

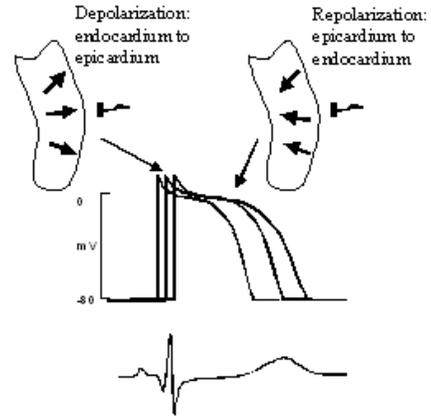
I. General Principles and Definitions

A. Depolarization: Electrical activation of the myocardium

B. Repolarization: Restoration of the electrochemical gradient of the myocardial cell creating a potential difference (voltage) across the cell membrane.

C. Sequence: Depolarization begins within the pacemaker cells of the sinoatrial (SA) node and travels through internodal tracts of to the left atrium and atrioventricular (AV) node. The signal then traverses the AVN to reach the Bundle of His, which divides into right and left bundle branches; left bundle branch divides into left anterior and posterior fascicles. The signal for depolarization is spread by this specialized conduction system to coordinate activation of contractile myocardium.

1. The spread of depolarization through the cardiac chambers creates the surface shifts in the electrical field that we record as a deflection on the ECG.



D. ECG: A galvanometer whose poles are a series of electrodes with six limb leads and six chest leads that records the electrical forces generated by the heart during depolarization and repolarization. The electrocardiogram is recorded on graph paper with 1 mm divisions. Each mm on the horizontal axis represents 40 msec and on the vertical axis 0.1 mV.

E. P wave: ECG deflection representing atrial depolarization.

F. QRS: ECG deflection representing ventricular depolarization.

G. T wave: ECG deflection representing ventricular repolarization.

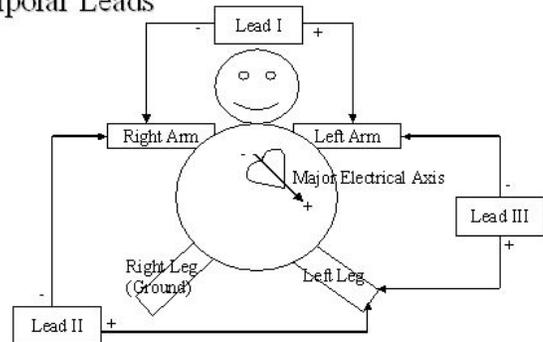
II. ECG Electrodes

A. Two lead arrangements are used for standard electrocardiography. The important concept to understand about their arrangement is that their ability to detect a change in the electrical field is limited. A lead arrangement is a “dipole” and may only detect field shifts that occur in the direction that the electrodes are “looking”, i.e., it can only see the component dipole vector of the total vector generated by the heart. Therefore, individual lead arrangements are placed to look in virtually every direction. Part of ECG interpretation is working backward to visualize both the direction and magnitude of the electrical field disturbance. The mechanism of doing so assumes that the human body is a uniform conductor and that lead arrangement is sufficiently symmetrical to allow use of a polar coordinate system. A detailed explanation follows under the heading Axis Determination.

B. Bipolar Lead: A potential difference is created between two electrodes. Disturbances in the electrical field are conducted to an amplifier and simple galvanometer whose deflection registers the changing electrical field.

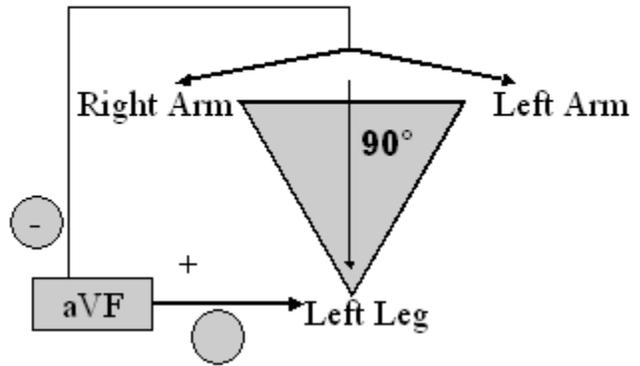
C. Unipolar Lead: An arrangement in which the electrodes attached to several locations on the body are attached to the negative terminal and a single “exploring electrode” is attached to the positive terminal.

Bipolar Leads



Unipolar Lead (aVF)

An example of unipolar leads is shown for aVF. The right and left arm electrodes are connected to the negative terminal of the ECG machine. The positive terminal is connected to the left foot. As a result, the ECG machine can "see" only along an axis that looks from the average location between the left and right arms toward the left foot.



D. Standard Limb Leads: These

constitute the various arrangements of the bipolar and unipolar leads attached to the limbs denoted I, II, III, aVR, aVL and aVF. Since their arrangement on the limbs is consistent with respect to anterior-posterior positioning, they explore the electrical activity in the heart in the anatomic coronal plane which is referred to as the frontal plane.

1. Bipolar Limb Leads: I, II, III; form what we assume to be an equiangular triangle (Einthoven's Triangle) surrounding the heart. Given that assumption, we may assign a direction or polar coordinate to each lead.

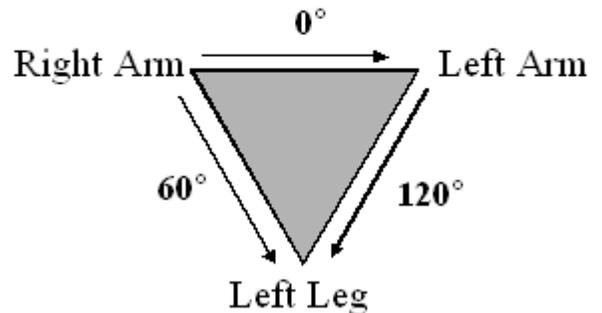
- a) Lead I: negative electrode on the right arm and positive electrode on the left arm.
- b) Lead II: negative electrode on the right arm and positive electrode on the left leg.
- c) Lead III: negative electrode on the left arm and positive electrode on the left leg.

2. This allows for something very interesting. If a given vector's components are measure by leads I, II and III, because of their arrangement, the net vector measured in lead I added to the net vector measured in lead III will provide the net vector measured in lead II.

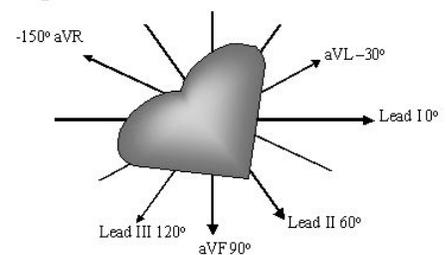
3. Unipolar Limb Leads: the signal recorded is low amplitude. Therefore, the voltage is "augmented" to correct for the parallel resistance of two electrodes attached to the negative terminal. This gives these leads the name, augmented voltage (aV) aVR (exploring electrode right arm), aVL(exploring electrode left arm), aVF(exploring electrode foot/left leg); form a set of axes 60° apart but are rotated 30° from the axes of the standard limb leads.

- a) You may notice that the components of aVR and aVL oriented on the 0 axis would give you lead I. Actually,
- b) $\text{Lead I} = \text{aVL} - \text{aVR}$
- c) $\text{Lead III} = \text{aVF} - \text{aVL}$
- d) $\text{Lead II} = \text{aVF} - \text{aVR}$

Einthoven's Triangle



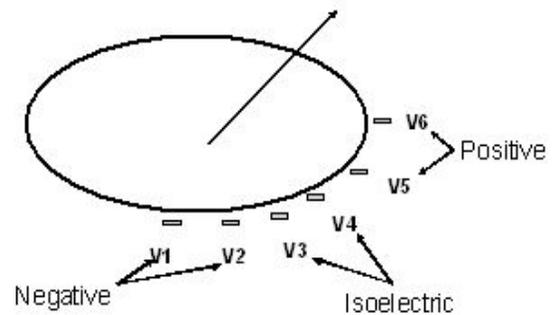
Directionality of Bipolar and Unipolar Leads



E. Chest Leads: An arrangement of unipolar leads whose negative terminal is the left arm, right arm and left leg with the “exploring” or positive terminal attached to the chest. V1-6 explore the electrical activity of the heart in the horizontal plane; i.e., as if looking down on a cross section of the body at the level of the heart.

