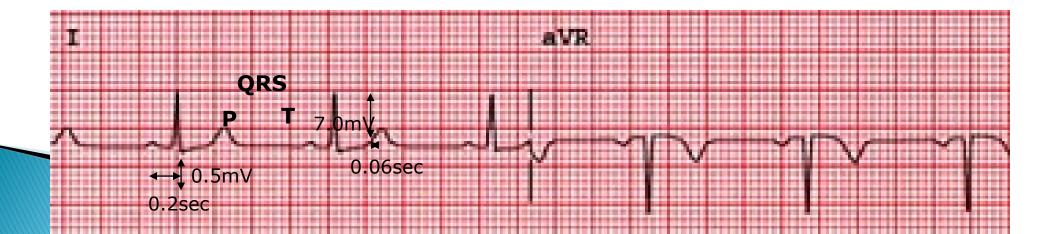
Negative QRS in Lead aVR.

R wave = 7-8 mm high in

Lead I = 7-8mV.

QRS wave = 0.06 seconds

long in Lead I.



Rhythm Summary



▶ Rate 90–95 bpm

Regularity regular

P waves normal

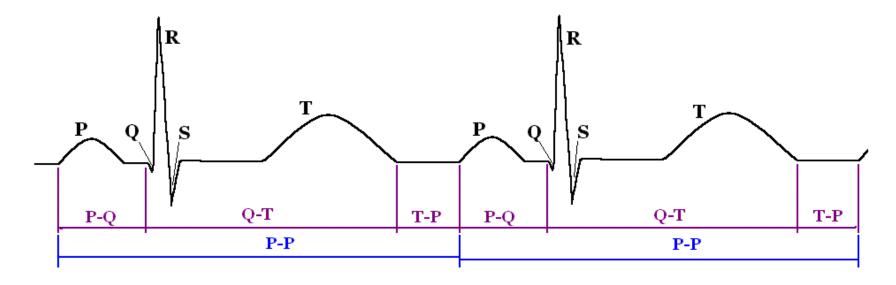
▶ PR interval 0.12 s

• QRS duration 0.08 s

Interpretation? Normal Sinus Rhythm

# Electrocardiogram

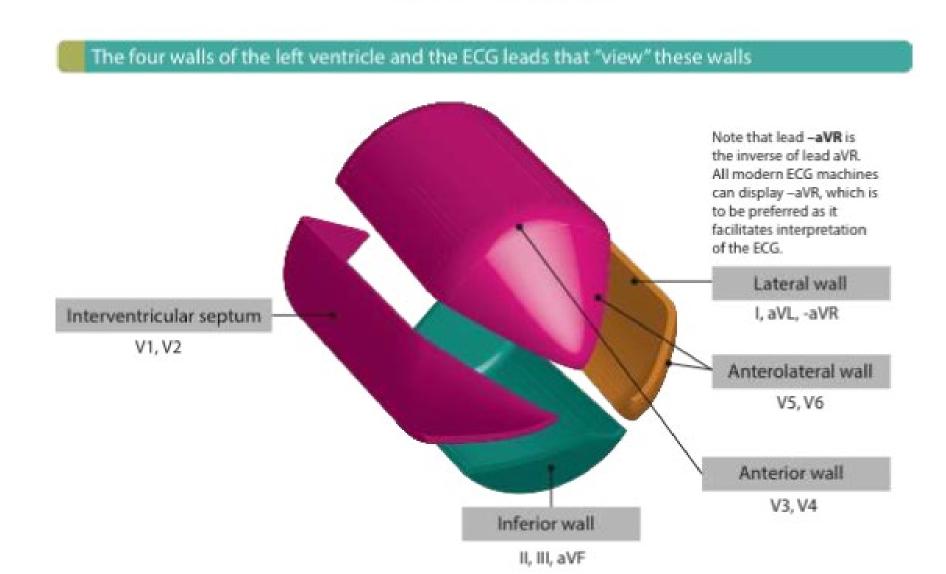
- Intervals show timing of cardiac cycle
  - P-P = one cardiac cycle
  - P-Q = time for atrial depolarization
  - Q-T = time for ventricular depolarization
  - T-P = time for relaxation



- 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

# The walls of the left ventricle and the leads that view these walls



# Anatomic Groups (Septum)

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

# Anatomic Groups (Anterior Wall)

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

# Anatomic Groups (Lateral Wall)

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

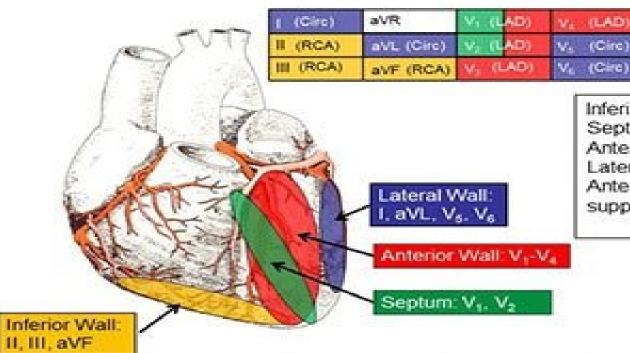
# Anatomic Groups (Inferior Wall)

l	aVR	V <sub>1</sub>	V <sub>4</sub>
Lateral	None	Septal	Anterior
II	aVL	V <sub>2</sub>	V <sub>5</sub>
Inferior	Lateral	Septal	Lateral
III	a∀F	V <sub>3</sub>	V <sub>6</sub>
Inferior	Inferior	Anterior	Lateral

# Anatomic Groups (Summary)

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

#### Which Leads Look Where?



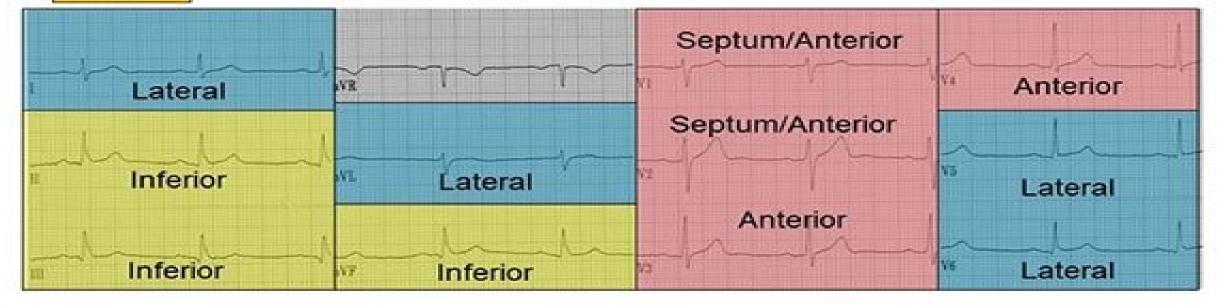
Inferior wall: II, III, aVF

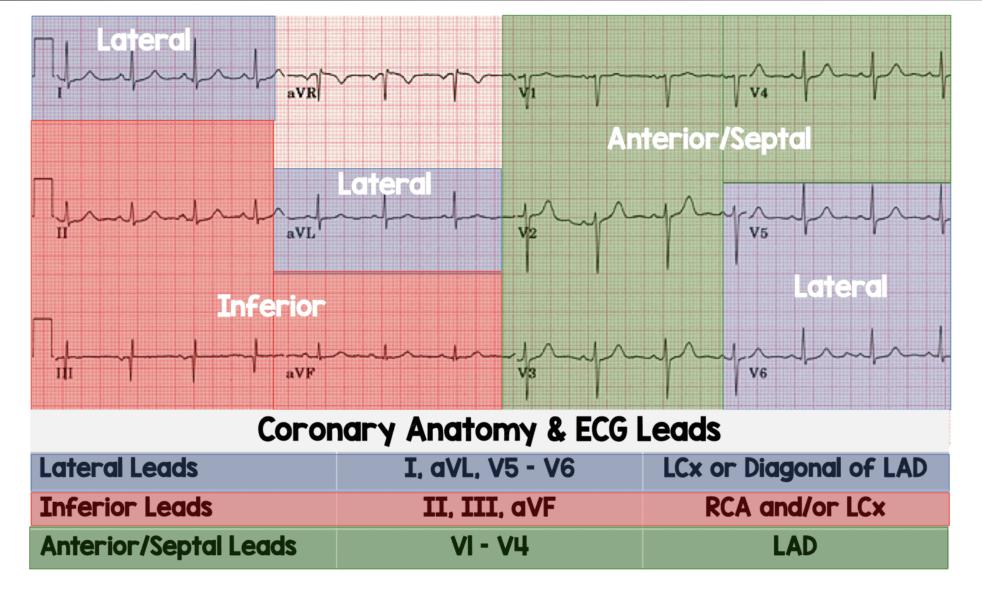
Septum: V1, V2 Anterior wall: V3, V4

Lateral wall: I, aVL (high lateral); V5, V6 (low lateral)

Anterior wall and septum often infarct together because both

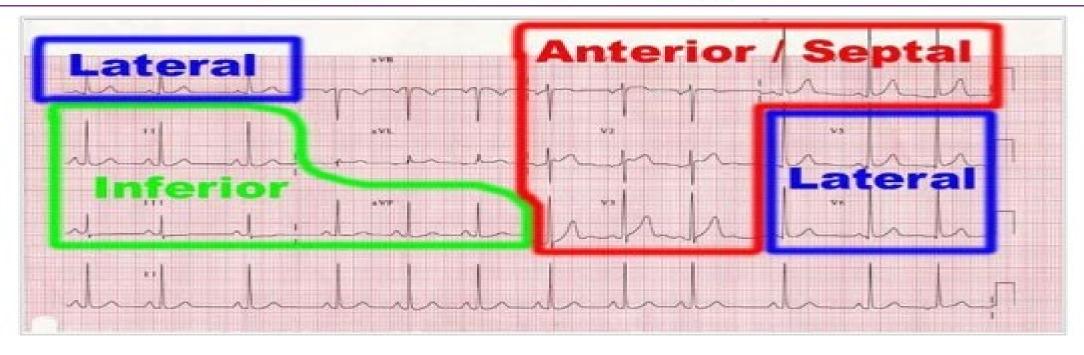
supplied by LAD, so anteroseptal MI shows in V1-V4







# Areas of the ECG to be concentrated upon to study the events e.g. MI

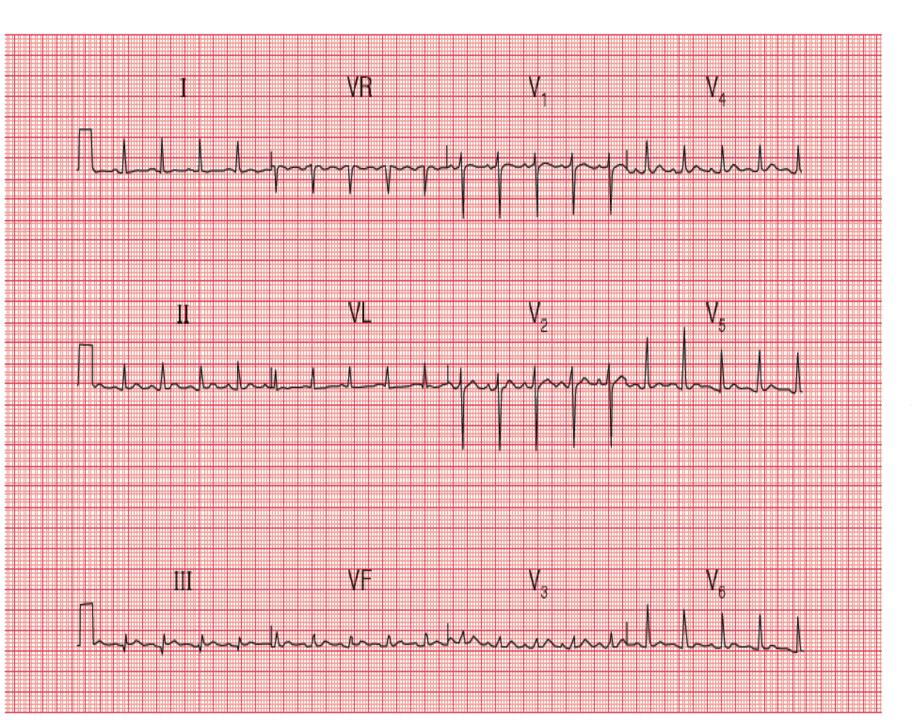


Position	Leads
Lateral	look on lead I,V5 ,V6
Inferior	look on lead II ,III ,aVF
Anterior/ Septal	look on V1,V2,V3,V4