1. V1: 4th intercostal space just to the right of the sternum.
2. V2: 4th intercostal space just to the left of the sternum.
3. V3: halfway between V2 and V4.
4. V4: 5th intercostal space in the mid-clavicular line.
5. V5: anterior axillary line at the same level as V4.
6. V6: mid axillary line at the same level as V4 and V5.

Chest Leads/Horizontal Plane



The standard chest leads are shown with the large arrow representing the normal mean electrical axis of the QRS. Its somewhat posterior direction (due to the dominance of the LV) is the reason for the variation in QRS appearance across the precordium.

III. Intervals

A. Dimensions of Grids on ECG Paper

Paper: Time is the horizontal axis. The paper speed is 25 mm/sec.. Therefore, small blocks are 40 msec duration large blocks are 0.2 seconds in duration. The vertical axis represents the voltage measured. Each small block represents 0.1 mV.



B. Estimation of Heart Rate

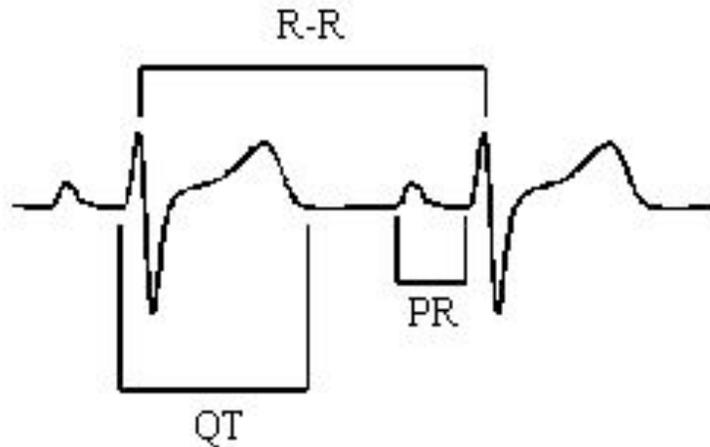
1. Heart rate may be measured in any of three ways.
2. $60 \text{ sec/min} \div 0.04 \text{ sec/mm} = 1500 \text{ mm/min}$. Therefore dividing the number of small squares in an R-R interval into 1500 provides the number of RR intervals/minute.
3. $60 \text{ sec/min} \div 0.2 \text{ sec/mm} = 300 \text{ lg boxes/min}$. Therefore dividing the number of large squares in an R-R interval into 300 provides the number of RR intervals/minute. Because the math is pretty simple, you can memorize the first few values and simply count down by the number of large boxes, 300, 150, 100, 75, 60, 45 but then it gets tougher to remember 43, 38, 33, 30, etc.
4. If the rhythm is irregular, you should take an average of several RR-intervals or more simply count the beats in a 10 second tracing and multiply by 6.

C. RR interval: The time between peaks of the R wave that represents the rate of ventricular depolarization.

D. PR interval: onset of P wave to first deflection of the QRS representing the time required for conduction to proceed through the atria, AV-node and HIS/Purkinje network.

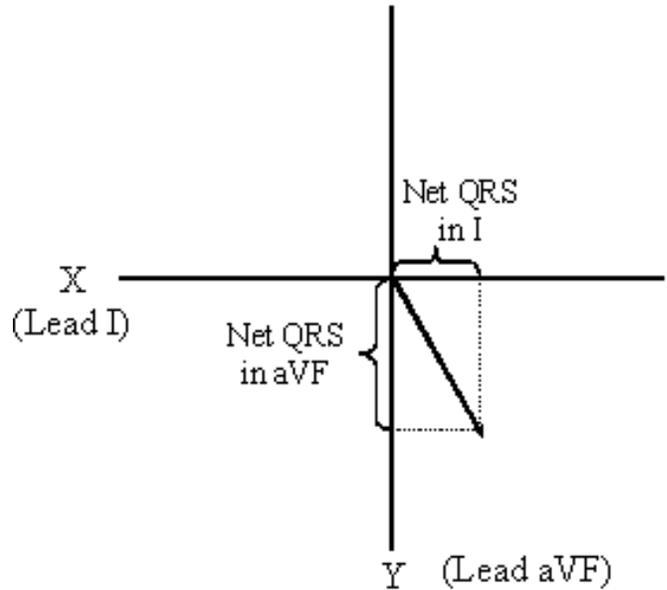
E. QRS duration: First deflection of the QRS to the last rapid deflection of the QRS measured in the lead where you see it best. This represents the time required for activation of the ventricles.

F. QT interval: The time from the first deflection of the QRS to the last deflection and return to baseline of the T wave. This reflects the time required to depolarize and repolarize the ventricles. The QT interval represents the average duration of the monophasic action potential throughout the ventricles. Because this is very dependent upon heart rate, the QT is often reported as corrected for heart rate (QTc). Of the many methods of correction available, Bazett's formula is most frequently used: $\text{measured QT} / \sqrt{\text{prior RR interval in seconds}}$.

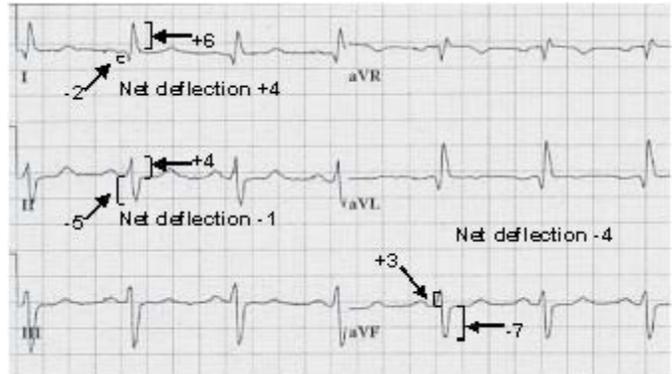


IV. Determination of Axis

A. Now that we have a series of leads or electrode combinations that give us some direction, we can describe what has happened using these coordinates. What were going to see with our leads is a component of the heart's electrical vector in a given plane, either frontal or horizontal. As you know, a vector has both amplitude and direction. We'll concern ourselves primarily with direction. For example, see the image to the right showing a vector in the left lower quadrant. The vector has components in both the X and Y axes. The vector can be described by its location on the graph, by a linear equation or by amplitude and polar coordinates. If we just use the coordinates we might describe the vector as directed toward 60° . Conveniently substituting frontal plane leads I and aVF, we may arrive at the polar coordinate using, $\text{Arctan}(aVF/I)$. Since none of us really has time for such calculations, there is fortunately a much easier way.



B. Mean Electrical Axis: Defined as the mean vector of any disturbance in the electrical field. It may be determined for the atria (p-wave) or the ventricles (QRS), for depolarization (QRS) or repolarization (T-wave). The mean electrical axis of the QRS and T-waves frequently provides critical information leading to a diagnosis. The first step in determining axis of the p, QRS or T wave is to determine the net deflection of the wave in a particular lead. This is shown for the QRS in the figure to the right.



1. Short version

- a) Check lead I.
- b) If lead I is negative, right axis deviation
- c) if I is positive, check lead aVF
- d) if lead F is positive, axis is normal.
- e) if aVF is negative, check lead II.

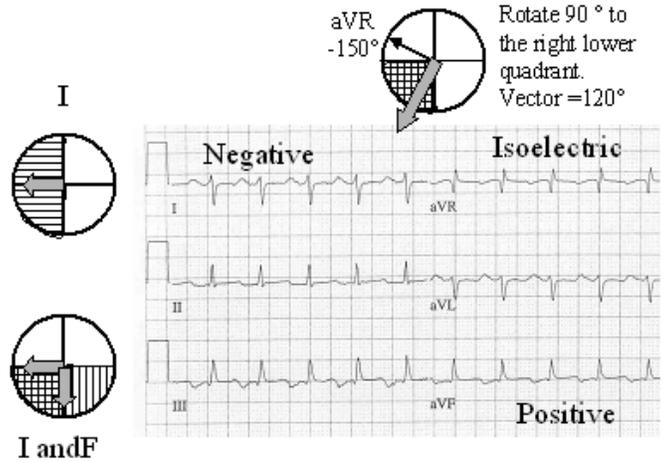
	Lead I	Lead aVF	Lead II
Normal			
Left Axis			
Right Axis			
Northwest Axis			

(i) if lead II is positive, the axis is normal.

(ii) if lead II is negative, there is left axis deviation.

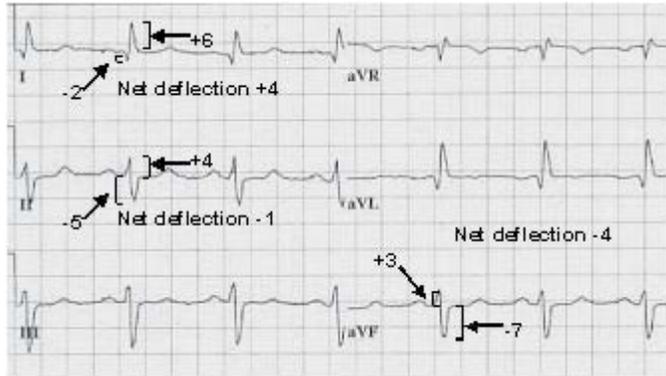
2. Estimating the numerical value of the mean axis using polar coordinates.

- Use leads I and aVF to assign the vector to a quadrant.
- Find the lead that is isoelectric (net deflection closest to zero).
- Using the known vector of the isoelectric lead, add or subtract 90° depending upon the quadrant that the mean vector has been assigned to.



Shown right is determination of a numerical axis estimate. Lead I has a net negative deflection while aVF is positive placing the axis somewhere in the left lower quadrant (shown in upper left). Lead aVR is closest to isoelectric. Its known vector is -150° . In order to be in the proper quadrant, the vector 90° distant to aVR is 120° .

Using the same method from the example used for the simple method shown above, the mean QRS vector is directed to the left upper quadrant. The isoelectric lead is again aVR. Therefore, the numerical estimate of the mean QRS vector is -60° .



C. Normal Axis: A mean vector between $+105$ and -30 degrees.

D. Right Axis Deviation: A mean vector of $> +105$ degrees.

E. Left Axis Deviation: A mean vector more negative than -30 degrees. Determining the axis of the mean vector:

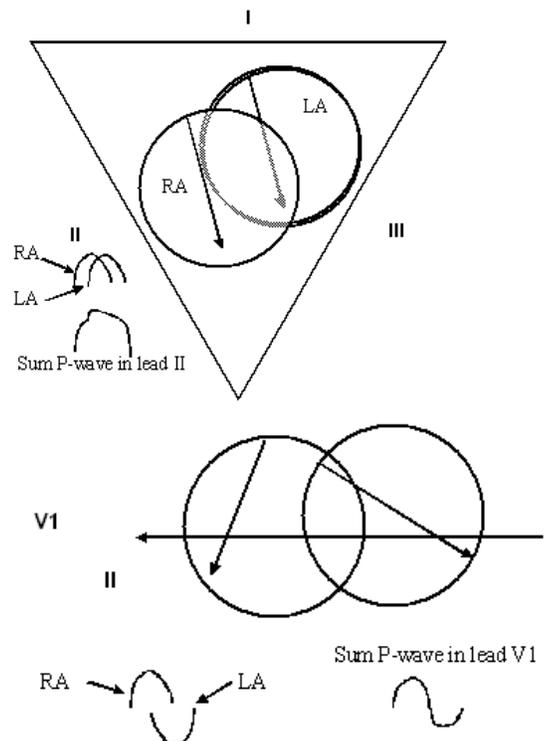
V. Hypertrophy

A. Generally, hypertrophy of a chamber will result in a change in wave amplitude, wave duration and often in mean electrical axis.

B. Atrial Enlargement: To evaluate atrial enlargement, look at the P waves in leads II and V1. The right atrium generates the early portion of the P wave, the left atrium generates the second half.

1. **Lead II:** Generally parallel to the axis of the atrial depolarization vector force.

2. **Lead V1:** Generally closest to the atria and perpendicular to the axis of the atrial depolarization vector force. Right atrial depolarization is seen as the early positive deflection and left atrial depolarization the



subsequent negative deflection.

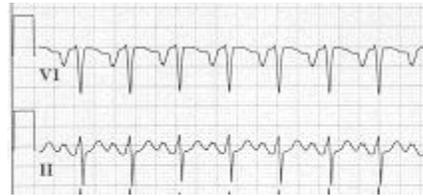
3. Right Atrial Enlargement:

Tall, pointed P wave, >2.5 mm tall in lead II. Early force >1.5 mm tall in V1.



4. Left Atrial Enlargement:

Increased P wave duration, >0.12 sec with distinct "humps" from each atrium. The humps should be >0.04 sec apart. In V1, the left atrium produces a deep terminal deflection >1mm deep and 1 mm wide.

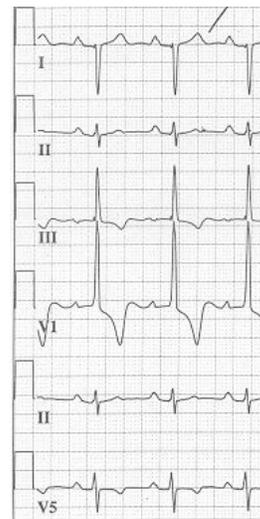


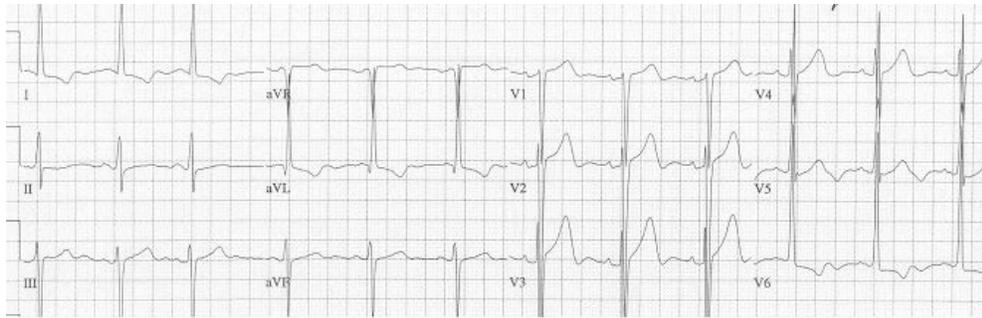
C. Ventricular Hypertrophy: Hypertrophy produces increased voltage from the affected ventricle that shifts the axis in the horizontal plane and may result in conduction abnormalities that result in axis deviation. Either conduction abnormalities or changes in energy supply to the affected ventricle frequently produce T wave changes.

1. Right Ventricular Hypertrophy (RVH): Anterior rotation of the axis in the horizontal plane or $R > S$ in lead V1 or $S > R$ in lead V6. Often accompanied by RAE, incompleter RBBB and T inversion in V1-3.