### 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

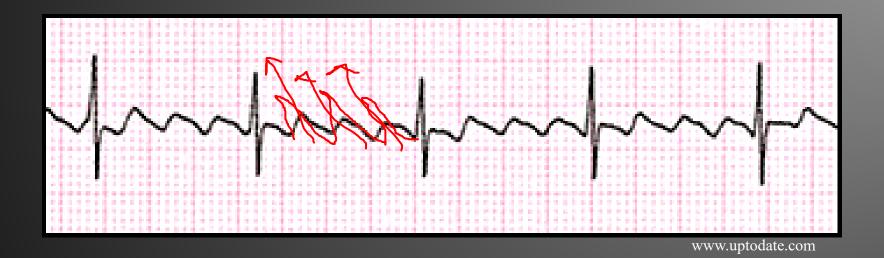
- Sinus rhythm, rate 110/min
- Normal PR interval (140 ms)
- Normal QRS duration (120 ms)
- Normal cardiac axis
- Normal QRS complexes
- Normal T waves (an inverted T wave in lead VR is normal)

### Interpretation

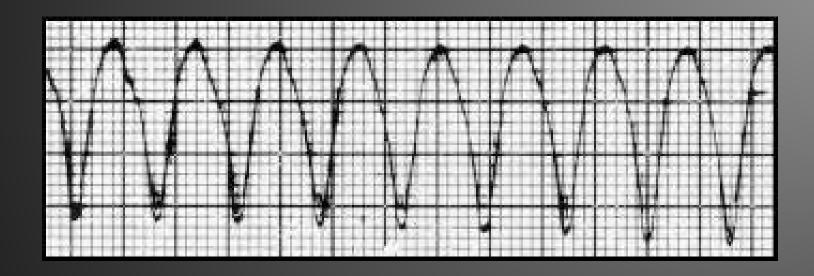
Normal ECG



(300 / 6) = 50 bpm



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



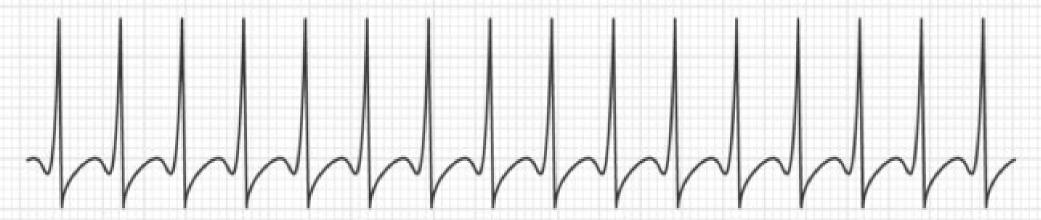
(300 / 1.5) = 200 bpm

## 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

# Superventricular tachycardia (SVT)



Standard calibrations: 25 mm/sec 10 mm/mV

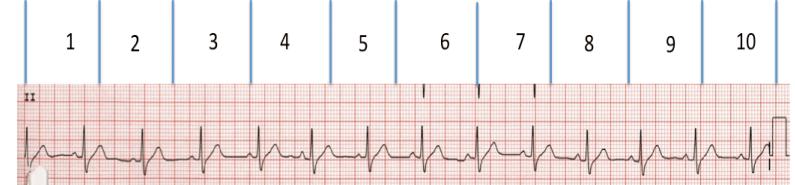
Like in the example identify two consecutive R waves and count the number of small squares (0.04s) between them. By dividing this number into 1500 (remember, this number also represents one minute) we are able to calculate a person's heart rate.

# What is the 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.

# What is the 6 second rule on ECG?

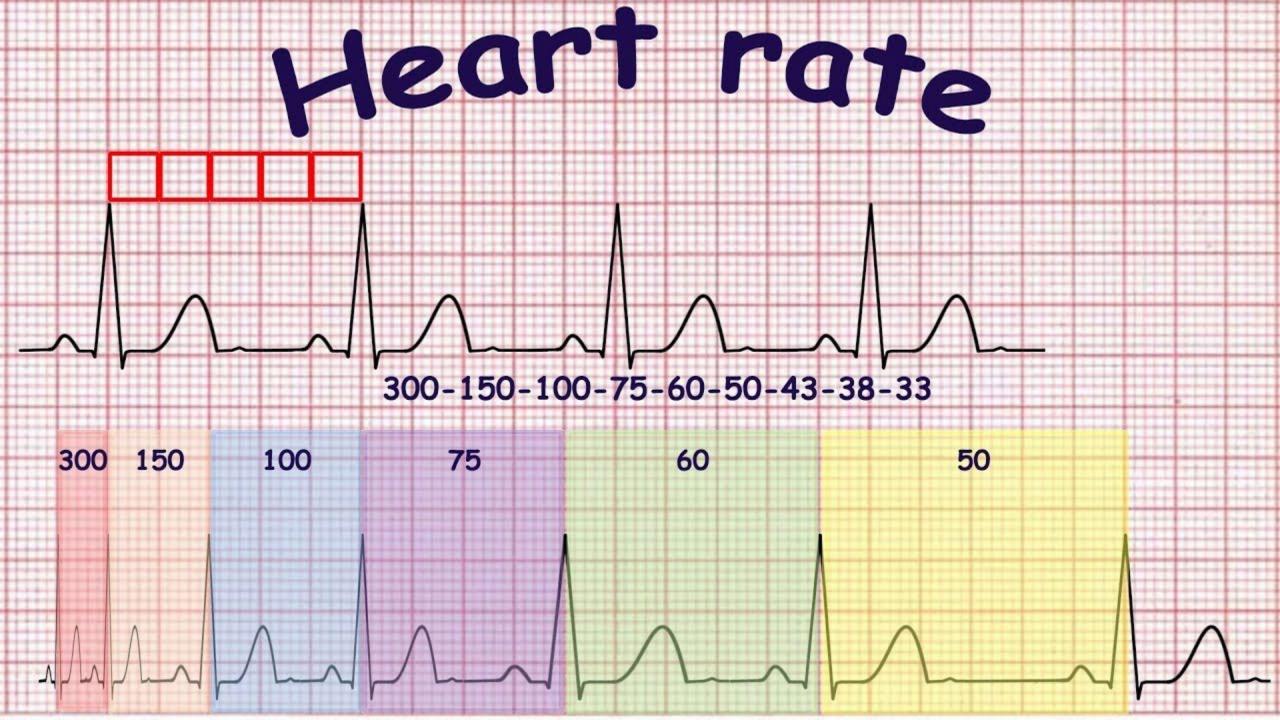
Count the number of R waves in a 6 second strip and multiply by 10. For example, if there are 7 R waves in a 6 second strip, the heart rate is 70 (7x10=70).



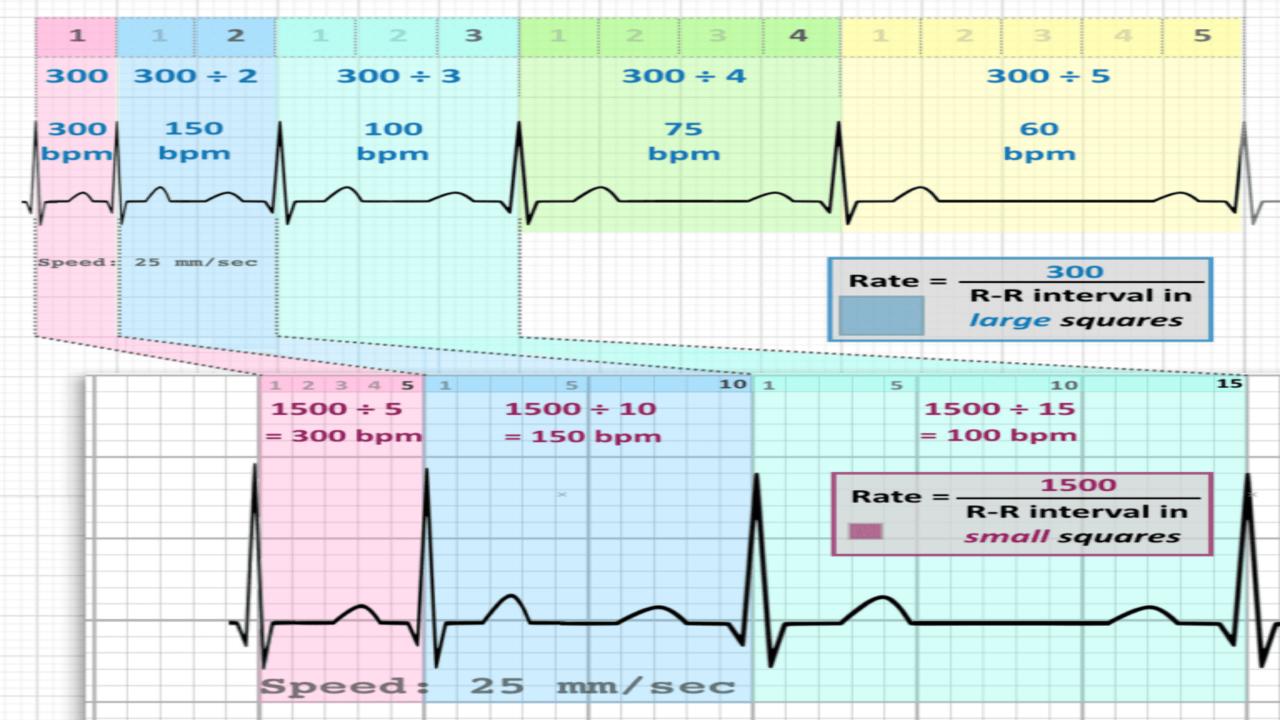
What is the 1500 rule for ECG?

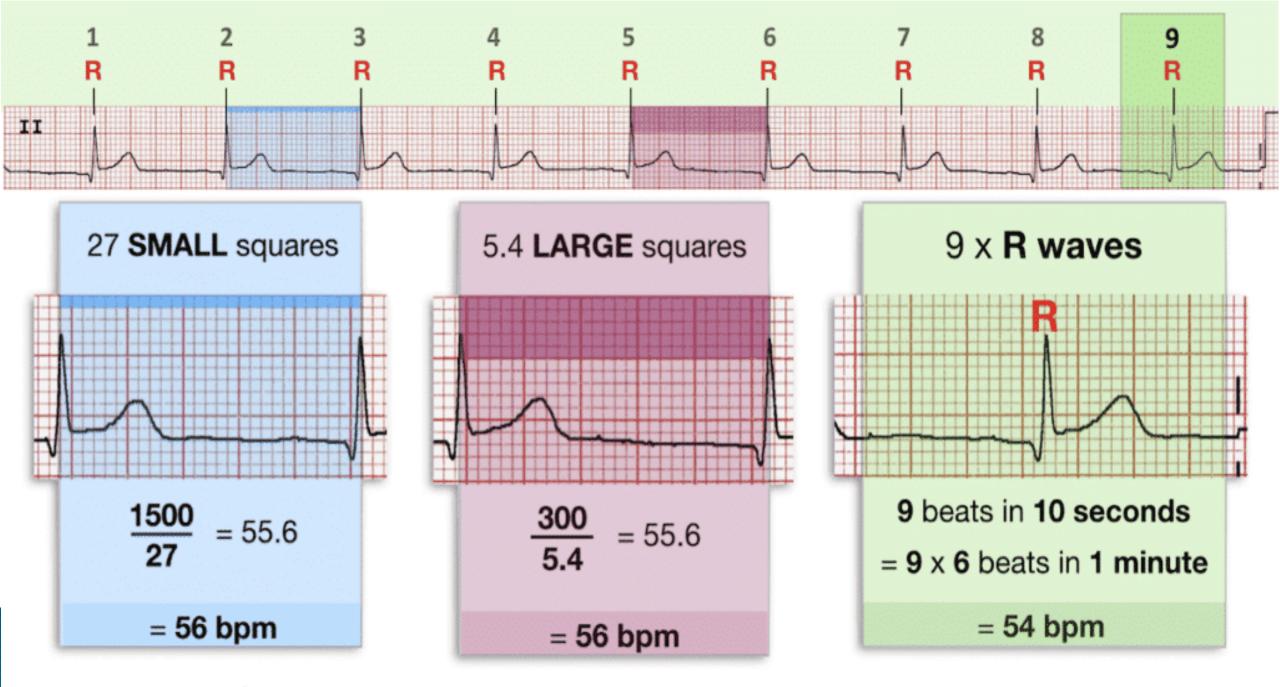
Count the number of small boxes between two successive R waves and divide this number into 1500 to obtain heart rate. This works well for faster heart rates.