Right ventricular Hypertrophy. The deep net negative deflection in I identifies right axis deviation (isoelectric in lead II, therefore approximately 150°). Combined with $R > S$ in V1, $S > R$ in V5(6) and the "strain" T-wave appearance in V, RVH is fairly certain. Taken from a patient with pulmonary valve stenosis and RV pressures in excess of systemic pressures.

2. Left Ventricular Hypertrophy (LVH): Posterior rotation of the QRS in the horizontal plane. LVH is present when the sum of the S wave in V1 and the R wave in V5 or V6 > 35 mm. Since left axis shift is frequently present in the frontal plane, its often helpful to look at the leftward leads I (>15 mm) and aVL (>11mm) or to combine the S in V3 with the R in aVL (>20 female, >24 male). LAE and T inversion in I, aVL and V5,6 are frequently present.

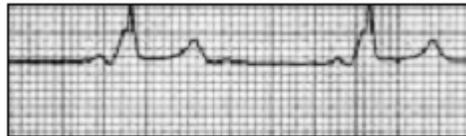




VI. The wide QRS

A. Pre-Excitation Syndromes: Pre-excitation syndromes refer to clinical Conditions in which the wave of depolarization bypasses the atrioventricular node as it passes from the atria to the ventricles. The time required for the wave to leave the sinoatrial node and arrive at ventricular muscle (P-R interval) is, therefore, shortened but the normal, specialized conduction system is not used giving rise to the wide, slurred QRS.

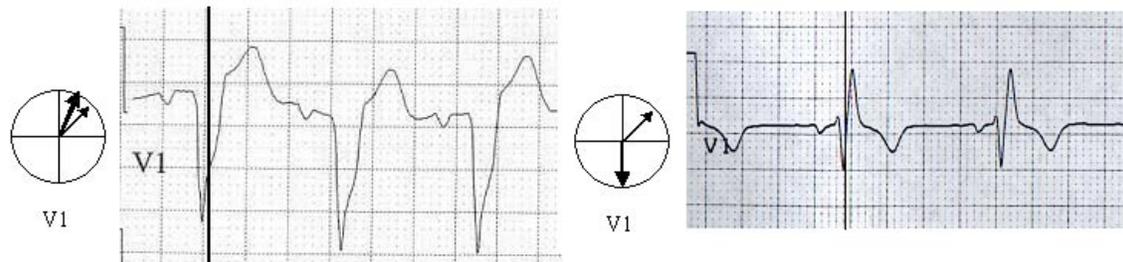
1. Wolff-Parkinson-White Syndrome (WPW): Patients with WPW possess an accessory pathway of depolarization, the *bundle of Kent*. Three electrocardiographic criteria for WPW are: (1) a short P-R interval, (2) a wide QRS complex, and (3) a delta wave. The QRS complex is widened by the delta wave in exactly the same amount as the P-R interval is shortened. The delta wave is a slurring of the initial portion of the QRS complex produced by early depolarization. Episodes of tachycardia are required for the diagnosis of WPW syndrome. Simply observing a delta wave is insufficient for the diagnosis and is termed “pre-excitation”.



B. Intraventricular Conduction Disturbances:

1. In the normal process of ventricular depolarization, the electrical stimulus reaches the ventricles by way of the atrioventricular (AV) junction. Then the depolarization wave spreads to the main mass of the ventricular muscle by way of the right and left bundle branches. The right bundle branch is undivided, while the left divides into anterior and posterior fascicles. Normally the entire process of ventricular depolarization occurs in less than 0.01 seconds. The QRS is generated by the change in electrical activity as this signal for depolarization spreads through the thickness of the left and right ventricles. Any process that interferes with normal depolarization of the ventricles may prolong the QRS width. In addition, abnormal depolarization begets abnormal repolarization. So, as a general rule, when a block is present, the T-wave points the opposite direction of the terminal forces of the QRS.

2. Block: QRS duration >0.12 sec (three small boxes) To determine the site of block, find the vector of the terminal 0.06 sec of the QRS in the horizontal plane. If its going to the back of the body, that's the vector of the left ventricle, LBBB. If its coming to the front of the body, that's the right ventricle, RBBB.

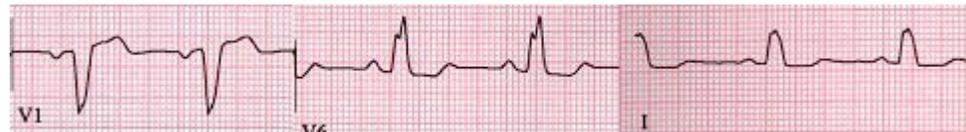
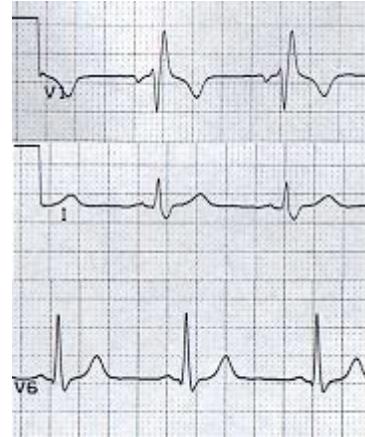


Examples of two blocks are shown with the vector of the first ½ of the QRS (small arrow) and last ½ (fat arrow). On the left, the wide QRS has been divided in half. The last half of the QRS is negative in V1 suggesting that the block results in an activation vector directed to the posterior of the body (away from V1). This vector is typical of the left ventricle. Therefore, the block has occurred in the left ventricle and may be in the left bundle branch. On the right, the last half of the QRS is directed toward V1, a vector typical of the right ventricle suggesting right bundle branch block.

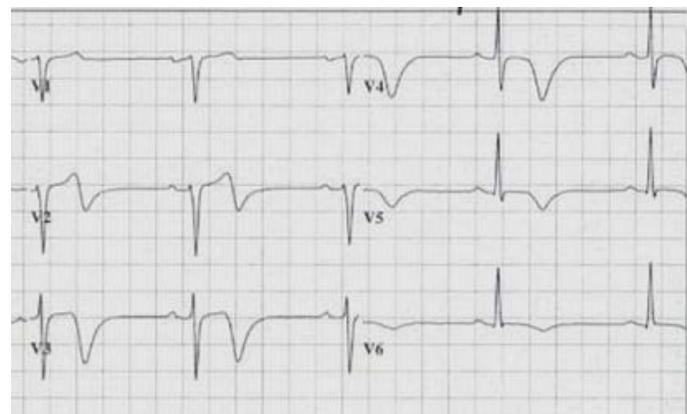
a) Right Bundle Branch Block (RBBB): RSR' in V1 and deep, wide S in I, aVL and V6. RBBB may occur in otherwise normal individuals. Activation of the left ventricle is normal. A diagnosis of myocardial infarction is possible when RBBB is present.

b) Incomplete RBBB: This shows the same QRS pattern as a complete RBBB; however, the QRS duration is between 0.1 and 0.12 seconds.

c) Left Bundle Branch Block (LBBB): Blockage of conduction in the left bundle branch prior to its bifurcation results primarily in delayed depolarization of the left ventricle. Wide, slurred RR' configuration in lead I, aVL and V6. Unlike RBBB, LBBB always is a sign of organic heart disease. The activation of the left ventricle is so altered by LBBB that infarction may not be visualized on the ECG when LBBB is present.



VII. Myocardial Ischemia produces three principle changes in the ECG, abnormal T waves,

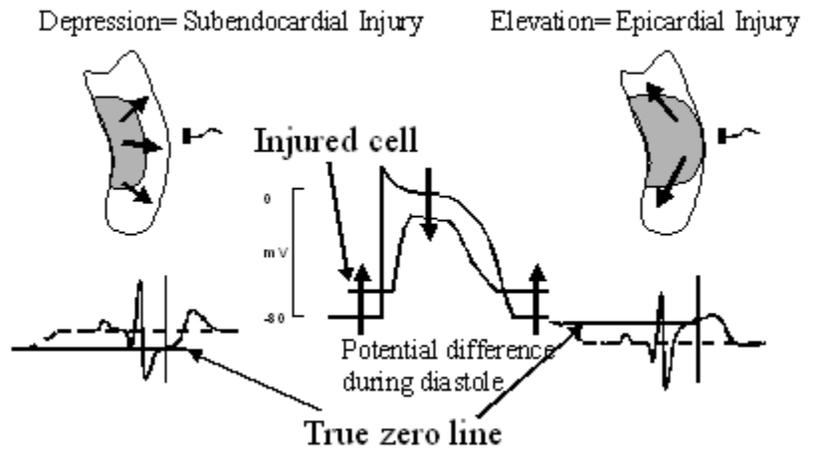


ST segment deviation and abnormal Q waves.

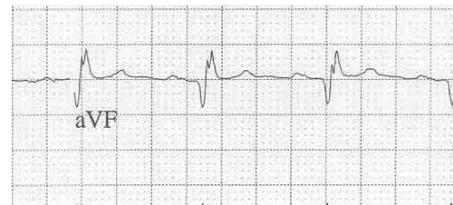
A. T inversion- With normal repolarization, the T-wave should generally be positive where the QRS is positive. Abnormal repolarization shifts the T-wave vector away from the affected region. *The T-wave runs away from trouble.* Repolarization is energy dependent and is most sensitive to ischemia. Therefore, abnormal repolarization felt to be the result of inadequate blood flow is termed *ischemia*. Ischemic T inversion is often symmetrical and associated with a long QT interval.

B. ST-deviation- deviation of the ST segment is in part due to inability of a region of myocytes to achieve and maintain resting ion gradients. Due to different concentrations of charged ions in the region, the heart acts as a battery during diastole and may assist current flow. When depolarization is complete, there is no longer any difference in electrical potential between any two regions of the heart and the ST segment represents the “true” zero line. However, we don’t calibrate to zero. Rather, we compare the ST segment to the baseline between the T wave and P wave. Therefore, it appears that the ST segment has moved. Inability to maintain resting gradients is termed injury. Injury may be,

1. *Subendocardial-* ST depression
2. *Epicardial-* ST elevation



C. Q-wave- The initial negative deflection of the QRS. If a QRS begins with an R wave, the next negative deflection is an S wave. When no R wave develops and the complex begins and remains a negative deflection, it is termed a QS complex. A pathological Q wave denotes absence of depolarization of a region of myocardium that usually reflects infarction/fibrosis of the region.

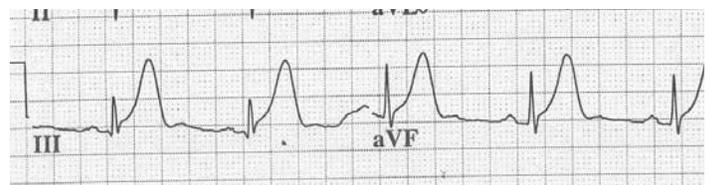


D. Myocardial Infarction

1. The pathological Q: A region of myocardium that has infarcted can no longer contribute to the vector of depolarization. As a result, the net vector points away from the region. The activation sequence of the heart develops in the first 0.04 sec. Therefore, a Q during this period may reflect infarction. Generally the Q of infarction is at least one box wide and >30% the size of the subsequent R wave though this rule isn’t written in stone. Keep in mind that a normal ECG may exhibit small Q waves in leads I, aVL, III, aVR, V5, and V6.

2. Time sequence of infarct evolution on the ECG:

- a) *Acute Injury-* ST elevation or hyperacute Twaves (below). Onset seconds, duration up to four

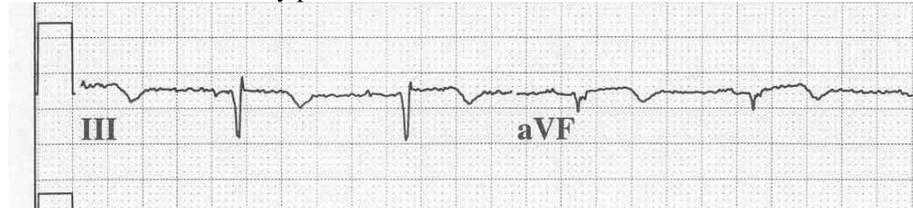


hours.

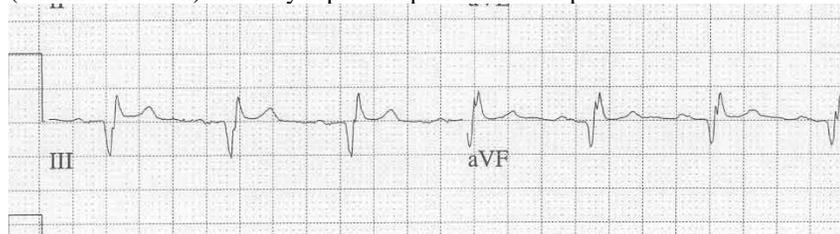
b) Acute infarct- Q waves appear from 1-4 hours after onset while ST elevation persists. As the ST segment comes down, Q waves deepen and T waves invert. Q with ST elevation may persist for up to two weeks.



c) Infarct age uncertain- When ST-segments have normalized, the Q and T inversion remain. This combination generally appears from a few hours to two weeks after onset and may persist forever.



d) Infarct Old- Q wave with normally directed T wave. This combination may appear from 6 months to one year after onset. Occasionally it is seen very early (within one week) and may represent peri-infarction pericarditis.



3. Localization of Myocardial Infarction: The left ventricle dominates early repolarization. Therefore, only infarction of the left ventricle may be reliably identified from the ECG. An infarct is described based upon Q wave localization of anatomic location: septal, anterior, antero-lateral, high-lateral, inferior, and posterior.

a) Anterior Infarction: Subdivided into septal, anterior, anteroseptal, and anterolateral infarctions. The causative lesion is in the LAD.

(i) *Septal: V1,2*

(ii) *Anterior: V3,4.*

(iii) *Anteroseptal Infarction: V1 through V4.*

(iv) *Anterolateral Infarction: V5,6 and sometimes I, aVL.*

b) High Lateral: I, aVL

c) Inferior Infarction: II, III, aVF.

d) Posterior Infarction: Does not generate Q wave in the conventional 12-lead ECG since there are no posterior exploring electrodes. Instead, subtle reciprocal changes in the magnitude of R waves in V1 and V2 occur. In posterior infarction, the R waves in V1 and V2 become 0.04 sec in duration and taller than or equal to the S waves ($R/S > 1$). Posterior infarction



frequently accompanies inferior infarction.

4. Classification of Infarction using the ECG

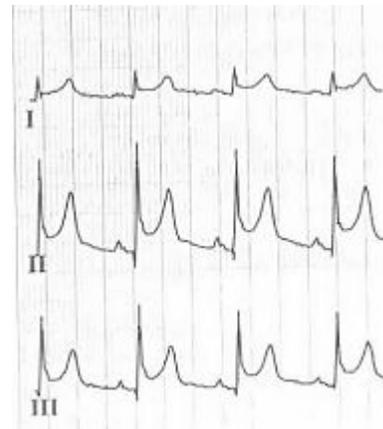
a) Non-Q wave Infarction: Diagnosis is established by typical symptom complex and abnormal cardiac marker determinations or autopsy. No Q wave is present. This may occur because of posterior infarction, LBBB or infarctions that do not greatly disturb early activation. The designation has little clinical significance but is commonly used.

b) ST-MI: Infarction marked by the presence of an injury current on the presenting ECG. Treatment is usually rapid. Q waves frequently follow.

c) Non-ST-MI: Infarction that is not announced by the presence of an injury current during its evolution. A very important designation because the diagnosis cannot be established in time to do anything about it. Q waves may not appear on subsequent ECG's.

d) Caveat: Old terms such as subendocardial and transmural infarction persist. These diagnoses cannot be established by ECG and require imaging study or autopsy. There is no place for their use in ECG diagnosis.