14 R waves in 10 secs  $14 \times 6 = 84 \text{ BPM}$ 

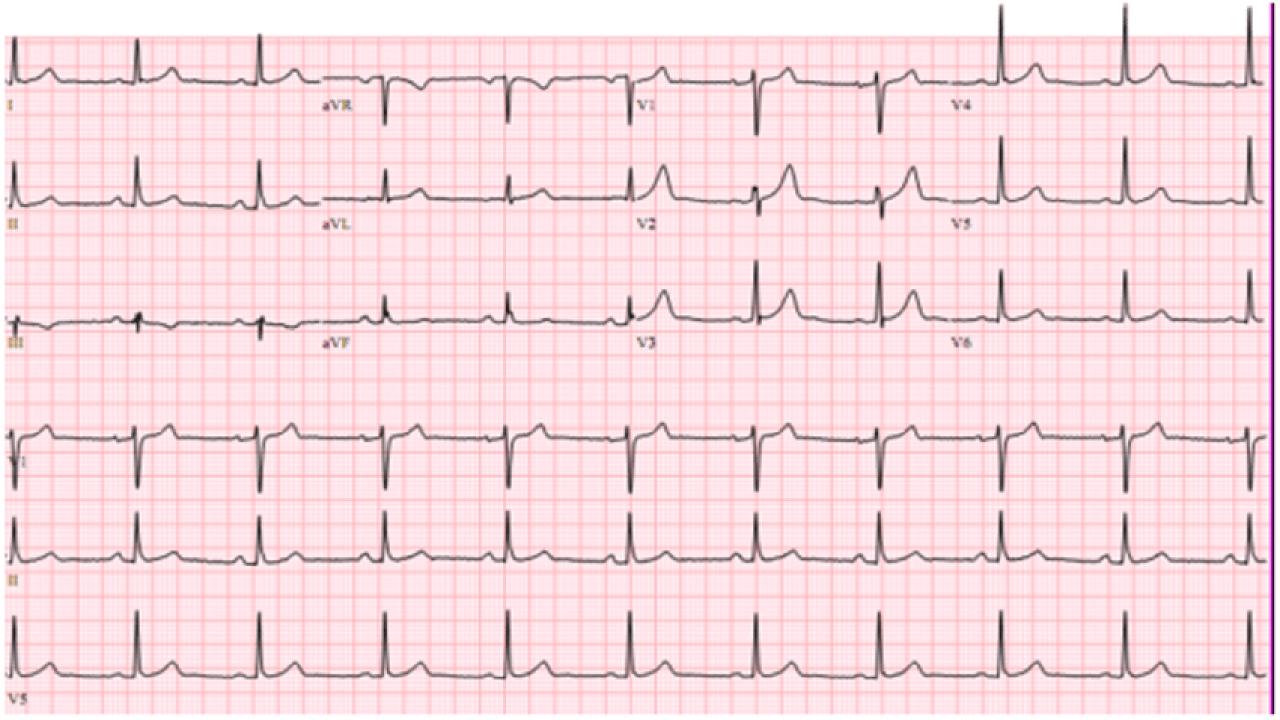








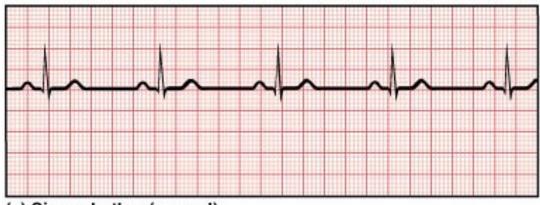
Speed: 25 mm/sec



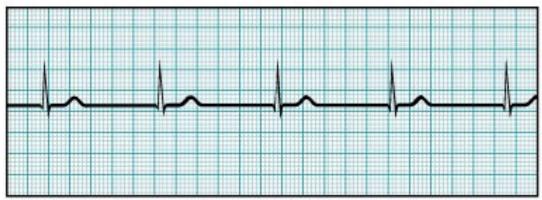
 $33 \times 6 = 198 \text{ bpm}$ 



# ECGs, Normal and Abnormal

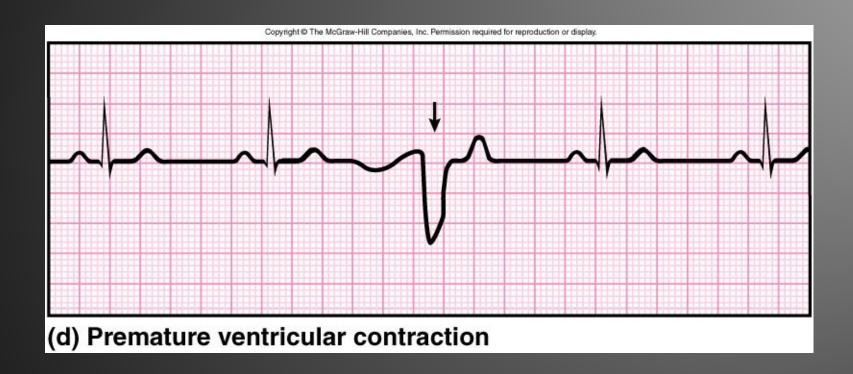


(a) Sinus rhythm (normal)



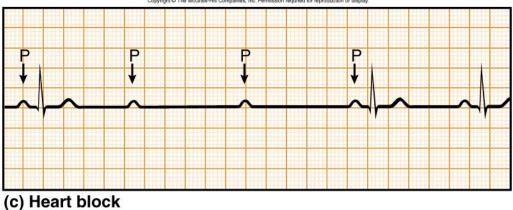
(b) Nodal rhythm - no SA node activity

## ECGs, Abnormal

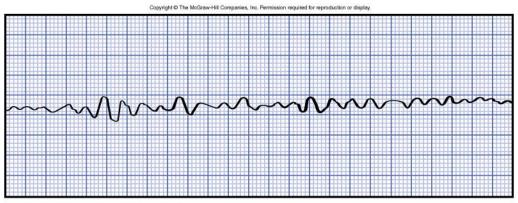


Extrasystole: note inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.

ECGs, Abnormal



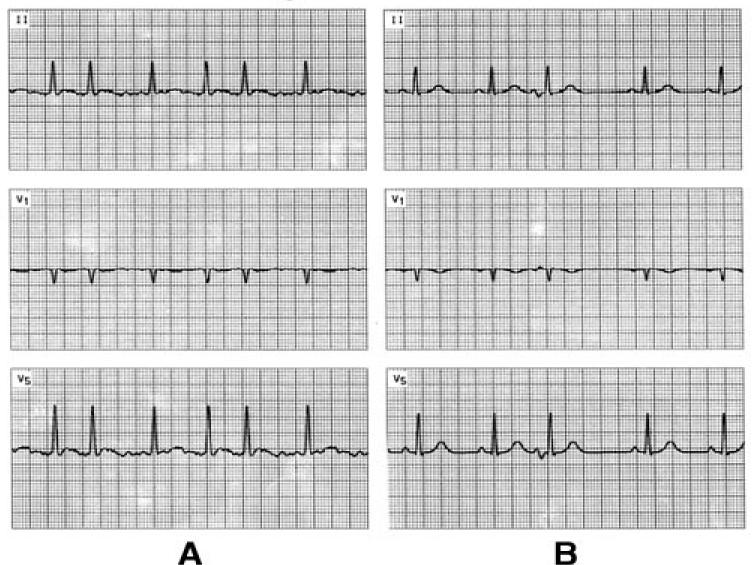
### Arrhythmia: conduction failure at AV node



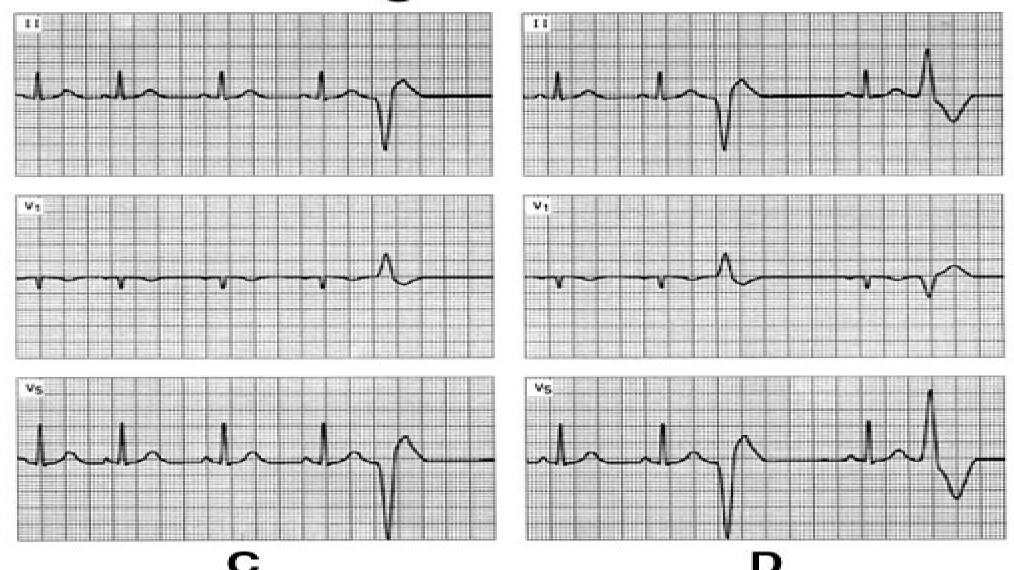
(e) Ventricular fibrillation

No pumping action occurs

# Irregular ECGs

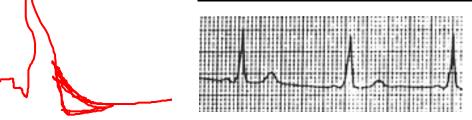


## Irregular ECGs



### PR interval

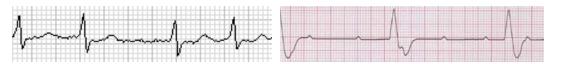
< 0.12 s	0.12-0.20 s	> 0.20 s
High catecholamine states Wolff-Parkinson-White	Normal	AV nodal blocks





### QRS complex

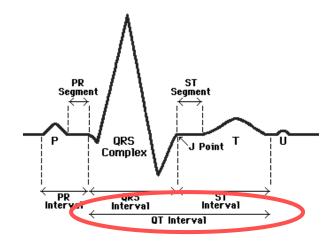
≤ 0.10 s	0.10-0.12 s	> 0.12 s
Normal	Incomplete bundle branch block	Bundle branch block PVC Ventricular rhythm



### QT interval

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

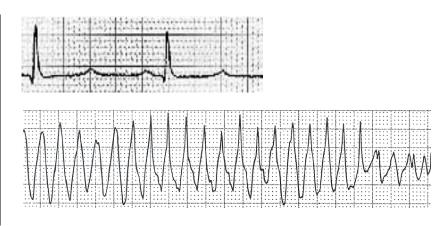
The faster the heart beats, the faster the ventricles repolarize so the shorter the QT interval. Therefore what is a "normal" QT varies with the heart rate. For each heart rate you need to calculate an adjusted QT interval, called the "corrected QT" (QTc):

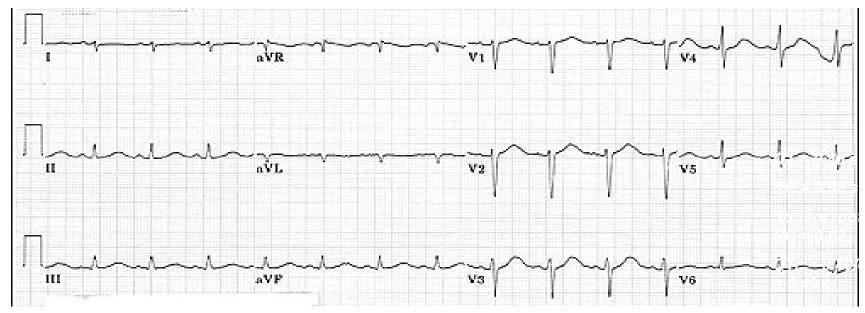


QTc = QT / square root of RR interval

### QTc interval

< 0.44 s	> 0.44 s
Normal	Long QT



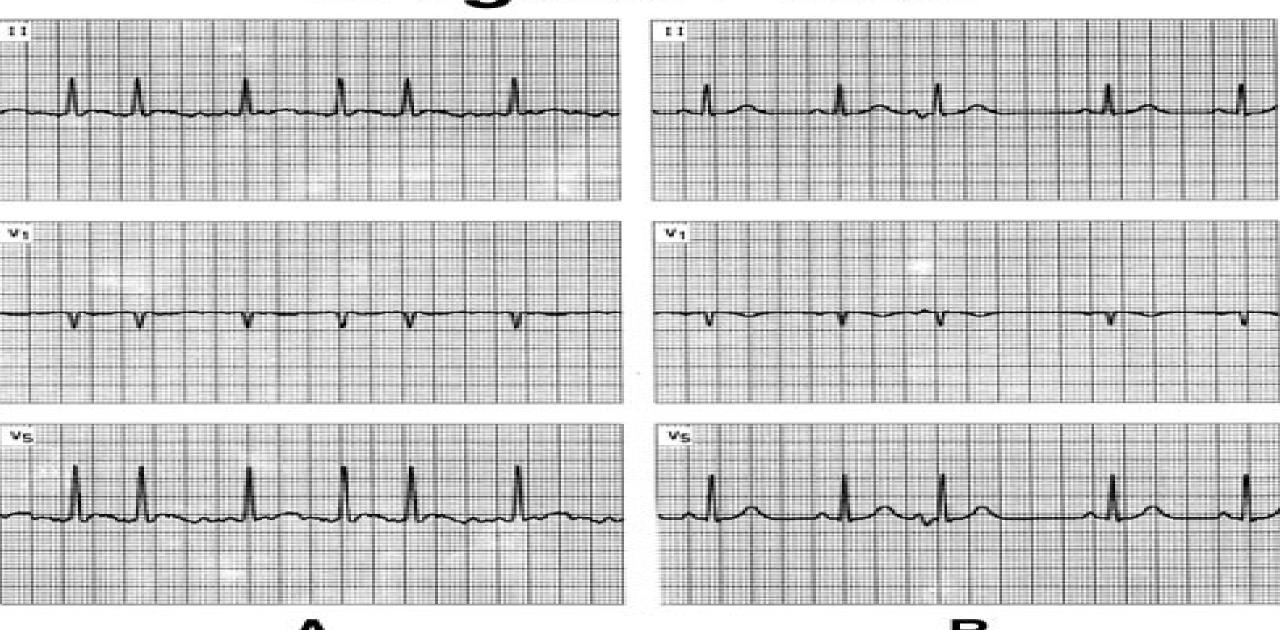


PR O.16 QRS width?