VIII. Pericarditis is inflammation of the pericardial lining or within the pericardial space. It produces an injury current (epicardial) or ST segment elevation whose vector oriented with the long axis of the heart. As a result, ST segment elevation may be apparent in almost every lead except aVR.



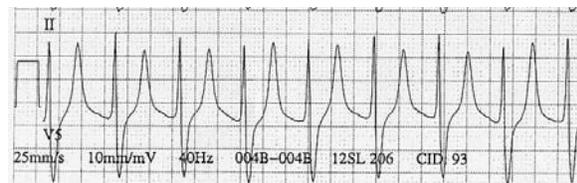
IX. Patterns Caused by Drug and Electrolyte Effects

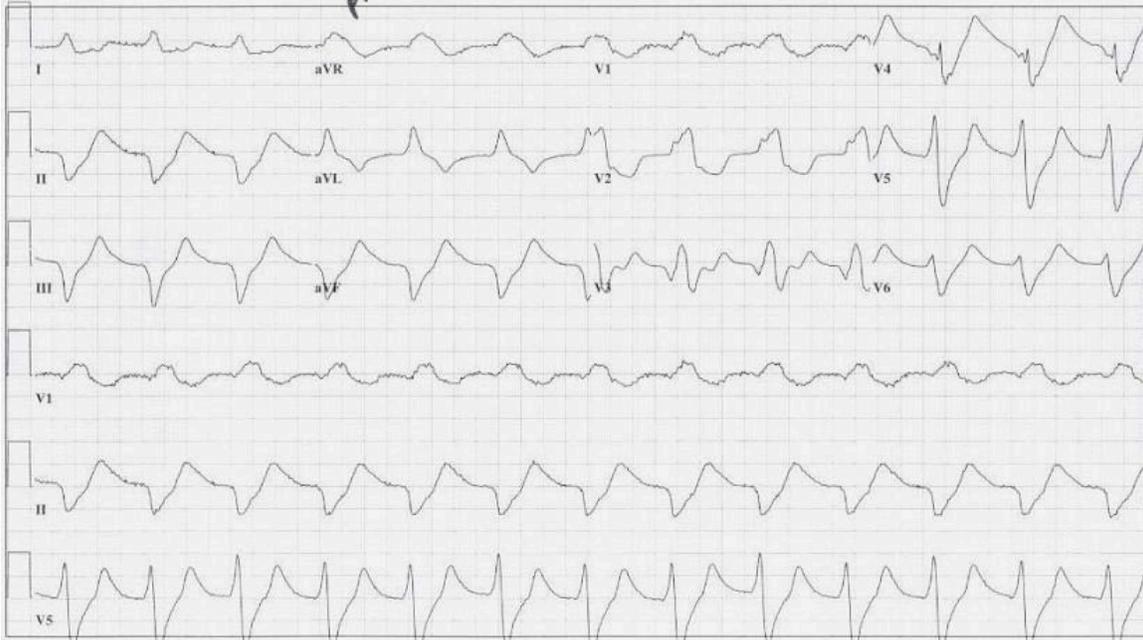
A. Background: Anything that may impact an ion channel or the development of the resting electrochemical gradient of myocytes may alter the ECG.

B. Digitalis: Na/K ATPase inhibitor that produces calcium loading and alters the ability of myocytes to generate normal resting gradients. Therefore, it produces changes in the ST-T contour, and enhancement of ectopic automaticity. The characteristic scooping of the ST-T segment is seen maximally in leads II and V5 and has been described as “Salvador Dali’s moustache”. With toxicity, digitalis can cause virtually any arrhythmia and all degrees of atrioventricular block.

C. Anti-arrhythmic drugs: These drugs generally affect the Na channels altering conduction and prolonging the QRS duration or K channels delaying repolarization and prolonging the Q-T interval.

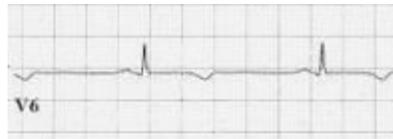
D. Potassium: *Hyperkalemia* produces tall, peaked T waves, widening of the QRS complex, and diminution of P wave amplitude. *Hypokalemia* produces flattening of the T waves and U waves more than 25% the size of the T wave or often larger. ST segment depression may occur.





The "sine wave" QRS of hyperkalemia. Potassium=8.5 meq/L

E. Calcium: Hypercalcemia shortens ventricular repolarization time, resulting in a shortened Q-T interval. Hypocalcemia prolongs the Q-T interval leaving the T-wave duration normal. The appearance is described as long Q to onset of T.



X. Non-specific ST-T Wave Abnormalities of the ST segment or T wave refer to non-diagnostic changes. By convention, more than 1mm of ST segment deviation is required to identify injury. ST-segment deviation less than 1 mm is termed a non-specific ST segment abnormality. Non-specific ST-segment changes are common with digitalis therapy and with hypokalemia. When T waves are flat throughout the ECG or when inversion is less than 1 mm, T wave abnormalities are termed non-specific. Also, a change in the T wave axis may be present that cannot be ascribed to ischemia or a conduction abnormality. This also is termed a non-specific T wave abnormality. Non-specific changes may be due to hypertrophy, medications, hyperventilation, hypothermia or a variety of other causes.

XI. Low Voltage Complexes can be caused by pericardial effusion, obstructive pulmonary disease, obesity, diffuse myocardial fibrosis, infiltration of the heart muscle by substances such as amyloid, and hypothyroidism. All complexes in the frontal plane are <5 mm and in the horizontal plane are <10 mm.

XII. Rhythms

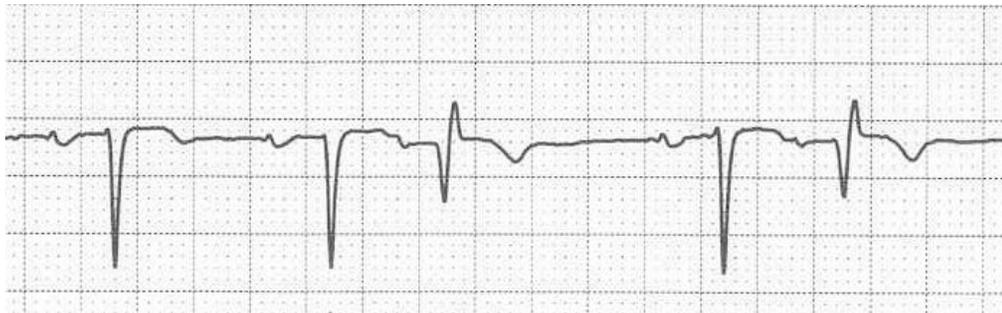
A. Sinus Rhythm Disturbances: Sinus rhythms originate in the sinoatrial node. Diagnosis of sinus rhythms requires examining leads II and aVR for the correct polarity of the P waves. The P wave is always positive in lead II and negative in lead aVR. A P wave will precede each QRS complex, and the P-R interval should be constant.

1. **Sinus tachycardia:** Sinus rhythm with a rate >100 beats per minute. With fast rates, P waves may merge with preceding T waves and be indistinct.
2. **Sinus Bradycardia:** Sinus rhythm with a rate <60 beats per minute.