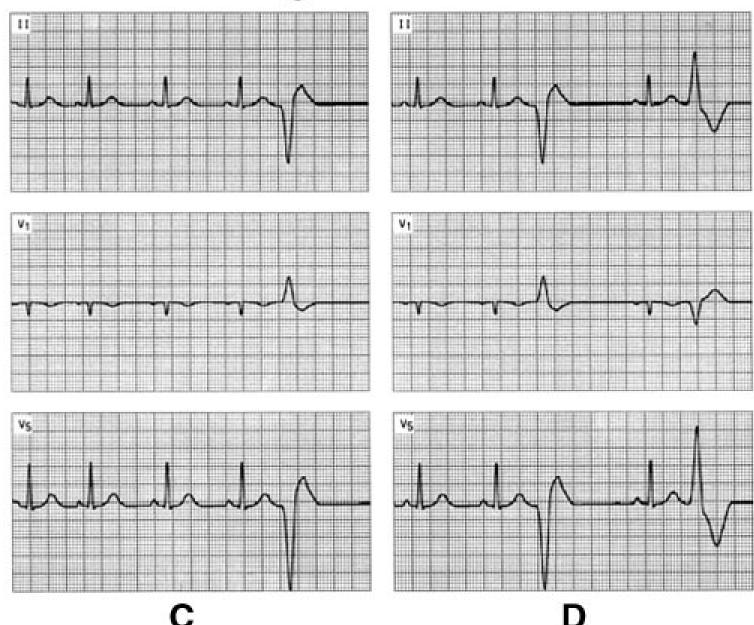
0.08 QTc 0.49 interval? seconds

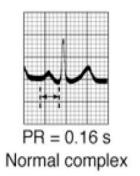
Interpretation of intervals? Normal PR and QRS, long

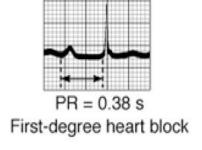
## Irregular ECGs

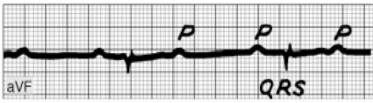


# Irregular ECGs





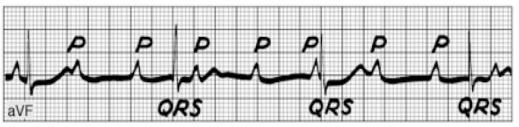




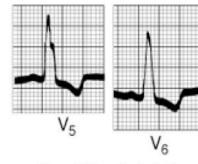
Second-degree heart block (2:1 heart block)



Second-degree heart block (Wenckebach phenomenon)

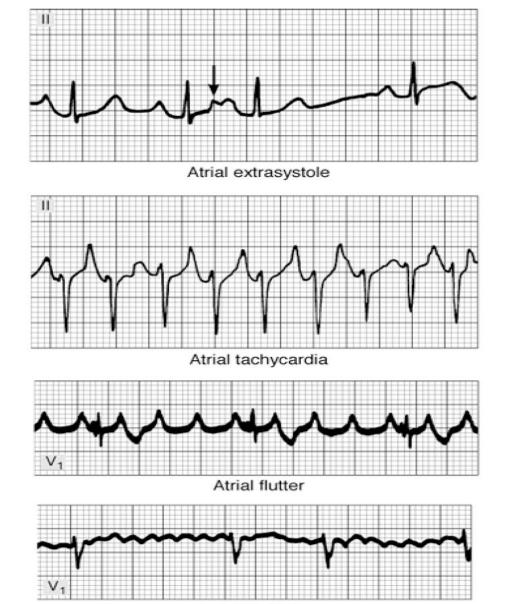


Complete heart block. Atrial rate, 107; ventricular rate, 43



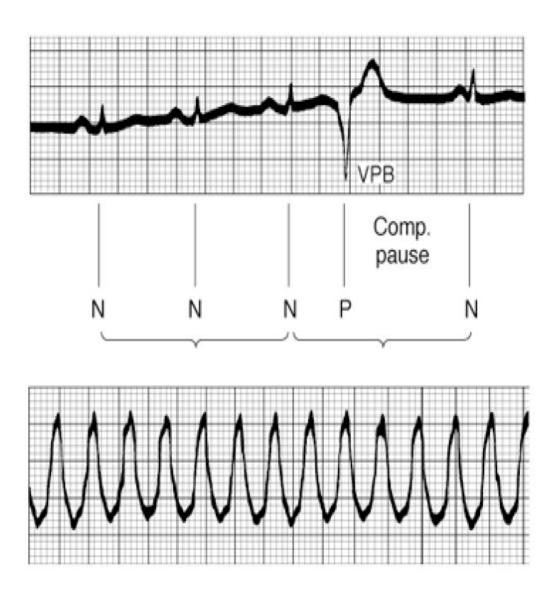
Two V leads in left bundle branch block

Figure 28-11. Heart block.



Atrial fibrillation

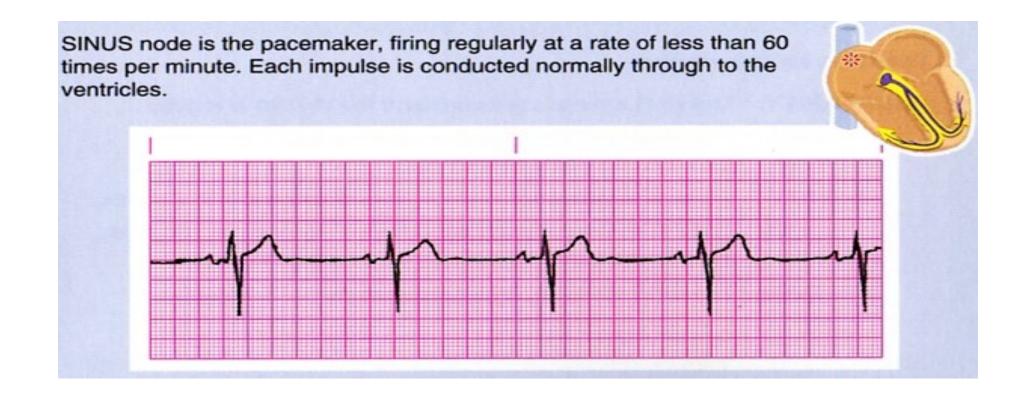
**Figure 28-13.** Atrial arrhythmias. The illustration shows an atrial premature beat with its P wave superimposed on the T wave of the preceding beat (arrow); atrial tachycardia; atrial flutter with 4:1 AV block; and atrial fibrillation with a totally irregular ventricular rate. (Tracings reproduced,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



# 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.

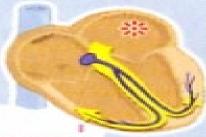


# Sinus Tachycardia

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.

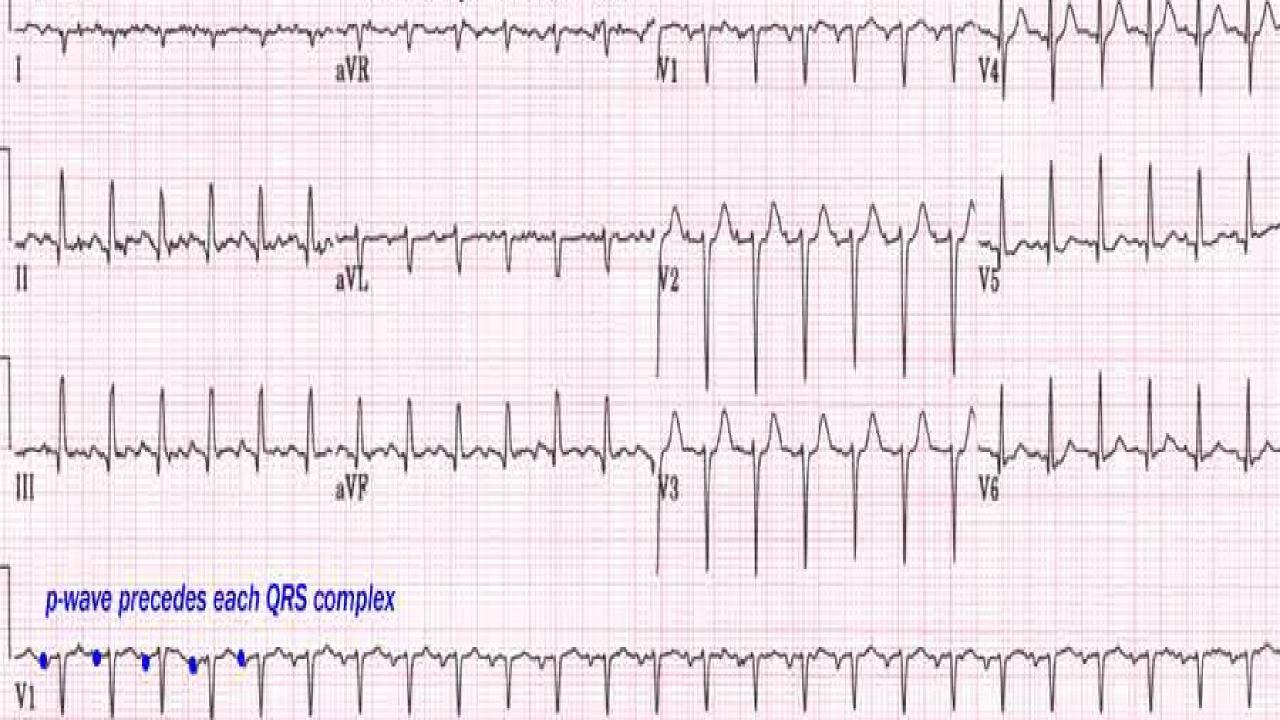
Here there is a focus other than the SA node that is firing, which causes a QRS.

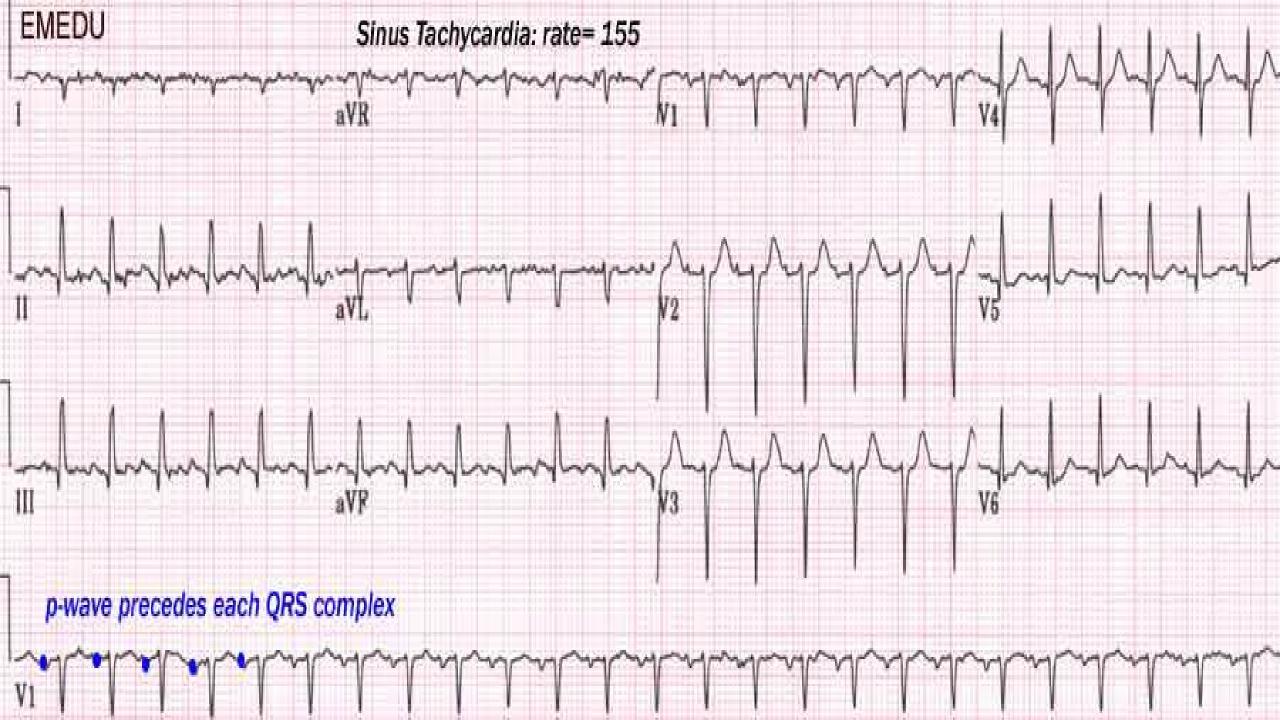
What is they underlying rate? 7\*10 using 6-second. Or, 75 using 4 big boxes per QRS.

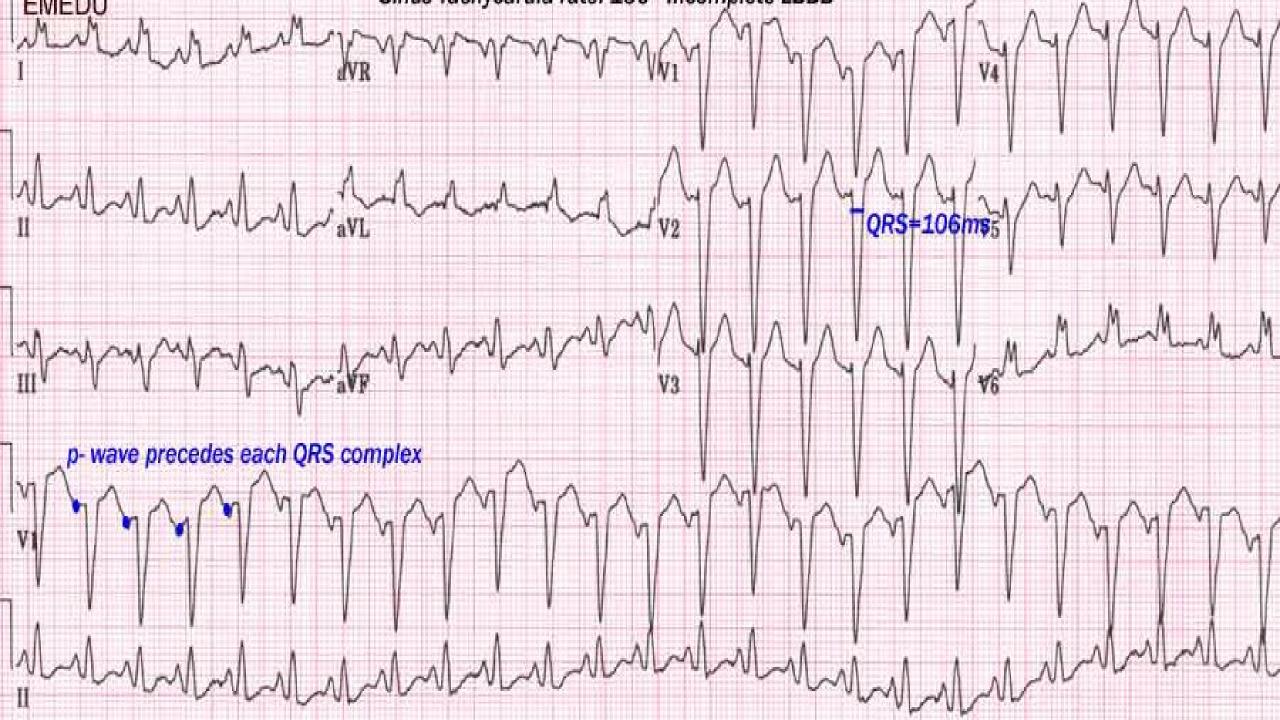
The PAC is the third QRS; you can see there is only about 1 big box between the 2nd and 3rd QRS. Usually the abnormal focus will cause a p-wave the looks different from the other p-waves (the SA node p-waves).

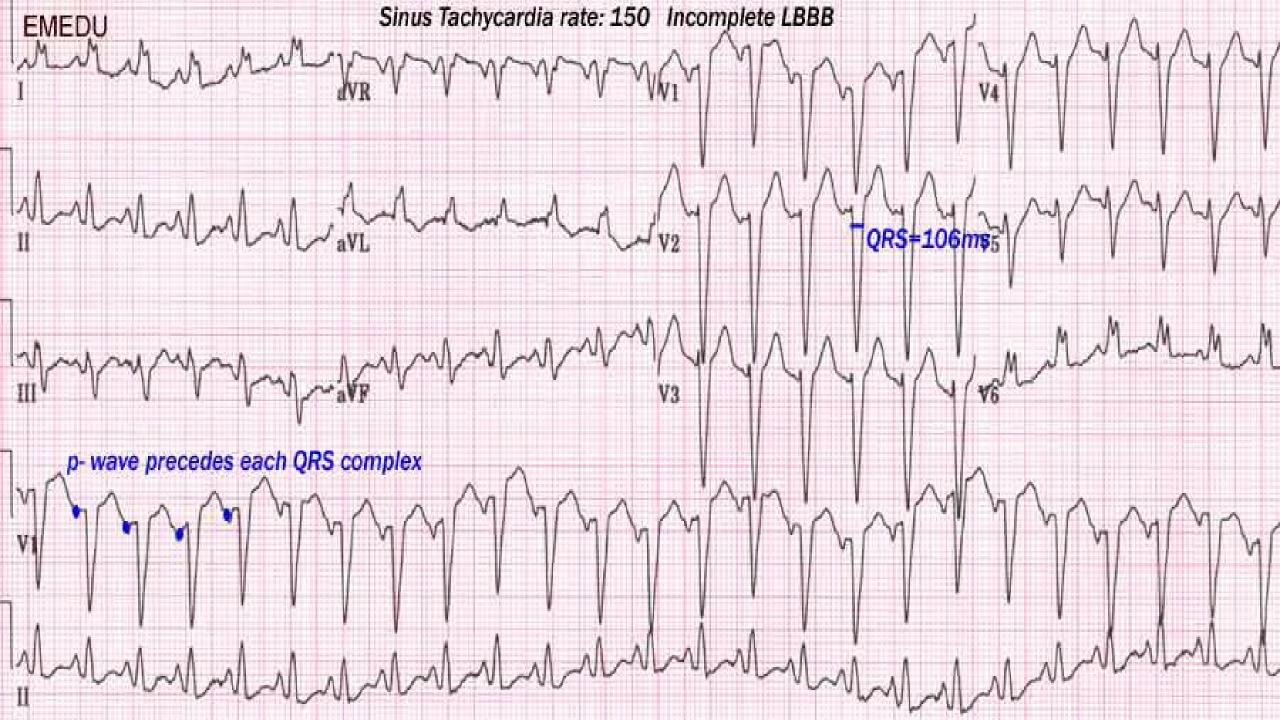
Here the aberrant atrial depolarization appears as a peaked p-wave in comparison.