B. Atrial Arrhythmias: Include premature atrial beats, paroxysmal atrial tachycardia, multi-focal atrial tachycardia, atrial flutter, and atrial fibrillation. Because the stimuli arise above the level of the ventricles, the QRS pattern usually is normal.

1. **Premature Atrial Contraction (PAC):** An ectopic beat arising somewhere in either

atrium but not in the sinoatrial node. Occurs before the next normal beat is due. The P wave has a configuration different from the normal P wave and may even be of opposite polarity. Occasionally, the P wave will not be seen because it is lost in the preceding T wave. If the premature atrial depolarization wave reaches the AV node before the node has had a chance to repolarize, it may not be conducted, and what may be seen is an abnormal P wave without a subsequent QRS complex. Some premature atrial depolarizations may be conducted to ventricular tissue before complete repolarization of part of the conduction system has occurred. In such cases, the subsequent ventricular depolarization may not take the “normal” route giving rise to a wide QRS complex. This is known as aberrant conduction.



PAC with aberrant conduction. The premature p wave may be seen deforming the T wave.

2. Paroxysmal Atrial Tachycardia (PAT): Defined as three or more consecutive PACs. PAT usually occurs at a regular rate, most commonly between 150 and 250 beats per minute. P waves may or may not be seen and may be difficult to differentiate from sinus tachycardia.

3. Multi-Focal Atrial Tachycardia (MAT): Results from the presence of multiple, different atrial pacemaker foci. This rhythm disturbance is characterized by a tachycardia with at least three different P wave morphologies.

4. Atrial Flutter: Instead of P waves, characteristic undulation or sawtooth waves are seen in leads II, III and V1. The atrial rate in atrial flutter is usually about 300 beats per minute. However, the AV junction is unable to contract at this rapid rate, so the ventricular rate is less-usually 150, 100, 75, and so on, beats per minute. The degree of AV conduction is usually described with the diagnosis of atrial flutter using the ratio of atrial to ventricular depolarizations. For example, atrial flutter with an atrial rate of 300 bpm and a ventricular rate of 150 bpm would be described as atrial flutter with 2:1 AV block.



Atrial Flutter. The classic “sawtooth” appearance of the flutter waves is best seen in lead II

5. Atrial Fibrillation: Here the atria are depolarized at an extremely rapid rate, greater than 400 beats per minute. This produces a chaotic pattern instead of normal P waves. Because the AV junction is refractory to most of the impulses reaching it, it only allows a fraction of them to reach the ventricles. The ventricular rate, therefore, is only 110-180



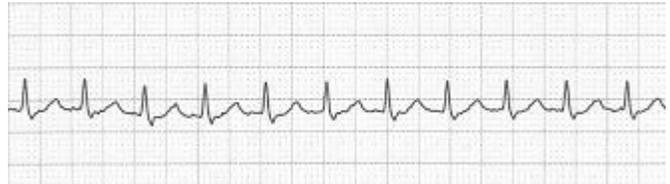
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per minute. Also characteristic of atrial fibrillation is an irregular ventricular rhythm. Atrial fibrillation with a ventricular rate >100 bpm is termed atrial fibrillation with rapid ventricular response.

C. Junctional Rhythms: The three types of junctional rhythms are *premature junctional contractions*, *junctional tachycardia*, and *junctional escape rhythms*. Junctional rhythms arise in the AV junction. P waves, when seen, are opposite their normal polarity. They are called *retrograde P waves*. These P waves may precede, be buried in, or follow the QRS complex. Since the stimulus arises above the level of the ventricles, the QRS complex is usually of normal configuration.

1. Premature Junctional Contractions: Can occur since the AV junction may also serve as an ectopic pacemaker. These are similar to PACs, in that they occur before the next beat is due and a slight pause follows the premature beat.

2. Atrioventricular Nodal Tachycardia: A run of 3 or more premature junctional beats. This is a common form of tachycardia that may result from a “short circuit” within the AV node. It is the most common cause of tachycardia that has a normal appearing QRS complex with no visible P waves.



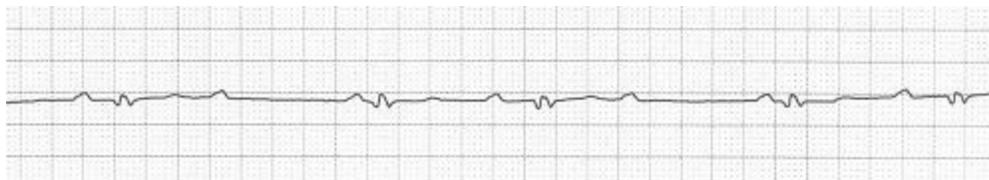
3. Atrioventricular Junctional Escape Beat: An escape beat occurs after a pause in the normal sinus rhythm giving evidence of the subsidiary pacemaker activity of regions of the heart other than the AV node. The diagnosis is established by a lapse in normal sinus depolarization (P wave) with a normal QRS complex (and no preceding P wave) appearing just a little bit later than the P wave would have been expected to appear. Junctional escape rhythm has a rate of 60 beats per minute.



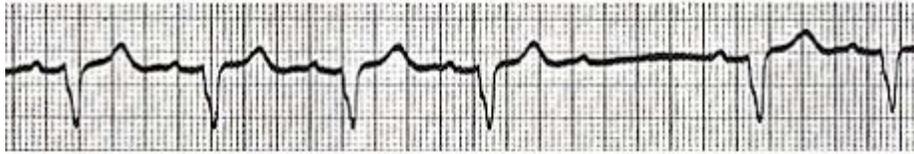
D. Atrioventricular Blocks: Impaired conduction of the atrial impulse to the ventricles. The site of disease may be within the AV-node, His bundle or Purkinje fibers. Heart block occurs in 3 forms: *first degree*, *second degree*, and *third degree*. Second degree heart block is divided into two types: *Mobitz type 1* and *Mobitz type 2*.

1. First Degree Heart Block: a P-R interval greater than 0.2 seconds. The site of disease cannot be identified.

2. Second Degree Heart Block, Wenckebach (Mobitz Type 1): The characteristic ECG is progressive lengthening of the P-R interval until finally a beat is dropped. The dropped beat is seen as a P wave that is not followed by a QRS complex. The site of disease is generally the AV-node.



3. Second Degree Heart Block, Mobitz Type 2: The characteristic ECG picture is a sudden failure of conduction to the ventricles without preceding change in the PR-interval. The site of disease is generally the His/Purkinje system. This type of block usually occurs with more severe heart disease.