## Premature Atrial Contraction









### What do you think?



A: normal

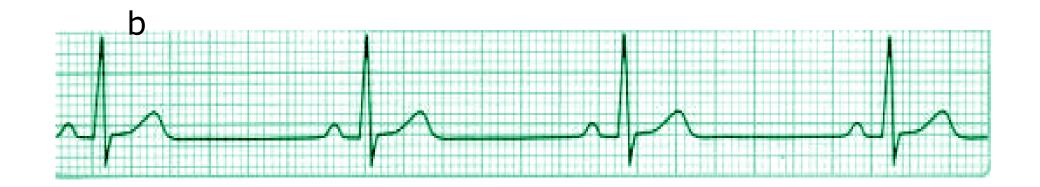
B: sinus tachycardia



### What do you think?

a

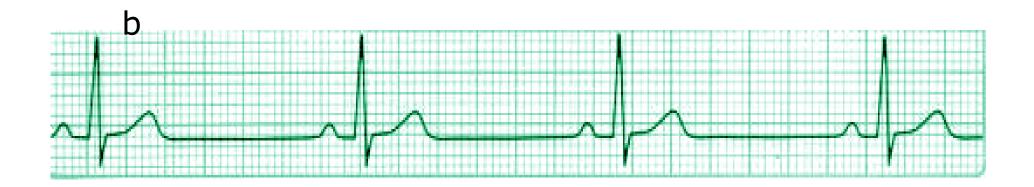




A: normal

a B:sinus bradycardia





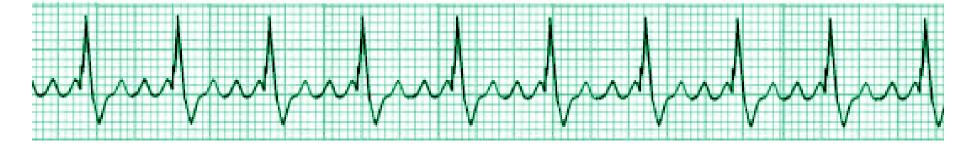




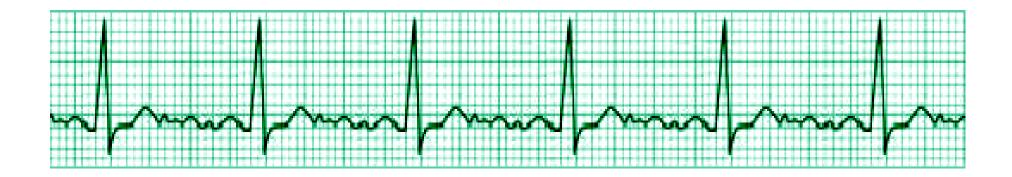
### Normal



### **Atrial Flutter**



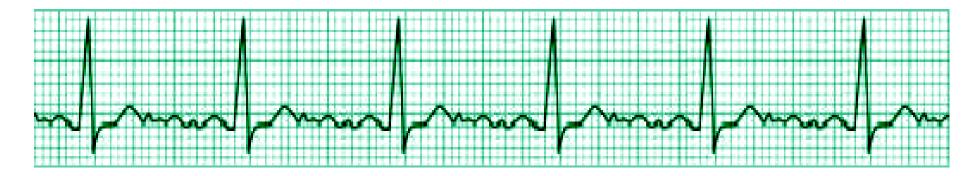


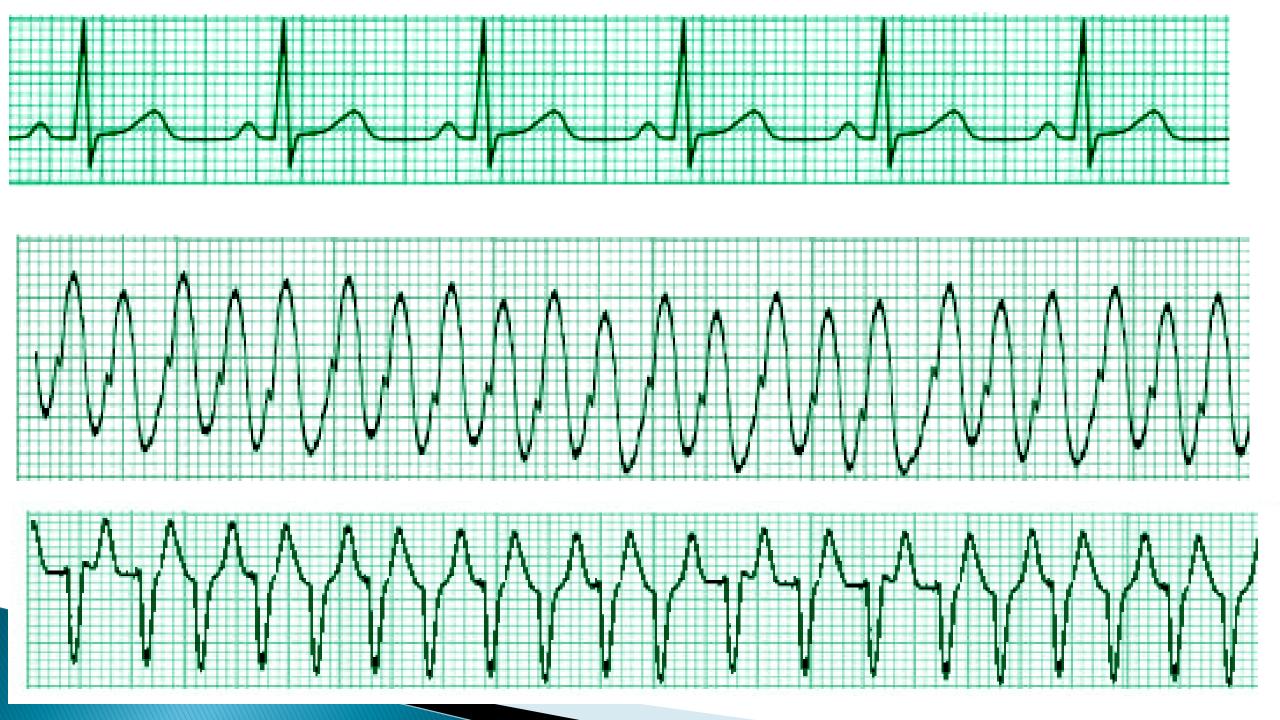


### Normal

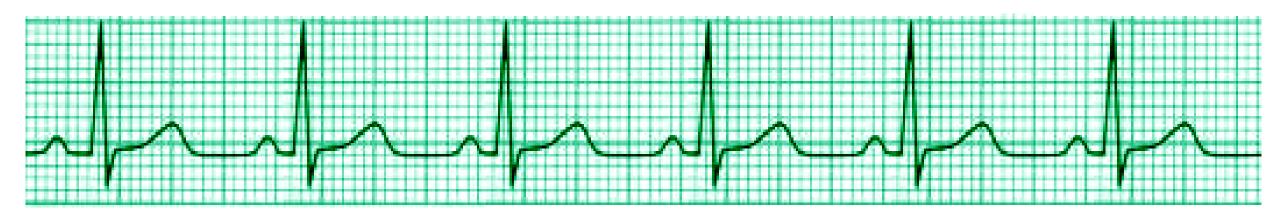


### **Atrial Fibrillation**

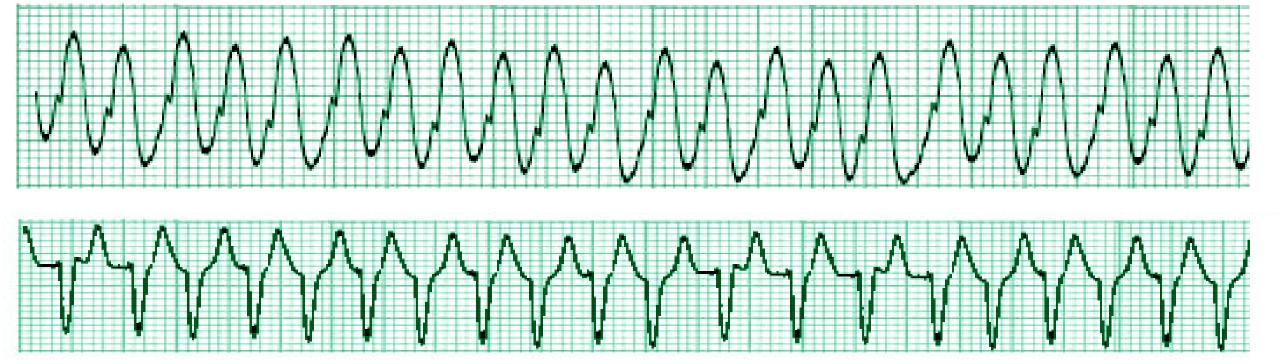


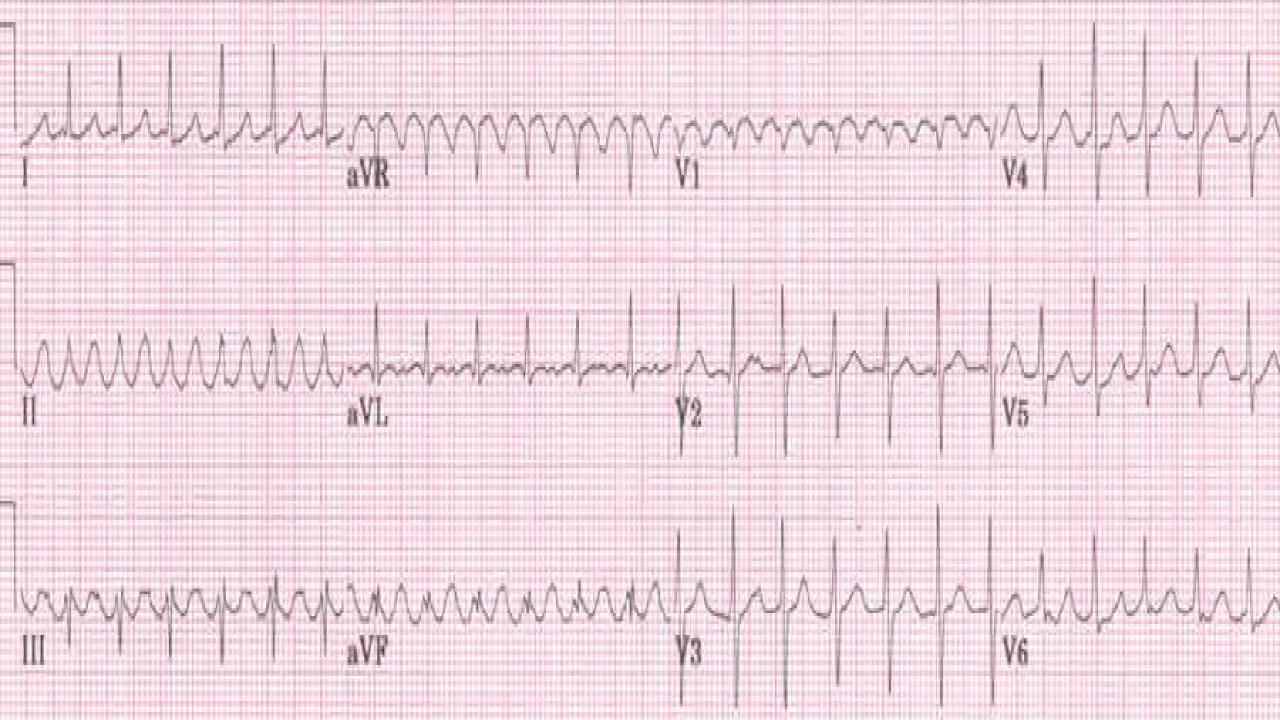


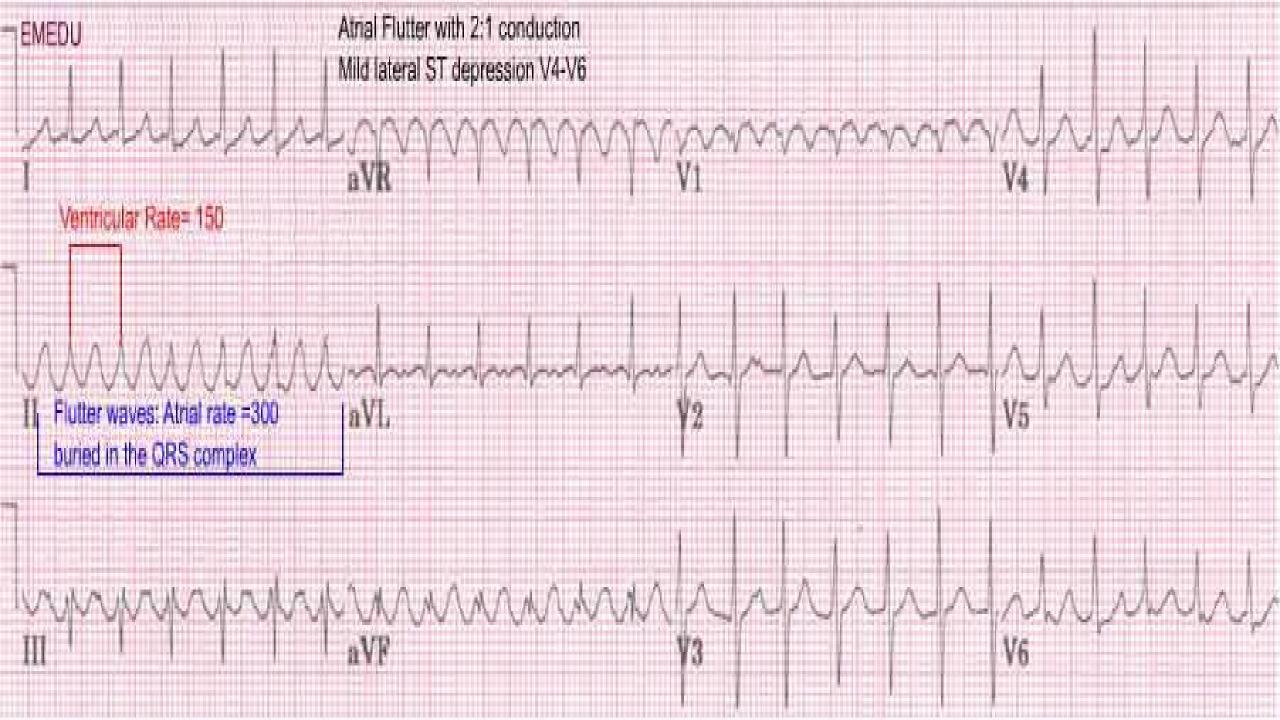
#### Normal



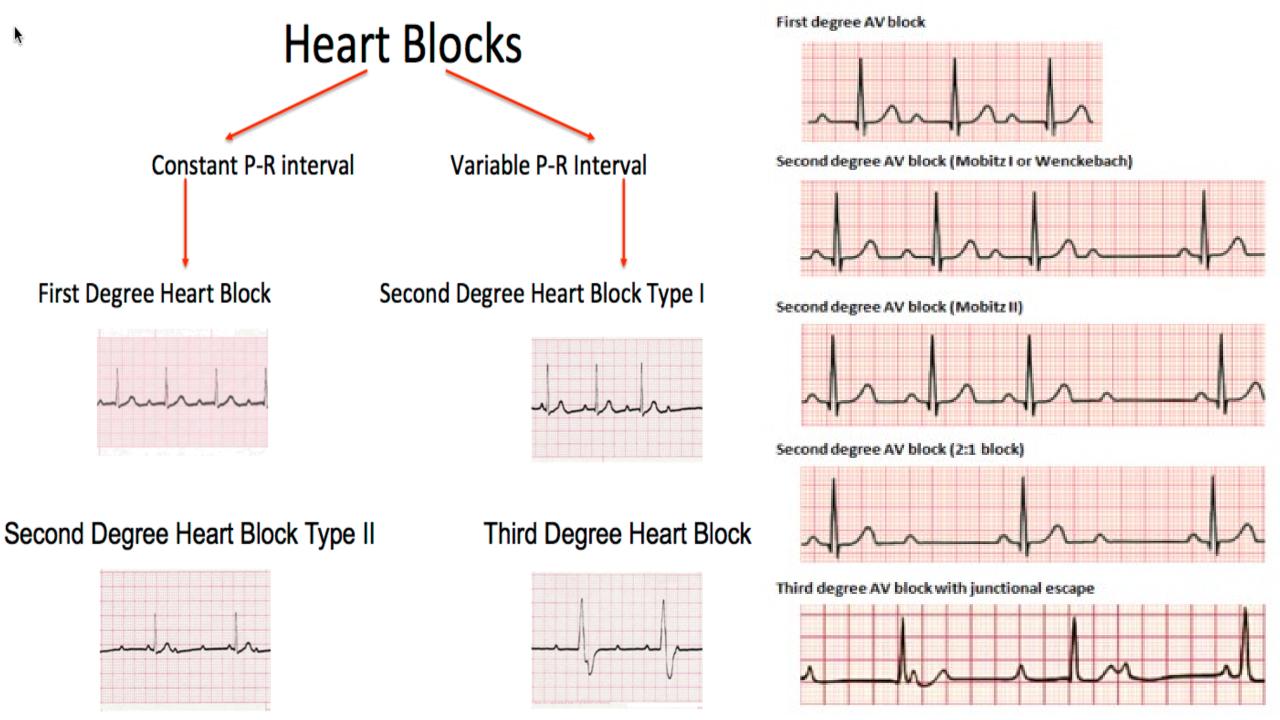
### **Ventricular Tachycardia (VT)**

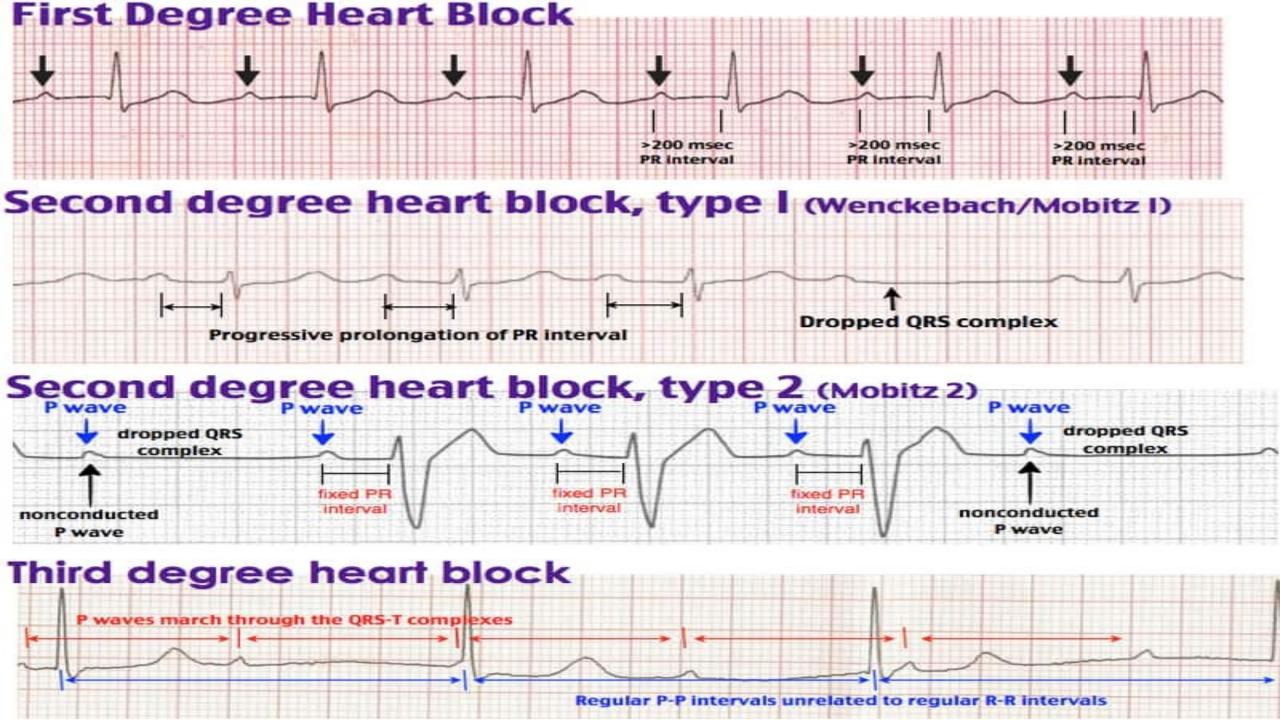












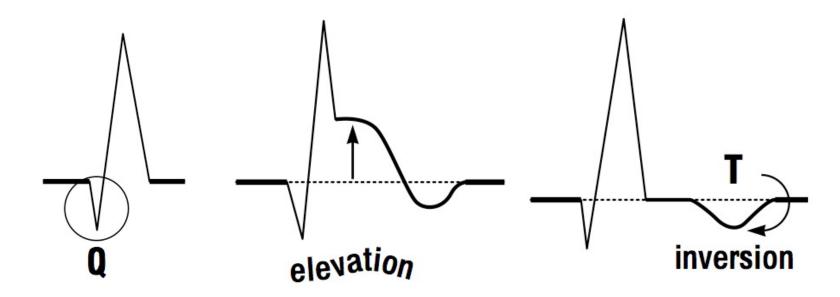
## **SUMMARY** Rate Rhythm Axis Intervals Hypertrophy Infarct

#### To summarize:

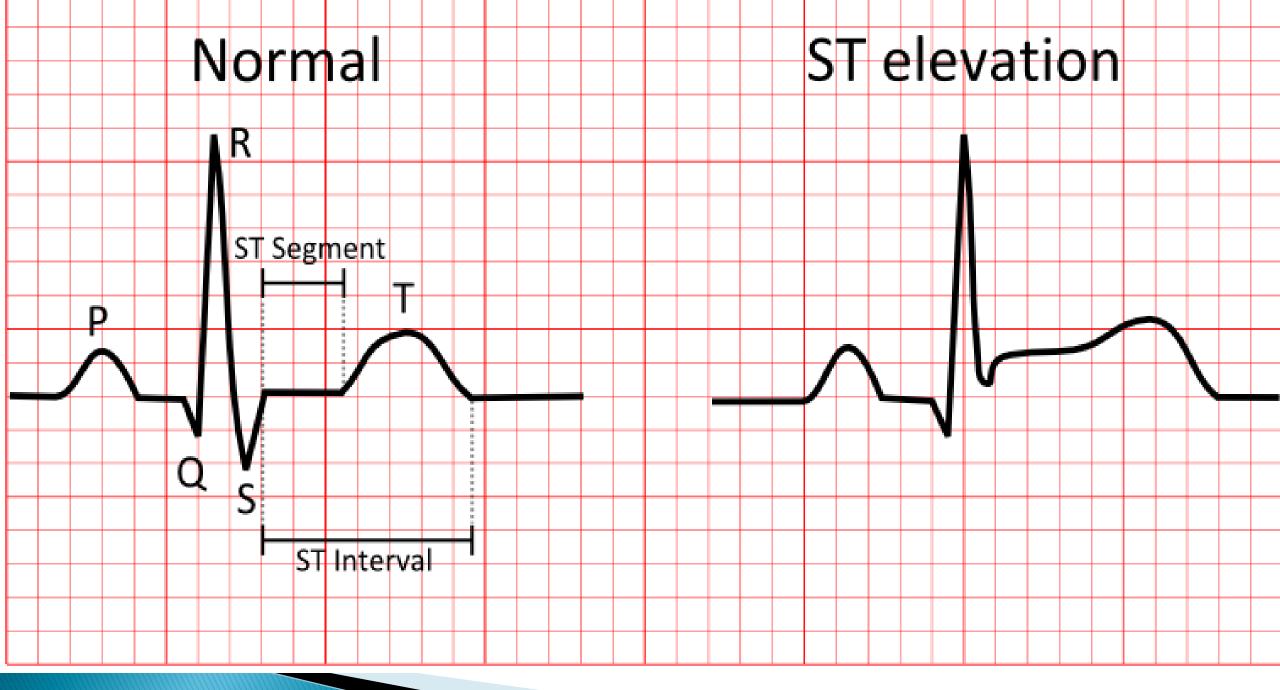
- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS

  - \_
  - \_
  - \_

- 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

 $I, V_2, V_3$ , and  $V_4$ 

I, aVL,  $V_5$ , and  $V_6$ 

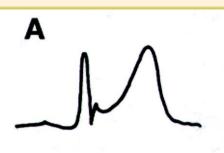
V<sub>5</sub> and V<sub>6</sub>

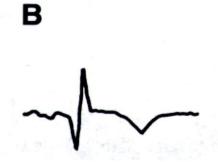
I and aVL (often with V<sub>5</sub>, V<sub>6</sub>)

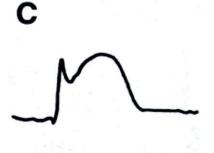
II, III, and aVF

II, III, aVF, and V<sub>6</sub>

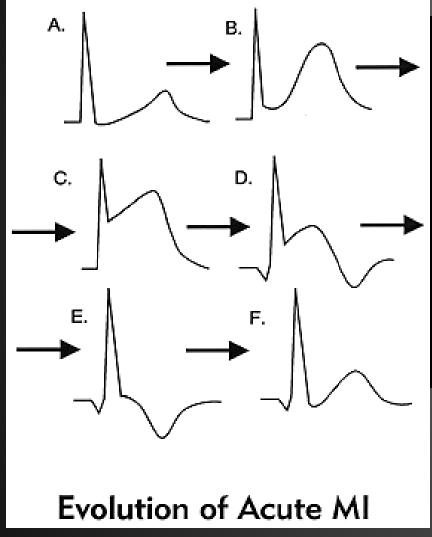
Reciprocal changes in V<sub>1</sub> and V<sub>2</sub>







## **MI** Location

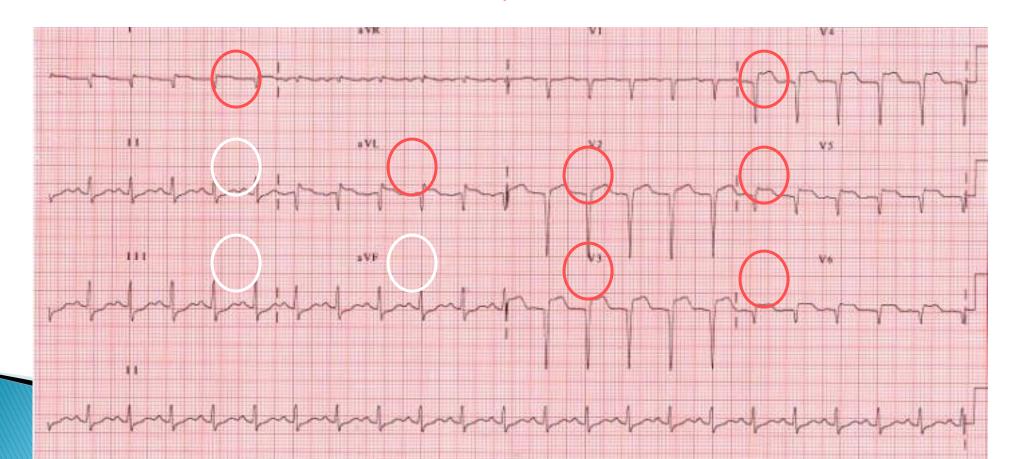


**Myocardial involvement EKG leads** Anterior **V2, V3, V4 (at least 2) Anteroseptal V1**, **V2**, **V3** (+**V4**) **Anterolateral V4, V5, V6 (+V3, +V2) Extensive Anterior** V1 through V6 (all) Lateral **V5**, **V6** (+**I**, +**aVL**) High lateral I, aVL **Inferior** II, III, aVF (at least 2) Inferolateral 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

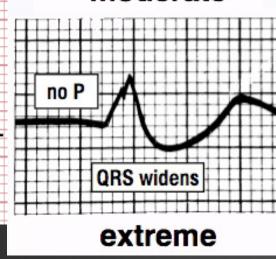
## ST Segment Elevation

#### DEPRESSED ST

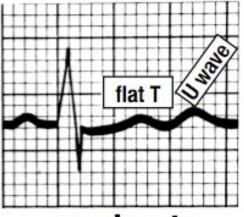
- Drooping valve (mitral valve prolapse)
- Enlargement or LV with strain
- Potassium loss (hypokalemia)
- Reciprocal ST depression (inferior MI)
- Embolism (PE)
- Subendocardial ischemia
- Subendocardial infarct
- Encephalon hemorrhage
- Dilated cardiomyopathy
- Shock
- Toxicity of digitalis, quinidine

# ST Segment Depression

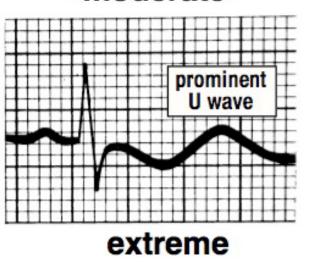
- Hyperkalemia • High K+ Peaked T Electro
- peaked wide moderate

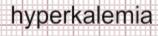


- Hypokalemia
  - Low K+
  - Flat T, U Wave



moderate





normal P waves



P waves flatten, prolonged PR



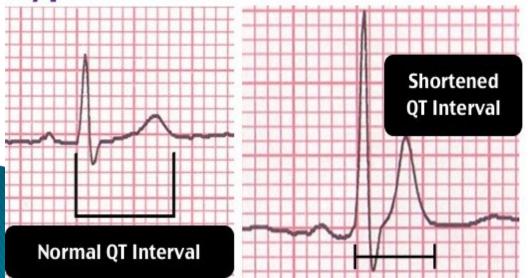
P waves disappear, bradycardia

### HYPERCALCEMIA

#### HYPOCALCEMIA

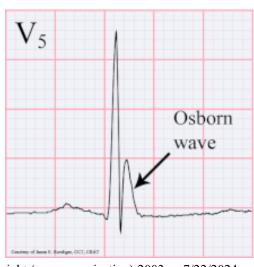


Hypercalcemia

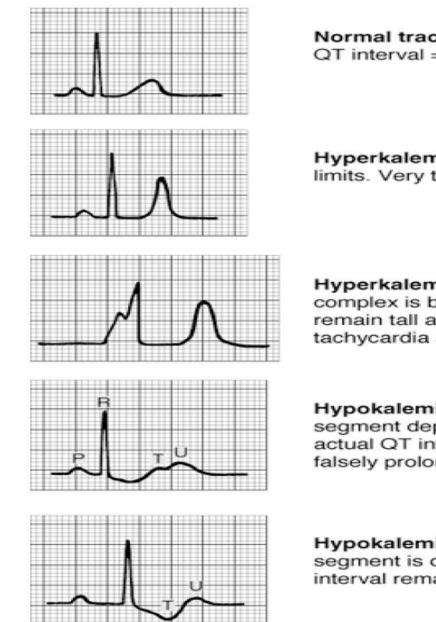


#### **Causes of Hypercalcemia**

- Malignancy
- · Milk-alkali syndrome
- · Vitamin D toxicity
- · Paget's disease
- Hyperparathyroidism
- Immobilization
- · Granulomatous disease
- Thiazide diuretics



pyright (your organization) 2003 7/22/2024



Normal tracing (plasma K<sup>+</sup> 4–5.5 meq/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 meq/L). The PR and QRS intervals are within normal limits. Very tall, slender peaked T waves are now present.