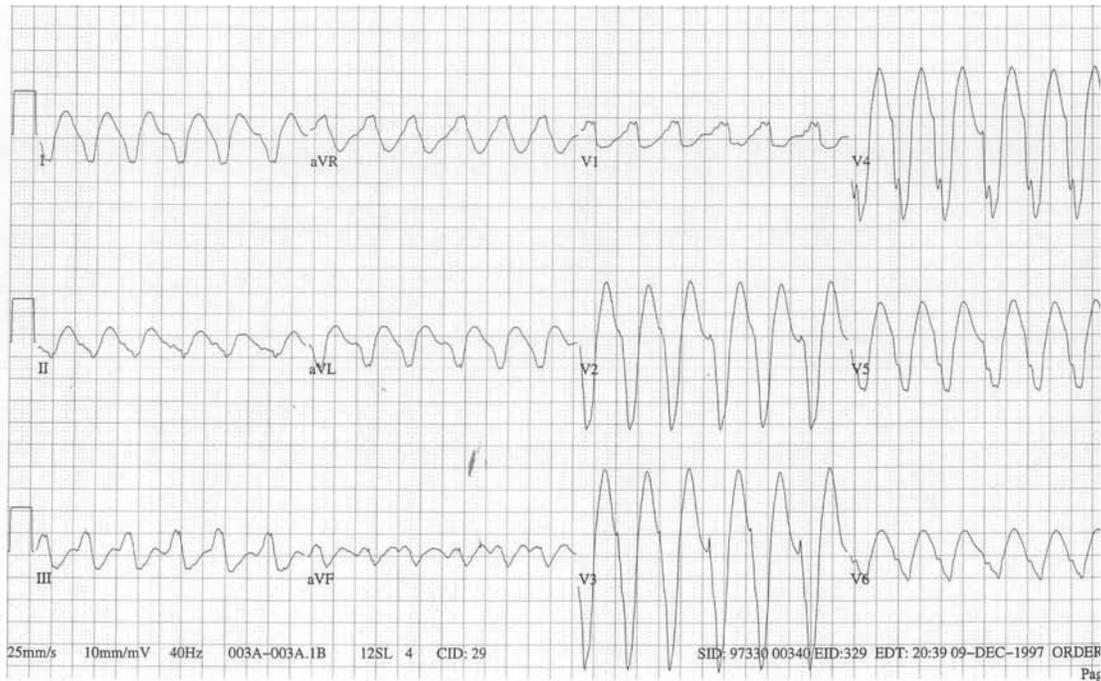
E. Third Degree Heart Block: Also known as: Complete Heart Block. The atrioventricular junction does not conduct any stimuli from the atria to the ventricles. Instead, the atria and the ventricles are paced independently. The characteristic ECG picture is: (1) P waves are present and occur at a rate faster than the ventricular rate; (2) QRS complexes are present and occur at a regular rate, usually <60 beats per minute; and (3) the P waves bear no relationship to the QRS complexes. Thus, the P-R intervals are completely variable. The QRS complex may be of normal or abnormal width, depending on the location of the escape pacemaker.



F. Ventricular Arrhythmias: Ventricular tissue is capable of spontaneous depolarization. When this occurs, a premature ventricular contraction (PVC) is initiated. Because the depolarization wave arises in the myocardium, it usually does not follow the normal path of ventricular depolarization. Therefore, the QRS complex is prolonged and bizarre in shape. Ventricular escape rhythms also occur when both the sinus node and AV-node are being lazy.

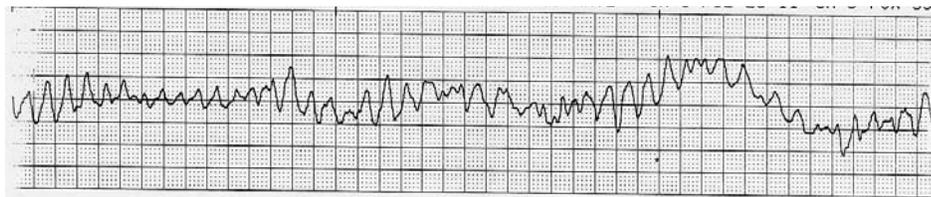
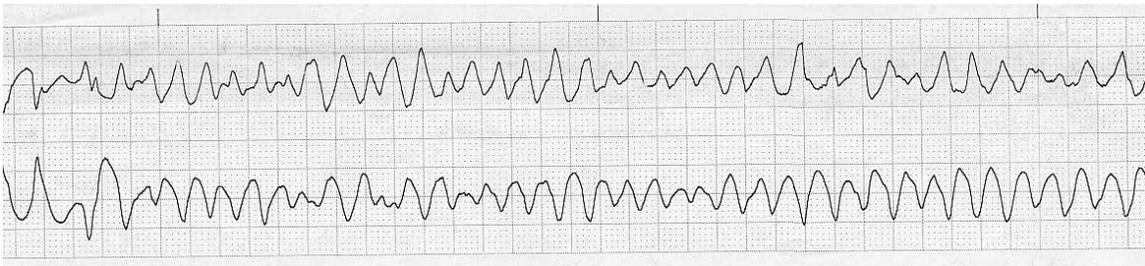
1. Premature Ventricular Contractions (PVC): PVCs are premature beats arising from the ventricles, and are analogous to premature atrial contractions and premature junctional contractions. PVCs have two major characteristics: (1) they are premature and arise before the next normal beat is expected (a P wave is not seen before a PVC), and (2) the QRS complex is always abnormally wide with the T wave and the QRS complex pointing in opposite directions. The PVC usually is followed by a compensatory pause. PVCs may be unifocal or multifocal. Unifocal PVCs arise from the same ventricular site, and as a result have the same appearance on a given ECG lead. Multifocal PVCs arise from different foci and give rise to different QRS patterns.

2. Ventricular Tachycardia: This is defined as a run of 3 or more PVCs and may occur in bursts or may be persistent until stopped by intervention. The heart rate is usually 120 to 200 beats per minute. Sustained Ventricular tachycardia (lasting >30 sec or until therapy is provided) is a life-threatening arrhythmia.



Ventricular Tachycardia. Rate >100 BPM with a QRS complex that is >120 msec in duration and frequently has a bizarre appearance. A “northwest axis”, as shown here, is closely associated with VT.

3. Ventricular Fibrillation: This occurs when ventricles fail to beat in a coordinated fashion and, instead, twitch asynchronously.



4. Ventricular Escape Beats: A ventricular focus may initiate depolarization when a faster pacemaker does not control the rate. They occur after a pause in the regular rhythm. If a higher focus fails to pick up the rhythm, ventricular escape beats may continue. When this occurs, the rhythm is called *idioventricular* and has a rate usually less than 100 beats per minute. The QRS complex is wide and bizarre. Idioventricular rhythms are usually of short duration and require no intervention.

5. Aberrant Ventricular Depolarization: Here the depolarization wave is initiated above the ventricular level and, because it is premature, reaches the ventricles when they are in a partially depolarized state, resulting in a wide QRS complex. The following rules can be used to determine aberrant ventricular depolarization: (1) the beat is aberrant if a P wave precedes the wide QRS complex with an unchanged PR interval, (2) the preceding

R-R interval is longer than the one under study (long-short), (3) most aberrant beats are conducted via the left bundle branch, giving the appearance of right bundle branch block, sometimes termed “Ashman’s phenomenon”.



PAC's are seen conducting to the ventricles with a right bundle branch block appearance or aberrancy.

XIII. The Normal ECG

- A. normal sinus rhythm
 1. each P wave is followed by a QRS
 2. P waves normal for the subject
 3. P wave rate 60 - 100 bpm with <10% variation
 - a) rate <60 = sinus bradycardia
 - b) rate >100 = sinus tachycardia
 - c) variation >10% = sinus arrhythmia
- B. normal QRS axis
- C. normal P waves
- D. normal PR interval 0.12 to 0.20 s (3 - 5 small squares)
 1. for short PR segment consider Wolff-Parkinson-White syndrome or Lown-Ganong-Levine syndrome
 2. for long PR interval see first degree heart block
- E. normal QRS complex
 1. < 0.12 s duration (3 small squares)
 - a) for abnormally wide QRS consider right or left bundle branch block, ventricular rhythm, hyperkalaemia.
 2. no pathological Q waves
 3. no evidence of left or right ventricular hypertrophy
- F. normal QT interval (Calculate the corrected QT interval (QTc) by dividing the QT interval by the square root of the preceding R - R interval. Normal = 0.38-0.46 s.)
- G. normal ST segment no elevation or depression
- H. normal T wave both contour and direction.

XIV. Approach the tracing with a method

- A. RATE
- B. RHYTHM/REGULARITY
- C. PQRST INTERVALS
- D. AXIS (P, QRS, ST-SEGMENT, T-WAVES)
- E. HYPERTROPHY AND BLOCK
- F. INFARCTION/INJURY AND REPOLARIZATION
- G. DRUG/ELECTROLYTE EFFECT