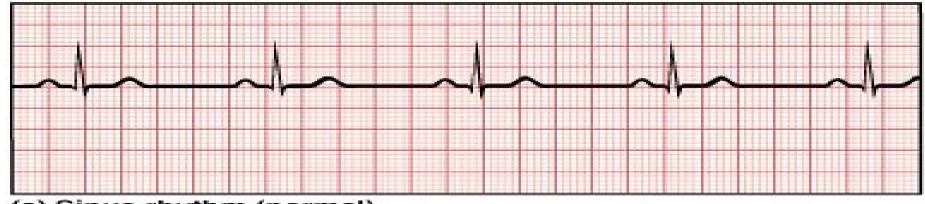
**Hyperkalemia (plasma K+** ±8.5 meq/L). There is no evidence of atrial activity; the QRS complex is broad and slurred and the QRS interval has widened to 0.2 s. The T waves remain tall and slender. Further elevation of the plasma K+ level may result in ventricular tachycardia and ventricular fibrillation.

**Hypokalemia (plasma K**<sup>+</sup> ±3.5 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.

**Hypokalemia (plasma K**<sup>+</sup> ±**2.5 meq/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<sup>+</sup> level and the ECG, assuming that the plasma Ca<sup>2+</sup> 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

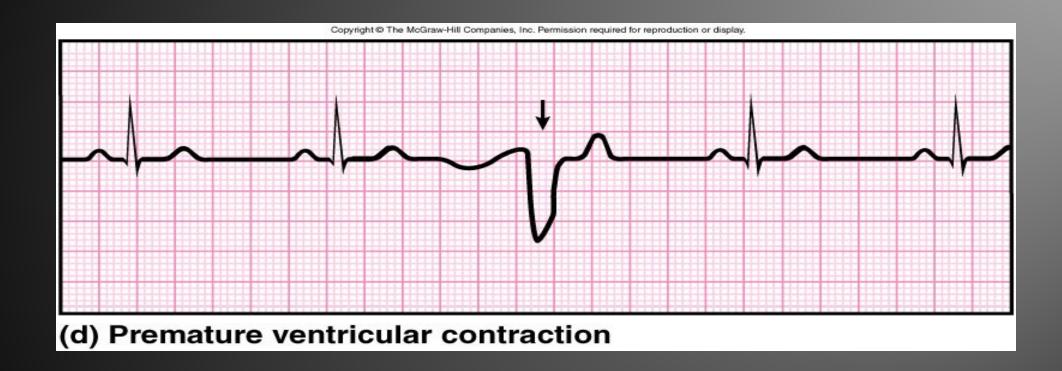


(a) Sinus rhythm (normal)



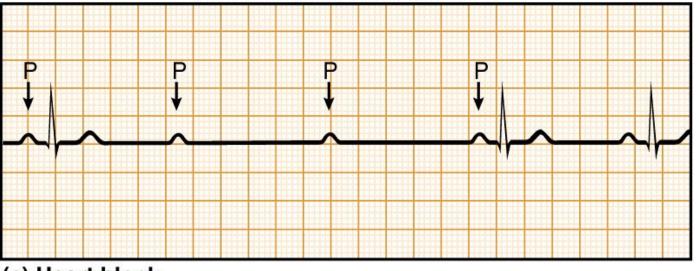
(b) Nodal rhythm – no SA node activity

# ECGs, Abnormal



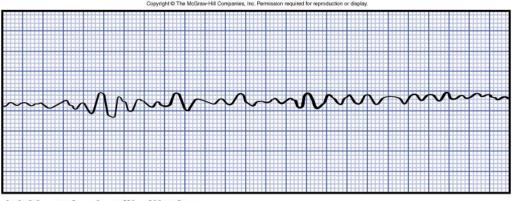
Extrasystole: note inverted QRS complex, misshapen QRS and T and absence of a P wave preceding this contraction.

# ECGs, Abnormal



(c) Heart block

### Arrhythmia: conduction failure at AV node



(e) Ventricular fibrillation

No pumping action occurs

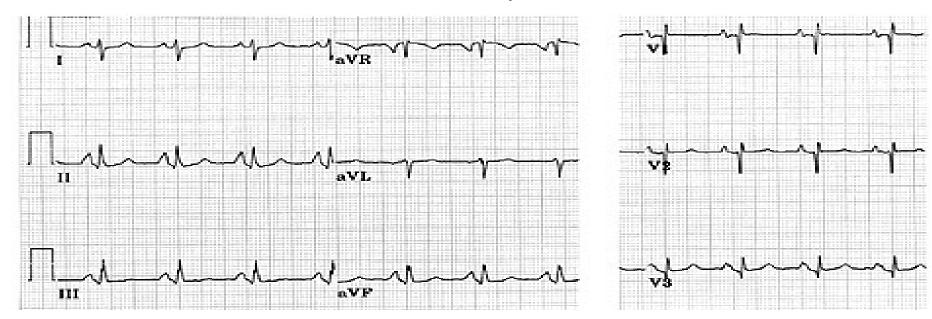
# THE ABNORMAL

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

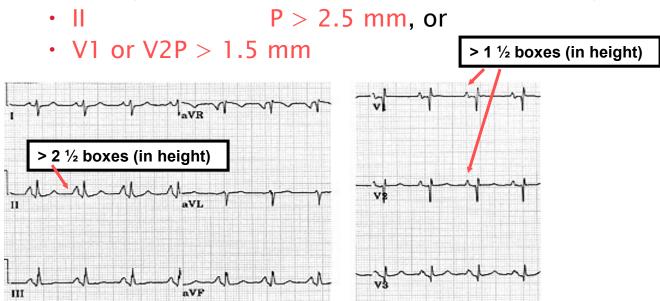
• Take a look at this ECG. What do you notice about the P waves?



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

### Right atrial enlargement

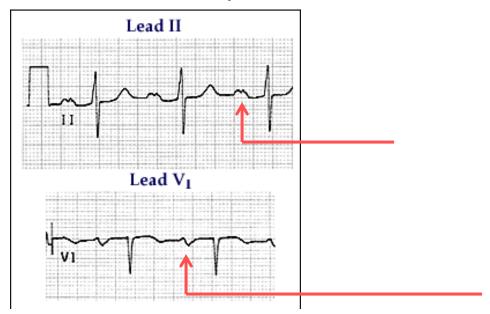
To diagnose RAE you can use the following criteria:



A cause of RAE is RVH from pulmonary hypertension.

### Left atrial enlargement

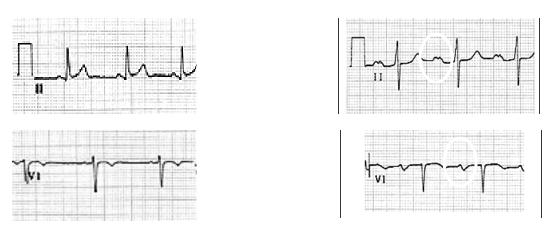
• Take a look at this ECG. What do you notice about the P waves?



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

#### Left atrial enlargement

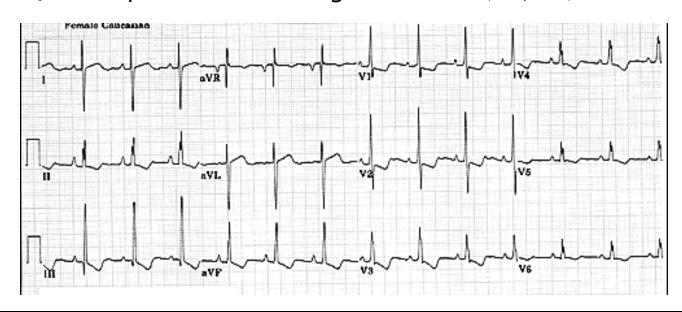
- To diagnose LAE you can use the following criteria:
  - II > 0.04 s (1 box) between notched peaks, or
  - V1 Neg. deflection > 1 box wide x 1 box deep



A common cause of LAE is LVH from hypertension.

### Right ventricular hypertrophy

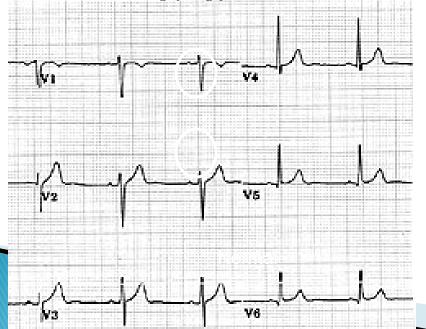
• Take a look at this ECG. What do you notice about the axis and QRS complexes over the right ventricle (V1, V2)?

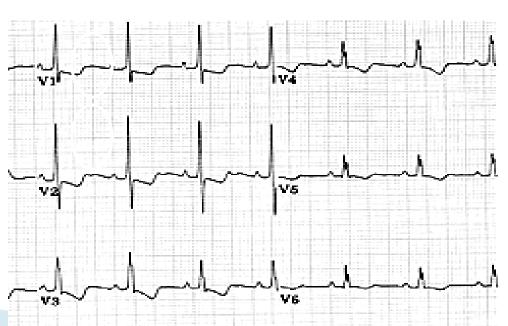


There is right axis deviation (negative in I, positive in II) and there are tall R waves in V1, V2.

### Right ventricular hypertrophy

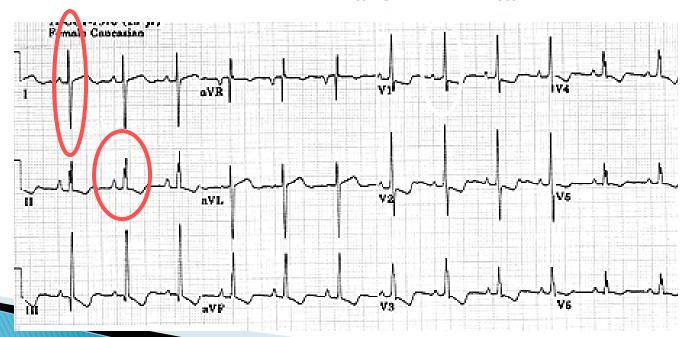
- Compare the R waves in V1, V2 from a normal ECG and one from a person with RVH.
- Notice the R wave is normally small in V1, V2 because the right ventricle does not have a lot of muscle mass.
- But in the hypertrophied right ventricle the R wave is tall in V1 V2





### Right ventricular hypertrophy

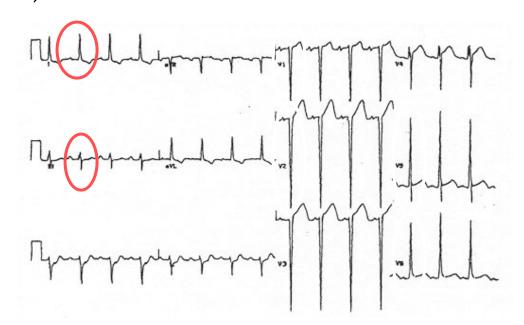
- To diagnose RVH you can use the following criteria:
  - · Right axis deviation, and
  - V1 R wave > 7mm tall



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)?

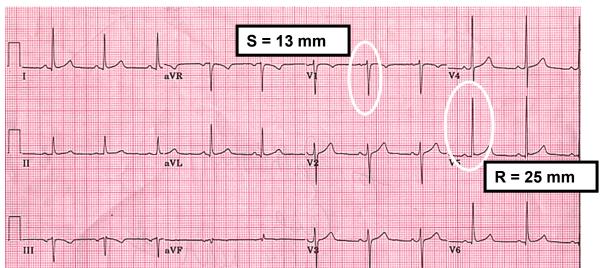


There is left axis deviation (positive in I, negative in II) and there are tall R waves in V5, V6 and

doon C wayaa in 1/1 1/2

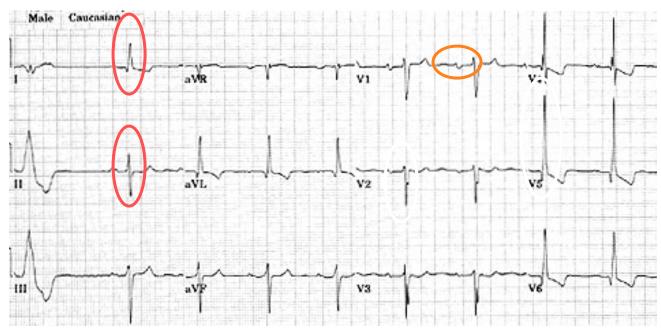
### Left ventricular hypertrophy

- 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

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